Edward Munro
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SAINT BARTHOLOMEW'S HOSPITAL REPORTS.

ARTICLE I.—*Pathological Anatomy of the Kidney.*

By REGINALD SOUTHEY, M.D. OXON.

THE structures of which the kidney is composed are a stroma of areolar tissue, a tubular gland system, bloodvessels adapted to a peculiar plan, nerves which accompany the bloodvessels, and lymphatics; the whole is enclosed in a fibrous capsule of connective tissue, interspersed with numerous elastic fibre filaments.

Each or all of these parts may become the seat of morbid changes, and be altered in such a manner as to abrogate or impair the functions of the gland. If it be important to understand how these structures are knit or combined together towards making any guess at the physiology of the organ, we may be assured that the vexed questions raised by its very diverse diseased appearances could not be set at rest without a most minute examination of the several component parts.

For the most part it may be strictly asserted, that morbid changes which commence in any one of the kidney's structures confer a special stamp upon the organ; still, in the natural train of pathological events, one tissue implicating another, the appearances finally attained may come to demonstrate great inter-resemblance. It remains for us only to premise, upon the good faith of our own observations, that the same goal can be reached by different roads; it will be our endeavour, as it is our task, in the following pages to show what these are, and how they lead to it.

Interstitial Disease * may go right through the kidney, its nidus, the stroma or connective tissue, extending everywhere in it. We shall consider it under three heads: *Hypertrophy*, *Hyperplasy*, Simple Fatty *Degeneration.*

Hypertrophy.

This is a simple increase in quantity of the intercellular elements or substances; it occurs not without some increase in the number of the cells, but this increase is proportionally slight. The cells remain very indistinct, the tissue presents its normal fibrillation, but its substance is thickened and more opaque than it should be; the distances between the elongated nuclei are abnormally great. If this change extend equally, and in all directions, the state of sclerosis is produced, the new substance coming to encroach upon and prevail over all the other structures. Now, as this tissue knits the capillary walls together, forms the outer sheath of the vessels, and passes over the Malpighian bodies as their enveloping capsules, it is easy to see that its mere thickening must interfere with the nutritive changes; but, after it has multiplied in its intercellular elements, a secondary change or contraction of these invariably follows, the afferent vessels are pressed upon, the capsular tufts collapse, the glomeruli degenerate; so do the contents of the tubules; the parts are shrunk and pitted, finely granular spots raised over above the rest marking the relics of the Malpighian bodies.

It is self-evident that such a change can be general or partial, primary or secondary.

If it be general and primary, at the stage when the Hypertrophy is greatest, and before contracture has set in, the kidney is found rather larger than it should be, denser, and tougher, taking the impress of the nail with difficulty, generally pale and transparent-looking, scarcely to be distinguished from amyloid disease, unless a fine section of it be submitted to the iodine test under the microscope; but at a later stage this kidney is tough, hard, and small, covered with a thickened and adherent but smooth shining capsule. Occurring partially, and

* To a proper comprehension of the morbid alterations affecting the stroma of this organ, a clear understanding upon the ultimate elements composing this tissue is indispensable; it consists of cells and intercellular substance; the cells are stellate, angular, or fusiform, and each one contains one elongated nucleolated nucleus; the cells often anastomose by their ends; the intercellular substance is the excretion or appurtenance of the individual cells.

The intercellular substances are not definitely limited, but merge into each other, and obtain an aspect of finest possible fibrillation when observed beneath high microscopic powers.

in the cortical portion, it may be regarded as strong evidence of past glandular affection, to which it is a secondary and re-cuperative stage. The same interstitial change may take place partially again in the tunica propria of the tubules in the papillæ, as a consequence of the irritation that arises from chronic catarrh of the bladder. A still more partial interstitial growth of a portion of the stroma in these parts, where it is naturally very thick, leads to the formation of those little fibrous tumours which are not at all uncommonly found in the medullary portion. They are apt to be mistaken for tubercle, but are usually of larger size, and present a more opaque white appearance than appertains to tubercular growths.

Occasionally these fibrous tumours completely fill a papilla, which then looks quite white, and is altered in length and shape, being contracted in its long, and correspondingly increased in its broad diameter.

We have found them also higher up in the cortical portion, but always in the straight tube territories.

Cellular Hyperplasy.

Under this head, we consider those changes which take their origin from the cells of the connective tissue.

The condition of fine fibrillation in the intercellular elements is not properly attained; the cells multiply by rapid division of nuclei at an early stage of their development, so that this tissue becomes filled with nuclei. These nuclei may subsequently pass into pus cells or fattily degenerate. Hyperplasy, too, may be general or partial; and, if general, it will correspond to such a condition of affairs as is understood by acute or active inflammation, leading to rapid pus formation and an entire destruction of the organ. This, however, is very rarely a primary disease, but is usually the result of the irritation produced by decomposing urine, which has distended pelvis and tubules, after an obstruction of the ureter by an impacted calculus.

As a partial affection, this multiplication of nuclei in the interstitial stroma, is the common complication, or secondary consequence, of changes which have commenced in the glandular elements; indeed, in the retrograde metamorphoses of catarrhal nephritis, when the affected structures degenerate into fat, both the glandular and interstitial elements course through identical stages to one common disorganisation.

Simple Fatty Degeneration.

Without experiencing any previous increase of its cells or intercellular elements, the connective tissue may fattily degenerate.

Of simple degeneration, we have nothing further to say here, than that we believe it to ensue only secondarily to some morbid change which has originated elsewhere.

The distinction made by us, between interstitial hypertrophy and interstitial hyperplasy, is of some importance, for attributing, as we do, both errors to some morbid stimulus or irritation, thus much is certainly observable, that, irritations which are chronic in their character, slight in degree, but long prolonged, induce true hypertrophy, or increase of intercellular substance; whereas those which are of an acuter kind start more rapidly disorganising alterations, such as those in which the cells are multiplied at the expense of the stability of the tissue.

Towards recognising alterations in the interstitial tissues, it is necessary that fine sections of the kidney should be made with a Valentin's knife across the bases of the pyramids, cutting the tubes transversely, and then again somewhat higher up in the cortical substance; the epithelium must be gently brushed out of the tubes with the aid of a camel's hair brush, and after being shaken about in a little water, the section can be examined under the microscope.

The points to be made out are, whether the increase of adventitious tissue appertains to the sheath of the vessels, to the capsules of the Malpighian bodies, or to the membrana propria of the tubules. The part most commonly first attacked in true chronic interstitial nephritis is the capsule of the Malpighian bodies, and the external supporting sheath of the vasa afferentia, whereas the tissue lying between the straight tubes, in the pyramidal portion, is much more often the seat of intercellular increase, as a consequence of repeated chronic catarrhal attacks.

If fatty degeneration has set in, it will be necessary to determine whether this is confined to the intercellular tissue, in which case a fine network of little opaque white dottings will be seen to run between the vessels and the tubes; or whether the tubes themselves are implicated, and also filled with large opaque masses, consisting of fat cells and granular glandular debris.

Under the head of Interstitial Changes we must further classify such new growths as tubercle and cancer, which appear to take their commencement in the interstitial stroma of this organ.

The pathological changes which commence in the vascular apparatus are:—

Thickening of walls (simple, amyloid).

Dilatation of vessels (simple, degenerative).

Obstructions of vessels (embolism of arteries, thrombosis of veins).

Simple Thickening of the External Walls of the Vessels.

This change is especially liable to be confounded with that increase of intercellular tissue which is due to interstitial nephritis. Upon transverse section the vessels appear more widely separated than they should be; now, unless we are able to satisfy ourselves, by examining an injected specimen, that this increase of connective tissue has taken place only upon the outsides of the smaller bloodvessels and of the capillary walls, and that there is no thickening of the membrana propria of the tubules, it will be impossible to arrive at any differential diagnosis.

Amyloid or Waxy Thickening.

Contrary to what might have been expected from the aspect of amyloid disease in the liver and spleen, in which organs this change can generally be examined from very characteristic naked-eye appearances, amyloid degeneration in the kidney is apt to escape observation, unless very particularly inquired for; we do not speak here of the advanced stages which are sufficiently easily recognisable, but of the early ones, when a superficial examination is often very delusive.

The kidney may be firm, level, smooth, rather large, the capsule non-adherent, the tissues somewhat over-translucent, and yet there may exist no amyloid change whatever in the vessels; again, it may present no apparent trace of disease, and yet prove amyloid in high degree. It may have every look of having been the seat of old glandular disease, be small, granular-looking, with an adherent capsule, and an irregularly pocked surface, with diminished cortical, and vascular and drawn-up

medullary portions, yet still be the seat of extensive amyloid degeneration.

The chemical composition of the lardaceous material we shall not discuss here; it is formularised by Schmidt * and Kekulé † thus:—C–53·58 H–7·00. N–15·4; let it suffice that it is a strictly albuminous compound, akin to vegetable cellulose only in appearance.

The albuminous degeneration, then, takes place first in the muscular coat of the afferent arteries, as these run to the Malpighian bodies; it next proceeds to involve the glomerulus, the capillary tuft in its interior, and finally the efferent vessel and vasa recta. But it may take a different course, occasionally implicating the vasa recta only. We have observed it affecting both afferent vessels and vasa recta simultaneously. Most rarely it is seen to be confined to the glomeruli alone.

The middle portion of the kidney (the neutral zone of Virchow, the intermedullary and cortical part), is that which is usually least, and certainly last, interfered with by this change. Should the degeneration go on, the membrana propria and the cells of the tubules are subsequently attacked; beyond this point it does not advance, the interstitial stroma remaining untouched, although usually increased in quantity.

The common course of the disease is for the walls of the arteries to thicken at the expense of their calibres, the capsules of the Malpighian bodies thickening and their contents appearing less nucleolated than is their wont. It is difficult to say whether they become larger or smaller, for they vary much in size at all times: my belief is that they are larger than normal; but the conspicuous feature about them is their glittering transparency; they stand out like tiny glass beads, and have quite lost their accustomed carnation pink hue. Supposing, as is by far most often the case, that it is the afferent vessels and the glomeruli which are thus encroached upon, it must be self-evident that an interference with the circulation of the blood in the kidney must ensue. The hindrance to the passage of the blood through the cortical portion is manifested by its yellowish-white translucent look, against which the vascularity of the medullary cones stands out very prominently, the vasa recta bundles are enlarged and distended, and the renal veins at the basis of the pyramids show very marked engorgement. It is by no means a rare thing to find the uriniferous tubes between the vasa recta bundles filled with extravasated blood;

* Schmidt, Ueber die chemische Const. des Thierischen Amyloid. (Annal. d. Chemie u. Pharm. vol. lx. p. 250. 1859.)

† Kekulé, Verhand. des Naturhist.-Med. Vereins, B. v. S. 144. Heidelberg, 1858.

indeed, the albuminuria accompanying this affection, and the epithelium and blood cells which escape into the urine in it, may fairly enough be attributed to a transudation from increased pressure, such as the intertubular capillaries in the medullary portion are inevitably subjected to. The circumstance of the blood still reaching the vasa recta, although it pass so scantily through the Malpighian bodies and rete corticale, is explicable upon those principles of collateral flux which have already been described in a former communication to these Reports (vol. i. p. 130). An important point is further illustrated by this form of pathological error—one, too, which holds good of all those changes, whether glandular or interstitial, that interfere with the forward current of the blood through the kidney—viz., that whatever diminishes the 'vis a tergo' tends to make the blood stagnate in this long and double capillary system, and therefore to accumulate in the renal veins.

Dilatation of Vessels.

Simple dilatation of the arteries needs no very special description. It has two main sources of origin—

1. Active, undue pressure,* such as is produced by a watery blood, or an hypertrophied left ventricle with deficient aortic valves, or any obstruction to the onward current of the blood in the aorta, beyond the giving off of the renal arteries (collateral flux, in a word).

2. Passive dilatation, such as can be due to a paralysis of the muscular coats of the arteries, or to dilatation with engorgement of the veins, such as may be produced by any hindrance to the current of venous blood between the renal veins and the right heart. The enumeration of the nature of such obstructions belongs more to the province of clinical medicine; we shall only specify those which we have had particular occasion to observe—abdominal tumours, aneurism of aorta pressing upon the vena cava, emphysema of lungs, cirrhosis of lungs, pneumonia, pleuritic affections, morbus cordis.

Dilatation with Degeneration of Vessels.

Like all degenerations, chronic but progressive in its course, arterial sclerosis, or atheromatous disease, may extend into the renal artery from the aorta, implicating in the first instance

* We can only further adduce from our own observations, that in almost all those cases in which fine sections of the kidney have revealed arterial dilatation, that the interstitial tissue has also been increased in quantity, the Malpighian bodies have been enlarged, and their interiors often filled with extravasated blood.

always the inner coat of the vessel. The aorta, however, is not invariably primarily diseased; the kidney arteries may be thus attacked alone. In some cases this affection begins in the afferent arteries, and extends forwards and backwards from them; the nuclei upon the capillary walls increase in number and size; then ensues a lengthening or enlargement of the loops of the tufts in the interiors of the glomeruli, so that they fill the Malpighian corpuscles more completely, and even distend them. Fatty degeneration commences by minute dottings between the nuclei; the walls experience a granular change as apart from their normal transparency; then the muscular coat is implicated, and we have all the ordinary train of pathological alterations induced by atheromatous changes, aneurismal dilatations from yielding of the walls, or the most extreme stenosis from calcareous deposition upon them.

Obstructions of Vessels.

The cause just mentioned, namely, atheromatous disease, may primarily or secondarily lead to the obstruction of the renal artery or its branchings.

1. The stenosis may reach the point of complete obliteration of the lumen.

2. The fragments or debris, from a similar change elsewhere, may be swept forward with the blood-stream and find lodgment in the renal vessels.

Embolisms, however, into the kidney, as into the spleen, take origin most frequently from the left heart, as fibrinous deposits consequent upon endocarditis: indeed, after the spleen, the kidney is the most common organ in which such embolism is found.

The pathological appearances due to the blood stasis vary according to the state of health of the individual in whom they occur, according to the nature of the foreign particles by which they are produced, and according to the size of the vessels which are thus obstructed.

Embolism supervening upon active endocarditis, or as an accident, in the course of acute sthenic disease, excites proportionably great disturbance; the irritation produced by the foreign body is great in the degree in which the parts about it are irritable, and the tissues run through successive changes towards pus formation the more rapidly. It is easy to foresee, that if the blood be in a state which renders the nutritive changes at the capillaries generally defective, the gravamen of an entire local defection will be more seriously resented by parts

around them, which are at best scarcely capable of preserving their own nutrition. This is neither more nor less than the failure of that reparative power in store implied by a state of perfect health. It is imperfect health, the strict logical contradiction which the term predisposition to disease compels us to adopt.

In place of congestion, or intense hyperæmia, which occurs as a halo round the stasis spot in a healthy animal that has been experimented upon, hæmorrhagic extravasation, or ecchymosis, commonly distinguishes embolism in the human kidney. If the vessel thus plugged be a very small one, the consequence is a tiny purple ecchymosis, like those so often seen upon the retina with the ophthalmoscope—little dottings, whose centres, first, and then whose substance become metamorphosed into a yellow mass. If the involved vessel is larger, the extravasation is more extensive in the first instance.

Such finer vessels will be found to be afferent arteries in the cortical portion, or perhaps special separate branches to the tissues about the bases of the pyramids. It is the obstruction of a main cortex or lobule branch, or vas ascendens, which originates those wedge-shaped masses that particularise the embolisms of this organ; why they are wedge-shaped is explained at once, when the course and distribution of these branches in the cortex is considered. The later changes are quite identical with those that occur in the smaller patches, and differ only through being upon a larger scale; the normal structures are lost, the ordinary pigmental alterations take place in the extravasated blood, and the centres soften into a yellow cheesy mass, which has frequently been mistaken for dried-up abscess. Finally, the parts shrink partly in consequence of the collateral hyperæmia, which swells the surrounding vessels, partly from the contraction of the interstitial tissues; then, if the patch have reached the surface, this is seen to be depressed and of a pale yellow colour.

The colouration of the spots is at times of a more dusky red, and, in place of extravasation around them, actual necrobiosis can supervene. For the most part, however, the infarcta shrink and become atrophied, heal but do not repair, while a sort of compensative hypertrophy ensues in the untouched lobules around them; this, of course, only in the event of life being long prolonged after their occurrence; and, finally, the embolised portions sometimes long afterwards, after being petrified with lime deposits and encysted, become 'irritamenta' of further 'malorum' in exciting future nephritis.

The immediate effect of an embolic plugging in any lobular

branch will be to congest other parts of the same organ by collateral fluxion; and if the obstruction be a large or important one, capillaries are often burst by the sudden and increased strain to which they are subjected. Hæmaturia occurring suddenly in the course of endocarditis is no such rare clinical event, and should at once awaken the suspicion of some such accident having befallen the kidney.

Thrombosis or Clot Formation in the Renal Veins.

This arises from one of two causes, or from both together.

1. Mechanical retardation of the blood-stream.
2. A cachectic alteration in the blood itself.

The sources of mechanical retardation are to be found in diminished 'vis a tergo,' such as interstitial or glandular disease, or amyloid degeneration, or extensive embolism can produce, or in obstructions 'a fronte,' such as originate from the further extension of clots commenced elsewhere in the vena cava. Clinical experience has shown us, that the pressure of tumours upon the vena cava more commonly determines such blood-clottings.

The cachectic alterations in the system which induce thrombosis are ichoræmia, puerperal fever, infantile marasmus.

Thrombosis of the renal veins is very rarely quite entire, a large part of the lumen of the vein may be obstructed, but still some bye-channel beside the clot usually allows some portion of the blood to pass away.

The consequence of this plugging is extreme venous hyperæmia of the kidney, reaching perhaps to the extent of extravasation, the blood then coming to escape into the urine tubes, the urinary secretion is probably diminished proportionately to the venous congestion of the organ. It is very rare for both renal veins to be thus occluded together; in the cases observed by Beckman * of 10 infants, who died marasmatic, with profuse diarrhœa, the plugging appeared to have begun in the large venous branches between the cortex and medulla; in no case did the clots extend so far as the vena cava, the left renal vein was that most frequently thus obstructed. The clots themselves may subsequently become the seat of carbonate of lime deposits, but of course only in those cases where the blood has found sufficient passage to admit of the kidney resuming its functions.

* Beckman in d. Verhandl. d. Wurzburger Physik in Med. Ges. Bd. ix. p. 201.

Glandular Disease.

Before entering upon the description of those pathological changes which have their seat and origin in the glandular structures of the kidney, it will be necessary for me very briefly to refer to the plan or order which prevails in the arrangement of the urinary tubules. The histology of these parts has already been discussed at p. 172, vol. i. of these Reports.

Speaking roughly, for purposes of general reference, the medullary portion of the kidney consists of straight tubes of various diameters arranged in bundles, having other bundles or tufts of blood-vessels between them. The cortical portion is composed of the further separation from each other, by means of their own tortuous prolongations, of the same bundles of straight tubes, which stretch up through the cortex to close beneath the kidney capsule.

We shall hereafter speak of the bundles of straight tubes in the cortical portion, as the straight tube territories, and of the alternating columns of curling tubes as the tortuous tube territories: it is in these last that the Malpighian bodies are always found.

In order to fix our landmarks still more precisely, it is advisable that we should pursue a plan first proposed by Professor Virchow, which consisted in further making three zones, taken breadthwise (Zonen der Verbreitung, as he termed them), that should transect the straight tubes, thus again dividing the kidney into three arbitrary sections.

1. An upper zone, formed by the upper two-thirds of the cortical portions terminated below by the anastomotic arches.
2. A middle or neutral zone, comprehending the origins of the vasa recta, including the upper one-third of the medullary portion, where these vessels run in bundles together, and terminating where their distinct brush-like appearance is merged and lost in their capillary distributions.
3. A lower or papillary zone, comprising the lower two-thirds of the medullary portion and terminating in the calices.

The upper might be called the glandular or secretory zone, the middle the vascular zone, the lower the excretory zone.

We shall treat first of those changes which especially affect the membrana propria of the tubules; secondly, of those which take their point of departure in the cells which line the tubes; and, thirdly, shall describe the site and nature of those various inorganic salt deposits which are also found in the tubes.

Cystoid Degeneration.

In a healthy state the membrana propria is very fine and transparent; the first change towards cystoid disease observable in it, is that its wall thickens; the tubule becomes larger, and at the same time varicose and beaded, or bulged and constricted, both above and below the bulging at pretty regular intervals the constricted walls close in, the opposite sides unite, and thus the tube comes to be divided off into a number of closed sacs or little cysts.

This change is almost invariably observed to take place in straight tubes, and therefore in straight tube territories; it affects a great many tubes at the same time, and they are always those which are lined with a distinct and well-formed epithelium, and this epithelium lining is quite traceable when the cysts are first completed; we further believe them to occur only in Henle's down-looping tubes. The cysts which are thus formed in a portion of a tubule do not enlarge equally; some quickly fill, and become distended with a yellow gelatinous substance, while others shrink and atrophy.

These cysts, then, are found in various parts of the kidney; in their early stage they can be teased out of the surrounding tissues so as to demonstrate their origin from the tubes; they may often be seen upon the surface of the kidney, where they frequently attain considerable size, and are distinctly traceable to the confluence of several contiguous sacs; such multilocular sacs are also occasionally observed to reach some size in the papillæ, pushing aside and displacing the tubules among which they lie.

The one point of interest about them is their contents, and the striking similarity which these bear to the so-called fibrinous cylinders, as they can be seen in situ in the tubules, or found in the urine; they are homogeneous, consist of a shining gelatinous colloid-looking material, and contain chemically a mucinous substance, combined with some low form of albumen; the opinion of many is, that they are formed by a metamorphosis of the epithelioid elements.

Clinically, it is well known that urinary casts resembling them are often found in acute pneumonia, and it is certain that these are alone no evidence of Morbus Brightii.

There are two forms of these cysts, the one above described, with thin walls, which after awhile become atrophied; and the other congenital, and having firm thick walls (which must be contra-distinguished, again, from hydronephrosis); both may

contain urine in place of the more usual albuminous fluid; and the urine sometimes decomposes, and sets up such an irritation as may excite abscess. The ordinary course, however, as previously mentioned, is, for these cysts to atrophy and degenerate when they have reached to about the size of a horse-bean, and never to give rise to any disturbance whatsoever of the kidneys' functions.

Changes in the Glandular Elements.

Catarrhal.—The urinary tubes do not usually become affected by that group of pathological symptoms which we together denominate catarrh, as a primary disturbance; indeed, just as a bronchial catarrh, beginning at the larynx, extends through the pulmonary mucous tract until it involves the uttermost alveoli, so a gonorrheal catarrh of the urethra, or a prostatitis, or a vesical catarrh, proves to be by far the most common cause of catarrhal changes in the tubuli uriniferi. Disease is much more apt to be conducted up to the glandular elements than to originate in them; but it certainly may also commence at the glomeruli, and in the glandular tubes proper, as contra-distinguished from the excretory tubes, being produced in these parts by the direct excitation of poisoned blood, or too highly stimulating diuretics.

We have, therefore, to consider the catarrhal affection of the kidney from two points of view; the first, regards that implication of the tubular linings by direct continuity of disease, stretching from below upwards, which resembles capillary bronchitis so closely; while the second takes account of an original change or error impressed upon the gland cells in their tortuous or secretory portion (for analogy, much more comparable to pneumonia), and which is conducted from above downwards into the excretory tubules.

With careful examination, the pathological appearances enable us to distinguish after death what course the disease has taken. It is impossible to describe the catarrhal changes from below upwards by any one set instance; the general nature of the cases, used for purposes of illustration, are those in which death has ensued, sooner or later, after the establishment of vesical catarrh, as the secondary consequence of lesions of the brain or spinal cord.

In the very first stage the mucous membrane of the pelvis of the kidney is swollen and red, generally hyperæmic, dotted here and there with spots of ecchymosis, which are usually situated in the centres of radiating and highly injected capillaries, the

apices of the pyramids may be caught darkly congested; this, however, is only a very transitory condition, for very shortly the epithelium lining the large excretory ducts swells up, so that the tubes, distended by their contents, squeeze the blood out of the fine capillaries which lie between them; the mamillary processes then become quite white.

The straight tubes continue to swell in all their length, the bundles of them stand out as widely prominent opaque white streaks, contrasting very markedly against the red bundles of vasa recta lying between them; thus it is, that the middle or neutral zone obtains that red and white striped look which is so highly characteristic of this stage of the affection.

The tubes are not merely swelled by an excess of cell contents, but the cells themselves, examined under the microscope, are individually larger, more granular than they should be, and often loosened from the membrana propria of the tubules. Upon pressing the pyramids, a greenish white secretion, consisting of casts, free epithelium, and mucous cells, can be squeezed out of the excretory orifices.

Let the disease extend up into the territories of the tortuous tubes, and we have still the same influences at work which have already been demonstrated as occurring below, the tubes swell and press the blood out of the capillaries between them, and the parts become white and opaque-looking to the naked eye: the glomeruli may be congested from the pressure, thus preventing the flow of blood through the efferent vessels, and here and there capillaries may have burst, and the colouring matters of the blood have passed into the tubes; but the prominent features presented to us in the section of such a kidney, are its larger than normal size, its greater than normal weight, the whiteness of its secretory zone, the redness of its vascular or middle zone, the pallor of its lower or excretory zone; venous congestion may be further marked by the stellate appearance of the superficial capsular veins.

If a moderate-sized section of the cortical portion be examined more closely, the interstitial tissues will be found soft and serously infiltrated, the straight tube territories will be disproportionately large and bloodless as compared with the tortuous tube columns.

As good an example as could be selected for demonstrating the naked-eye aspect of a kidney which has recently, during life, been the seat of catarrhal changes beginning at the Malpighian bodies, is presented to us in persons who have died of cholera.

The blood here is suddenly subjected to so grave a water-drain, and becomes so thick, that it circulates with great diffi-

culty through all the capillaries of the body; it stagnates at the Malpighian bodies just as it stagnates everywhere else. The 'vis a tergo' fails more and more as the heart's muscular nutrition is interfered with, and its powers are unduly taxed. The urine is suppressed.

After death the kidney is found highly venously congested; its substance, in the cortical portion especially, is soft and œdematous; patches of stellate veins stand out upon the capsular surface; the papillary zone is white, an exudation of milky juice, consisting of mucinous casts and entangling blood, and epithelium cells can be forced out of the mamillary openings, the vascular zone is dark red; the bundles of tubes between the vasa recta are often blood-stained, but otherwise opaque-looking and swelled; in the upper or secretory zone the capillaries in the straight tube territories are observed to be injected and distended, although nearly, if not quite, empty in the tortuous tube districts.

The tortuous tubes themselves are shining, of a yellowish-white colour, distended with gland cellular contents, crowded with urinary solids, and in part filled with fattily degenerate cells. Here and there, to make the contrast doubly striking, ecchymoses may have taken place from bursting of the distended capillaries between the straight tubes, or from transudation of the blood in these districts.

The prominent features, then, by which catarrh, beginning in the tortuous tube territories, can be distinguished from that which has crept up to these parts from below, must be sought in the upper cortical or secretory zone.

Is the appearance streaky?—we must inquire further into which set of capillaries are congested. Are they the long meshes between the straight tubes?—then the swelling cannot have taken place here first, and the catarrh can only have spread from above downwards. Are they the close meshes of the tortuous tubes?—then the glandular swelling has travelled up from below to these parts, has congested them by interfering with the efflux of blood from them, or else has extended so far up into them, as to make them participate in the parenchymatous swelling, the general pallor and opacity, which has pervaded the cortex.

Article II. — *On Congenital Cystic Tumour.*

By Thomas Smith.

THE disease to which this paper relates has been termed congenital cystic hygroma, and has been well described under that name by Dr. Adolph Wernher;* while in this country, the term congenital hydrocele has been applied to one form of the same disease.

Cases of a similar or identical affection in adults have been recorded by Professor Manoir, of Geneva;† by Mr. Lawrence;‡ by Dr. O'Bierne;§ by Delpech and Mr. Paget;‖ while Von Ammon;¶ Otto;** Henke;†† Meckle;†† Sömmering;‡‡ Volkers;§§ Ebermeyer;‖‖ Nicod. d'Arbent;¶¶ Droste;*** and others, have described the disease, as it occurs, as a congenital affection; and have given copious information on its pathological significance. Their knowledge, however, having been chiefly obtained by the post-mortem examination of still-born infants and fœtuses, there is little to be found in their writings bearing on the surgical aspect of the disease, and nothing to guide one to the correct diagnosis of the affection in living children, or to its successful treatment.

The surgery of this disease has received more attention from Dupuytren, Wurtzer, Wernher, Gurlt,††† Storch,‡‡‡ Guersant;§§§ but, as Wernher remarks, the recorded cases are hitherto too scattered to admit of eliminating from experience any well-grounded plan of treatment.

The disease, as an affection of infants, was, I believe, first, or at all events, best described in this country by Mr. Cæsar

* Die angeborenen Kysten-Hygrome. Von Dr. A. Wernher. Giessen, 1843.
† Mémoires sur les Amputations: l'hydrocèle du cou. 1815.
‡ Med. Chir. Trans. vol. xvii.
§ Dublin Jour. of Med. and Chir. Soc. vol. vi. p. 834.
‖ Surgical Pathology, p. 398.
¶ Die angeborenen chirurg. Krankheiten. Tab. xiii. Fig. 1, 2, 3. Tab. xxxiii. Fig. 7, 8, 9, 10.
** Monstrorum sexcentorum descriptio. Nos. 576, 577, 578, 579, 580, 581, 582.
†† De tumoribus fœtuum cysticis. Halæ, 1819.
‡‡ Abbildung und Beschreibung einiger Missgeburten.
§§ Hygroma cellulosum am Halse eines Neugeborenen. 1837.
‖‖ Casper, Wochenschrift für die gesammte Heilkunde. 1836.
¶¶ Bull. de Thérapie, tome xix. p. 54.
*** Hygroma celluloso-cysticum am Halse eines Neugeborenen. Hannoversche Annalen, 1839, p. 295.
††† Cystengeschwülste des Halses. Berlin, 1855.
‡‡‡ Journal für Kinderkrank. 1861. Band xxxvii., S. 68.
§§§ Notices sur la Chirurgie des Enfants. Dixième fascicule. Paris, 1864.

Hawkins, in a paper on a peculiar form of congenital tumour of the neck.* More recently, Mr. Holmes has written on cervical tumours of this kind, and has related three cases that occurred under his own care.†

Congenital Cystic Tumour, as I propose to call this disease, is a tumour formed of an admixture of cystic and solid substance, in varying proportions.

The cystic element consists of one or more circumscribed cavities, having generally thin walls, and containing either a clear serous fluid, a brownish-coloured serum, or serum stained to a greater or less degree by admixture with blood. More rarely the fluid is of a grumous gruelly consistence, and of greater density.‡ The walls of the component cysts in a large growth of this kind often differ much in thickness, and in the degree to which they are distended by their fluid contents. Thus while the larger cysts may be flaccid and thin-walled, the smaller ones, in the same growth, may be thick-walled and tensely filled with serum; so as to communicate to the touch the sensation of solid substance. In a large growth the cysts often vary much in size; *i.e.* from the size of a pea to that of a large orange. The inner surface of the cyst walls is generally smooth, glistening, and finely fibrous in appearance.

The solid element which, together with the cysts, makes up the tumour, is a species of connective tissue; being tough, succulent, and fœtal in its character; having the appearance and consistence of the tissue surrounding the vessels in the umbilical cord.

These tumours may surround, and therefore contain within them, lymphatic glands, as in a case observed by Mr. Hawkins; but the glands in such cases are only incidentally involved. One case is recorded by Otto, where firm fleshy masses were found within the growth.§ The same condition was met with by Henke; in both these instances, however, the growth was situated in the perineal region, where often the solid element of these tumours predominates over the cystic.

Congenital cystic tumours are, I believe, always situated beneath the muscular fascia, and are—on their superficial aspect at least—generally freely movable beneath the skin to which they are unadherent. Their deeper connections are, however, for the most part extensive and firm; often insinuating themselves both widely and deeply beneath the muscles, and around the deep vessels of the part. In a case, related by Mr. Hawkins,

* Med. Chir. Trans., vol. xii. † Lancet, 1864, vol. i. p. 577.
‡ See a case by Otto, Seltene Beobachtungen, ii. p. 65.
§ Seltene Beobachtungen, ii. p. 165.

the growth surrounded the portio dura and the external carotid; all the vessels and nerves at the base of the jaw were involved in some of the cysts, which also surrounded and separated the common carotid, jugular vein and vagus nerve; while some of the growth was found extending in front of the spine behind the pharynx and œsophagus—from the base of the skull to the sixth cervical vertebra.

The external appearance and characteristics of these tumours are easily recognisable, and there is little chance of an error in diagnosis if the cyst be unilocular—a condition, however, quite exceptional. More frequently the growth is unevenly elastic, and lobulated on its surface; in parts, it is soft and compressible, and elsewhere it may feel as if it contained solid masses or lymphatic glands. If any of the cysts are near the skin and their walls are tense, they may communicate to the latter a bluish tinge, like that of a subcutaneous nævus; or the more prominent parts of the growth may be evidently translucent.

From the lobed character of many of these tumours, their consistence, and the occasional dimpling in of the skin over them, they often bear a great resemblance to fatty growths. It may be still more difficult to distinguish them from large subcutaneous nævi; both on account of their general characters, and from a venous staining of the skin, which not unfrequently exists; while the resemblance may be much increased by the swelling up and turgescence of the growth when the child screams or struggles—a symptom particularly noticeable in congenital cystic tumours of the neck.

In a doubtful case, the existence of a nævus growth can generally be determined by observing the effect of firm pressure; which, while it produces some amount of collapse when applied to a nævus, has no such effect on a cystic tumour.

It often happens that these tumours are placed over the median line of the spinal column, where, from their evidently fluid contents, it is difficult to distinguish them from spina bifida. I believe the most reliable symptoms on which to found a diagnosis—in a doubtful case of this kind—to be, 1, the healthy condition of the skin over the tumour in cystic disease as distinguished from the discoloured and often highly attenuated integuments over the sac of a spina bifida; and, 2, the absence of any signs of deficient innervation in the trunk and lower extremities. I have had two such cases under my own care—one in the cervical region, where I relied on the signs above-mentioned to establish a diagnosis; and a second case, where the tumour was over the spines of the dorsal vertebræ,

and the difficulty of diagnosis had been to a great extent solved by a previous medical attendant, who had plunged a lancet into the tumour. In the cervical region there often exists down the centre of the growth, over the spinous processes of the vertebræ, a longitudinal furrow, which serves to distinguish the disease from spina bifida.

It is probable that this disease is more frequent in the female sex, and more than probable that first-born children are exceptionably liable to it.

Congenital cystic disease may be situated anywhere in the cervical region, on the trunk, about the gluteal region and perineum, and in internal organs. I have neither read of, nor seen, any case of the disease occurring as a tumour on the extremities of living children.

In the neck, the most frequent position of this disease is near or beneath the angle of the jaw, or deep in the lower part of the posterior triangle; though not unfrequently the tumour is placed symmetrically, either beneath the chin, or over the spines of the cervical vertebræ; in fact, no part of the neck is exempt. On the trunk I have seen two instances of the disease, both being situated on the side of the thorax. Sandifort gives an account of one in the axilla.* One of Mr. Hawkins's cases occurred in this region. Tofft has also recorded a similar case,† while numerous instances are related of the disease occurring in the gluteal and perineal regions.‡

Wernher believes that the affection may occur in all parts and in all organs of the body, and enumerates four parts on the external surface as especially liable to be the seat of these tumours; namely, (1) the front of the neck, especially below the jaw, between the mastoid process and the middle line; (2) the nape of the neck; (3) the axillary region; (4) the gluteal region and perineum.

Among internal organs, the kidney is liable to be affected. Indeed, notwithstanding Wernher's assertion, I know of no instance of the disease occurring elsewhere in visceral organs.§

A case of congenital cystic disease of the kidney was brought before the Pathological Society by myself for Mr. Marsh;‖ it well illustrates the rapid growth and large size to which these

* Otto, Bibliothek für Läger, 1839.

† Sandifort de Singulari Membranæ Cellulosæ Degeneratione, Obs. Anat. Patho. L. iv. cap. 2, p. 21.

‡ Vide Wernher, Otto, Von Ammon.

§ See a case of cystic degeneration of the kidney, by Dr. G. Duffey, Med. Times and Gaz. 1866, vol. i. p. 14.

‖ Path. Soc. Transac. vol. xvi. p. 171, where the growth is erroneously headed 'Medullary Tumour'—an error for which I am myself greatly responsible. The case was under Dr. West's care.

tumours may attain. The tumour weighed fourteen pounds, and was removed from a child seventeen months old, whose weight was more than half made up by the disease. It was first noticed at the age of three months, when it appeared the size of an orange. Although from the first the disease made very rapid progress, it was not accompanied by any special cachexia, and until by its size and weight the tumour interrupted the functions of the abdominal viscera, the child's general health was unaffected. When this, however, took place, the child rapidly emaciated, and died of inanition. During life the disease had been diagnosed by Dr. West, and by others, as a renal tumour.

After death, the tumour was found to lie behind the parietal layer of peritoneum, which was tightly stretched over its anterior surface. It was covered by a distinct capsule, and had formed no connection either by adhesion or infiltration with surrounding parts. It originated in the substance of the left kidney, the remains of which, unaltered in structure, were found spread out in a thin layer over its posterior surface. The ureter was healthy; there was no affection of the lymphatic glands, and the remaining viscera were unaffected.

On dissection, the tumour was found to contain numerous cysts embedded in a coarse fibrous or reticulated structure. The cysts were of various sizes; the larger ones contained others of smaller dimensions springing from their inner surface. On microscopic examination, the solid parts were found to be of a fibro-cellular structure, the cellular element predominating over the fibrous. The cysts contained a clear, serous fluid.

Originating during intra-uterine life, these tumours, after birth, seem to be in no way influenced by the growth or development of the structures among which they are situated. They may, from the time of birth, rapidly increase and destroy life, or they may as rapidly wither up and disappear. The same tumours, after growing for a while vigorously, may cease to increase, and finally become absorbed. While occasionally such a tumour, after remaining stationary for years, will causelessly start into active growth and attain a large size.

A case is related of a man, æt. 50, where the disease had existed since birth, and had not commenced growing until he was advanced in life.* One of O'Bierne's cases, in an adult, had existed since the earliest childhood. A case also occurred under my own care, in a boy, æt. 8, where the tumour had existed at birth, but had not altered in size until shortly before I saw him. Mr. Prescott Hewett has been kind enough to give me the par-

* Revue Médicale, Mars 1834.

ticulars of the following case, where the tumour remained dormant for many years, and then started into active growth. From an infant girl, six months old, a congenital cystic tumour was incompletely removed. The growth was situated in the lower part of the posterior triangle of the neck, extending beneath the clavicle, and under the trapezius muscle. It consisted of a bunch of watery cysts; and the small portion of it that could not be removed was intimately connected with the subclavian artery. From this operation the child quite recovered, and until the age of thirteen no reappearance of the disease took place. At this time, however, a swelling formed in the site of the former growth, which gradually increased in size for one year, when Mr. Hewett first saw the case. There was then, in the lower part of the posterior triangle, a tumour as large as a fist. It lay partly under the trapezius, and extended deeply beneath the clavicle. It was cystic in its nature, and had over its more superficial parts the appearance of translucency.

On April 23, 1849, the front part of the tumour was punctured with a grooved needle, and a quantity of bloody serum escaped: this puncture only emptied the front part of the swelling. Two days later, the posterior part was tapped through the trapezius with a trocar, and two ounces of the same kind of fluid were let out, and a double ligature was then passed from behind, forward through the tumour, to act as a seton. The introduction of this was followed by severe constitutional disturbance and local inflammation. At the end of two days one thread was withdrawn. The tumour became hard, and in a day or two suppuration occurred in the posterior part, and the patient's general condition began to amend. Everything progressed favourably for a few days, when the patient was suddenly attacked by a rigor, intense pain in the tumour, followed by swelling and induration. An incision was made through the trapezius, dividing the tumour, which was found to consist of a number of small cysts in a tough fibrous stroma; a quantity of sanious pus was evacuated. No hæmorrhage occurred. After this, the edges of the wound swelled up and became everted, and there was a profuse serous discharge.

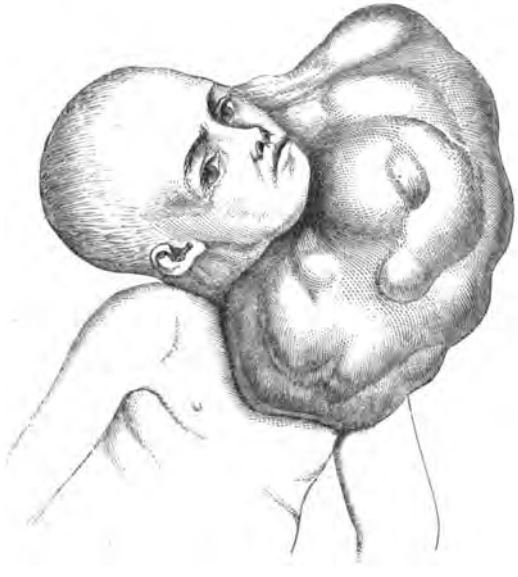
May 9.—Sixteen days after the seton was passed, the severity of the symptoms began to abate, though there still remained a most troublesome cough, like whooping-cough. The resonance of the chest was unimpaired. A month after the first operation the tumour had nearly disappeared, and the wound had nearly healed.

At this time the patient was suddenly attacked by vomiting,

and brought up a very large quantity of fetid pus—a quart or more—and henceforward the convalescence was uninterrupted. At the present date—seventeen years having elapsed—the patient is in perfect health, and there is no trace of the tumour.

Some idea of the large size to which congenital cystic tumours may grow may be gathered from the instance of this disease of the kidney, given above. Here the tumour almost outweighed the body from which it was removed. In Von Ammon's plates are contained excellent examples of the hideous proportions to which the disease may attain. The accompanying woodcut of a child, aged ten months, taken from Von Ammon's work, will serve to illustrate the characteristic appearance of a large growth of this kind.

Fig. 1.



Death may result from this disease as the effect of the general emaciation and exhaustion which usually attends the rapid growth of the tumour. Or if the growth be in the neck, death may occur from pressure on the larynx, pharynx or trachea, or the deep vessels of the part.* Sandifort relates a case where

* See a case by Mr. Hawkins, Med.-Chir. Transactions, vol. xxii. p. 237.

death occurred, three weeks after birth, from spontaneous inflammation of the tumour.*

This spontaneous inflammation may produce a cure. Such a case is related by Tofft.† On the other hand, without assignable cause, congenital cystic tumours may, of themselves, wither away. Three such cases occurred under my own observation, and one—which I proceed to relate—is given in Wernher's Monograph.

The patient, a strong female first-born child, had a tumour at birth, on the left side of the neck, which filled the whole space from the jaw to the shoulder; and from the ear to the middle line of the neck. It was of a bluish colour, and here and there it was streaked over with ramifying blood-vessels. It was tense, elastic, and fluctuating; towards the front part was a small abraded spot. Respiration and the power of swallowing were unaffected. When the infant was ten days old this tumour had notably increased in size. Dr. Wernher, under all the circumstances of the case, determined not to interfere, unless symptoms endangering the child's life should occur; but more to satisfy the mother than with any trust in its remedial effects, he ordered a liniment of two grains of iodide of potassium to an ounce of oil. After some days the posterior part of the swelling became prominent and tense; the skin gave way, and a very small opening formed, through which a dark brownish-red syrupy fluid flowed for some days. No suppuration occurred. The tumour sensibly diminished, and a depressed adherent cicatrix formed at the orifice in the skin. After a time, in a similar way, other openings formed in different cysts composing the growth; and the whole tumour began to shrink away from the jaw towards the clavicle. Finally, after eight weeks, the swelling consisted of a firm—no longer fluctuating—mass, which lay immediately above the clavicle. It seemed to be close beneath the skin to which it was connected by the cicatrised openings, and with which it could easily be raised from the deeper parts. Six months afterwards the child died of small-pox, and on post-mortem examination, the cure of disease was found to be perfect.

The following cases occurred under my own observation. In the first the tumour had grown to most formidable dimensions by the time the child was three weeks old, and three months later was found to have spontaneously diminished to less than a third of its former size:—

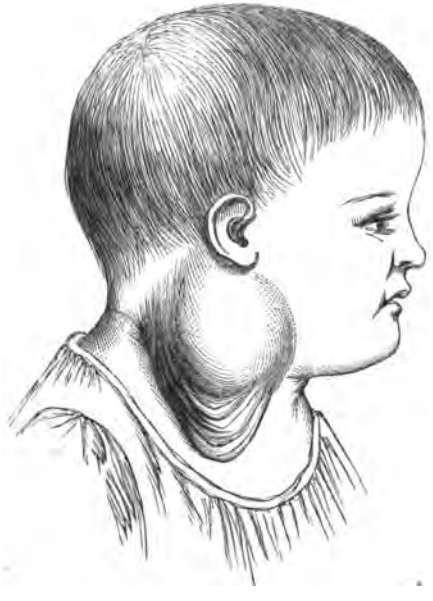
* Sandifort, Museum Anatomicum.
† Otto, Bibliothek für Läger, 1839.

Case I.—Amy White, aged three weeks, was brought to me at the Children's Hospital, September 14th, 1865. She was in good health and condition. Immediately after birth a swelling was noticed in the neck, which had since rapidly increased in size. There was, at the time I saw her, a large, partly cystic and partly solid, growth occupying almost the whole of the left side of the neck; extending, from under the jaw, widely and deeply into the posterior triangle down to the clavicle. It presented all the characteristic features of a congenital cervical cystic tumour, growing rapidly. The mother declined any treatment.

December 13th, 1865. I saw the child, who was in good health. The growth had most remarkably diminished in size. There was at this time nothing to be felt but a loose, flabby, cystic mass, in the bottom of the posterior triangle, not much larger than a hen's egg. The skin over it was shrivelled, loose, and baggy.

March 19th, 1866. A drawing was taken which shows that the tumour had still further diminished in size.

Fig. 2.



Case II.—Amelia Bishop, æt. 1 year and 6 months, was brought to me December 31st, 1864. She was a healthy child, and in good condition. At the time of birth a swelling was found to exist on the nape of the neck in the middle line, which, accord-

ing to the mother's account, was soft and fluctuating. The mother thought it had not increased much in size, but had become rather more firm to the touch. A friend of the mother, who accompanied her, maintained that the tumour had grown rapidly since birth.

There was at the time I saw the child, a soft fluctuating tumour, evidently containing fluid, filling all the hollow between the occiput and the spine, and extending from ear to ear. It measured seven inches and a quarter from side to side; and three inches from above, downwards. It seemed to lie deeper than the subcutaneous tissue: it was fluctuating and soft when the muscles were relaxed; but very firm when the muscles of the back of the neck were put in action. It was apparently painless. The skin over it was natural.

The spines of the vertebræ in the upper clavicular region could not be felt. There was no symptom of deficient innervation of the lower parts of the body.

I advised that no treatment should be adopted unless the tumour increased in size.

A year later (November, 1865) the mother wrote me, saying that the swelling had greatly diminished, indeed was hardly perceptible; and the child was in good health.

February 19th, 1866. Three months later, I saw and examined the child, and could only detect a slight fulness in the back of the neck; so little as would pass notice except on careful examination.

I venture to draw attention to the natural state of the skin covering this tumour, and to the due innervation of the lower parts of the body, as furnishing most valuable assistance in distinguishing the tumour from a cervical spina bifida.

The following case has many points in common with the last:—

Case III.—Rebecca C. F., æt. 3 months, was brought to me on January 7th, 1866. There was a large fluctuating swelling in the dorsal region of the spine, quite central in position—in appearance like a spina bifida. The integument over it was of natural thickness and colour. It measured five and a half inches by six inches, in its transverse and longitudinal diameters respectively; while its circumference round the base measured eleven inches and a half. Its contents seemed to be fluid, and its walls thick and tense. It was very prominent, and it could be swayed about with difficulty; being as much restricted in its movements as the sac of a spina bifida. Owing to the size and tense condition of the tumour, the spinous processes of many of the dorsal vertebræ could not be felt.

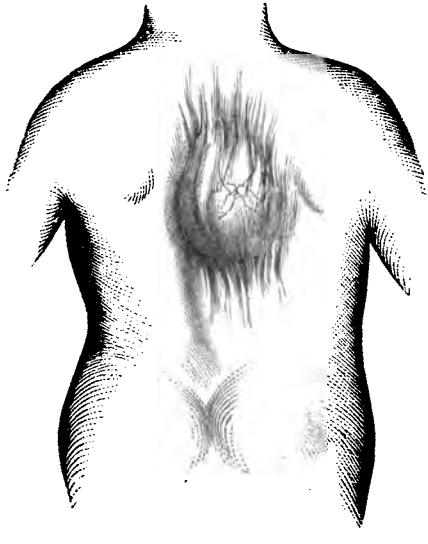
The child seemed in vigorous health, and to be free from pain, nor was there any tenderness on pressure. There was no symptom of any kind indicating deficient innervation of the lower parts of the body.

This tumour at the time of birth was about the size of a walnut: since that time it had grown quickly. About six weeks before I saw it, a small puncture had been made with a lancet; the mother stated that nothing but blood had escaped at the time.* A gutta percha shield was moulded to the tumour, and being lined with wash leather, was firmly bandaged on.

March 5th. Two months later, I saw the child—the tumour was firmer and smaller, measuring in its diameters four inches and five inches, and in circumference nine inches: the child was in good health. The treatment was continued.

February 9th, 1866. The child was in good health; the tumour had been gradually diminishing in all its diameters—it was now so flat that it was difficult to measure its circumference. It was more oval in shape than when seen a year ago,

Fig. 3.



and was now not exactly median in position; it felt baggy and flaccid; the skin over it was dimpled and wrinkled, and was here and there puckered in and adherent to the growth,

* The puncture probably did not penetrate to the cavity of any of the cysts of the growth.

which latter felt lobulated, and was evidently of a polycystic nature, though from the cysts being empty, little remained but the fibrous skeleton of the growth. The appearance was like that presented in Case I. and is represented in the accompanying woodcut.

The spines of all the dorsal vertebræ could now be plainly felt. These were normally prominent and regular.

There can be little doubt but that congenital cystic tumours are not of a cancerous or malignant nature, and that they do not spread, by vascular or lymphatic absorption, from one region of the body to another. At least there exists no evidence on the side of malignancy, and everything to prove the contrary. It is also well established that in many instances the disease is amenable to treatment, and that a cure when once obtained is permanent.

It is certain that a spontaneous cure may take place. Such was the case in two cases recorded by others,* and in four instances that have come under my own observation; and therefore it is evident that measures, either operative or medicinal, for the treatment of this disease are out of place, unless necessitated by the locality of the tumour, or by its rapid growth. When situated in the neck (the most common position) the bulk or deep attachments of these tumours may seriously interfere with respiration, deglutition, the power of sucking, or the circulation of blood in the brain.

On the other hand, on the thorax, back, and perineum, they may be of but little inconvenience, and cause no danger to life. Remedial measures are generally urgently required in the event of the tumour beginning to increase quickly, since this rapid growth is for the most part attended by a commensurate wasting of the rest of the body and by a failure of the general health.

Various means have been employed for the cure of this disease, of which the following are the principal:—(*a*) Puncture. (*b*) The application of so-called discutients. (*c*) Laying open the cysts. (*d*) The injection of Iodine. (*e*) Complete removal by the knife. (*f*) Setons.

(*a*) Puncture with a small trocar is a perfectly harmless and speedy method of relief; it is often unavoidable, and of great service in cystic tumours of the neck, which from their size or tension, interfere with respiration, deglutition, or the circulation. Puncture, however, is merely a palliative measure; it seldom does more than afford a temporary relief, and this is often only partial, since, out of the many cysts forming the growth, but one is usually opened by the puncture, and the deeper cysts, from the

* Wernher and Tofft.

removal of pressure, often seem the more rapidly to increase. I am not aware of any recovery from this disease that could be attributed to the treatment by simple puncture, whether single or often repeated; yet, the emptying of the larger cysts of a growth by a fine trocar may be the only means of affording immediate relief, or temporarily averting danger; while in other cases it may be usefully employed to postpone the necessity of more heroic treatment until the tender age of early infancy be passed. Or, again, its employment may assist the efficacy of other remedies. . There is but one instance recorded of death caused by puncture. The case occurred to Volkers, who tapped a cervical cystic tumour in an infant eight days old—the child dying of trismus,* a casualty which, standing alone, need scarcely weigh against the proceeding.

(*b*) From the local use of the so-called discutients, such as tincture of iodine, iodide of potassium ointment, I have never witnessed any success which could fairly be attributed to the effect of the application. In Wernher's case, which I have related as an instance of spontaneous cure, friction with iodide of potassium and sweet oil were employed. Whether the drug or the friction had any share in causing the evacuation of the cysts is more than doubtful — indeed, Wernher himself seems to attach little value to the application. And others also regard the case in the same light.† One of the cases recorded by Mr. Hawkins was treated in this way—the tumour here was repeatedly punctured, and friction, with iodide of potassium ointment, was employed during a whole year. At the end of this time it was one third of its original size, and some years afterwards Mr. Hawkins heard that the tumour had disappeared. In this case, if the use of the iodide of potassium was the cause of the diminution in size which the tumour underwent, it was at best a tardy means of relief.

Though I am unable to produce any instances of cure distinctly referable to the use of an external application, I would venture to recommend the use of weak vermilion ointment in a suitable case, as the most powerful of external remedies of this kind, and as the most likely to effect a cure.

(*c*) The laying open of the cysts forming these growths (how-

* Dr. Storch, Ueber das angeborene Hygrom des Halses, Journal für Kinderkrankh., Band xxxvii. p. 68. Dr. Storch has collected twenty-four cases of congenital cystic tumours of the neck; ten of these recovered—one spontaneously, and nine through operative interference. Twelve died; of these, four were treated on the expectant plan; one died of pneumonia, seven died from the operation, viz., six from suppuration, and one from trismus; in two cases the final result was not recorded.

† Dr. Storch, loc. cit.

ever suitable for analogous tumours in adults) is a perilous plan of treatment for infants. A case is related by Mr. Hawkins of a child one month old, in whom this plan was adopted; four months afterwards the proceeding was repeated, and an attempt was made to remove the tumour: this being found to be impracticable, a ligature was applied around the deeper part of the growth. The ligature did not separate until three months later, and during this period repeated attacks of erysipelas occurred. The child is reported ultimately to have done well; but whether the disease was cured, or how long after the cessation of the treatment the cure took place, is not stated.

A case is related,* where Volkers laid open a cystic growth in the neck of a new-born child, who died sixteen days afterwards in consequence of the operation.

The same plan of treatment was adopted in a case by A. Bednar, with a similar result. The growth was situated in the neck; when the child was two months old one of the superficial cysts was laid open; profuse suppuration occurred—emaciation, and death on the 14th day.†

(*d*) The puncture of the cyst, or cysts, forming the tumour, and the subsequent injection of iodine solutions, has proved successful in several instances. Mr. Holmes bears testimony to its occasional good effects,‡ and M. Roux has obtained good results from its use. It is a means of treatment, however, which is uncertain, and not wholly devoid of danger in young infants. Still it is a hopeful remedy in suitable cases, and can be employed in any case where the rapid cure of the disease is not of paramount importance. Suitable cases for treatment by iodine injection are those where the cyst is unilocular (an exceptional condition). Mr. Prescott Hewett informs me that recently he cured a large congenital cervical cyst in an infant by a single injection of iodine. Here the cyst was unilocular.

(*e*) Complete removal.—It may almost be laid down as an axiom, that the most careful investigation of congenital cystic tumours of the neck, before an operation, cannot precisely determine the connections of the growth either in their depth or extent.§ And post-mortem examination of such growths in this situation often reveals connections so intimate and extensive, as would make the most daring pause before attempting their removal during life. In Mr. Arnott's case, related by Mr.

* Casper's Wochenschrift, 1837, No. 44, p. 704.
† Gurlt, Cysten-Geschwülste des Halses, p. 27. Berlin, 1855.
‡ Lancet, 1864, and System of Surgery, p. 963.
§ Wernher, Die angeborenen Kysten-Hygrome, p. 88.

Hawkins, of a congenital cystic tumour of the neck, the operation could not be completed: this also occurred in Mr. Hewett's case, before related. In Mr. Arnott's case, the child's life seems to have been put in great danger; in Mr. Hewett's, the remains of the disease took on active growth thirteen years afterwards.

Mr. Holmes extirpated a tumour of this kind from the neck of a boy aged eight years;* the disease had existed since birth, but had only recently begun to grow. The tumour was very large, was multilocular, was situated on the right side of the neck, chiefly in the posterior triangle; its connections were extensive, deep, and uncertain. The boy had been for some time under my own care, and I had tapped some of the cysts at different times without any permanently good effect. The parents being anxious for an operation, which I was unwilling either to recommend or undertake, Mr. Holmes took charge of the case, and removed the tumour with the knife, only a small portion of the cyst wall remaining behind.

The final result was all that could be desired; the boy perfectly recovered. Indeed, the issue of the case did credit to the skill and discrimination of the operator, and fully justified the proceeding in this particular case; yet the difficulties and dangers of the operation were so considerable, and the constitutional effects upon the boy were for a time so serious, that I should be loth to adopt the same treatment in a similar case.

Nélaton† adopted this treatment for the cure of a large cervical cystic tumour of congenital origin, having first punctured the growth, to admit of its more easy manipulation. The child did well for a time, but the final result of the case is not given. Broca‡ also removed a large and deeply-situated cervical tumour of this kind from an infant, who died three weeks later, the fatal result being due, apparently, as much to the neglect of the parents as to the consequences of the operation.

(*f*) The treatment by seton was adopted with success in Mr. Prescott Hewett's case, but only at a very serious risk to the patient's life; a risk, however, under the circumstances well worth incurring, since the size of the growth and its rapid increase urgently called for interference; while its situation and structure respectively rendered removal impossible and the treatment by iodine hopeless.

* For a detailed account of the case, see Mr. Holmes's paper, Lancet, 1864, vol. i. p. 577.

† Gaz. des Hôp. 1854, No. 78, p. 310.

‡ Journal für Kinderkrankheiten, 1861, No. 37, p. 82.

The following cases, under my own care, were treated by the introduction of fine silk threads as setons:—

Case IV.—George A., aged eight months, was brought to me, May 21, 1864. He was a healthy, well-nourished child. At the time of his birth a swelling was noticed behind the right ear, about the size of a hen's egg. This had gradually increased up to the time when I saw him; it was then as large as one's fist, and was situated in the posterior triangle of the neck, running up behind the ear, and towards the angle of the jaw. Its deep connections were of uncertain extent; it felt irregularly cystic, and evidently contained fluid. Here and there its surface was of a bluish tint, like a cervical hydrocele. In parts it was tense and elastic, and elsewhere it felt flaccid, semi-solid, and lumpy.

I passed a seton, formed of a single thread of fine silk, through the most prominent part of the tumour. The immediate result of this was to diminish the size of the swelling considerably, and subsequently to cause inflammation, suppuration, and discharge from two of the cysts which formed the growth. Three weeks after the introduction of this seton, these had quite collapsed, and the tumour had lost about two-thirds of its former size.

On June 20, a silk thread was passed through what appeared to be the only remaining cyst, and was withdrawn after three days. This cyst inflamed, consolidated, and subsequently contracted without any suppuration.

A month afterwards, the tumour had almost disappeared, though there still remained a thin-walled bluish-looking cyst, about the size of a marble, in the lower part of the neck. In October of the same year, five months after the commencement of the treatment, the child was in good health, and there still remained a very small, nearly empty cyst; all the solid part of the growth had disappeared.

January 13, 1866.—His father reports, 'that there is only a slight projection left, which is not easily perceived, and which appears to die away as he gets older.'

I learnt subsequently, from additional experience, that it was unnecessary to leave the seton in long enough to produce suppuration, but that, as was done in the preceding case, after the passage of the second seton, it was better to withdraw the thread as soon as inflammation and some amount of induration occurred in the cyst.

Case V.—Mary Lyons, æt. 3, came under my care at St. Bar-

tholomew's Hospital in January, 1865. There was a large cyst, or rather growth, formed of cysts and solid matter, in the posterior triangle of the neck on the right side. It had existed since birth, and was increasing in size. It felt flaccid, and thin walled, uncertain in its connections, but evidently extending deeply towards the clavicle in the posterior triangle.

The growth had been tapped more than once, at another metropolitan hospital, without any permanent effect. A single thread of fine silk was passed through the most prominent part of the swelling, and about an ounce and a half of transparent fluid drained away at the time of puncture. The silk was not withdrawn until considerable inflammation had been set up, when it was removed, and a poultice was applied. There was, however, no suppuration in the growth, but the inflammation was accompanied by an apparent consolidation of texture. As the inflammation subsided, the tumour diminished in size; and on April 29, three months after the introduction of the thread, the child appeared in good health. The only trace of the tumour was a very obscure and indistinct sense of fluctuation deep in the posterior triangle of the neck.

January, 1866.—The child remained free from any return of the disease.

Case VI.—Ada Wilkinson, an infant, was brought to me at the Children's Hospital, April 29, 1865.—She was a fat, healthy child. Two months previously a small lump was noticed in the left side of the neck. There was at the time I saw her a cystic tumour in the posterior triangle, about the size of an orange; a large portion of it extended beneath the trapezius; the rest was more superficially situated; it was rapidly increasing in size.

April 29, a single silk thread was passed through the centre of the growth, giving exit to the usual transparent fluid. Four days later, the size of the growth had greatly diminished, and one large cyst at least was completely empty. As the thread had produced but little inflammation or discomfort, it was allowed to remain 'in situ' for one week, when it was withdrawn.

A fortnight after the introduction of the thread, there was still a smaller semi-solid lump in the posterior triangle.

January 29, 1866.—The child is now fifteen months old; the disease has quite disappeared, and she is in very good health.

Case VII.—George Fawcett, æt. 14 weeks, came to me, at the Children's Hospital, Oct. 24.—There was a loosely filled congenital cystic growth, with bluish translucent walls, the size

of a small hen's egg, situated on the lower and posterior part of the thorax. It was quite movable beneath the skin; it was increasing in size. A single strand of fine silk was passed through it, and transparent serum escaped. On the fourth day, the growth was very tense and bright red coloured, and serum was running freely from the punctures. The silk was withdrawn, and a poultice was applied.

January 17, 1866.—The place on his side is much smaller, but has not entirely disappeared.

Case VIII.—Edith M. Harvey, aged two years, was brought to me on June 22, 1864. She was a healthy child. Soon after birth, the mother noticed a swelling on the left side of the chest as large as a nut. At the level of the seventh rib, rather nearer the spine than the sternum, was a hemispherical, and apparently solid growth, about the size of a large chestnut. This was said by the mother to be decreasing in size. Connected with this, and extending from it upwards, and inwards towards the breast, was a puffy, ill-defined swelling, feeling like a cystic or nævus growth. It was as large as the palm of a man's hand, and at its anterior border, just beneath the nipple, apparently in its substance, there was another firm lump, much like the one in the back in shape, but rather smaller. As it seemed likely that this growth was either diminishing in size, or stationary, I advised that the child should be taken back into the country and watched.

A year afterwards, June 21, 1865, the child came again. The larger of the apparently solid portions, namely, that in the back, was about the same size, and was of a bluish tint on its surface. The rest of the growth was in much the same condition as it was on the first visit. The disease was evidently either a large degenerate nævus, or a partly solid and partly cystic tumour.

The softer part of the growth was punctured, and a brownish-yellow serum escaped. A single thread was left in as a seton. A week afterwards the cyst had suppurated, and was opened, giving exit to pus. At the same time a thread was drawn through a neighbouring cyst, and left in situ.

January 20, 1866.—There is now only a fulness in the side, the tumour has disappeared, and the lump in the back is about the size of a large pea, the one on the outer side of the nipple as big as a hazel-nut.

Though the result of one's personal experience is favourable to the treatment of congenital cystic tumours by means of

setons, it is but right to record the opinion of others on this subject. Gurlt* considers this plan as generally inapplicable to the disease, and as dangerous on account of the risk of diffuse suppuration and purulent infection. He thinks it occasionally admissible for single outlying cysts, or the deeper parts of a compound cyst, where the main bulk of the disease has been cured by excision, or some other means. Storch† considers the treatment by seton as not particularly satisfactory. He has collected six cases, three of which recovered and three died under this plan of treatment. It does not appear that either of the above-named authors speaks from personal experience. Guersant,‡ however, who must have had unusual experience in these cases, speaks favourably of the seton as a means of cure.

In making use of the seton, there are certain precautions to be adopted, which it may not be out of place to mention. The seton employed should be a single thread of fine silk or sewing cotton. It should be passed, if possible, through but one cyst of a growth at a time, and to this end should not be plunged deeply into the tumour, but made to pass through the part nearest the surface. It should be withdrawn as soon as any appearance of inflammation with induration is set up, and if possible before the occurrence of suppuration, the occurrence of this process being undesirable.

No incisions into the growth should be made, unless suppuration has unmistakably occurred. It is not always necessary to continue the introduction of setons into each of the separate cysts of a large growth, as when once the tumour has begun to contract, the process will often continue spontaneously, as was the case in three of the patients under my own care.

No plan of treatment for congenital cystic tumour can be of universal application. The age of the patient, the locality of the disease, its rapid growth, or decrease, its dimensions, and its composition, whether unilocular or otherwise, must chiefly weigh in the selection.

While freely acknowledging that some cases of this disease are from the first incurable, and admitting the value of iodine injections in many cases, and the advisability of excision in rare instances, I think that we have, in the treatment by seton, a comparatively safe, and certainly effectual method of dealing with many formidable cases of congenital cystic tumour. This treatment, if carefully carried out, after the plan above de-

* Cysten-Geschwülste des Halses, Berlin, 1855, p. 35.
† Loc. cit. p. 81.
‡ Notices sur la Chirurgie des Enfans, deux^me^ fasc. p. 71.

scribed, enables one to attack the disease in detail, and thus materially to diminish the danger to the patient.

It will be found, in the cases of treatment by the seton, recorded as fatal, that very large setons were used, and that they were passed through the main bulk of the tumour, and then were allowed to remain in position until either severe inflammation or excessive suppuration was set up, under which the patients died.

Article III.—*Practical Observations on Diphtheria.*

By W. Newman, M.D. Lond.

THE growing tendency of medicine in the present day would seem to be the recognition of general constitutional disturbance as the cause and factor of definite local changes, in place of the older creed, which treated with little of doubt or cavil, as special entities, local conditions, yet owning a common origin. Hence the acknowledgment of diseases as due to alterations in the circulating fluid itself; and as one in the now long catalogue of blood disorders I would wish to consider and to write of Diphtheria, a disease manifesting local distinctive characters, yet general from its earliest commencement.

The analogies of the affection might lead to the impression (by some authors indeed advanced) that it was an exanthem: yet though with scarlet fever, for example, certain similarities may be traced, there are many more points of well marked divergence, notably in some such directions as: That an attack of scarlatina confers on the patient no immunity from subsequent diphtheritic seizure and vice versâ, no peculiar skin rash of definite onset and duration is noted in diphtheria, though a roseolous efflorescence may occur for some few hours, not followed by desquamation. The throat and fauces are implicated in both diseases, yet the ulceration and tissue destruction of the scarlatinous affection have little in common with the exudation of lymph characteristic of the other disease. Again, too, the one has sequelæ, indicative of intense nervous implication, while the former induces, by somewhat less evident link, mischief either about the chest or the kidney.

I believe it more accurate and more wise to look on diphtheria as a disease in which some morbid material is received into the blood—the system as a whole sympathizing subsequently more or less severely, and the further progress being marked by the exudation of lymph about the mucous membrane lining the

throat, pharynx and upper part of the air passages, while the general skin, especially if denuded of its epidermal layer, may show the same state. Yet to speak of the affection as if this local state were the constant or necessary attendant, or as if from the physical changes about the throat the constitutional disturbance were afterwards evolved, I hold to be incorrect in observation and untrue in theory; for the sufferers, too, a belief fraught with possible serious mischief if it, by logical deduction, lead the practitioner to deal with the tangible mischief, instead of addressing himself to the whole systemic depression.

I cannot substantiate by aid of statistical tables the impressions which I have derived from a fairly large experience of the disease in question; the statements put forward as to its characters and treatment must be accepted as personal deductions, and claim only that weight which may reasonably be asked for the honest declarations of a single observer.

Isolated cases of the disease are not unfrequently met with —more commonly an epidemic seems to sweep through a village or district; any single case, however slight in its symptoms, may prove a focus for spreading the affection. Whether the resulting attack be severe or trivial, seems to depend more on the exact health standard and general vigour of the recipient, rather than on the conditions of the primary source.

In this point of view, experience teaches that children suffer most, and the younger the patient the less the chance of recovery. Direct contact with an invalid is not necessary to the inducing diphtheritic mischief, but the breathing a tainted atmosphere, or close dealing with the secretions poured out from the affected surfaces, may readily determine its transmission.

I believe that the poison may be handed on to others by the wearing clothes on which expectorated matters have remained, and that this, from poverty and carelessness, is by no means uncommon with the poorer classes.

Defective hygienic conditions do not seem to develop these disorders, yet I have no question but that they intensify their virulence, and, most probably, by the indirect mode of diminishing the normal vigour of the residents; thus, in one block of cottages isolated, sadly deficient in space, air, and water supply, and abominably dirty, nearly two-thirds of the cases attacked died—a far larger proportion than that furnished by the rest of the district; and in those, too, who lived through the active stage the sequelæ were well marked, and recovery very tedious.

As a rule, the period of incubation seems to be short, most so

when the exposure to the toxic influences has been very complete. In no case that I have been able to watch has the time appeared to exceed seven or eight days; most frequently it has been two or three only.

Systematic writers have with reason marked out different forms and stages of this disease; but however well such divisions may answer the purpose of pointing a moral to a class of students, or of defining sharply the indications for one or other special plan of treatment, there is yet the same substratum, the essential part of the disease is alike throughout, varied as its after appearances may be from local changes or constitutional peculiarities.

Adopting the most usual classification of diphtheritic affections, I would refer to them as simple or malignant; grouping the laryngeal form under the first-named, and the nasal complication under the last division.

Simple Diphtheria.—The early symptoms are most frequently severe; burning skin, pains in the limbs, and malaise, usher in the attack, with usually but a brief prelude of shivering. Not unfrequently the sudden invasion of illness has led me to expect the after development of diphtheritic exudation, an impression verified by the result.

A common local occurrence is that of œdematous infiltration of the mucous membrane of the uvula and tonsils; the uvula becomes thickened, glistening, and lies helpless on the tongue; swallowing is thus difficult, and the voice is altered from its natural tone. Or, again, there may be no special œdema of the lining membrane, and then exudation of lymph is more speedily noted, and there is more complaint of soreness about the throat generally.

In either case the uvula, palate, or fauces, soon exhibit one or more spots of lymph deposit which may remain distinct centres, or are covered with a whitish film of more uniform thickness. The surrounding mucous membrane is intensely injected, and when a few hours have gone by, the punctated spots will be seen to have become larger and to have coalesced, or the general filmy coating will have increased in thickness and extent.

Many of the simple cases hardly exhibit so much of local change. The effused lymph soon clears away, and the throat resumes a fairly natural appearance. Yet much more actual debility will be found to result than the precursory symptoms would have seemed likely to warrant, and this perhaps also will be followed by conditions of disturbed innervation.

More serious cases, still simple in character, are to be noted. The exudation tends to increase in extent and cohesiveness,

so forming a complete pellicle, which may often be stripped off with forceps, then leaving bare the mucous membrane, either raw and bleeding, or but little altered from its ordinary state. The pellicle is in such cases usually distinctly fibrinous to microscopic examination, while in a different state of system, or more serious form of the disease, it is found to be more friable, less adherent, and corpuscular in its intimate structure.

The duration of this form of the disease is from a week to ten days in the milder forms; from fourteen days to three weeks where there is more local mischief.

There are too cases seen in practice, where diphtheritic poison seems to seize on a patient without producing local manifestation, or at the utmost the throat may be somewhat sore, and the fauces be found to exhibit a trace of redness, without lymph deposit. Subsequent marks of depressed health and nervous disturbance are to be met with.

Such instances seem to happen in the same house with, or in the vicinity of, already existing cases; and, probably, like the masked form of scarlatina, owe their being to a partial implanting of the poison in an uncongenial soil, the constitution sympathising more or less, although the customary local changes do not appear.

Nor can such instances be treated as of light moment. In 1860, I was asked to see a lad about fifteen years of age, who was living with and working for a baker. He had been in contact with some diphtheritic cases, and was weak and prostrate, but there was no pellicle about the throat, nor anything more than a shade of general redness of the fauces. The depression and rapid pulse were the only symptoms of serious moment.

The master wished me to sanction his immediate removal home, a distance of fourteen miles; but this I declined to do, and spoke plainly of the risk the lad would run. The next day, however, the fifth day of his illness, the father took him home in a covered cart. The patient was very much exhausted at the end of the journey, and died very soon afterwards, no change in symptoms having occurred.

On these comparatively simple cases it would seem as a rule that the laryngeal complication supervenes—usually, too, but a few days from the first declaration of illness. So that, in any given case, if the patient have been suffering for eight or ten days, the probability of the downward extension of the exudation to the air-passages is very much lessened.

Where this complication does occur, the earliest symptom seems to be a little, husky, dry cough, with alteration in the voice; no special renewal of febrile disturbance; then follows

whistling inspiration, with metallic, croupy cough, and the ordinary sequence of symptoms which point unmistakeably to narrowing of the channel of the air-passages. In by far the great majority of these cases, death closes the scene in a comparatively short time.

Malignant Diphtheria sometimes commences thus:—a child is noticed to be more still than usual, to have some pallor of the face, loss of appetite, and restlessness.

Close inquiry elicits no history of sore throat, but the tonsils have fragments of deposit upon them, and the submaxillary glands are slightly swollen; very soon the pulse becomes excessively rapid, the local conditions of the throat suddenly change for the worse, and the child sinks into a hopeless state of exhaustion.

Again the malignant form may supervene on symptoms which have been for several days of the ordinary simple kind; or, lastly, it may be well marked from the very first, and manifest very serious conditions without any premonitory illness.

The duration of inflammatory or feverish symptoms is very short. The local exudation is produced with great rapidity, less firm, less closely attached to the subjacent textures than in the simple form, and under the microscope presents corpuscular appearances. Local, and even severe, hæmorrhages may occur, staining the coriaceous material deposited on the mucous membrane of the fauces, and adding materially to the weakness and discomfort of the patient. Not unfrequently ulceration of the exposed mucous membrane may be noted, and the destructive process so commencing may run on into actual sloughing of the deeper tissues. Hence much of the fœtor which attends on the later stages, and also certain states of the general system, which are probably due to absorption of the putrilage from the decomposing surface. When these are noticed, analogous somewhat in their characters to surgical pyæmia, there is practically no hope left for the sufferer.

Published opinions vary much on the value which should be attributed to the coincident appearance of engorgement and infiltration of the glands below the jaw. I confess that I have been led to look on early and marked swelling of these structures as a symptom of impending malignancy of type; and in this view I have rarely been deceived. A certain amount of enlargement may in the most simple cases be met with, and is in them of little practical moment; but when the glands become considerably swollen, and with great rapidity, I think the prognosis should from this fact alone be a very guarded one.

The supervention of laryngeal implication on this septic form

of diphtheria is, I believe, very rare; the breathing may be oppressed, and more or less cough and dyspnœa be present, but the fatal termination is due to asthenia, not to apnœa.

On the other hand, the invasion of the nasal mucous membrane should be looked on as a variety of the malignant type.

The first warning of the extension is the appearance of mucous discharge from the nostrils, which reddens and excoriates the nasal orifices and the upper lip; then swelling, and the characteristic exudation, followed very speedily by oozing of blood from the much-injected mucous membrane. Remedial agencies have appeared of very little service, probably in part from the extreme difficulty of dealing with the affected surfaces. The great majority of such cases that I have seen have terminated fatally, two of them by profuse and frequently recurring loss of blood.

The foregoing pages take little cognizance of anything other than the tangible characters of diphtheritic affections, yet one symptom, on which much stress has at times been laid, demands a passing remark. I refer to the occurrence of albuminous urine. Some years ago, in a number of consecutive cases, I examined the urine day by day; in the majority there was evidence of the presence of albumen, but I was not able then or since to link its appearance with any special tendencies or peculiar virulence of the attack. In several of the worst cases it was not to be found, while in some less severe the urine contained albumen continuously and for some time. I have rather looked upon its existence as a comparative accident, often but of short duration, and not of material import; owing its being, too, far more to the vitiated condition of the blood than to any implication of the structure of the kidney.

The natural history of the class of diphtheritic affections does not end with the outbreak and its subsidence, for certain well-marked sequelæ are commonly met with. After an interval of apparent progress to convalescence, these consequences usually declare themselves and are to be referred to disordered innervation—varying from defective controlling power over some one or more sets of muscles, to a more or less completely defined paralysis. Nerves of motion and sensation appear almost equally susceptible of the benumbing influence of the poison of diphtheria, though the former have seemed sometimes most affected.

The whole spinal system may be implicated, and the extremities lose for a time much of their power, or the condition

may be that of almost complete paraplegia, the arms remaining unaffected.

Special nerves suffer—notably those nearest to the original seat of the local manifestations—thus, very commonly, the voice becomes husky, swallowing is not accomplished without difficulty, and fluids are apt to return through the nose. The velum palati hangs loose and flaccid, has lost much of its sensibility, and fulfils none of its purposes as a muscular curtain. So, too, I have twice seen the portio dura affected, and in one case the external rectus of the eyeball was paralysed. Vision may be defective, the eyes easily wearied, and objects after a short time blurred and indistinct—a state of things due to defective innervation, relieved by the use of convex glasses, and ultimately disappearing as physical health is restored.

Another fairly well-marked class of cases must be referred to loss of power about the 'vagus,' as evidenced by slowness and feebleness of heart-beat, slow and infrequent respiration, and failure of digestive power.

Recovery is often tedious, yet has commonly happened. Those cases where the spinal cord is involved are of most grave future.

Treatment.—In diphtheritic affections, just as in the wider division of fevers, a cure is not to be sought for. As yet no specific has been found which will enable the medical attendant to cut short the attack; and in the present state of medical knowledge, it is simply open to us to tide the patient over a disease more or less severe, more or less prolonged, and attended very commonly by serious complications.

On this view, then, I have little sympathy with the directions laid down by some authors to treat the outbreak on so-called general principles; to give saline and diaphoretic medicines, because the skin may in the early stage be hot and dry; aperients, because the bowels may not have acted with all due regularity, and when the opposite condition of depression is established, then, and then only, to have recourse to tonics, wine, and supporting measures.

The natural history of the disease is of material moment in settling this question of treatment. Many cases will recover speedily, many more will lapse almost as rapidly into a state in which but little of good can be looked for; but the one essential characteristic of well-developed cases of diphtheria is that of a distinct tendency to death from general failure of strength.

If then the asthenic stamp be so strongly impressed, it seems to me imperative that nothing be done which may lower the existing standard of vigour, and that everything be done which

may husband the strength for a time of critical endurance. I write strongly, for I believe any other doctrine to be erroneous and fraught with extreme danger. A very short time will turn the trembling balance in one direction or the other.

Therefore, from the very first onset and recognition of the affection, I would urge that the patient be kept in the recumbent position, and be spared any unnecessary exertion; that he be fed frequently rather than largely; and that the air of the room be often thoroughly changed. Do not, blindly following a mischievous routine, administer aperients; least of all, those containing mercurials. A simple injection of warm water will probably meet every requirement. Nor is it advisable to encourage perspiration by sedative or lowering medicines.

In this, as in all septic diseases, I would ask for constant circulation of fresh air through the apartment. Screen the patient from direct draught, but yet by continuous fire and open windows ensure that he shall not breathe again and again an atmosphere vitiated by his own excretions.

Medical treatment will embrace

1. *Local applications.*—So soon as the exudation is fairly manifested, and the co-existence of vivid redness of the surrounding mucous membrane points to its probably rapid increase, I hold it the best to use at once, as the most efficient application, the strong hydrochloric acid, diluted either with honey or with water, in equal bulk. A piece of sponge tied firmly on a stem of stick or whalebone, and then well saturated with the diluted acid, will be best for the application; and this once made the patient will usually declare himself much relieved. It seems to do away with the burning sensation about the fauces at the cost of a comparatively momentary pain, and furthers the curling up and detachment of the coriaceous layer. Once or twice again it may be well to resort to this potent caustic at an interval of twelve or twenty-four hours, rarely more frequently.

Then I have used as an after application the undiluted Beaufoy's solution of chlorinated soda, also used with sponge or large brush every four or six hours.

To make any of these applications thoroughly, the tongue must be well kept down with a spatula, or drawn forward out of the mouth.

Gargles are of little value. It is true that they wash out the mouth and the anterior part of the fauces, but they do not go back to the special seat of the disease.

In those not very infrequent cases where fœtor exists from decomposition of the secretions, or from commencing sloughing, I have had recourse to a tolerably strong solution of the chloride

of zinc, and I think with decided advantage; and as an addition to the patient's comfort, the permanganate of potash (Condy's fluid), with six or seven parts of water, may be employed to cleanse the mouth from stringy saliva and offensive discharges.

In the earlier cases the nitrate of silver was used as an application, and then totally discontinued. This agent has always seemed to me inefficient as a caustic, apt to deceive the eye in subsequent examinations from the coating which is left behind after its use, and unsatisfactory in any influence for good in the removal of exudation or effect on the tissues.

Of external applications I have little to say, other than to deprecate very positively any resort to those which may remove the cuticle or damage its condition. More than once I have met with cases where after the local use of blisters, or liniments of liq. ammoniæ, the irritated surface has been speedily covered with the characteristic lymph exudation, but in no one instance has even temporary benefit appeared traceable to such means.

For the comfort of the patient and indeed with some actual value I think it well to apply an evaporating lotion of spirit of wine to the swollen and infiltrated structures beneath the jaw.

2. *Internal Remedies.*—The class of internal remedies from which the most good may be hoped is that of tonics, and from many conditions which are present in severe diphtheria preference should be given to preparations of iron. These, in one form or other, I would give even from the first declaration of the disease, not waiting until increased pallor of integument and advancing debility make such agents palpably needful.

The ordinary tincture of the sesquichloride is for several reasons most to be advocated, not merely from its effect on the circulating fluid, and from its power as a general nervine tonic, but also from its presumably good effect on the affected tissues as a direct astringent.

Again, it would seem that all compounds containing and readily parting with chlorine are of service, so the chlorate of potash may be given in conjunction with the iron; or, for the good purpose of combining in one, an efficient local application, and a means of introducing chlorine readily into the system, the formula given below may be employed with advantage.*

* ℞ Potass. Chloratis ʒij.
Acid. Hydrochloric. ℥ij.
Aq. destillat. ℥ij.

Mix the acid and water together first; add the mixture to the chlorate of potash, and keep the whole in a dark place.

ʒij. of this solution to be mixed with a pint of water.—Dose, one or two tablespoonfuls, according to age, for internal use; or as a wash for mouth or gargle.

Several of the worst cases of diphtheria which I have seen have seemed to owe their recovery to the steady use of this compound; specially one where life for days together hung by a thread, and where a huge coherent cast of the pharynx and back of the throat was expelled.

Cinchona, as the decoction or tincture, I do not think of material value; and quinine, although I have commonly given it where there has been much loss of appetite, excessive perspiration, and so forth, has seemed to carry with its use only an indirect good.

3. *Food and Stimulants.*—These play no unimportant part in the medical dealing with so serious a malady. Small quantities of nutritious and easily digestible food should be given at very short intervals; and no existence of feverish symptoms should for one moment be deemed warrant for confining a child to 'low diet.' I believe that, humanly speaking, very many deaths from diphtheria have resulted from inattentive nursing, and from an unwillingness to press on a listless sufferer the prescribed supplies of food.

New milk is of very much value, and may be used in large quantities, and, at intervals with this, beef tea, essence of beef, or other nutritious foods. Farinaceous compounds are least to be trusted to.

Sometimes, and indeed as if it were an integral part of the affection, there may be absolute repugnance to food; a condition, if lasting beyond some short time, which renders ultimate recovery almost impossible. Yet here even much may be done by quiet perseverance, and, failing other means, nutrient enemata should certainly be tried.

Stimulants should be used early and without hesitation; as in cases of fever, the first-noted feebleness of the heart-beat, or softness and rapidity of the pulse, furnish full reasons for their employment. Wine is probably to be preferred to other alcoholic compounds.

Accidental complications, very apt to occur, will call for changes of treatment, and a wearisome illness cannot throughout be treated by the same remedies; but the declared principles hold good that the disease is eminently asthenic in type, and that no lowering agencies are to be deemed admissible.

Extension of local mischief to the larynx may occur in the most simple cases, and certain expedients should at once be tried. Emetics (the sulphate of zinc, for example) may be given, and the child made, as in croupy attacks, to breathe an atmosphere loaded with warm vapour, with other means which may promise relief.

If these, however, fail, tracheotomy may be suggested; in four well-marked cases I have myself done the operation, substituting thereby an easy for a most terrible death, and giving some hours more of life when life was on the point of passing away. The details of the necessary operation may be found in any surgical work. Cautiousness and slowness are the two most necessary points to be observed in performing it. Yet although I would in a suitable case propose and carry out the same procedure, it would still be with the fear that recovery would be most uncertain; not from the local throat mischief, or in any sense from the operation itself, but from the wide-spread taint of the general system. Relief may be promised with fair confidence, a painless death will take the place of an agonising struggle for breath, but a very small percentage of cases ultimately do well.

Supporting treatment, enforced and long-continued rest, with change of air, form the best rules for dealing with the consequences of diphtheritic affections; loss of nerve power is but slowly repaired, and though, in the great majority of cases, recovery may be certainly expected, it is only after a very prolonged convalescence.

The preceding remarks must be looked on as suggestions, hardly indeed doing justice to the very interesting subject which has been put into my hands for this volume of St. Bartholomew's Hospital Reports. For a satisfactory treatment of the affection, and a due recognition of the labours and suggestions of others, very much more space and time would be required.

Enough for my purpose, if I add a fragment to that practical knowledge of every-day disease, without which no useful advances in medicine can be made.

Article IV.—*On the Relation of Phlebitis and Thrombosis to Pyæmia.* By William S. Savory, F.R.S.

THE history of our knowledge of Pyæmia is an instructive one. The formation of abscesses in distant parts after wounds or local injuries, naturally enough, in the first instance, attracted the attention of surgeons. At an early period, the explanation most generally adopted, after some fanciful theories had been set aside, was that they were due to the absorption, transference, and deposition of pus. The abscesses which were discovered after death, or detected during life, were regarded as collections of pus, which had been merely transferred as such by the circulation from the original source to the structures in which they were afterwards found.

This idea was overthrown, chiefly by the celebrated experiments of Cruveilhier. By injecting mercury and ink into the veins, and by introducing mercury into the interior of bones, whence it passes into the circulation, he pointed out that the purulent collections, these secondary abscesses, which are so commonly found in various organs after death, are not simply passive deposits of pus, which has been absorbed and carried by the circulation to this or that part, but that the pus is formed in the situation where it is found.* His experiments led him to the conclusion, that 'every foreign body introduced in the living subject into the venous system occasions, when its discharge from the emunctories is impossible, visceral abscesses, completely resembling those consequent to wounds and surgical operations; and such abscesses are the result of capillary phlebitis in these same viscera.'

* 'I inject mercury into the jugular or into the femoral vein. You will see, if the mercury is in large quantity, that the animal will become extremely oppressed, and succumb in twelve, eighteen, or twenty-four hours. . . . You find the whole of the mercury in the lungs, which are not inflamed, but loaded with serum, which you can express so as to make it overflow. If the quantity of mercury is less, the animal will survive a longer time after the experiment, and you will find then a focus of red induration around each globule of mercury; later purulent collections, still later tuberculous masses, or for the most part a mixture of pus and tuberculous matter; at last, when the animal survives two or three months, you will find tubercles, in the centre of which is a globule of mercury.

'I have destroyed the medulla of the femur; for the medulla I have substituted mercury. The animals (for it was upon dogs that I have practised all these experiments) have lived four or five days, and have presented during their last days great oppression. In the examination I have found all the mercury disseminated in the lungs, and each globule surrounded by a small halo of inflammation. . . . Once I introduced a single globule of mercury into the medullary cavity of the femur, and this globule I found a month afterwards in the lungs; it was divided into many globules extremely small, each of which occupied the centre of a tuberculous abscess.' Cruveilhier, Anatomie Pathologique, liv. xi. p. 5.

Others, however, had, before Cruveilhier, injected mercury into the veins and arteries, and produced abscesses around the globules, which are arrested in the minute vessels. See especially the experiments of Gaspard.*

Dr. Saunders, previous to these, had injected mercury into the crural vein of a dog, and after death found the lungs studded with small indurated masses, which he called tubercles, and small circumscribed abscesses. In the centre of each was a small globule of mercury.

Experiments of injecting oil and other substances into the circulation, which obstructed the minuter channels and produced inflammation, had been performed long before even this.

And it is only fair to relate, that Morgagni,† and others, while they accepted the doctrine of the formation of secondary abscesses, by the absorption and mere passive deposition of pus, which in their time prevailed, yet conceived that in some cases the purulent collection was increased by inflammation.

But notwithstanding the evidence furnished by experiment and clinical research, the idea still continued to prevail in the minds of some, that these purulent collections are simply passive depositions of pus. Those who have thus regarded the secondary abscesses have appealed to the insidious manner in which they are formed, as proof that the pus cannot arise in the part as the result of acute inflammation. They are often formed so insidiously that their existence is not suspected during life; whereas inflammation, it has been argued, attended by such striking and rapid results, must be marked by obvious local disturbance. But the circumstances under which these abscesses are formed, and the condition of the system, must be taken into the account. When the blood is poisoned, sensibility is blunted, for the functions of the nervous system are especially impaired. Local mischief, then, makes but little impression. Witness the insidious character of pneumonia, and of other local affections, during fever.

Another objection taken to the doctrine that pus is formed where it is found, is the comparatively trifling amount of inflammation which is often seen around these purulent deposits, which often accumulate with amazing rapidity. In a great number of cases not the least vestige of inflammation has been found.‡ It has been argued that in acute and rapid cases the

* Journal de Physiologie, 1821, vol. i. p. 165, &c.
† De Sed. et Causis Morb. lib. iv.; epist. li. sect. 23.
‡ Velpeau, Leçons Orales, 1841, p. 74.

inflammation bears no proper proportion to the suppuration. But there is no constant or defined relation between the degree and extent of inflammation and the amount of pus formed. It would be very easy to point out other cases where much pus is found associated with evidence of comparatively little, or even no inflammation; for that which at first existed may have rapidly subsided.

The purulent deposits are, indeed, usually surrounded by a halo of congestion or inflammation; but the degree and extent of this varies widely. Sometimes none can be discovered; at others, the redness extends far into the surrounding tissues. In the joints especially large collections of pus are often found without any visible evidence of accompanying inflammation. Thus an articular cavity may be filled with pus, and when this is washed out, the synovial membrane and cartilages may appear quite natural and healthy.* It may seem at first sight as if well formed pus had been simply poured into a healthy joint. Sometimes, however, there is obviously increased vascularity, and even ulceration. What relation these visible changes bear to the pus, and the influence of time in their production, are considerations of great interest and importance, for the question of the propriety of early and complete evacuation of the pus will mainly turn upon the answer.

When, from observation of the morbid condition which the veins involved in the local mischief are apt to present in these cases, phlebitis was believed to be of common occurrence, the supposed presence of pus in the inflamed vessels was regarded as a demonstration of its source. The first idea that the pus was absorbed from without by the vessels was supplanted by another—that it was produced in and by the inflamed veins.†

* Velpeau, Op. cit. pp. 10, 11.

† The absorption of pus, as pus, is, as Berard, Sedillot, Virchow, and others have shown, a physiological impossibility. Berard says, 'The dimensions of the globules of pus are such that it is stupid to suppose that these globules can pass through the walls of vessels;' and Sedillot observes, that if the globules of pus could thus pass in, there is no reason why the blood globules should not pass out. The fluid portion or serum may pass into the blood, but the cells must remain. They may part with the fluid they contain and shrivel, but there is no means by which the cell wall in its integrity can find its way through uninjured blood vessels.

Nevertheless, it is not correct to say, as Virchow says (Cellular Pathology, p. 196,) that recourse was had to the doctrine of phlebitis to explain the presence of pus in the blood, because it was found necessary to abandon the original view, that pus made its way through an opening in the wall of the vessel or its yawning extremity; for this idea, unlike that of the physiological absorption of pus—that is the absorption of pus by uninjured vessels—involves no impossibility. It is possible that pus may pass into the blood current through a wound or perforation of a vessel—nay from time to time this actually occurs. The doctrine that phlebitis was the source arose from actual observation, that a puriform substance, believed to be genuine pus, was found in the interior of inflamed veins.

Arnott, in his masterly essay in the Medical and Chirurgical Transactions,* after referring the formation of the abscesses in the lungs, liver, and joints, which follow injuries of the head, parturition, great surgical operations, and suppurating wounds, to inflammation of the veins of the part primarily affected and the entrance of pus into the circulation, says, it becomes a question whether the occurrence of phlebitis, and the passage of pus from an inflamed vein into the circulation, are not sufficient of themselves to account for the secondary affections of wounds, without its being necessary to resort to an absorption of the same fluid from their suppurating surfaces.

For some time, however, it was believed that the symptoms of what is now called pyæmia, were due to the extension of inflammation along the veins to the heart.

'In some cases of amputation the veins; in others, the arteries; and in others, again, both the veins and arteries, will be found inflamed, from the point of the stump to the very auricle and ventricle, and in many parts lined with coagulable lymph, or filled with purulent matters, to various distances.'†

'When a vein is wounded, the inflammation, which is the effect of the injury, sometimes extends along the lining of the vessel into the principal venous trunks, and in some instances even to the membrane which lines the cavities of the heart.'‡

Hunter wrote,§ 'it may either be that the inflammation extends itself to the heart, or that the matter secreted from the inside of the vein passes along that tube in considerable quantity to the heart, and mixes with the blood.'

He says farther on, 'in all cases where inflammation of veins runs high, or extends itself considerably, it is to be expected that the whole system will be affected. For the most part the same kind of affection takes place which arises from other inflammations, with this exception, that where no adhesions of the sides of the veins are formed, or where such adhesions are incomplete, pus passing into the circulation may add to the general disorder, and even render it fatal.'

Mr. Arnott‖ showed the fallacy of the doctrine just alluded to, and argued 'that death in cases of phlebitis does not take place from inflammation extending to the heart, but that the entrance of pus or even of some other product of inflammation from the inflamed part of the vein into the circulation is the source of the alarming and fatal indisposition.'

* Vol. xv.
† Hennen's Military Surgery, 3rd edition, p. 271.
‡ Hodgson, On the Diseases of Arteries and Veins, p. 511.
§ Works, 1837, vol. iii. p. 584. ‖ Loc. cit.

Cruveilhier * argued that 'the first effect of all phlebitis is a coagulation of the blood with adherence to the walls of the vessel.'

And again, that the deposit of 'pus' is not between the vein and the clot formed in its interior, but in the very centre of the latter. But he gave a fanciful explanation of this occurrence.

Another fact pointed out by Cruveilhier is that no ordinary mark of inflammation is ever noticed upon the inner surface of the vein in the various stages of phlebitis, that is to say, no injection of capillary vessels can be discovered. The deep red colour is a stain due to imbibition.

Now pyæmia is often witnessed without any evidence whatever of phlebitis. The worst forms of phlebitis often exist without producing pyæmia. Indeed it is very rare to see pyæmia following upon well marked phlebitis during life. Pyæmia is rarely preceded by any obvious affection of the veins.

'It is quite the exception,' says Dr. Wilks,† 'to discover phlebitis as a cause of infection.' Again, 'Amongst the various causes of pyæmia, phlebitis may take its place, although it is very remarkable how seldom the veins can be found affected.' And again, 'So far from phlebitis being a cause of pyæmia, it is remarkable how often the former occurs without any contamination of the blood whatever; that is, if we call that phlebitis where we find a vein and its branches quite closed by coagulum or adherent fibrine.'

Indeed all agree now that pyæmia often occurs without the least evidence of any affection whatever of the veins.

It must of course be admitted that when after death from pyæmia no phlebitis can be discovered, it may nevertheless exist, and escape detection. But this argument has been unduly pressed into the service of those who regard pyæmia as always consequent on phlebitis or some affection of the veins. This objection has been repeatedly urged against the fact that pyæmia often exists when no traces of inflammation of a vein can be discovered. But before any weight can be given to this plea, the connection between pyæmia and phlebitis, or even thrombosis, must be established upon better evidence than that by which it has been hitherto supported.

Then Gulliver ‡ demonstrated the important fact that what

* Op. cit. Dict. de Méd. et de Chir. Prat. Phlebite.

† Guy's Hospital Reports, vol. vii.; also, Velpeau, Leçons Orales, 1841, p. 74.

‡ Medical and Chirurgical Transactions, vol. xxii.

was regarded as pus in the interior of the veins was, although a puriform, not a purulent fluid. The experiments and observations recorded in his admirable paper led him to the following conclusions:

1. 'That coagulated fibrine, when removed from the body and subjected to a blood heat, commences to soften in about forty hours, assuming the colour and consistency of pus, but easily distinguishable from it by microscopic and chemical examination.

2. 'That the purulent-like fluid found in the fibrinous clots of the heart and arteries, and so frequently in the veins, is essentially distinct from pus, and analogous to, if not identical with, softened fibrine.

3. 'That the softening of coagulated fibrine is an elementary pathological condition of frequent occurrence, distinct from suppuration, and constituting a considerable proportion of the cases generally denominated suppurative phlebitis.'

Dupuytren,* but after Gulliver, remarked that the so-called pus in the veins may be due to an alteration effected after death. The greyish clots and puriform appearance may be but an effect of stasis of the blood, of fever during life and of the temperature which remains some time after death.

Veins may inflame. But Virchow and others have shown that phlebitis does not lead to pus in the canal of the veins; nor even, it is affirmed, always to coagula.

Phlebitis 'is an inflammation which really affects the walls, and not the contents of a vessel. In the larger vessels the most different layers of their walls may become inflamed, and enter upon every possible phase of inflammation, and yet all the while their channel may remain entirely unaltered.' 'Hitherto no experimenter who carefully prevented the blood from streaming into the vessels has succeeded in producing an exudation which was deposited in their cavity. On the contrary, when the wall is inflamed, the exuded matter passes into the wall, which becomes thicker, cloudy, and subsequently begins to suppurate. Nay, even abscesses may form, which cause the walls to bulge on both sides like a variolous pustule, without any coagulation of the blood ensuing in the cavity of the vessel. At other times, certainly, phlebitis, properly so-called (and in like manner arteritis and endo-carditis), is the cause of thrombosis, in consequence of the formation of

* Traité Théorique et Pratique des Blessures par Armes de Guerre. Tom. ii. p. 104.

inequalities, elevations, depressions, and even ulcerations upon the inner wall, which favour the production of the thrombus. Still, whenever phlebitis, in the usual sense of the word, takes place, the alteration in the coat of the vessel is almost always a secondary one, and indeed occurs at a comparatively late period.'*

That the fibrinous deposit so often found upon the lining membrane of veins is from the blood, and not lymph from the walls, seems proved by the following facts, which may be repeated here.

It never occurs in a part from which blood is excluded; and lymph has never been produced upon the lining membrane of a vein in any experiment when blood was excluded from the canal.

It can be detached, for the most part, with facility from the lining membrane, which then presents a natural aspect, or is at least only somewhat blood-stained.

Such deposits of fibrine are often continuous with undoubted coagula, which have not yet lost their colour.

Thus the old idea that lymph may be poured out from the lining membrane of a vein, and that pus may be formed in the canal, depended upon a misinterpretation of what was observed in veins which had been plugged by coagula. When clots form in veins they, after awhile, gradually lose their colour, and usually at length disintegrate. Such disintegration, advancing most rapidly in the centre of the clot, gives rise to a granular and oily matter which is puriform to the naked eye, while around there is a shell of fibrine presenting the characters of ordinary lymph.

There is much evidence, but of a very contradictory nature, concerning the existence of pus in veins. However, it may be affirmed that it is not yet established that pus is ever found in veins except under peculiar and extraordinary circumstances, such as those already alluded to.† Just as the evidence of inflammation of the lining membrane of veins, such as the presence of effused lymph, has broken down under critical examination, so the presence of pus in the interior of a vein becomes more rare and exceptional the more thoroughly the subject is investigated.

But Virchow‡ and Kirkes§ have shown how fragments of fibrine, being detached from the cardiac valves or from the interior of vessels where they had formed, or becoming disintegrated in the manner described by Gulliver, may pass into

* Virchow's Cellular Pathology, translated by Dr. Chance, 1860, pp. 202, 203.
† See Virchow, op. cit., p. 183. ‡ Loc. cit.
§ Medical and Chirurgical Transactions, vol. xxxv.

the circulation, be transported to distant parts, and at length, according to their size, block up arteries or capillaries. To this process of obstruction of remote vessels by fragments which have been detached from a disintegrating thrombus, or clot formed in a vein, Virchow gave the name of *Embolia.*

Thus arose the great doctrine of the cause of the secondary deposits of pyæmia, which Virchow has expounded with such masterly skill. According to this, the metastatic deposits have their origin in embolia. A clot, which has formed in some vein, softens and crumbles away, or minute particles are detached from the surface of a cardiac valve. The granules, thus set free, are carried to distant parts, and at length block up the finer vessels. Thus, mechanical obstruction is the starting-point of the mischief. In explanation of the small and very numerous deposits which are often found, Virchow goes farther, and declares it to be a necessary inference 'that, when a considerable fragment of a thrombus becomes wedged at a certain point in an artery, it may, in its turn, crumble away through the onward pressure of the blood, and thus the minute particles to which this crumbling of the larger plug gives rise be conveyed into the small branches into which the vessel breaks up.'*

But clots in veins often occur, and softened ones too, without the occurrence of pyæmia. 'It may be observed,' said Gulliver in the paper referred to, 'that the softening of fibrine certainly occurs sometimes without appreciable suppuration in any part of the body.' This, however, constitutes no fatal objection to Virchow's doctrine, for in such cases conditions may, and often do, exist which prevent the passage of the granules of fibrine into the circulation. But not only may softened clots exist without pyæmia, but, what is more to the point, pyæmia may occur without any evidence whatever of the existence of softened clots.

And when in pyæmia the veins are found affected and filled with coagula, in what relation does this stand to pyæmia? May not both be effects of a common cause—of the absorption of poison which at once contaminates the blood and causes its coagulation in the nearest veins through which the poison first passes, perhaps inflaming, or at least irritating, their walls?† May it not be that thrombi, and even phlebitis, are the local, and 'pyæmia' the general, effect of the passage of the morbid poison into the blood? But neither phlebitis nor thrombi, nor

* Op. cit., p. 207.

† See Dr. F. W. Mackenzie. Researches on the Pathology of Obstructive Phlebitis, and the Nature and Proximate Cause of Phlegmasia Dolens. Medico-Chirurgical Transactions, vol. xxxvi.

any affection of the veins whatever, is a necessary link in the production of pyæmia.

Thus, as the relation formerly supposed to exist between phlebitis and pyæmia was set aside because there is no evidence to support it, so the relation of thrombosis—which has been substituted for phlebitis—to pyæmia is, in its turn, open to at least this objection, that many cases of pyæmia occur which cannot fairly be referred to this cause; many even in which there is no evidence to show, and no reason to believe, that they have been preceded by the formation and softening of any clot.

In the present state of our knowledge, then, the relation in which an affection of the veins stands to the pyæmia appears to be this.

They may be throughout perfectly healthy. For it will not do to argue that when, in pyæmia, no morbid condition whatever of any vein can be discovered, that nevertheless it exists, and escapes detection. This is possible, of course; but in the absence of evidence to support it, with the proof that such a condition is by no means necessary to the event, and that all the effects witnessed in pyæmia may be otherwise produced, such a doctrine has no claim to acceptance.

The veins may be inflamed. Now the only acceptable explanation of the way in which pus—as pus—can pass into the circulation, except in extremely rare and exceptional instances, is that it is formed in the interior of veins as a consequence of phlebitis. Yet not only do cases of pyæmia occur without the slightest evidence of phlebitis, but—assuming that our knowledge of phlebitis is still very defective, and that the present views need some qualification—evidence is wanting to show that in phlebitis any matter is added to the blood, or any change induced in it which causes pyæmia. Yet it is quite possible or probable that the veins, when inflamed, may, like other inflamed structures, furnish some morbid matter which affects the blood.

One or more veins may be occupied or filled with coagula more or less adherent, and in various states; firm and solid throughout, or softened or even diffluent. But, in the majority at least of cases of pyæmia, there is no evidence of the existence of such softened clots, or that such matter has passed into the circulation.

What relation exists between thrombosis and phlebitis?

Among the preparations of affections of the veins, in our Museums of London, are specimens variously described as

phlebitis, thickening of the coats, clots or coagula in veins, in which—

A clot, more or less filling the canal, occupies the interior of an apparently healthy vein, no thickening or alteration of texture of the walls being visible.

A clot, more or less shrunken and decolourised, or a layer or layers of fibrine, occupies the interior of a vein, the walls of which are obviously more or less thickened, and otherwise altered in texture.

But I do not find specimens where the walls are obviously thickened, presenting those changes which are attributed to phlebitis, the canal of which is nevertheless free from clot or fibrine.

So that here is evidence that a vein may be plugged without such previous alteration of structure as is attributed to phlebitis, although such changes may ensue upon the plugging, but there is no evidence that veins undergo the changes caused by phlebitis without any plugging or deposit of fibrine within the canal.

Now Virchow says: 'We do not know that inflammation, as such, has any necessary connection with coagula; on the contrary, it has turned out that the doctrine of stasis rests upon manifold misinterpretations. Inflammation may unquestionably exist when the current of blood within the vessels of the affected part is perfectly free and unobstructed.'*

Although here, and in another passage previously quoted, Virchow speaks positively of the existence of phlebitis, and even of its consequences, such as pus in the walls, without any coagulation of the blood in the cavity of the vessel, such a condition must surely be, to say the least, extremely rare.

When there is pain and much tenderness in the course of the vein, with an increase of heat, when there is swelling and some hardness, with a blush along the surface over the affected vessel—when, in a word, that condition exists to which the term phlebitis is generally applied, there is usually evidence that the vein is plugged with clot. Not that this fact can be by any means always clearly made out, for a thickening of the coats of the vessel themselves will give to the touch the sense of solidity; but, making full allowance for this, the signs of thrombosis during life are often confirmed by examination after death, which discloses a clot throughout the vessel. So too, in these cases, the structure of the vein is generally affected. The coats, especially the outer ones, are thickened and hardened, so that the wall has lost its natural pliancy and elasticity,

* Op. cit., p. 198. See also Lee on Diseases of the Veins, 1866, p. 11.

and is unnaturally rigid; the vessel standing out like an artery when divided. Now, in what order are these changes induced? Which occurs first—the changes in the wall of the vein or the coagulation of the blood in its canal?

In some cases unquestionably the coagulation, for it sometimes happens that a vein, which previously gave no sign whatever of being in any way unhealthy, will suddenly become plugged throughout, and at this time the most careful examination can detect no change whatever in the walls of the vessel. In such a case the clot may shrink or disappear, and the vein may resume its natural condition, no traces of the previous mischief remaining, or there may ensue upon the thrombosis those symptoms of phlebitis and those changes which lead to the alterations of structure just alluded to.

On the other hand, but these cases are undoubtedly more rare, the symptoms of phlebitis seem to precede the formation of a clot in the canal. For some time after pain and tenderness have existed in the course of a vein evidence may be obtained, by pressure and otherwise, that the blood still flows through it. If the mischief progresses, however, this evidence soon disappears.*

Thrombosis, then, without phlebitis, is evidently of more frequent occurrence than phlebitis without thrombosis.

But if either may exist without the other, what is the cause of thrombosis?

It must be confessed that thrombosis sometimes occurs, and these are the most striking cases, under circumstances in which the cause of it is altogether a mystery. When clots form in portions of veins that have been torn or bruised, or otherwise damaged, we may explain the mode in which they are produced. When they arise in veins which communicate with wounds or diseased structures, or even in veins which are compressed so that their current is interrupted, we may still assign a cause for the result. In an altered condition of the venous channel, in some abnormal state of the blood passing through it, or in some obstruction to its flow, we may perhaps discern something of the cause which determines its coagulation. But in our museums specimens may be found of veins which are plugged with clot, the coats of which are nevertheless apparently quite healthy. If, then, we conclude that healthy vessels may become plugged, we must seek elsewhere than in the vein for the cause of the coagulation. But it may be that incipient changes in the lining membrane of the veins, which

* See Lee, op. cit., pp. 13 and 24; by whom this question of the relation between thrombosis and phlebitis is admirably discussed.

may not yet have advanced sufficiently to be observed, may still have determined the coagulation of the blood. It may be so when the coagulum is recent. But this idea is hardly tenable against the fact that there are specimens of veins which contain old clot, clots which have existed long enough to undergo the usual changes in vessels, the walls of which are still free from any visible morbid change. If the formation of clot had been determined in these cases by the morbid state of the vein, it is reasonable to believe that during the time the clot has been undergoing the ordinary changes the disease of the vessel would have become apparent. From the consideration of these latter cases, then, the conclusion is drawn, not only that clots may form in healthy veins, and that, therefore, in some cases at least, the cause of the coagulation must be independent of any disease of the vessels themselves, but that the existence of a clot in the canal does not necessarily provoke disease in the walls of the vessel.

Now when a certain vein, or series of veins, apparently in all their integrity, communicating with no diseased or injured part, and carrying blood like that which circulates through neighbouring and other veins—when a set of veins under these circumstances becomes almost suddenly filled throughout with clot, where, in what direction, are we to look for the cause? Is it in the vessels or in the blood? The vessels have given no previous evidence whatever of any morbid condition; nor, perhaps, will any be discerned afterwards by the most minute scrutiny. No local disease exists, nor has any injury been sustained. And let it be observed that, even for the moment assuming one of these, it will not account for the extent of the clot, for coagula formed in such cases are limited in their extent. Again, if the blood be in fault, why is the effect so local, limited to certain veins, or, indeed, to the veins at all? for it would appear, as a rule at least, that arterial blood deposits fibrine more readily than venous. The assumption of some spot of mischief as a starting-point would not go far enough; the assumption of some affection of the blood would seem to prove too much.

Can it,—for this has been suggested,—be due to a very languid movement of the blood through the vessels in which it at length coagulates? Such a condition of the circulation, however it may assist, can hardly determine the formation of the clot. If this were the cause, why is the effect so limited? Lee remarks, 'a retardation in its movement can hardly yield the explanation; since, then, coagula ought to be found in the smaller tributary veins and capillaries, out of all proportion

to those in the larger venous channels.'* And again, although a feeble venous current is, no doubt, often associated with thrombosis, yet it is by no means always so. Veins are sometimes plugged in individuals in whom there is no evidence of any unusual failure of the circulation. And lastly, blood is not very prone to coagulate even when at rest in healthy vessels. It is a common remark how long it will remain fluid under these circumstances. Nay, even when effused from vessels into surrounding parts, one often observes how long it will remain fluid. No, a retardation of the current cannot be the sole cause. To be sure, clots will sometimes form in the vessels of the dying. Occasionally evidence is presented of the formation of clots even of considerable magnitude before death, and this is, no doubt, then associated with a very feeble failing circulation. But the languid circulation and the clot must not be viewed in the relation simply of cause and effect, without any consideration of other influences which are probably acting.

It has been remarked that thrombosis is more common in the left than in the right lower extremity, and suggested that this may be due to some retardation of the returning current in the left limb from the pressure to which the left iliac vein is subjected as it passes under the artery. Such a condition may assist, but can hardly determine the formation of the clot, or why is it not produced by any considerable pressure upon the principal vein? Why does it not follow every attempt to cure popliteal aneurism by pressure?†

Again, it has been broadly asserted that the blood in these cases is charged with a great excess of fibrine, but I am not aware of the evidence which establishes the fact.‡

In truth, the suggestions just alluded to show how very far we are at present from any knowledge of the pathology of thrombosis.

* Op. cit., p. 16.

† Dr. Mackenzie, speaking of Phlegmasia Dolens, has noticed the greater liability of the left lower extremity to the disease, as compared with the right, and remarks that the relative frequency corresponds very nearly with that of the attachment of the placenta to the left side of the uterus as compared with the right. And he says that, if the relation here indicated should be hereafter borne out by further facts, it would furnish an argument in favour of the origin of the disease from vitiation of the blood. 'For it is where the placenta has been attached that the uterine wound will be left on its removal, and it is from this that unhealthy secretions will be poured forth in cases of local injury, inflammation, or constitutional disorder, whilst here, also, these secretions will be most favourably placed for reabsorption, by being in contact with the orifices of the uterine veins which had been torn across by the removal of the placenta.' Op. cit.

‡ In reference to these questions, see Humphry on the Coagulation of the Blood in the Venous System during Life, 1859.

Still in the midst of the darkness may be found, perhaps, some clue to the direction from which light may be hereafter thrown upon the subject.

However much the formation of clots in veins may be influenced by the quality of the blood and the rate of its movement, the determining or essential cause must surely be, in many cases, a local one; otherwise it would seem that coagulation ought to be more general, and not limited to this or that particular vein. It might be, on the one hand, that such local cause would often prove inoperative unless assisted by an increased disposition to coagulate on the part of the blood, and yet, on the other hand, if sufficiently intense, it will speedily induce the formation of a clot, be the condition of the blood what it may. Thus thrombosis appears to be the almost inevitable result of certain changes in the walls of the veins, and I do not remember to have seen a vein which was thickened and indurated, and exhibiting the changes usually attributed to phlebitis, the canal of which was nevertheless free from clot. That the disposition of the blood to coagulate is powerfully influenced by the nature of the surface with which it is brought in contact, seems to be a well-established fact. It was pointed out by Hunter, has been more or less clearly recognised ever since, and recently more fully demonstrated by the experiments of Lister. Thus, whatever may be the cause of it, there is a striking difference in this respect between dead and living parts, and living parts themselves possess different degrees of influence. So Hunter, after pointing out that 'the want of disposition to coagulate whilst moving in the living vessel does not arise from the motion,' goes on to say, 'This harmony of the blood with the solids is more observable in some parts than others. The parts with which it is in the greatest harmony are the vessels; this is evident from its retaining its fluidity longer in contact with them without motion than in any other part of the body, though equally enclosed in living parts.'* But this, of course, applies to the blood and the vessels only in a natural and perfectly healthy state, and it is not difficult to believe that any deviation from this—whereby the harmony is disturbed—must proportionately tend to determine the formation of clot.†

* Works, vol. i. pp. 232, 233.

† Dr. Mackenzie has argued, and adduced experiments in demonstration of the fact, 'that whilst phlebitis may occur in a variety of forms, without giving rise to extensive obstruction of veins,' the coagulation of the blood in their canal may be referred 'to irritation or excitation of their lining membrane immediately induced by the presence of morbid matters in the blood,' such coagulation depending 'upon a disturbance of the relations which normally subsist between the blood and the

With reference to the causes of thrombosis, and more especially to the tendency of the blood itself to coagulate, it should be observed that the clots found in the interior of veins may fairly be divided into two classes. In one, the clot is a simple solid cord, and presents on section an uniform structureless surface. In the other, the clot is distinctly laminated, and a section shows a number of concentric layers. This difference, which is equivalent to that observed in the interior of aneurismal sacs, must imply a difference in the mode in which the clot was formed. In the first case, it would seem as if the whole of the blood with which a vein is filled had throughout coagulated, the vessel subsequently contracting upon and adapting itself to the clot as it shrunk. In the second, it would seem as if that portion only of the blood in direct contact with the wall of the vessel had deposited a layer of fibrine, and that within this another had afterwards formed, and so on. This view is strengthened by what is often observed in the examination of these cases. In recent ones, sometimes the whole canal of a distended vein is found filled with a large dark soft uniform clot. In other cases, perhaps old ones, when the canal of a large vein is laid open, it may, at the first glance, appear of its natural size, and little or no thickening of the wall may be observed, but more minute observation shows that upon the lining membrane is deposited a thin layer of fibrine. Further, it is to be observed, what from this point of view would be anticipated, that when the contents of veins of about the same normal diameter are compared, those clots which are formed of concentric layers usually far exceed in calibre those which are uniform throughout.

But to conclude, the sum of all that has been said amounts to this—

Thrombosis may exist without any evidence of phlebitis, and very often occurs without being followed by pyæmia.

Phlebitis may occasionally exist without thrombosis, and often occurs without being followed by pyæmia.

Pyæmia often exists without any evidence of thrombosis or phlebitis; still oftener it occurs without any evidence whatever

lining membrane of the veins, and that this is immediately due to irritation of the latter.' Op. cit.

But while it may be easily conceived, and indeed demonstrated, that a change in the lining membrane of a vein will induce coagulation of the blood in its canal, it is difficult thus to account for the ordinary phenomena of thrombosis. There is no proof or probability that in many of these cases the blood is vitiated, and assuming this, why should the effects be so local—why should certain veins only suffer? But further, as a matter of fact, the blood may be, and often is, vitiated in various ways without, as a consequence, the occurrence of thrombosis.

that it has been preceded by either of these, or of any other affection of the veins.

It has therefore not been satisfactorily shown that either phlebitis or thrombosis stands, in any especial or peculiar manner, in relation to pyæmia, as cause and effect.

Although the ancient idea that the affection termed pyæmia depends on the presence of pus in the blood is no longer generally received, yet there can be little doubt that it still continues to influence largely the views which are taken of its pathology. That the disease is still so commonly associated with what was called phlebitis is due to the old idea that in this way pus is introduced into the circulation. It seems to be a great step onwards to recognise the fact that the disease may occur independently of the presence of pus, or of any affection of the veins whatever—that it is not due to any specific or peculiar matter formed in this or that particular part, but that it is the effect of blood-poisoning; due to the introduction into the circulation of morbid or putrid matter. No doubt pyæmia is often associated with the formation of clots in certain veins, but this coincidence by no means implies the relation of cause and effect. When veins are found occupied by disintegrated clot after death from pyæmia, it has still to be shown that this was the cause of the disease. On the contrary, it can be shown, so far indeed as the most careful and critical examination can go, that pyæmia may occur independently of any affection of the veins whatever. And it is a question whether, in those cases in which the veins are plugged or inflamed, thrombosis and phlebitis are not the local, and pyæmia the general, effect of the same cause.

No doubt pyæmia is almost always associated with the presence of pus somewhere; but this association does not of necessity imply the relation of cause and effect. The almost constant formation of pus in these cases may be otherwise accounted for, and there is no more evidence of any especial relation between pus and pyæmia than this, that pyæmia is the result of the passage of putrid fluid into the circulation, and that pus is of very common occurrence, and liable, like other animal fluids, to become putrid. It is only when pyæmia is disentangled from any special or specific local affection that there will be any chance of a full and free investigation of its pathology.

ARTICLE V.—*Case of Intra-cranial Cyst containing Hair—also a Case illustrating the Physiological Action of Iodine.* By WILLIAM TURNER, M.B. Lond., F.R.S.E.

CASE I.—Intra-cranial cysts with hairy contents rank amongst the rarest of morbid products. A short time ago I met with a small specimen within the cranium of a male child æt. 23 months, whose body was undergoing dissection in the anatomical rooms of the University of Edinburgh. When the dura mater was stripped off the inner surface of the occipital bone, a small well-defined tumour, about the size of a French bean, was seen to be situated outside it, and lodged in a small depression on the inner surface of the occipital bone, immediately below the lateral angle of the left lateral sinus, close to the occipito-mastoid suture. Where the cyst was lodged the bone was bare, thinned, and somewhat roughened, but there was no aperture of communication with the exterior. Except at its upper end, where a fibrous band connected the cyst to the lower margin of the groove for the lateral sinus, the cyst was perfectly free, and could be turned out of its fossa with the handle of the knife.

The cyst possessed a perfect wall of thin, delicate membrane, through which the white contents could be seen without difficulty. When this membrane was slit up, it was found to enclose a mass of opaque, almost pearly-white, substance, in the midst of which a small lock of loose hair was imbedded. When examined with a magnifying power of 200 diameters, the white substance was seen to consist of flattened, oval, or somewhat angular cells, in which no distinct nucleus was visible. Their flattened, scale-like form reminded one of the pavement epithelium, or superficial cells of the cuticle, with which indeed they may be compared. From the pearly lustre of the mass I thought that crystals of cholestearine would have been present, but none were detected after a careful examination.

The hairs varied in size from a quarter of an inch to an inch in length. Some were fine, others much coarser. Some were complete in form, with bulb, shaft, and point; others, again, were broken at the extremities. In their structure they presented the customary characters of human hair. No other characteristic skin structure was observed.

The cyst evidently belonged to that group of tumours which Mr. Paget has designated as cutaneous proliferous cysts; one of the best marked examples of which is the specimen in the

museum of St. George's Hospital, in Mr. Cæsar Hawkins's collection. From a carefully drawn up description of this specimen, recently published by Dr. J. W. Ogle, in the 'British and Foreign Medico-Chirurgical Review' for 1865, it would seem that in its epithelial and hairy contents my specimen exactly corresponded with the one in the St. George's Museum; but in their external relations a very important difference existed, for whilst the ossification of the cranial bones in the former was complete for the time of life of the child, in the latter the depression in which the cyst was lodged communicated with the exterior of the skull through a foramen, situated in the mesial line of the supra-spinous part of the occipital bone.

Case II.—Cases of local Iodism, produced by the direct action of the vapour of iodine on the mucous membranes, are not of very frequent occurrence. As a case of this kind, however, came under my notice a few years ago, a brief narration of the effects produced may prove perhaps of interest. An experimental chemist had been occupied for several hours on each of two successive days in laboratory work, in the course of which the fumes of iodine and hydriodic acid were constantly being evolved from a retort, so that the atmosphere of the room had become gradually impregnated with them. On the evening of the second day his eyes felt sore, and smarted painfully, and the secretion of tears was increased in quantity. These symptoms became more marked during the night, and the next morning the eyelids on the cutaneous and mucous aspects were much swollen, very red and painful, and lachrymation was profuse. Exposure of the eyeballs to light produced intolerable smarting, so that the patient was obliged to sit in a darkened room. Movement of, or pressure on, the eyeballs greatly increased the pain. At the same time there was a most profuse 'watering' from the nose, the secretion literally running from it. There was occasional frontal pain, increased on exposure to light, but no salivation or soreness of the gums. Beyond a feeling of depression, no constitutional symptom existed; the tongue was clean, pulse quiet, appetite good. Towards evening the irritation of and discharge from the nose and eyes were greatly relieved, and the next morning they had almost entirely disappeared. No medicine of any kind was taken.

Writers on therapeutics have long been familiar with a class of symptoms occasionally produced by the administration, in-

ternally, of iodine and its chief medicinal compound, the iodide of potassium. Dr. Christison (Poisons, p. 202) states that the most remarkable idiosyncratic effects are such as imitate sometimes catarrh, at others cold in the head, and again, at p. 197, that during a course of iodine, chronic irritation of the Schneiderian mucous membrane is apt to be occasioned. Various cases have been published by Erichsen, Lawrie, and others, which show that, in persons constitutionally predisposed, a few grains taken internally have produced great irritation of, and discharge from, the nasal and conjunctival mucous membranes. In the present case, symptoms of a similar kind were produced by the stimulating action of the vapour, diffused throughout the atmosphere, on the membrane itself. The action was local, not constitutional, for on testing the air expired from the lungs, by passing it through a cold solution of starch, no blue colour was produced, which would seem to prove that the iodine had not been absorbed into the system.

Article VI.—*Remarks on the Use of the Thermometer in Disease.* By John Southey Warter, M.D. Edin.

In most observations of the thermometry of disease that have been previously published, the patient's temperature has been taken both morning and evening, and from these two daily observations conclusions have been deduced. The following remarks, however, are founded on a single daily temperature taken at the time at which the patient's other symptoms were observed, all the observations having been taken in London during the last two years, between the hours of 12 and 4 p.m., and with an English thermometer obtained of Mr. Casella.

The healthy temperature of the body seems to differ slightly in different persons; but probably, after a patient has been an hour in bed at midday, anything below 96 and above 99·5 is abnormal, and I believe the exceptions are very rare in healthy persons where the temperature will be found below 97 or above 99 degrees Fahrenheit at that time. A rise of a degree of temperature is accompanied normally by a rise of about ten beats in the pulse, but this ratio does not hold good in many diseases.

In health there is a normal daily elevation often from 2 to 2·5 degrees Fahrenheit, the morning temperature being the lowest and the evening one the highest. A full meal and the ingestion of alcohol is also said to lower 'pro tempore' the heat of the body.

The actual temperature of the body cannot be estimated by the hand, the skin sometimes feeling hot when the temperature is normal, and again a high temperature being registered by the thermometer when the hand perceives nothing abnormal; this fallacy is supposed to depend on the amount of moisture present on the skin.

In some diseases, as pneumonia and typhus fever, the heat of the patient's hand or lower arm may generally be taken as a guide in guessing at the heat of the body. In others, as typhoid fever, the hand may feel perfectly cold, while the temperature of the body, as marked by the thermometer in the axilla, may be as high as 102 to 104 degrees Fahrenheit; this, apart from thermometry, is a practical fact which it is well never to lose sight of. Again, in some cases the mercury in the thermometer mounts very rapidly, and soon attains its greatest height; while in others it seems to creep up, and the instrument must be left some time in the axilla before the temperature is finally registered.

In many diseases the pulse and temperature rise and fall together, so that from the observation of one we may often infer the other correctly; this, however, is not always the case, as in typhoid fever, for instance, the pulse ranges low at first, while the temperature is high. Then, on the other hand, we may have a rapid pulse arising from weakness or nervousness, the temperature being nearly normal; and in illustration of this I may state that I have taken a pulse beating 184 in the minute, when the temperature of the patient at the same time has been only 98°·8 Fahrenheit.

The frequency of the respirations does not seem to influence the temperature of the body so much as we might expect, and this may no doubt be explained by the fact that the number of respirations per minute do not indicate the amount of air taken into the lungs, or the actual oxygenation of blood that is going on in the system. The influence, however, that aeration exerts on temperature is very evident; thus, in a case of croup, of which I took notes, the pulse was 183, the temperature 99°·4, and the respirations 50, on the first day of the disease. Tracheotomy was performed the same evening, and on the next day, though the pulse had only risen 3 beats, and the respirations 13, the temperature had risen very nearly 3°·5. Surely, then, in cases like this we cannot doubt that the temperature was kept down at first by the blood not having sufficient oxygen supplied to it for its proper and complete aeration.

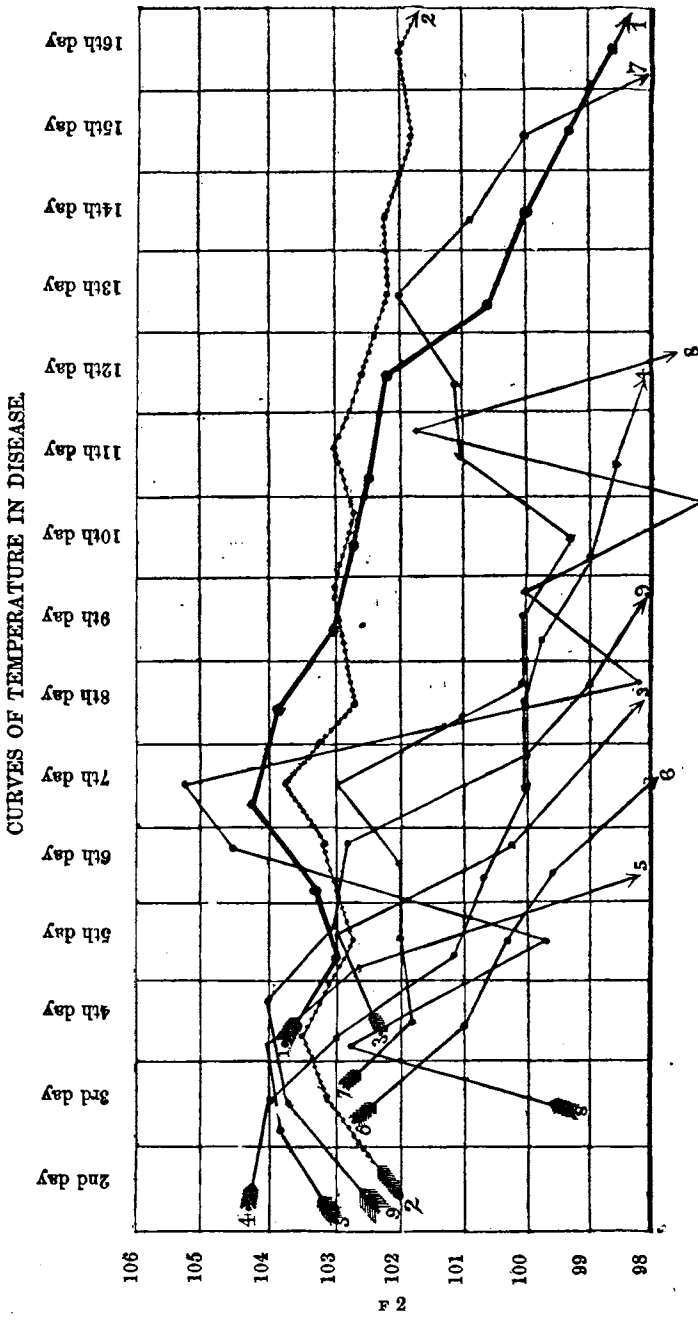
In many cases recovering from acute disease where the temperature sinks rapidly, it falls down for a time below the standard of health, and then again mounts up slowly to the normal.

The objection having been often made to me that, as morning and evening temperatures vary considerably, one single mid-day observation is of no value, I have attempted to see if any general rules could be given by which a physician might tell in what diseases any great variety in this respect is likely to occur. As far as I can ascertain, two great classes of disease seem to exist:—In the first, which includes pneumonia, scarlet fever, measles, typhus, rheumatism, natural and modified small-pox, the difference between the morning and evening temperatures varies from 1° to 3°, usually, however, being only from 1° to 2°. In the second class, on the contrary, 3°, 5°, and even 8° difference is to be commonly noted; and under this head are to be found simple and protracted ephemera, erysipelas, and intermittent fever: typhoid fever, according to its stage, partakes first of one and then of the other class, in the first fortnight the difference between morning and evening being about 2°, while after that the temperature will rise during the day as much as 4° or 5° Fahrenheit. The time at which changes of temperature seem to occur most is from night to morning, and this holds good more or less in all diseases, both in the increase and decrease of the thermometric wave.

Blood Diseases.—These general observations having been made, I now pass on to discuss thermometry in more detail, and the first class of diseases I shall take up is the one entitled Blood Diseases, typhus and typhoid fever being those of which I shall first speak: before commencing, however, I think it but fair to state that about half the mid-day temperatures on which these remarks are founded were taken by my friend Dr. Compton;* though for the diagrams and conclusions set forth in this paper I am alone responsible.

As far as I have noticed in typhoid fever, as a general rule, the temperature of the body rises high in the first week, while the pulse remains comparatively low, and this is not the case in typhus or in many of those other diseases for which this fever is apt to be mistaken in its early stage. Thus, in typhoid fever I have only one or two records of a pulse up to the eighth day higher than 120, and this number is uncommon, most of them ranging from 90 to 110 beats per minute; in all these cases, however, the temperature was as high as from 102°·4 to 104°·3 Fahrenheit. Exceptional cases to this rule occur, as might have been expected, in young children, on account of the normal rapidity at which the pulse beats at this age.

* An article by Dr. Compton, giving the results of his observations, conducted in the wards of St. Bartholomew's Hospital, is published in the Dublin Quarterly for August 1866.—Ed.



In typhus fever running its usual course there seems a direct ratio between the rapidity of the circulation and the heat of the body; the pulse and temperature rising together till the fever reaches its climax, when the pulse ranges generally from 110 to 144, and the temperature from 103° to 104°·5. At one time I used to think that a low pulse and high temperature at the commencement of fever always indicated that it was specifically typhoid, but more extended observation has taught me that there are many exceptions to this rule; thus occasionally in febricula, acute rheumatism, tonsillitis, exhaustion from suckling, and phthisis pulmonalis, a temperature of 102° or 103° may occur with a low pulse-rate; and these exceptions are the more worthy of our attention as the aspect is in them often typically that of the early stage of typhoid fever; thus in all there may be a clear, white, and perspiring skin, red lips, tinted cheeks, and dilated pupils, and hence without some closer investigation a superficial observer may be often easily deceived in his diagnosis. Perhaps the cases of enteric fever in which the diagnostic use of the thermometer is most striking, are those in which malaise and perhaps some diarrhœa have existed for several days and yet the patient has got about; one day at length, however, the doctor is consulted, and finding the tongue clean and the pulse quiet he is thrown off his guard, and is about to think but little of the attack; wishing to make sure, however, ere he dismisses his patient, he places his thermometer in the axilla, and the mercury rising to 102° or 104° shows him that it is typhoid fever, and not a simple diarrhœa that he has to deal with. Again, in how many cases of supposed typhus does the thermometer, by marking a low temperature, contradict every other symptom! There may be a history of exposure to infection, a rigor may have occurred, the face may be muddy, the eyes injected, the pupils small, the pulse rapid, and the tongue thickly furred; still, if with all this the temperature at mid-day is below 99°·5 or 100°, typhus fever, at any rate, may be stated positively not to exist. Of course in saying this I do not mean to include those cases in which the rash is out and the patient is collapsed, or those cases in which the fever is really past but the black tongue and rapid pulse of exhaustion exist; these tell their own tale and require no such instrument as the thermometer to aid in their diagnosis.

The curve given to represent typhus may be taken as a fair type of the afternoon ranges of temperature in this disease. The most common days on which the climax of temperature is reached are the seventh and ninth from the first rigor, and of these perhaps the ninth is the most common.

The diagram represents the seventh as the highest temperature, as this seems the most simple form of the disease. In a fair proportion of cases the acme of typhus is reached on the eighth day, and it may occur, as a rare exception, as late as the tenth, twelfth, or possibly the thirteenth. There seems a decided tendency in many cases of typhus for a rise of temperature to take place on alternate days, and especially on the odd ones; of these a rise on the thirteenth day is most common, and after this a rise on the eleventh. The fever of typhus ends usually by lysis (gradually) about the fifteenth, sixteenth, or seventeenth day, but it may end suddenly in any case by crisis a day or so after the highest temperature has been reached.

The curve given to represent typhoid fever may also be taken as a fair sample of this disease; the highest afternoon temperatures in it occur probably as early as about the fourth day. In it also there is a tendency to rise on alternate days, especially on the thirteenth, and the ninth, eleventh, and fifteenth; but it runs a course longer than typhus, ending generally some time from the twentieth to the thirtieth day. High temperatures may be occasionally reached at any time during its course, and its decline is gradual, and, as far as my experience goes, seldom ends by crisis.

In some cases I believe that at the end of typhoid fever constipation may keep up the mid-day temperature. We are all so afraid of giving purges in this disease, that at its termination after diarrhœa has been once checked, the bowels are apt to get loaded and irritated, and possibly in consequence of this the temperature of the patient keeps up abnormally high. At any rate, as a fact, I know that in many cases when the patient is getting better, the temperature comes to a stand and keeps up more or less high for some days, no reason for it apparently existing except that the bowels are confined; then in the course of time a free action of the primæ viæ takes place, the temperature sinks at once, and the patient rapidly gets well. I believe also that in cases of exhaustion confined bowels will elevate the temperature, but these two latter statements require confirmation, and so I only throw them out as hints for other observers to keep in mind.

As regards prognosis in typhus and typhoid fever, the most important rules which can be derived from the records of a single daily temperature seem to me to be two; namely, that a sudden rise of temperature in either indicates some intercurrent disease, as congestion of the lungs, and that a sudden fall of the thermometer in typhoid fever indicates either diarrhœa or hæmorrhage.

In treating fevers, at any rate towards their terminations, I think we may derive some hints from the use of the thermometer, for by its marking a normal temperature when perhaps the pulse is frequent, the patient constantly delirious, and the whole mouth black with sordes, it shows us plainly that these symptoms are not the result of cerebral inflammation, but depend in all probability on exhaustion; hence nutrients and stimuli appear the rational curative agents, versus leeches, cupping, and other lowering remedies. Counter-irritation behind the neck may, however, be employed with advantage in these cases; the rationale of its action is not very apparent, but I suspect that in some way by reflex action it increases the tone of the cerebral arteries, and that thus again a healthy balance is restored between these vessels and the tissue which they supply.

The study of thermometry in typhus and typhoid fever, puts, I think, an end to the question as to whether these two depend on separate poisons, or are really one and the same disease. In typhus the pulse and temperature rise more or less together until the seventh, eighth, or ninth day, when they are at their highest, then they begin to decline, and drop down steadily day by day in unison, both becoming normal about the fifteenth or sixteenth day. In typhoid fever, however, all is chaos; the pulse and temperature rise and fall irregularly, taking separate and distinct courses. Both, however, remain, on the whole, moderately high from about the sixth to the fifteenth day, then a fall takes place pretty rapidly, and between this and the twentieth day the normal mid-day temperature is again reached in most cases.

One more interesting problem will, I think, be ultimately solved by our thermometrical tables. I allude to the much-argued change of type of disease, for surely if fifty years hence we find the curves of temperature in cases of pneumonia and typhus the same as they are now, it will go far to prove that much of the so-called change cannot really have taken place.

The range of temperature in measles is a short and rapid one, thus contrasting well with that of typhus fever, for which this disease is apt to be mistaken. As far as I have observed, it rises to its highest point about the fourth, fifth, or sixth day, and then again sinking rapidly, it falls to the temperature of health about the seventh or eighth. Thus, after the sixth day, at any rate, its course is diametrically opposite to that of typhus; for while in measles after that date both the pulse and temperature are falling rapidly, in typhus fever they are both rising, and on the seventh or eighth day, when the temperature of a case of measles is normal, the temperature of

typhus is at its greatest height, ranging from 103° to 105° Fahrenheit. The highest temperature reached in measles is not very great, being rarely higher than 103°; the disease ends usually by rapid lysis, the pulse and temperature falling together. In the curve which is given to represent measles, it is shown how, after reaching its acme of temperature comparatively early, the thermometric wave takes a rapid downward course, being thus entirely distinct from the one representing typhus fever. It may be well to give a caution here as to the fallacy of intercurrent lung disease setting in, for pneumonic congestion of the lung occurring about the fourth or fifth day in measles will keep up the patient's temperature, and make the thermometrical range, taken alone, simulate typhus. As, however, in such cases the lungs would naturally be ausculted, this fallacy is not likely to interfere with actual practice.

In scarlet fever as a rule, the temperature rises to its greatest height from the second to the fifth day, the pulse also being highest at this early date and by its extreme rapidity often aiding in diagnosis; from this point the temperature declines gradually, attaining to the normal about the tenth day. From the study of those cases which I have taken, I imagine that the temperature of a fairly severe case on the second or third day would usually range as high as 104°; in mild cases, however, the temperature need not be elevated to any great extent, and I have taken one on the third day as low as 99° Fahrenheit. In the curve given to represent this fever it will be seen that although this disease reaches its highest point of temperature sooner than measles, it yet takes a longer time in subsiding to the normal standard.

Febricula has no special temperature that is diagnostic, as far as I have observed, though it sometimes closely simulates typhoid fever, having in common with it a low pulse-rate, moderately high temperature, moist or sweating skin, and furred tongue. It runs, however, a shorter course, no rash appears, and diarrhœa is not a prominent feature among its symptoms. The curve representing it has only been drawn to give an idea of the usual course of this disease, and not to indicate any absolute specific character of its rise and fall in temperature. As regards prognosis, high temperatures in it do not necessarily imply danger.

Tonsillitis at its commencement has sometimes many symptoms in common with typhoid fever, and when it comes on in a patient convalescent from that disease, may simulate a relapse both in temperature and general symptoms; an inspection of

the throat will, however, at once reveal the real 'fons et origo mali,' and it is well to look out for this in doubtful cases, as sore-throat is often a thing very little complained of. The same remark applies to the curve representing the temperature in this disease as applies to that of febricula, and the time during which fever is present in it is generally of short duration.

The afternoon temperature of patients suffering from acute rheumatism does not seem to rise very high. Out of 142 observations that I made in this disease, some of the cases being complicated with pericarditis, there was not one where the mid-day temperature exceeded 103°, and in 130 out of the above number the temperature rose no higher than 102° Fahrenheit. I have collected also out of Dr. Davies' pamphlet 'On the Treatment of Acute Rheumatism by Free Blistering,' 135 observations. Of these only five were above 102°, one being 103°·5, and the other four 103°.

Now this seems to me to be a point that may possibly help the surgeon in the diagnosis of acute pyæmia coming on after an operation, for in this latter disease the temperature generally ranges high, and hence differs considerably from that of acute rheumatism, where certainly a temperature above 102°·5 at mid-day is a rare exception. In cases of chronic pyæmia the temperature does not keep high as a rule, and so taken alone is not characteristic; still, in a doubtful case, a pulse above 120°, and a lowish temperature, may add one more point to other symptoms in forming a diagnosis of pyæmia, as cases of rheumatism where the pulse is above that number, and the temperature is low, are certainly rare. Out of 196 observations of the number of the pulse per minute in acute rheumatism, in 160 it was below 111°, in 29 below 121°, and in only one did it attain 124°. The curve given represents fairly the range of temperature during the course of acute rheumatism, but is not meant absolutely to imply that on a given day, as the 14th, the temperature is always to be 100°·9. In all cases of acute rheumatism auscultation should never be omitted, as the stethoscope will detect the onset of cardiac disease before either the pulse or temperature begins to rise. Rheumatic pericarditis does not give rise to a very high temperature, the majority of cases being between 102° and 103°, or even lower. Rheumatic pneumonia also, as far as I have seen, does not raise the temperature of the body so high as idiopathic pneumonia does, the difference being nearly 2°; apart from the difference of temperatures in the two diseases, I am sure that, as a rule, rheumatic patients will live with considerably more mischief in the cavity of the thorax than can be borne by a

patient who has not been previously prepared, so to speak, by the rheumatic poison.

In facial erysipelas with relapses, the temperature rises and falls in the most extraordinary manner, but does not seem to me, as far as the erysipelas is concerned, to give any information of more value than can be gained from other symptoms. As, however, the erysipelas subsides, the use of the thermometer comes into play, for should the temperature persist some degrees above normal when the eruption has faded, it points to the occurrence of some of the sequelæ which we know to be common after this disease. In this disease, though the temperature may range very high, the pulse is comparatively a low one; as, for instance, a pulse of 116 with a temperature of over 105°. The curve given to represent this exanthem is not meant to be typical, but only to show its extraordinary, erratic course when complicated with relapses.

In active tuberculosis the temperature is always elevated some time during the day, and it also follows the usual rule of being higher in the evening than in the morning. Mid-day temperatures in phthisis pulmonalis, usually range from 99°·2 to 102°, though sometimes in this disease much higher temperatures are observed. Although I should not consider a single temperature of even 99°·5 abnormal in an apparently healthy person, I should be inclined to regard a persistent temperature above 99° in a strumous patient as indicative of some active mischief, even though the elevation were at mid-day only two decimals above that number; at any rate in such a case evening temperatures might advantageously be taken, for at that time if there was really anything serious going on, the temperature would be decidedly elevated. In the so-called acute phthisis, I believe the pulse will generally be found to be very rapid, and the temperature commonly as high as from 102° to 104°, or even 107° in very rare cases. Even in this variety the temperature may sink down for a day to about the normal range, but the pulse still keeps rapid, and in twenty-four hours the temperature will again be found to have risen to its previously high febrile standard.

Cancer in its ordinary form does not give rise to any increase of temperature, but though this statement is generally true, it is occasionally subject to exceptions. In a very interesting case which recently came under my notice, where cancer involved the tonsils and almost all the lymphatic glands of the body, running only a course of six weeks, the mid-day temperature taken for seven days was never below 101°, and mostly ranged from 101°·4 to over 102° Fahrenheit.

Diseases of the Lungs.—Of these pneumonia is the one of which I shall first speak, and in this affection, on the whole, I have taken the greatest number of high temperatures.

In pneumonia the temperature seems to rise to a considerable height at once, for in the only case in which I took it on the first day, the thermometer rose to 102°·8; rising from or about this point gradually, it seems to attain its greatest elevation on the fourth, fifth, or sixth day of the disease, the fourth being the most common; on this date temperatures of 104° or 105° are often to be met with at mid-day. After this the temperature falls pretty rapidly together with the pulse, most often by lysis, then by crisis, and then by a mixture of the two. Should the declining temperature of a case of pneumonia take a fresh rise, some new strip of pleurisy or pneumonia may be stated almost positively to exist, and this return of inflammation, as far as I have seen, increases out of all proportion the gravity of the prognosis. Contrary to what we might expect, double pneumonia does not seem to cause more febrile disturbance, as far as the curve of temperature is concerned, than is set up by inflammation of only one lung; and the most fatal cases of pneumonia are certainly not those where the temperature ranges highest, as shown by the thermometer. It is important to remember that the temperature having dropped to normal in this disease, only indicates that fever has ceased, and not that the solid lung has cleared up. I have taken normal temperatures where there has been bronchial breathing and mixed crepitation from top to bottom in one lung, and where even the other had been also partly involved.

Bronchitis in its chronic form does not increase the temperature of the body; and even in some cases where it assumes a more active character, the patient's temperature is nearly normal. As a rule, however, in acute cases, the temperature ranges from 99°·5 to 102°; and though in some few instances the mercury in the thermometer rises to 103°, lower temperatures are by far the most commonly met with.

Of pure idiopathic pleurisy in its early stage I have no record, the only case of this disease that I have taken early being one in which pleurisy was set up by the advancing irritation of œsophageal cancer. In this case, on the third day of the disease the temperature was 102°·4, on the fourth 103°, and on the sixth a fall had taken place, the mercury of the thermometer only rising to 99°·6. Eleven days after this the patient died, the temperature being from the sixth day always below 98°·5, except once, on the tenth, when it rose to 99°·6. In this case pericarditis came on after the heart was displaced,

but the temperature nevertheless continued low. I believe that in most cases in healthy persons after serous effusion has taken place, the temperature will fall to normal if not below it; though, as all the cases I have taken have been complicated with pneumonia, or have occurred in patients exhausted with suckling, I cannot speak positively on this point. In cases where the patient is somewhat exhausted before the attack, as from suckling for instance, the temperature often keeps abnormally elevated for a long time; thus, in one case of this kind that I observed, the temperature did not reach the normal till from the thirty-fifth to the fortieth day, ranging from the fifteenth to the twenty-second between 101°·6 and 102°·2, and after that marking below 100°. In another case also the temperature ranged from the nineteenth to the fiftieth day between 100°·6 and 102°·6, nineteen days after that even having only sank to 99° Fahrenheit. Pleurisy, at any rate in its later stages, is not a disease marked by a very high temperature, for I do not recollect having ever taken a case in which the mercury rose higher in the thermometer than 103°.

Of phthisis pulmonalis I have already spoken under the head of tuberculosis, and it is therefore only necessary to repeat the axiom that in all active cases of this disease the temperature of the body is elevated above the normal standard. This fact is sometimes useful in aiding the diagnosis of thoracic diseases which though giving more or less the same physical signs as tubercle in the lungs do not cause the temperature to be elevated. In cancer, dilated bronchi, and the ordinary bronchitis grafted on emphysema the temperature is normal. In the few cases of supposed cirrhosis of the lung that I have taken the temperature has not been abnormally elevated, but, as I have not yet had the opportunity of verifying by post-mortem examination the diagnosis formed in these cases, I cannot give any absolute statement as regards the temperature in this affection.

Before leaving the subject of diseases of the chest, it may be as well to state that, with a mid-day temperature ranging within, or very nearly within, normal limits, the patient may yet be the subject of pneumothorax, hydrothorax, or complete pneumonic consolidation of a good part of his lungs; in these cases, however, there must be of necessity no active inflammatory action going on, or the patient must be in a state of collapse.

Abdominal Diseases.—Of the value of the thermometer in these I can give little or no information. I have taken temperatures in jaundice, colonitis, limited and general peritonitis,

cancer of the liver and of the abdominal glands, albuminuria, ascites, suppuration of the kidney, &c., but I have not found the thermometer at present give much more information in these diseases than can be gained from other symptoms. In the diagnosis of hysterical peritonitis no doubt the thermometer will be found valuable, as in hysteria the temperature is normal, while in peritonitis, before collapse sets in, the temperature is certainly elevated. I do not think, however, that peritonitis is a disease in which fever, as estimated by the thermometer, runs very high. Out of five cases which I have taken, and one of which ended fatally, the highest temperature observed was only 102°·8, and the great majority of observations were considerably below this point. Albuminuria in its chronic form does not elevate the temperature of the body, but when it arises from acute nephritis the heat of the body is increased, as we might naturally expect. Connected with this disease is the state called uræmia, and in the only two cases of this in which I applied the thermometer the temperature was elevated above the normal standard.

With regard to the use of the thermometer in diarrhœa, I need only refer to the remarks previously made on this point under the heads of typhus and typhoid fever.

Brain Diseases.—In congestion, meningitis, and so forth, as far as I have at present seen, the temperature, though elevated, ranges moderately low, as about or under 102° at mid-day. In cerebro-spinal meningitis I once took a temperature of 103°, and in congestion of the brain I have taken one of 103°·2, but these are exceptional instances. Should further observations confirm this statement, temperature will certainly be useful in aiding a diagnosis between doubtful cases of cerebral disease and continued fever where the eruption is absent. It is probable that coma and cerebral irritation arising from reflex causes do not increase the temperature of the body, but I have only two cases at present illustrative of this point. Of delirium tremens uncomplicated I have only taken one case, and in this, though the temperature was normal, the patient died the same night. Another fatal case complicated with pleuro-pneumonia has lately come under my notice, in which the temperature ranged from 102°·8 to 100°·8, the patient coming under observation on the fourth, and dying on the eleventh day of his disease. This case is interesting from the comparative low temperatures taken, as, in a healthy person struck down with the same amount of disease, a temperature of 104° would have been reached almost to a certainty. Examination after death

disclosed recent pleurisy of the left side, and pneumonic consolidation of the whole of the lower lobe of the left lung, and also of the upper lobe of the right.

Diseases of the Spinal Cord.—In these perhaps the temperature varies more, and rises occasionally higher than in any other instance. In one case of softening of the spinal cord which I observed, the lesion being opposite the tenth, eleventh, and twelfth dorsal vertebræ, the temperature ranged as low as 98°, and at no time exceeded 101°. Again, out of about seventy observations that I made on another spinal case, where the arms, legs, body, and sphincters were paralysed, the temperature never exceeded 101°·6, and was mostly below 99° Fahrenheit. I have, however, records of a case of softening of the spinal cord where the temperature ranged commonly as high as from 103° to 104°, and where on one occasion it rose as high as 106°, which is certainly a temperature not often observed at mid-day, even in our acute English specific fevers.

Acute chorea has a rise and fall of temperature, as occurs in any other febrile disease, but my experience of it is confined to one or two cases, and I have not found the thermometer here of any practical value. In chronic chorea the temperature is not elevated. As an interesting fact to prove that amount of disease does not always give rise to a very high temperature, I may quote a case where chorea, rheumatism, pericarditis, endocarditis, and pneumonic consolidation of one lung were present at the same time; in this case the temperature was only 101° Fahrenheit.

In all the cases of sciatica and pleurodynia that I have taken up to the present time I have found the temperature ranging within normal limits. In two strange nervous cases also, of which spasmodic muscular movements were the most prominent features, I found the temperature of both patients normal. The first was a case of severe clonic muscular spasm, sometimes partial, sometimes general, causing the patient to leap at times almost half out of bed; the second was one of those cases that used to be included under St. Vitus's dance, but which is now classed under hysteria, where frequent rhythmical movements of different parts of the body took place, as flexing and extending the limbs alternately without intermission for a considerable time.

From these last cases the transition is natural to hysteria, and I may perhaps be blamed for putting this at the last when it plays so great a part in every-day practice. My reason, however, for doing so was, that I thought, when the tempe-

rature of real disease had been studied, the fact would come more home as a differential one in diagnosis, that the temperature of hysterical patients is not elevated above the normal standard. I do not say that real disease does not often exist with a low temperature—indeed, I have given several instances to prove that it does—but I do say, and all experience goes to prove, that when this Proteus simulates acute disease, as pleurisy, peritonitis, and other maladies, the thermometer when placed in the axilla not rising above the normal temperature, will differentiate this nervous shadow from real acute inflammatory disease.

Of the use of the thermometer as a prognostic agent, I cannot speak with satisfaction. Indirectly it is certainly of use, as by often establishing a correct diagnosis it enables us to foretell the ordinary course of a disease; directly I do not think it is of much value, but the five following rules include nearly all the lessons I have been able to learn under this head:—

TABLE TO SHOW THE CORRELATION OF PULSE AND TEMPERATURE IN DIFFERENT DISEASES.

	Typhus Fever.		Typhoid Fever.		Measles.		Scarlet Fever.		Febricula.		Tonsillitis.		Rheumatic Fever.		Facial Erysipelas with relapses.		Pneumonia.	
	T	P	T	P	T	P	T	P	T	P	T	P	T	P	T	P	T	P
1st day	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	102·8	123
2nd day	..	..	102	98	..	..	104·2	144	103	99	..	..	..	..	..	..	102·3	120
3rd day	104·8	108	103·1	98	..	..	104	148	103·7	103	102·6	102	..	..	99·2	102	103·6	122
4th day	103·6	113	103·4	110	102·3	130	103	134	104	105	101	104	101·8	105	102·7	104	104	126
5th day	103	114	102·7	107	103	124	101·2	122	102·6	99	100·3	100	102	114	99·6	114	103	122
6th day	103·2	122	103·2	104	100·2	112	100·6	108	98·4	99	99·6	96	102	116	104·4	108	102·8	122
7th day	104·2	124	103·7	107	98	102	100	106	..	..	97·4	84	103	120	105·2	116	100	114
8th day	103·8	122	102·5	108	N	98	100	110	..	..	..	..	100	90	98·2	72	99	94
9th day	103	113	103	108	..	80	99·8	108	..	..	..	..	100	96	100	105	N	78
10th day	102·7	117	102·6	111	..	..	99	100	..	..	..	..	99·4	86	94	82	..	..
11th day	102·4	119	103	111	..	..	98·6	104	..	..	..	..	101	104	101·6	102	..	..
12th day	102·2	108	102·5	112	..	..	N	84	..	..	..	..	101	102	97	97	..	..
13th day	100·5	106	102·2	108	..	..	..	..	..	..	..	..	102	100	..	..	..	..
14th day	100	100	102·4	109	..	..	..	..	..	..	..	..	100·9	100	..	..	..	..
15th day	99·4	98	101·8	107	..	..	..	..	..	..	..	..	100	88	..	..	..	..
16th day	98·7	92	102	100	..	..	..	..	..	..	..	..	98	90	..	..	..	..
17th day	98·4	90	101·4	100	..	..	..	..	..	..	..	..	99	64	..	..	..	..
18th day	98·2	..	99·8	98	..	..	..	..	..	..	..	..	102	96	..	..	..	..
19th day	..	..	101·4	105	..	..	..	..	..	..	..	..	103	102	..	..	..	..
20th day	..	..	100·2	100	..	..	..	..	..	..	..	..	101·6	100	..	..	..	..
21st day	..	..	99·8	98	..	..	..	..	..	..	..	..	101·7	104	..	..	..	..

In this table T stands for temperature, P for pulse, and N for normal.

A sinking temperature and a rising pulse existing together day by day are unfavourable; and a comparatively low temperature, as 102°, in the later stages of pneumonia and typhus fever, does not at all exclude the chance of death occurring.

In erysipelas, in some few cases of pneumonia and febricula, and in some spinal affections, temperatures above 105° may be met with without being necessarily of evil prognostic omen; but in most other diseases a mid-day temperature above 105° is to be considered a decidedly grave symptom.

If the pulse and general symptoms of a patient are favourable, the thermometer, in spite of this, marking a high temperature, the former symptoms are to be chiefly relied on in forming a prognosis, though the latter should make us still more careful in searching for any intercurrent disease, and especially for inflammation of the lungs.

In cases in which the general symptoms are perplexing, one counter-balancing the other, a very high temperature registered by the thermometer throws one more weight in the scale for forming an unfavourable prognosis, and vice versâ.

When the pulse and general symptoms on one hand indicate danger, the temperature on the other hand not raising alarm from being either very high or very low, the pulse and general symptoms are by far the most trustworthy in forming a correct prognosis.

Article VII.—*On the Treatment of Enlarged Bursa over the Patella.* By William S. Savory, F.R.S.

Amongst the diseases which the surgeon is called upon to treat, there are some which, although they do not threaten life or produce much suffering, are yet important, because they not only cause considerable annoyance, but obstinately resist or resent attempts to cure them. Enlarged bursæ are examples of these diseases. They will not readily yield to mild measures, while more decided plans of treatment are too often followed by very unpleasant consequences. Of these enlarged bursæ, that over the patella and its ligament is the one which is, by far, most frequently diseased, and which most urgently demands remedy, for, occurring almost always in women, whose daily occupation obliges them to kneel much—the disease being indeed thereby produced—it seriously interferes with their ability to earn their daily bread. Now, one has

only to recall the numerous and very various plans of treatment of the enlarged bursa on the patella which have been proposed and practised by surgeons, to show that there is, as yet, no single method generally recognised as at once safe and sure. The milder plans of treatment, such as by blisters, iodine, or mercurial ointments, are, at the best, slow and tedious, and in most cases will not succeed at last, unless the patient is laid by; while the more decided measures are either—as extirpation—severe in themselves, or are apt—as the seton—to be followed by mischievous and even dangerous consequences.

These considerations have induced me to ask for a trial of a plan of treatment which I have now adopted in a great number of instances, and, I think, I may say with uniform success. It is no novelty to many, and is mentioned among other plans of treatment cursorily; but as I do not think it has received the attention it deserves, I will venture to describe it. It is shortly this—The enlarged bursa is punctured with a lancet, or small knife, at its most prominent point; all its contents are carefully expressed, and then it is subjected to firm pressure by a pad of lint, strapping, and bandage, so that its walls may be everywhere kept in contact, and its cavity obliterated. This practice, according to my observation, is followed by one of two results. Either the bursa is obliterated outright, and there is an end of the case in a few days, or, in spite of the pressure, the bursa tends to refill. In this latter event, the fluid is again evacuated through the same puncture, and the bursa is again compressed. Then it may be cured, or may partially fill again, but perseverance in this plan soon succeeds. It must be observed, however, that when the evacuation is repeated, the character of the fluid which is discharged is gradually altered. It becomes more puriform. Therefore after two, or perhaps three, evacuations, particularly if there be any tenderness, a poultice is substituted for the pressure until the bursa subsides. But even in these last-mentioned cases, in which the plan is followed by the least simple result, a cure is, as a rule, accomplished more quickly and satisfactorily than by other means.

This simple plan of treatment may be adopted, I believe, in almost every case. It cures most quickly when the walls of the cyst are thin, and can be readily pressed together. It is most likely to require repetition, and to be followed by some suppuration, when the walls of the bursa are thicker and firmer. In some extreme cases, where the walls are very thick and the cavity a mere slit, the bursa appearing as a solid tumour, perhaps extirpation may be preferable, but I think

this operation should be reserved for extreme cases. When the bursa is inflamed, a few days' previous rest is advisable. In the smaller bursæ the fluid has usually the characters of ordinary synovia; in the larger and thinner ones it is often more or less bloodstained from previous bruising, but this, according to my experience, does not at all interfere with the efficacy of the plan of treatment I am advocating. There can be no doubt, that, as a rule, the cure is more certain and rapid when the limb is not used while the pressure is kept up, but, in the great majority of cases, one need not insist upon quietude. Indeed, amongst the poorer classes, this plan of treatment has the great advantage, in many cases, of not depriving them of a single day's work.

The chief points to be attended to in the management of these cases are—

To secure, after the puncture, by gentle and careful manipulation, the complete evacuation of the contents of the bursa.

To take care that the subsequent pressure is equable and efficient, so that no cavity can remain.

To keep up the pressure long enough to prevent any chance of subsequent refilling. In every case the pressure should be continued for at least a week after the cure seems complete. It is safer, in some cases, to keep it up for a period twice or thrice as long as this. Indeed there is no excuse for discontinuing it too soon, for, if properly applied, it produces no inconvenience.

This plan of treatment is, of course, applicable to other bursæ, whether normal or adventitious, when enlarged and troublesome, provided it can be carried out. But in most cases there is little chance of being able to keep up firm pressure; there is nothing against which the bursa can be steadily compressed. But for the treatment of the ganglions which sometimes appear in the neighbourhood of the wrist, either in front, or on the back of the hand, this treatment seems preferable to any other. It is simple, safe, almost painless, and when thoroughly carried out, it rarely fails.

Article VIII.—*On Gouty and some other Forms of Phlebitis.* By James Paget, F.R.S.

I HAVE met with certain cases of phlebitis, the like of which I cannot find on record. I propose, therefore, to give some account of them. They are all examples of the so-called adhesive phlebitis; the disease in which inflammation of the coats of a vein is associated with clotting of blood in its canal, but not with suppurations, or pyæmia. Of many of them, indeed, I cannot tell, any more than of certain other forms of phlebitis, whether the inflammation or the clotting were the first event, nor, therefore, whether they are to be referred more properly to phlebitis, or thrombosis. But I give the former name to them all because it is in most common use amongst us; and is probably correct, for at least one part or stage of every case.

Many of the varieties of adhesive phlebitis have been so well described, that I need only refer to them for the sake of comparison.* Such are—

1. The traumatic; including those due to distension.
2. Those occurring in exhaustion during, or after, either acute or chronic disease.
3. Those due to extension of inflammation or of blood-clotting from ulcers, morbid growths, or gangrenous or acutely inflamed parts.
4. Those of the so-called idiopathic, or rheumatic form which Dr. Mackenzie very fully described; but among which I am convinced that a closer study would lead to the distinction of different forms, associated with as many differences of constitutional affections.
5. The pyæmial.
6. The puerperal; among which it is probable that examples of all kinds, only modified by the puerperal state, are grouped.

Any of these forms of phlebitis may be modified by occurring in veins already varicose; but, passing by these, I proceed to the more proper subject of the paper.

* Such descriptions may be found in, or by means of, Callender, Art. 'Pyæmia;' Holmes's Syst. of Surgery, vol. i., and 'Diseases of Veins,' in the same, vol. iii.; S. Weber, Handbuch der Chirurgie, von v. Pitha u. Bilbroth, B. iii. Abth. ii. p. 113; Humphry, 'On the Coagulation of the Blood in the Venous System during Life;' Mackenzie, Pathol. and Treatment of Phlegmasia Dolens, 1862; Henry Lee, Diseases of the Veins, 1865; Virchow, Cellular Pathology; by Chance, lect. ix. x. 1860; Rokitansky, Pathologische Anatomie, B. iii.

Gouty Phlebitis.—The use of this name is, I believe, justified by the number of cases in which phlebitis is associated with ordinary gouty inflammation in the foot, or joints, and occurs, with little or no evident provocation, in persons of marked gouty constitution, or with gouty inheritance. In such cases the phlebitis may have no intrinsic characters by which to distinguish it; yet, not rarely, it has peculiar marks, especially in its symmetry, apparent metastases, and frequent recurrences. Gouty phlebitis is far more frequent in the lower limbs than in any other part; but it is not limited to the limb that is, or has been, the seat of ordinary gout. It affects the superficial rather than the deep veins, and oftener occurs in patches, affecting (for example) on one day a short piece of a saphenous vein, and on the next day another separate piece of the same, or a corresponding piece of the opposite vein, or of a femoral vein. It shows herein an evident disposition towards being metastatic, and symmetrical; characters which, I may remark, by the way, are strongly in favour of the belief that the essential and primary disease is not a coagulation of blood, but an inflammation of portions of the venous walls. The inflamed portions of vein usually feel hard, or very firm; they are painful, aching, and very tender to the touch; such pain, indeed, often precedes the clearer signs of the phlebitis, and not rarely begins suddenly. The integuments over the affected veins, (where they are superficial), are slightly thickened, and often marked with a dusky reddish flush. When superficial veins alone are affected there may be little œdema; but when venous trunks, as the femoral, the whole limb assumes the characteristics of complete venous obstruction. It becomes big, clumsy, featureless, heavy, and stiff; its skin is cool and may be pale, but more often it has a partial slight livid tint, with mottlings from small cutaneous veins visibly distended. The limb thus enlarged feels œdematous all through; but firm, and tight-skinned, not yielding easily to pressure, and not pitting very deeply. By this state almost alone the disease must sometimes be recognised, for it may be very marked when only a small portion of vein is affected, and that (as the lower part of the popliteal) so deeply seated as to be scarcely felt.

The constitutional disturbance associated with this condition is that of slight feverishness, or of an ordinary gouty attack, more or less acute, in different cases. The effects of the disease I have never had an opportunity of examining by dissection; for in the only fatal case that I have seen, no autopsy was allowed. So far as one may judge of them, by after-events during life, the veins which may have been obstructed

become, in some cases, pervious again; for in some instances the clearing-up of the œdema, and the restoration of the healthy condition of the limb, are complete. Yet the veins remain apparently very susceptible: — they ache exceedingly during fatigue, or trivial illness, or in changing weather; and I have known phlebitis excited, by trivial causes, in the same veins three or four times. In other instances, however, (but I think they are rarer than in other forms of phlebitis), the obstruction of the veins appears complete, and permanent; and then, if they be trunk-veins, the limb remains permanently enlarged, cumbrous, and heavy. Its superficial veins may, after some time, become varicose; and others may enlarge for collateral blood-streams; and I believe that an increased growth may take place in some of the tissues, especially the muscles of the limb.

Equally with the other forms of phlebitis, but as rarely as in any, that which occurs in gout may be fatal or very dangerous by embolism. I think that incomplete pulmonary embolism occurred in two cases in which—during gouty or rheumatic phlebitis—embarrassed breathing and tremulous action of the heart almost suddenly ensued, and then slowly but completely subsided. In another case such embolism was fatal. A member of our profession, whom I saw with Dr. Ferguson and Mr. Morgan, was suffering with a severe and protracted attack of gout, such as he had had more than once before. During its course he had signs of phlebitis in scattered portions of the veins of the right thigh and leg (having previously had phlebitis three times from accidental causes). He was sufficiently recovered to be down-stairs, and engaged in writing, and thought himself convalescent; but having walked up-stairs to his bedroom, he fell down as if in a deep syncope, and remained nearly an hour, breathing very faintly, scarcely conscious, and with a feeble fluttering pulse. In a few hours he seemed quite recovered, and next day, and two days later, we could find nothing additionally wrong about him, except a fresh attack of similar phlebitis in the opposite thigh. We examined his chest, and detected only some slight crepitus and faint breathing about the root of one lung. All appeared going on well for three days, and he had no sign or warning of severe illness; but five or six days after the previous 'fit,' as he was sitting on the night-stool he fell forward, and rapidly died with a renewal of the signs of syncope, and feeble breathing. No examination after death was made, but from the likeness of the manner of death to that which I have seen, in cases of ascertained embolism from systemic veins into the pulmonary artery, I cannot doubt what happened

here. It is probable that in the first fit, the obstruction of the pulmonary artery was partial; or that the clot was broken up, and its fragments dispersed; and that in the second, another clot remained blocked in the main artery, or was heaped on the adherent fragments of the previous clot.

I believe that gouty phlebitis is often hereditary. A patient, who had phlebitis in successive patches of both saphenous veins during an attack of acute gout, told me that his father and his maternal grandmother were gouty; and that, among his relatives on the maternal side, his mother, two uncles, grandmother, and two cousins, had inflammations of veins. And I can scarcely doubt that among the cases of phlebitis which are called 'common,' and are supposed to be referable to cold, or some wholly external cause, many might be traced to the gouty diathesis, however diluted and modified in its hereditary transmission.

In the management of cases of gouty phlebitis, there has never appeared to me any need of active treatment. Leeches do no good; mercury (I think) would do harm, if anything; purgatives seem unnecessary; colchicum has the same limited value as in other forms of gout, and appears useful in direct proportion to the severity of the symptoms. Alkaline drinks are certainly comfortable, and very probably useful; and certainly useful are a diminution of food and of stimulants, and an increase of water-drinking. But more important than all these is rest, with the trunk and limbs level; for in this condition there is the best opportunity for the adhesion of the clot, and its union with the walls of the vein; and the least risk of its detachment. Among local applications none seem more useful than frequent fomentation, and wrappings of the limbs with hot wet flannels.

Among the rarest diseases is a phlebitis extending through large portions of branches of both the superior and inferior *venæ cavæ*. I have seen only one well-marked case of the kind.

A man, 42 years old, had been ill for three weeks, when he first came under my care, on October 19th, 1864. I found him restless, looking very distressed, breathing about thirty times in the minute, but not with any conscious difficulty, lying on his right side across his bed. He had pain, and difficulty in moving either arm or leg, (especially the latter). Both arms were swollen, and œdematous; the hands quite bloated; and both legs, especially the left below the knee. The cephalic vein to the shoulder, and several other subcutaneous veins of the arm felt like hard, closely beaded cords; and had

dusky or ruddy marks over them; but they were scarcely tender. On the legs all the saphenæ veins felt similarly hard, and over some branches of the right saphena in the thigh there were diffuse branching red bands and blotches, very tender to the touch, and painful in movements of the limbs. His tongue was large, thinly furred, dry along the middle, and at the tip: he was thirsty and his mouth was all clammy; he hated food, but was no longer sick. His pulse was 120, small, and rather weak; his breathing as above noted. The heart's sounds were natural; so were the percussion and respiratory sounds over all the front of the chest. Over the lower half of the back of the right lung percussion was all dull, and there was a moderately fine crepitation audible to the same extent. The same kind of crepitation was in the lower and posterior third of the left lung; but here there was a less degree of dulness. The skin was all moist; at the head rather hot, and perspiring, at the hands cool. The bowels appeared disposed to act regularly, but were confined by the opiates he took. His mind was quite clear. All the signs of pneumonia with increase of the pulse from 80 to 120 had come on during the last twenty hours. He was advised to take fifteen grains of Dover's powder at bedtime, and to continue the use of chlorate of potash and ammonia, with some bark; and about ten ounces of wine, and some beef tea, and to remain in perfect rest.

October 20th.—He passed a very restless night, with frequent delirium. This evening his pulse was weaker, very soft and feeble; his breathing more free, but of about the same quickness. His abdomen was rather distended; he had had all day frequent hiccough, and had been sometimes sick. He sweated profusely, almost constantly, especially at the head; and had a short shivering fit in the morning. His general strength appeared much lower. The swelling of both legs was increased, and the fore part of the left foot was dusky bluish, cold, insensible—evidently gangrenous. Thus it had been for about 12 hours. The femoral pulse corresponded with the radial: the pulses of the tibials could not be felt on account of the œdema. From this time he rapidly became weaker, and without any marked new symptom, unless it were commencing gangrene of the left hand; he died at 1 p.m. on the 21st.

The history of this case, for which I am indebted to Dr. Corbould, was that the patient had been an active and generally healthy man, till five years before, when he had Smyrna ague, followed by a severe attack of 'Aleppo buttons.' From that time he had had less good health than before, and had often boils; and whenever he was unwell, was apt to have chills,

and feverish attacks reminding him of his old Smyrna ague. His family had no known disease, except consumption. Of his children, three had died recently in an epidemic of Scarlet Fever, and it was believed that a drain ran under his house (at Sydenham), but there was no clear evidence of this.

His illness was of three weeks' duration, and began with irregular chills, and a fit of shivering, and heats, and sweatings, which he regarded as a renewal of his old malady. After a few days he had soreness of the throat, which went on until a large abscess formed in one tonsil. It was opened, and discharged freely. All this part of his illness was attended with an ordinary amount of fever, and much sweating, but with no unusual symptoms. About a week before his death, without any accession of other new symptoms, the affection of his cutaneous veins began. Those of the arms were first affected, then those of the legs. First, part of the course or branchings of a vein would be marked with rather diffused red vascular bands (like those common over inflamed lymphatics); and with these were pain and tenderness. Then the vein would feel as if becoming hard, and at last quite hard, and closely knotted; and with this change the discoloration of the skin would gradually change to dusky brown, or nearly black, and then slowly disappear.

In both the progress and general distribution of the disease in the veins, there was an evident plan of symmetry; and usually the progress was from superficial to deep veins, and œdema followed at a distinct interval, the external signs of phlebitis.

The body was examined by Mr. W. M. Baker, who gave me the following report of it.

Post Mortem examination.—Left leg.—The long saphenous vein was plugged with coagulated blood, apparently in its whole extent. The femoral, popliteal, posterior, and anterior tibial veins were in the same condition; and so were their branches, muscular and others, as far as was seen, either in dissecting them out, or whenever they happened to be cut across. Here and there the colouring matter of the blood had oozed through the femoral vein, and stained the coats of the artery, and other neighbouring parts.

Left arm.—The radial, and other superficial veins of the hand and fore-arm were plugged in the same manner as those of the leg; and on dissecting out the brachial artery, its venæ comites were found in the same state. The deep radial, and ulnar veins were not examined, but they were doubtless in a similar condition.

The external iliac, common iliac, and inferior cava veins were healthy, and free from clot.

The coats of the plugged veins appeared somewhat thickened, and the clots, which completely filled and uniformly distended them, were slightly adherent to their lining membrane. The right limbs were not dissected, but there appeared no reason to doubt that their vessels were in a like condition to that which has been described as existing in the left.

The arteries were apparently quite healthy, and everywhere in the limbs, as far as they were seen in dissection, entirely free from clot. They were traced down to the gangrenous part of the left foot, and here also were found quite pervious.

The heart was very flabby and fatty, but with no other disease than this, and all its cavities were remarkably free from either fluid or coagulated blood. Their lining membrane was deeply blood-stained. The pulmonary arteries on both sides were pervious and empty, excepting one branch of the right, which contained a small clot, apparently recent. The substance of both lungs was here and there emphysematous, and throughout congested and very œdematous. No secondary deposits were seen in any part of them. The right pleural cavity contained a considerable quantity of deeply blood-stained fluid. The liver was pale and fatty; apparently not otherwise diseased. The kidneys were flabby, soft, pale, and fatty.

I cannot venture to say on what, if on any, manner of blood-poisoning the development of this singular disease depended. It may be only by chance that, in the only other case at all resembling it that I have met with, there was also some reason for believing that the disease had its origin in poisoning with foul air from a drain. This was the case of a clergyman, Mr. A.; a generally healthy man, with no known tendency to disease. In 1859 he superintended the opening of an old well, which proved so foul that it was at once closed again. Two of the men who had worked at the well were ill for some days after, with sickness, headache, and depression: and he himself felt slight nausea, for which he took a little brandy. A few days subsequently, and after a fatiguing journey, he had what appears to have been an attack of pneumonia, accompanied with fever and difficulty of breathing, and for which he was treated with leeches, and poultices. In about ten days, considering himself much better, he went down-stairs, but on reaching the drawing-room was suddenly seized with such excruciating pain in the left leg, that he was obliged to go to bed again; next day he was told that he had phlebitis. The attack of

phlebitis soon subsided, and he returned by easy stages into the country.

A few days after this he was seized with 'low fever:' this lasted six weeks, and was attended with alarming symptoms; rapid pulse, great heat of skin, unconsciousness, and delirium; and slight hæmorrhage from the ears, and nose, and from the stomach, bladder, and intestines. During a lingering convalescence from this illness there was great swelling of the legs, especially of the right, (the left having been the seat of the previous phlebitis): 'a fearful straining sort of pain in the region of the left kidney;' and a frequent recurrence, for about ten days, of 'most violent shivering fits, succeeded by fever, and profuse perspirations.' There were frequent, and violent fits of hiccough; and 'the throat was covered with an appearance of thrush of a yellowish-white colour.' At length Mr. A. regained his ordinary health. Thus the case ended with apparently complete recovery from the extensive inflammation of the veins of the lower limbs (and as we may assume), of those of the kidneys, intestine, and other parts from which hæmorrhage occurred. But ever since, there has remained a singular readiness for phlebitis in the trunk, and lower limbs. Thus in September 1861, after an unusually long walk, Mr. A., on examining a tender spot on the inner side of the right leg, found a red streaking of the skin over the internal saphena vein, with a hard cord-like condition of the vessel, for about two inches of its length. With horizontal rest this phlebitis, which did not extend itself, passed off. In the beginning of December in the same year, phlebitis occurred in the veins of the right groin, and spread to those of the abdomen, producing tenderness and redness of the skin, and leaving, as these passed away, a bruise-like discoloration of the surface. The veins affected in this attack remained much enlarged, and varicose. During the autumn of 1864, Mr. A., after a fatiguing walk, discovered a small inflamed spot in a vein of the left groin, and from this centre phlebitis spread throughout all those veins of the abdomen which had remained varicose from the attack in 1861. At the end of three weeks the affection suddenly left the abdominal surface-veins, and fixed itself in those of the inner side of the right thigh, causing considerable pain, and for the time, wholly disabling the limb. Treated with entire rest of the part in the horizontal posture, the disease subsided, and Mr. A. was well again, and able to take clerical duty till January 1865, when, after some precursory tenderness, the fourth onset of phlebitis showed itself in the veins of the right

calf. This was of short duration, but left the limb much weakened.

In strong contrast with these cases of widely diffused phlebitis, are those in which a single small portion of a great vein becomes obstructed. I have referred to some of these as occurring in connection with gout; but I have seen other instances in which no trace of gout or other general disease could be detected.

A man about 50 years old, thin and moderately muscular, and usually healthy, observed, during a September, that his right arm was growing larger, and, as he thought, stronger, and fitter for work. But, as it still increased, it became inconveniently heavy, and certainly weaker; and then he applied for advice. I found the upper arm two inches more in circumference than the left, and the whole limb enlarged in the same proportion. It looked full, round, and muscular, and felt firm, and œdematous, not only in the subcutaneous tissue but throughout; the skin was tense, cool, and pale. In nearly four inches of its course the axillary vein felt large, hard, and cord-like; and in one or two spots over it pressure caused pain: but with this exception no pain was felt in any part of the arm. Some of the superficial veins in the arm, and over the upper and front part of the chest, were enlarged, and when the arm hung down for a long time the hand became dusky. No cause whatever could be traced for this condition: no injury or pressure; no known inheritance of disease; no disturbance of the general health, past or present.

With the help of the hot douche, warmth, and friction, the swelling of the arm very gradually subsided; and, as it did so, the cord-like feeling of the obliterated axillary vein became more distinct. A year elapsed before the vein regained its completely natural condition; but it has now for more than two years been well.

Very similar to this case, was that of a regimental servant, 27 years old, previously healthy, and very active, who was sent to me on April 12th, 1855, by Mr. Bossey, on account of the condition of his right arm. This was swollen, and, when it hung down, he had a feeling of weight and fulness as if the blood could not return from it. The arm was indeed about a quarter, or a third larger than the other; but its chief enlargement felt as if due to great muscular development. Besides this, however, it had probably some general swelling, which might be from slight œdema of its deeper tissues; its subcutaneous veins were all over-full; there were small bluish spots

over the deltoid, as if from small clusters of varicose veins, and the veins over the right pectoral muscles were fuller than those over the left. This condition of fulness extended as high as the deltoid's origins:—the shoulder especially was remarkably broad and large, and there was fulness and some prominence of the upper part of the right great pectoral. The heart's action and sounds were natural; so was the pulse at both wrists, equal and moderately full. It was uncertain how long this state of the arm had existed; it had been observed only a week; its rate of increase was unknown. The patient remained in about the same condition till May 4th, when he was taken into the hospital, and ordered milk diet; six leeches every third night; and hydrarg. c. cretâ gr. iij every night and morning. He was under this plan for about a fortnight, and certainly improved, the arm decreasing, and its veins becoming less full. Then he had an attack of scarlatina, and while this was running its course all signs of the affection of the arm disappeared. It regained its natural size; the veins were scarcely fuller than in a healthy man; he lost the sensation of numbness, and believed himself well at the end of May.

A marked feature, in both these cases, was the apparent, and, I believe, real enlargement of the muscles of the limb. I referred to this in a paper published in the 'Medical Times and Gazette' of March 1858, and soon afterwards received a letter from the late Professor Laurie of Glasgow, from which the following is an extract:—

'I am the subject of one form of that peculiarity which I suspect is not very common, and which depends, as you hint, on diseased, or varicose veins.

'In the year 1831, I had an almost fatal attack of Typhus. During convalescence, I was seized with Phlegmasia dolens of my left lower limb, attended with exquisite pain in the ham and calf. When the pain subsided, and I commenced to leave my bed and dress, I was astonished to find that while my right thigh and leg were emaciated, as they usually are after such an illness, my left was nearly, if not actually, as large as when I took to bed. It was not in any way mis-shapen, or œdematous—simply plump, and full sized. When I recovered so far as to be able to walk, I found the veins enlarged, and, by night, the whole limb œdematous, but in the morning the soft swelling had disappeared, and the muscular enlargement round the calf remained. The excess was at least an inch. For many months I was unfit for much walking; indeed, but for a large laced stocking, I should have been compelled to relinquish my profession. I wore one for two or three years, and then was

able to lay it aside, but as I got older, I was obliged to return to its use, and for years back I have worn an elastic stocking. The veins are now decidedly varicose, and the limb becomes œdematous after unusual fatigue. The soft swelling readily disappears under rest, and, as formerly, the muscular enlargement remains. It is, however, now less obvious than formerly, as I am unhappily much less muscular than I was ten years ago.'

The occurrence of acute phlebitis during, or at the beginning of pyæmia, is well known. It is, I think, less considered that cases of the less acute forms of phlebitis are frequent after all the suppurative phenomena of pyæmia have passed by. Some instances of this were related in the last volume of the Reports (vol. i. p. 5). I would not maintain that such cases are peculiar sequences or residues of pyæmia. They may rather be reckoned among a class of cases of what may be called 'post febrile phlebitis,' for after any illness attended with acute fever, and without any very marked exhaustion, it is not rare to find one or more veins of the limbs becoming almost suddenly painful and hard, and then to see œdematous swelling of the parts supplied by them. Such attacks of phlebitis usually subside without treatment. Among many instances, I have seen nothing worse than an interruption or a delay of convalescence.

The overgrowth of parts whose veins are obstructed, to which I have referred, is very notable in the rare instance of phlebitis in young children. I have lately seen a child five years old, whose right lower limb has for three years been growing larger than the left. In the day, and when long dependent, it becomes œdematous; but during the night the œdema disappears, and the limb only looks much too large. Many parts of the thigh are mottled, dusky, and pink, and part of the skin at the knee is coarse-textured and warty. The whole aspect of the limb is like that of the limbs of adults in which, as in Professor Laurie's, the femoral, or common iliac vein, has been long obstructed. One might suppose it a case of simple hypertrophy, but that the limb is cold, not over-warm; or one of obstructed lymphatics, with growth from retained lymph, but that the blotches on the skin are characteristic of obstructed veins.

ARTICLE IX.—*Case of Congenital Myopia; with a faulty perception of colours, limited to a small portion of one retina, of recent origin.* By BOWATER J. VERNON.

THE subject of these changes is a very intelligent member of the medical profession, and it is mainly by his patient assistance that the following observations on the peculiarities of his vision are now recorded. As far as he can describe them, the phenomena of which he complains may be told almost in his own words.

He has always been short-sighted, but has never required to use glasses; he has used his eyes much in reading and writing, and at one time worked a great deal with the microscope, using especially the right eye, but he has been obliged to discontinue the last kind of work on account of pain and other symptoms of asthenopia. He was quite unaware of any other peculiarity of his eyes, and until lately is sure that one eye could distinguish and appreciate shades of colour equally with its fellow. About four years ago he was engaged in examining the spots on the sun with the aid of a powerful telescope, having previously guarded his eye—the right—with a double layer of smoked glass. Having completed his observations, he resumed his ordinary work, *i. e.* his books and his microscope, and did not for a time notice that his sight was in any special way affected. One day, however, when the question of the proper colour of a flower had been raised, he found that he differed from his friends as to its nature. As he was rather puzzled at this, he set about investigating the cause of it, and, after much patience, he found out that his two eyes could not equally distinguish shades, and he was able, by means of coloured dots upon paper, to illustrate the different tints which an object appeared to assume when observed singly with either eye. His observations at that time correspond with those which he has just now made.

His eyes are rather small, the irides bluish grey, and moderately active, but the right pupil is slightly larger than the left; the motions of the eyes are perfectly regular.

He is myopic with both eyes, $=\frac{1}{12}$, and when tested with Snellen's types his acuteness of vision is about $\frac{20}{30}$ with the right, and $\frac{20}{40}$ with the left. In addition, he is astigmatic with both eyes, the right eye very slightly so, but the left to a much greater extent, though this is perfectly remedied by the use of cylindrical glasses. He is very sensible that the vision of his left

eye is hardly as acute as that of his right, and, without being able to account for it, he has observed that the vision of his left eye can be improved by making slight lateral pressure with the point of the finger on the globe.

When the ophthalmoscope was used three years since, there was no marked change to note in the fundus of either eye; now, however, the ophthalmoscope verifies the existence of myopia; in the left eye there is a slight disturbance and irregularity of the pigment of the choroid on the apparent inner and upper margin of the optic papilla (inverted image); in the right eye, however, the choroidal changes are more marked. The inner border of the optic papilla is bounded by a small distinct, irregular 'myopic crescent;' and between this and the situation of the yellow spot, the choroid looks thinned and its pigment irregular. Both eyes are rather sensitive to light, but there is nothing else to note in their appearance.

When tested with colours the vision of the left eye is quite accurate, and he can readily distinguish and appreciate very slight degrees of difference in shade. With the right eye, however, his vision is very different. At a distance of five inches, at which he generally reads, he cannot appreciate green, but that colour seems to him to be a very pale blue; a light blue seems to him to be the same tint as an emerald green; a bright magenta dot appears to him to be a bright scarlet; with this latter exception, however, all bright colours seem to possess a duller tint.

By making him fix his eye steadily upon small circular dots of the brightest coloured ribands, it is possible to mark out, and represent by a map upon white paper, the portion of his 'field of vision,' within which these peculiarities are observed, by making a sketch much in the same manner as one would map out a 'field of vision.' This faulty portion of his retina seems to be situated to the inner side of the yellow spot, between the latter, in fact, and the optic entrance. With this right eye the oblique rays give him a proper notion of colours, although it is very difficult to determine this accurately; however, he is certain himself that it is so, and that 'there is but one small spot' in his retina 'which gives a faulty impression.'

With this eye his power of definition is greater than in the left; and after carefully tracing out his 'field of vision,' I cannot ascertain that any one spot is deficient in power of ordinary perception. Very bright colours serve best to show the above changes, and especially the bright colours of flowers.

I think there can be no doubt that in this case a small portion of the retina has lost the power of appreciating tints,

while its functional power does not seem to be otherwise impaired; and in trying to come to some rational conclusion as to the cause of such loss of power, we may, in this case, fairly put aside all idea that the imperfection is a congenital one; the history is very clear upon this point, and there is a complete want of any of the other phenomena which have been said to attend upon cases of colour blindness. With the sound eye his appreciation of colours is decidedly more accurate and acute than that of most men. There is no imperfection of any other sense, and his ear for music, and appreciation of musical sounds, are very acute.

Cases in which some severe illness has been for a time followed by faulty perception of colours are recorded here and there, but the phenomena seem to have been dependent principally upon imperfect conditions of the circulation. Several such cases have been mentioned by White Cooper,* and Mackenzie† records their association with double amaurosis. As the ophthalmoscope was not in use when the last mentioned case came under observation, it is impossible to say what structural changes had occurred in the choroid or retina. I cannot find any recorded cases where this defect has been associated with evident structural changes; my friend thinks the glare of the sun was too powerful for his retina; but it is very interesting, I consider, that the seat of disease should be just the spot where the structural changes due to myopia are to be seen. Supposing the choroidal changes to become more marked, there can be no question that the ordinary acuteness of vision at this spot will also become more notably diminished. The investigation would not be easy, and with unintelligent people, almost impossible, but would yet be of the greatest interest, if by it we could ascertain that with the earlier stages of choroidal or retinal disease, there was manifested, first of all, an imperfection in the appreciation of coloured tints. Without hazarding too strong an opinion upon this one case alone, it seems not unreasonable, I think, that here, at least, the functional imperfection of the retina is dependent upon the commencing choroidal changes ordinarily associated with myopia.

* White Cooper. Art. Vision. Cyc. Anatomy and Physiology.
† Diseases of the Eye, 3rd Edit., p. 799.

ARTICLE X.—*Respecting Rupture of the Axillary Artery in reducing Dislocations at the Shoulder Joint.* By GEORGE W. CALLENDER.

IN the following pages I propose to narrate some cases in which the axillary artery has been torn; their importance is evident, if only from the mortal nature of the injury, and the consequent necessity of guarding, as far as possible, against its occurrence.

J. F., æt. 61, was admitted under my care into Abernethy Ward with the following history. During the month of February whilst pursuing his occupation, that of a gardener, he was engaged one day in repairing the thatch of a plant-house. By some mischance he slipped from the ladder on which he was standing and fell heavily to the ground, a distance of several feet. His left arm being extended was driven upwards, and the head of the humerus was forced out of the glenoid cavity.

This dislocation was reduced, but unfortunately the patient and his friends persisted in using passive movements at too early a date and the dislocation recurred. At the expiration of the sixth week he was sent to St. Bartholomew's. This was on the 11th of April, 1865.

On the 13th of April he was taken into the operating theatre. Being a spare man the dislocation was unusually well marked. The head of the bone occupied the axilla, drawn up towards the coracoid process. The signs of this displacement were typical. Chloroform was first administered and reduction was then attempted by simple extension, the foot being placed in the axilla, and also by extension forwards, but these measures after a fair trial were abandoned as unsuccessful. I then sought to replace the humerus by circumduction, and to this end the arm was made to describe a half-circle over the face and head. In moving it round after this fashion, with the exercise of very slight force, and as the arm passed over the patient's head, I felt and heard, as it were, the humerus re-entering the glenoid cavity, and on bringing the arm to the patient's side it was found that the dislocation had been reduced.

At the moment of placing my hand over the shoulder to ascertain the result of the manœuvre, a swelling, rapidly lifting

itself and projecting the pectoral muscle, attracted attention. This swelling did not pulsate, and as the radial artery beat naturally at the wrist, I concluded, as did those of my colleagues who happened to be present, that a vein, probably of some size, or a muscular artery or arteries, had been ruptured in the round-twisting of the arm. Such an accident, though rare, has occasionally occurred, and was the cause, no doubt, of the symptoms noticed in the cases known amongst French writers as examples of the 'tumeur aërienne.'

Desault's account of his remarkable case singularly coincides with the symptoms which we observed. 'A peine,' he writes, 'la réduction fut-elle achevée, que l'on vit une tumeur s'élever subitement sous le grand pectoral, se propager vers l'aisselle, et en occuper toute l'étendue.' Subsequently there was great pain with fever, but on the thirteenth day this tumour was gone, and on the thirtieth day the patient was discharged. Pelletan recognised, as he thought, a similar accident, and looked for an equally favourable result, but the tumour was opened and the patient died from hæmorrhage, for here, as was afterwards ascertained, the main artery had been torn across. Flaubert narrates two cases and Malgaigne also gives an account of a considerable blood tumour rising up beneath the great pectoral muscle after reduction of a dislocation, the swelling subsequently subsiding and the patient in each instance making a good recovery. A swelling which Malgaigne is disposed to refer to rupture of some of the muscular arteries, rupture of a principal vein, independently of other injuries, being he asserts a very rare accident. He had met, he adds, with only one such case, and, with the exception of a second, to which reference will be presently made, I believe it is still the only example of its kind on record.

Experiences such as these serve to illustrate some of the difficulties which from the first surround these accidents to the vessels of the axilla, and show it was not unreasonable to expect that the hurt my patient had sustained might prove to be no very serious evil; for looking at the slight amount of force which had been used in effecting the reduction, although the possibility was admitted, I did not believe that, like Pelletan, I had been deceived as to the nature of the accident and that the main artery had been torn. So the arm was confined by a bandage, the patient was removed to his bed and some Arnica lotion was applied to the bruised shoulder. The swelling, having attained considerable size, ceased to grow larger, and as the man recovered from the chloroform there was no complaint of local pain.

The following morning the patient's general condition was good, and here it may be mentioned that he was sound and hearty for his years, though nervous and anxiously unquiet about his hurt and its consequences. The swelling beneath the pectoral muscle had become more diffused and now mixed itself up with the fulness resulting from the general bruising of adjacent parts, so that it extended around and behind the shoulder. There was also considerable ecchymosis which spread, chiefly through the subcutaneous cellular tissue, as low as the buttock, and the entire arm was somewhat œdematous. No change occurred, save that the bruising and ecchymosis began to clear up, until the fourth day. Then, after straining at stool, he complained of pain about the shoulder, and it was evident that some small quantity of blood had been afresh effused. The œdema of the arm was, however, subsiding rapidly, and the radial and ulnar arteries corresponded in their pulsations with the strength in beat of the vessels of the opposite fore-arm.

Until the twenty-fifth of May his history limited itself to the continuance of the swelling which, although it formed altogether a very considerable tumour, did not materially increase in size. But now it again enlarged itself, though to a comparatively slight extent and without any recurrence of pain. Now, as before, no pulsation existed in the swelling, nor was any bruit detected, carefully as it was naturally sought for. The arm below the shoulder had regained its natural condition, but, cramped and bandaged, he had lost to a great extent all command over its muscles, its sensation being, however, unimpaired. All this while the patient had been kept at rest in bed, in good general health, though about this time it became evident that the confinement and the local mischief were beginning to tell upon him.

Looking to the progress of the case and the history of the accident, the prospect for the patient, if things were left to themselves, seemed so unfavourable that I considered it desirable to take the opinion of my colleagues as to the propriety of laying open the swelling, and of endeavouring to secure the vessel from which the bleeding was recurring. What this vessel might prove to be was a matter of some doubt, for although the absence of pulsation and of bruit are well-known occurrences in cases of diffused aneurism of the axillary, yet the natural condition of the wrist pulse and the occurrence of the occasional hæmorrhages could only be explained, supposing the main artery to have been torn, by the existence of an opening in the vessel of such small size as to permit of its complete closure from time

to time, a condition scarcely to be looked for with a rent occurring under such circumstances and in so considerable an artery as the axillary. It seemed desirable, however, in selecting an operation, to choose one which would admit of my reaching the main artery, in case it should prove to be the source of the bleeding.

As the result of our consultation the patient, on the same day, was placed under the influence of chloroform, and was removed from the ward to the operating theatre.

The plan of the operation was based upon that described by Mr. Paget in a case which will presently be narrated. In this instance, I proceeded to lay open the blood tumour by two incisions. The first of these extended along the entire lower border of the pectoralis major, from its costal to its humeral attachment; the second, at a right angle to the first, through the mid-thickness of the muscle as high as the clavicle. Before making this second cut, my finger was passed beneath the muscle into a quantity of clot debris, and acting as a director, enabled me to divide freely from without. The large cavity now exposed was filled with various coagula and fluid blood, which I baled out by handfuls as quickly as possible, no bleeding as yet occurring. Thus the bulk of the effused blood was removed, and the space it had occupied was exposed and defined, the blood being seen to have made way far beneath the deltoid, and also into the shoulder joint; the size of the cavity being measured by the under surface of the pectoral muscles, of the deltoid, of the subscapularis, and latissimus dorsi, as well as by the distended lower boundaries of the axilla. When viewing the extent of the extravasation, greater than the outward signs had led me to expect, I regretted that the operation had not been performed at an earlier date.

The pectoralis minor lay across the bottom of the cavity, and seeing some clots projecting from beneath it, I scooped them out with my fingers, on withdrawing which a gush of arterial blood immediately followed.

Before the operation was commenced it had been found impossible, from the swollen state of the parts, to command the pulse by pressure on the subclavian. Mr. Wormald, however, had the kindness to compress the distal end of the axillary, and, with the occurrence of the rush of blood, Mr. Paget took charge of the artery as it came over the second rib. Whilst the bleeding was thus readily checked, I cut through the lesser pectoral muscle from beneath which the blood had issued.

The axillary plexus of nerves, with the accompanying vessels, was now in view, and I saw, having cleared the artery

to a slight extent, that the bleeding came from a small roundish aperture on the upper wall, and by pressure upon this spot all hæmorrhage was arrested. The vessel was now more completely exposed, and a ligature was passed round it on the distal side of the wound, and then tied. A second ligature was placed around the vessel about one inch above the first, and, after tying this, the artery was cut across midway between the two, and, all bleeding having ceased, it was evident that no branch opened into the main trunk between the applied ligatures. The walls of the vessel were considerably thickened.

On looking at the adjacent structures, the principal veins and nerves were found to be uninjured, with the exception of a small nerve passing across the axilla, which was so bruised and spoilt that it was thought well to remove it. It may further be noticed that none of the bones adjacent to the artery had been the seat of fracture. Taking a general view of the condition of the parts it was decided, the question having of course arisen, that the arm should not be amputated, as there seemed yet a chance that the patient might retain it and recover. So some strips of linen were placed in the wound, and the whole was covered with oiled lint. The patient, exhausted from the effects of the operation, was, after a short time, removed to his bed, and the arm was wrapped in cotton wool, its temperature being very low.

The subsequent history of the case may be briefly stated. For four days after the operation the patient did well, but on the fifth day the arm became gangrenous as high as the lower limit of the insertion of the pectoralis major, where, on the following evening, a line of demarkation was established. The afternoon of the seventh day he died suddenly, with symptoms of pulmonary embolism.

Although injuries somewhat similar to the above have been recorded, rupture of the axillary artery in reducing a dislocated shoulder is a rare accident, and the cause of the rupture, probably from the isolation of the cases, has not been made out so clearly as, I believe, it may and ought to be, now that the number of such cases admits of their general consideration. The references to thirty-one are placed at the end of this paper. Fourteen of these cases will be found in a group by themselves, and although in these instances the rupture of the vessel was not connected with the reduction of dislocation at the shoulder, yet the reasons for referring to them will be obvious as we proceed. The seventeen cases which remain may be conveniently and, I think, fairly arranged in four divisions.

The first of these consists of five cases. Four of the number are loosely referred to by Malgaigne and Pelletan, but, as I am unable to obtain any accurate history of them, they must be regarded as worthless for comparison with the rest. The fifth mentioned by M. Rigal seems after all to have been a doubtful case of rupture of the artery, for the death of the patient is said to have been almost instantaneous, so that Malgaigne justly suspects there must have been some other cause for the fatal issue, as in no one instance has such immediate death resulted from mere tearing of the vessel.

In the five cases which form the second group, great and, it should be added, undue violence was evidently resorted to. In the one referred to by C. Bell as having occurred in the Newcastle Infirmary, the muscle, as well as the artery was torn by the ambe; in Platner's case great violence is reported as having been employed, but in neither of the two are the particulars given, so that after all they are of little comparative value. Warren's patient had his artery ruptured, as chief amongst other violent attempts at reduction, by the pressure of a boot against the axilla. Pelletan tells us of a woman who had fallen and hurt her arm two months before her admission into the Hôtel-Dieu, but neither fracture nor dislocation could be detected by the surgeon who first examined her. Six weeks after her accident she consulted a 'paysan rebouteur,' who, after telling her the bone was out of joint, endeavoured to effect its reduction. To this end he made violent attempts at extension, and in fine, after having given her a great deal of pain, assured her that she was cured. The axillary artery was ruptured by the violence he employed, and the patient eventually died from hæmorrhage. The amount of violence used in such cases passes belief; quite recently, for instance, a bone setter at Leicester employed twelve or sixteen men to pull at the arm of a man, æt. 60, who was said to have dislocated his shoulder some time previously, and the following were the fatal results—the pectoral muscle was torn through, the plexus of nerves was ruptured just below the clavicle, where also the artery was torn across. The bones of the fore-arm were dislocated at the elbow, and the bones of the arm and fore-arm were fractured.

It is manifest that all these cases, some of which are habitually referred to in surgical works, are quite useless for any pathological inquiry into the causes of the accident, by reason either of the imperfect account we have of them, or because it is so palpably evident that the violence used was far greater than any surgeon should venture to employ, and in three

instances was certainly directed by unskilled and ignorant men.

Two cases, which form the third group, (they have been so often quoted that I do not refer to them at length) are instances in which the dislocation was associated with fracture; in one the inner edge of the glenoid cavity had been broken off (Flaubert); in the other about one-third of the lower portion of the glenoid cavity, and the extremity of the acromion, had been detached, and the greater tubercle of the humerus was cracked through (Gibson); so that in addition to the force employed in reducing a dislocation (in one case of eleven days' duration, in the second of long standing, and by which the bones, which had been broken and partially re-united, were again separated), some sharp edges or points were present, against which the vessel might have been torn. I do not mean to assert that the wound was caused in either of these cases by fragments broken off from adjacent bones, but the existence of fracture with the dislocation certainly increased the risk, at least we may fairly presume so from the history of such cases as the following—

A female, æt. 55, slipped in walking, and fell violently to the ground with her arm thrown up in an extended position. One or two days elapsed before she was seen by a surgeon, who detected and reduced, it was said, a dislocation at the shoulder. After three or four weeks, the patient began to complain of a swelling in the armpit, which being punctured some blood escaped from the opening. She was then sent to St. Bartholomew's, where she was admitted under the care of Mr. Skey, three months after the original accident. After careful consideration of the case, the swelling was laid freely open, the blood turned out, and the axillary artery tied above and below an opening in its canal. The patient subsequently died, and on examination of the shoulder the humerus was found to be fractured obliquely in its upper third, the artery having been wounded by a pointed piece of detached bone. No dislocation had occurred.

For the notes of the following case I am indebted to Mr. Holden, to whom they were given by Mr. Stanley. A male fell and injured his shoulder, and was told, by the surgeon who first saw him, that he was suffering from a dislocation, and accordingly the surgeon set about reducing it. Not being quite satisfied with the result of his efforts, for the head of the humerus still appeared to project more in front than it ought

to do, and than it did on the opposite side, he sent for Mr. Stanley, who thought it might be a case of partial dislocation forwards (much spoken of at the time by Mr. Abernethy). In this view they bound the arm tightly across the chest, with the hand resting on the opposite shoulder. Calling in to see the case a few days afterwards, Mr. Stanley's attention was roused by the fact that there was no perceptible pulse at the wrist of the injured arm. The bandage was immediately removed, but without restoration of the pulse. Many years afterwards the patient died, and Mr. Stanley made a careful examination of the parts. He found that there had been a fracture through the anatomical neck of the humerus, with obliteration of the axillary artery, opposite the broken part of the bone.

I fancy that in this case of Mr. Stanley's only the inner and middle coats of the vessel had given way, probably at the moment of the accident, just as in Bérard's case, where the injury, however, was followed by death in consequence of the arm becoming gangrenous after the obliteration of the artery. It is worth noting, as we pass, that Bérard's is the only instance on record in which the vessel has been torn at the time of the dislocation without accompanying hurt to either veins or nerves, the hurt having been recognised before any attempt at reduction; except, perhaps, the interesting case narrated by Adams, by whom a false aneurism of the axillary was detected before a recent dislocation was reduced, both having been caused by direct violence to the shoulder, a hurt which may be placed in the same list with that of Nicholas Pochard, who had an aneurism of his brachial artery, caused by blows from the lock of a gun.*

There are two other conditions, not yet referred to, which invite or even cause rupture of the artery, and to these I will revert, after narrating a case for the notes of which I am indebted to Mr. Paget.

Rupture of the Axillary Artery.

The patient in whom this accident occurred was a female, 40—45 years of age, of rather feeble health. In the summer of 1855 she had a severe rheumatic inflammation of her left shoulder, which left the joint completely stiff, with the upper

* Cases of injury to the axillary artery from strains and blows, without any external wound, have been mentioned by Aston Key, Le Gros Clark, Liston, Syme and Gibbs. Such cases, with many interesting examples of various hurts to the vessel in question, have been collected and arranged in two tables in the Nouveau Dictionnaire de Médecine et de Chirurgie pratiques.—p. 365. Paris, 1866.

arm close to the side. It remained thus fixed until early in 1857, when Mr. Ingoldby began to treat it with friction, galvanism of the wasted muscles of the arm, and movements. Forcible elevation of the arm was frequently practised, by the help of chloroform, and by degrees such an amount of movement was acquired as to enable her to touch her head with her hand.

On the tenth of January, 1858, wishing to show her sister how much her arm was improved, the patient rested her elbow on the mantelpiece and then lowered her trunk, till the arm was horizontal. She felt no remarkable pain or straining at the time, but, about half an hour afterwards, there came great pain and swelling in and about the axilla, increasing and extending by degrees about the shoulder and upper half of the arm.

When first seen by Mr. Paget, at Mr. Ingoldby's request, on the nineteenth of January, she looked pale, feeble, and reduced by pain. The whole axilla was filled out, as much as the fixed position of the shoulder would allow, with a tense firm swelling, raising in front the pectoralis major muscle, and still more prominent behind under the teres major and latissimus dorsi. In every part of the swelling distinct pulsation could be felt; with each pulsation a rough blowing sound could be heard all over, and even for a little distance above and below, the swelling. No pulsation could be detected in the arteries of the arm below the axilla, though the radial, in the fore arm, was distinct and even full; the upper arm was somewhat œdematous.

A projecting cervical rib, or a long growth from the last cervical vertebra, left scarcely room behind the clavicle for the finger to be pressed down upon the third part of the course of the subclavian, but pressure on the axillary artery below the clavicle put a stop to the pulsation in the tumour.

After careful consideration of the case it was thought advisable to recommend, for the time, complete rest, diminished food and anodynes.

For a time the symptoms were less severe, but on March the ninth the swelling was larger, and was rather softer, especially in the axillary part. The scapula and humerus were evidently pushed further away from the side, and the integuments over the lower part of the swelling had a dusky light brownish colour, the swelling of the arm was rather increased and more general, and the local pain had become more severe, such pain coming on suddenly with hardness and great tension of the skin and intense aching, followed by tingling. But pulsation was no longer perceptible in any part of the swelling, or in the axillary artery above it, and the bruit was no longer audible. Below the tumour no pulse could be felt.

Thus the events that had occurred during this second month after the accident, suggested that, whatever changes might be taking place in and about the extravasated blood, the ruptured artery was closed or closing.

It therefore seemed still right to avoid active interference.

On the twentieth of April, another month having elapsed, the swelling was larger, especially in front and towards the axilla, where, also, it was more thinly covered and formed more prominent knobs. The increase had occurred especially in the last ten days, with renewal of paroxysms of pain. There was still no pulsation either in the swelling or in any part of the arm below it, but the general œdema of the extremity was much increased, and was now equal in every part of it. With this she had become much paler and thinner, so that it appeared imperative to give her relief, if possible, by emptying the blood from the swelling and tying the artery.

On the twenty-third of April, in accordance with a suggestion of Sir B. C. Brodie, Mr. Paget punctured the lower and posterior part in the axilla, where the skin was very thin and dusky, when only a few drops of dark and thick blood escaped. The same result having followed a second puncture, 'I therefore cut along the whole length of the axilla, just behind, and parallel with, the lower border of the pectoralis major muscle, and made a second cut at right angles with this, and from its middle, through the skin of the front wall of the axilla. Having secured a pretty large superficial artery divided in this cut, I passed my finger through the lower incision, under the pectoral muscle, and then, on my finger, cut straight upwards through its whole width. Raising the angular flaps of the ⊥ shaped wound, the surface of the great mass of clot was exposed. On scooping it out with my fingers, small streams of arterial blood rushed into the cavity from two small divided arteries, and a larger stream from what we soon discovered to be an opening in the posterior wall of the axillary artery. This vessel with its vein, and the axillary plexus of nerves were stretched across the axillary cavity, in contact and close union with the integument of the lower part of the axilla. They appeared compressed into a thick firm band, in which all were matted together. The opening in the vessel was oval, with its long axis parallel with that of the artery, regular, with smooth borders about one quarter of an inch in length and one tenth in width. Above and below it the artery was apparently healthy.'

The bleeding was not controlled by such pressure as could be made on the subclavian, though arrested, of course, by pressure on the wounded vessel. Ligatures were applied above and be-

low the opening, and then tied, when all bleeding ceased. The artery was divided between them, and the remaining blood clots were removed, chiefly through an opening in the lower part of the armpit.

The walls of the cavity which had been occupied by the blood effusion, whose several diameters were from 4″ to 6″, were all the normal boundaries of the axilla, and one could distinguish, see, and feel the front surface of the neck of the scapula, the edge of the glenoid cavity, the front and lower part of the head and neck of the humerus. All these were thinly covered by the softer tissues compressed firmly upon them, but with no appearance of disease. In short the cavity was the anatomical axilla exactly filled and distended.

The anterior vertical wound was united with sutures; oiled lint was laid over all and gutta percha.

The clot was of various consistence. Its outermost parts were firm, brownish-black, half dry, irregularly laminated. Its inner parts black, soft, and without layers. There was very little, if any, fluid blood, till the artery began to bleed.

The loss of blood during the operation was under six ounces. The patient was all the time fully under the influence of chloroform and her pulse was scarcely affected.

The recovery from the operation was without interruption. The chief things to be noted during the patient's convalescence were—1. The greater part of the wound healed with immediate union; the rest, with its edges never wide apart, slowly closed with granulations. 2. The pain she had suffered at the seat of the swelling was never felt after the operation. 3. The walls of the cavity from which the blood was removed contracted with remarkable rapidity, and, in a week the shoulder had regained the size it had before the rupture of the artery. 4. Nearly the whole of the pus from the cavity, as well as from the wounds, escaped through the opening which had been made for a drain near the posterior wall of the axilla. 5. The œdema of the limb began to decrease in five days, but very slowly. 6. The pulse at the wrist returned at the end of three weeks, feebly, and at times, and was constant after about five weeks. 7. The temperature of the limb was not at any time diminished.

The patient left her room eight weeks after the operation, and in one week more the wounds were healed, except that from the one at the posterior margin of the axilla there was a very thin watery discharge.

The movements of the shoulder-joint were not regained, but the patient remains free from all trouble at the wound.

Amongst the causes which may have led to the accident just described, it is evident that we should bear in mind the stretching to which the artery was subject at the moment of the yielding of the anchylosis; indeed examples are not wanting to show that stretching, especially sudden stretching, of the vessel may be the sole cause of its tearing. Pelletan gives a case in which the axillary artery was rent across from violent efforts, a male, æt. 40, having been suspended for some time by the hands from the bolts of a scaffolding; and in the Museum of our Hospital* there is part of a brachial artery which was torn straight across by external violence. The patient, æt. 69, fell with his arm stretched out. At first he seemed little injured; but pulsation was lost in the radial and ulnar arteries. In a few hours the arm became enormously swollen and livid, and amputation near the shoulder was performed.

Bearing in mind what happened in Mr. Paget's case, and in those to which I have just alluded, we may proceed to examine the circumstances under which the artery was torn, during reduction of a dislocation, in the remaining cases which, with one exception, constitute the last of my divisions. In the one which I first related it is quite certain that the vessel gave way as the humerus was raised to its highest point above the shoulder, and consequently when the head of the bone was turned down into the axilla bending before it, and extending the vessels and nerves, which in dislocations of this kind lie close to it on the anterior and inner aspect.† As the arm was twisted round pretty quickly the strain may be said to have been sudden in its action. In Blackman's case, a physician, æt. 50, presented himself with a dislocation downward and inwards, of sixteen weeks' duration, one unsuccessful attempt at reduction having been made two weeks after the occurrence of the accident. Chloroform having been given, the arm was adducted, rotated and abducted; these measures failing it was next elevated, and in ten minutes a swelling was noticed in the pectoral region, and it was then found that the radial and ulnar arteries had ceased to pulsate. The artery was tied in the upper part of its course, but the patient died on the twelfth day, from secondary hæmorrhage.

Some years ago, at the Great Northern Hospital, the late

* Series xiii. 88. In the museum of the College of Surgeons, is an axillary aneurism, caused by rupture of the artery, from a man falling on ice with his arm extended. Series xxv. 1695.

† See Museum of St. Bartholomew's Hospital.—Series iii. 112. Malgaigne has shown that rupture of the axillary artery has occurred with most of the common forms of dislocation at the shoulder.

Mr. Price was reducing an old dislocation at the shoulder of an aged female, when the axillary vein, as was subsequently ascertained, was torn across, the patient dying on the following day. In this case also the rupture (of the vein not of the artery) occurred at the moment of greatest tension of the vessel, that is, whilst the dislocated humerus was fully raised above the shoulder in effecting what is known as White's method of reduction. The second accident of the kind which happened to Mr. Gibson, occurred to a male, æt. 50, of intemperate habits. Three weeks after the hurt five strong men pulled at the arm without effect. Five weeks later he was bled to twenty-four ounces, and attempts at reduction were made, first with pulleys, then by five or six assistants, and again with pulleys, but the patient becoming faint these efforts were discontinued. Already a considerable swelling was apparent beneath the muscles around the shoulder, when eventually, after two slight rotatory motions, the head of the bone slipped suddenly into its place. But the artery had been torn across; the swelling increased, the patient became blanched, and died some hours after the operation.

It is true that in the last case some adhesions between the artery and the head of the bone had been broken through, but no portion of the vessels had been rent off with them, and from the fact that the artery had been torn directly across it seems to me quite clear that the injury must have been sustained during the varied and prolonged extensions, irrespectively, that is to say, of the rupture of the adhesions. Although under somewhat varied circumstances, it is true, for each of these cases, that the vessel (in one instance the vein) gave way at the time of its being subject to a considerable strain, a condition indeed, under which an artery, rather than either veins or nerves, is likely to succumb.

This strain, even if no great force is being employed by the operator, is really most severe when, as in two of the instances just referred to, the artery is bent over the head of the bone in a somewhat sharp curve, in carrying out the ordinary movement of circumduction or of extreme extension upwards, under which conditions it is manifest that the tension is not fairly borne by the walls of the vessel but falls upon them unequally, that is to say, on one side more than on another. In Gibson's second case, the extension downwards seems to have been carried to an extreme degree, and probably in this instance, as in Flaubert's, the fact of the artery being adherent (in the case last mentioned to the second rib) may have increased the risk, as the length of vessel for bearing the strain was of course

lessened by its upper portion being fixed, and so rendered practically unyielding. As to the opinion expressed by Flaubert that such accidents to vessels may be due to the changes they have undergone from local inflammation with softening, we are not sufficiently acquainted with the changes produced under such circumstances to justify the assumption which he advances.

With the exception of one of Gibson's cases, the one complicated with several fractures of adjacent bones, the patient was in each instance over fifty years of age; in the case quoted by Nelaton, the age, said to be advanced, is not given, but in Astley Cooper's the accident happened to a person advanced in life; so that there is a strong presumption that the arteries were predisposed, so to speak, for the accident by previous disease of their tissues. In the case which came under my own notice the walls of the vessel were undoubtedly thickened. Astley Cooper expressly refers to the artery as being diseased and rigid, and, although his utterance is not clear, I fancy a like condition must have been present in Nelaton's patient.

In the majority of the cases as may be seen by referring to the list which I have appended, the dislocation has been of several weeks' duration, but in three at least the accident has happened in reducing a recent displacement, in two of these the artery was diseased, and in the third there was the complication of a fracture.

The conclusions to be drawn from the cases are these—

Experience shows that the artery of the arm has occasionally given way from the effect of blows or strains.

Rupture of the axillary artery in reducing a dislocated shoulder is a rare accident, the more so if we set aside the instances in which the hurt has occurred with the complication of a fracture, and those in which rude attempts have been made by ignorant persons to extend a limb, sometimes, even, not the seat of a dislocation.

Rupture of the artery, in the remaining well authenticated cases, has occurred during extreme extension, when the head of the bone has been twisted round in effecting the reduction, or has been otherwise turned down into the axilla.

The accident has happened to persons over fifty years of age, in whom, for the most part, old dislocations were being treated, or when the dislocation was recent the artery has been diseased.

The occasional occurrence of this accident does not rule against the recognised practice of attempting the reduction of old dislocations, but should make us cautious of using move-

ments calculated to over-stretch the vessel, such as circumduction and extreme extension, as by White's method.

These movements must be especially avoided when the patient, either from age or from other causes, is likely to be the subject of degeneration (atheroma) of the artery.

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1. *Cases of Rupture of the Axillary Artery in reducing dislocation at the shoulder of which the history is incomplete.*

Verduc (Malgaigne.—Traité des Fractures, tome ii. p. 151) saw, in the reduction of the humerus, an axillary artery torn, and, in consequence, an aneurism speedily fatal.

Petit (Malgaigne as above) witnessed a similar accident.

Pelletan (Clin. Chir. tome ii. p. 49) refers to a tradition at the Hôtel-Dieu that a four months' dislocation of the humerus was sought to be reduced. There was a painful crack, and a tumour rose up; an emphysema it was said, but it was punctured, and the patient bled to death.

Flaubert (Sur plusieurs cas de Luxation, Répertoire Général, tome iii. p. 55 et seq.) gives a case of 'tumeur aërienne' which proved fatal to the patient, but the cause of death is not satisfactorily explained.

Delpech (Case mentioned by M. Rigal to Malgaigne, op. cit. p. 152). See ante, page 101.

2. *Cases in which great violence was used in effecting the reduction.*

Platner (Malgaigne, op. cit. p. 151) cites a case of rupture of the artery and of the axillary veins in consequence of violent extension.

C. Bell (System of Operative Surgery, p. 247). See ante, page 101.

Warren (Amer. Journal of Medical Science, vol. xi. N.S. 1846). A male, æt. 30, dislocated his shoulder whilst drunk. The reduction was immediately effected, not without great violence and notably by extending whilst the operator's foot, from which the boot had not been removed, was forced into the axilla. Thirty-eight days later, the tumour which formed in the armpit broke, and two hæmorrhages ensued. The subclavian was then tied in the third part of its course, and the life of the patient was saved; but four months later sensation and the power of movement were still wanting in the limb.

Pelletan (op. cit. p. 83). The case of a female æt. 66, see ante, page 101.

Case referred to at page 101.

3. *Cases in which the dislocation was complicated with fracture.*

Flaubert (op. cit. tome iii. p. 55). A sailor, æt. 57, with a dislocation of eleven days' standing. After fixing the limb, two assistants guarded the counter extending rope from slipping, whilst eight intelligent pupils made steady extension downwards and backwards. This attempt brought the head of the bone from over the second rib well into the axilla. A second attempt, extension being made downwards and outwards, effected the reduction. Immediately, however, before relaxing the extension, an enormous swelling rose up under the pectoral muscle. There was great local pain, a feeble pulse at the wrist, pulsation in part of the tumour, and the whole arm became cold and livid. 'The only "tentative" ligature of the subclavian artery of which we could by rights think was forbidden by the general condition of the limb.' Some days later a quantity of blood escaped from two openings, three fingers' breadth below the hollow of the axilla, and the patient died an hour later. The upper end of the broken artery adhered to the rib surrounded by cellular tissue, whose density was augmented by thick lymph deposits. The inner edge of the glenoid cavity was fractured.

Gibson (Amer. Journal of the Medical Sciences, vol. ii. p. 136). A male, æt. 35, with a nine weeks' dislocation of the left humerus, for which four attempts at reduction had been made. Severe operative measures were employed, and after one hour and three quarters the bone snapped into the glenoid cavity. At eight the following morning the swelling was observed, at three p.m. the next day the subclavian was tied, and the tumour ceased to pulsate. The arm passed into what I fancy must have been a gangrenous condition (the account is not clear) and the patient died on the sixth day after the operation. The upper end of the wounded artery was adherent to the head of the bone and to the articular capsule (*sic*) by dense cellular substance. Respecting the fractures see ante, page 102.

4. *Cases incidentally referred to.*

Desault (Surgical Works, vol. i. p. 380). A male, æt. 60, with a dislocation of one month and a half, see ante, page 97.

Flaubert (op. cit. tome iii. p. 55 et seq.). Two cases of 'tumeur aërienne,' see ante, page 97.

Malgaigne (op. cit. tome ii. p. 150). A carman, æt. 44, dislocated his humerus forward beneath the clavicle, and after numerous previous failures, the reduction was just being effected, 'le succès semblait assuré,' on the sixty-eighth day,

when a swelling suddenly rose up in the subclavian region and extension was discontinued. There was no bruit heard in the swelling, the radial beat naturally, so that Malgaigne felt assured that the artery was not torn. He feared, however, that the vein might have given way. An enormous ecchymosis subsequently appeared, but on the ninth day absorption of the blood had commenced, and on the twenty-second the tumour and the ecchymosis had disappeared. See ante, page 97.

Froriep (Veraltete Luxationen, Weimar, 1834. p. 35). A scrofulous subject, æt. 26, twenty days after the accident, was found to have sustained a dislocation at the shoulder. At the second attempt, two distinct sounds were heard and the dislocation was reduced, but at the same moment the shoulder became swollen. The patient died one hour and a half after the operation, and the axillary vein was subsequently found to have been torn completely across. See ante, page 97.

Skey, see ante, page 102.

Stanley, see ante, page 102.

Bérard (Nelaton, tome ii. p. 368), see ante, page 103.

Adams (Cyclop. of Anat. and Physiology, art. Shoulder, p. 616) reports the case of a male æt. 50, who suffered from rupture of the axillary artery from a blow, the humerus being also dislocated. The dislocation was at once reduced, and, on the tenth day after, the subclavian was tied, the patient recovering. See ante, page 103.

Pelletan (op. cit. p. 14) gives the case of Nicholas Pochard, see ante, page 103.

Paget, see ante, page 103.

Pelletan (op. cit. p. 49) mentions the case of Gabriel Longpré, æt. 40, referred to at page 107.

Museum of St. Bartholomew's Hospital. – Series xiii. 88. See ante, page 107.

Price, see ante, page 107.

5. *The remaining cases of rupture of the axillary artery in reducing dislocation at the shoulder.*

Blackman (Hamilton on Fractures and Dislocations, p. 554 et seq.), see ante, page 107.

Gibson (Philadelphia Journal of Medical Science, vol. vii.), see ante, page 108.

Nelaton (op. cit. p. 368). The humerus was displaced below the glenoid cavity of a woman advanced in years. Reduction was easily effected, but a false aneurism formed in the axilla. Three months later the subclavian was tied, but

the tumour broke open, and eventually the patient died. The two inner walls of the artery had been slightly torn, and the aneurism which formed in consequence of this hurt communicated with the cavity of the joint.

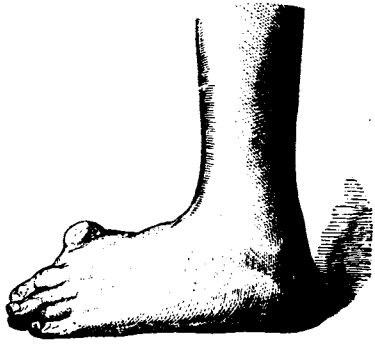
A. Cooper (On Dislocations and Fractures of the Joints, p. 371) mentions a case in which reduction was easily effected, but a false aneurism formed, and, the sac having ruptured, a fatal hæmorrhage ensued. The artery was diseased and rigid. In this instance the dislocation was of recent date.

Case reported at page 96.

Article XI.—*Remarks on the rectangular Talipes Equinus of Orthopœdic Surgery.* By Holmes Coote.

I EMPLOY this term, 'rectangular talipes equinus,' not because it is well selected, but because it has long been used by those engaged in the treatment of deformities, to express a condition of the foot and ankle which is very common among all classes, although little recognized in general surgery.

The patient does not walk on the extremities of the toes, as the horse; nor on the extremities of the metatarsal bones, as the dog or the cat; but on the sole of the foot; so that at rest the limb presents no abnormal appearance. The toes may be freely pointed downwards, as in the act of dancing, but any attempt to raise the foot beyond the right angle to the leg meets with irresistible or nearly irresistible opposition from a tense condition of the tendo Achillis.



In order to ascertain this fact, the limb must be examined in a state of complete extension; the surgeon's left hand indeed

should press upon the thigh immediately above the knee, while the right grasps and moves the foot; and, moreover, it must be remembered, that firm and continued pressure will, in milder cases, produce an amount of elevation of the foot, which is not readily attainable by the patient himself in progression.

The contraction, in many cases, becomes progressively worse, while in others it is paroxysmal. I have known some cases recover on the removal of uterine and other disturbances; but in most instances, the division and subsequent elongation of the tendo Achillis have become necessary.

The symptoms are as follows—The patient's walk is stiff and ungainly, and he soon gets tired, especially on standing. It is remarked that he bears more upon the front part of the sole of the foot than upon the heel. The latter often does not touch the ground. The front part of the shoe is worn. The foot soon shows a tendency to turn inwards. Corns of thickened cuticle form on the front part of the sole. In walking the toes are apt to catch in any irregularity, and the patient often feels an inclination to pitch forward. He can, it is true, run upstairs easily enough, but finds a difficulty in coming down. He cannot sink the leg on the foot, in consequence of the stiffness of the Achilles tendon, and he prefers going down sideways as in the companion-ladder of a ship.

After a time the arch of the foot sinks, owing to elongation of the calcaneo-scaphoid ligament. This process is always attended with a constant aching pain. It has been remarked that girls in this condition can dance without fatigue, but cannot stand long at a time. The reason is this—In the exercise of dancing, there is no constant pressure on one set of tendons or ligaments; the weight of the body is shifted from part to part, or from limb to limb. In the act of standing the whole weight falls steadily on the calcaneo-scaphoid ligament, and the tendon of the tibialis posticus. These structures, and particularly the former, become intolerably painful in yielding to steady and continued pressure.

It is often noticed that the great toe is pulled up by the extensor tendon; and this latter structure is so tight and prominent, that surgeons have been tempted to divide it. Such an operation is unnecessary.

In cases where the patient can ride and drive about at pleasure, this state of inconvenience may be bearable; but among those forced to work for their living the evil soon becomes so grave, even in children, as to induce them to submit to treatment, including an operation.

I have met with cases in the very young and in the adult, but it does not seem to be a congenital affection.

The causes are generally to be referred to disturbances of the functions of the cerebro-spinal axis, reflex or otherwise, by which the proper amount of muscular equilibrium is destroyed. Thus it supervenes in the irritation induced by difficult dentition; it follows an attack of scarlet fever, of measles, or other of the exanthemata; it supervenes in the adult female on painful menstruation.

It may be produced artificially by casing the legs in tight heavy boots; or as a sequence of wounds and subsequent cicatrisation.

In very many instances the nerve lesion is confined to the spinal cord; in others the brain is also involved, when symptoms of slow or feeble intellect are readily detected.

Treatment.—In so brief a notice it may be sufficient to say that in all cases the removal of any exciting cause of disturbance is in the first place clearly indicated. The mouth should be carefully examined; decayed teeth should be removed; tense and swollen gums should be freely lanced; the sequelæ of fevers should be rigidly investigated, and uterine disturbance put right.

Small doses of tartar emetic may often be given with advantage; the alimentary canal should be cleared of offending matter by doses of rhubarb and mercury and chalk, or even stronger purgatives. A stimulating liniment may be rubbed along the spine and limb. The foot should have free play. Where mechanical treatment is needed, steady and continued extension, easily effected by means of the Scarpa's shoe, has proved sufficient, combined with constitutional treatment, in milder cases.

But in the majority, the division of the Achilles tendon becomes ultimately necessary. After re-union, its elongation to the requisite and normal extent must be slowly effected. After which the patient must wear, for a period of twelve months or more, a boot with double irons and a stop-joint at the ankle to prevent the tendon again becoming morbidly tight.

In conclusion, I would say that all these operations succeed better where the nervous system is in a quiet state; and that so long as patients are known to complain of thirst and heat of skin, or are observed to start in their sleep and toss about the bed, or grind the teeth, it is better to defer the division of the tendon until a more suitable time.

ARTICLE XII.—*On the Treatment of Irreducible Hernia.*

By JOHN LANGTON.

OF all the various phases which Hernia may assume, none has been so cursorily dealt with, or summarily disposed of by surgical writers, as the irreducible condition of it.

So long as life is not imperilled, the surgeon usually contents himself with telling a patient to procure a truss. In the case of an ordinary reducible hernia, such an injunction is scarcely consistent with good surgery, because the patient is sure to get a truss which suits only his ideas or his feelings. But when the hernia is irreducible (a condition which would naturally demand the anxious care of the surgeon), the common practice is still nearly the same—the patient is usually confided to the care of the bandage maker. No doubt warnings are given, but, as a rule, there is little treatment attempted by the surgeon after a few days' confinement to bed on low diet. It is not, however, always the fault of the surgeon. No warnings are effective to persuade some patients to submit to the tedium and discomfort of prolonged treatment for the sake of procuring reduction of the hernia, so long as there is no pain; and pain (that is, local pain referable by the patient to the region of the hernia, and recognised by him as due to it) is by no means a frequent concomitant. Whatever pain there may be attendant upon this condition is (as all surgeons know) usually referable to the epigastric or umbilical regions.

Nor is it surprising that such cases should be lightly regarded, when so little is to be found in the writings of surgical authorities on the subject. Mr. Lawrence, speaking broadly, says: 'An irreducible hernia must be left in great measure to itself;' and this in spite of the statement which immediately follows, that 'its bulk and gradual increase are sources of inconvenience, and the constant possibility of strangulation exposes the patient to considerable danger.'* Richter also says: 'The patient is obliged to leave an irreducible hernia entirely to itself, because it is not often possible to be reduced, and, consequently, no bandage can be employed.'†

But it is not only the danger to life that demands the surgeon's attention. He has also to aim at rendering life tolerable by maintaining or restoring the normal relations of parts. There is in a hernia unreduced and ungoverned, not

* Lawrence on Ruptures, 5th edition, page 133.

† Richter, Abhand. v. den Brüchen, 1785, page 479.

only danger to life, but also loss of physical power, partly positive and real, and partly through apprehension and pain. It is surely not advisable, then, to leave such cases 'to themselves;' nor is it well to rest satisfied with an attempt to stay the progress of the complaint. It should be the surgeon's aim and purpose ultimately to reduce the hernia, and maintain it in reduction. This may not always be possible, but it should always be attempted. If the patient be earnest in the cause, success may be more often achieved than writers on this subject lead us to suppose. The case must be advanced and involved with serious complications, for us to say with Pott, in his chapter on irreducible hernia, that 'they are capable of no relief from surgery but the application of a suspensory bag, to take off or lessen the inconvenience arising from the weight of the scrotum.'* Old people who have allowed their herniæ to get into an irreducible condition, through years of neglect, rightly come under this category; but younger patients, so long as their health endures, as a rule, should never be despaired of.

The question, then, is—What is the plan of treatment to be pursued in order to arrive at this consummation? It has been urged by writers, that whenever reduction is attempted, 'the proper method,' in the words of Pott, 'is by absolute rest, in a supine posture, for a considerable length of time; by great abstinence, and the use of evacuants, so to lessen the size of the parts in the hernial sac as to render them capable of passing back again into the belly.'† This is also, in the main, the plan of treatment recommended by Arnaud,‡ Le Dran,§ Earle,‖ Lawrence,¶ and others; mechanical appliances being regarded by most as of secondary importance.

Rest is, without question, essential, but prolonged confinement to bed is not the only form of rest; indeed it may happen that (with ill-health and cough) it may be no rest at all, 'quoad' the rupture. This form of so-called rest is frequently impossible to the working-man, and, indeed, is undesirable at any time, if it can be proved to be unnecessary. It is of the first importance that the patient's health should be, as far as possible, maintained, and a coerced recumbency is likely to endanger this. It is the experience at the City of London Truss Society that herniæ, of whatever kind, and in whatever condition, are

* Pott's Works, edited by Earle, 1808. Article, Treatise on Ruptures, vol. ii. page 48.

† Op. cit., page 51.

‡ Arnaud on Hernia, 1748, pages 187 to 296.

§ Le Dran, Traité des Opérat., page 114.

‖ Earle, London Medical Gazette, vol. v. page 223.

¶ Lawrence on Ruptures, 5th edition, page 136.

but little troublesome or difficult to manage so long as the health of the patient continues good. A typical example of this common fact is extracted from the Society's books. A well-known London waterman, ruptured on both sides from the age of 19, came for a new truss in 1859, at the age of 31, having just finished an aquatic tour in France. He had pulled the stroke oar in all the races, wearing a broken and almost useless truss, and yet his rupture had not troubled him, and had but slightly increased.

On the other hand, the following case, quoted from the same source, is an example of an opposite condition, but bearing out the same rule. A working mason, æt. 68, came to the Society in 1860 ruptured on both sides for four years. At the time of examination, he was afflicted with chronic bronchitis and emphysema, and such general laxity of tissue that every abdominal outlet bulged with hernia. The protrusions through both inguinal and femoral apertures, and the umbilicus, were so large and lax as to prevent retention by any mechanical means. After his condition had become somewhat improved by medicines, the mechanical treatment became available and effective, and his herniæ were governable by simple appliances. He was, at the age of 70, still under treatment for his general condition, and the herniæ also continued governable.

Again, as a link between these two extremes, cases may be referred to (and they are not of rare occurrence) in which the patients, whose trusses have been (prior to the supervention of a debilitating illness) adequate and efficient, come, after such loss of health, complaining that their ruptures are no longer kept up. The difficulty of maintaining them remains so long as the illness continues; but the same or a similar instrument is again found sufficient as soon as the health of the patients is restored. These facts show that the maintenance of health is essential alike for the treatment of difficult herniæ, as for the control of simple ones.

Since, then, confinement to bed is likely to endanger health, and health is essential for the treatment of ruptures, it would seem desirable to attempt reduction of irreducible hernia without supine confinement, by great abstinence, and the use of evacuants, considered so essential by Pott, Petit, and others.

The cases for which Pott advises such formidable treatment are irreducible scrotal herniæ, which are, however, of much less frequent occurrence than formerly.

The only classification required for the treatment of the irreducible hernia is into the three fairly recognisable conditions —1. Epiplocele; 2. Enterocele; 3. Entero-epiplocele.

The care which writers take to classify the adherent from the impacted forms need not engage our attention, even if it were possible, at all times, to arrive at the distinctive characteristics; for the same treatment will apply to both, and neither should be considered incurable.

1. *Epiplocele.*

When omentum alone constitutes the hernia, without any fluid in the sac or sign of intestine, the protrusion will bear with impunity considerable pressure; in this case it is fair at the outset to apply the taxis freely and forcibly without much apprehension. It would be little credited by some how much force may safely be employed under such circumstances; but if there be much fluid in the sac, together with much tenderness, it would be unwise to use such an amount of force. In case the epiplocele resist the taxis, the object is to maintain, while the patient pursues his ordinary occupation, the most continuous and firm pressure that can be tolerated.

The irreducible femoral epiplocele, which is so commonly met with in women, and generally of such small size as to allow of the application of uniform pressure over it by means of a well-fitting cup, cannot be included in Pott's list, because the lesion is too trivial for such severe treatment. It is well known that cases of this description are frequently reducible by the means referred to.

Femoral epiplocele is not, however, always confined to these easily-managed dimensions. When neglected—and femoral hernia in the female is very commonly neglected—it is liable to become large and difficult to manage. When it passes forward through the saphenous opening, and, partly by fascial attachments, and partly by flexion of the thigh, rides over Poupart's ligament, be its size what it may, it is still within treatment by a cupped truss. But when it descends down the thigh along the course of the saphena vein, it is necessary to append to the hollow truss pad, a circular thigh-belt with a soft pad shaped to fill Scarpa's triangle comfortably; by this means the mass of omentum may be pressed up against the opening, through which it descended, and then be treated as in the foregoing cases. The junction of the thigh-belt with the cupped truss allows free mobility of the thigh, while it affords the greatest obstacle to the escape of the hernia.

Case I.—Sarah L——, æt. 67, ruptured since the age of 36, came first under notice in February 1866, with irreducible femo-

ral epiplocele on the left side. The enterocele which descended above was reducible, but there remained a large mass (larger than a hen's egg) of omentum, which could not be returned, by reason, it appeared, of its size, and not from adhesions. The plan of treatment just alluded to was adopted in this case, with the effect of rendering the omentum reducible in a few months, without any alteration in her mode of living, so that on August 3 of the same year a solid-headed truss was adjusted, the thigh-belt being still worn for additional security.

If the portion of omentum be small enough to be pushed either into the canal or as far as the external abdominal ring, it should be maintained in this position by the continuous pressure of a well fitting cup or hollow pad with a soft thigh strap and cross-piece, after the fashion of a spica bandage.

It will often happen that reduction may be effected after a few days, but this result will necessarily depend much upon the duration of the hernia and the perseverance of the patient.

If the mass be too large to be so treated, the scrotum in the male must be closely gripped by an accurately fitting bag, which should be capable of being laced tighter by means of a running tape, so as to follow the decreasing size of the protrusion. This should be applied so as to keep the mass closely pressed against the external ring; the bag itself being suspended from a belt which should be fitted tightly round the pelvis, just below the crests of the ilia, and should firmly gird the iliac and hypogastric regions of the abdomen, as tightly as the patient can endure it. When the bag has been adjusted so as to drag up the hernia well against the external ring, the flat pad of a Salmon and Ody's patent ball-and-socket truss should be applied, partly over the inguinal canal and partly over the neck of the protrusion, to prevent the mass from being pressed over the sharp edges of the external abdominal ring, to direct the pressure in the proper course, and also to hinder any protrusion of intestine. The apparatus should be applied while the patient is in the recumbent position, not only for ease of adjustment, but also to return as much of the hernia as is reducible. The result effected by the pressure of the truss is the gradual diminution in size of this part of the omentum, probably by absorption of the fat which is normally present, the omentum becoming smaller and indurated. The bag should be worn without intermission day and night; the truss also should be kept on, if the patient can tolerate it. If he be unable to do so, it should be taken off after he goes to bed and re-applied before rising in the morning.

CASE II.—I. W——, æt. 50, came to the Truss Society, April 11, 1865, with an inguinal hernia on the right side which had existed many years; part of which, he stated, was returnable, but the larger portion had remained irreducible for some years. On examination, there was a large protrusion, of which the enterocele was reducible, but a considerable amount of omentum still remained, and was adherent to the upper portion of the testicle. He was supplied with a truss having a hollow pad. He continued to wear this till April 25, 1865, when he again presented himself to the surgeon, the omentum being in much the same condition. A tight suspensory bag was then adjusted, so as to compress the contents of the scrotum well up to the external ring; over the inguinal canal was placed a Salmon and Ody's truss.

July 12.—Omentum, since last note, had very much decreased in size, and with a little taxis was entirely reducible into the abdominal cavity and retained completely by an ordinary truss. The testicle was dragged up to the outer ring.

Sept. 16.—Omentum is easily retained by a truss, but readily escapes on its removal.

He came again April 16, 1866, and the omentum was then perfectly retained under the truss and did not escape when the latter was removed. During the whole time he was under treatment he followed his ordinary occupation, which was that of a gold chain maker.

CASE III.—E. M——, æt. 30, came July 19, 1866, with inguinal hernia on both sides: it first appeared on the left side at the age of 6. He has been wearing an inefficient truss for the last twelve months, which has entirely failed in retaining the hernia on the left side; it has, however, been always reducible at night till three weeks ago; when it could no longer be returned. For the last few days it has been down to a greater size and has occasioned him much discomfort. His bowels are opened regularly.

There is now a crepitating mass of omentum, with fluid, in the sac; the hernia forms one swelling with the testicle which lies below and is distinct from the hernia, which is probably in an unclosed funicular portion of the vaginal process of the peritoneum. Some of the protrusion can be reduced, but only a very insignificant amount. The attempt causes considerable pain, evidenced not only by the man's expression, but also by the profuse cold sweat over his body and face. A closely fitting, strong, cotton-stocking bag was adjusted, which pressed the hernia tightly up to the external abdominal ring, while the

flat pad of a Salmon's truss was allowed to press gently upon the inguinal canal. He was allowed to continue his ordinary vocation. July 23.—The hernia was much less in size, most of the fluid having disappeared. After some firm pressure the mass was reduced into the canal and ultimately into the abdomen. The suspensory bag has been worn without intermission; the truss has been removed at night, and during the second day was worn only a few hours at a time; he wore it, however, during the whole days of July 21 and 22. An ordinary truss was then applied.

The two last cases (out of many that might be quoted) illustrate the good results that may attend the use of firm pressure; the patients all the time they are under treatment pursuing their ordinary occupations.

2. *Enterocele.*

This condition is much less common than either the irreducible epiplocele or entero-epiplocele; it being rarely met with except in the state of strangulation. The diagnosis between the pure irreducible enterocele and entero-epiplocele is by no means so easy as might at first sight appear.

Pure enterocele must be treated more cautiously than the irreducible epiplocele. In most cases the suspensory bag is the only mechanical appliance which can be recommended; but in some, the addition of the flat pad of a Salmon's truss may be employed with great advantage. The bag should be of strong calico, with a running tape, so that it can be made smaller as the hernia decreases. The sensations of the patient must be taken more into consideration than is necessary in the case of a simple irreducible epiplocele; these will be evidenced by general pain, a sense of sickness, together with faintness. These sensations are not uncommon at first, but in a short time the patient frequently expresses himself as feeling great support from the truss; if, however, they should continue, the use of the truss will have to be discontinued. It is advisable, in all cases of irreducible enterocele, to take off the truss at night; the patient, after doing so, being further instructed to apply the taxis gently, so as to procure reduction of the intestine if possible.

Case IV.—A healthy farmer, æt. 38, came under notice in 1864, with a large scrotal hernia, larger than the two hands could grasp. He could give no date to its commencement. There had been more fulness on that side as long as he could remember.

The hernia was mainly, and it seemed entirely, enterocele. It covered the testicle completely, so that that organ was completely lost in the tumour. It had gradually grown to its present size, and was still increasing. The patient did not complain of it for any other reason than that it was a local inconvenience. His bowels were sluggish, but not constipated. He did not suffer from flatulence or nausea. Taxis was but slightly attempted; it caused more pain than the patient was disposed to tolerate.

A strong calico bag was made to fit the tumour, and so managed by a running tape that it could be made smaller in the long axis of the hernia. The swelling so constricted was braced up tightly to a leather girdle with shoulder straps, so that there was a double bearing from the hips and the shoulders. This was tightly worn for nearly a month; the bag being tightened to the swelling as it decreased. The taxis was occasionally employed, but not vigorously. The patient went about his farm as an overlooker. At the end of a month he found that he could reduce the tumour completely. It was with difficulty retained by a truss for the next two or three months on account of the size of the opening and the magnitude of the protrusion. The testicle after reduction was found small and undeveloped. There was no omental adhesion.

3. *Entero-epiplocele.*

This form, although not so frequent as the epiplocele, is much more common than pure enterocele. The non-reducibility of this condition is often, in all probability, due to the presence of omentum in the sac, and when the adhesions are removed, the enterocele, not infrequently, is completely reducible. The plan of treatment is first to treat the protrusion as a pure enterocele, with the suspensory bag, and sometimes by the ball-and-socket truss of Salmon; frequently the enterocele is speedily reduced, and when this has been effected, the further treatment of the tumour limits itself to that described under the head of irreducible epiplocele.

Case V.—J. H——, æt. 35, an engine-keeper, came to the Truss Society, April 10, 1861, with a large scrotal hernia on the right side. He stated that he had had a swelling there as long as he could remember. He attributes its increasing size to a cough to which he has been subject for some months. When seen he had chronic bronchitis with occasional urgent paroxysms of dyspnœa. He was a fat, bulky man, with a small weak pulse. He states that he has lately been getting rapidly thinner.

The hernia was a large entero-epiplocele which covered the testicle, and only part of the enterocele was reducible. A strong suspensory bag hanging from a leather band, tightly grasping the pelvis, was adjusted to constrict the tumour and press it up firmly to the external abdominal ring. The flat pad of a Salmon's truss was applied over the neck of the protrusion. At the end of a week (the patient in the meanwhile continuing his occupation) the intestine was reducible. After three weeks his general condition becoming much improved under treatment, the hernia was so far reducible as to warrant the use of an ordinary truss instead of the ball-and-socket truss. The suspensory bag was still worn. The patient presented himself weekly for the next three months. He became considerably thinner, but much stronger and more healthy. He had still the cough when he ceased attendance at the institution, but under treatment it was much less troublesome. He was able at that time (Aug. 13) to maintain his rupture completely by means of an ordinary truss; the testicle being, however, dragged up to the external ring.

This plan of treatment will be seen to stand the test of experience. It is the custom at the Truss Society to treat such cases in the manner already described, and with such a fair amount of success as frequently to reduce those herniæ which have been long 'left to themselves.' The treatment of irreducible herniæ, by means of steady and uniform pressure, has been advocated by some writers, although it does not appear to have been adopted as extensively as it might be. I have endeavoured to show in this paper, that this plan is assisted more by allowing the patients to continue their ordinary occupation, and by improving their general condition during the time they are under treatment, than by confinement to bed, low diet, and so forth, considered so essential by most writers on this subject.

Article XIII.—*Case of Wound of the Right Lumbar Region, involving and laying bare the Kidney; complete Recovery.* By Bowater J. Vernon.

THE kidney is so placed and connected as to be less exposed to injury by ordinary external violence than the liver and the spleen, and injuries of its substance are certainly less

fatal in their issue than is the case with these latter organs. It is not difficult to find reasons why this should be; besides being a duplicate organ, the kidney is situated behind the peritoneum, embedded in much cellular tissue and fat, with a very definite capsule of its own, conditions which very materially tend to limit effusion of blood or urine, provided the escape be gradual and moderate in amount, and therefore of the greatest value in warding off or limiting* peritonitis, while the ureter serves admirably as a channel by which blood or foreign substances† may be removed from the abdomen; for though we seldom meet with cases of extensive injuries of the kidney, yet we, by no means infrequently, see cases, in which, after blows or heavy falls, there is an immediate and considerable escape of blood with the urine, and where we may suppose there has been some limited damage done to the substance of the organ, such as cracks or rents through the living membrane of its central cavity. Where, as in the present instance, the case is complicated with an extensive external wound, there is every facility for the escape of hurtful fluids, which in itself is of importance in bringing about a successful issue.

CASE.—William Morris, æt. 14, was admitted into Kenton Ward, under Mr. Paget's care, in the afternoon of May 8, 1864. While walking along the wall of a house which was in course of being pulled down, he became giddy, and losing his footing, fell from a height of forty feet upon a heap of rubbish and old iron. Nobody saw him fall; he says he was not stunned; he got up and walked some little distance without help, and then asked a man to carry him to the Hospital, as he had hurt his back; he was brought to St. Bartholomew's about twenty minutes after his fall; he was quite calm and collected, very intelligent in his answers, and, except that he was pale, there were no signs of shock about him, and you would not have thought him seriously hurt. On turning him over on his face, we found a wound of the soft parts immediately above the right iliac crest, semicircular in shape, and about six inches in length, stretching across the spine; on the left of the spine the wound was superficial and involved integument only; on the right of this, however, the crest of the right ilium was exposed, and the erector muscle had been torn through, so that a large bruised piece of its substance hung downwards. Into the gap thus formed there protruded the lower end of the right kidney, a portion of it as large as a walnut being fairly ex-

* Cases of Rupture of Ureter, by Mr. Stanley. Medico-Chirurgical Transactions.
† Hennen's Military Surgery.

posed; this was of a dull red-brown colour, and moved freely downwards with each contraction of the diaphragm. A piece of its substance had been chipped out, leaving a gap which would admit the finger-end, its wounded surface having a fibrous appearance, free from blood, but certainly distilling urine; lastly the whole exposed portion seemed to have been deprived of its capsule. There had been very little blood shed externally, and there was no bleeding at the time of his admission.

Mr. Paget saw the lad very soon after he was put to bed. A portion of integument was secured over the wound in such a manner as to protect the exposed kidney. The wound was lightly dressed with oiled lint, and the lad placed upon his side in such a position as to favour a free drainage of all discharges. There was a large ragged hole in his clothes through which the hand could be passed, corresponding to the wound; and as far as could be ascertained from his own account and from an examination of the rubbish upon which he fell, the injury must have been inflicted by some broken iron, the remains of an old fire-grate.

Early next morning the lad became very restless and feverish, and his symptoms increasing during the day he was ordered small but frequent doses of opium. No water was passed until fourteen hours after admission, and that was scanty and blood stained. As time went on he had all the ordinary symptoms of extreme reaction after shock, but his breathing was very laboured as well as quickened, and he evidently suffered on each movement of his diaphragm; his pulse varied between 110—130, and he complained much of thirst. For some hours there was no discharge whatever from the wound.

From this date until the twelfth of May his condition may be thus described: the reaction passed off, but he became very feeble, breathed with great difficulty, and with much cough; looked sallow and wasted, and though generally sleepy was nearly always delirious. The integument covering the wound sloughed away, and the torn pieces of muscle, looking sloughy, were removed. It was no longer possible to keep the lad in the same position, and we could not ensure a free outlet for discharges. There was some limited tenderness of abdomen, but the main thing to note was the extreme difficulty with which his diaphragm moved; as far as we could ascertain there was no pulmonary complication, but he had an eruption of herpes around his lips, and breathed with dilated nostrils; he would take very little nourishment, but continued the small doses of opium (about ℥ xxv of Liq. Opii in the twenty-four

hours, in doses of ℥ v each). About the fifth or sixth day his condition improved; his symptoms subsiding, there was a free discharge of urine from the wound, and suppuration commenced. Up to this time he had passed a small quantity of blood-stained urine every day in the natural way, but occasionally his doing so caused him great pain in the penis; after this date no blood was passed with the urine.

On the eighteenth day his bowels acted freely after a simple injection; and by this time the wound had begun to suppurate profusely, and was always washed with urine escaping from the wounded surface of the kidney; this now could hardly be distinguished among the surrounding florid granulations, but its movements with each inspiration were still very apparent.

From this time there was not much to note in his steady recovery; the wound gradually closed, and six weeks after his admission, was reduced to the condition of a sinus through which urine escaped; this state of things continued for some weeks, during which the lad's progress occasionally flagged, his symptoms, pain and general fever, being invariably preceded by a cessation or diminution for some hours of the discharge of urine from the wound; then a sudden reappearance of the urine would be noticed, and all uneasiness would subside. This occurred some five or six times. No more urine was passed from the wound after the ninth week; it soon completely healed over, and the lad was discharged apparently in perfect health. Since his discharge he has been quite well and able to lead an active life.

On reading the account of this case, it must strike everybody as very remarkable that there were present none of the ordinary phenomena of shock; for these are generally so marked after blows upon, or very slight injuries of the abdomen, and are almost invariably shown in an extreme degree when there has been a lesion of any internal organ, since, besides the intense disturbance of the sympathetic nerve, there is usually more or less internal hæmorrhage, which assists in deepening the depression. True, there was in all probability very little loss of blood in this instance, but the force of the lad's fall from such a height must have been very great, and very considerable violence alone could have produced such a wound; moreover the proximity of the wound to the spine, and the fact of such an important organ as the kidney being involved in it, might themselves have led one to anticipate the occurrence of shock in a very marked degree. About the same time there were in Kenton Ward eight men who had been injured

by the sudden falling of some scaffolding upon which they were working; the local injuries were mostly of a very trivial kind, but they one and all were brought to the Hospital in a state approaching to collapse, so much so indeed as to require active surgical treatment. One of them however was thought to have sustained some injury of his kidney, and for some weeks his life was in considerable danger from peritonitis with considerable hæmaturia. The contrast between these latter cases and that of the lad was most striking.

Surgeons but seldom have the opportunity of inspecting any of the internal organs, so that anything which is observed of their condition during life and health is not unworthy of note: the colour of the kidney in this instance was very much that of a so-called healthy kidney after death. In this present case, there was very considerable difference of opinion as to the actual colour of a kidney during life, as nobody had happened to see one before, and in so young a subject, one might reasonably enough have suspected the colour would have been brighter and more florid, or in this case it might have been expected to be in some degree blanched from loss of blood; the fibrous brittle appearance which its injured surface presented was very notable; the escape of urine from it when the wound was dressed an hour after admission was undoubted, and carefully verified.

Another remarkable feature was the very slight hæmorrhage which occurred; of course we could take no note of the amount of internal extravasation, and the after symptoms of local inflammation which appeared render it not improbable that there was at least an appreciable amount, but the wound was so direct and patent, that there could hardly have been any serious internal loss of blood without a corresponding escape of it externally.

The symptoms of undoubted peritoneal mischief which arose were in all probability due to extravasation of urine through the surrounding cellular tissue; from the position of the wound, it was hardly possible to maintain the lad in such a posture as to ensure an absolutely free discharge from it, and during the restlessness of his delirium this was clearly out of the question, and what little urine escaped from the kidney during the first day of his illness, soaked its way through the cellular tissue, and set up a corresponding inflammation; this, in all probability, involved the crus of the diaphragm, and very considerably impeded its action, though there was some reason to suspect the existence of slight pleuritic mischief as well.

The lad complained occasionally of pain in the penis when

passing water, but never of the pain in the loins or testicle said to occur after such injuries as this. The complete soundness with which so large a wound eventually healed shows very well to what an extent, and in what perfection, any amount of local injury may be repaired in young subjects. Up to the present time the boy is in perfect health, but it is an interesting question how far an actual wound of substance, and deprivation of its capsule to a greater extent, may be the origin of remote yet insidious structural changes in the injured organ.

ARTICLE XIV.—*The Inheritance of Cancer, and its Relation to Questions concerning the local or constitutional Origin of the Disease.* By W. MORRANT BAKER.

THE question whether cancer is a general or local disease, at its beginning, has been much and often discussed; and the weight of evidence has seemed, probably at all times, to some to be on one side, and to others on the other. And this will not seem strange if we take into account the exceeding uncertainty which hangs about those parts of the life and history of cancer, from which alone satisfactory data concerning its nature can be obtained. The differences of opinion, moreover, which prevail, have been also increased, probably, by the term 'general' or 'local' not always expressing the same idea to different people. Thus some, in employing the term 'constitutional' to express that condition which they believe to exist before the development of any local cancerous growth, use it in the same sense, and to denote the same thing, that is believed by others to exist only after the system has become infected from a so-called primary tumour. On the other hand, many who believe in the presence of some peculiar state of the blood, or, more correctly perhaps, of the body in general, antecedent to the growing of a tumour, have in their mind a notion of something different from the disease itself, but yet issuing at last in its development. In discussing this subject, therefore, it is necessary to keep in view the difference of opinion, or rather of idea, as much of the pertinence of any argument concerning the nature of cancer will depend on the notion that is entertained of this pre-existing condition. Thus, it may be,

and indeed has been argued,* that the comparative rarity of congenital and infantile cancer favours the opinion that inheritance has but little to do with the production of this disease. And on the supposition that cancer, as such, is transmitted from parent to child, this argument is a strong one. But on looking at the fact from the other point of view, namely, that which regards the inheritance as one of a tendency to the disease, not of the disease itself, it is seen to agree with what occurs in other diseases of which the frequent inheritance is undoubted, but the time of appearance of which, in the offspring, although in most cases earlier, corresponds tolerably closely with the period at which it showed itself in the parent.

In the present state of our knowledge, it is, doubtless, impossible to say with certainty whether cancer is, at the first, a local or general disease, but it may be of some use to direct attention to those parts of the records of the disease, from which an inference concerning this question may be drawn. For much of what is frequently brought forward is of no real value as evidence, because, although it tells to some, and even a great extent in one direction, it tells at least equally in the other,—its relative value for one or the other side, like relative height in perspective, depending entirely on the point from which it is viewed. And indeed there is no subject that could illustrate better than cancer how much may be said, equally true and equally forcible, on both sides of an argument. It becomes, then, especially necessary to consider carefully what can and what cannot serve as a help in settling this disputed point, and among the comparatively few matters in connection with cancer that have a real bearing on the question, whether it is at first a local or general disease, the most important, perhaps, is that of inheritance.

I. *The Inheritance of Cancer.*

The inheritance of cancer is sometimes treated as if its acknowledgment as a fact must settle the point in dispute, and decide in favour of this disease being a general one. And, at first sight one is apt to agree with this; because the inheritance of constitutional affections is more common, or, at least, more noticeable than that of local defects. But, on further examination, this is evidently an unwarrantable conclusion. Many deformities, and, nearer still, the common epidermal cysts, might be cited in proof. Both are commonly reckoned local affections, but their occasional, and in the latter instance fre-

* The Antecedents of Cancer: by C. H. Moore, F.R.C.S. 1865.

quent, transmission by inheritance is undoubted.* The proving, then, of cancer to be hereditary, will not prove it to be a constitutional disease; but the manner in which it is transmitted from parent to offspring is of considerable importance in relation to the question before us. The bearing, indeed, which the inheritance of cancer has on the subject under discussion may be said to narrow itself to this point,—is cancer inherited always in corresponding organs in parent and offspring? If it were so, indeed, the merely local nature of the disease would not be proved, because all diseases seem to have a special liking for particular tissues or organs, and it might be expected that in the child they would show the same preference that they did in the parent. But such an occurrence would afford a very strong reason for believing the disease to be the property of a particular organ rather than of the body in general. The facts, however, as they have been hitherto observed, show that no such confinement of cancer to one organ in successive generations is noticeable. The proportion of cases of inheritance in which there occurs transmission of this disease from one organ in the parent to another in the offspring, has but little to do with the general question whether a constitutional condition precedes or accompanies the local development of cancer. For if it be acknowledged to occur in one case out of a hundred or a thousand, the merely local nature of cancer cannot be assumed. Nor can it be said that in the remaining cases the disease is only local, until some difference can be, at the same time, shown between them and the single case in which the disease is transmitted in a different manner. Of course, the smaller the proportion of instances in which the disease attacks different organs in successive generations, the greater is the chance of such an occurrence being the result of coincidence, and of inheritance having nothing to do with it; but for the assumption of cancer being only local in its nature, it is necessary to deny altogether its transmission otherwise than as a local disease,—from organ to organ, so to speak.

Again, the occurrence of the same kind of cancer in both parent and offspring tells neither way in an attempt to settle this subject. For it might be expected beforehand that the transmitted disease would be like that from which it had been handed down. But if it can be shown that the inherited is sometimes a modification of the old disease, and one that is

* Mr. Paget remarks of these sebaceous or epidermal cysts: 'It is interesting to notice the frequent hereditary origin. Perhaps, in the majority of cases, the bearers of these have known one or more members of their family similarly endowed.' Lect. on Surg. Path., 2nd ed. p. 436.

prone to affect other organs, there will be proof of a relationship between the different kinds of cancer, and a strong argument for their constitutional origin.

For a complete and final settlement of this subject, if, indeed, such a thing can be, it will be necessary to make a far greater number of exact observations than have been yet recorded. Those at my disposal, however, are sufficient to justify the belief, for the present, that a special constitutional condition precedes the formation of a local cancerous growth. And on these grounds—1. That the disease is not always in the same organ in both parent and child, nor in the same always in all those relatives who are affected by the same inheritance. 2. That the transmitted disease is often a modification of that of the parent from whom it is inherited.

Of the great value of the statistics to be immediately quoted, I have no hesitation in speaking, because they are not my own, but kindly placed at my disposal by Mr. Paget. A portion of them has been already published by him.* The remainder has been collected since. None of them have been chosen with reference to any particular result, but recorded simply in the order in which they came under his own observation.

No. of Case and Sex.	Organ affected in Patients.	Relatives having Cancer.	Organ affected in Relatives.
1 F.	Breast	Cousin	Breast.
2 M.	Great-toe; medullary	Father	Lip.
3 F.	Breast	Grandmother	Breast.
4 F.	Labia; epithelial	Aunt, cousin, sister	Breast.
5 F.	Breast	Sister	Breast.
6 F.	Breast	Mother	Breast.
7 M.	Lower lip; epithelial	Grandmother	Breast.
8 F.	Breast	Mother	Breast.
9 F.	Breast; medullary	Mother, sister	Breast.
10 F.	Breast	Mother's uncle	Lip.
11 M.	Cheek; epithelial	Aunt	Breast.
12 M.	Gum; scirrhus	Mother	Breast.
†13 M.	Larynx; epithelial	Brother	Scrotum; epithelium.
14 F.	Mole on the foot; melanotic	Great-grandmother	Breast.
15 F.	Breast	1st Cousin	Uterus.
		1st Cousin	Lip.

* Med. Times and Gaz., August 22, 1857.
† Both the brothers were chimney-sweeps.

No. of Case and Sex.	Organ affected in Patients.	Relatives having Cancer.	Organ affected in Relatives.
16 F.	Breast	Maternal aunt, 2nd cousin	Breast.
17 F.	Breast	Mother	Breast.
18 F.	Breast	Great-grandfather	Face.
19 F.	Femur; medullary	Father	Face.
20 F.	Breast	Father's mother, Father's sister	Breast.
21 F.	Breast	Aunt, 2nd cousin	Breast.
22 F.	Conjunctiva; epithelial	Sister	Breast.
23 F.	Breast	Mother	Breast.
24 F.	Breast	Aunt	Breast.
25 F.	Breast	Brother	Eye.
26 F.	Breast	Sister	Breast.
27 F.	Breast	Sister	Breast.
28 F.	Scalp; melanotic	Mother	Brain.
29 F.	Shoulder; melanotic	Mother	Face.
30 F.	Breast	Mother	Breast.
31 F.	Breast; medullary	Mother, sister	Breast.
*32 M.	Lower eyelid; epithelial	Brother.	Scrotum, epithelium.
33 F.	Axillary glands; scirrhus	Sister	Breast.
34 M.	Tongue; epithelial	Sister	Breast.
35 F.	Pharynx; epithelial	Great-grandmother, grandfather, great aunt	Unrecorded.
		Mother	Uterus.
36 F.	Breast	Mother	Int. cancer.
37 F.	Breast	Sister	Breast.
38 F.	Breast	Sister	Breast.
39 F.	Breast	Grandmother, great aunt	Unrecorded.
40 M.	Finger; epithelial	Mother	Breast.
41 F.	Breast	Sister	Stomach.
42 F.	Breast	Sister	Breast.
43 F.	Breast	Aunt	Breast.
44 F.	Breast	Grandmother	Breast.

* Both the brothers were chimney-sweeps.

No. of Case and Sex.	Organ affected in Patients.	Relatives having Cancer.	Organ affected in Relatives.
45 F.	Breast	1st Cousin . .	Breast.
46 F.	Breast	Aunt . . .	Breast.
47 F.	Breast	Sister, cousin .	Breast.
48 F.	Breast	Mother . .	Uterus.
49 F.	Breast	Mother, aunt .	Unrecorded.
50 F.	Breast	Mother . .	Breast.
		Brother . .	Stomach.
51 F.	Breast	Grandmother .	Breast.
52 F.	Breast	Sister . . .	Breast.
53 F.	Breast	Mother . .	Unrecorded.
54 F.	Breast	Sister . . .	Unrecorded.
55 F.	Breast	Two aunts, great aunt . .	Unrecorded.
56 F.	Breast	Sister . . .	Uterus.
57 F.	Breast	Sister . . .	Breast.
58 F.	Breast	Great aunt . .	Breast.
59 F.	Breast	Two 2nd cousins, 3rd cousin .	Unrecorded.
60 F.	Breast	Mother . .	Unrecorded.
61 M.	Tongue; epithelial . .	Grandfather . .	Mouth.
62 F.	Breast	Two 2nd cousins .	Unrecorded.
63 F.	Breast	Father . . .	Under arm.
		Sister . . .	Breast.
		Sister's daughter .	Breast.
64 F.	Breast	Grandfather . .	Face.
65 F.	Breast	Grandmother .	Unrecorded.
66 F.	Breast	Mother . . .	Unrecorded.
67 F.	Breast	1st Cousin . .	Stomach.
68 F.	Breast	Mother's sister .	Unrecorded.
69 F.	Breast	Father's sister .	Uterus.
70 F.	Tongue; epithelial . .	Mother, sister .	Unrecorded.
71 M.	Œsophagus; medullary . .	Sister . . .	Unrecorded.
72 F.	Breast	Aunt . . .	Uterus.
73 F.	Breast	Sister . . .	Breast.
74 F.	Breast	Grandmother .	Breast.
75 F.	Breast	Mother . .	Breast.
76 F.	Breast	Mother's sister .	Unrecorded.
77 F.	Breast	Mother's sister, mother's 1st cousin	Breast.

No. of Case and Sex.	Organ affected in Patients.	Relatives having Cancer.	Organ affected in Relatives.
78 F.	Breast	Mother	Breast.
79 F.	Cancer under axilla	Mother's mother, mother's mother's mother	Uterus.
80 M.	Tongue; epithelium	Father	Abdominal cancer.
81 M.	Breast	Father	Leg, melanotic
		Sister	Unrecorded.
82 F.	Breast	Mother	Breast.
		Cousin	Unrecorded.
83 F.	Cheek	Mother	Liver.
		Sister	Œsophagus.
84 F.	Uterus	Father	Jaw.
85 M.	Breast	Mother	Breast.
86 F.	Breast	Father's sister	Breast.
87 F.	Breast	Mother's sister	Breast.
88 F.	Breast	Father	Cancer under the arm.
89 F.	Breast	Mother	Uterus.
90 F.	Stomach	Daughter	Breast.
91 F.	Œsophagus	Daughter	Cervical glands.
92 F.	Breast	Uncle	Scalp.
93 F.	Breast	Mother	Breast.
94 F.	Breast	Aunt, cousin	Breast.
95 F.	Breast	Mother's sister	Breast.
		Mother's mother	Eye.
96 M.	Shoulder; medullary	Daughter	Breast.
97 F.	Breast	Mother's aunt	Face.
98 F.	Breast	Grandmother, sister	Breast.
99 F.	Tongue	Father's brother	Cervical glands.
100 M.	Tongue	Mother	Uterus.
101 F.	Breast	Mother	Intestines.
102 F.	Breast	Mother	Abdominal walls.
103 F.	Breast	Mother's sister	Rectum.

In this list of cases there were altogether 45 instances (in 41 families) of direct inheritance from father or mother; and of these, the disease was in the same organ in both parent and child in 19, and in different organs in 26 instances.

It is a curious fact that in all the cases but one in which the disease occupied the same site in both generations, the breast was the organ affected; the exception was the uterus. As might be expected from this fact, almost all these patients were females, only one case occurring of cancer of the breast inherited by a male.

Among the cases of direct inheritance from a parent, but in which the disease was transmitted to a different organ in the child, 9 were instances of inheritance from the father, and 17 from the mother.

There were 16 instances (in 14 families) of inheritance from a grandparent or great-grandparent, or both. Of these, 8 were cases of cancer in the same, and 8 in a different organ, in the two generations. As before, the cases of disease of the same site in both generations were cases of cancer of the breast, with a doubtful exception (Case 61).

There were 49 families—some of them have been included in the former statement—two or more members of which had cancer, the relationship between whom, however, was not that of parent, or grandparent, and child. Of these, 28 were families in which the disease was seated in the same organ in all the relatives affected, 21 in which different organs were attacked.

There were 25 instances of the disease in brothers or sisters, or in brother and sister. In 14 of these, the same organ suffered in both the relatives affected, in 11 a different one. The whole of the 28 instances of cancer of the same organ in all the affected relatives were cancers of the female breast, and 14 of them were in sisters.

The cases in the tables in which the kind of cancer and its site in the relative were unrecorded, could not, of course, be used in drawing out the foregoing results; but it seemed best to leave them where they stand, for the sake of the information which they afford concerning the comparative frequency of inheritance from or in common with various relatives.

The only way by which doubt can be cast on the result drawn from these tables must be by doubting whether many of the so-recorded inheritances are really inherited, and are not rather only coincidences of disease in the same family. And that the occurrence of cancer in relatives is sometimes accidental cannot be doubted. But we are scarcely justified, at least in the present state of our knowledge, in supposing that more than

a small minority are so. The question, however, can be finally answered only by discovering the proportion of cancerous relatives belonging to those not cancerous; and this, for a fair comparison, would be somewhat difficult. In the paper by Mr. Paget,* before referred to, it is stated that among 147 patients with innocent tumours (neither cancerous nor recurring), only 6·8 per cent. were aware of any relative having cancer, while 22·4 per cent.† of the cancerous had one or more relatives with the same disease. So far as it goes, therefore, this would seem to show that the number of accidental coincidences of cancer in the same family is but small; and it is probably balanced, or more than this, as Mr. Paget remarks, by the addition that would have to be made, if the cases of inheritance from patients with unknown internal cancers could be reckoned also. If then it be granted that the preceding tables represent, practically, real inheritances, we may suppose that more than one-half of the inherited cancers are transmitted to a different organ in the child from that which they affected in the parent. And if this be so, then cancer must be allowed to be not merely a local disease.

II. *The Results of Operation.*

No part of the history of cancer has, perhaps, so much to do with any question relating to its local or constitutional nature as the manner of its inheritance; but it may be well to refer to one or two other points which are often used in discussions concerning this disease. The results of operation are very frequently brought forward in support of the argument for the constitutional character of cancer, because they show how hopeless of permanent good any undertaking for its removal by these means must almost always be. And, indeed, the records of surgical interference with cancer furnish a strong reason for the notion that the disease is not merely a local one. But they cannot afford any proof of this. If there is a tumour to be

* Med. Times and Gaz., August 22, 1857.

† In a more recent paper (Med. Chir. Trans. 1862) containing a résumé of Mr. Paget's statistics, the per centage of cancerous patients who had cancerous relatives is noted as 24·2; and this high number is probably to be attributed, in great measure, to the cases having been gathered largely among patients in private practice, the family history of whom would be much better known than that of the hospital cases. From this circumstance, as well as from the fact (of which I am informed by Mr. Paget) that cancer appears, commonly, in the offspring at an earlier age than in the parent, it may be inferred that all statistics of this disease, and especially those which are taken from hospital records alone, must give too low an estimate of the frequency of its inheritance.

removed, there is also a source whence the blood may have been tainted. And by those who uphold the theory that cancer is propagated by seeds of disease gathered from the primary tumour by the blood, and thence scattered, it might be said that the system was infected before the source of evil was removed.

Local recurrence after operation, moreover, is no proof of the constitutional nature of cancer, because it is so impossible to say what is really recurrence, and what the continuance of disease that has escaped the knife of the operator. And by this is not intended disease that with greater care might have been seen and removed, but that which at the time of the operation is invisible to the naked eye, and would be so, probably, under the microscope also. For it cannot be doubted that an area of disease in such a stage of imperfect development must always surround and precede, so to speak, the perceptibly developed tumour, to which alone, in ordinary language, the name cancer is applied, and therefore must, frequently, be in part left outside the line of incision that limits the portion to be removed.

But, on the other hand, no argument for the local nature of cancer can be drawn from the same source. Whether it is possible for a constitutional disease to exist, and yet be prevented, for an indefinitely long time, from showing itself locally, on account of the want of an appropriate place for its development, is a question that might be fairly argued, even were it a fact that the removal of the first local manifestation of such a disease prevented its further production; for the kind of constitutional affection commonly supposed to exist, in this particular instance, is a very different thing from that which is produced secondarily by absorption from the primary disease. It is unnecessary, however, to enter into any such discussion until it has been proved that the removal of a primary cancer does prevent its production in other parts. Without doubt, removal of the local disease does often much good, and on any theory respecting the nature of cancer, it might be expected so to do, both by removing the local evils which exist, and by preventing those which afterwards would have arisen from its presence. It is quite true, moreover, that many cases are on record in which, after the removal of a cancer, the patient has remained free from apparent disease for so long a period as to justify the belief that at least for a time the malady was in abeyance. But it is doubtful whether such cases are too numerous to be balanced by those in which, without operation, the patient lives for many years with an

old cancer, that shows no sign of activity and gives no trouble. Nobody is in a position to deny the possibility of extirpation of cancer by operation, and say that the notion is absurd; but surely there is at present no evidence of permanent cure by these means sufficient for the foundation of an argument against the constitutional nature of cancer. The comparative number of successes to failures will, of course, always have a different effect on different minds, and it may be that exceptional instances of permanent good from operation are accumulating at a more rapid rate than are those in which, without operation, patients live an equally long time. But the question now is only whether there are at present facts sufficient to justify a belief in the merely local nature of this disease, and it must, I think, be confessed that statistics of the result of operation in cancer are among the least hopeful sources of support for any such notion.

III. *Multiple Cancers.*

The simultaneous development of many cancerous growths is frequently supposed to indicate that this disease is constitutional. But the support to be derived from this occurrence is not strong. It may be, possibly, as Mr. Moore believes,* that such cases are not met with under the more exact observation of the present day. It is very doubtful, however, if, in the present state of our knowledge, it is possible to decide whether an outbreak of cancerous tumours is a simultaneous development, or a consequence of rapid dissemination from one primary source. Supposing a case to come under the notice of two equally good observers—a case, say, in which, among many cancers, no one was, in respect either to size or situation, sufficiently eminent to rank indisputably as the first tumour formed—it would by no means follow that their opinions would agree about the character and manner of the cancer's development. To the mind of one it might seem a convincing example of widespread disease following a constitutional taint—to the other, only an instance of how difficult it sometimes is to decide which is the parent and which the offspring.

But even were it allowed that the local manifestation of cancer begins always in one part of the body, it by no means follows that the later developments are offsprings from this single spot. It cannot be doubted, of course, that some of them so arise, as, for example, those in the lymphatic glands belonging

* Loc. cit., p. 9.

to a cancerous tissue or organ. But there is no proof that all the growths which are developed secondarily, in point of time, to the so-named primary disease, are secondary to it in any other sense. That a disease should manifest itself locally, first in one part of the body, then in another, is no proof of any direct dependence of the later upon the earlier manifestations. It is no more than happens in constitutional syphilis, in gout, in tuberculous disease. The argument from analogy is as forcible for one side as the other. The sequence is a fact; it may be explained as well by supposing a primary constitutional disease as a local one.

The settlement, however, of this doubt, would still leave unanswered the main question, whether a special constitutional condition precedes the first local growth.

The object of this paper, it need scarcely be said, has been rather to show what matters relating to cancer have any real connection with existing doubts concerning its nature, whether local in its origin or constitutional, than to bring forward all the arguments that might be used on one side or the other. It has been rather an attempt to demonstrate what seems the more reasonable view of the subject from the standing point of what evidence now exists, than an endeavour to show that the question can now be settled. So little is known about this disease, that any doctrine concerning it must be taken reservedly, and only on trust. But of the two ideas concerning the origin of cancer—that which is involved in the notion of a pre-existing constitutional condition being necessary for its local development, and that which supposes local changes alone to be sufficient for its production—I cannot but believe that the former is the one which, on the evidence now before us, can be the more reasonably supported.

Article XV.—*Observations on the Pathology and Treatment of Joint Disease.* By Holmes Coote.

THE following two cases of disease of the knee-joint present some points of pathological interest to which I would direct attention.

In the study of diseases of joints generally, it is without doubt necessary to be acquainted with all the morbid changes to which the component parts of a joint, i. e. synovial membrane, bone, cartilage, ligaments, and so forth, are subject; but the sequence of such changes, and the causes which produce them, are matters of equal, if not greater importance in practice.

Thus, inflammation of the synovial membrane leads to the effusion of synovial fluid into the joint in large quantity. Injuries, exposure to cold, rheumatism, and other causes are sufficient to excite such inflammation as a primary disease, and in the greater number of cases the disease is limited to its primary seat and is easily curable. In other and rarer cases the joint becomes filled with pus, and in such cases the disease is less manageable, and is often incurable; but the cause of its incurability lies, not, as has been supposed, in the want of power on the part of the absorbents to remove pus, but in the fact that the inflammation has spread to other structures, and assumed a more general and destructive character.

In other cases, agonising pain is felt when the patient bears on the limb; the sleep is disturbed by nocturnal cramps, and the contour of the knee is altered. This disease is called 'ulceration of cartilage.' But more accurate investigation shows that the primary seat of disease is the cancellous tissue of the head of the bone, the inflammation being excited, perhaps, by some local injury; thus, in consequence of such local inflammation, the articular cartilage becomes destroyed at its deep-seated surface by a layer of vascular and highly sensitive granulations springing from the bone. The disease should be more properly designated inflammation of the 'joint,' or inflammation of the cancellous tissue of the head of the bone, with shedding of the cartilage, and the treatment would consist in the application of such measures as would restore the bone to its healthy and normal state.

Another group of diseases depends upon errors of nutrition, independent of any process which we can strictly term 'inflammation.' Fibrillation, and the gradual removal by absorption of the articular cartilage, occur in persons the subjects of chronic

rheumatism. We meet with this condition symmetrically, and in very many of the joints in the same subject. No local treatment seems to arrest it. In other cases, the articular extremities of the bones become soft and friable. Fractures occur on the application of the least force. We have here to search the cause in all that produces textural atrophy. There may be progressive loss of nerve power, either local, as in partial paralysis, or universal, as in general paralysis. Lowly organized deposits, such as tuberculous matter, may encroach upon and produce absorption of the bone; soft cancerous growths may spring into existence, or the whole limb wither from disuse.

It is with the view of illustrating some of these points that I offer the following remarks.

Case I.—John P——, æt. 39, hoop-mounter, a strong, active man, of dark complexion, somewhat over five feet seven inches in height, states that last August twelvemonth, being then in full work, he became affected, without apparent cause, with pain in front of the left knee joint. He went to another hospital, where he received medicines which in the course of six weeks afforded him the necessary relief. After five months, however, the pain returned and he again sought advice as an out-patient; the pain became more severe, so that he was obliged to walk with the aid of a stick—but he persisted in his work, for the benefit of his family. Towards the end of last November there was a very great aggravation of his symptoms, but he did not give over work until April, when he was forced to take to his bed. He applied for relief at St. Bartholomew's Hospital, May 3, 1866, when he was admitted under my care.

Upon inquiry I could find no adequate cause for the disease, unless it were a fall which befel him three years ago, when the affected limb 'bent under him.' His present aspect was sallow and unhealthy: he looked like one who had gone through a long course of suffering; limbs shrunken. The knee of the affected limb measured sixteen inches, that of the sound limb fourteen and three-eighths. The thigh of the affected limb measured thirteen inches; that of the sound limb thirteen and three quarters. The leg of the affected limb just below the knee measured twelve inches; that of the sound limb eleven and three quarters. There was a slight amount of displacement of the left tibia and fibula backward and outward. He had experienced occasional startings of the leg, perhaps twice in a fortnight, just as he was dropping off to sleep; but in general his rest had been unbroken and the appetite unimpaired. I regarded this as a case of chronic inflammation of the cancel-

lous tissue of the head of the tibia, for the following reasons—The constant and abiding pain in the joint, unattended by synovial inflammation; the occasional startings of the limb, indicating an incipient separation of the cartilage from the head of the tibia; the increase of pain after bearing on the limb in work; the enlargement in the circumference of the head of the tibia; and the pain always excited by pressure or by examination. The great indication was obviously rest, combined with counter-irritation; the administration of opiates when necessary, and good nutritious diet. Accordingly he was ordered to remain in bed; to have applied a succession of blisters of moderate size; meat diet, and a pint of porter daily; steel medicines and an opiate when necessary.

On the 14th there was increase in pain, when ice was applied in a bladder, and on the 17th the transcurrent cautery was used, and repeated, but without any obvious effect. On the 18th he was ordered six ounces of port wine daily.

On June 23, the knee was covered with lint spread with camphorated mercurial ointment and then strapped and rolled; no perceptible improvement ensuing, it was discontinued after a fair trial. Quinine was next administered, and finally a lotion containing opium was ordered, which seems to have been most soothing to him. In this state he continues; being about to try the effect of sea-air in a Convalescent Hospital.

The question may be asked why the limb, then the seat of an obstinate and unmanageable disease, has not been removed by amputation. The patient's objection to an operation is insuperable; and both his age and general aspect are scarcely such as would warrant us in urging on him such a proceeding. The rate of mortality after amputation of the thigh in the adult of his age varies from 50 to 80 per cent.! The operation of resection was wholly inadmissible.

Case II.—Mary H——, æt. 60, married, always in the enjoyment of good health, was engaged in household work until about five years ago, when she became affected with erysipelas of the right leg. On recovery she resumed her usual avocation, but soon went into a union in consequence of increasing infirmities. She says that early in May she twisted the left leg, and feeling great pain, walked about three miles to a hospital to obtain relief. She returned with the proper medicines, and in the course of a fortnight came to St. Bartholomew's Hospital, where she was admitted May 26, 1866. No inquiries could elicit from this patient a more connected history of her case. The limb, which was greatly swollen, was put

into splints, well padded, and a lotion was applied. In the course of a week a more accurate examination was made, when an irregularity was detected, clearly indicating a fracture of the head of the tibia. She was naturally restless, and, being free from pain, moved the limb without thought. Repeated suppurations had formed; but in other respects her health was good.

August 1.—She lies crippled in bed; the leg near the ankle is much shrunken, but the upper part is swollen and out of shape; there is sufficient union to allow of the limb being moved with care, but it is not strong enough for use. There is a small sore on the heel. Her general condition is unaltered.

Of all the structures composing a joint, two only are the subjects of primary inflammations, namely, the synovial membrane and the cancellous tissue of the bone: and of all joints, the knee is most commonly the seat of disease, in consequence of its extent of surface, and the shocks and other injuries to which it is liable. In the consideration of diseases affecting this as all other articulations, then, we speak of synovitis, and inflammation of the cancellous tissue, as types of inflammation; atrophy of cartilage, elongation of ligaments, or gradual distension of the capsule, we regard as errors of nutrition, not necessarily of inflammatory origin.

Primary inflammations of joints therefore are thus divided: —

a. Synovitis—acute or chronic.
b. Inflammation of the joint itself, commencing in the bone and spreading thence to other structures.

In the diagnosis of a difficult case this information is valuable. A patient, as in the case of John P——, is the subject of a diseased knee—but we ascertain that there is no history of synovitis—we therefore naturally direct our attention to the extremities of either the femur or the tibia; and in this case find that there is the usual amount of general atrophy of the limb, except around the circumference of the tibia, where there is an enlargement to the extent of at least a quarter of an inch in the affected member.

Pathology teaches (Museum of St. Bartholomew's Hospital) that in inflammation of the cancellous tissue there is an immediate increase in the vascularity of the part, accompanied by loosening of the connections of the articular cartilage (Ser. ii. No. 37): that between the bone and the cartilage there is a layer of exceeding sensitive granulations. The under surface of the cartilage is irregular and eroded. Superficial ulceration

of the bone is very common (Ser. ii. Nos. 1, 4, 5, 8), while deep seated ulceration may destroy the normal shape.

But there are yet other effects of inflammation on the cancellous tissue. It may become dark coloured, sodden, soft, and friable, when the patient for months, and even for years, feels easy only when at complete rest, but suffers on the least exertion. In this state an abscess may form, the progress of which, always painful, is marked by swelling and thickening of the walls of the shaft, consolidation of the remaining cancellous tissue, ulceration to give the confined matter vent, and irregular deposits of bone around and over the ulcerated surface. The cavity of the abscess is sometimes lined by a soft vascular membrane (Ser. i. No. 82), or the excavation may be irregular and the bone porous and spongy (Ser. i. No. 163). In some instances there are numerous cavities (Ser. i. No. 184). In others there is an abundant growth of new bone externally (Ser. i. No. 201).

In these abscesses, portions of dead bone are often found, or the necrosed bone may still adhere to the living parts. A specimen (Ser. i. No. 123) was taken from a young woman, the subject of necrosis of the tibia for many years, in whom amputation of the limb was performed on account of the inflammation of the joint then excited. A section of the head of the tibia shows a small portion of dead bone in the centre of its cancellous texture. A fistulous passage extended from the skin, covering the front part of the head of the tibia, to the dead bone in its centre. The synovial membrane was very vascular, and covered on its internal surface by a layer of lymph, into which injection freely passed from the vessels of the synovial membrane.

CASE I.—I regard this case as illustrating the earlier stages of this formidable disease, and although it appears in a chronic form, its stages, unless checked at the commencement, are not the less sure. Among the wealthier portions of society, the imposition of absolute rest can, in most cases, be carried out effectually; this measure, combined with change of air and all necessary accessories, usually suffices to effect an early cure. But among the working classes the case is different; the patient feels himself bound to work to the very last, and thus exposes himself to the morbid changes here described, which in an advanced stage are uncontrollable.

What medicines can avail against such a disease? Iodide of potassium or mercurials; opium to allay pain; blisters, the actual cautery; incision down to the bone; the use of the

trephine; the hypodermic injection of morphia, have all in succession been tried and approved for the relief of different symptoms. In some cases they have done good, while in others they have been found wanting. The great principle of treatment is rest, long continued and unremitting, until the healthy nutrition of the parts has been restored, and the tissues are fit to sustain the duties imposed on them.

CASE II. illustrates another form of disease. A woman aged 60, both mentally and bodily feeble, fractures the head of the tibia by some sudden movement of the body. The fracture in all probability was incomplete and impacted, but so trifling an accident could not have been followed by such an effect in a healthy person.

The first point, to which, irrespective of the local injury, my attention was directed, consisted in her imperfect utterance, and incomplete articulation; in her forgetfulness of facts, and incapacity to give any connected account of her previous life or present accident. She laid in bed, with no care or thought of the future, indulging in occasional fretfulness, but otherwise contented. Her condition appeared to me to be that of a person in the earlier stages of 'general paralysis,' a disease to which my attention has often been directed through the kindness of Dr. Rhys Williams, the present superintendent of the Royal Hospital of Bethlehem. Among the many symptoms of this affection none is more constant than the peculiarity of utterance, and the dropping of certain syllables or words. In this state of disease there is gradual deterioration of all the tissues of the body; the muscles become fatty, and the bones light and friable. I saw a patient thus affected some months ago who had sustained a fracture of the long bones on two occasions from trivial causes; osseous union, however, took place in the usual time.

The pathological condition to which the bone is reduced may be thus described. The walls of the bone are thin and soft, and extremely light, their lamellæ being partially separated. The place of the medullary and cancellous tissue is occupied by a soft jelly-like fat, often of various hues. In one preparation (Ser. i. No. 233) scarcely any of the medullary texture remains, except a thin layer beneath the articular surface of the bone. The periosteum and articular cartilage are mostly healthy. The articular extremity, after immersion in spirit, seems to consist of a number of small cavities, which in the fresh state are filled with semifluid fat. Any sudden shock would break such a structure, and drive the portions

together in an impacted form. The shaft of a long bone may be reduced to a complete shell, filled with semifluid fat, no trace of cancellous tissue remaining. This morbid condition has been called 'eccentric atrophy,' the normal outline of the limb remaining.

In this case the patient's age and general want of power were strong arguments against amputation, and she herself was little disposed to listen to such a suggestion.

Finally, I would call attention to a remark which I have already made in a former volume of these Reports—namely, the vast and unmistakeable difference which exists between atrophy from simple disuse of parts, and atrophy from impairment or arrest of nerve power. The former is slow in its progress, and easily repaired by renewal of exercise. It consists chiefly in simple diminution in bulk. But the atrophy from deficient nerve power is textural, and consists in a positive change in the material. Thus bony matter is replaced by soft semifluid fat; muscular tissue by granular albuminous material. In the young there is immediate arrest of growth in length as well as circumference, and these symptoms are the more marked, the more complete the state of paralysis.

I know of no instance of recovery from this condition.

ARTICLE XVI.—*On the prejudicial effect of Inter-articular Pressure in Joint Disease; and the application of continuous extension, by means of a weight, as a remedy for this condition.* By F. HOWARD MARSH.

IT is an obvious fact that many chronic and troublesome affections arise out of a trivial injury, the repair of which is prevented by some single adverse circumstance. Thus, a chronic ulcer of the tongue may be the result of a scratch which would at once have healed but for the constant irritation of its surface by the sharp edge of a broken tooth. Fissure of the anus is an ulcer which cannot heal, because it is frequently torn by the contraction of the muscular fibres on which it rests. An abrasion of the skin over the tendo Achillis becomes a troublesome sore, because it is often disturbed by the movements of the subjacent tendon. In any of these cases, if the one adverse circumstance is removed, the affection ceases to be unmanage-

able, and may soon be cured. Many cases of joint disease seem to belong to this catalogue. They begin after a slight injury, or after exposure to cold, as a trivial inflammation, and they progress from bad to worse, till the articulation is destroyed; yet their tendency is thus dangerous not because the patients in whom they are met with are the subjects of any faulty constitutional state, or because the inflammation with which the joint is affected is essentially intractable; but because the process of repair is constantly hindered, and the present mischief increased by injury which befalls the diseased structures from without. This injury consists of pressure of the articular surfaces against each other; and it is caused both by any ordinary movement of the limb, and also by abnormal contraction of the muscles which surround the joint.

I would not be understood to believe that the destructive tendency exhibited by diseases of the joints depends in every instance on this one condition of inter-articular pressure, but I feel assured that many cases are made serious solely by its influence, and that every case in which it is present is greatly aggravated by its action.

In the present paper I wish to dwell more especially on the bearings, on joint disease, of pressure produced by abnormal muscular action; but reference will also be made to that which is caused by any ordinary movement, active or passive, of the limb.

No formal proof of the presence of abnormal muscular action in association with diseases of the joints need be offered. It is a complication which is only too conspicuous. Its effects are most plainly seen in the early stages of disease, in the jumpings and startings of the limb, which are so agonizing to the patient; and in the later, in the distortion which it produces; —witness the displacement of the head of the tibia backwards and outwards towards the ham in old disease of the knee. This abnormal condition of muscles has been especially studied by Mr. Hilton, who has pointed out that it is the result of reflex irritation. Mr. Hilton says,* 'When the interior of a joint is in a state of inflammation and irritation, the influence of this condition is carried to the spinal marrow, and thence reflected to the various muscles of the joint through the medium of the associated motor nerves, the muscles being supplied by the same nerves that supply the interior of the joint.'

It is evident that whenever muscular action takes place there must result pressure of the surfaces of the joint to which the

* Lectures on Rest and Pain.

muscles belong, against each other; for a long bone is a lever, moved by the muscles inserted into it, and having its fulcrum at that joint which is its centre of motion; and the first event in the action of a lever is that it is driven against its fulcrum.

The influence of such pressure as that which muscular action produces on the surfaces of a diseased joint is in the highest degree prejudicial; it is sufficient to alter the whole course and tendency of any case in which it is allowed to take effect; it is capable of converting a trivial wrong, which, but for it, would soon be repaired, into an intractable disease, which at length destroys all the structures of the articulation. Indeed, that this should be so cannot excite surprise, when it is remembered how powerful the force exerted by muscular action is, and that this force is constantly taking effect on surfaces which are inflamed and very tender, or covered with a layer of exquisitely sensitive granulations, or which are formed by exposed and ulcerating cancellous tissue. Given this pressure, and the condition of the articular surfaces, and it is evident that great injury must be produced. But clinical evidence on this point may easily be obtained. While house surgeon at the Hospital for Sick Children I several times performed the following experiment, in order to ascertain how much of the pain present in hip-disease is due to inter-articular pressure. A child, who on admission presented well-marked signs of acute disease, including startings of the limb and night-screamings, was placed in bed with no other treatment than a weight suspended from the foot of the affected limb, for the purpose of drawing the diseased joint surfaces asunder. In almost every instance the acute symptoms quickly subsided. When the child had become free from pain, which was usually the case in from three to eight days, I went during the night, while he was asleep, and raised the weight and lodged it in the bottom of the bed, thus setting the muscles free from restraint, and giving them liberty to contract. In a very few hours many of the children so treated became restless in their sleep, and presently awoke with the old pain, and screaming. They soon fell asleep again when the weight was allowed to hang down so that the muscles were again restrained.

In many cases of acute joint disease the patient is free from pain during the day, or as long as the limb is not moved, and while abnormal reflex action is restrained by an effort of the attention; but upon any movement of the limb, or the jarring of the bed by heavy footsteps in the room, or during sleep, when irregular reflex action is allowed, pain becomes severe.

The common method of testing the amount of increased

sensibility of the structures of an affected joint is by direct pressure, or by jarring the limb so that the surfaces are brought into sudden contact. Even slight pressure carefully applied frequently causes very severe pain.

Patients with joint disease suffer great pain during the movements of the limb, which are unavoidable in re-arranging the bed, or in changing a splint. It will be found that this pain may be very much reduced, or wholly prevented, if, during any movement, the joint surfaces are kept apart by an assistant who pulls gently at the limb grasped below the joint,—at the leg when the knee is diseased, at the thigh, or the leg in the case of the hip. Some children with acute hip disease, although they scream when they are turned on the affected side, or when the limb is jarred, may be drawn pretty quickly down the bed by the affected limb without complaining, for this movement can be performed without throwing the joint surfaces into contact. I have often found that children who scream on any movement of the limb when this plan of extending the part is omitted, may, when it is adopted, be carried from one bed to another, or be thoroughly washed, without giving evidence of pain.

Seeing how grave an influence is exerted on the course of joint disease by the pressure and bruising which the articular surfaces inflict upon each other, both during any movement of the limb, and during muscular action, it is obviously of importance in treatment (1) to keep the surfaces at all times from contact, and (2) to control the muscles. The only means for keeping the joint surfaces from contact is by applying extension to the limb; and this to be efficient must be continuous in its action, and appropriate in its amount. Now, as a matter of fact, in the ordinary treatment of joint disease, no extension is employed except in the management of hip disease by means of Liston's long-splint, in conjunction with the perineal band. But even in this instance the apparatus for extension is not satisfactory. The perineal band is generally made of small size and of inelastic material, so that, if it is fastened tightly enough to secure adequate extension, there is great risk of its leading to excoriation of the skin of the groin; while, if it is safely loose, it wholly fails in its purpose. Indeed, many surgeons find it so inefficient that they do not employ it at all, but treat diseases of the hip as they do those of other joints, by simply securing rest in a fixed posture by the use of a rigid splint. Thus it may be said that in the ordinary treatment of joint disease no extension at all is employed; or, in other words, that no provision is made for keeping the diseased surfaces asunder.

It may be objected that a joint is so tightly braced-up by its ligaments, and by the surrounding tendons, that no amount of extension which can be safely applied will be sufficient to separate the articular surfaces from each other. But this is not really the case. On the contrary, anyone may easily convince himself that every joint is constructed loosely enough, and that its ligaments are long enough, to allow a certain separation of its surfaces from each other. In the trick of making the fingers crack by pulling at them, it can be seen after the 'crack,' and when extension is continued, that the surfaces of the metatarso-phalangeal joint are distinctly separated from each other. Some persons are born with hereditary 'loose joints,' others have loose joints from general weakness; and in both cases the abnormal looseness differs only in degree from that which is natural in healthy joints. A certain amount of play in a joint is necessary for its free movement. From a physiological point of view the object of this looseness is evident; there is in it a provision for the functional rest of the articular cartilage. The principal office of cartilage is to resist pressure, and from this it requires intervals of rest. In a healthy joint this rest is provided-for, for the articular surfaces are but lightly in contact at all times, except when the joint is under the influence of muscular action, or when it supports the weight of some part of the body. The necessity of providing artificially for this relief from pressure in disease is obvious, when it is remembered that in the spontaneous course of events, not only is the pressure constant, but also that it is greatly increased in its amount by abnormal muscular action. Extension, by means of a weight, is well adapted for supplying this need.

The second indication in the management of joint disease to which I have referred is to control irregular muscular action. As this abnormal action is due, as we learn from Mr. Hilton, to the inflamed condition of the joint, it may be expected to subside when the inflammation passes off. Until this is the case, however, it will be found that it is perfectly controlled by continuous extension. By this force the muscles are tired out, and are soon made to capitulate.

It detracts nothing from the advantages of the weight as compared with the use of splints, even combined with extension, to allow, what is certainly the fact, that muscular action commonly ceases when the splint is used; because the firm bandaging by which the splint must be fixed, not only causes some wasting of the limb, but is also prejudicial to the inflamed joint textures; for example, firm bandaging of the thigh, in knee disease, unless it is very carefully regulated, has a ten-

dency to interrupt the circulation, and so to cause engorgement of the vessels of the affected structures. If extension by the weight is adopted as a means of quieting the muscles, either no splint at all need be employed, or the limb may rest lightly in a splint, to which it is bandaged so loosely, that no pressure on soft parts is produced.

Having had an opportunity of observing the results of treating diseased joints by simple extension, applied by means of a weight, I propose to mention the practical advantages which this method offers, and to relate some illustrative cases; but, as the remarks that I have to bring forward are founded principally on cases of hip disease, I shall first mention, as briefly as possible, what are the chief objections to the use of the long splint and perineal band—the apparatus most commonly employed for the management of this affection—in order that the comparative advantages of the treatment by simple extension may be more clearly set down.

It is true that the long splint prevents any movement of the limb, and restrains muscular action; and that symptoms quickly subside under its use. It, however, inflicts an amount of inconvenience which is quite unnecessary, except during the most acute stage of disease. A joint which is seriously diseased must be kept, for the first few weeks, at absolute rest; but, as the acute symptoms subside, and the tenderness, which was due in great part to injury inflicted on the articular surfaces by movement of the limb or by muscular action, passes off, more liberty may be safely allowed, provided that no close contact of the surfaces is permitted. The child may turn on the sound side, or sit up and amuse himself. Now the long splint does not allow an abatement of the restraint which it inflicts to go on 'pari passu' with the abatement of the acute symptoms; the patient, when convalescent of his disease, must submit to the same degree of confinement as when his disease was at its worst.

In many conditions the long splint cannot be satisfactorily adjusted; as, for example, when there is twisting of the pelvis, and projection of the hip; or when the thigh remains, as it frequently does, even after very forcible extension, more or less flexed upon the pelvis, and inverted; when there is abscess, either presenting or discharging on the outer aspect of the thigh, (splints interrupted at the hip I believe allow of so much motion at the joint as to be useless); or when sinuses are so situated as to prevent efficient bandaging.

The long splint requires an amount of frequent readjustment, which it is sometimes, especially among out-patients, not easy

to carry out; and it makes the observance of cleanliness a matter of difficulty. Space need not be occupied by the notice of other objections to this apparatus; and I may go on at once to speak of the weight.

This appliance was recommended by Sir Benjamin Brodie in the first edition of his well-known work on 'Diseases of the Joints,' in the following terms: 'I will not say that the effect of such a continuance of extension (by the weight) is to prevent the shortening of the limb altogether, but I am satisfied that it will, in many instances, render this less than it would have been otherwise; at the same time preventing, or very much diminishing, the excessive aggravation of the patient's suffering, with which shortening of the limb is usually accompanied.' This statement is founded on practical experience, and is independent of any doctrine as to the pathology of joint disease; but the scientific explanation of the good effects of this 'continuance of extension' is contained in the words of Mr. Hilton:* 'Is it not indisputable that if we allow an inflamed knee-joint to lie on a soft pillow unconstrained by mechanical means, the muscles of the joint, stimulated to undue exertion, never allow the articular surfaces to be kept quietly in apposition without pressure upon each other?'

In spite of Sir Benjamin's advocacy, and although it has been used from time to time by many of the highest surgical authorities, the weight has failed to come into general use in England; probably for the want, which has till recently existed, of some simple and safe method of adjusting it to the limb. American surgeons, however, employ the weight very largely in the case of all the joints; and by the help of the 'strapping stirrup,' invented by Professor Pancoast, of Philadelphia, are enabled to apply it with the greatest facility. Although Pancoast's stirrup is very commonly adopted in England for making extension from the foot by fastening this to the end of the long splint, in the treatment of fractures of the thigh, I shall venture to describe the manner of its application when the weight is required for disease, either of the hip or the knee. A long piece of stout adhesive plaster, from two to four inches wide, according to the size of the limb, is bent upon itself at its middle, and made to adhere to the leg as high as the knee, one half running up the outer, and the other up the inner aspect of the limb; a loop or 'stirrup,' between four and five inches deep, being left below the sole. The two pieces running up the leg are secured by circular strips and a

* Op. cit.

lightly applied bandage. The weight is suspended in mid-air, at the foot of the bed, by a stout cord, tied into the stirrup.

It will be found convenient to attend to the following details in the use of the weight. Time should be allowed for the strapping to become thoroughly adherent to the surface before the weight is applied, otherwise the stirrup is apt to be dragged off. A few hours (six or eight) are sufficient for this. The strapping ought to be of stout material, and recently made. Care should be taken to defend the malleoli from too great pressure by the sides of the stirrup; for this purpose, American surgeons are in the habit of using what they term a 'spreader.' This consists of a thin piece of wood, a little longer than the foot is wide, which they place transversely in the stirrup, just below the sole, like the 'set-stick' in a horse's trace. A pad of cotton wool placed above each malleolus will answer the same end. Some patients, when the weight is applied, acquire the habit of sliding down in bed till the weight lodges on the floor. This may be effectually prevented by raising the bottom end of the bed six or eight inches, by placing books, or wood blocks, or bricks under its feet. The amount of weight which it is proper to apply is a point of great importance; but it must often be a matter of experiment to ascertain, in any case, how much is required. As a general rule, for children between the ages of six and nine years, four pounds are appropriate; but, while I have seen it necessary to apply as much as twelve pounds, gradually accumulated upon four, in a case of recurring muscular contraction in old hip disease, in the person of a child eight years old (Case No. IV.), I have found in other instances that children of this age complain of any weight exceeding three pounds. The weight should be as small as is consistent with its purpose of relieving pain, and preventing or removing distortion; so that it is advisable to diminish the amount as symptoms subside, and not to continue to employ a heavy weight after it has become unnecessary to do so.

The weight bears favourable comparison with the long splint in the following particulars. It may be applied by one person without the use of chloroform and without any violence to the limb, and if care is taken, without any pain or fright to the patient. This is so even in the most acutely sensitive condition of parts. It relieves patients from all unnecessary restraint. Children, during the acute stage of disease, will remain by choice in the horizontal posture, but when the urgent symptoms have subsided they experience great relief from the liberty which may safely be allowed them of turning over towards the sound side, or of raising themselves slowly into the sitting posi-

tion, so that during the day they may amuse themselves with toys, or by watching what is passing around them. It will readily be conceded that the less irksome and wearisome any method of treatment is, the greater is the likelihood of its being fairly carried out. The weight-apparatus does not interfere either with the circulation, or with nutrition, as the long splint is apt to do through the circular bandaging of the whole limb which it necessitates. As it leaves the whole limb above the knee completely exposed, it does not obstruct examination of the seat of disease, or the observance of cleanliness, or the application of local remedies, such as the actual cautery, or blisters, or poultices. Mr. Holmes, in the after management of cases of excision of the hip, now, instead of employing a long splint interrupted opposite the wound, uses the weight along with sand-bags for the lateral support of the limb. Several cases in which he has adopted this plan have been successful. The 'stirrup' is easily adjusted and easily kept in order, and if well put on, will last good two or three months without renewal. It may be applied in the out-patients' room, and the child may be consigned to the management of its mother, and it will then be sufficient that the progress of the case is from time to time reported on. Mr. Smith is in the habit of so using the weight among his out-patients, and it is found that mothers who are fairly intelligent and careful are able to conduct the treatment satisfactorily with the help of occasional advice. While the long splint is adapted only for confining the limb in the straight position, into which, in the majority of cases, it must first be brought by extension, which must often be injurious to the diseased structures, the weight may be used while the distortion is still present, and this it will generally remove by overcoming the abnormally contracted muscles. In some instances of old-standing disease, there is, along with deformity of the limb, so atrophied and weak a condition of the femur that forcible extension preliminary to the putting on of the long splint involves a considerable risk of fracture, and here the gentle action of the weight is very advantageous.

For liberty to use the following cases I am indebted to the kindness of Mr. Holmes, under whom they have been treated at the Hospital for Sick Children.

CASE I.

A. Vernon, æt. 6, not evidently strumous, and in whose family there was no history of consumption, was sent up from the country to the Children's Hospital early in the winter of 1864, for the treatment of a diseased hip. I have no note of the exact date of the first symptoms, but the disease was said by the father to be due to a fall. The child had not walked for fifteen weeks. In the three weeks preceding her admission she had been in very severe pain, often screaming loudly enough to be heard across the street. The pain was in the hip and in the ham. It was stated that the head of the femur was dislocated, and that it had been reduced several times, but had always become displaced again. The child was evidently in great agony. She had wasted quickly, and now was hectic, and with an expression of countenance indicative of great terror. On an examination, necessarily conducted under chloroform, the leg was found flexed upon the thigh, and the thigh on the pelvis. The head of the femur was plainly felt upon the dorsum ilii. Reduction and re-dislocation could be practised with the greatest ease; the head of the bone (which seemed scarcely altered in size) jerking visibly over the rim of the acetabulum; grating between the head of the femur and the floor of the acetabulum was distinctly felt. The dislocation having been reduced, the limb was placed in the straight position between sand-bags; and a weight of four pounds fixed to the foot by means of the strapping stirrup. Nothing else was done. From this time the child became absolutely free from pain, except when she was carelessly moved, and then she immediately screamed out and showed all her former terror. She slept through the first night almost without waking. She very soon became fresh-looking and merry, playing with other children in the ward, and saying that she was 'nearly well.' In a few days she got into the habit of raising herself on her elbows to play with toys, and within the first fortnight she sat up in bed almost all day long. Dislocation did not recur. On December 27, Mr. Holmes ordered a leather splint to be moulded to the hip, the weight being removed. For three or four days no return of symptoms was observed, but on January 1 the child began screaming, and subsequently had such bad nights that on January 4 the weight was again put on. On the night of January 4 she slept well, and from that date was wholly free from pain. The weight was left off a second time on February 4, and that night the pain again returned, and became so severe, that in the two days

which elapsed before Mr. Holmes's next visit the child lost her appetite, looked pale and anxious, and screamed almost constantly during the night. The weight was then by Mr. Holmes's order immediately re-applied, and the pain was again directly removed. She was, after the lapse of several weeks, able to dispense with the weight, and at length left the Hospital apparently convalescent, but with directions still to remain in bed, and to wear a leather splint for the hip.

Case II.

W. Smith, æt. 5, came into the Hospital under Mr. Holmes on October 17, 1864, for the treatment of a diseased hip. The disease had been caused by a fall fourteen months previously. He had become lame, and of late pain had been extremely severe, so that for many nights his parents had been obliged to sit up with him. He took little food, was very much wasted, and had profuse night-sweats. There was found on examination a considerable prominence of the hip, and so much twisting of the pelvis that a long splint could not be applied. There was extreme tenderness about the joint, and the child screamed loudly when moved. A weight of four pounds was applied to the foot: nothing else was done. For the first night the child screamed much as usual, but afterwards the screamings became less and less frequent, and within four days they had all but ceased. The twisting of the pelvis and the projection of the trochanter were much diminished, and the limb had assumed a much better position. The sweating ceased, and the appetite became good. It is fair to say that within four days after his admission the child was relieved of all severe pain. He left the Hospital after some months' treatment apparently cured, with the limb and pelvis free from distortion.

Case III.

W. A. Williamson, æt. 7, was admitted into the Children's Hospital under the care of Mr. Holmes, in July 1864. Her history was that four months before she had fallen upon her left hip, and a fortnight afterwards had become lame: the lameness had increased, but she had had scarcely any pain. Her condition on admission was the following:— The left thigh was drawn up and rotated outwards to a very marked degree, and the leg was flexed upon the thigh to about a right angle; thus the limb was in a very odd position, the foot being up in the

air nearly on a level with the opposite knee—about as it would be supposing the child should sit nursing the foot on the opposite knee, and should then stand upright upon the opposite leg, changing the position of the affected limb as little as possible. On examining the hip, no tenderness or alteration in shape, except that due to posture, could be detected. A weight was applied, at first of three pounds, afterwards increased gradually to eight. The limb came down into the natural position within a week; the child complained of no pain, and after six weeks was discharged with the limb quite straight, with directions to continue the weight till permission was given to leave it off. The parents were poor and neglected her, and it was afterwards ascertained that the symptoms developed themselves into those of confirmed hip disease.

Case IV.

A. P., æt. 9, came under Mr. Holmes's care in 1864, for the relief of a deformity of the left lower extremity from old hip disease. A number of old abscess-scars were seen about the hip. The limb was much shorter than its fellow, and so drawn up that the toe could not touch the ground by nearly ten inches. Under chloroform, firm fibrous anchylosis was made out. The hip was straightened as far as Mr. Holmes thought safe, and a weight of four pounds was applied. No mischief followed the straightening. The limb was not again drawn up, but the weight failed to bring it further down. In a fortnight the limb was still further straightened, and again at the end of a week. A long splint was now applied, as the weight, increased to eight pounds, did not keep the limb in a good position. Owing, however, to the remaining deformity, the splint did not fit well, and the child, who was very restless, was able still to bend up the thigh. A splint was therefore put along the front aspect of the limb and abdomen as high as the ribs, and a weight of twelve pounds hung from the foot. This plan answered well enough, the limb gradually straightened, and subsequently the tendency to re-contraction passed off, so that the child at length left the Hospital with a straight leg, and with a certain amount of movement at the hip. She now walks well with a high-heeled boot. In this case the amount of extension required to keep the limb straight could not have been made by the help of the perineal band; and, as was found, the long splint could not be kept in position. The anterior splint was applied more for the sake of securing additional restraint, and to prevent re-contraction, than for bringing down the limb. It had little effect of this nature, and was not readjusted.

Case V.

A boy, 7 years old, was admitted into the Children's Hospital under the care of Mr. Holmes, with strumous disease of the knee. The joint was considerably enlarged, the leg was drawn up, and the head of the tibia was displaced in a very marked degree towards the ham. The child complained of pain when the knee was touched, and screamed frequently in the night. A weight of four pounds was suspended from the foot, and sand-bags, to give lateral support to the limb, were used. All acute symptoms passed off in a few days, and in a month the patient was discharged with a tin splint adjusted to the ham, and a weight of three pounds at the foot. A month afterwards, when he was brought to the Hospital, the limb was quite straight; there was scarcely a trace of the former backward displacement of the head of the tibia; there was no pain or heat about the joints; and the child had a good appetite, and had gained flesh.

Before concluding I may mention two cases in which the weight was useful in straightening limbs contracted by the scarring of extensive wounds produced by burns.

Case VI.

T. S., æt. 5, was admitted into the Children's Hospital, with her leg drawn up to within a right angle by the contraction of a scar following an extensive burn in the ham, and back part of the leg and thigh. By dividing the cicatrix Mr. Holmes was able to straighten the limb; and a large piece of skin was turned from the outer aspect of the thigh to fill up the wound. In consequence of the poor health of the child, the transplanted flap sloughed, and thus a very extensive wound remained; the whole ham, and a portion also both of the thigh and leg, being without skin. In this condition of parts no splint for preserving a straight position of the limb could be applied, but by means of a weight hung from the foot perfect extension was maintained till cicatrization was complete. The child at length left the Hospital with the limb quite straight, and with the scar soundly healed.

Case VII.

A. B., a boy aged 6, was taken into the Children's Hospital under Mr. Holmes, with both arms flexed at the elbows to their

full extent by the contraction of burn scars, which extended on the front aspect of each limb from near the wrist to the middle of the upper arm. The scars formed large, cord-like masses, which were continually ulcerating at different parts of their surface. The boy was placed in bed, and a weight of three pounds was suspended from either hand (by means of strapping made to adhere to the palmar and dorsal aspects), and allowed to hang over a raised cross-piece at the foot of the bed. Great trouble was caused by the constant ulceration of the surface of the scars, but the cord-like bands gradually yielded, and became flattened out, and within a month the arms were straight. For many weeks healing of the ulcers about the scars went on slowly, and there was a very strong tendency to recontraction; but at length the boy was allowed to be up, his arms being bandaged to straight splints placed on the dorsal aspect of the limbs during the day, and having the weights still applied during the night. He finally left the Hospital with his arms in a straight position, and with the old scars soundly healed.

Article XVII.—*Notes on Oxaluria.* By Dyce Duckworth, M.D. Edin.

IT has not hitherto been proved that oxalic acid is a normal constituent in the urine of healthy persons. That it is not fair to seek to establish this fact from observations conducted upon hospital patients will hereafter be shown.

There seems to be sufficient evidence that this acid is a natural constituent of the blood, probably combined, as in the case of certain vegetables, with protein matters.

All observers, subsequent to Golding Bird, have agreed in adopting his two divisions of the cases furnishing oxalate of lime deposits in the urine, viz. 1, those suffering from a temporary or accidental oxaluria, and 2, those affected with permanent or confirmed deposits.

Now it is certain that cases of the first class have no claim to be considered as pathological entities, while those that may fairly be referred to the second category are not so frequently met with as is perhaps supposed.

As is well known, certain articles of diet, especially of the vegetable class, induce the first or temporary form of the affec-

tion, but seldom with symptoms of any importance except in the cases of some gouty persons, or those subject to articular rheumatism. In these instances to partake of cooked rhubarb or English champagne is almost certain to bring on return of pain in addition to dyspeptic symptoms.

The oxalate of lime is found in the urine, on microscopic examination, immediately after being passed, and if the specimen be permitted to stand, a deposition of the crystals occurs in a very characteristic manner, which will be presently described more in detail.

In most of these instances soluble oxalates are introduced into the blood, and we have only to imagine a very simple transformation taking place, dependent on the well-known affinity of oxalic acid for lime. In the case of rhubarb, however, we have abundant octohedral crystals of oxalate of lime taken into the stomach,—anyone can satisfy himself of this by teasing out cooked rhubarb in water, and examining the sediment which occurs: this, though so insoluble a salt when taken by the mouth, certainly reappears in a short time in the urine, and in the form of octohedral crystals.

I lately made some experiments with a view to induce temporary oxaluria by the ingestion or administration, sometimes of lime water, and sometimes of oxalic acid. I first ascertained that the urines were free from oxalates on all occasions before experimenting. This was done by examining microscopically the morning urine of the day previous, it having stood for twenty-four hours.

Experiment I.

Took at 10 p.m. f. ℥iij of Liquor Calcis in the same quantity of spring water. Passed urine at 11 p.m.; let it stand over night. In eight hours a mucoid deposit had occurred in it; in this abundant octohedral crystals were detected under the microscope. In the urine passed the next morning, only a slight deposit took place: this, on examination, was seen to contain a few small octohedral crystals. Some were likewise found in the urine passed at mid-day, after it had stood for ten hours.

Experiment II.

Took f. ℥j of Liquor Calcis at bedtime. Examined morning urine of next day, found large octohedra of oxalate of lime, and some globular masses of carbonate of lime.

Experiment III.

Gave f. ℥j of Liquor Calcis to a strong healthy man at bed-time. Octohedra of oxalate of lime were found on microscopical examination of the morning urine of the next day.

Experiment IV.

Took at 11 p.m. a third part of a solution of three grains of Oxalic Acid in spring water. Feeling cramping pain in the stomach, I did not take the whole of the solution as intended (it was well diluted). In about an hour passed f. ℥ij of urine. Examined this next morning, found a characteristic mucoid deposition in it, and recognized myriads of octohedral crystals under the microscope.

Experiment V.

Took a grain of Oxalic Acid in f. ℥ij of distilled water. Passed urine an hour afterwards. On examination next morning found octohedral crystals, and some granular masses which responded to the chemical reactions of oxalate of lime. Suffered no gastric uneasiness on this occasion.

Thus it appears that by taking from half to two grains of lime in the form of liquor calcis (an ounce of which contains about half a grain of caustic lime), oxalate of lime can be produced in the urine. A similar result also ensues if a grain of oxalic acid in solution be swallowed.

I have never met with dumb-bell-shaped crystals either in the natural or artificially-induced temporary form of oxaluria.

The second, permanent or confirmed, form of oxaluria may usually be diagnosed by the accompanying constitutional symptoms, as well as by certain physiognomical signs presented by the patient. It is certainly more common in town than in country life: I have, however, observed severe cases in farm labourers and sailors. An amount of languor and mental depression, with hypochondriasis disproportionate with the evidence of disease as elicited by any physical signs, a dingy muddy hue of complexion, more or less dyspepsia, and a certain degree of wasting, will point to the probability of persistent formation of oxalate of lime. The peculiar burning pains across the anterior lumbar regions which Golding Bird described in these cases, I have several times observed in a

marked degree.* Sometimes fixed pains are referred to the dorsal region, and weakness in the loins is complained of. The tongue constantly presents fissures running in a longitudinal direction, *i. e.* parallel to the raphé, and on pulling out the organ laterally these are rendered more apparent. It is likewise pale and more or less sodden in appearance, seldom foul or coated.

In watching these cases from day to day under hospital treatment, several points worthy of notice regarding the urine present themselves. The specific gravity is generally high, according to my own observations varying from 1.013 to 1.037. The urea is in decided excess. Deposits of phosphates and urates sometimes accompany the oxalates, and on some days alternate with them. Sometimes the urina sanguinis is more dense than the urina chyli, and 'vice versâ.' The colour is usually a full yellow, and after standing for some hours the characteristic deposit occurs. This is apt to be mistaken for mucus, and in truth in the majority of cases a considerable amount of epithelium is present with it. The upper surface of the deposit is disposed in a regular wavy and flocculent manner; it is more dense than in the lower strata; and if urates or phosphates are precipitated also, they fall subsequently to the oxalates, and are therefore seen lying on the top of the wavy surface.† Not unfrequently the urates are tinged with the morbid pigment purpurin. The reaction is always decidedly acid.

Octohedral crystals are not always to be found in the urine immediately on micturition. Sometimes they cannot be seen for some hours afterwards. Dumb-bell-shaped crystals are not unfrequently seen, generally in small proportion; and they are, as a rule, significant of the confirmed cases.

I have not observed that patients suffering from chronic oxaluria have manifested a decided inclination for sweet articles of diet, though this has been asserted by some; nor have I noted the tendency to boils and carbuncles, except in one case.

The frequency with which oxalate of lime occurs in the urine of hospital patients is truly remarkable. The cases yielding it would naturally, if classified, be referred to the temporary form. In examining lately the urine passed daily

* One patient remarked that he felt at times as if standing before the blast of a furnace.

† The peculiar disposition of the oxalate of lime deposit seems to be due partly to the form of the crystals, and partly to the presence of epithelium. It is quite characteristic.

from fifty patients in medical wards, I found it to be the rule for convalescents from most acute diseases to have oxaluria for a few days before the various secretions returned to the natural condition. Especially was this the case after scarlatina, typhus, typhoid, and rheumatic fevers. This peculiar urinary phase seemed to come on about the end of the first, or during the second week of convalescence. In some cases it continued for ten days, though with an occasional day or two of intermission. I also observed the occurrence of oxalate deposits in cases of phthisis, bronchitis with emphysema, saccharine diabetes, dyspepsia, lead poisoning, epilepsy, anæmia, neuralgia, hysteria, lumbago, chorea, erysipelas, eczema, impetigo, favus, psoriasis.

I am quite disposed to believe that this condition bears an intimate relation to the change of diet usually adopted about the period when it is observed. It is also partly explicable, no doubt, by other circumstances which are entailed by a hospital life, such especially as limited exercise or indoor confinement, and a fuller allowance of food than can be fairly disposed of by the inefficient pulmonary and other transformations.

Seeing then that oxaluria in one of its forms is of so frequent occurrence amongst hospital inmates, it would not, I think, be fair to draw any inference from their cases with a view to establish the existence of oxalic acid as a normal urinary constituent. Such an investigation should be conducted only upon persons in good health, living under varying conditions, provided always that these are in accordance with the principles of hygiene.

It would seem that confirmed cases are best treated out of hospital, an active life in the purest air being amongst the most potent therapeutic measures.

Article XVIII.—*On the Treatment of Acute Pericarditis with Opium.* By Frederic J. Farre, M.D. Cantab.

ONE of the most legitimate and obvious uses of a hospital journal is to publish the treatment and result of diseases which are more frequently met with in hospitals than elsewhere. One of these diseases is, I believe, Rheumatic Pericarditis. It occurs most frequently among the poorer classes, and the subjects of it generally find their way to the hospitals. Thus, I have had in the last three years eleven cases of rheumatic pericarditis, three of which were admitted on three successive days. These eleven cases have, with one exception, been treated mainly alike, though in a few of them the complications which occurred required the use of some additional remedies. I have selected rheumatic pericarditis for this paper because I am not altogether satisfied with the ordinary treatment of this disease, and because I think that a better result may be obtained by other means. The ordinary treatment in this country consists, I believe, in the frequent administration of calomel and opium, perhaps combined with antimony; the moderate abstraction of blood, chiefly from the region of the heart, by leeches or cupping; and the occasional application of a blister when there are signs of pericardial effusion. I use blisters, not only when effusion has taken place, but in every case as soon as pain is felt in the heart, and generally find that they afford the same relief as leeches or cupping. If, however, they fail to do so, or if the pain is very severe, I have recourse to the latter remedies. It is in the omission of mercury, except as a purgative, that my practice chiefly differs. I have been in the habit, like others, of considering mercury the most efficient means of controlling inflammation of serous membranes tending to fibrinous effusion; but the result of the mercurial treatment in this disease has so often disappointed me, that I have been led to consider whether some other mode of treatment might not be more successful. Certainly nothing can be more opposed to my own experience than the doctrine recently advanced, that the action of mercury is merely antisyphilitic, and that it has no general power of arresting the effusion of fibrine, or of promoting its absorption. I fully believe that it has this power, whether the inflammation be syphilitic or not, but there are circumstances in the case of pericarditis which interfere with the exercise of this power. One difficulty consists in 'the obstinacy with which the system often resists the influence of

mercury;' and another arises from the almost incessant motion of the heart, which is an unfavourable condition for an inflamed part under any circumstances, but is particularly so in this disease, on account of the greatly increased frequency of the movements; so that irreparable damage is generally done before the mercury can arrest or even modify the morbid action. Everyone who has treated cases of acute iritis knows how important it is to keep the inflamed iris perfectly quiet while mercury is doing its work. My first object, therefore, was to quiet as much as possible the increased action of the excited heart, ever liable to participate in the inflammation of its covering membrane. The remedies adapted for this purpose are to be found in the order Sedatives. Such are, among minerals, nitrate of potash, and tartarated antimony; and, among vegetables, digitalis, lemon juice, camphor, hyoscyamus, belladonna, hemlock, and opium. I may here state that I almost always use in acute articular rheumatism the salts of potash, generally the bicarbonate or the acetate; and that if pericarditis supervenes, I frequently substitute the nitrate, or combine it with one of the former; but I do not rely on these, nor do I make much use of antimony, though antimony is well adapted to diminish the activity of the vascular system in acute sthenic inflammation. Large doses of lemon juice will undoubtedly reduce both the frequency and force of the heart; but when the heart is inflamed I believe such doses are not free from danger. The same remark will apply to colchicum and digitalis, the latter of which, however, I sometimes employ when effusion has taken place. But the remedies which are most calculated to tranquillise both the heart and the nervous system are camphor, opium, hyoscyamus, and belladonna. Possibly any of these would accomplish the purpose, though not equally well. Camphor, hyoscyamus, and belladonna are very useful in palpitation arising from cardiac disease, but opium is peculiarly beneficial in rheumatic inflammation. It tranquillises the heart; it relieves pain; it procures sleep; it prevents delirium, which is not an unfrequent symptom of pericarditis; and it produces its effects speedily, and with tolerable certainty. Opium, especially when used alone, is generally regarded as an objectionable remedy in inflammation; but it is, nevertheless, frequently employed in enteritis, dysentery, cystitis, and incipient pulmonary catarrh. It is also of great service in peritonitis. In this disease it is usual to combine it with mercury, when it only plays a secondary part. But peritonitis has been treated very successfully by opium alone, in large and frequent doses. Such successful treatment of peritonitis will be found in a pamphlet

published by Mr. Bates, of Sudbury, and quoted in Watson's 'Principles and Practice of Physic.' Sir Thomas Watson especially points out the injurious effect of purgatives in increasing the peristaltic action of the bowels, and the great importance of the horizontal posture. Dr. Stokes and Dr. Graves bear testimony to the good, and even sometimes curative, effects of opium in the worst forms of peritonitis—those which arise from perforation of the intestines, or the bursting of an abscess in the peritoneal cavity. The whole chapter on the treatment of acute peritonitis in Watson's 'Practice of Physic' illustrates the importance of the principle I am here advocating. I have, myself, treated peritonitis successfully with opium, carefully avoiding purgatives, or only in case of necessity using an enema; so that the bowels were kept undisturbed—a condition as important in peritonitis as the tranquillity of the heart in pericarditis.

The eleven cases related in this paper are all the cases of rheumatic pericarditis which have come under my own care in St. Bartholomew's Hospital during the last three years. Two others, not included among them, have also been admitted into my wards during my absence, but have been differently treated. I shall relate, first, those cases of well-marked and pure rheumatic pericarditis, which were free from any except the articular complication.

CASE I.—*Acute Articular Rheumatism with Pericarditis—Recovery.*

George Dawson, æt. 32, a lithographic printer, admitted December 18, 1863, with acute rheumatism and pericarditis. He had been a steady man the last six or seven years, but was previously somewhat irregular, though never intemperate. Never ill before.

Three weeks ago he had rigors, became feverish, and left his work. All the joints, small and large, became, in succession, swollen, painful, and tender. A week before admission he had much pain in the cardiac region. No medical treatment had been used. On admission he was thin and pale, with pain in all his joints, none of which, however, were red or swollen. There was a little pain in the heart. The skin perspired moderately. He slept badly. The pulse was 96, regular, soft, and full, but rather undulating. The respiration was natural, and there was no cough. The tongue had the usual moist creamy appearance, but the appetite was good, and there was no thirst. The bowels acted regularly, and the motions were healthy.

The valvular sounds of the heart were natural; but friction was heard over the lower two-thirds of the heart, not at the base.

A blister was applied over the region of the heart; one grain of opium and twenty grains of bicarbonate of potash were directed to be taken every six hours; and tincture of iodine was, according to my usual practice, applied to the joints. On the following day, Dec. 19, the friction was less audible; on the 21st it was barely heard; and on the 23rd quite gone, unless the stethoscope was pressed against the chest, when a trace of it could be heard for four or five days later. After commencing the treatment, the pain in the heart quickly ceased; and the pulse fell to 80 on the 19th, and to 76 on the 21st, at which state it remained, continuing all the time soft and regular. The pain in the joints was removed in four days. In eight or ten days he was convalescent, and quickly recovered his strength and ordinary condition with the aid of meat and quinine and cod-liver oil. He was kept for a little time for observation, and then left with the heart's action and sounds and the pulse quite natural.

The pericarditis in this case had existed a week before admission, and the friction was only heard over the lower two-thirds of the heart. As the inflammation generally commences at the base of the heart, at the reflection of the pericardium, it had probably extended, during the week before admission, from the base to the apex; and it had either ceased at the base, or adhesion had already taken place there, before he came in. The very gradual fading away of the friction, and the possibility of reproducing a trace of it by pressing together the roughened surfaces of the pericardium, for four or five days after it had apparently ceased, seem to indicate that there was little, if any, adhesion in this case. Probably a 'white macula,' without adhesion, was ultimately formed.

CASE II.—*Acute Rheumatism with Pericarditis—Recovery.*

Lucy Duff, æt. 16, admitted December 19, 1863, with acute rheumatism. She was a healthy-looking girl, but, according to her own account, was always delicate and sickly. About six months ago she had a slight attack of acute rheumatism in the back and feet, which lasted only three days, but was accompanied with pain in the region of the heart.

There was, besides pain in the joints, some pain in the heart, and the fatigue of coming to the Hospital had caused palpitation. The pulse was 100, full and regular; the sounds of the heart were natural. The bowels had been confined three

days. Ordered four grains of calomel, and one grain of opium, to be followed, after four hours, by a draught of salts and senna, twenty grains of bicarbonate of potash every six hours, and a mustard poultice to the præcordium.

Dec. 21.—The bowels acted freely after the purgatives, the motions being lumpy and dark. The pain in the heart continued to be felt during inspiration, but the sounds were natural. Pulse 100, feeble and regular. A blister was ordered to be applied to the chest.

Dec. 23.—The pain was relieved by the blister, but friction was heard at the base of the heart. Six leeches were applied below the left scapula, and she was ordered to take one grain of opium every six hours, and to continue the bicarbonate of potash.

Two days afterwards I found her much relieved. The friction continued, but the pain of the heart had ceased, and the joints were better. There was, however, some headache, the headache being apparently caused by the opium. I therefore combined with it three grains of camphor. This had the desired effect; and I heard no more of the headache. The friction lasted altogether ten days; but after the pain had been relieved, the inflammation gave her so little inconvenience, that I had difficulty in persuading her that her illness was not, what she called it, 'a very trifling affair.'

Whenever the bowels are much confined, as they were in this case, I begin the treatment with a large dose of calomel and opium, followed by a purgative draught, and repeat the dose daily, according to the plan of Dr. Hope, until the motions are natural.

The leeches were probably useful here, though they were not applied until the pain had been relieved by the blister. Possibly, an earlier application of them, before the friction commenced, might have stopped the pericarditis at once, but I had no special reason to anticipate pericarditis. Endocarditis was more probable, and, in either case, I thought a blister sufficient. I have generally found the addition of camphor to the opium prevent the occurrence of headache, so that I usually order this combination. When the friction ceased, the opium was discontinued.

In this case the friction never extended beyond the base, and the recovery was apparently so perfect, that I feel sure there was no adhesion beyond this part.

CASE III.—*Acute Rheumatism—Pericarditis—Recovery.*

James Beale, æt. 22, a cocoa-nut mat weaver, was admitted December 20, 1863, with acute rheumatism of fourteen days' duration; his first attack. The pain in the joints was not severe, but there was some effusion in one knee. There was no pain in the chest, but some cough; and the first sound of the heart was a little prolonged. The pulse was 74, and regular. Bicarbonate of potash and ipecacuan wine were ordered to be taken every six hours, and a blister to be applied over the heart. Tincture of iodine to the joints as usual.

During the next five days the joints improved, the cough subsided, and the sounds of the heart became natural. He was therefore allowed a little meat. Still continuing to improve, he began on January 1 to take some decoction and tincture of cinchona, the potash was diminished to five grains, and the ipecacuan omitted. With the exception of a slight return of pain in the joints on the 5th, all went on well, and I already regarded him as convalescent; but on January 13 there was pain in the heart, with pericardial friction, double at the base, single at the apex; and the pulse had risen from 80 to 120. Pericarditis had commenced. I immediately applied leeches below the left scapula and a blister over the heart, and ordered a grain of opium every six hours, and twenty grains of bicarbonate of potash night and morning. Next day the pain was gone and the friction less. No alteration was made in the treatment during the four following days, at the end of which time there was very little friction at the base and none at the apex. There was no pain, and the pulse had fallen to 104. On the 6th day of the pericarditis the friction had entirely ceased, the pulse was 80 and regular, and there was no pain anywhere. The opium was now omitted. It had never caused any constipation. After taking tonics for a few days he was discharged cured on the tenth day from the commencement of the pericarditis, without any alteration in the natural force and action and rhythm of the heart, and without any sign of valvular disease.

In this case the leeches and the blister removed the pain and arrested the inflammation, and five or six days' treatment with opium was sufficient to put an end to it. The decrease in the pulse during the 6 days' treatment from 120 to 80 is very important. It is probable that some adhesion occurred at the base of the heart, but the rapid cessation of friction at the apex without signs of effusion leads me to conclude that

the adhesion was only partial, and at all events the muscular substance and the lining membrane and valves escaped injury.

CASE IV.—*Acute Rheumatism with Pericarditis and delirium, followed by Endocarditis—Incomplete recovery.*

Lucy Nesbitt, æt. 17, not a very strong girl, was admitted May 24, 1864, with pain in the heart and in several of her limbs of five days' duration. She had been delirious before admission. The joints were painful, but not red or swollen. The pain in the heart and the delirium before admission prepared me for pericarditis, which was indicated by a friction sound at the base of the heart. A blister was placed over the heart; and half a grain of opium was given every four hours, with bicarbonate of potash and chloric ether.

The following day (May 25) the pain being relieved, but not removed, six leeches were applied below the left scapula, which succeeded in removing it. During this and the third day the friction continued, and the pulse was 110. She now passed a week nearly free from pain. No delirium had occurred after admission, but the friction still continued; and on the tenth day the pulse became for twenty-four hours a little irregular. Probably some effusion occurred about this time; for a day or two afterwards the friction suddenly diminished, and, though it was faintly heard for a week longer, it never became conspicuous again. The pulse also became much more frequent. She was therefore ordered fifteen minims of tincture of digitalis every six hours, and the opium was continued. The digitalis did not diminish the frequency of the pulse, but, as it caused sickness, the tincture of veratrum viride was substituted for it, in doses at first of five, afterwards of ten and fifteen minims. The pulse, notwithstanding, still further increased in frequency, and rose one day to 160. On the 27th day pain was felt in the heart, and a blister was again applied. The pain was removed by the blister, and the pulse, which had resisted the opium, the digitalis and veratrum, began to fall.

The friction disappeared soon afterwards; but it had continued a month, and a week after it ceased a systolic murmur was heard at the apex. The still somewhat rapid pulse appearing now to arise from debility, she was ordered to take tincture of perchloride of iron in infusion of quassia and to leave off all other medicines. She left the Hospital about eight weeks after admission free from rheumatism, but with a rather unsteady pulse of 92, and a slight regurgitant mitral murmur.

The delirium which formerly gave rise to the idea of me-

tastasis to the brain, but which is no longer considered to indicate cerebral inflammation, is sufficiently common in rheumatic pericarditis to lead us to consider pericarditis as its ordinary exciting cause. But it sometimes occurs in acute rheumatism without inflammation of the heart or any other organ; so that we may have only the ordinary disturbing effects of rheumatism, pain, want of sleep and consequent exhaustion, and the poison of rheumatism, to account for it. Opium is most valuable in this state just as it is in the delirium of drunkards; but I find in practice that it sometimes fails when given alone, and that the addition of a volatile stimulus such as chloric ether greatly assists it, whenever there is much exhaustion. The girl Nesbitt had no delirium after admission, and, as I have often seen it occur before during pericarditis, I believe that its non-occurrence in this or any of the other cases related in this paper was in some degree due to the opium treatment.

Nesbitt, however, did not escape the subsequent endocarditis, while the pericardium became generally adherent, and the muscular substance of the heart, no doubt, inflamed also; so that the recovery was very imperfect.

CASE V.—*Acute Rheumatism with Pericarditis—Endocarditis —Incomplete recovery.*

Mary Lamb, æt. 16, was admitted on Saturday, November 4, 1865, with pain and redness in the hands and feet, and some pain in front of the chest. The rheumatism had existed eight days, and it was her first attack. The heart's sounds were natural, but the pulse was rather frequent. A mustard poultice was applied over the sternum and tincture of iodine to the joints, and a draught of acetate of ammonia was ordered to be taken during the day and Dover's powder at night. I saw her first on Monday the 6th. The articular pains were much relieved, but not the pain in the chest. Pericardial friction was now audible below the nipple towards the sternum: the pulse also had risen to 140, but was regular. A blister was placed over the heart, and she was ordered half a grain of opium and two grains of camphor every six hours, and twenty grains of bicarbonate of potash with the draught. The blister gave much relief. The next day, November 7, the heart was free from pain, the friction was for a time inaudible, and the pulse fell to 128. November 8, pain and tightness returned in the chest, and friction was heard again, though the pulse continued to fall, being 104. Eight leeches were applied below the scapula, which removed the pain, but not the friction.

November 9, the opium was increased to three-quarters of a grain with three grains of camphor. A mustard poultice was found sufficient to relieve any thoracic pain when it occurred. The pulse was 90 and regular. The friction continued more or less, being heard all over the heart, till the 21st, when it began to disappear at the apex. On the 23rd it was quite gone, and the increased dulness showed that its cessation was due to effusion. Another blister was applied, and in two days (25th) the friction reappeared and with it an endocardial murmur. By the 27th adhesion had taken place, and the friction finally ceased. The pulse a little increased in frequency during the effusion, rising to 100 and 108, but the heart's action was not at all laboured, and was always regular.

The endocardial murmur, which was not audible behind, became musical, and was probably caused by some cohesion or other alteration of the chordæ tendineæ. Mary Lamb remained in the Hospital nearly a month longer, taking quinine and wine and cod-liver oil till her health was quite restored; and when she left, the murmur, which had long ceased to be musical, and was often absent for several days together, had almost entirely disappeared.

In this case the pericarditis continued full three weeks and terminated in effusion and adhesion, and was followed by some endocardial lesion probably, however, not implicating the valves. The latter was of little importance, and was hardly perceptible when she left. The subdued action of the heart was very marked. The pulse, which at the commencement of the pericarditis was 140, fell in a week to 90, and the regular action of the heart was never disturbed. How far was this due to the tranquillising effect of the opium?

CASE VI.—*Acute Rheumatism with Pericarditis and Pleurisy —Recovery.*

Reuben Whitehead, æt. 16, was admitted September 28, 1863. He was a grocer's boy, frequently exposed to wet and cold, and had previously enjoyed good health. On September 20 he got very wet and wore his wet clothes all day. The following day he had an attack of acute rheumatism in his wrists, knees, and ankles. On the 23rd, the joints still becoming more painful, he felt much dyspnœa and uneasiness in the chest. These symptoms continuing, he came to the Hospital on the 27th. When placed in bed he looked pale and anxious, and lay on his back, being afraid to move on account of the thoracic pain. The pulse was 84, soft and regular; the respirations 28 in a

minute; and there was cough and scanty tenacious expectoration. Deep inspiration caused cutting pain. Most of the joints were painful, and the other symptoms were those of acute rheumatism. On listening to the chest a double superficial friction sound (pericardial) was heard at the base of the heart, and also a pleuritic friction sound on the left side, only during respiration. He was seen by Mr. Wood, who ordered a warm bath and a mustard poultice to the chest, to be followed by a blister, also thirty grains of bicarbonate of potash every six hours, and two grains and a half of calomel and half a grain of opium immediately.

I saw him on the following day, September 29: the pleuritic rubbing had disappeared, and the pain, both in the chest and in the joints, was much better. I applied myself, therefore, to the inflammation of the pericardium, the friction of which continued very distinct, and directed him to take a grain of opium every six hours, and to continue the potash. The pleurisy, however, though checked, was not removed, but returned two or three times with sufficient acuteness to require blisters, which always relieved it. The pain in the heart also returned on September 30, and October 2 and 5, and was each time removed by six or eight leeches below the scapula. Notwithstanding the pleuritic, the pericardial, and the articular pains, the opium procured him good nights. The pericardial friction continued, though it never extended beyond the base of the heart. On October 7 he expressed himself much better and quite free from pain, though there was a feeling of weight at the heart. On examining him I found that effusion had taken place; the friction had ceased, and the pulse was rapid, 120, and indistinct. I ordered ten minims of tincture of digitalis three times a day, and this was continued as well as the opium until the 14th. The sense of weight left him the next day, and the friction returned at the same time, and then gradually became less distinct till the 14th, when it disappeared. The pulse also gradually diminished with the friction sound, and had fallen on the 14th to 86, continuing always regular, but feeble; the opium and digitalis were now omitted, and with quinine and iron, meat and wine, he rapidly improved. At the end of the month he was quite well, having been under treatment four and a half weeks. The heart's sounds were quite natural, but he had occasional slight attacks of palpitation.

No doubt adhesion took place here; but as friction, though it continued fourteen days, was never heard beyond the base, the adhesion was probably confined to that part. The occasional palpitation appears to indicate that the muscular substance did

not wholly escape. No traces remained of the pleurisy, and he left feeling quite well.

CASE VII.—*Acute Rheumatism—Pericarditis, and Pleuro-pneumonia of right lung—Recovery.*

John Mahon, æt. 25, a miner, was admitted February 7, 1865. My notes of this case are imperfect. When admitted he had pain in the left wrist, for which he was blistered and purged with calomel and jalap. The following day it appears that he had some dyspnœa, with pain and crepitation at the base of the right lung, pericardial friction at the base of the heart, and a hot and dry skin. A blister was applied over the heart, and was directed to be kept open with mercurial ointment; and he was ordered five grains of calomel and a grain of opium immediately, and a draught of acetate of ammonia with nitre every four hours. I saw him on the 10th; the pulse was 124 and feeble; there was pain in the right side, with bronchial breathing, and pericardial friction at the base of the heart. He was ordered one grain of opium and three of camphor every six hours, bicarbonate and nitrate of potash with ipecacuan wine, and a blister to the right side. Under this treatment the pericarditis ceased in about a week, but the pleuro-pneumonia continued. The opium was now omitted. On the 17th there was still bronchial breathing and dulness, and some pleuritic rubbing on the right side, but no pericardial friction. The pulse was 112, regular, but feeble. The counter-irritation on the right side was continued; and as the pneumonia assumed an asthenic character, carbonate of ammonia was given with acetate of ammonia and ipecacuan wine, and a little wine allowed. The pleuro-pneumonia slowly subsided, and the heart retained no trace of the recent inflammation. He was discharged cured at the end of five weeks.

I cannot speak very positively of this case, but, as the pericardial friction was confined to the base, and ceased within a week, and there were no signs of effusion, I conclude that there was only partial adhesion.

CASE VIII.—*Rheumatic Pericarditis and Pneumonia of left lung—Recovery.*

John Broughton, æt. 22, admitted October 2, 1863. A strong, healthy young man, employed in a tobacconist's shop, of steady habits, well nourished, but rather pale. He had rheumatism at the age of fourteen in the joints and chest.

Eight days ago, September 25, he felt giddy and feverish, and had slight cough. The next day he had dull throbbing pain in the region of the heart, extending backwards between the shoulders. On admission, the pain in the heart still continuing, he was cupped below the left scapula to four ounces, a blister was applied over the heart, five grains of calomel and a grain of opium were given immediately, and acetate of ammonia with nitrate of potash every eight hours. I saw him on Monday, October 5. He complained of sharp cutting pain in the heart on coughing or deep inspiration; the pulse was 100, soft and regular, the respirations were 40, there was a hacking cough, and rusty difficult expectoration, and he slept badly. There was no pain in the limbs. I found pericardial friction at the base of the heart, and fine crepitation at the base of the left lung; the right lung was healthy. He was ordered a grain of opium three times a day, and acetate of ammonia with nitrate of potash and ipecacuan wine every six hours. The pain in the heart and the friction immediately began to diminish, and in four days, October 9, were quite gone. On this day the left wrist became painful and swollen, and continued so until the 12th. Meanwhile the pneumonia improved; the sputa became less rusty and tenacious, and the cough easier; the crepitation also became large and moist; and on October 12, a week after I commenced the treatment, nothing remained but a little ronchus and slight cough, with grey bronchial sputa. The opium was continued until the termination both of the pneumonia and the pericarditis.

From this time improvement was rapid, and he left the Hospital in three weeks, quite well; the lungs everywhere resonant and healthy, and the heart, as far as I could discover, quite natural.

This was rather an unpromising case. Here was not only pericarditis, but well marked pneumonia with painful cough and rusty sputa. I should not have given opium had this been a case of ordinary pneumonia, and I am aware that in doing so I acted contrary to the usual practice; but the former history, and the occurrence of articular rheumatism during the present thoracic attack, indicated, in my opinion, that the pneumonia was as much connected with the rheumatism as the pericarditis. The cupping no doubt assisted the recovery.

CASE IX.—*Acute Rheumatism with Pneumonia in both lungs—Pericarditis—Recovery.*

Edward Booth, æt. 19, a compositor, was admitted May 20, 1866, with acute rheumatism, cough, and pain in the right

side. He was not a strong man, and looked as if he had been hydrocephalic in infancy, which, however, I was informed was not the case. He had suffered from articular rheumatism three weeks, and during the last four or five days with pain in the chest, especially on the right side. He now complained of pain in the hands and right side, and dry cough. Pulse 124. The sounds of the heart were natural. A blister was applied to the right side, and he was ordered to take a saline draught with antimonial wine. I saw him on Monday the 21st. There was fine crepitation in both lungs behind, and pain in the left side. Heart still natural. A blister was applied to the left side, and nitrate of potash was added to the draught. Five grains of calomel were also ordered, to be followed by castor oil, which opened the bowels well. 23rd, the pain in the side was relieved by the blisters, but the expectoration was scanty and rusty, and the breathing was bronchial. There was also an obscure friction sound right of the sternum. 25th, the friction accompanied the heart action, and was evidently pericardial: the signs of pneumonia continued. He was ordered to take a grain of opium and three grains of camphor every six hours, and to continue the draught. On the 28th the pericardial friction and the crepitation had both disappeared, and the expectoration was no longer rusty: the pulse also had fallen from 124 to 80. The opium was now directed to be taken only at night. No friction was heard after this date, and only some large muco-crepitation for about ten days longer, with slight occasional pleuritic pains, probably due to adhesions, which were always relieved by mustard or flying blisters. The articular rheumatism had also ceased. He remained till the 18th, taking only tonics and cod-liver oil, and left without any pain or cough, or any trace of the pulmonary and cardiac inflammation, the heart's action and sounds being perfectly natural.

The rapidity with which the pericarditis subsided in this case was very marked. Both the cardiac and pulmonary inflammation were of a low character, rheumatic in their origin and nature, and afforded a favourable illustration of the good effect of the soothing or sedative treatment which I am advocating.

CASE X.—*Pericarditis after acute Rheumatism—Endocarditis —Carditis—Pleuro-pneumonia—Death.*

Thomas Major, æt. 12, admitted January 10, 1864. Two months ago this boy had acute articular rheumatism. The inflammation of the joints had now ceased, but during the last four days pain had been felt in the heart.

Jan. 11.—Pericardial friction was heard over the lower two-thirds of the heart, and more faintly over the base. He was directed to take three grains of calomel and a grain of opium immediately and afterwards castor oil, half a grain of opium six times, and twenty grains of acetate of potash three times a day, and to have a blister over the heart. The blister relieved the pain, and the next day (Jan. 12) the friction was only heard at the base. The sudden disappearance of the friction except at the base made me suspect pericardial effusion, and the pulse being 123, I directed the potash draught to be taken four times a day, with eight minims of tincture of digitalis, continuing the opium. Under this treatment the pulse quickly fell to 80, remaining regular. The friction continued, as before, only at the base, but a slight endocardial murmur was also heard. No further alteration occurred until the eighth day after admission (Jan. 18), when the friction was heard over a larger space, and the pulse became irregular. Another blister was applied over the sternum. On the tenth day (Jan. 20) the pulse was 72, feeble and irregular, and pleuro-pneumonia was detected on the right side. The digitalis was now omitted, and some wine was ordered, and ether. The next day (Jan. 21) I was informed that 'he died suddenly in a fit.' I was unable to ascertain by post mortem examination the precise cause of death. I have no doubt the heart was much injured, probably in its substance as well as both its surfaces; and pleuro-pneumonia, added to the very severe cardiac lesion, may easily have destroyed a boy of 12 years; but the sudden death, which was called 'a fit,' though I could not learn that it resembled epilepsy, seems to point out the digitalis as its probable immediate cause.

CASE XI.—*Acute Rheumatism with Pericarditis, Endocarditis, and Pleuro-pneumonia of both lungs—Recovery.*

John Briggs, æt. 29, a printer, was admitted on the 3rd of February, 1866, suffering from acute rheumatism. He had had a previous attack of articular rheumatism in May 1864, for which he was treated in Guy's Hospital. He was in the hospital fourteen weeks and had no pain in the chest, nor was any treatment specially directed to that part of the body. On the 2nd of January he took cold, and after a fortnight's catarrh was seized with pain in his knees and chest, which continued till his admission on Saturday, February 3rd.

He was a fairly nourished young man of dark complexion. The joints were not much swollen, but there was pain in the

chest and left shoulder. He was seen on admission by Mr. Wood, who detected pericarditis, and ordered a blister over the heart, twenty grains of bicarbonate of potash three times a-day, and mercury with chalk and Dover's powder, of each two grains and a half night and morning. The next day (Feb. 4) a double friction sound was heard under the sternum, and a systolic murmur at the base of the heart. I saw him on Monday, Feb. 5; the pericardial friction was audible enough, but I was informed that it was less distinct than on the 4th. The pulse was 116, and there was a little crepitation in the right lung behind. He was ordered to take a grain of opium and two grains of camphor three times a-day, and to continue the potash draught. The following day the friction had ceased, nor was it ever heard again. The endocardial murmur continued nearly a fortnight; the pleuro-pneumonia on the contrary rapidly increased, extending to both lungs, and the sputa became thick, greenish, and tinged with blood, while the cough was accompanied with much pain; the pulse was 120, and the skin hot and dry. This was so serious a complication, implicating both lungs, that I feared to rely on the opium alone in large doses, nor did I care to do so, as the pericarditis had already ceased. I therefore adopted the more usual treatment of calomel one grain, and opium a quarter of a grain, every six hours, and acetate of ammonia with ipecacuan wine, and applied mustard daily to the back. The mercury and opium were continued for above a fortnight, when the gums became tender, and the pill was only given at bedtime for a fortnight longer. The pleuro-pneumonia meanwhile advanced to the stage of consolidation in both lungs, with effusion on the right side; but as soon as the gums became tender the fluid began to be absorbed, and the lungs to recover their permeability to air. On leaving, April 16, about ten weeks after admission, the heart's action was slightly increased, the endocardial murmur which existed on his admission had ceased, and the pulmonary sounds were quite natural, except that there was a little dulness on the right side from pleural adhesion.

I should not have given this case had I not undertaken to relate all the cases of pericarditis that have come under my care in St. Bartholomew's Hospital in the last three years; for though the pericarditis was the first complication, it ceased early, and the case became one of simple pleuro-pneumonia. I do not think that opium alone would have succeeded in this case, and it was one which I think would have justified a moderate abstraction of blood. Neither do I think that in this case the opium cured the pericarditis. The pericarditis had probably existed several days before his admission, and adhesion had al-

ready commenced, and was completed the day after he began the opium.

In estimating the degree of recovery in these cases I must consider the amount and importance of the injury remaining or likely to follow. The worst injury is, I conceive, inflammation of the muscular substance of the heart. Hollow muscles and muscular tubes after inflammation lose in some degree their power of contracting, and if continually acted upon by pressure from within dilate. In none of these cases, except IV. and X., and perhaps VI., were there any symptoms of myocarditis, such as irregular action of the heart or duplication of one or both beats, or great faintness. Possibly there was some myocarditis in other cases when the pericarditis lingered long, as in Case V. General adhesion of the pericardium, however, such as occurred in Case IV., is scarcely a less evil. It may not cause immediate inconvenience; but had it occurred in the case of a labouring man, it would, by confining and impeding the heart's action, have rendered it incapable of sustaining any extraordinary effort. It must be remembered too, as Dr. Latham has pointed out, that a heart which has once inflamed in rheumatism will, being unsound, often inflame again, even without any rheumatism of the joints. These remarks, however, do not apply to partial adhesions such as occurred in Cases II., III., VI., and VII. Such adhesions are met with after death in persons who have felt little or no inconvenience from them and who were quite unconscious of their existence, and are consequently of scarcely more importance than the partial adhesions of pleurisy.

I offer these remarks with great deference, feeling that many more cases are required to substantiate the value of the treatment I propose, but believing that at all events I have been guided by a sound principle.

Article XIX.—*Surgical Cases, Devon and Exeter Hospital.* By Philip Chilwell Delagarde.

WHEN I delivered the 'Address in Surgery' before the British Medical Association in 1860, I gave a large, although somewhat unconnected series of surgical cases—all of which, however, had been under my own observation. My object was to throw light on some doubtful points of operative surgery. This address I printed for private distribution. My

present contribution comprises several operations which I have since performed in the Devon and Exeter Hospital.

As far as I know, they all possess novelty, but that was not the motive. Every surgeon who operates should indeed be acquainted with the text-books of surgery; but the surgeon of a great hospital would hardly render justice to his position if he had not sufficient self-reliance so to alter old operations, or devise new ones, as to meet each varying contingency of disease or accident. I dealt with difficulties as they occurred, and I desire to show how I accomplished my purpose.

I add a case of lock-jaw. I have now cured five severe cases of traumatic tetanus, and it seems desirable that I should show, in a detailed report of at least one case, by what means I have succeeded.

I.—*Restoration of the upper eye-lid after a burn.*

Elizabeth Boynes, æt. 9, came to the hospital from the union-house, Newton Abbot. The upper eye-lid of the right eye had been nearly obliterated by a burn in infancy. The tarsal cartilage, with its lashes, was preserved, but all between it and the eyebrow was destroyed. At the outer third it was firmly attached—looped up as it were angularly—to the superciliary process of the frontal bone. From this angle projected a large fold of conjunctiva, which protected the eye in some degree—for the eye could be rolled under it, and thus wiped—but the lids would not approach each other at any point. Plate I. fig. 2.

Sept. 3, 1861.—First, the band connecting the tarsus to the superciliary ridge was divided. Then the tarsus was carefully separated from the eye-brow by an incision carried between them. The expanded tendon of the levator palpebræ superioris was exposed and freed from its adhesions to the skin and cicatrix. The skin on each side of the incision was raised, down to the tarsus, and up to and under the eye-brow. Then a large willow-leaf shaped piece of skin was dissected from the temple, turned forward into the incision between the tarsal cartilage and eye-brow, and secured by several silver-wire sutures. Plate I. fig. 1.

A perfect union took place. The eye-lid assumed almost a natural appearance, and could be nearly closed through its whole breadth. The conjunctival fold shrank up considerably, but I intended, if it did not disappear under the edge of the eye-lid, to snip it off, as it was the only hindrance to a perfect closure of the lids.

I heard no more of this child (although I had desired, when she returned to the Union, that she might be sent up again) for several years. She was then brought up from Torquay, where she was living in a school for training domestic servants. A hair, which originally belonged to her temple, but which I had transferred to her eye-lid, growing as she grew in age and stature, tickled her. My opinion was required as to what could be done for it. The skin on which it grew could not be spared, so I suggested that it should be plucked out with tweezers from time to time. The fold of conjunctiva had disappeared. She could open the eye thoroughly, and winked at will.

II.—*Restoration of the upper eye-lid after small-pox.*

William Bagwell, of Honiton, æt. 21. Eight years before I saw him he had had confluent small-pox. The cicatrizations were of the worst description, giving him the appearance of a fat and very coarse old man. The upper eye-lid of the left eye had almost disappeared, and the eye itself was destroyed by unceasing exposure and by ulceration. The right eye had a semi-opaque cornea, and was otherwise in a very bad state of chronic inflammation. The lids had not been closed, night or day, for eight years. He was almost blind.

April 16, 1862.—The same operation was performed as in the preceding case, Plate I. fig. 1; but the greater part of the flap taken from the temple was thin cicatrix. As much sub-cuticular tissue as could be found was taken out. With this object the incisions were oblique and divergent from without inwards, so as to make the base of the flap wider than the surface (Plate I. fig. 3); but under the cicatrix there really was no sub-cuticular tissue. The fibres of the orbicularis were carefully preserved. The flap was secured by interrupted sutures, and adhered; but after several days that part which was nearest the nose sloughed, not perhaps through its entire depth. It was a cicatrised spot that sloughed. In three weeks it healed. He could close the lids, and the inflammation began to subside; but afterwards, as the wound on the temple healed, the flap was dragged up. Even after that the lids could be closed, but only by a great effort. On the 1st of July I sent him home for a month—to refresh himself previous to a second operation which I contemplated.

August 3, I cut away the root of the flap from the cicatrix on the temple. Then I continued the incision across the eye-lid, above the flap, to the inner canthus. I introduced pins

Fig 1.

Fig 2.

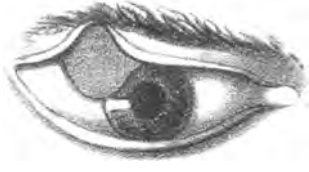


Fig 5.

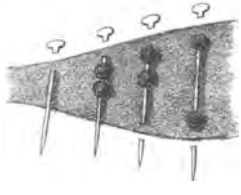
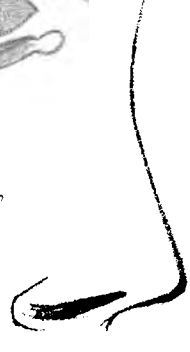


Fig 3.

Fig 4.



with perforated shots on them (see Plate I. fig. 4) and kept the edges wide apart until the wound was healed. Plate I. fig. 5 shows the successive steps in fixing each pin. This required unceasing attention, for I had to shift the pins to new places every three or four days to prevent their ulcerating out. The wound healed in a month, when I could no longer use the pins. But I had the lid firmly bandaged down instead, and directed him to close his eye, with as much force as he could exert, continually. Thus the cicatrix was prevented from contracting; and by constant and energetic exercise of the orbicularis he squeezed out a narrow rim of conjunctiva, which supplemented the tarsus, and enabled him to close the lids without any unusual effort. I kept him in the hospital, where he made himself useful, until January 10, 1863. In April, finding there was no disposition to contract, I discharged him, cured.

He wears a pad on his left eye; but, if that should ever trouble him, I have this operation in store. I will cut away the tarsal cartilage from either eye-lid close up to the punctum. Then I will make incisions, one on the upper and one on the lower lid, each in the direction of the fibres of the orbicularis, but I will divide the tendon of the levator palpebræ superioris. Then I will unite the cut edges, from which the cartilages have been removed, by interrupted sutures. They will be sound in three or four days, and, after that, the wounds, above and below, may heal at their leisure. Thus I will shut up his blind eye, and take care that it does not worry its working fellow.

During this year I have seen him frequently, for he has come into this neighbourhood to live. The upper lid has greatly improved. The rim of conjunctiva has become firm from exposure, and quite answers its purpose as supplementary to the skin. The cornea is perfectly clear, and Bagwell is a useful and powerful labourer on the railway.

III.—*Resection of the head of the Femur for unreduced dislocation into the Ischiatic notch.*

Robert Harford, of Tiverton, labourer, æt. 51, about five months before the operation had had the left lower extremity crushed and lacerated by the falling of a cob wall. The thigh bone was broken in two places, and its head dislocated into the sacro-ischiatic notch. The knee was badly cut, and the leg bruised and broken. Four months after the accident he was, on the 1st of August, 1861, brought to the Hospital. I found

the fractures well united, but of course no attempt had been made to reduce the dislocation.

The man's state was deplorable. The slightest attempt to move the thigh made the head of the femur press on the sciatic nerve, producing throughout the limb a most peculiar numbness, which was intolerable. He lay as straight as if a spit had been thrust from his heel to his head. His health was rapidly sinking, and I determined on removing the head of the thighbone.

The head of the femur was lodged so deeply in the upper part of the notch, and the neck lay so buried in the soft parts, that I foresaw I should not be able to saw it off, and prepared accordingly.

Sept. 1.—I made a deep incision from the trochanter to the sacrum through the glutæus, and laid bare the neck and head of the femur. Upon these an adventitious capsule had been formed. The ligamentum teres was strained, but not broken. The pyriformis tendon passed under the neck with the obturator tendons. I had the limb rolled inward, and carefully passed an elevator under the neck of the femur, so as to lift the head off from the sciatic nerve. The semilunar limb of a very strong bone-cutter was passed under the neck, near the trochanter, but the bone was so hard that not the slightest impression could be made. It was withdrawn, and I tried a large trephine. The pivot could hardly be made to pierce the bone, but at length I got the teeth to bear, and with great labour took out a circular die. I gouged out the cancellated structure until I reached the opposite wall. Then I introduced a smaller trephine, and worked until I supposed I had almost got through. I passed a strong, narrow-bladed saw into the cavity, and sawed as well as I could the bone remaining between the two circles. Plate II. fig. 1. I fixed a very strong pair of bone forceps, first in one groove and then in the other, and thus cut off the head of the bone. The splintered portions were nipped or filed down. The sciatic nerve was laid bare for an inch and a half. It was curiously flattened, and moulded to the head of the femur. This operation took three-quarters of an hour.

The relief was immediate. The wound healed in six weeks. He could then sit up in bed with the thigh at a right angle with the body. Gentle exercise on parallel bars soon gave great freedom of motion. He was discharged on the 16th of January, 1862, with a serviceable limb, and in good health.

He died in the summer of 1864, of pneumonia, having slept in the rain one Saturday night, instead of going home as a sober man would.

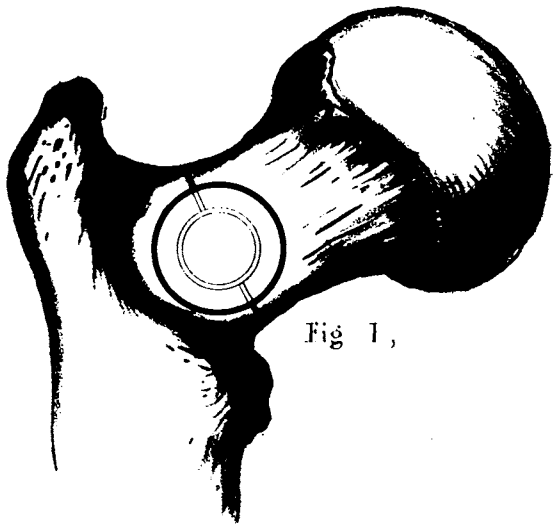


Fig 1,

Fig 2,

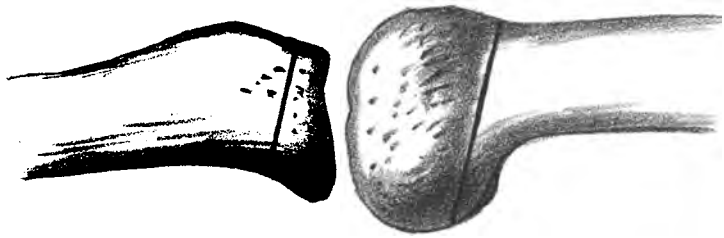
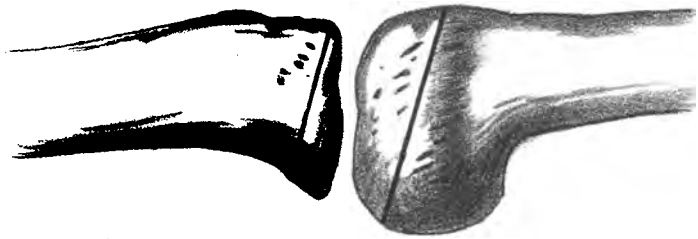


Fig 3,



IV.—*Note on figures 2 and 3, Plate II.*

Every one familiar with resection of the knee is aware of the tendency of the tibia to slide up behind the femur. This I prevent by taking the ends of those bones off obliquely —a simple expedient, not much known, but worth knowing. I studiously preserve the hinder parts of the condyles for the protection of the popliteal nerve and vessels, and for the extension of the foot.

V.—*Removal of the breast and extensively diseased skin.*

In removing the female breast, the disease cannot always be included in the ordinary elliptical incisions. In such cases I try to remove the contaminated parts, and yet retain integument enough to cover up the wound. The two following cases are examples. I had, however, another, and not less important object. I had to forbid the hope of absolute cure; but I told the poor creatures that life might be prolonged, and that when the disease did return, it might bring them to their end with far less distress than if allowed to take its course. This seems in course of fulfilment.

Jane Thorn, æt. 58, had long-neglected scirrhus of the right breast. Immediately above the nipple was a hard heavy tumour, the size and shape of an orange. A tubercle as large as a hazel-nut stood forward, distending the skin. This skin, on and above the tubercle, was a disk, broad and conspicuous, white, glossy, hard, and thickened; presenting a defined edge quite perceptible to sight and touch. The rest of the skin was healthy, and the gland was freely moveable on the pectoral. There was no disease in the axilla, or above the clavicle.

Dec. 26, 1860.—As the breast was large and pendulous, I thought I could remove all the diseased parts, yet close up the wound. I made a V shaped incision from the sides of the nipple to the bottom of the breast; then laterally from the sides of the V, right and left, and then semicircularly from the transverse incisions, so as to include the tumour and all the diseased skin. The whole was then cleared out down to the pectoral. Plate III. fig. 1.

The V was brought together by twisted sutures. The skin from the lower part of the breast was then lifted up, and attached by wire sutures to the skin below the clavicle, filling

up the semicircle. The whole was healed by Feb. 7, 1861. The tumour proved decidedly cancerous, and the skin above the nipple divided like the rind of pork.

She remained quite well until January 1865, when she had neuralgic pains of the right crural nerve. She went into the hospital again on the 15th of June. Three weeks afterwards a tumour suddenly protruded from the middle of the femur. In a few days the bone separated into two parts and the pains ceased. In a month the tumour occupied the thigh, from its middle to the groin. The thigh was bent at right angles, and is now so fixed as to convey the impression of reunion. The tumour is apparently intruding into the abdomen. She is thinner, but looks healthy and fresh. There is little pain. Although she is no longer under my own charge, I have had sufficient opportunity of watching the case.

VI.—*Removal of the breast, diseased skin, and axillary glands.*

Eliza Wright, of Burlescombe, æt. 48, had had a scirrhus of the breast for two years. Menstruation had ceased. There was a hard knob, the size of a small orange, above the nipple. The skin as well as the nipple adhered to the whole tumour. It was livid, and about to ulcerate. There were two scirrhous glands midway between the breast and armpit. She had a palpitating heart, and I would not venture on chloroform.

Nov. 22, 1861.—First, I removed the two scirrhous glands. Then I made an incision across the breast, below the nipple and areola. I dissected the skin from the lower portion of the mammary gland, which was broad, flat, and thin. The gland was then detached from the pectoral muscle, from below upwards, as high as it could be conveniently. I then made two incisions from a point at the upper part of the breast; they diverged, and when prolonged to the lower incision formed a triangle, including the nipple and diseased skin. Plate III. fig. 2. The two flaps were dissected off from the upper part of the mammary gland. The gland, the tumour, and the diseased skin were then separated from the parts beneath. Thus I removed the whole breast.

The two flaps were brought together by twisted sutures. The flaps thus united were then attached to the skin below by interrupted sutures, and the wound was covered.

She remained quite well until Michaelmas 1865. I saw her in December. A knob, hard, discoloured, and immovable, had risen up behind the clavicle. Although as large as a walnut,

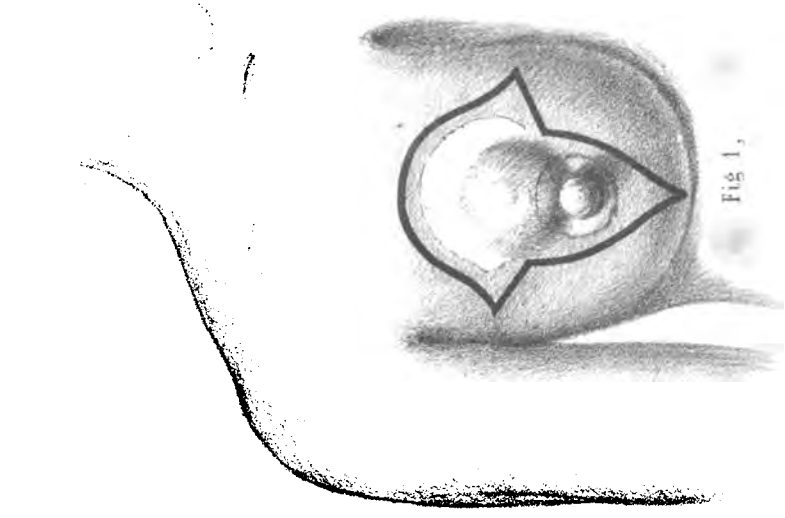


Fig 1.

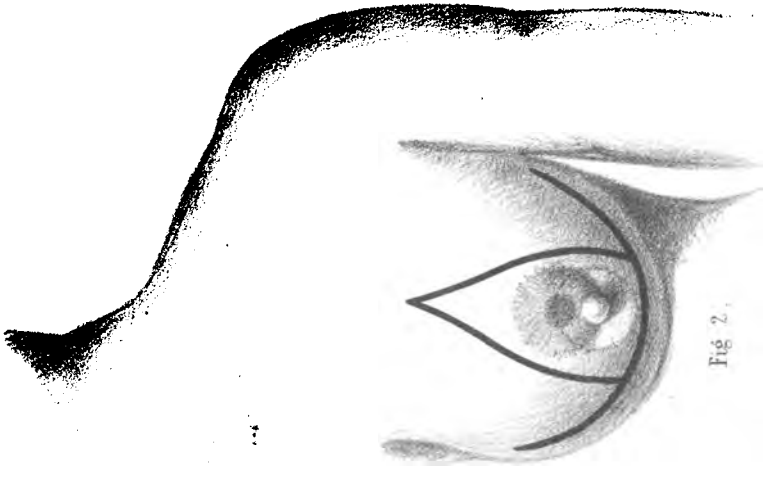


Fig 2.

W. Spreat Lith. Exeter

it was neither painful nor inconvenient. She looked and felt in good health. No enlarged glands in the axilla.

VII.—*Case of Traumatic Tetanus.*

Ellen Venus, of Lympstone, æt. 15, a remarkably fine girl, a brunette of the Celtic type so prevalent in the South of Devon and in Cornwall, fell under a waggon, the wheels passing over both thighs. The skin was crushed and torn just above the knees. In the right all the subjacent tissues, from the patella to the inner condyle, were destroyed down to the capsule of the joint. The synovial membrane by the side of and above the patella, although not penetrated, was bared to the size of a half crown. She was brought to the hospital at once, Aug. 21, 1865. As the wounds were full of gravel and dirt, bread poultices were applied, with fomentations. After a few days, when the sloughs began to separate, and granulations to form, she was put on the ordinary house diet, with first half a pint, and then a pint of good strong ale daily. As the discharge became copious, four ounces of port wine were added.

The left thigh had nearly healed, and the wound of the right was full of healthy granulations, when, seventeen days after the accident, she spoke of slight starting. She had had some general stiffness, which was ascribed to a cold. I had not heard of these symptoms, which were in some measure disguised by her high spirit. On the 11th of September, I was called early; she had tetanus. I had been taken off my guard; for of the multitude of cases I had seen, none had occurred earlier than the fourth, nor later than the eleventh day. Her countenance was anxious, the muscles of her face tense, her speech impeded, and the mouth distorted by the characteristic grin. A general rigidity was perceptible. The tongue was clean and moist; the skin disposed to perspiration. The pulse 112 and small.

I ordered an enema of gruel, with castor oil and turpentine, which procured a sufficient evacuation. Five grains of quinine, with a little sulphuric acid, every second hour; after five doses had been taken, every third hour. I directed extract of belladonna and mercurial ointment, in equal parts, to be rubbed along the course of the crural nerve.

Sept. 12.—She was comforted by the quinine, but disliked the ointment. I had used it in my first successful case, but had no great confidence in it, so it was given up. The pulse was upwards of 100, and the skin relaxed. Her neck was strongly arched back, burying the occiput in the pillow. The

back stiff, and the abdomen hard as a board. She evidently suffered a great deal, but she was full of courage and made no complaint. Her cheeks were creased by spasm, and the expression of anguish was intensified by her fierce dark eyes. To take 12 ounces of port wine daily. Amongst other food she sucks in finely minced meat between her teeth. Quinine mixture every four hours. In the night she had slight diarrhœa.

13.—The symptoms much as yesterday. Perhaps there was less spasm. She thought herself better. Pulse 110. Remedies to be continued.

The next three days the arching of the neck decreased, and the jaws were less stiff; but the thigh jerked without ceasing, preventing sleep. It had to be continually held down. Skin moist, tongue clean, pulse 115, appetite good. She had lost flesh, but was less distressed. The bowels were on the whole loose, but actual purging was controlled by opiate injections. At such times brandy was given instead of wine. The quinine was given without acid every six hours; an extra dose allowed should the spasms be worse. I directed a chloroform liniment to be frequently sprinkled on the thigh; it relieved the jerks, and she slept.

During the next week the rigidness of the neck ceased, and the constraint of the jaws diminished, though her speech was still affected. The thigh started during sleep, but she could sit up in bed comfortably. Pulse 115. Bowels regular. Quinine as before, three times a day, but with the addition of one grain of sulphate of iron in each dose, as it was time she should menstruate. She was much thinner, but the appetite kept up. Slept well, except when disturbed by starting of the limbs.

28.—The improvement continued, the spasms having almost ceased. The wound had nearly healed. The quinine and iron twice a day, and the chloroform liniment now and then. On the 30th she was well as regarded the tetanus, and the medicine was reduced to once a day. But she was still haggard, and the pulse would not go below 115. I gave up medicine, and reduced the quantity of wine. I was puzzled; something was wrong, and I could not tell what. A fortnight afterwards, a large but painless abscess was discovered between the flexors above the popliteal space. It broke, and a great quantity of pus was discharged. It soon healed.

As I had seen a patient die at very short notice after an apparent cure of tetanus, I kept this girl in the hospital until the 14th of December. She was then made an out-patient, but I would not discharge her as cured until the 1st of March, 1866.

She remains quite well, strong, active, and in good spirits; but while her aspect is that of an energetic, it is that of a care-worn woman, although not seventeen.

ARTICLE XX.—*Case of Profuse Venous Hæmorrhage from the left meatus auditorius externus, consequent on a fall upon the back of the head.* By LUTHER HOLDEN.

HÆMORRHAGE from the ear, after an injury to the head, is not of uncommon occurrence. The following instance is, however, in many respects so remarkable, that it ought to be recorded. As far as I know it is without precedent.

On the evening of October 28, 1865, John Reid, æt. 35, a painter by occupation, while staggering homewards intoxicated, fell and struck the back of his head with considerable force against the bottom of a lamp-post. He was picked up immediately insensible, bleeding profusely from the left ear, and carried to the house of a surgeon close at hand, who very judiciously plugged the ear with cotton wool; and, after instructing the attendants to press firmly upon the plug with the finger, sent the man to St. Bartholomew's Hospital.

Mr. V. F. Eck, the house-surgeon on duty, with several of his dressers, bear witness to the man's condition on his arrival at the hospital, about fifteen minutes after the accident. His clothes, on the left side, were saturated with blood. He was insensible and pulseless. The plug was still held firmly in the ear. After a careful examination of the head, the house-surgeon could detect no external injury. He was about to remove the plug from the ear, when one of the bystanders implored him not to do so, exclaiming 'he was sure his friend would bleed to death.' However, the plug was removed. To use Mr. Eck's words, 'its removal was followed by the most profuse venous hæmorrhage from the ear, filling the whole calibre of the meatus, and flowing as a stream would pour from the mouth of a full teapot. The blood was extremely dark in colour, not to say black. I instantly re-applied a plug in the meatus, and placed a compress and tight bandage over the ear. In about half an hour afterwards I could detect a feeble flickering pulse at the wrist, and the extremities became warmer. With this improvement in the circulation, there was some return of consciousness. By and by he began to talk incoherently, like a drunken man. Soon afterwards he fell asleep, and slept quietly through the night.'

On the following morning (Oct. 29) he was quite himself, and sat up in bed. Very little seemed amiss beyond a blanched countenance, a feeble pulse, beating forty-eight times in a minute; a slight frontal head-ache, and great thirst.

On the third morning he expressed himself as 'quite well.'

Nothing worthy of note occurred until the morning of the twelfth day, when, on the removal of the plug, about half an ounce of bloody fluid spurted out over the bed-clothes.

On the thirteenth day he complained of head-ache and thirst. His skin was hot and dry, and his tongue furred. These feverish symptoms subsided in a few days under appropriate treatment.

On the twenty-fourth day he appeared in all respects well. The plug was carefully removed. There was no hæmorrhage. He had lost all hearing in the left ear.

After remaining in the hospital, under close observation, until Dec. 31, *i.e.* sixty-four days after the accident, he was discharged, with strict injunctions as to temperance, and the necessity of reporting himself from time to time.

This man has been under our observation on several occasions since he left the hospital; and we have every reason to believe that, up to the present time, ten months since the injury, he continues well, and actively employed.

The question naturally arises—what was the source of the hæmorrhage? The most rational explanation would appear to be, that there was a fracture of the mastoid process of the temporal bone, and a laceration of the lateral sinus. The blood would flow through the mastoid cells into the tympanum, and thence escape through the lacerated membrana tympani.

ARTICLE XXI.—*Case of Aneurism of the lower part of the Common Iliac, of the External Iliac and Femoral Arteries, successfully treated by Pressure of the Abdominal Aorta.* By VINCENT F. ECK.

JOHN CUSACK, aged 40, a thin, spare, and ill-nourished man, was admitted into St. Bartholomew's Hospital on April 17, 1866, under the care of Mr. Holden, suffering from a pulsating tumour of the abdomen.

He gave the following history. He was a night-watchman at a railway-station, formerly a constable in the Irish police. Had been temperate, and always enjoyed excellent health

up to within the last two years and a half. In the early part of 1864 he felt obscure pains across the loins, which prevented him stooping, turning his body round, or walking fast. He applied to Mercy Hospital, Cork, where it was first noticed that the right foot and lower part of the leg were slightly swollen and red. After a rest of three weeks he was discharged relieved, all symptoms subsiding. He returned to his work, and after a few days the limb began to swell again, the swelling this time extending half way up the thigh. He continued his work, however, for a few days longer, when one night, while on duty, he was about to 'shove' open a gate, and finding the hinge out of order, he put his shoulder to the gate and exerted himself violently to push it open; his foot slipped backwards on the muddy soil, and he felt at once a 'beating' in the groin. His groin felt very stiff, the beating continued, and in a few days the leg and thigh swelled up 'enormously.' Shortly afterwards the veins on the upper and outer aspect of the thigh became exceedingly varicose and tortuous, and the skin gave way just above the inner ankle, leaving an ulcer about the size of a five-shilling piece. In this condition he was admitted into Tralee Infirmary, whence, after a rest of five weeks, he went to Dublin, and became a patient in Dr. Steevens's Hospital. He was confined to bed, absolute rest forming the leading feature of the treatment. After five months he was discharged, his condition but little improved.

He returned to Tralee, and on September 10, 1864, he entered the Union Infirmary, where he remained eight months. For six weeks of this period pressure was kept up on the common iliac artery for an hour daily, by means of bags of shot. At the same time he was kept on a low scale of diet, consisting of meat three ounces, bread seven ounces, butter one ounce, milk four ounces, water four ounces. He was greatly reduced by this restricted diet, but all his symptoms were improved, the limb being diminished in size, and the impulse of the tumour becoming somewhat less. He left for Dublin, and entered St. Vincent's Hospital June 1, 1865, and remained there four months. During this time compression of the vessel above and below the tumour was employed twice—on the first occasion by means of a tourniquet over the common iliac, and by another tourniquet over the femoral artery, at about the junction of the upper with the middle third of the thigh. Pressure was kept up in this manner for three hours, with occasional intervals of rest. Two weeks subsequently pressure was again applied as before, for a period of two hours. No decrease in the impulse of the tumour followed this plan of treatment,

but the swelling of the leg and thigh was greatly diminished, and the ulcer of the leg healed. His general condition and health were greatly improved. In the beginning of October 1865 he again entered the infirmary at Tralee, and after a rest of six months he determined to come to London.

On examination, a large fusiform swelling was detected in the right iliac fossa, extending from just above the situation of the division of the common iliac, running downwards in the direction of the external iliac artery, passing under Poupart's ligament, and occupying about three inches of the upper part of the thigh.

The tumour pulsated strongly, and gave a heavy, heaving, and distensile impulse to the hand. This pulsation was arrested by firm pressure on the common iliac artery, and returned at once on remitting the pressure. The stethoscope revealed a loud, whizzing bruit, synchronous with the impulse of the heart. The whole limb of the affected side was considerably enlarged; the foot and lower part of the leg were livid, and lower in temperature than the other limb by 2° Fahr. Sensation was considerably diminished on the diseased side. A large, varicose, and tortuous condition of the superficial veins was observed on the upper and outer part of the right thigh. The greatest breadth of the tumour, which was about 3 inches above the crural arch, measured 3 inches; as it passed under the ligament its breadth was reduced to 2 inches; from this point it gradually tapered downwards till it was lost in the healthy artery of the thigh, 3 inches below the arch. The circumference of the uppermost part of the thigh measured 25 inches, that of the sound side a little under 19.

He complained of great pain and stiffness in the limb, and was quite unable to walk without the aid of a crutch. The 'beating in the groin' troubled him greatly, and prevented him sleeping. He was subject to occasional giddiness and severe pain over the præcordial region.

Examination of the heart indicated considerable hypertrophy, with obstructive and regurgitant disease of the aortic valves. The pulse of aortic regurgitation was well marked.

His general condition was that of extreme emaciation and ill-health, resulting from prolonged suffering, and aggravated no doubt by constant sickness and want of sleep during a very rough sea passage from Cork to London.

April 17.—He was directed to keep his bed, and ordered a liberal diet. In two weeks he was so far improved in health and condition that it was deemed advisable to try again the effect of pressure high up on the common iliac artery. On

May 3, as a preliminary treatment, his bowels were well cleared out with castor oil, and early on the morning of May 5 an injection was administered so as thoroughly to empty the large intestine.

May 5.—At 9·30 A.M. pressure was applied by Mr. Holden by means of Lister's tourniquet over the bifurcation of the aorta, so as to control only the passage of blood through the right common iliac, leaving the left as far as possible free. All pulsation in the tumour was arrested. At 10 A.M. the temperature was taken, and the right foot found to be 75°, the left 79° Fahr. Up to this time he bore the pressure well. At 10·5 he complained of pain at the seat of pressure and across the epigastrium, and was sweating on the forehead and face. Pulse 64, regular, but feeble. Countenance pale. At 10·15 the pressure was slightly relaxed. The pulse improved in strength and volume, and rose to 74 beats in the minute. At 10·30 he became very restless, was sweating profusely, and asked for chloroform to be administered. He was accordingly put under its influence, without any unfavourable symptoms resulting. The tourniquet was screwed up a little tighter, and he went on well for the next hour, being kept just within the influence of the anæsthetic. Shortly before 12, noon, his breathing became embarrassed, his pulse intermitted, and for a few seconds was not to be felt at the wrist. The chloroform being now suspended, he rallied at once, and recovered from the influence of the anæsthetic. At 12·15 he was suffering a great deal from the pressure, and begged for a continuance of the vapour, which was administered very cautiously from time to time, and suspended for good at 1·30 P.M. He was becoming very faint, with hardly any pulse, and it was thought advisable to remove the pressure by slow degrees.

Faint pulsation was then detected in the tumour, with very feeble impulse. At 2 P.M. the impulse had increased, at 3 P.M. more so, but over a much less extended space than previously to the operation. His expression was now haggard, and indicative of great exhaustion, countenance pale, surface clammy. Pulse 76, regular, but very feeble. Respiration natural. He complained of nausea and giddiness in the head. Had no pain in the abdomen, but the skin felt sore where the pressure was applied. Ordered a little brandy and water, and ice-bag to the tumour.

At 8 P.M. he had regained his usual expression, and appeared at ease. Surface warm and moist. Pulse 84, regular, with more power. He had been sick two or three times, but was

now quite comfortable, save feeling rather faint. Ordered one sixth of a grain of acetate of morphia.

May 6.—Slept for about two hours during the night, and appeared a little fatigued, but otherwise his general condition was satisfactory. Pulse 80, regular, of moderate volume. Tumour pulsating much less strongly than before the operation.

On the following day, May 7, he was progressing very satisfactorily, and all constitutional irritation had ceased. He had slept well after taking one-sixth of a grain of morphia. Had no pain in the abdomen, and there was no extension of impulse in the tumour.

From this date he has progressed uniformly well. The tumour seems to be consolidating, and is less in all its measurements by more than one half than when noted on the day of his admission. Pulsation, which was plainly visible through the walls of the abdomen, is greatly diminished, and can only be felt by making deep pressure with the finger in the iliac fossa. The area over which the impulse can be detected is considerably less than that noted one hour and a half after pressure was suspended on the day of operation. The leg has been measured from time to time, and (August 20) is twenty-one inches in circumference at the uppermost part of the thigh, the unaffected side measuring nineteen and a half inches. The varicose condition of the superficial veins is greatly diminished, and the leg is nearly normal. The whole limb is perfectly limber and supple.

Three weeks after the operation he was allowed to sit up in the ward, and in another week he went out into the Hospital Square, and no increase in the impulse observed in the tumour followed this exertion. In a few days more he was able to walk about the square for half an hour at a time, with the aid of a stick.

He now looks in good health and condition, and is no longer troubled with the beating in the groin. He walks about the Hospital Square best part of every day without aid or support, and without fatigue, or any injurious effect upon the tumour.

Article XXII.—*Medical and Surgical Landmarks.*

By Luther Holden.

'Chirurgus mente prius et oculis agat quam manu armata.'

In going round the wards of the Hospital I often have occasion to point out, on the surface of the living body, any landmark—such as a line, an eminence, a depression—which is a guide to, or an indication of, deeper-seated parts; and I find this practice all the more useful, because many, even advanced students of anatomy, are not so ready as they ought to be in their recognition of parts when covered by skin. They are familiar enough with the bones, muscles, and blood-vessels displayed in the dissected subject, but are often at fault when they come to put this knowledge into practice. For instance, ask a student to point out the exact place where he would feel for the head of the radius, the coracoid process of the scapula, the tubercle of the scaphoid bone in the foot; ask him to compress effectually one of the main arteries, to show the line of its course, to place the stethoscope over the precise position of the several valves at the base of the heart, to trace along the walls of the chest the outline of the pleura, to demonstrate the bony prominences about the joints, and their relative positions in the different motions of the joints; test him about the muscles and tendons which can be seen or felt on the surface as they stand out in relief or remain in repose; let him introduce his finger into the several orifices of the body, and say what parts are accessible to the touch: questions such as these, even a good anatomist, unaccustomed to deal with the living subject, might possibly find himself at a loss to answer.

The following observations are not made with the idea of exhausting the subject, but rather to induce in students the habit of looking at the living body with anatomical eyes, and with eyes too at their fingers' ends; for in expressing the importance of a perfect knowledge of anatomy to the operating surgeon, it may certainly be said that the various structures of the body should be pictured in his mind as they lie naturally grouped and connected together, and that he should see them with the same clearness and accuracy as if the whole substance of the body were perfectly transparent.

I would also invite teachers of anatomy to follow the example of Sir C. Bell, who was in the habit of introducing, from time

to time, a powerful muscular fellow to his class, 'in order to show them how much of the structure of the body, such as the articulations and the muscles, might be learned without actual dissection.'*

The amount of anatomy which is obvious to the eye and the finger depends in great measure upon the quantity of subcutaneous fat. In some the anatomy stands out beautifully clear; in others it is masked by corpulency. Selecting, therefore, for study a moderately lean person, let us begin with—

The Head.

The great density of the scalp, more especially at the back of the head, is owing to its intimate connection with the cranial aponeurosis, the scalp vessels and hair bulbs intervening. This density often obscures the diagnosis of tumours on the cranium, and creates a difficulty in discriminating whether they arise from within or without the skull-cap. Tumours, the removal of which was undertaken in the idea that they were only encysted, and could be easily taken out, have been found to originate within the cranium. The density of the scalp gives rise, in certain cases of blood effused beneath it, to that peculiar feel so exactly resembling fracture with depression of bone, that, in a first examination, a surgeon is almost sure to be deceived, notwithstanding any previous caution.

The pulsation of the supra-orbital artery can be felt just above the supra-orbital notch; of the temporal, about one inch and a quarter behind the external angular process of the frontal bone; of the occipital, near the middle of a line drawn from the occipital protuberance to the mastoid process; of the posterior auricular, behind the apex of the mastoid process. All of them can be effectually compressed against the subjacent bone.

The skull-cap is rarely symmetrical. This want of symmetry is often obvious. It may occur in men highly gifted, as in the celebrated French anatomist Bichat. As to shape and relative dimensions, no two heads are exactly alike, any more than two faces. It is beside my present purpose to go into the question of craniology more than to say that, although the cranium does not follow the brain in all its eminences and depressions so as to be, as it were, a cast of its surface, yet it certainly indicates the impressions of the great cerebral masses. The frontal and parietal eminences and the occipital region

* Some pertinent remarks on this subject have been made by Mr. C. Heath in a pamphlet 'On Anatomy in relation to Physic.'

may be taken as a general expression of the development of the corresponding lobes of the brain. The breadth and height of the frontal region is a measure of the intellectual capacity; the breadth and height of the parietal region measures the development of the moral feelings; the occipital region measures the animal propensities. To ascertain the relative proportions of these three regions, let a thread be passed from one meatus auditorius to the other, across the front, the middle, and the back of the head.

The bony prominences on the forehead, formed by the separation of the two tables of the skull, and termed the 'frontal sinuses,' may vary much in size in different persons and at different periods of life. This has an important bearing on wounds of the forehead and on trephining in this situation. These 'bumps' are not prominent in children, because the tables of the skull do not begin to separate before puberty. From an examination of many skulls, I find that the absence of the external prominence, even in middle age, does not necessarily imply the absence of the sinus itself, since it may be formed by a retrocession of the inner wall of the skull. In old persons, as a rule, when the sinuses enlarge, it is the inner table which encroaches on the brain case. The skull wall here follows the shrinking brain. It is, therefore, important to bear in mind that an adult, and more especially an elderly person, may have a considerable frontal sinus without any external indication of it.

The same observations apply to the air cells of the mastoid process, which can be felt behind the ear.

The occipital protuberance can be distinctly felt at the back of the head. This is always the thickest part of the skull-cap, obviously for the purpose of protection.

A line drawn over the head from the root of the nose to the occipital protuberance corresponds with the superior longitudinal sinus. Another line drawn from the occipital protuberance to the front border of the mastoid process corresponds with the lateral sinus. The trunk of the middle meningeal artery runs along the anterior inferior angle of the parietal bone, about one inch and a half behind the external angular process of the frontal bone. The application of the trephine on any of these parts of the skull would be objectionable.

The average thickness of the cap of an adult skull is about $\frac{1}{8}$ of an inch. The thickest part is at the occipital protuberance, where it is often $\frac{3}{4}$ of an inch. Every one in the habit of making post mortem examinations knows how much the skull-cap differs in thickness in different persons, and in different

parts of the same skull. In the anatomical museum at Pavia is the skull of a child in which an angry cock had pecked a hole. In old persons it is often in some parts not thicker than a shilling, owing to absorption of the diplöe. Another point of interest is that the inner plane of the cap does not always correspond with the outer. Hence, in applying the trephine, this is not a bad rule—'Think you are operating on the thinnest skull ever seen, and thinner in one half of the circle than the other.'

The lower level of the anterior and middle lobes of the cerebrum corresponds with a line drawn from the external angular process of the frontal bone to the meatus auditorius. Another line drawn from the meatus to the occipital protuberance corresponds with the lower level of the posterior lobe. The lower level of the cerebellum cannot be defined by external examination. It depends upon the extent to which the occipital fossæ bulge into the nape of the neck; and this bulge varies in different skulls.

The Face.

The approaches to the organs of the senses, their ever varying expression, their numerous muscles, and their rich profusion of vessels and nerves, give the face great anatomical importance, which has a most valuable bearing, not only on the practice of surgery, but also on the physiognomy of health, and in the diagnosis of disease.

As a surgeon may be called upon to divide either of the three branches of the 5th nerve, he looks with interest to the precise situations where they leave their bony foramina. The supra-orbital notch or foramen can be felt about the junction of the inner with the middle third of the supra-orbital margin. From this point a perpendicular line drawn with a slight inclination outwards, so as to cross the interval between the two bicuspid teeth, passes over the infra-orbital and the mental foramina.

By pressing the thumb beneath the internal angular process of the frontal bone, the cartilaginous pulley of the superior oblique muscle can be distinctly felt. We should be careful not to interfere with this pulley in any operation about the orbit.

The working of the condyle of the jaw can be distinctly felt in front of the ear. When the mouth is opened wide, the condyle advances out of the glenoid cavity on to the eminentia articularis, and returns into its socket when the mouth is shut. The muscle which causes this advance is the external pterygoid; and the object of it is to give the jaw a greater freedom of lateral motion.

The posterior margin of the ramus of the lower jaw corresponds with a line drawn from the condyle to the angle. In opening abscesses in the parotid region, the knife should not be introduced behind this line for fear of wounding the carotid artery. Punctures to any depth may be safely made in front of it. They are often necessary where inflammation of the parotid gland ensues after eruptive fevers, and runs on to suppuration. The swelling, tension, and pain are most distressing. Owing to the fibrous framework of the gland, the matter is not in one sac, but diffused. One puncture is not enough. Three or more may be requisite. The blade of the knife should be held horizontally, so as to be less likely to injure the branches of the facial nerve. We are not to be disappointed if no matter flows. The punctures give relief, and matter will exude the next day.

A line drawn from the bottom of the lobe of the ear to midway between the nose and the upper lip gives the track of the parotid duct and of the branch of the portio dura which supplies the buccinator.

Between the root of the zygoma and the ear can be felt the pulsation of the trunk of the temporal artery. The facial artery can be distinctly felt as it passes over the body of the jaw at the anterior edge of the masseter, again near the corner of the mouth close to the mucous membrane, and, lastly, by the side of the ala nasi. By holding the lips between the finger and thumb the coronary arteries are felt under the mucous membrane. The facial vein does not accompany the tortuous artery, but runs a straight course from the inner angle of the eye to the front border of the masseter.

The opening between the eyelids varies in size in different persons; hence more of the eyeball is seen in some than others, and the eye appears larger. Although human eyes do vary a little in size, yet the actual difference is by no means so great as is generally believed. The size of the fissure has more to do with it. Contrast the narrow elliptical fissure of the Chinese and Mongolian races, and the apparent smallness of their eyes as compared with Europeans. As a rule the external angle of the lid is a trifle higher than the internal. When not exaggerated, it gives the face an arch and pleasing expression.

Evert the lids to see the Meibomian glands; observe their perpendicular arrangement, and that they lie in the substance of the lid cartilages.

The free borders of the lids are not bevelled, as described by J. L. Petit and most anatomists, 'so as to form with the globe of the closed eye a triangular canal for the flow of the

tears.' On the contrary, it is easily seen that the lid margins, when closed, come into accurate contact. Their plane is not exactly horizontal, but slightly inclined upwards.

Every time the eye is shut, the ball moves upwards and inwards, so that the cornea is completely covered by the upper lid. This may be well seen by raising the lid of a sleeping infant; and also in cases of low fever, when the lid is not completely closed. The reason of this upturning of the eye is obviously to clear the cornea, and to protect it from the light.

A careful examination of the motion of the lower lid in the act of shutting the eye proves that it is a double motion. The lid is not only slightly raised, but drawn inwards about $\frac{1}{12}$ of an inch. The object of this second movement is to draw any particles of dust towards the inner canthus.

The puncta lachrymalia are distinctly visible at the inner angles of the lids. The lower punctum is larger and a little more external than the upper, so that they may not be exactly opposite. The direction, too, of the puncta deserves notice. Their open mouths look a little backwards, ready to imbibe the tears. When, from any cause, their proper bearing is lost, as in facial paralysis or a cicatrix near the lid, the tears overflow the cheek. The length of the lachrymal canals is from three to four lines. The lower is a little shorter and wider than the upper. As they both make a little angle in their course, soon after their orifices, the lid should be drawn outwards to straighten it when we introduce a probe.

To find the lachrymal sac, feel for the inner edge of the orbit, and draw outwards the eye-lids to tighten the tendo oculi, which crosses the sac a little above its middle. A knife introduced just below the tendon close to the edge of the orbit would enter the sac. A probe directed in a line with the inner edge of the orbit, *i.e.* downwards, outwards, and backwards, would traverse the nasal duct, and appear in the inferior meatus of the nose.

The tendo oculi serves many purposes. One, often overlooked, is to pump the tears into the lachrymal sac. Place a finger on the tendon, and feel that it tightens every time the lids are closed The tendon, being intimately connected to the sac, draws, as it tightens, the sac wall outwards and forwards, and in this way pumps along the lachrymal canals any fluid collected at the angle of the eye.

The nasal duct is from six to eight lines long, and narrowest in the middle of its course. Its termination in the inferior meatus lies under the inferior spongy bone, about a quarter of an inch behind the bony edge of the nostril. An inspection of

the orifice in the dry bone conveys no idea of its size and shape in life;* for it is diminished by a valve-like fold of mucous membrane, so as to become, in most cases, a mere slit, not exceeding a line in diameter.

The facility with which instruments can be introduced into the nasal opening of the duct depends upon its position as well as its size. This position varies in different instances. Sometimes it opens directly into the roof of the inferior meatus, in which case the hole is large and round, so that tears readily drop into the nose. In other instances the opening is situated on the outer wall of the meatus, and is then always such a narrow fissure as to be hardly discernible. The practical conclusion then is, that a probe can be easily introduced when the opening is in the roof of the meatus, but not without difficulty and laceration of mucous membrane when on the outer wall. This difficulty indeed may be increased by the narrowness of the meatus, arising from an unusual curvature of the spongy bone.

The close connection of the skin to the cartilage of the nose admits of no stretching; hence the acute pain felt in erysipelas and boils on the nose. The external aperture varies in size and shape in different individuals. It is always placed a little lower than the floor of the nostril, so that the nose must be pulled up before we can inspect its cavities.

Looking into the nostrils, we find that the left is, in the majority of cases, narrower than the right, owing to the inclination of the septum towards the left. A communication sometimes exists between them, through a hole in the septum, as was the case in the celebrated anatomist Hildebrandt. By dilating the alæ nasi, we can get a view of the end of the inferior spongy bone. The middle spongy bone cannot be seen: its attachment to the ethmoid is high up, nearly opposite the tendo oculi. The cavities are so much narrowed transversely by the spongy bones, that in the extraction of polypi it is better to dilate the blades of the forceps perpendicularly, and near the septum.

What can be seen and felt through the mouth? The upper surface of the tongue, 'speculum primarum viarum,' is a study in itself. We notice, on its under surface, a median furrow, on each side of which stands out the ranine vein, lying upon the prominent fibres of the lingualis. On the floor of the mouth is the 'frenum linguæ,' with the orifices of the ducts of the submaxillary glands on each side of it. The glands themselves can be felt immediately beneath the mucous membrane.

* Alluded to by Morgagni, Adv. Anat. xli.

When the tongue is put out, the anterior fibres of the genio-hyoglossi can be felt in strong action. The previous division of their attachment behind the symphysis of the jaw would enable a surgeon to draw the tongue more freely out of the mouth in any attempt to remove carcinoma extending far back into its root.

There is great difference in the shape of the hard palate; this difference depends upon the depth of the alveolar processes. In some it forms a fine broad arch; in others it is contracted, and rises almost to a point like a Gothic arch, and materially influences the tone of the voice.

To examine the throat well, the nose should be held so as to compel inspiration through the mouth. Thus the soft palate will be raised, the palatine arches widened, and the tonsils and the back of the pharynx fairly exposed.

Put the finger into the mouth, and feel the anterior border of the coronoid process of the jaw. On the inner side of this process, between it and the tuberosity of the upper jaw, is a recess, where a deeply-seated temporal abscess is likely to burst, or might be opened. On the inner side of the last molar tooth of the upper jaw we can distinctly feel the hamular process of the sphenoid bone; also the lower part of the pterygoid fossa, and the internal pterygoid plate. Behind, and on the outer side of the last molar, can be felt part of the back of the antrum and of the external pterygoid plate.

When the mouth is wide open, the pterygo-maxillary ligament forms a prominent fold, readily seen and felt beneath the mucous membrane. A little below the attachment of this ligament to the lower jaw we can easily feel the gustatory nerve, as it runs close to the bone below the last molar tooth. The exact position of the nerve can be ascertained in one's own person by the acute pain on pressure. A division of the nerve, easily effected by a small incision in the right place, gives much temporary relief in cases of advanced carcinoma of the tongue.

To feed a patient in spasmodic closure of the jaw, it is well to know that there exists behind the last molar teeth a space sufficient for the passage of a small tube.

Lift up the upper lip and examine the front wall of the antrum. The proper place in which to tap it is above the second bicuspid tooth.

A surgeon's finger should be familiar with the feel of the posterior nares, and of all that is within reach behind the soft palate. This is important in relation to the attachment of polypi, and to plugging the nostrils. In the examination of

the back of the throat it is necessary to throw the head well back, because, in this position, nearly all the pharynx in front of the basilar process comes down below the level of the hard palate, and can be seen as well as felt. On the other hand, when the skull is at a right angle with the spine, the basilar process is concealed from view, and the hard palate is on a level with the margin of the foramen magnum.

The head, then, being well back, introduce the fore-finger behind the soft palate, and turn it up towards the base of the skull. We feel the strong grip of the superior constrictor. Hooking the finger well forwards, we can feel the contour of the posterior nares. Their size depends upon the anterior, but rarely exceeds a small inch in the long diameter, and a small half-inch in the short. Their plane is not perpendicular, but stands a little forwards. We can feel the septum formed by the vomer, and also the posterior end of the inferior spongy bone in each nostril.

Before taking leave of the throat, look well at the position of the tonsils between the anterior and posterior half arches of the palate. In a healthy state they should not project beyond the level of these arches. In all operations upon the tonsils, we should remember the close proximity of the internal carotid artery to their outer side. Nothing intervenes but the pharyngeal aponeurosis, and the superior constrictor of the pharynx. Hence the rule in operating on the tonsils always to keep the point of the knife inwards.

A word or two on the lines of the face as indicative of expression. Every one pays unconscious homage to the study of physiognomy, when, scanning the features of a stranger, he draws conclusions concerning his intelligence, disposition, and character. Without discussing how much physiognomy is really worth, there can be no doubt that it is a mistake to place it in the same category as phrenology, since the latter lacks that sound basis of physiology which no one can deny to the former.

The muscles of the features are generally described as arising from the bony fabric of the face, and as inserted into the nose, corners of the mouth, and the lips. But this gives a very inadequate idea of their true insertion. They drop fibres into the skin all along their course, so that there is hardly a point of the face which has not its little fibre to move it. The habitual recurrence of good or evil thoughts, the indulgence in particular modes of life, call into play corresponding sets of muscles which, by producing folds and wrinkles, speak a language all can understand, and which rarely misleads.

Schiller puts this well when he says that 'it is an admirable proof of infinite wisdom that what is noble and benevolent beautifies the human countenance; what is base and hateful imprints upon it a revolting expression.'

The Neck.

We must notice first the direction of the subcutaneous veins. The course of the external jugular corresponds with a line drawn from the angle of the jaw to the middle of the clavicle; it is, exceptionally, joined by a branch which runs over the clavicle, and is termed 'jugulo-cephalic.' The anterior jugular generally runs along the front border of the sterno-mastoid.

Immediately below and nearly on a level with the lower jaw we feel the body of the os-hyoides, and can trace backwards on each side the whole length of the cornua. They might easily be broken by the grasp of a garotter. Below the body of the os-hyoides is the gap above the thyroid cartilage. This gap corresponds with the apex of the epiglottis: so that in cases of cut throat in this situation, nearly the whole of the epiglottis lies above the wound. Between the thyroid notch and the bone there is a large bursa, to facilitate the play of the cartilage beneath the bone in deglutition. The notch in the thyroid cartilage, or 'pomum Adami,' varies in prominence in different persons. It does not appear till puberty, and is, throughout life, much less distinct in the female than the male. The finger can trace the upper borders and cornua of the thyroid cartilage, and its lower cornua can be felt by the side of the cricoid.

On each side of the thyroid cartilage we can recognise the lateral lobes of the thyroid gland. On the upper and front part of the gland we can distinctly feel the pulsation of the superior thyroid artery. This pulsation, coupled with the fact that the gland rises and falls with the larynx in deglutition, is the best distinction between a bronchocele and other tumours resembling it.

Below the angle of the thyroid cartilage we feel the interval between it and the cricoid, which is occupied by the crico-thyroid membrane. In laryngotomy we cut through this membrane transversely close to the upper edge of the cricoid cartilage, in order that the incision may be as distant as possible from the attachment of the vocal cords.

The projection of the cricoid cartilage is a point of great interest to the surgeon, because it is his chief guide in opening the air passages, and can always be felt even in infants, however

young or fat. It corresponds with the fifth cervical vertebra. The commencement of the œsophagus lies behind it: here, therefore, a foreign substance too large to be swallowed would probably lodge, and might be felt externally.

Those who have not directed their attention to the subject, are hardly aware what a little distance there is between the cricoid cartilage and the upper part of the sternum. In a person of the average height sitting with the neck in an easy position, the distance is about one inch and a half. When the neck is stretched about half-an-inch more is gained. Thus, we have generally not more than eight or nine rings of the trachea above the sternum. None of these rings can be felt externally. The second, third, and fourth are covered by the isthmus of the thyroid gland. The trachea, it should be remembered, recedes from the surface more and more as it descends, so that, just above the sternum in a short, fat-necked adult, the front of the trachea would be one inch and a half from the skin.

In the dead subject nothing is more easy than to open the trachea: in the living, no operation may be attended with greater difficulties. You must expect to find the patient with his head bent forwards, and the chin dropped, to relax as much as possible the parts in the neck. On raising his head, a paroxysm of dyspnœa is almost sure to come on, threatening instant suffocation. The elevator and depressor muscles draw the trachea and larynx up and down with a rapidity and a force which may bring the cricoid cartilage within half-an-inch of the sternum. The great thyroid veins which descend in front of the trachea are sure to be distended. There may be a middle thyroid artery. In children the two lobes of the thymus may rise in front of the trachea as high as the thyroid gland, so that the whole length of the air-tube may be covered by important parts which ought not to be divided. Considering all these possible complications, the least difficult mode of proceeding is to open the trachea just below the cricoid cartilage, and if more room be requisite, to divide the cricoid itself.

The sterno-mastoid muscle stands out in bold relief when the head turns towards the opposite shoulder. It is the great surgical landmark of the neck. Its inner border overlaps the common carotid, which can be easily compressed for a short time against the spine about one inch below the pomum Adami. The precise course of the artery corresponds with a line drawn from the sterno-clavicular joint to the angle of the jaw.

Between the sternal origins of the sterno-mastoid is the fossa above the sternum, more or less perceptible in different necks.

As it rises and falls, especially in distressed breathing, it was called by the old anatomists 'fonticulus gutturis.' In beautiful necks it is filled up by fat.

Notice the interval between the sternal and clavicular origins of the sterno-mastoid. Behind this lies the internal jugular vein, which here dilates into a small bulb. This is a good situation to listen to a venous murmur.

Many important parts lie behind the sterno-clavicular joint. There is the confluence of the internal jugular and subclavian veins; behind this confluence comes the division of the arteria innominata on the right side, and the common carotid on the left. Deeper still, the apex of the pleura and lung rises into the neck.

The extent to which the apex of the lung rises into the neck is greater than is generally supposed. Many observations in reference to this point lead me to the conclusion that the lung rises behind the sterno-mastoid, on an average, one inch and three quarters above the clavicle; in persons with long necks, as much as two inches. As this cervical portion of lung is peculiarly obnoxious to tubercular disease, it should be carefully examined. Its condition may be ascertained by percussion on the clavicle.

The depression above the clavicle, between the sterno-mastoid and the trapezius, is very manifest in emaciation and old age. In some necks only a small depression is visible, particularly when the trapezius has a broad insertion into the clavicle, and comes well forwards, so that its front border gives a graceful contour to the base of the neck.

In this depression, near the border of the sterno-mastoid, we feel the pulsation of the subclavian artery. Here the artery lies upon the first rib, and can be effectually compressed. But the pressure must be made in the right direction, or the artery will be pressed off the rib instead of against it. The plane of the rib is such, that the pressure, to be effectual, must be made in a direction downwards and inwards.

It is worth remembering that the outer border of the sterno-mastoid corresponds pretty nearly with the outer edge of the scalenus anticus, which is the surgical guide to the subclavian artery.

By pressing deeply at the upper part of the supra-clavicular fossa, the transverse process of the seventh cervical vertebra can be distinctly felt.

In long and thin necks, a thin cord is perceptible, running nearly parallel with the clavicle. It is the posterior belly of the omo-hyoideus. See it rising and falling in breathing and making tense during inspiration that part of the cervical fascia

which lies over the cervical portion of the lung. Thus it counteracts atmospheric pressure, and is in all respects a muscle of inspiration, co-operating with the sterno-mastoid and scaleni. In the language of transcendental anatomy, we may say that the central tendon of the omo-hyoid represents a rudimentary cervical rib. Its posterior belly is analogous to a serration of the serratus magnus; its anterior belly to a sterno-hyoid.

The Chest.

As a rule, the right half of the chest is larger than the left. Of ninety-two persons of the same sex and good constitutions, seventy-one had the right side the larger; eleven the left; ten had both sides equal. The maximum of difference in favour of the right was $1\frac{1}{4}$ inch. The measurements were made on a plane with the nipple.

The course of the arteria innominata corresponds with a line drawn from the middle of the junction of the first with the second bone of the sternum, to the right sterno-clavicular joint. When the artery rises higher than usual into the neck, its pulsation can be felt in the fossa above the sternum.

In fat persons, it is often difficult to count the ribs; hence the following rules may be useful:—

1. The transverse projection, slight, but always to be felt, at the junction of the first with the second bone of the sternum, corresponds with the level of the cartilage of the second rib.
2. The nipple in the male is placed, in the majority of cases, between the fourth and the fifth ribs, about three-quarters of an inch external to their cartilages.
3. The lower external border of the pectoralis major corresponds with the direction of the fifth rib.
4. A line drawn horizontally from the nipple round the chest cuts the sixth intercostal space midway between the sternum and the spine. This is a useful rule in tapping the chest.
5. When the arm is raised, the first visible digitation of the serratus magnus corresponds with the sixth rib. The three lower visible digitations correspond respectively with the seventh, eighth, and ninth ribs.
6. The scapula covers the ribs from the second to the seventh, inclusive.
7. The eleventh and twelfth ribs can be felt even in corpulent persons.

Immediately below the clavicle we recognise the interval between the pectoralis major and the deltoid. This space

varies in different individuals, depending on the vicinity of the muscles to each other. It is important, because it is a guide to the coracoid process and to the axillary artery. In a case of injury to the shoulder, to ascertain whether the coracoid process is broken, carry the arm outwards, to put the deltoid and pectoral muscles on the stretch, and make manifest the space between their opposite borders. Pressing the thumb into the space we can feel the inner side of the coracoid process, the apex being under the fibres of the deltoid; thus it is easy to ascertain whether it be broken. Moreover, this space corresponds with the line of the axillary artery; here its pulsation can be distinctly felt, and here it can be compressed against the second rib.

To define that part of the præcordial region which is less resonant on percussion, make a circle of two inches in diameter round a point midway between the nipple and the end of the sternum. This circle will define, sufficiently for all practical purposes, that part of the heart which lies immediately behind the wall of the chest, and is not covered by lung.

The apex of the heart pulsates between the fifth and sixth ribs, two inches below the nipple, and one inch to its sternal side. The place and extent, however, of the heart's impulse, vary a little with the position of the body. Of this anyone may convince himself by leaning forwards, backwards, on this side and on that; feeling, at the same time, the heart. Inspiration and expiration also alter the position of the heart. In a deep inspiration it descends from half to three-quarters of an inch, and can be felt beating at the pit of the stomach.

The aortic valves lie behind the third intercostal space, close to the left side of the sternum.

The pulmonary valves lie behind the junction of the third rib, on the left side with the sternum.

The tricuspid valves lie behind the middle of the sternum, about the level of the fourth costal cartilage.

The mitral valves lie behind the third intercostal space, about one inch to the left of the sternum.

Thus these valves are so situated that the mouth of an ordinary sized stethoscope will cover a portion of them all, if placed over the junction of the third intercostal space with the sternum. All are covered by a thin layer of lung; therefore we hear their action better when the breathing is for a moment suspended.

Now let us trace on the chest the outline of the lungs, with as much precision as their expansion and contraction in breathing permit.

The apex of each lung rises into the neck behind the clavicle and sterno-mastoid muscle as much as 1½ inch. From the sternal ends of the clavicles the lungs converge, so that their edges almost meet in the mesial line on a level with the second costal cartilage. Thus there is little or no lung behind the first bone of the sternum. From the level of the second costal cartilage to the level of the fourth, the margins of the lungs run parallel, close behind the middle of the sternum, consequently their thin edges overlap the great vessels at the base of the heart.

Below the level of the fourth costal cartilage the margins of the lungs diverge, but not in an equal degree. The margin of the right corresponds with the direction of the cartilage of the sixth rib: the margin of the left, being notched for the heart, runs behind the cartilage of the fourth. A line drawn perpendicularly from the nipple would find the lung margins about the lowest part of the sixth rib. Laterally, *i.e.* in the axillary line, the lung margin comes down as low as the eighth rib: posteriorly, *i.e.* in the dorsal line, it descends as low as the tenth.

The reflection of the pleura from the wall of the chest on to the diaphragm corresponds with a sloping line drawn from the bottom of the sternum over the cartilages of the ribs down to the lower border of the last rib. Therefore, a wound in the back penetrating the last intercostal space would certainly enter the pleural sac, though it would not necessarily injure the lung.

The Back.

Notice first the median furrow caused by the prominence of the erector spinæ on each side. The bottom of the furrow corresponds with the spine of the last lumbar vertebra.

A little friction with the fingers down the backbone will cause the spines of the dorsal and lumbar vertebræ to be tipped with red, so that they may be easily counted and any deviation from the straight line detected. Still it is worth remembering that the spine of the third dorsal is on a level with the commencement of the spine of the scapula—the spine of the seventh dorsal on a level with the inferior angle of the scapula—the spine of the last dorsal on a level with the last rib.

Opposite the spine of the third dorsal vertebra is the division of the trachea. In front this division is on the level of the junction of the first with the second bone of the sternum.

The root of the spine of the scapula is marked by a slight dimple in the skin. This is on a level with the third intercostal

space. A stethoscope placed on the inner side of this dimple would cover the bronchus, more especially the right, since it is nearer to the chest wall.

The place where the kidney is most accessible to pressure is below the last rib, on the outer edge of the erector spinæ.

The best line for opening the descending colon is a transverse incision beginning at the outer edge of the erector spinæ, midway between the crest of the ilium and the last rib, and continued for three inches or more, according to the amount of subcutaneous fat.

It is useful to know opposite what vertebræ the spinal nerves in the different regions arise from the spinal cord. They arise as follows—

The origins of the eight cervical nerves correspond to the interval between the occiput and the sixth cervical spine.

The origins of the first six dorsal nerves correspond to the interval between the sixth cervical and the fourth dorsal spines.

The origins of the six lower dorsal nerves correspond to the interval between the fourth and the eleventh dorsal spines.

The origins of the five lumbar nerves correspond to the interval between the eleventh and twelfth dorsal spines.

The origins of the five sacral nerves correspond to the spines of the last dorsal and the first lumbar vertebræ.

The movements of which the spine is capable are three-fold. 1, Flexion and extension; 2, lateral inclination; 3, torsion. Flexion and extension are freest between the third and the seventh cervical vertebræ, between the eleventh dorsal and the second lumbar, and between the last lumbar and the sacrum. This is well marked in severe cases of opisthotonos, where the body is supported on the back of the head and the heels.*

Still better may it be observed when a mountebank bends backwards, and touches the ground with his head.

The lateral movement is freest in the neck and the loins.

The movement of torsion or rotation round its own axis may be proved by the following experiment—Seated upright, with the back and shoulders well applied against the back of a chair, we can turn the head and neck as far as 70°. Leaning forwards so as to let the dorsal and lumbar vertebræ come into play, we can turn 30° more.

There are a few points worthy of observation about the scapula. It covers the ribs from the second to the seventh inclusive. We can feel its superior angle covered by the

* See a beautiful illustration of this in Sir C. Bell's Anatomy of Expression, p. 160.

trapezius. The inferior angle is covered by the latissimus dorsi, which keeps it well applied against the ribs in the strong and athletic; but in weak and consumptive persons the lower angles of the scapula project like wings—hence the terms 'scapulæ alatæ.' A line drawn horizontally from the spine of the sixth dorsal vertebra over the inferior angle of the scapula gives the upper border of the latissimus dorsi. Another line drawn from the root of the spine of the scapula to the spine of the last dorsal vertebra gives the lower border of the trapezius.

The sliding movement of the scapula on the chest can be best understood only on the living subject. It can move not only upwards and downwards as in shrugging the shoulders —backwards and forwards as in throwing back the shoulders, but it has a rotatory movement round a moveable centre. This rotation is seen while the arm is being raised from the horizontal to the vertical position, and is effected by the co-operation of the trapezius with the deltoid. The glenoid cavity is thus made to look upwards, and the inferior angle slides forwards, and is well held under the latissimus dorsi.

[*To be continued.*]

ARTICLE XXIII.—*Note on the Termination of the Second Case of Poisoning by Mercuric Methide.* (Reports, vol. i. p. 144.) By GEORGE N. EDWARDS, M.D. Cantab.

THIS patient continued in much the same state as reported on July 4, 1865, till within a few days of his death, which took place on April 7, 1866, the immediate cause being an attack of pneumonia. Occasionally he appeared better, and gained flesh slightly for a short time, but soon relapsed into his former condition, and on the whole his symptoms varied very little, his mental powers not improving at any time.

The post-mortem examination was made, forty hours after death, by Dr. Andrew, to whom I am indebted for the following notes:—

Body greatly emaciated, and generally anæmic. Rigor mortis present. Incipient greenish discoloration of the abdomen. Wasting general. Muscles of neck somewhat less wasted than others. Right pupil rather more dilated than left. Scalp slightly adherent. Bones of skull-cap natural. Longitudinal sinus contains a very few small clots. No adhe-

sion in sac of arachnoid; considerable amount of cerebro-spinal fluid. Convolutions somewhat deeper than usual, certainly not flattened; thickening of arachnoid in longitudinal fissure, which appears to pass across at a higher level than usual. Grey matter somewhat pink, in other respects of natural appearance; cavities of ventricles rather large, but not dilated, choroid plexuses and velum also appearing natural. Arachnoid over cerebellum somewhat opaque. Weight of brain with medulla, forty-one ounces. Pericardium contains ʒj. of serum. Pleuræ: Left, recent soft yellow lymph over lower lobe; membrane beneath ecchymosed. Right, one or two old adhesions. Heart, valves all healthy. Muscular substance firm and brown, not mottled: weight, six ounces. Bronchial glands enlarged, especially those connected with the root of the left lung. Trachea and bronchial tubes contain viscid mucus. Lungs: Left, the whole of the lower lobe in a state of pneumonic consolidation; the upper lobe in a state of grey hepatization. Right, appears natural, the lower lobe œdematous posteriorly. Stomach of enormous size, filling a large portion of the abdomen. Mucous membrane natural. Liver somewhat mottled and œdematous; weight, two pounds nine ounces. Bile duct pervious. Gall bladder contains a little pale viscid bile. Spleen moderately firm; weight, ten-and-a-quarter ounces. Supra-renal capsules natural. Kidneys: Left, capsule slightly adherent, leaving surface rough and congested; on section great congestion of pyramids; cortical substance increased in amount, congested in lines and dots of a fawn tint, mottled with minute specks; weight, six-and-a-half ounces, texture moderately firm, slightly œdematous, congestion of mucous membrane of pelvis and calices. Right, same as left; weight, five-and-a-quarter ounces. Bladder almost empty, mucous membrane posteriorly ecchymosed and congested. Intestines natural throughout, except that there is a good deal of congestion of the small intestine.

Article XXIV.—*On an Operation for the Relief of a Stricture of the Urethra.* By George W. Callender.

THOMAS C., æt. 26, was admitted into Darker Ward during the month of September, 1864, having been sent to St. Bartholomew's from Cornwall with the following history.

Seven and a half years ago he suffered from gonorrhœa, and from the beginning of the attack to the end of the fourth year

he seems scarcely ever to have been free from urethral irritation, and gradually there arose a narrowing of the urethra, so that, as time passed by, his water was voided with slowly increasing difficulty.

About this time he placed himself under the care of Mr. Hichens of Redruth, his difficulties having culminated in an attack of retention of urine. After the use of the hot bath and opium a number three elastic catheter was introduced, and after some few days a number five was passed with comparative ease. 'Had he continued his attendance at this time, I am satisfied,' writes Mr. Hichens, 'that most of the difficulties of his case would have been overcome: this, however, he declined doing, and I saw no more of him until the seventeenth of March, 1864, when he was again suffering from urgent symptoms of retention of urine.'

Despite attempts to introduce a catheter, carefully made and aided by the use of a hot bath, of opium, and eventually of chloroform, it was found impossible to draw off the water by the urethra; and the symptoms becoming most severe, the bladder was punctured above the pubes on the eighteenth of March. An elastic catheter was kept in the wound to draw off the urine, and the patient remained tolerably well until the twentieth of April, when he suffered from diffuse inflammation of the scrotum, with formation of matter, requiring free incisions. Up to July various attempts were made to pass instruments through the stricture, but without success. About this time, through his moving from home, the canula (a silver tube was now substituted for the elastic catheter) was allowed to remain in the bladder for three weeks, during which time it became coated with phosphates, and had to be removed by enlarging the supra-pubic opening.

In the progress of the case the wounds in the perineum had shrunken into sinuses, through which matter occasionally discharged, and which led in the direction of the urethra; but no urine escaped from them. And these, the opening above the pubes, and the closure of the urethra were his ailments when he was first seen at the Hospital on the twenty-fifth of September. His general health was good, but he was out of heart and desponding. On examining the perineum three sinuses were seen leading towards the urethra. One of them opened just below, the other two discharged in front of, the scrotum, right and left of the middle line. The opening above the pubes was occupied by a canula, around which the tissues were tightly contracted, and through which the urine was drawn off several times in the course of the day without inconvenience.

The urethra was closed; neither through it, nor yet through the openings in the perineum, did any of the contents of the bladder escape; and although I several times attempted to get various instruments past the stricture, the endeavour was always unsuccessful. An instrument could be passed beyond the site of the anterior fistulæ through a narrow passage, but then, deflecting to the patient's right side, it came to a stop about the site of the posterior sinus, and it remained uncertain whether this point had been reached by the natural or by a false passage.

As I was quite in the dark as to the extent of urethra contracted, and had nothing to guide me for making an ordinary perineal section, for there was no dilatation even of the urethra behind the stricture, I considered myself barred from attempting to restore the urethra in that way. On thinking over the difficulty, and after referring to the various proposals which have been made for the treatment of similar cases, it seemed to me desirable to attempt an operation which was first suggested, but not practised, by John Hunter, whose remarks upon the subject I here add, because in some notes of cases by foreign writers they have not received the attention which they deserve. That his observations were familiar to the older school of French surgery, for example, is evident from the following quotation from his essay which is thus rendered * by one of its number: 'Peut-être, ajoute le même auteur (M. Hunter), sera-t-il plus sûr, et moins douleureux pour le malade, d'introduire par la vessie, jusque dans l'urètre, l'extrémité courbée de la canule. . . . . Il n'est pas bien difficile, en effet, de pénétrer par cette voie dans le méat urinaire.' In a foot-note to the paragraph from which this passage was translated, Hunter† adds: 'Where this operation (the supra-pubic puncture) is performed in consequence of a stricture, I have conceived that by passing a catheter into the urethra from the bladder till it comes to the stricture, and then passing another straight canula from the glans down the urethra, that the two may nearly meet, only having the stricture between them; and a piercer may be passed down and forced into the end of the one from the bladder, and afterwards either a bougie or hollow catheter introduced.'

Following these suggestions, I proceeded to the operation, (October the twenty-seventh), with the assistance of my colleague Mr. Thomas Smith, the patient having been first

* Encyclopédie Méthodique, Part. Chir., Art. Paracentèse de la Vessie. Paris, 1790–9.

† Hunter's Works, ed. by Palmer, vol. ii. p. 292.

placed under the influence of chloroform. A number eight elastic catheter was passed into the bladder by the supra-pubic opening, after which it was armed with a stout stilette bent to the curve of an ordinary urethra. Guided by the finger, introduced for this purpose into the rectum, the catheter passed readily through the prostate, but was stopped in the membranous portion of the canal, and then, by withdrawing the stilette a little, the point of the instrument was tilted up, and it passed easily into, and was felt in the perineum.

A straight metal catheter introduced from the glans penis as far as the level of the posterior perineal sinus, reached, without actual contact, to the site of the instrument passed from the bladder, lying a little to its right side. Cutting from the perineum upon the extremity of the latter, and then examining the parts from time to time with a probe, the incision was prolonged forward through callous tissue for about an inch and a half, when the probe slipped up and struck the straight catheter at a level with the anterior fistulæ, and then emerged through the orifice of the urethra. Following this probe, passing through the wound in the perineum, and following also the elastic catheter as it was drawn backwards through the supra-pubic opening, a number eight elastic catheter was now readily introduced into the bladder, and the treatment was subsequently continued as for an ordinary perineal section.

From this operation the patient recovered rapidly, requiring only on one or two occasions to have the passage stretched as the wound in the perineum closed (for which purpose I made use of Matthieu's modification of the ingenious dilator invented by Moutin of Lyons, an instrument similar to the one employed by Mr. Henry Thomson). He was discharged on the twenty-seventh of January, the fistulæ closed, his urethra admitting the passage of a number eight catheter—eleven months later he continued perfectly well.

Before, however, he left the Hospital he had to be relieved of another trouble which had befallen him in the course of his greater disease. The examination of the bladder had, at an early period, acquainted us with the existence there of calcareous matter. No inconvenience was caused by its presence, but when the canal of the urethra had been restored, and it became necessary to close the supra-pubic opening, the removal of the calculi had to be provided for.

On the twentieth of November the hypogastric puncture was accordingly dilated, by means of ordinary sponge tents, to a size sufficient to admit two fingers. The opening into the bladder was then enlarged towards the pubes by incision,

and two calculi were extracted with a pair of ordinary straight forceps. They were composed of phosphates, and weighed seventy-six and forty-two grains respectively, the larger one measuring one inch by six-tenths of an inch in its long and short diameters respectively. After this operation the opening above the pubes closed rapidly and completely.

The first of the calculi extracted, being very friable, was broken somewhat in its removal. By introducing the finger into the rectum so as to lift its anterior wall, and by examining the interior of the bladder with the forefinger of the right hand, there was no trouble in ascertaining that every particle of calculus had been removed. I mention this because the difficulty of ensuring the removal of all such fragments has been, and is now, named as one of the objections to the performance of the supra-pubic operation for lithotomy.

ARTICLE XXV.—*On the Value of Palpation in the Diagnosis of Tubercular Disease of the Lungs.* By GEORGE N. EDWARDS, M.D.

FOR a considerable time past I have felt confident that the assistance which palpation affords in the diagnosis of tubercular disease of the lungs has not been estimated at its true value, and that the parietes of that part of the chest which is situated over the diseased portion of lung impart a totally different sensation to the cushiony ends of the fingers, on touch, from that which is felt over the rest of the chest walls. Having assured myself that it was possible, in a large majority of cases at least, to determine in this way the position of tubercle in the lungs, I instituted a series of observations, in which my own diagnosis, arrived at from palpation, was corroborated by others from auscultation and percussion.

The following method of proceeding was adopted. Taking a patient who had not previously attended at the Hospital, but who, from the symptoms and general appearance, seemed likely to be the subject of phthisis, I laid the palm of the right hand on the bare chest beneath the clavicle, the ends of the fingers being upwards towards that bone; and then, carefully avoiding the production of sound, made a very gentle stroke from the wrist upon the parietes. I repeated this over various parts of the chest, especially under both clavicles. I then pointed out to the observer the situation in which the physical signs would

probably be discovered, and left him to examine the patient by auscultation and percussion. In the accompanying table will be found the results of 50 cases out of 75 which have thus been examined consecutively, in all of which the position of tubercle as diagnosed by myself was confirmed by the auscultatory phenomena observed by others. I may add that the observer was in every case well versed in the physical examination of the chest, being in some instances one of my colleagues, in others Mr. Power, Resident Medical Officer at the Hospital for Diseases of the Chest, and in all the rest some gentleman who had been for a long time clinical clerk in the medical wards, and who was thoroughly acquainted with the use of the stethoscope, and that I always examined the patient myself by auscultation after hearing the report of the observer.

It is almost impossible to express in words what is the difference between the sensation imparted by that portion of the chest situated over tubercular and that over healthy lung: perhaps it may rather be described as a want of elasticity than anything else. I only know that it exists, and may be easily perceived by any one who will examine a sufficient number of cases.

TABLE.

Sex and age of patient	Position of tubercle as diagnosed by palpation	Auscultatory Signs
M. æt. 21	Beneath left clavicle	Tubular breathing, large crepitation, undue resonance of voice beneath left clavicle. Right side natural.
F. æt. 25	Beneath right clavicle	Coarse inspiration, prolonged expiration, and great resonance of voice beneath right clavicle, no moist sounds. Left side natural.
F. æt. 29	Beneath left clavicle	Feeble inspiration, prolonged expiration, a little crepitation beneath left clavicle. Right side natural.
F. æt. 19	Beneath right clavicle	Cavernous breathing and pectoriloquy beneath right clavicle. Left side natural.
F. æt. 37	Beneath right clavicle	Cavernous breathing, large crepitation, gurgling, and pectoriloquy beneath right clavicle. Left side natural.
F. æt. 20	Beneath right clavicle	Feeble inspiration, prolonged expiration, with click at end of forced inspiration beneath right clavicle. Left side natural.
F. æt. 53	Beneath left clavicle	Cavernous breathing, large crepitation, gurgling on cough beneath left clavicle. Right side natural.
M. æt. 34	Beneath left clavicle	Tubular breathing and some crepitation beneath left clavicle. Right side natural.
F. æt. 20	Both sides, left more advanced	Tubular, almost cavernous, breathing on left side, with abundant crepitation. Prolonged expiration, with click, on right side.

Sex and age of patient	Position of tubercle as diagnosed by palpation	Auscultatory Signs
M. æt. 36	Beneath left clavicle	Tubular inspiration and expiration, without moist sounds, on left side. Right side natural.
F. æt. 29	Both sides, right more advanced	Tubular inspiration and expiration, with some crepitation, on right side. Divided inspiration and prolonged expiration on left.
F. æt. 31	Beneath right clavicle	Cavernous breathing and crepitation on right, good breathing on left, side.
F. æt. 18	Beneath right clavicle	Tubular breathing, crepitation, and great resonance of voice on right side. Left side natural.
F. æt. 19	Beneath left clavicle	Tubular breathing with scanty crepitation beneath left, exaggerated breathing beneath right, clavicle.
M. æt. 23	Beneath right clavicle	Cavernous breathing, gurgling, and pectoriloquy on right side. Left side natural.
F. æt. 20	Beneath right clavicle	Tubular inspiration and expiration, with crepitation and increased vocal resonance, on right side. Left side natural.
F. æt. 33	Both sides, right more advanced	Cavernous breathing, gurgling, and pectoriloquy on right, tubular breathing and a little crepitation on left side.
F. æt. 25	Beneath left clavicle	Tubular breathing and crepitation on left side. Right side natural.
M. æt. 37	Beneath right clavicle	Cavernous breathing and some gurgling on right side, exaggerated breathing on left.
M. æt. 16	Beneath right clavicle	Cavernous breathing, crepitation, and very loud resonance of voice at humeral end of right clavicle. Left side natural.
M. æt. 14	Beneath right clavicle	Coarse inspiration, very prolonged expiration, and scanty crepitation on right, no unnatural sound on left side.
M. æt. 22	Beneath right clavicle	Cavernous breathing, gurgling, and pectoriloquy at upper part of right side. Left side natural.
F. æt. 28	Both sides, left more advanced	Tubular, almost cavernous, breathing, and pectoriloquy on left side. Prolonged expiration and scanty crepitation on right side.
M. æt. 19	Beneath left clavicle	Tubular inspiration, prolonged expiration, resonance of voice, no moist sounds, beneath left clavicle. Exaggerated breathing on right side.
M. æt. 25	Both sides, right more advanced	Cavernous breathing, large crepitation, and gurgling beneath right clavicle, a little crepitation on left side.
M. æt. 18	Beneath left clavicle	Tubular breathing beneath left, good beneath right clavicle.
M. æt. 30	Beneath left clavicle	Tubular breathing, click on deep inspiration, beneath left clavicle. Exaggerated breathing on right side.
F. æt. 45	Both sides, left more advanced	Cavernous breathing, gurgling, and pectoriloquy on left side, prolonged expiration and some crepitation on right.
F. æt. 27	Beneath left clavicle	Coarse inspiration, prolonged expiration, rhonchus, and a little crepitation beneath left clavicle. Right side natural.
F. æt. 26	Beneath right clavicle	Prolonged expiration, some crepitation and resonance of voice beneath right, good breathing beneath left clavicle.

Sex and age of patient	Position of tubercle as diagnosed by palpation	Auscultatory Signs
M. æt. 19	Beneath left clavicle	Coarse, divided, inspiration and prolonged expiration beneath left, exaggerated breathing beneath right clavicle.
F. æt. 32	Beneath left clavicle	Bronchial breathing, with abundant crepitation on left side; good breathing on right.
F. æt. 33	Beneath right clavicle	Tubular inspiration and expiration, with click under right, good breathing under left, clavicle.
F. æt. 38	Beneath right clavicle	Cavernous breathing, large crepitation, gurgling, and pectoriloquy beneath right clavicle, left side normal.
F. æt. 52	Both sides, right more advanced	Cavernous breathing, large crepitation, and pectoriloquy on right, tubular breathing and scanty crepitation on left, side.
M. æt. 22	Beneath right clavicle	Tubular breathing and large crepitation on right side. Slight click at end of inspiration on left.
M. æt. 35	Beneath left clavicle	Scarcely any air entering upper part of left lung, crepitation in left inter-scapular space, right side normal.
M. æt. 33	Beneath left clavicle	Breathing almost cavernous, with crepitation, beneath right, normal beneath left, clavicle.
M. æt. 20	Beneath left clavicle	Cavernous breathing, crepitation on cough, and pectoriloquy beneath left clavicle. Right side normal.
M. æt. 18	Beneath right clavicle	Tubular inspiration and expiration, without moist sounds, beneath right, good breathing beneath left, clavicle.
M. æt. 25	Beneath left clavicle	Tubular inspiration and expiration with great resonance of voice beneath left, good breathing beneath right, clavicle.
F. æt. 25	Beneath right clavicle	Tubular breathing with click, and great resonance of voice beneath right, good breathing beneath left clavicle.
F. æt. 24	Beneath right clavicle	Tubular inspiration and expiration, with some crepitation beneath right clavicle. Left side normal.
F. æt. 25	Both sides, right more advanced	Breathing very bronchial, with some crepitation, beneath right clavicle. Prolonged expiration beneath left.
M. æt. 21	Beneath left clavicle	Tubular breathing and some crepitation, beneath left, exaggerated breathing beneath right, clavicle.
M. æt. 38	Beneath left clavicle	Breathing very tubular, with crepitation and great resonance of voice, beneath left, good breathing beneath right, clavicle.
M. æt. 20	Beneath left clavicle	Tubular breathing, crepitation and great resonance of voice beneath left clavicle. Right side natural.
F. æt. 39	Beneath left clavicle	Cavernous breathing, gurgling and pectoriloquy beneath left, exaggerated breathing beneath right, clavicle.
F. æt. 29	Beneath right clavicle	Tubular inspiration and expiration beneath right clavicle, with crepitation at its sternal end. Left side natural.
M. æt. 38	Both sides, right more advanced	Cavernous breathing, gurgling and pectoriloquy on right side. Tubular breathing and a little crepitation on left.

ARTICLE XXVI.—*Extracts from the Statistical Report of the Hospital for the year* 1865.

THE following tables have been selected for republication from the annual report of the Registrars to the Hospital (Dr. Edwards and Mr. Willett), printed by order of the Governors; and attention is particularly requested to those numbered IV., V., which are intended to show the cases of disease bred in the wards of the Hospital during the year, thus indicating the amount of hurt, so to speak, which is suffered by our in-patients from this cause. This inquiry, commenced in 1863, may, it is hoped, be the means of directing more general attention to a subject the importance of which has been always recognised, whilst data for our information, or for comparison, have been wholly wanting.

It may be added respecting these cases, that not all the erysipelas, not all the fever, tabulated as beginning in the Hospital, is to be credited to our wards; for it is notorious that patients are constantly being admitted, ready charged, as it were, with erysipelas, or poisoned by fever, which breaks out and shows itself whilst the patients are recovering from some chance hurt for which they have been taken in. Practically it is, however, difficult to distinguish such cases in a tabular report.

During the year 1865 as many as 84 patients died within 24 hours of admission into the Hospital.

In Medical Wards . .	43; or, 11·50 per cent.
In Surgical Wards . .	41; or, 21·24 per cent.
From Disease .	13; or, 6·73 per cent.
From Accident	28; or, 14·50 per cent.
Patients brought in Dead	16
Number of Beds in Hospital . . .	650
Number of Patients under treatment in the wards during the year	6125

TABLE I.

SHOWING THE WHOLE NUMBER OF MEDICAL CASES UNDER TREATMENT, WITH THE RESULTS.

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in Days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER I.											
1. Smallpox	...	4	4	...	...	4	...	1	12		
2. Chickenpox	...	1	1	1	...	...	...	...	12		
3. Measles	...	1	1	1	...	...	...	...	53		
4. Scarlatina	4	41	45	43	...	...	2	36	36	4·44	0·54
(*a*) Dropsy after Scarlatina	4	35	39	30	...	...	9	69	36	23·08	2·41
6. Quinsy	2	18	20	20	...	...	...	11	40		
7. Croup	...	10	10	6	...	...	4	7	16	40·	1·07
8. Continued Fever	1	2	3	3	...	...	...	...	54		
(*a*) Typhus	16	76	92	61	...	...	31	46	41	33·69	8·82
(*b*) Typhoid	4	60	64	55	...	...	9	42	55	14·06	2·41
(*c*) Febricula	6	32	38	37	...	1	...	30	27		
9. Hooping-cough	...	7	7	5	...	...	2	68	51	28·57	0·54
10. Diarrhœa	...	27	27	22	...	3	2	9	20	7·41	0·54
11. Dysentery	1	7	8	5	...	2	1	66	...	12·50	0·27
13. Catarrh	...	2	2	2	...	...	...	7	...		
15. Ague	...	1	1	1	...	...	...	17	...		
16. Erysipelas	2	26	28	27	...	...	1	29	35	3·57	0·27
18. Pyæmia	...	4	4	...	...	...	4	4	20	100·	1·07
21. Rheumatism	55	253	308	285	19	4	...	43	42		
24. Syphilis—											
(*b*) Secondary	3	11	14	10	1	3	...	51	30		
(*c*) Hereditary	...	1	1	1	...	...	...	3	...		
26. Purpura	...	9	9	7	...	1	1	38	24	11·11	0·27

TABLE I.—*continued.*

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in Days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER I.—*continued.*											
27. Scurvy	1	1	2	2	...	...	...	84	...		
32. Worms	...	2	2	2	...	...	...	62	26		
33. Hydatids	1	2	3	1	2	...	...	53	115		
ORDER II.											
1. Gout	...	2	2	1	1	...	...	25	...		
2. Dropsy	3	3	6	2	2	1	1	46	55	16·66	0·27
3. Anæmia	2	10	12	11	...	...	1	...	35	8·33	0·27
4. Cancer—											
(*b*) Of Internal Organs	5	43	48	9	13	2	24	10	45	50·	6·42
9. Tumours	...	8	8	...	1	4	3	23	21	37·50	0·80
14. Scrofula	1	2	3	2	1	...	...	186	42		
15. Psoas Abscess, including Pelvic	...	5	5	...	2	...	3	8	84	60·	0·80
17. Tubercular Peritonitis	...	3	3	1	...	...	2	...	5	66·66	0·54
18. Phthisis	15	143	158	61	21	9	67	52	55	42·40	14·71
19. Hydrocephalus	...	1	1	1	...	...	...	84	...		
ORDER III.											
1. Meningitis	...	6	6	2	...	...	4	59	6	66·66	1·07
4. Apoplexy	...	10	10	...	...	...	10	1	...	100·	2·67
5. Paralysis	...	...	...	...	...	...	...	...	...		
(*a*) Hemiplegia	6	39	45	27	10	...	8	47	104	17·77	2·14
(*b*) Paraplegia	2	15	17	6	6	3	2	50	57	11·76	0·54
7. Chorea	7	28	35	32	...	...	3	54	59	8·57	0·80

TABLE I.—*continued.*

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in Days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER III.—*continued.*											
8. Epilepsy	...	52	52	31	8	9	4	14	26	7·69	1·07
10. Hysteria	1	14	15	13	...	2	...	...	23		
11. Convulsions	...	7	7	2	...	3	2	14	10	28·57	0·54
12. Insanity	...	3	3	1	...	2	...	11	8		
(*a*) Hypochondriasis	...	3	3	1	2	...	...	16	9		
14. Neuralgia	...	8	8	5	2	1	...	37	23		
16. Disease of Brain	2	5	7	2	3	...	2	40	28	28·57	0·54
17. Delirium Tremens	...	11	11	5	...	1	5	2	...	45·45	1·33
ORDER IV.											
2. Pericarditis	1	1	2	1	...	...	1	63	...	50·	0·27
5. Disease of Heart	2	57	59	15	5	3	36	38	37	61·02	9·63
6. Aneurism of Aorta	...	16	16	3	2	1	10	39	32	62·50	2·67
7. Do. of other Arteries	...	1	1	...	1	...	...	15	...		
11. Phlebitis	1	1	2	2	...	...	...	50	...		
ORDER V.											
1. Epistaxis	...	1	1	1	...	...	...	...	32		
2. Hæmoptysis	1	11	12	10	...	2	...	26	47		
3. Laryngitis	...	8	8	5	1	1	1	61	...	12·50	0·27
5. Bronchitis	9	77	86	52	3	4	27	38	33	31·40	7·22
6. Pleurisy	...	7	7	7	...	...	...	41	28		
(*b*) Empyema	3	7	10	7	...	1	2	70	80	20·	0·54

TABLE I.—*continued.*

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in Days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER V.—*continued.*											
8. Pneumonia	2	47	49	30	...	2	17	36	69	34·69	4·55
9. Pleuro-pneumonia	5	27	32	25	...	...	7	46	77	21·87	1·87
12. Disease of Lungs	...	1	1	...	...	...	1	28	...	100·	0·27
ORDER VI.											
3. Stomatitis	...	2	2	1	...	...	1	21	...	50·	0·27
4. Gastritis	...	4	4	4	...	...	...	24	9		
5. Enteritis	...	5	5	4	...	1	...	30	27		
6. Colonitis	...	4	4	4	...	...	...	37	...		
7. Peritonitis	...	6	6	1	...	...	5	25	65	83·33	1·33
8. Constipation	1	15	16	14	1	1	...	27	35		
10. Stricture of Intestines	...	1	1	...	...	...	1	6	...	100·	0·27
11. Ulceration of Intestines	...	2	2	...	...	...	2	12	4	100·	0·54
14. Dyspepsia	2	16	18	16	1	1	...	29	42		
17. Hæmatemesis	1	8	9	6	...	1	2	34	63	22·22	0·54
18. Melæna	1	1	2	2	...	...	...	70	...		
22. Disease of Spleen	...	4	4	...	1	3	...	36	47		
23. Disease of Liver	1	12	13	2	1	...	10	52	84	76·92	2·67
24. Jaundice	2	13	15	12	1	2	...	22	26		
25. Hepatitis	...	2	2	2	...	...	...	36	...		
26. Cirrhosis	...	2	2	1	...	...	1	23	...	50·	0·54
28. Ascites	...	14	14	3	2	1	8	45	30	57·14	8·14
29. Colic	...	7	7	3	...	4	...	13	15		
30. Lead Poisoning	2	21	23	20	1	2	...	33	57		

TABLE I.—*continued.*

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in Days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER VII.											
2. Albuminuria	8	57	65	25	8	6	26	65	66	40·	6·95
5. Diabetes	1	6	7	2	3	...	2	4·5	26	28·57	0·54
7. Hæmaturia	1	2	3	1	2	...	...	83	...		
ORDER VIII.											
4. Hæmatocele	1	5	6	4	1	1	...	...	38		
7. Metritis	1	9	10	7	...	3	...	...	60		
8. Ovarian Dropsy	1	4	5	5	...	...	...	...	51		
9. Ovarian Tumour	1	2	3	3	...	...	...	...	31		
11. Uterine Tumour	2	5	7	2	3	2	...	..	43		
12. Uterine Polypus	...	6	6	5	1	...	...	...	28		
16. Disorder of Menstruation	...	22	22	22	...	...	...	...	33		
17. Disease of Uterus, &c.*	7	165	172	159	9	2	2	...	38	1·16	0·54
ORDER IX.											

* Under this head are included 73 patients who were admitted for the application of leeches, and only remained one day in the Hospital.

TABLE I.—*continued.*

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in Days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER X.											
2. Boils	...	1	1	1	...	...	...	...	59		
3. Abscess	...	3	3	2	...	...	1	78	51	33·33	0·27
7. Urticaria	...	1	1	1	...	...	...	15	...		
12. Scabies	...	1	1	1	...	...	...	34	...		
26. Erythema Nodosum	1	3	4	4	...	...	...	28	11		
ORDER XI.											
1. Intemperance	...	28	28	26	...	2	...	2	1		
2. Privation	2	2	4	4	...	...	...	1	36		
8. Debility	5	39	44	38	3	3	...	36	39		
9. Poison	...	21	21	19	...	...	2	15	7	9·53	0·54
12. Drowning	...	1	1	1	...	...	...	32	...		
Diseases not classed	4	28	32	...	1	30	1	17	18	3·12	0·27
Total	213	1866	2079	1426	145	134	374	...	...	...	...
Remaining January 1, 1866	...	...	...	...	215						

TABLE II.

SHOWING THE WHOLE NUMBER OF SURGICAL CASES UNDER TREATMENT, WITH THE RESULTS.

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in Days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER I.											
6. Quinsy	...	9	9	9	...	...	...	14	17		
16. Erysipelas—											
(*a*) Simple	7	32	39	39	...	...	...	24	19		
(*b*) Phlegmonous	4	12	16	12	...	1	3	43	36	18·75	1·55
(*c*) Cellulitis (diffuse)	14	110	124	114	...	4	6	31	26	4·83	3·10
(*d*) Elephantiasis	...	1	1	...	...	1	...	...	...		
18. Pyæmia—											
(*a*) Acute	...	...	...	...	...	...	...	...	...		
(*b*) Chronic	...	1	1	...	...	1	...	...	...		
24. Syphilis—											
(*a*) Primary	23	147	170	143	1	26	...	26	35		
(*b*) Secondary	38	197	235	193	4	36	2	33	41	·85	1·03
(*c*) Inherited	...	10	10	9	...	1	...	29	48		
25. Gonorrhœa, including Bubo and Phymosis	20	206	226	174	...	51	1	23	27	·44	·51
27. Scurvy	...	...	...	...	...	...	...	...	...		
ORDER II.											
4. Cancer—											
(*a*) Of External Organs	5	50	55	31	18	...	6	18	35	10·90	3·10
6. Lupus	...	5	5	5	...	...	...	38	32		
8. Polypus	...	2	2	2	...	...	...	8	25		
9. Tumours	4	59	63	57	3	1	2	24	38	3·17	1·03

TABLE II.—*continued.*

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER II.—*continued.*											
10. Mortification—											
(*a*) Primary	...	...	...	...	...	...	...	...	...		
(*b*) Secondary	...	1	1	...	...	...	1	40	...	100·	·51
11. Phagedæna	...	...	...	...	...	...	...	...	...		
12. Bed-sores	...	2	2	1	...	...	1	72	34	50·	·51
13. Fistula	...	9	9	9	...	...	...	38	29		
14. Scrofula	1	12	13	7	3	...	3	55	52	23·07	1·55
15. Psoas Abscess, including Pelvic	1	18	19	8	5	2	4	40	37	21·05	2·07
ORDER III.											
9. Tetanus	...	3	3	1	...	...	2	17	...	66·66	1·03
14. Neuralgia	...	12	12	7	2	3	...	18	20		
18. Disease of Spinal Cord	...	2	2	1	1	...	...	41	...		
19. Conjunctivitis	2	17	19	17	...	2	...	18	25		
20. Amaurosis	...	6	6	2	4	...	...	23	8		
21. Cataract	...	4	4	1	3	...	...	24	...		
22. Iritis	...	2	2	2	...	...	...	37	...		
23. Corneitis	2	11	13	11	...	2	...	43	26		
24. Disease of Eye	2	20	22	16	3	3	...	24	32		
25. Disease of Ear	...	1	1	1	...	...	...	27	...		
ORDER IV.											
7. Aneurism	...	1	1	...	...	...	1	35	...	100·	·51
11. Disease of Arteries	...	2	2	...	2	...	...	...	6		

TABLE II.—*continued.*

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER IV.—*continued.*											
12. Phlebitis	1	8	9	8	...	...	1	31	27	11·11	·51
13. Disease of Veins	2	19	21	17	2	2	...				
(*a*) Rupture of ditto	...	4	4	4	...	...	...				
14. Nævus	...	5	5	5	...	...	...	15	9		
ORDER V.											
1. Epistaxis	...	6	6	6	...	...	...	4	7		
3. Laryngitis	1	...	1	1	...	...	...	23	...		
ORDER VI.											
1. Stricture of Œsophagus	...	1	1	...	1	...	...	11	...		
3. Stomatitis	1	3	4	4	...	...	...	28	16		
(*a*) Glossitis	...	3	3	3	...	...	...	9	12		
10. Stricture of Rectum	...	5	5	4	...	...	1	41	38	20·00	·51
11. Ulceration of ditto	...	1	1	1	...	...	...	18	...		
13. Hernia—											
(*a*) Femoral	...	31	31	24	...	...	7	20	33	22·58	3·62
(*b*) Inguinal	...	23	23	18	...	...	5	17	6	21·73	2·59
(*c*) Otherwise	...	3	3	1	...	...	2	...	36	33·33	1·03
19. Hæmorrhoids	1	10	11	10	...	1	...	15	19		
ORDER VII.											
7. Hæmaturia	1	2	3	3	...	...	...	...	...		
8. Stone	4	18	22	16	...	1	5	54	15	22·76	2·59

TABLE II.—*continued.*

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER VII.—*continued.*											
10. Cystitis	...	10	10	8	2	...	...	17	...		
11. Disease of Prostate Gland	2	8	10	6	2	1	1	26	...	10·0	·51
12. Stricture of Urethra, including Retention of Urine	5	43	48	44	...	2	2	28	...	4·16	1·03
13. Extravasation of Urine, including Urinary Abscess	2	6	8	6	...	...	2	31	...	25·00	1·03
14. Incontinence of Urine	...	1	1	...	1	...	...	8	...		
ORDER VIII.											
2. Orchitis	2	4	6	6	...	...	...	17	...		
3. Hydrocele	3	9	12	12	...	...	...	20	...		
4. Hæmatocele	...	3	3	3	...	...	...	32	...		
5. Disease of Testicle	...	6	6	5	...	1	...	47	...		
9. Ovarian Tumour	...	6	6	...	2	...	4	...	33	66·66	2·07
18. Disease of Female Organs (External)	...	24	24	18	2	3	1	...	38	4·16	·51
ORDER IX.											
1. Synovitis	10	56	66	59	5	2	...	42	50		
2. Periostitis	3	8	11	11	...	...	...	28	33		
3. Exostosis	...	2	2	2	...	...	...	48	17		
4. Caries	1	23	24	18	3	1	2	34	46	8·32	1·03
5. Necrosis	6	65	71	55	6	5	5	41	54	7·04	2·59
6. Disease of Bone	...	6	6	5	1	...	...				
7. Curvature of Spine	1	2	3	3	...	...	...	26	43		

TABLE II.—*continued.*

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER IX.—*continued.*											
8. Disease of Joints	17	95	112	68	22	8	14	58	50	12·5	7·25
(*a*) Disease of Glands	3	39	42	39	1	1	1	23	22	2·80	·51
(*b*) Ditto of Bursæ, Ganglia, &c.	8	84	92	89	1	2	...				
ORDER X.											
1. Carbuncle	2	15	17	15	...	...	2	32	20	11·76	1·03
3. Abscess	16	151	167	156	...	6	5	21	24	2·99	2·59
(*a*) Chronic	...	12	12	9	2	...	1	53	...	8·33	·51
4. Ulcer	12	65	77	70	1	2	4	29	36	5·19	2·07
5. Disease of Skin	...	4	4	4	...	...	...	35	31		
8. Eczema	1	16	17	16	...	1	...	38	40		
12. Scabies	...	...	...	...	...	...	...	...	...		
13. Pemphigus	...	4	4	2	1	1	...	27	...		
18. Erythema Nodosum	...	7	7	7	...	...	...	24	18		
21. Psoriasis	...	5	5	4	...	1	...	46	44		
26. Gangrena Senilis	...	4	4	...	...	...	4	29	...	100·	2·07
ORDER XI.											
5. Malformations	4	30	34	29	2	2	1	35	38	2·94	·51
10. Violence and Injury—											
(*a*) Burn and Scald	14	130	144	104	...	19	21	25	24	14·58	10·87
(*b*) Contusions	14	290	304	289	...	10	5	17	18	1·64	2·59
(*c*) Sprains	4	66	70	70	...	...	...	21	27		
(*d*) Gunshot Wounds	...	2	2	2	...	...	...	25	...		

TABLE II.—*continued.*

DISEASES	Remaining January 1, 1865	Admitted during 1865	Total Patients under Treatment	Discharged Cured and Relieved	Discharged Unrelieved	Discharged for other than Medical Reasons	Died	Mean Residence of Patients in Hospital, in days		Per-centage of Deaths to Number of Patients of each Disease	Per-centage of Deaths to Mortality from all Causes
								Male	Female		
ORDER XI.—*continued.*											
10. Violence and Injury—											
(*e*) Laceration of Limbs	2	10	12	10	...	...	2	51	...	16·66	1·03
(*f*) Wounds	14	252	266	253	...	6	7	12	19	2·63	3·62
(*g*) Concussion of Brain and Cord	2	102	104	97	...	2	5	15	11	4·80.	2·59
(*h*) Fractures—											
Simple	36	291	327	288	2	4	33	48	37	10·09	17·09
Compound	7	65	72	57	...	...	15	41	35	20·83	7·77
Ununited and Mal-union of	...	4	4	3	...	1	...	14	...		
(*k*) Dislocations—											
Simple	2	25	27	25	...	...	2	36	26	7·40	1·03
Compound	...	2	2	1	...	...	1	29	15	50·	·51
13. Suicide (Cut Throat)	...	8	8	7	...	1	...	28	30		
Diseases not classed	...	25	25	22	1	2	...	11	7		
TOTAL	327	3192	3519	2995	109	222	193			5·48	

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SUB-TABLE (FRACTURES AND DISLOCATIONS).

DISEASES	Under 5 years		5–10		10–15		15–25		25–35		35–45		45–55		55–65		65–75		75–85		Above 85		Total		Deaths		Per-centage of Deaths	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
SIMPLE FRACTURES—																												
Skull, Vault	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..	..	2	..	2		100·
,, Base	..	1	..	..	..	..	1	..	..	..	6	2	4	..	8	..	1	..	..	..	..	..	15	3	9	2		
Spine, Cervical	..	..	..	..	..	..	2	..	1	..	1	..	..	..	..	..	..	..	..	..	..	..	4	..	4	..	100·	
,, Dorsal	..	..	..	..	..	..	1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	2	..	2	..	100·	
,, Lumbar	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..		
,, Sacrum and Coccyx	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Bones of Face	..	..	..	1	1	..	2	..	1	..	..	..	1	..	1	..	..	..	..	..	..	..	6	1	1	..	16·66	
Sternum	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	1	..	100·	
Ribs	1	..	2	..	2	..	2	..	2	..	8	2	8	1	6	1	1	..	1	..	..	..	33	4	4	..	12·12	
Clavicle	..	..	..	..	..	..	..	2	1	..	..	..	..	..	..	1	1	1	..	..	..	..	2	4	..	..		
Scapula	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Humerus	..	..	..	..	..	..	5	..	2	..	2	..	..	..	1	1	2	..	..	..	..	..	12	1	..	1		100·
Radius and Ulna	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1	1	..	..		
Bones of Hand	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..		
Pelvis	..	..	..	..	..	..	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	1	..	1		100·
Femur, Neck	..	..	..	..	..	..	..	..	..	..	..	..	1	..	2	..	..	5	2	3	1	..	6	8	1	2	16·66	25·
,, Shaft	7	8	14	4	3	..	9	..	6	..	7	1	5	3	7	1	3	1	1	3	..	..	62	21	1	2	1·63	9·52
Patella	..	..	..	..	..	..	1	1	4	2	2	1	2	5	1	..	..	1	..	1	..	..	10	11	..	..		
Tibia and Fibula, or Tibia alone	3	2	2	..	7	1	16	..	15	1	14	1	11	3	6	..	4	2	1	1	..	..	69	11	..	..		
Fibula alone	..	..	1	..	2	..	5	..	6	3	8	..	5	..	1	2	..	..	..	..	..	..	28	5	..	..		
Bones of Foot	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..		
Total	11	11	19	5	15	1	47	3	38	8	50	7	39	12	28	6	12	11	5	9	1	..	265	73	23	10		
Deduct multiple fractures of separate bones																							7	4	..	..		
Total patients under treatment																							258	69	23	10	8·91	14·50

SUB-TABLE (FRACTURES AND DISLOCATIONS)—*continued.*

DISEASES	Under 5 years		5–10		10–15		15–25		25–35		35–45		45–55		55–65		65–75		75–85		Above 85		Total		Deaths		Per-centage of Deaths	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
COMPOUND FRACTURES—																												
Face	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..				
Skull, Vault	..	..	..	..	3	..	..	..	..	..	1	1	1	..	..	..	..	..	..	..	..	..	5	1	3	..	60·	
Clavicle and Scapula	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Sternum and Ribs	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Humerus	..	..	1	..	..	..	1	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	4	..	1	..	25·	
Radius and Ulna	..	..	..	..	1	..	..	..	..	..	1	..	3	..	..	..	..	1	..	..	..	..	5	1	..	..		
Bones of Hand	..	..	..	..	4	1	9	..	5	..	1	..	1	..	1	..	1	..	..	..	..	..	22	1	..	..		
Pelvis	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	100·	
Femur	1	..	..	..	..	..	..	..	1	..	..	..	..	..	1	1	..	..	..	..	..	..	3	1	1	1	33·33	100·
Patella	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Tibia or Fibula	..	..	1	..	1	..	3	..	5	..	4	..	4	1	2	..	2	..	1	..	..	..	23	1	6	1		100·
Bones of Foot	..	..	..	..	1	..	..	..	..	1	2	..	..	..	..	..	..	..	..	..	..	..	3	1	..	1		100·
Total	1	..	3	..	10	1	14	..	12	1	9	1	10	1	4	1	3	1	1	..	..	..	67	6	12	3		
Deduct multiple fractures of separate bones																							1					
Total patients under treatment																							66	6	12	3	18·18	50·
DISLOCATIONS, SIMPLE—																												
At Shoulder Joint	..	..	..	..	..	..	..	..	1	..	..	..	..	..	2	2	..	..	..	..	..	..	3	2	1	..	33·33	
„ Elbow „	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..		
„ Hip „	..	..	1	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..		
Of Patella	..	..	..	..	..	..	..	..	..	..	1	..	..	1	1	..	..	..	..	..	..	..	2	1	..	..		
At Ankle Joint	..	..	..	..	..	..	2	..	4	..	4	1	2	1	1	..	..	..	..	..	..	..	13	2	..	1		50·
„ Knee „	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..	..	..		
Total	..	..	1	..	..	..	2	..	7	..	5	1	2	2	4	2	1	..	..	..	..	..	22	5	1	1	4·54	20·
DISLOCATIONS, COMPOUND—																												
At Elbow Joint	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
„ Wrist „	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
„ Ankle „	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..		
Of Digits, Tarsal	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	1	..	1		100·
Total	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	2	..	1		50·

SUB-TABLE SHOWING TOTAL NUMBER OF CASES OF ERYSIPELAS, PYÆMIA, &c.

DISEASES	Under 5 years		5–10		10–15		15–25		25–35		35–45		45–55		55–65		65–75		75–85		Above 85		Total		Deaths		Per-centage of Deaths	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
1. Erysipelas—Admissions	1	1	1	3	..	..	6	7	7	8	3	4	6	3	2	3	..	..	..	..	..	..	26	29	2	1	7·69	3·44
2. Do. occurring in Hospital	..	..	2	..	1	1	3	8	3	2	2	3	5	..	..	1	..	1	1	..	..	..	17	16	3	..	17·64	
3. Do. do. after Operations	..	..	1	..	1	..	4	1	..	1	1	2	..	1	2	..	..	..	..	1	..	..	9	6	..	3		50·
1. Pyæmia—Admissions	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..		
2. Do. occurring in Hospital	..	..	..	..	..	1	2	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	2	2	2	1	100·	50·
3. Do. do. after Operations	..	..	..	..	1	..	2	1	..	1	1	..	..	1	1	..	..	..	..	..	..	..	5	3	4	2	80·	66·66
1. Phagedæna—Admissions	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
2. Do. occurring in Hospital	..	..	..	..	..	..	4	1	1	..	1	..	1	..	1	..	..	..	..	..	..	..	8	1	..	..		
3. Do. do. after Operations	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
1. Gangrene—Admissions	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	1	..	100·	
2. Do. occurring in Hospital	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	..	1		100·
3. Do. do. after Operations	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	2	..	1	..	50·	
1. Diffuse Cellulitis—Admissions	2	1	5	2	7	2	15	4	14	13	16	4	18	4	9	4	2	2	..	..	..	..	88	36	5	1	5·68	2·77
2. Do. occurring in Hospital	..	..	..	..	..	..	2	..	1	..	1	..	2	..	..	..	..	1	..	..	..	..	6	1	..	1		100·
3. Do. do. after Operations	..	..	..	..	1	..	3	1	3	..	1	..	3	..	..	..	1	..	..	1	..	..	12	2	2	1	16·66	50·
1. Tetanus—Admissions	..	..	..	..	..	..	..	..	..	..	1	..	2	..	..	..	..	..	..	..	..	..	3	..	2	..	66·66	
2. Do. occurring in Hospital	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	2	..	2	..	100·	
3. Do. do. after Operations	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
Fever, including Scarlatina—																												
2. Do. occurring in Hospital	2	2	..	..	..	..	1	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	8	5	1	..	33·33	
3. Do. do. after Operations	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..		
2. Rheumatism — occurring in Hospital	..	..	..	..	..	..	1	1	..	..	..	..	..	..	2	..	..	..	..	..	..	..	3	1	..	..		
3. Do. do. after Operations	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..		
2. Œdema Glottidis, Croup, Bronchitis, Pleurisy, Pneumonia —occurring in Hospital	..	..	..	..	..	..	1	..	1	..	..	..	1	..	3	2	..	1	..	..	..	..	6	3	3	3	50·	100·
3. Do. do. after Operations	..	..	..	..	..	..	1	1	..	..	..	..	1	..	..	1	..	..	..	..	..	..	2	2	1	2	50·	100·
2. Delirium Tremens—occurring in Hospital	..	..	..	..	..	..	..	..	1	..	5	..	4	2	2	..	..	..	..	..	..	..	12	2	1	..	8·33	
3. Do. do. after Operations	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..		
3. Hæmorrhage after Operations, Continued	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..		
Do. Recurring	..	..	..	..	..	..	4	1	1	1	..	1	..	..	..	..	..	..	..	..	..	..	5	3	..	..		
Do. Secondary	1	..	..	..	..	..	1	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	4	..	..	..		

TABLE III.

STATEMENT OF DISEASE OCCURRING WITHIN THE HOSPITAL IN PATIENTS ADMITTED FOR OTHER DISEASES.

Smallpox	Medical Wards	1		
	Surgical „	3	Total	4
Measles	Surgical „	1	„	1
Scarlatina	Medical „	7		
	Surgical „	4	„	11
Typhus	Medical „	8		
	Surgical „	3	„	11
Typhoid	Medical „	3		
	Surgical „	2	„	5

Erysipelas	Surgical Wards	48	Total	48
Cellular Inflammation	Surgical „	22	„	22
Pyæmia	Surgical „	12	„	12
Rheumatism	Surgical „	5	„	5
Phagedæna	Surgical „	9	„	9
Gangrene	Surgical „	3	„	3
Tetanus	Surgical „	2	„	2
Delirium Tremens	Surgical „	14	„	14

In the general tables all these patients are placed under the primary diseases for which they were admitted into the Hospital.

MEDICAL WARDS.

Smallpox	Recovered	0	Died	0	Removed to Smallpox Hospital	1	Total	1
Scarlatina	„	7	„	0			„	7
*Typhus	„	7	„	1			„	8
†Typhoid	„	2	„	1			„	3

* Recovered—4 patients, 3 nurses. Died—1 nurse.

† Recovered—1 patient, 1 nurse. Died—1 nurse.

TABLE IV.

DISEASE OCCURRING WITHIN THE HOSPITAL IN PATIENTS ADMITTED FOR OTHER CAUSES IN THE SURGICAL WARDS.

No. of Cases	Sex		Age	Complication	Disease or Injury
2	M.	F.	35—45	Erysipelas	Erysipelas.
2	...	F.	15—25	,, ...	Primary Syphilis.
1	...	F.	35—45	,, ...	Sec. Syphilis, Ulcers.
3	...	F.	15—25	,, ...	Gonorrhœa. Bubo. Condylomata.
1	...	F.	25—35	,, ...	Scrofula. Ulcer.
1	...	F.	15—25	,, ...	Psoas Abscess.
1	M.	...	25—35	,, ...	Neuralgia. Sciatica.
1	...	F.	35—45	,, ...	Conjunctivitis.
1	...	F.	55—65	,, ...	Ophthalmia.
1	...	F.	25—35	,, ...	Disease, Joint.
1	...	F.	35—45	,, ...	Synovitis.
1	...	F.	15—25	,, ...	Necrosis of Femur.
1	M.	...	25—35	,, ...	,, Metatarsal Bone.
1	M.	...	35—45	,, ...	Abscess.
1	...	F.	10—15	,, ...	Ulcer.
1	...	F.	15—25	,, ...	Eczema.
1	...	F.	55—65	,, ...	,,
1	M.	...	5—10	,, ...	Wounds.
2	M.	F.	15—25	,, ...	,,
2	M.	...	25—35	,, ...	,,
4	M. 3	F. 1	45—55	,, ...	,, (M 2) Fatal.
1	M.	...	5—10	,, ...	Fractures, Femur.
1	...	F.	75—85	,, ...	,, ,, Cervix. Fatal.
1	M.	...	45—55	,, ...	Compound Fracture, Tibia and Fibula.
1	M.	...	15—25	,, ...	Primary Amputation, Fore-arm.
2	M.	...	55—65	,, ...	Amputation for disease, Leg. (1) Fatal.
1	...	F.	75—85	,, ...	Herniotomy, Femoral. Fatal (from Exhaustion).
2	...	F.	45—55	,, ...	Excision, Cancer of Breast. (1) Fatal.
2	...	F.	25—35	,, ...	,, Tumours. (1) Fatal.

TABLE IV.—*continued.*

No. of Cases	Sex		Age	Complication	Disease or Injury
1	M.	...	5—10	Erysipelas	Excision, Joints.
2	M.	...	15—25	„	„ „
1	M.	...	10—15	„	Removal, Sequestrum.
2	M.	F.	15—25	„	„ „
1	M.	...	35—45	„	Division, Fistula in ano.
1	M.	...	10—15	Pyæmia	Necrosis.
2	M.	F.	15—25	„	„ Fatal.
1	...	F.	35—45	„	Abscess. Fatal.
1	M.	...	10—15	„	Amputation, Thigh. Fatal.
2	M.	F.	15—25	„	„ „ Fatal.
1	M.	...	35—45	„	„ Leg. Fatal.
1	M.	...	55—65	„	Lithotrity. Fatal.
1	...	F.	25—35	„	Excision, Tumour.
1	...	F.	45—55	„	„ „ Fatal.
1	M.	...	15—25	„	„ Joint.
3	M.	...	15—25	Phagedæna	Primary Syphilis. Bubo.
2	M.	...	25—35	„	„
1	M.	...	45—55	„	„
1	M.	...	55 – 65	„	„
2	M.	F.	15—25	„	Gonorrhœa. Bubo.
1	...	F.	55—65	Gangrene	Comp. Frac. Femur. Fatal.
1	M.	...	10—15	„	Amputation, Leg.
1	M.	...	55—65	„	Ligature of Artery (Axillary). Traum. Aneur. Fatal.
1	M.	...	15 – 25	Diffuse Cellular Inflammation (including Suppuration and Sloughing of Cellular Tissue) . .	Necrosis. Inferior Maxilla.
1	M.	...	15—25	„ „ „	Wound.
1	M.	...	25—35	„ „ „	„
1	M.	...	35—45	„ „ „	„
1	M.	...	45—55	„ „ „	„ Fatal.
1	...	F.	65—75	„ „ „	„ „

TABLE IV.—*continued.*

No. of Cases	Sex		Age	Complication	Disease or Injury
1	M.	...	5—10	Diffuse Cellular Inflammation (including Suppuration and Sloughing of Cellular Tissue) . .	Fracture. Femur.
1	M.	...	45—55	,, ,, ,,	Compound Fracture, Radius.
1	M.	...	25—35	,, ,, ,,	Primary Amputation. Fore-arm.
1	M.	...	15—25	,, ,, ,,	,, ,, Finger.
1	M.	...	25—35	,, ,, ,,	Amputation, Thigh.
1	M.	...	15—25	,, ,, ,,	,, Leg.
1	M.	...	25—35	,, ,, ,,	,, ,, Fatal.
3	M.	...	45—55	,, ,, ,,	,, Fingers.
1	M.	...	35—45	,, ,, ,,	Herniotomy, Inguinal.
1	M.	...	65—75	,, ,, ,,	,, ,, Fatal.
1	...	F.	75—85	,, ,, ,,	,, Femoral.
1	...	F.	15—25	,, ,, ,,	Excision, Tumour.
1	M.	...	15—25	,, ,, ,,	,, Testis.
1	M.	...	10—15	,, ,, ,,	Circumcision.
1	M.	...	65—75	Tetanus	Burn. Fatal.
1	M.	...	45—55	,, ...	Comp. Fract., Tibia and Fibula. Fatal.
1	M.	...	Under 5	Fevers.—Rubeola	Burn and Scald.
1	M.	...	Under 5	,, Scarlatina	,,
2	...	F.	15—25	,, ,, ...	Secondary Syphilis.
1	...	F.	Under 5	,, ,, ...	Tenotomy.
1	...	F.	15—25	,, Typhoid	Gonorrhœa.
1	M.	...	15—25	,, ,, ...	Bed-sores. Fatal.
1	...	F.	Under 5	,, Variola	Disease, Joint.
1	...	F.	Under 5	,, ,, ...	Scald.
1	...	F.	10—15	,, ,, ...	Removal, Sequestrum, Humerus.
1	M.	...	55—65	Rheumatism	Diffuse Cellulitis.
1	M.	...	15—25	,, ...	Synovitis.
1	...	F.	15—25	,, ...	Contusion.
1		...	55—65	,, ...	,,

TABLE IV.—*continued.*

No. of Cases	Sex		Age	Complication	Disease or Injury
1	M.	...	35—45	Rheumatism	Tracheotomy.
1	...	F.	55—65	Inflammation of Respiratory Organs	Ulcer. Fatal.
1	M.	...	25—35	,, ,, ,,	Contusion.
1	M.	...	55—65	,, ,, ,,	,,
1	M.	...	15—25	,, ,, ,,	Fracture, Clavicle.
1	M.	...	45—55	,, ,, ,,	,, Skull-base. Fatal.
2	M.	F.	55—65	,, ,, ,,	,, Femur. Fatal.
1	M.	...	55—65	,, ,, ,,	,, Humerus.
1	...	F.	65—75	,, ,, ,,	,, Femur cervix. Fatal.
1	...	F.	15—25	,, ,, ,,	Amputation, Thigh. Fatal.
1	...	F.	55—65	,, ,, ,,	Herniotomy, Femoral. Fatal.
1	M.	...	15—25	,, ,, ,,	Excision, Joint. Fatal.
1	M.	...	45—55	,, ,, ,,	Lig. of Artery (Femoral), Aneurism. Fatal.
1	M.	...	25—35	Delirium Tremens	Diffuse Cellulitis.
6	M.	...	35—45	,,	Contusion, 2. Wounds, 3. Fracture, 1.
6	M. 4	F. 2	45—55	,,	Diff. Cell. (M. 1), Fatal. Contus. (M. 1). Frac. (F. 1). Comp. Fract. (M. 2) (F. 1).
1	M.	...	55—65	,,	Phlegmonous Erysipelas.
1	M.	...	15—25	Hæmorrhage (after Oper.) Continued	Excision. Testicle.
1	M.	...	15—25	,, Recurrent	Amputation for Disease. Thigh.
1	...	F.	25—35	,, ,,	,, ,, ,,
1	...	F.	35—45	,, ,,	,, Cancer of Breast.
2	M.	...	15—25	,, ,,	Excision. Tumours.
1	M.	...	25—35	,, ,,	,, ,,
1	...	F.	15—25	,, ,,	Excision. Joint.
1	M.	...	15—25	,, ,,	Removal Sequestrum.
1	M.	...	25—35	,, Secondary	Primary Amputation. Fore-arm.
1	M.	...	25—35	,, ,,	Amputation for Disease. Thigh.
1	M.	...	Under 5	,, ,,	Tenotomy.
1	M.	...	15—25	,, ,,	Perineal Section.

TABLE V.

Causes of Death in Fifty Patients after Operation.

No. of Cases	Sex		Age	Operation	Cause of Death
1	M.	...	50—60	Primary Amputation, Leg	Collapse. 1st day. Internal Injuries.
1	...	F.	60—65	,, ,, ,,	Exhaustion. Fatty Degeneration of Organs. (P.M.) 14th day.
1	M.	...	65—70	,, ,, ,,	Collapse. 2nd day.
1	...	F.	20—30	,, ,, Toes	Thrombosis. 14th day.
1	M.	...	15—20	Secondary ,, Thigh	Pyæmia. 25th day.
1	M.	...	20—30	,, ,, ,,	Exhaustion. 6th day.
*1	...	F.	20—30	,, ,, ,,	Exhaustion. 3rd day.
1	...	F.	20—30	Amputation for Disease, Thigh	Recurrent Hæmorrhage. Diffuse Cellulitis. 10th day.
1	...	F.	20—30	,, ,, ,,	Phthisis. 62nd day.
1	...	F.	20—30	,, ,, ,,	Pyæmia. 23rd day.
1	M.	...	40—50	,, ,, ,,	Collapse. Chloroform Vomiting. 4th day.
1	M.	...	40—50	,, ,, ,,	Collapse. Fatty Degeneration of Heart. (P.M.) 2nd day.
1	...	F.	40—50	,, ,, ,,	Exhaustion. 15th day.
1	M.	...	50—60	,, ,, ,,	Phthisis. 98th day.
1	M.	...	10—15	,, ,, Leg	Phthisis. 110th day.
1	M.	...	20—30	,, ,, ,,	Exhaustion. 20th day. (? Fatty Degeneration of Organs.)
1	M.	...	30—40	,, ,, ,,	Diffuse Cellulitis. 10th day.
1	M.	...	40—50	,, ,, ,,	Pyæmia. 55th day.
1	...	F.	40—50	,, ,, ,,	Exhaustion. Disease of Kidneys. 60th day.
1	M.	...	60—65	,, ,, ,,	Phlegmonous Erysipelas. 22nd day.
1	M.	...	50—60	,, ,, Finger	Diffuse Cellulitis. Diabetes. 5th day.
1	M.	...	50—60	Herniotomy, Inguinal, with opening of Sac	Peritonitis. 2nd day. (P.M.)
1	...	F.	50—60	,, ,, ,,	Peritonitis. 3rd day. (Present on admission.)
1	M.	...	70—75	,, ,, ,,	Diffuse Cellulitis. Sloughing of Sac. 9th day.

TABLE V.—*continued.*

No. of Cases	Sex		Age	Operation	Cause of Death
1	M.	...	Over 75	Herniotomy, Inguinal, with opening of Sac	Peritonitis. 4th day. (P.M.)
1	...	F.	40—50	,, Femoral, with opening of Sac	Peritonitis. 4th day.
1	...	F.	50—60	,, ,, ,,	Exhaustion. 4th day. (No P.M.)
1	...	F.	60—65	,, ,, ,,	Bronchitis. 27th day. (Wound healed.)
1	...	F.	70—75	,, ,, ,,	Erysipelas. Exhaustion. 126th day. (Wound healed.)
1	...	F.	Over 75	,, ,, ,,	Vomiting. Exhaustion. 4th day. (P.M.)
1	...	F.	Over 75	,, Femoral, without opening of Sac	Diffuse Cellulitis. 6th day.
1	...	F.	...	,, Umbilical, with opening of Sac	Gangrene of Bowel. 12th day.
1	M.	...	Under 5	Lithotomy	Peritonitis. 4th day.
1	...	F.	15—20	,, ...	Tuberculosis. Advanced Phthisis. (P.M.) 4th day.
1	M.	...	30—40	,, ...	Disease of Kidneys. Uræmia. 2nd day.
1	M.	...	50—60	,, ...	Cystitis. 3rd day.
1	M.	...	50—60	Lithotrity	Pyæmia. 21st day.
1	...	F.	30—40	Excision of Tumours, Cancer of Breast	Intussusception of Ileum. (P.M.) 3rd day.
1	...	F.	30—40	,, ,,	Erysipelas. 13th day.
1	M.	...	70—75	,, Cancer of Lip	Diffuse Cellulitis. Exhaustion. 6th day.
1	...	F.	30—40	,, not Cancerous	Erysipelas. 11th day.
1	...	F.	50—60	,, ,,	Pyæmia. 5th day.
1	M.	...	15—20	Excision of Joints, Wrist . .	Diphtheria. 40th day.
1	M.	...	20—30	,, ,, Knee . .	Pyæmia. 16th day.
*	...	F.	20—30	,, ,, ,, . .	Exhaustion. (Sec. Amp. Thigh.) 56th day.
1	M.	...	30—40	Trephining Spine for Injury .	Asphyxia. (Present before operation.) Died during the operation.

TABLE V.—*continued.*

No. of Cases	Sex		Age	Operation	Cause of Death
2	...	F.	30—40	Ovariotomy	Peritonitis. 3rd day.
1	M.	...	40—50	Ligature of Arteries for Aneurism, Femoral	Pneumonia. 13th day.
?1	M.	...	60—65	Ligature of Arteries for Traumatic Aneurism, Axillary	Gangrene of Arm. 7th day.
?	M.	...	60—65	Reduction of Old Dislocations, Humerus	Rupture of Axillary Artery. Formation of Traumatic Aneurism. (Ligature of Axillary Artery.) 53rd day.
	M.	...	50—60	Perineal Section . . .	Exhaustion. Disease of Bladder and Kidneys. 6th day.

* ? Refer to the same Patient.

ST. BARTHOLOMEW'S HOSPITAL & COLLEGE.

PRESENT MEMBERS.

Consulting Physician, DR. BURROWS.

Physicians to the Hospital.

DR. FARRE, DR. JEAFFRESON, DR. BLACK, DR. MARTIN.

Consulting Surgeons, MR. SKEY, MR. LAWRENCE.

Surgeons to the Hospital.

MR. WORMALD, MR. PAGET, MR. COOTE, MR. HOLDEN.

Assistant Physicians to the Hospital.

DR. EDWARDS, DR. HARRIS, DR. ANDREW, DR. SOUTHEY.

Assistant Surgeons to the Hospital.

MR. SAVORY, MR. CALLENDER, MR. T. SMITH, MR. WILLETT.

Physician Accoucheur, DR. GREENHALGH.

Apothecary, MR. WOOD.

SCHOLARS.

1861	Baker, W. Morrant	Jeaffreson, Horace	1859
1856	Barford, James G.	Newman, William	1854
1864	Bateman, F.	Paine, William	1860
1855	Best, Henry D.	Rhind, Samuel	1850
1860	Brook, Charles	Richards, F. W.	1863
1851	Brown, John A.	Sadler, Michael J.	1855
1866	Butcher, W. D.	Senior, Charles	1858
1852	Callender, George W.	Sharpin, Henry W.	1849
1864	Cole, T.	Shepard, W. L.	1865
1857	Crowfoot, William M.	Snook, J. W.	1864
1861	Done, John	Square, W.	1866
1865	Garrett, W. J.	Thompson, Charles R.	1850
1856	Goodall, Ralph	Turner, William	1853
1858	Helm, George F.	Vernon, Bowater J.	1862
1862	Hingston, Albert	Winkfield, William B.	1857
1853	Humphry, Frederick A.		

Abercrombie, J., MD Cantab. 1848, FRCPL 1849
Ackland, W., LSA 1823
Ackland, W. H., MRCS 1847, MD St. And. 1862
Acton, W., MRCS 1840
Adam, J., MRCS 1835, MD Edin. 1855
Adams, E. B., MRCS 1850, LSA 1852
Adams, J., MRCS 1859, MD St. And. 1859
Adams, J., MRCS 1848, LFPS Glas. 1859
Adams, J. D., MRCS 1858, MD St. And. 1862
Adams, J. O., MRCS, LSA 1865
Adams, R., MRCS 1854, LRCP Edin. 1860
Addison, C. E., MRCS 1862, LKQCP Ireland 1865
Aldrich, P., LSA 1835, MRCS 1859
Alger, J. S., LSA 1831, MRCS 1842
Allas, L., MRCS 1857
Allen, A. B. de Lisle, MRCS 1845
Allen, E., MRCS, LSA 1842
Allen, G., LSA 1845, MD St. And. 1856
Allen, J., MRCS 1855, LSA 1856
Allen, J. W., MRCS 1855, LSA 1857
Allen, R., LSA 1833, MRCS 1834
Alsop, E., LSA 1843, MRCS 1847
Amsden, J. G., LSA 1837, MD Jena 1853
Anderson, D. H. B., MRCS 1866
Anderton, C., MRCS, LSA 1834
Andrew, J., MD Oxon. 1863, FRCP 1866
Andrew, J. M., MRCS 1855
Andrew, W. W., MRCS 1860, MB Cantab. 1861
Andrews, E., MRCS, LSA 1848
Andrews, F. F., MRCS 1850, MD St. And. 1862
Angier, G. A., MRCS 1861, MD St. And. 1862
Angier, J. H., MRCS, LSA 1826
Antrobus, D., LSA 1836, MRCS 1859
Archer, E., MRCS 1848, MD Paris 1865
Archer, J., LSA 1830, FRCS 1859
Archer, L. H., MRCS 1851
Arding, W., MD Edin. 1827
Armstrong, J., MRCS 1828, MD Aber. 1851
Armstrong, S., MRCS, LSA 1861
Arnold, J., MRCS 1864
Arrowsmith, J. Y., MRCS 1817, FRCS 1843
Ascroft, E. H., MRCS, LSA 1830
Ashford, J. B., MRCS 1843, MD Aber. 1851
Ashton, J. H., MRCS 1863
Atherley, J., in practice prior to 1815
Atkinson, H. M., LSA 1829, MRCS 1830
Atkinson, R., LSA 1822, MRCS 1823
Attfield, G., MRCS 1850
Atwood, W. A., MRCS 1860
Austin, T. J., LSA 1842, MRCS 1843

Badger, S., MRCS, LSA 1844
Badley, J. P., MRCS, LSA 1847
Badley, J., MRCS 1803, FRCS 1843
Bage, W. F. J., LSA 1828, MRCS 1830
Bagg, G. W., MRCS 1843, LSA 1845
Bailey, F. C., LSA 1860, MRCS 1864
Bailey, J., LSA, MRCS 1833
Baillie, A., MRCS 1825
Baillie, M. T. B., LSA 1849, MRCS 1858
Bainbridge, W., MRCS, LSA 1833
Baker, A., MD Montreal 1848
Baker, A., MRCS 1837, FRCS 1852
Baker, J., LSA 1828, LRCPE 1859
Baker, R., LSA 1820, MRCS 1821
Baker, R. L., LSA 1841, FRCS 1861
Baker, S. I., MRCS, LSA 1853
Baker, T. J., LSA, MRCS 1833
Baker, W., MRCS 1811
Baker, W. M., MRCS 1861, FRCS 1864
Ball, D., LSA 1824, FRCS 1844
Baller, J. H., MRCS 1829, MD St. And. 1861
Baly, G., MRCS 1855
Bancks, T., LSA 1834, FRCS 1860
Banks, W. R., LSA 1853, MD St. And. 1855
Barbeau, A., MRCS 1851
Barford, J. G., MRCS 1857, LRCP 1861
Barker, E., LSA 1823, FRCS 1849
Barker, E., jun., MRCS 1856, LSA 1858
Barnes, C. H., LSA 1823, FRCS 1852
Barnes, F. W., MRCS 1845
Barnett, H., LSA 1828, MRCS 1834
Barreau, F. H., MRCS 1859
Barrett, C. A., MRCS 1854, LSA 1859
Barringer, T. S., MRCS 1839, LRCP Edin. 1860
Barrow, B., MRCS 1836, FRCS 1862
Barsham, J. B., MRCS, LSA 1836
Barter, C. S., LSA 1859, FRCS 1865
Barter, T., LSA 1825, MRCS 1826
Bartlam, E. G., MRCS 1839, LSA 1840
Bartleet, F., LSA 1824, FRCS 1852
Bartley, R. T. H., LSA 1840, MD St. And. 1815
Barton, S., MRCS 1811, FRCS 1844
Barwise, J., LSA 1840 MD St. And. 1862
Basley, C., MRCS, LSA 1840
Bass, A., MRCS 1852, MD St. And. 1857
Bateman, H., LSA 1828, FRCS 1855
Bateman, F., MRCS, LSA 1866
Batt, A., MRCS 1852, MD St. And. 1857
Batten, R. W., MRCS 1857, MD Lond. 1860
Batten, W., LSA 1832
Batty, B., MRCS 1815
Battye, W., MRCS 1864
Baxter, H. F., MRCS 1838
Bazeley, W., MRCS 1861, LSA 1862
Beach, H., LSA 1828, MRCS 1829
Beale, J. S., MRCS 1846, LM 1853
Beale, T. M., LSA 1855, MRCS 1856
Beard, C. T., MB Cantab. 1855, MRCP 1859
Beattie, H. M., MRCS 1861, MD St. And. 1862

Beckett, F. M., MRCS 1864
Bedwell, H., MRCS 1840, FRCS 1860
Bedwell, J. R., LSA 1831, MRCS 1832
Beecroft, J., MRCS, LSA 1847
Bell, G. W., MD Edin. 1835
Bell, J., LSA 1825
Bellis, B., LSA 1828, MRCS 1829
Bendall, J., MRCS 1861, LSA 1862
Benfield, T. W., MRCS 1843, FRCS 1859
Bennett, F. G., MRCS, LSA 1864
Bennett, F., MRCS 1854
Bennett, G. J., MRCS 1859, LSA 1860
Bennett, J., MRCS, LSA 1839
Berry, S., LSA 1830, FRCS 1852
Best, F. A., MRCS 1863, LSA 1864
Best, H. D., MRCS, LSA 1855
Betham, J., MRCS 1855
Bett, R. L., MRCS 1859
Beveridge, J. E., MRCS 1836
Bewley, C., MRCS 1852, LSA 1856
Bickersteth, E. R., MRCS 1851, FRCS Edin. 1855
Bicknell, H., MRCS 1855
Bidden, R. C., MRCS 1834
Biddle, H., LSA 1822, MRCS 1823
Billett, J., in practice prior to 1815
Birch, W., MRCS 1823, FRCS 1844
Bird, H., LSA 1834, FRCS 1858, Ext. LRCP Lond. 1858
Bird, J., LSA 1821, MRCS 1825
Bird, J., MRCS, LSA 1834
Birt, A. W., MRCS 1862
Bishop, E., MD. Erlang. 1856
Black, P., MD Oxon. 1839, FRCP 1845
Blackman, F., MRCS 1854, LSA 1855
Blackman, F., MRCS 1856, LM 1858
Blackmore, H. P., MRCS 1857, MD St. And. 1857
Blackmore, S., LSA 1828, MD St. And. 1852
Blaker, E. S., MRCS 1859, LRCP Edin. 1860
Blaker, H. M., MRCS 1839, LSA 1840
Blasson, T., LSA 1823, FRCS 1858
Blenkinsop, H. LSA 1834, FRCS 1852
Bletchly, W. E., MRCS, LSA 1862
Blick, T. C., MRCS 1832, LSA 1833
Blomfield, J., MD Aber. 1855, FRCS 1855
Bloxam, J. A., MRCS 1864, LSA 1866
Blundell, J. W. F., MD Giessen 1851, LRCP Edin. 1859
Blundell, T. L., MRCS 1812, MD Glasg. 1826, FRCS 1852
Blunt, T., MRCS 1863, MD Edin. 1865
Blyth, E. J., MRCS, LSA 1850
Bodilly, J. D., MRCS 1857, LSA 1860
Bodington, G., LSA 1825, LRCP Edin. 1859
Bodman, J. B., LSA 1834
Bogg, E. B., MRCS, MD St. And. 1859
Bogg, J., LSA 1821, MRCS 1822
Bond, E. T., MRCS, LSA 1820
Bond, F. St. Q., MRCS, LSA 1858
Bond, H. H., LSA 1826, MRCS 1827
Bond, H. J. H., MD Cantab. 1831, FRCP 1835
Bond, W. Y., LSA 1817
Boodle, R. H., MRCS, LSA 1843
Bore, G. H., LSA 1834, MRCS 1835
Borrett, J., MRCS 1827, MD Edin. 1830
Bossy, A. H., LSA 1851, MRCS 1859
Bostock, E. J., MRCS 1864, LSA 1865
Bostock, J. A., MRCS 1838
Bott, T., LSA 1829, MRCS 1830
Boughton, J. H., MRCS 1848
Bowen, W., MRCS 1855
Bowes, R., MRCS, LSA 1831
Box, J., LSA 1826, MRCS 1827
Bradshaw, A. F., MRCS 1857
Brake, W. N., MRCS 1844
Bramley, L., LSA 1829, FRCS 1853
Bratt, W., MRCS, LSA 1849
Brendon, P., MRCS 1817, FRCS 1852
Brent, R., MRCS 1844, MD St. And. 1845
Brereton, C. Le Gay, MRCS 1851
Brewer, A., MRCS 1832, LSA 1834
Brewer, A. H., MRCS 1864, LRCP 1865
Brewer, C. C., MRCS 1864, LSA 1865
Brewer, H. M., MRCS 1864, LSA 1865
Brewer, J., LSA 1825, FRCS 1852
Brewer, T., MRCS, LSA 1863
Brewer, W. H., MRCS 1839
Brickwell, C. J., LSA 1832, MRCS 1833
Brickwell, H., MRCS 1839
Brickwell, J., LSA 1829, MRCS 1830
Brickwell, J., senr., MRCS, LSA 1829
Brickwell, J., MRCS, LSA 1856
Bridger, J., LSA 1857, MRCS 1859
Brigstocke, H., MRCS 1828, MD St. And. 1861
Brigstocke, R. W., MRCS, LSA 1859
Brine, J. E., MRCS 1844, LSA 1845
Bringloe, C., MRCS 1851, LSA 1852
Briscoe, J., MRCS 1842, LSA 1844
Broadbent, J. C., MRCS 1862, LKQCP Ire. 1864
Brodhurst, B. E., MRCS 1844, FRCS 1859
Brodribb, W. P., MRCS 1823, LSA 1822
Bromley, F., MRCS 1853, LSA 1854
Brook, C., MRCS, LSA 1861
Brooke, C., FRS, MA Cantab. 1827, FRCS 1844
Brookman, H., MRCS 1820, LRCP 1842
Broughton, R. D., MRCS, LSA 1865
Brown, F. W., MRCS 1861
Brown, J. H., MRCS, LSA 1862
Brown, J. A., MRCS, LSA 1851
Brown, J. M., MRCS 1851
Brown, J. W., MRCS, LSA 1861
Brown, R. G., MRCS, LSA 1854
Brown, N. B., LSA 1829, MRCS 1830
Browne, B., LSA 1832, LRCP Edin. 1859
Browne, F. H., MRCS, LSA 1860
Brownless, A. C., MRCS 1841
Bruce, J., MRCS 1863
Brunskill, W., LSA 1823
Brush, J. R., MD Heid. 1839, FRCS, MRCP 1860
Bryan, E., LSA 1863, MRCS 1864
Bryan, J. M., MRCS 1833, MD Aber. 1852
Bryant, W. H., MRCS 1864
Buchanan, W., LSA 1814, MD Erlang. 1841
Buck, H. J., MRCS 1859, LRCP Edin. 1860

Buckle, F., MD St. And. 1862, LRCP 1863
Buckley, N., FLS, MRCS 1842, MD St. And. 1843
Bucknill, H. W., MRCS, LSA 1851
Bullen, G., senr., MRCS 1813, FRCS 1844
Bullen, G., junr., MRCS 1845, LSA 1849
Bullmore, F. C., LSA 1830, MRCS 1831
Bullocke, J. J. A., LSA 1824, MD New York 1825
Bulmer, W., LSA 1828, MRCS 1829
Bunn, W. P., MRCS, LSA 1819
Burd, E., MB 1851, MD Cantab. 1859
Burgess, J. MRCS 1843, MD Aber. 1847
Burlton, T., LSA 1834, FRCS 1854
Burman, T. S., LSA 1836, MRCS 1838
Burn, W. B., MRCS 1865
Burroughs, J. B., LSA 1827, MRCS 1828
Burrows, G., FRS, MD Cantab. 1831, FRCP 1832
Burrows, W. A., LSA 1852, MRCS 1859
Burt, G. R., LSA 1835, LRCP Edin. 1860
Burt, S. J., MRCS 1830, LSA 1853
Burton, B. T., MRCS 1839, LSA 1840
Bury, J. W., MRCS 1858, MD Giessen 1865
Bush, E., LSA 1828, MRCS 1829
Bushnan, J. S., LRCS 1830, MD Heid. 1836
Buswell, R., LSA 1853, MRCS 1854
Butler, W. H., MRCS 1829
Butler, T. M., MRCS, LSA 1860
Butlin, C. H., MRCS 1863
Button, B., MRCS, LSA 1847

Caird, T. W., MRCS 1850, LSA 1851
Caird, W., LSA 1825, MRCS 1826
Callaway, E., MRCS 1858, LSA 1861
Callaway, Rev. H., MRCS 1842, MD Aber. 1853
Callender, G. W., MRCS 1852, FRCS 1855
Cam, T., MRCS 1837, FRCS 1844
Camden, G. J., LSA 1825, MRCS 1826
Campion, H., MRCS 1856
Candy, J., LSA 1827, MD Aber. 1860
Cann, T., MRCS 1859, MD St. And. 1860
Cape, L., MD Edin. 1833, FRCP Lond. 1857
Carden, H. D., MRCS 1829, FRCS 1843
Carter, A. P., MRCS 1858, LSA 1859
Carter, D. D., LSA 1830, MRCS 1831
Carter, J., LSA 1836
Cartwright, J. T., MRCS 1832, LSA 1834
Cary, W. H., LSA 1823, MRCS 1825
Cattell, T. W., MRCS, LSA 1831
Cattlin, W. junr., MRCS 1859, LDS 1860
Cautley, J., MRCS 1852, MD St. And. 1852
Cautley, W. C., MRCS, LSA 1849
Chadwick, C, MD Edin. 1837, MRCP Lond. 1859
Chadwick, P. C., MRCS, LSA 1835
Chamberlaine, J., LSA 1852, MRCS 1860
Chance, F., MB Camb. 1855, FRCP Lond. 1863
Chapman, E. G., MRCS 1848, LSA 1849
Chapman, G., LSA 1822, MRCS 1823
Chapman, H. T., MRCS 1828, FRCS 1843
Chapman, T., LSA 1829, MRCS 1840
Chappell, J. J., MRCS 1857, MD St. And. 1859
Charlton, E., MD Edin. 1836, MD Durham 1856
Chatto, J., MRCS, LSA 1832
Chavasse, T., LSA 1822, FRCS 1844
Cheese, J., MRCS 1860
Chippendale, W., MRCS 1855, MD St. And. 1862
Cholmeley, W., MD St. And. 1850, MRCP 1859
Cholmondeley, J., MRCS 1816
Chune, H. C., MRCS 1860
Church, W. J., LSA 1821, FRCS 1852
Church, W. S., MB Oxon. 1864, MRCP 1864
Churchill, S., MRCS 1848, MD Aber. 1849
Clark, A., MRCS 1846, LSA 1850
Clark, J. E., LSA 1822, MRCS 1830
Clark, J. L, LSA 1835, MRCS 1854
Clark, R. O., MRCS, LSA 1848
Clarke, E. G., MRCS 1861
Clarke, J. S., LSA 1833, MD King's Coll. Aber. 1861
Clarke, W. M., MRCS, LSA 1850
Clifton, N. H., LSA 1841, FRCS 1856
Close, H. A., MRCS 1859, LRCP 1863
Clubbe, W. H., MRCS, LSA 1850
Coalbank, T., MRCS, LSA 1866
Coates, G., LSA 1827
Coates, G. V., LSA 1852, MRCS 1854
Coates, M. G. W., LSA 1836, MRCS 1838
Coates, W. M., LSA 1832, MRCS 1833
Coats, G. A. A., MRCS 1864
Coathupe, C., MRCS 1862
Cocker, J., LSA 1827, MD Erlang. 1845, FRCP Edin. 1858
Cocker, W. H., MRCS 1861, LSA 1862
Cockey, E., MRCS, LSA 1842
Cole, T., MRCS 1866
Coleman, A., MRCS, LDS 1860
Coleridge, E., MRCS 1826
Collins, C. H., LSA 1845, MRCS 1846
Collins, J. B., MRCS, LSA 1860
Collyns, G. N., MRCS, LSA 1855
Collyns, J. B., MRCS 1845, LSA 1846
Collyns, W., LSA 1844, MRCS 1845
Colquhoun, A. G., MRCS 1861
Colthurst, J., LSA 1831, FRCS 1844
Combe, H., MRCS 1808
Compton, T. A., MRCS 1864, LRCP 1865
Cooke, A., LSA 1832, MD Albany 1833
Cooke, A. S., LSA 1863, MRCS 1864
Cooke, W., MRCP 1866, MD St. And. 1822
Cooke, W., MRCS 1822, LM 1860
Cooke, W. R., MRCS 1838, LSA 1839
Cooke, W. H., MRCS 1855, MD Aber. 1860
Coombs, J., MRCS 1859, MD Erlang. 1864
Cooper, A., MRCS 1861
Cooper, G. L., MRCS 1834, FRCS 1843
Cooper, Sir H., MRCS 1830, MD Lond. 1841
Cooper, F. S., LSA 1839, FRCS 1854
Cooper, W. W., MRCS 1838, FRCS 1846
Coote, H., MRCS 1838, FRCS 1844
Cope, R., MRCS 1857
Copeland, G. F., MRCS 1832
Corfe, G., LSA 1831, MD Aber. 1849

Cornish, S. H., MRCS 1854, LSA 1856
Cornwall, J., MRCS 1840, LSA 1841
Corsellis, N. C., LSA 1820, MRCS 1831
Costall, J., MRCS 1816
Costerton, H., MRCS 1852, LSA 1857
Cotterell, T. R., MRCS 1837, LSA 1845
Cotterell, T. S., MRCS 1833, LSA 1834
Cotton, T., MRCS 1816, MD St. And. 1861
Coucher, M. S., MRCS 1855, MD St. And. 1856
Courtney, C. F. A., MRCS, LSA 1847
Coveney, J. H., MRCS, LSA 1843
Covey, C. E., MRCS 1868, LSA 1865
Covey, G., MRCS 1830
Covey, G., jun., MRCS 1864, LSA 1865
Covey, W. H., MRCS 1826, FRCS 1852
Cowen, H. L., MRCS 1839, FRCS 1859
Craven, R. M., MRCS 1846, LSA 1847
Crawford, D., MRCS 1820
Cregeen, J. J., LSA 1851, MD St. And. 1853
Cresswell, J., MRCS, LSA 1841
Cripps, E., MRCS 1845, LSA 1846
Croft, J. Mc G., MRCS 1846, MD Aber. 1856
Croft, R.C., MRCS 1853, LRCP Edin. 1859
Crompton, S., MRCS 1833, MD St. And. 1862
Crookes, J. F., MRCS 1833, FRCS 1843
Cropp, F. J., MRCS 1864, LRCP Edin. 1864
Crosby, T., MRCS 1831, LSA 1832
Cross, W., LSA 1837, FRCS 1852
Crosse, T. W., LSA 1847, FRCS 1860
Crowfoot, W. E., LSA 1828, FRCS 1845
Crowfoot, W. J., MRCS 1813, MD Leyden 1830
Crowfoot, W. M., MRCS 1858, MB Lond. 1859
Cuddeford, T., MRCS, LSA 1865
Cuff, R., LSA 1852, MRCS 1856
Culling, R., MRCS 1843, LSA 1860
Cupiss, F. P., LSA 1850, MRCS 1851
Curgenven, J. B., MRCS, LSA 1852
Curling, T. B., FRS, FRCS 1843
Currie, J. L., MRCS 1863, LRCP Lond. 1864
Currie, J. T., LFPS Glas. 1838
Curtis, A., MRCS 1857, LSA 1860
Curtis, C. E., MRCS 1865
Curtis, F., MRCS, LSA 1822
Curtis, W., LSA 1824, MRCS 1825
Cutcliffe, J. L., MRCS, LSA 1847

Daglish, G., LSA 1826, FRCS 1853
Dain, F. R., MRCS 1839, LSA 1843
Dalley, C. T., MRCS 1864, LSA 1865
Dakeyne, B. B., MRCS 1833, LSA 1834
Dalley, W. C., LSA 1823
Dalley, W. C., jun., MRCS, LSA 1855
Daly, O., FRCSI 1844, MD Dublin 1857
Dandy, C., MRCS 1813
Danford, F., MRCS, LSA 1838
Daniel, J. S., LSA 1825, FRCS 1852
Daniel, T. P., MRCS 1858, LSA 1859
Daniel, W. J., MRCS, LSA 1856
Daniel, W., LSA 1861, MRCS 1862
Daniell, A. H., MRCS 1848, LSA 1850
Daniell, W., LSA 1838, MRCS 1840
Daniell, W. C., MRCS 1856, LSA 1857
Dansey, G. F., MRCS 1861
Dashwood, W. H., MRCS 1858
Daubeny, C. G. B., FRS, MD Oxon.
Davenport, C., MRCS 1852, LRCP Edin. 1860
Davey, A. G., MRCS 1860, MD St. And. 1862
Davey, J. G., LSA 1833, MD St. And. 1863
Davey, R. G., LSA 1830, MRCS 1858
Davey, R. S., LSA 1853, MD St. And. 1862
Davies, F., LSA 1834, FRCS 1852, MD St. And. 1862
Davies, H., MRCS, LSA 1860
Davies, H. H., MRCS, LSA 1858
Davies, J., MRCS 1819, MD Aber. 1850
Davies, J., MD Aber. 1845
Davies, N. E., MRCS, LSA 1866
Davies, R., MRCS 1816
Davies, T., LSA 1824, MRCS 1827
Davies, T., MRCS 1864
Davies, T. G. D., LSA 1852, MRCS 1853
Davies, T. H. W., MRCS 1864, LSA 1865
Davies, W., MRCS, LSA 1847
Davis, A. A., MRCS 1854, LRCP Edin. 1859
Davis, E., in practice prior to 1815
Davis, T., MRCS, LSA 1823
Davis, T., jun., MRCS 1855, MD Lond. 1861
Davy, J. W., LSA 1829
Dawson, J., LSA 1831, MRCS 1860
Dawson, R. H., MRCS, LSA 1861
Dawson, T., MRCS, LSA 1835
Dawson, W., LSA 1834, MRCS 1835
Dawson, W. H., MRCS, LSA 1860
Day, E. H., MB Cantab. 1856, MRCS 1862
Day, J. S., LSA 1841
Dayman, H., LSA 1832, FRCS 1854
Deane, J., MRCS 1834, MD Glas. 1854
Deane, R., LSA 1828
Dearden, J., MRCS, LSA 1856
Dearden, J., MRCS, LSA 1858
De Crespigney, E. C., MRCS 1845
Delagarde, J. L., MRCS 1855, MB Lond. 1857
Delagarde, P. C., MRCS 1819, FRCS 1843
Denne, T., MRCS 1857
Denny, C. J., LM Dub., MRCS 1864
Denny, J., MRCS 1857, LRCP Edin. 1860
Dew, E., MD Edin. 1831, MRCP 1831
Dewes, E., MRCS 1843, MD Glas. 1844
Diamond, H. W., LSA 1829, MD Kiel 1834
Dickin, J., FRCS 1843
Dickinson, T., LSA 1819
Dingley, W., MRCS, LSA 1850
Diver, T., LSA 1857, MD St. And. 1858
Dixon, C., LSA 1816, MRCS 1819
Dixon, T. G., MRCS 1841, MD St. And. 1853
Dobell, H. B., MRCS 1849, MD St. And. MRCP 1856
Dolton, W. B., MD Heild. MRCP 1859
Donald, J. R., LSA 1838, FRCS 1865
Douglas, R., LSA 1823, MRCS 1825
Dover, F., MRCS 1829, FRCS 1843
Dow, J., MRCS 1858, LSA 1857
Dowker, F. W., MRCS, LSA 1859
Dowling, T., LSA 1823, MRCS 1860
Drage, C., MRCS 1848, MD Aberd. 1857
Drake, A., LM Dub. 1848, MB Cantab. 1849

Druce, W., LSA 1857
Ducket, C. A., MRCS 1852, MD St. And. 1862
Duckworth, D., MD Edin. 1863, MRCP Lond. 1865
Dudley, J. G., MRCP 1859, MD Cantab. 1860
Dudley, W. L., MRCS 1846, MD St. And. FRCS 1860
Duff, W. H., MRCS, LSA 1838
Dunderdale, W., MRCS 1862, MD St. And. 1862
Dunn, A., MRCS 1862
Dunn, C. B. N., MRCS 1860
Dunn, G., MRCS, LSA 1848
Dunne, F. W., MRCS 1855, LSA 1856
Dyer, W. T., LSA 1833, MD St. And. 1859

Eagles, G. M., MRCS, LSA 1850
Eaton, F. J., MRCS 1855, LSA 1858
Eaton, J. C., MRCS, LSA 1866
Earle, E. S., MRCS 1856, FRCS 1860
Ebbage, LSA 1833, FRCS 1858
Eccles, A., MRCS 1843, FRCS 1858
Eccles, G. H., MRCS 1862, LRCP Edin. 1865
Eccles, J. H., MRCS, LSA 1839
Eccles, W. S., MRCS 1864, LSA 1865
Eck, V. F., MRCS 1864
Edginton, G. W., LSA 1818
Edkins, C., LSA 1832, MRCS 1833
Edlin, E. H., MRCS, LSA 1862
Edmunds, J., MRCS 1852, LSA 1852
Edwardes, C. L., MRCS 1856, LSA 1858
Edwardes, T., MRCS 1835, LSA 1837
Edwards, C. T., MRCS 1833
Edwards, H. N., MRCS 1864, LSA 1865
Edwards, D. P., MRCS 1828, FRCS 1860
Edwards, G. N., MD Cantab. 1859, FRCP 1863
Edwards, H. J., MRCS, LSA 1857
Edwards, W., MRCS, LSA 1861
Edye, S., MRCS 1859, LSA 1860
Eland, J. L., MRCS 1836
Eldershaw, F., MRCS 1860
Elkington, T., MRCS 1854, LRCP Edin. 1860
Elliot, R. L., MRCS 1862, LRCP 1863
Elliott, J., LSA 1826, MRCS 1827
Elliott, G., MRCS 1862
Elliott, W., LRCS Edin. 1815, MD 1828
Elliott, E., MRCS 1839, MD Aberd. 1852
Elliott, G. H., MRCS, LSA 1863
Elliott, J. R., MRCS 1862, LRQCPI 1863
Elliott, P. MRCS 1834
Ellis, H. D., MRCS 1861, LSA 1863
Ellis, T. S., MRCS, LSA 1861
Ellis, W. H., MRCS, LSA 1866
Ellison, J., MRCS 1840, MD Lond. 1845
Else, J. O., MRCS, LSA 1825
English, T., MRCS 1848, MD St. And. 1849
Evans, A., MRCS, LSA 1857
Evans, C., MRCS 1859
Evans, C. J., MRCS, LSA 1856
Evans, F. G., MRCS, LSA 1849
Evans, G., MRCS 1820
Evans, G. F., MD Cantab. 1839, FRCP 1859
Evans, H. N., MRCS 1860, MB Oxon. 1862
Evans, H. N., MD Glas. 1824, FRCS 1845
Evans, J. T., MRCS 1864, MD Aberd. 1865
Evans, N., MRCS 1859, MD St. And. 1859
Evans, O., MRCS 1827, MD Erlang. 1839
Evans, S. H., LSA, MRCS 1834
Evans, T., MRCS 1842, LSA 1844
Evans, T., MRCS 1837, LSA 1838
Evans, T., MRCS, LSA 1837
Evans, T. G., LSA 1823, MRCS 1825
Evans, T. M., MRCS, LSA 1861
Evans, W. L., MRCS 1862
Evers, C., MRCS 1861, MD St. And. 1862
Exell, E. MRCS 1866
Eyles, R. S., MSA 1812

Fairbank, T., MRCS 1864, MD Lond. 1865
Fairless, H. W., MRCS 1864
Faithorn, G., MRCS, LSA 1830
Falconer, R. W., MRCP 1860, MD Edin. 1859
Fall, J., MRCS 1864
Falwasser, F., MRCS 1858, FRCS 1866
Farncombe, W., MRCS 1852
Farr, J., in practice prior to 1815
Farrar, C., LFPS Glas. 1854, MD Heidl. 1854
Farre, A., FRS, MB 1833, MD Cantab. FRCP 1843
Farre, F. J., MD Cantab. 1837, FRCP 1838
Farrington, A. C., MRCS 1865
Farrington, W. H., MRCS 1859, MD St. And. 1862
Farwell, A., MRCS 1862
Farwell, J., MRCS, LSA 1829
Favell, W. F., MRCS, LSA 1853
Fenn, F., MRCS, LSA 1861
Fenn, T. H., MRCS, LSA 1838
Fennell, T., MRCS 1865, LSA 1866
Fenton, H., MRCS, LSA 1844
Fereday, T., LSA 1827, FRCS 1845
Ferris, G. T., MRCS 1837
Ferguson, G., MRCS 1857, MB Lond. 1861
Finch, J. E. M., MRCS 1865, LSA 1866
Firth, G. W. W., LSA 1835, FRCS 1845
Fish, J. C., MB Cantab. 1861
Fisher, F., MRCS 1856
Fitzgerald, C. E., MRCS 1856, MD St. And. 1862
Fletcher, M., LSA 1817, MRCS 1818
Folliott, J., MRCS 1863, LSA 1865
Foote, C. N., MRCS 1849, MD St. And. 1862
Foquett, H. R., MRCS, LSA 1852
Ford, B. B., MRCS 1860
Forder, J., MRCS 1818
Forshall, F. H., MRCS 1855, LRCP 1863
Forster, J. E., MRCS 1850
Fortescue, W., MRCS 1837, FRCS 1852
Foster, W. F., MRCS, LSA 1857
Fowke, F. G., LSA 1836
Fowler, O., MRCS 1835, LSA 1836
Fowler, O., MRCS 1863
Fowler, W., MRCS 1818
Fox, D., LSA 1821, FRCS 1843
Fox, G. F., MRCS 1843, LSA 1846
Fox, L. O., LSA 1833, MD St. And. 1859
France, W. H., MRCS 1860, LSA 1861

Francis, J., LSA 1834, MRCS 1835
Francis, M., MRCS 1846, LSA 1852
Franco, M. R. F., LSA 1832
Franey, E., MRCS, LSA 1858
Franklin, A. W., MRCS 1836, LSA 1837
Freeman, J., LSA 1839, MD Erlang. 1841
Freeman, R. T., MRCS 1864, LRCP Edin. 1865
Freeman, S., LSA 1825, MRCS 1827
Freeman, T. A., LSA 1836, MRCS 1859
Freer, A., MRCS 1850, LSA 1852
Freer, G. R. V., LSA 1835, MRCS 1842
Freer, R. L., MRCS 1839, LSA 1840
Freer, T., LSA 1822, MRCS 1828
Freer, W. C., LSA 1840, MRCS 1841
Fryer, T. W., MRCS 1854, LSA 1856
Fulcher, G. A., MRCS 1853, LSA 1853
Fullerton, J. C., LSA 1828
Furnell, M. C., MRCS 1850
Furley, E., LSA 1834, MD St. And. 1856
Furnivall, E. T., MRCS 1855

Gabb, A. W., MRCS 1843, LSA 1844
Gabb, D. H., MRCS 1845, LSA 1846
Gabb, F. E., MRCS, LSA 1862
Galton, J. C., MRCS 1866
Galton, R. C., MB Cantab. 1856, MRCP 1857
Gardiner, G.G., MRCS 1857, MD St. And. 1862
Gardner, J. T., LSA 1823, MRCS 1824
Gardner, S., MRCS 1861
Garlike, S. W. R., MRCS 1863
Garlike, T. W., MRCS 1839, FRCS 1858
Garlick, W., MRCS 1864
Garrington, W. H., LSA 1826, MRCS 1845
Gaskell, R. A., MRCS, LSA 1849
Gatehouse, C., MRCS 1821, LSA 1819
Gatty, W. H., MRCS, LSA 1851
Gaved, A., MRCS 1846
Gay, J., MRCS 1811
Gay, J., MRCS 1841, LSA 1842
Gay, J., MRCS 1834, FRCS 1843
Gaye, C., MRCS 1819, LSA 1818
Gaye, H. S., MRCS, LSA 1850
Gaye, W., LSA 1818
Gayleard, J., LSA 1833, MRCS 1834
George, H., LSA 1850, MD St. And. 1862
George, H., LSA 1834
George, R. F., MRCS, LSA 1820
Geoghegan, R. T., MRCS 1863, LM Dublin
German, J., MRCS, LSA 1849
Gerrans, J. G., LSA 1831, MRCS 1832
Gibb, G. D., MD Montreal 1846, MRCP 1859
Gibbs, J. H., MRCS 1835, MD Aberdeen 1850
Gibbon, S., MB Cantab. 1851, MRCP 1852
Gibson, G., MRCS, LSA 1835
Gilbertson, J. B., MRCS 1849, MD Aber. 1859
Gilbertson, R., MRCS 1840, LSA 1841
Giles, W. F., MRCS 1858, LSA 1860
Gillard, F. J., MRCS 1837, LSA 1838
Gillott, E. D., LSA 1830, MRCS 1831
Gimson, W.G., MRCS 1860, MD St. And. 1862
Girdlestone, T. M., FRCS 1849
Girdlestone, W. T., MRCS 1856, LSA 1857
Gisborne, H. F., MRCS 1827
Glasier, G. W., MRCS 1859, LSA 1860
Glynn, T. R., MRCS 1863
Goddard, L. M., LSA 1832, MRCS 1843
Goddard, R. C., MRCS 1864
Godfray, C. Le V., MRCS, LSA 1860
Godfrey, R., MRCS 1813
Godrich, F., LSA 1818, MRCS 1819
Godwin, A., MRCS 1855, MD St. And. 1862
Gooch, W. H., MRCS 1834, MD Edin. 1832
Goodacre, F. B., MB 1853, MD Cantab. 1860
Goodall, J., MRCS 1866
Goodall, R., MRCS 1857, LSA 1859
Goodeve, H. H., MRCS 1828, FRCP 1860
Goodfellow, S. J., MD Lond. 1840, FRCP 1849
Goodwin, C., LSA 1834, MRCS 1845
Goodwin, J. W., MB 1849, MD Cantab. 1854
Goodwin, J. H., LSA 1836
Gore, R. T., LSA 1820, FRCS 1843
Gorham, R. V., LSA 1836, MRCS 1837
Goude, J. F., LSA 1837, MRCS 1838
Gover, R. M., MRCS 1856, LSA 1859
Gowar, F. R., MSA 1814
Graham, A. R., MC Cantab. 1863, MB 1864
Graham, T. H., MRCS 1841, LSA 1849
Graham, T. J., MRCS 1814, MD Glas. FRCP Edin. 1861
Grammer, S. R., MRCS 1850, LSA 1857
Grant, K., MD Edin. 1826, MRCS 1829
Gray, Charles. MRCS 1858
Gray, E. B., MRCS 1856, MD Oxon. 1861
Gray, T. S., MRCS 1855, MD St. And. 1857
Gray, W., MRCS 1855
Grayling, J., MRCS 1832, MD Aberdeen 1851
Greatrex, A. B. W., LSA 1852. MRCS 1855
Greatrex, A. B., LSA 1862, MD St. And. 1862
Greaves, W. T., MRCS 1831
Greeves, J., LSA 1833
Green, T., MD Edin. 1824, FRCS 1843
Green, J. C., MRCS, LSA 1834
Green, T., LSA 1822, MRCS 1823
Greenhill, J. R., MRCS 1860, FRCS 1864
Gregory, B., MRCS, LSA 1859
Grellett, C. J., MRCS, LSA 1864
Griffin, F. C. G., MRCS, MD Oxon. 1864
Griffin, R., LSA 1826, MRCS 1830
Griffith, S. C., MRCS 1864, MD St. And. 1847
Griffith, J. W., MRCS 1841, MD St. And. 1842
Griffith, T. T., LSA 1816, FRCS 1844
Griffiths, G. H., LSA 1849, MD St. And. 1862
Griffiths, W. H., MRCS 1861, LSA 1862
Grigg, N. B., MRCS 1860
Grime, H. A., MRCS 1840, LRCP Edin. 1860
Gross, J. E., MRCS 1857
Growse, J., MRCS 1843, LSA 1845
Gulliver, G., FRCS 1843
Guppy, T. S., MRCS 1849, MD Aberd. 1859
Gutteridge, T., LSA 1826, MRCS 1827
Gwillin, W., LSA 1819, MD Erlang. 1831

Haffenden, T., MRCS 1807
Haig, J. B., MRCS 1842
Hall, E., MRCS, LSA 1846
Hall, E. F., MRCS 1858, MD St. And. 1860
Hall, F. R., MRCS 1852, LSA 1853

Hall, H. J., MRCS 1858
Hall, S., MRCS, LSA 1863
Hall, T. W., LSA 1839
Hall, W., LSA 1820
Hall, W., LSA 1840, FRCS 1862
Hall, W., LSA 1834, MRCS 1835
Hallowes, F. B., MRCS 1858, LSA 1860
Hallowes, P. B., LSA 1822, FRCS 1843
Hammond, A., LSA 1829, MRCS 1832
Hammond, C. C., LSA 1818, MRCS 1819
Hammond, E. C., MRCS 1860, LSA 1863
Hammond, F., MRCS 1860
Hammond, J., LSA 1832, FRCS 1843
Hanbury, C., jun., MRCS 1849, LSA 1850
Hands, B., LSA 1819, FRCS 1853
Hanslip, T., LSA, 1853, MRCS 1859
Harbroe, E., LSA 1829, MRCS 1830
Harcourt, G., LSA 1821, FRCS 1841, MD St. And. 1846
Hardey, K., MRCS 1851
Harding, J. F., LSA 1835, FRCS 1852
Harding, W. W., MRCS 1855
Hardwicke, E. J., MRCS 1866
Hardwicke, H., MRCS 1840, LSA 1841
Harington, J. H., MRCS 1861, MB Oxon. 1862
Harper, T., LSA 1837, MRCS 1838
Harries, C. A., LSA 1829, MRCS 1830
Harris, A., MRCS 1862
Harris, B., MRCS 1860, LSA 1861
Harris, F., MB 1854, MD Cantab. 1859
Harris, J. S., MRCS 1863, LKQCP Irel. 1864
Harris, R. D., MRCS 1847, LSA 1856
Harris, S., MRCS 1854, LSA 1855
Harris, T., MRCS 1815
Harris, W., LSA 1831, FRCS 1853
Harris, W. J., MRCS 1859, LSA 1860
Harrison, C., MRCS 1860, MD St. And. 1861
Harrison, C. H. R., MRCS 1834, FRCS 1843
Harrison, E. P. D, MRCS 1842, LSA 1843
Harrison, R., MRCS, LSA 1859
Harsant, M., MRCS, LSA 1837
Hartill, J., LSA 1826, MRCS 1856
Hartill, W. H., LSA 1860, MRCS 1861
Hartley, J., MRCS, LSA 1852
Harston, A. D., LSA 1839, FRCS 1859
Harvey, O., MRCS 1860
Harvey, W., MRCS, LSA 1834
Harvey, W., MRCS 1864
Haslam, G. H., MRCS, LSA 1861
Hatherly, N. C., MRCS 1845, MD Aberd. 1857
Havers, J., MRCS 1837, FRCS 1849
Haviland, H. J., MB 1849, MD Cantab. 1854
Haward, F. R., MRCS, LSA 1865
Haward, W., MRCS 1854
Hawett, T., MRCS, LSA 1822
Hawkes, J., MRCS 1853, MD St. And. 1861
Hawkins, C. J., LSA 1836, FRCS 1854
Hawthorn, F., LSA 1836, MRCS 1842
Hawthorn, H. O., LSA 1831, MRCS 1842
Haycock, G., MRCS 1862, LSA 1864
Haydon, N. J., MRCS 1837, MD St. And. 1860
Hayes, H., MRCS, LSA 1842
Hayman, H., MRCS 1834, FRCS 1858
Haynes, F. H., MRCS 1866
Haynes, R. L., LSA 1841, FRCS 1849
Hayward, J., MRCS, LSA 1845
Hazard, J., MRCS 1852, LSA 1857
Head, R. T., MRCS, LSA 1859
Head, T., MRCS 1822, MD St. And. 1845
Headley, W. B., MB & MC Cantab. 1864
Heatley, J., MRCS 1839
Heaton, C. L., LSA 1837, MRCS 1838
Heaton, F. L., MRCS 1862, MB Oxon. 1863
Hedges, J. A., MRCS, LSA 1862
Heelas, M. L., MRCS 1866
Hele, H. H., LSA 1825, MRCS 1826
Helm, G. F., MRCS 1859, FRCS 1863
Hemborough, J. W., LSA 1865, MRCS 1866
Henderson, H. G., MRCS 1863
Henderson, W., MD Aber. 1857, MRCS 1849
Henry, M., MRCS 1847, FRCS 1854
Hepworth, G. A. H., MRCS, LSA 1847
Hester, J., MRCS 1853
Hester, J. T., LSA 1821, FRCS 1852
Hewer, J. H , MRCS, LSA 1852
Hewlett, C., MRCS 1853
Hewlett, T., MRCS, LSA 1858
Hews, F., in practice prior to 1815
Hickens, G. L., MRCS 1854
Hichens, J. S., MRCS, LSA 1851
Higgins, H., LM 1857, LRCP Edin. 1860
Hilder, H. H., LSA 1824, MRCS 1826
Hill, R. S., MRCS, LSA 1840
Hill, T. J., MRCS 1858, LSA 1859
Hill, W., MRCS, LSA 1842
Hillier, J. T., MRCS 1850, LM 1853
Hilton, C. S., LSA 1862, MD St. And. 1862
Hine, S. D., MRCS, LSA 1856
Hine, W. C., MRCS 1861, LSA 1862
Hingston, W., LSA 1824, MRCS 1825
Hingston, C. A., MB Lond. 1864
Hinton, J., MRCS 1848
Hitch, S., MRCS 1822, MRCP 1859
Hitchcock, C., MRCS 1834, LRCP Edin. 1860
Hitchcock, T., MRCS 1831, MRCP 1859
Hitchins, C. V., LSA 1854, MRCS 1855
Hoare, W., LSA 1826, MRCS 1827
Hoare, W., MRCS 1858
Hodges, F. S., LSA 1845, MRCS 1847
Hodgson, J., FRS, FRCS 1843
Hodgson, W., MRCS 1855, MD St. And. 1862
Hodson, C. F., MRCS 1837, LSA 1838
Holbrow, A., LSA 1818, FRCS 1862
Hole, J., LSA 1826, MRCS 1828
Hole, J. C , LSA 1822
Holden, G., MRCS 1856, MD St. And. 1859
Holden, L., MRCS 1838, FRCS 1844
Hollis, W. P., MRCS 1861, LM 1863
Holmes, A., MRCS, LRCSJ 1858
Holmes, C., LSA 1838, FRCS 1861
Holthouse, C., LSA 1832, FRCS 1843
Holttum, —, LSA 1840, FRCS 1855
Hooper, C., MRCS 1859
Hooper, J. D., LSA 1830

Hooper, J. T., MRCS 1845, LSA 1858
Hooper, L. G., MRCS 1852, LSA 1854
Hope, H., MRCS, LSA 1864
Hore, H. A., MRCS, LSA 1844
Hore, W., LSA 1826, MRCS 1858
Horne, T. B., MRCS 1845, LSA 1846
Horsfall, J., MRCS 1866
Hosking, R., MRCS, LSA 1852
Hoskins, E. J., MRCS 1858, MD St. And. 1860
Hoskins, H. R., LSA 1848, LRCP 1860
Houghton, J. H., MRCS, LSA 1838
Houseman, —, MRCS, LSA 1863
Howell, H. S., MRCS 1861, MD St. And. 1862
Hue, J. W., FRCP 1856, MD Cantab.
Hughes, D. W., MRCS 1861, LSA 1862
Hughes, J. B., MRCS 1866
Hughes, H. H., MRCS, LSA 1831
Hughes, H. R., MRCS 1839
Hughes, J., LSA 1840, FRCS 1858
Hughes, J. E., LSA 1861, MRCS 1862
Hughes, T. H., LSA 1858, MRCS 1858
Hughes, W. E., MRCS 1847, LSA 1857
Hughes, W. F., MRCS 1863
Hulme, E. C., MRCS 1844, FRCS 1849
Hulme, J. D., MRCS 1849, MD St. And. 1862
Humphry, A., MRCS, LSA 1866
Humphry, C. H., MRCS, LSA 1862
Humphry, F. A., LSA 1853, FRCS 1856
Humphry, G. M., FRS, LSA 1842, MD Cantab. 1859
Hunt, E., LSA 1830
Hunt, H., MRCS 1840, FRCP
Hunt, T. O., MRCS 1856
Hunt, W. C., LSA 1844, MRCS 1845
Hunter, J. C., MRCS 1821, LRCP 1863
Hunter, R. H., MRCS 1861, LSA 1862
Hunter, T. D., MRCS 1825, LSA 1832
Hurlock, Rev. J., MD 1816
Husband, C., MRCS 1845, LSA 1846
Husband, H. A., MRCS 1866
Hussey, E. L., FRCS 1849, MRCP Edin. 1859
Hutchins, W., LSA 1825, MRCS 1826
Hutchinson, F., LSA 1828, FRCS 1852
Hutchinson, J., MRCS 1850, FRCS 1862
Hutchinson, J. R., MRCS 1845, LSA 1855
Hutchinson, T., MRCS 1849, LRCP Edin. 1860
Hutchinson, W. B., MRCS 1829, FRCS 1852

Iles, A., MD St. And. 1861
Ilott, E., LSA 1848, FRCS 1863
Ilott, J. W., LSA 1836, MRCS 1837
Ingman, J., MRCS 1847
Ingram, C. P., MB Cantab. 1856, MD Cantab. 1860
Ingram, W. G. L., MRCS, LSA 1852
Irving, W., LSA 1830, FRCS 1852
Ireland, J., LSA 1853

Jebell, E. J., MRCS 1841
Jackman, T. S. H., MRCS 1848
Jackson, A., MRCS 1866
Jackson, H. W., MRCS 1855, LSA 1857
Jackson, J. H., MRCS 1856, MD St. And. 1860
Jackson, J. T., LSA 1841, MRCS 1842
Jackson, M., MRCS, LSA 1827
Jackson, M, MRCS, LSA 1866
Jackson, P., MRCS, LSA 1844
Jackson, R., MRCS 1832
Jackson, W., MRCS 1814, FRCS 1853
Jacob, E. L., MRCS, LSA 1855
Jacques, J. T., MRCS, LSA 1865
Jallard, R., MRCS 1854
James, E., MRCS, LSA 1853
James, E., MD Edin. 1835
James, E. M., MRCS 1860
James, H. H., LSA 1841, FRCS 1856
James, J., MRCS 1834, LSA 1862
James, J., MRCS 1862
James, J. D., MRCS, LSA 1857
James, J. H., MRCS 1811, FRCS 1843
James, T., MRCS, LSA 1836
James, T., MRCS, LSA 1846
Jeaffreson, C., MRCS 1863
Jeaffreson, H., MD Cantab. 1838, FRCP 1839
Jeaffreson, H., MRCS 1860, MD Lond. 1865
Jeaffreson, J., MRCS 1862
Jeaffreson, J. B., MRCS, LSA 1858
Jeaffreson, W., MRCS 1820
Jefferys, W. E., LSA 1848, MRCS 1849
Jenneret, H., LSA 1824, MD Edin. 1825
Jenkins, H. J., MRCS, LSA 1829
Jenkyns, C., MRCS 1861, LSA 1862
Jepson, E. C., MRCS 1835, FRCS 1855
Jerrard, J. C., MRCS 1818, LSA 1819
Jeston, A. F. W., MRCS, LSA 1824
Johnson, J. G., LSA 1820, FRCS 1843
Johnson, T. R. R., MRCS 1851, LSA 1854
Johnston, J., MRCS, LSA 1823
Johnstone, J., MD Cantab. 1832, FRCP 1834
Jones, A. B., MRCS 1848, LDS 1863
Jones, D., MRCS 1851, LRCP Edin. 1860
Jones, G. T., MRCS 1842, LRCP Edin. 1859
Jones, H., jun., MRCS 1853, LSA 1855
Jones, H. D., MRCS 1835, FRCS 1852
Jones, H. P., MRCS, LSA 1834
Jones, J., MRCS 1834, LSA 1838
Jones, J., MRCS, LSA 1862
Jones, M., MRCS 1855
Jones, M., MRCS 1856
Jones, M., MRCS 1861, LRCP 1861
Jones, O., MRCS 1862
Jones, R. O., MRCS, LSA 1861
Jones, R., MRCS, LSA 1836
Jones, R. A., MRCS 1864, LSA 1865
Jones, T., MRCS 1841
Jones, T., MRCS, LSA 1860
Jones, T. E., MRCS, LSA 1856
Jones, W., MRCS 1841, MD St. And. 1856
Jones, W., MRCS 1863
Jotham, G., LSA 1826, MRCS 1827
Jowers, F. W., MRCS 1854, LSA 1855
Julian, J. P., MRCS 1852

Karkeek, P. Q., MRCS, LSA 1865
Kay, J., LSA 1840, MRCS 1848

Keal, W., MRCS, LSA 1854
Kelly, J., MRCS, LSA 1833
Kempe, A., LSA 1838, FRCS 1861
Kesteven, W. B., LSA 1837, FRCS 1854
Kiernan, F., FRS, MRCS 1825, FRCS 1843
Kiernander, W., MRCS 1864
King, E. P., MRCS 1858, LSA 1859
King, F., MRCS 1848
King, R., MRCS 1844
Kingdon, J. A., MRCS 1849, FRCS 1861
Kinsey, R. H., MRCS 1864
Kipling, W., LSA 1827
Kirkman, J. M., LRCS Edin. 1833, MRCS 1835
Kitching, A., MRCS 1850, MD Aber. 1860
Kite, W. J., MRCS, LSA 1846
Knapp, F. W., MRCS 1864
Knight G. T., LSA 1847, MRCS 1848
Knight, H. J., MRCS, LSA 1858
Knott, T. B., MRCS, LSA 1848
Kough, O'B., LSA 1860, MRCS 1861

La Farque, P. A., MRCS 1847, LSA 1854
Laity, R. J., MRCS 1837, LSA 1838
Lamb, B. W., MRCS, LSA 1866
Lancaster, W. J., MRCS, LSA 1860
Lanchester, H. T., MRCS 1861, MD Lond. 1864
Lanchester, T. W., LSA 1826, MD Erlang. 1839
Lang, W., MRCS, LSA 1832
Langdon, C. G., LSA 1866
Langdon, H. W., MRCS 1854, LSA 1855
Langdon, T. C., LSA 1858, FRCS 1863
Langford, E. C., LSA 1855, MRCS 1856
Langshaw, J. P., LSA 1837, FRCS 1854
Langton, J., MRCS 1861, FRCS 1865
Langworthy, G. V., MRCS, LSA 1864
Langworthy, J. M. B., LSA 1852, MRCS 1854
Langworthy, R., MRCS, LSA 1856
Langworthy, S., MRCS 1851, LSA 1852
Langworthy, W. F., jun., MRCS, LSA 1861
Lansdowne, J. G., LSA 1827, MRCS 1828
Lascelles, R. M., MRCS, LSA 1836
Latham, C., MRCS, LSA 1839
Latham, J., MRCS 1823, LRCP Edin. 1860
Latham, P. M., MD Oxon. 1816, FRCP 1818
Latham, P. W., MRCP 1861, MD Cantab. 1864
Latham, R. G., MD Cantab. FRS
Latten, R., LSA 1836, MRCS 1837
Lavery, T., LSA 1834, MRCS 1835
Lawrance, F., MRCS 1857, LSA 1859
Lawrence, G. R., MRCS 1862, LSA 1864
Lawrence, H. C., MRCS 1864, LRCP 1865
Lawrence, J. T., MRCS 1853
Lawrence, L. A., LSA 1839, MRCS 1848
Lawrence, R. M., MD Berl. MRCP 1849
Lawrence, W., MRCS 1805, FRS, FRCS 1843
Lawrence, W. F., MRCS 1841, LSA 1842
Lawton, J., LSA 1822, MRCS 1824
Lawton, R., LSA 1827
Laycock, W., MRCS 1837,
Leach, H., MRCS, LSA 1858
Leather, P. W., MRCS, LSA 1839
Ledgard, J. A., MRCS, LSA 1831
Ledsam, J. J., MRCS 1820, MD St. And. 1851
Lee, H. B., MRCS 1833
Lee, H., LSA 1830
Lee, J., LSA 1825
Lee, J., jun., MRCS 1858, LSA 1859
Lee, N. B. C., MRCS 1857
Lee, R., LSA 1825, MRCS 1848
Leech, E., MRCS, LSA 1822
Leech, H. P., MRCS 1848, LSA 1849
Leeds, T., MRCS, LSA 1861
Leggatt, R. S., MRCS 1841, LSA 1842
Leigh, P., LSA 1834, MRCS 1835
Lemon, H. M., LSA 1860, MD St. And. 1861
Leppington, H. M., LSA 1830, MRCS 1831
Lestourgeon, C., LSA 1831, FRCS 1843
Leverton, E. J., MRCS, LSA 1866
Leverton, H. S., MRCS 1851, LRCP Edin. 1860
Levy, J. L., MRCS 1866
Lewis, D. W., MRCS 1861
Lewis, R. B., MB Lond. 1850, MRCS 1852
Lewis, W. T., MRCS 1853, LSA 1863
Ley, W., MRCS 1831
Lindop, J. C., MRCS 1862, LSA 1863
Lindop, W., LSA 1829, FRCS 1856
Lindgard, A. R., MRCS 1839
Linneker, E. H., MRCS 1858, LSA 1859
Linnecar, E. H., LSA 1824, MRCS 1843
Little, J. C., MRCS 1839, LSA 1840
Little, T. S., MRCS 1842, LSA 1843
Livett, H. W., MRCS 1837, MD Pisa 1837, LRCP Edin. 1859
Llewellyn, E., MRCS 1859
Lloyd, D., MRCS, LSA 1838
Lloyd, D., MRCS, LSA 1859
Lloyd, F., MRCS 1859
Lloyd, J. A., MRCS 1829, MD Aberdeen 1854
Lobb, H., MRCS, LSA 1850
Locock, MD Edin. 1821, FRCP 1836
Lombe, T. R., MRCS 1843, MD Aberdeen 1856
Long, E., LSA 1823, MRCS 1824
Long, T., LSA 1833
Longhurst, A. E. T., MD St. And. 1862, MRCS 1863
Lovekin, L. F., LSA 1833, MRCS 1835
Lovedess, W. K., LSA 1830, MRCS 1831
Lovell, F. H., MRCS 1865
Lowdell, C., MRCS 1847
Lowdell, LSA 1835, FRCS 1852
Low, A. J., MRCS 1864, LSA 1866
Lowe, LSA 1837, FRCS 1863
Lowne, B. T., MRCS, LSA 1844
Lowne, B. T., jun., MRCS 1861
Ludlow, E., MRCS 1862, MB London 1864
Ludlow, T., MD St. And. 1863
Luke, H., MRCS 1858
Lush, W. G. V., MRCS 1864, MB Lond. 1865

Macaulay, F. E., MRCS 1861, LSA 1862
McCandlish, W., MRCS 1864
McCheane, W., MRCS 1843, LSA 1844
Macilwain, G., MRCS 1818, FRCS 1843
McKechnie, A., MD Glas. 1829, MRCP 1860
Mackenzie, G. W., MRCS 1848, LSA 1851

Mackenzie, J. T., MRCS 1858, MB Cantab. 1861
Mackinder, D., MRCS 1847, MD St. And. 1853
Mackintosh, H.R. D., MRCS 1863, MD Erlang. 1864
McLeod, A. W., LSA 1827, MRCS 1828
M'Nab, D. R., LSA 1841, MRCS 1842
McNicoll, D. H., LSA 1836, MD Glasgow 1842
Maling, E. H., LSA 1822, MRCS 1832
Mallet, S. E., MRCS 1861
Manby, A., LSA 1880, MRCS 1831
Manifold, W. H., MRCS 1849
Mann, R. M., MRCS, LSA 1845
Manning, J , LSA 1856, MRCS 1857
Mant, N., MRCS 1840, LRCP 1860
Marchant, R., MRCS 1815, LSA 1816
Margetson, P., MRCS, LSA 1835
Marrack, W., MRCS, LSA 1854
Marriott, J., MRCS 1850, LSA 1852
Marsh, A. J., LSA 1839, MRCS 1841
Marsh, F. H., MRCS 1861, FRCS 1866
Marsh, J., MRCS 1850, LSA 1856
Marshall, T. H., MRCS 1846, LSA 1849
Marson, J. T., LSA 1829, FRCS 1862
Martin, H. E., MRCS 1859
Martin, H. V., MRCS 1834, FRCS 1850
Martin, P., LSA 1863, MRCS 1864
Martin, Sir R., CB, FRS, FRCS 1843
Martin, R., MRCS 1813, FRCS 1844
Martin, R., MB 1851, FRCP 1859, MD 1859 Cantab.
Martyn, T. D., MRCS, LSA 1832
Maskelyne, H., LSA 1825, MRCS 1829
Mason, J. B., MRCS 1858, LRCP 1860
Mason, R., MRCS 1848, LSA 1856
Master, A., LSA 1837, MRCS 1838
Mathew, J. E., MRCS 1841, FRCS 1858
Mathias, J. E., MRCS 1838
Matterson, W., MRCS 1837, MD St. And. 1862
Matthews, J., LSA 1838, MD St. And. 1853
Maturin, H., MRCS 1864, LSA 1865
Maughan, J. B., MRCS 1840
Maxwell, E. C., MRCS 1866
May, A. S., LSA, MRCS 1864
May, E. C., LSA 1820, FRCS 1853
May, E. H., LSA 1854, FRCS 1856
May, J. H. S., MRCS, LSA 1859
May, J., LSA 1830, FRCS 1860
Mayne, R. F., MRCS 1854, MD Jena 1858
Mayo, C., MRCS 1861
Mayo, C., MRCS 1811, FRCS 1844
Mead, G. B., MRCS 1854, MD Giessen 1859, FRCP 1861
Meade, H., MRCS, LSA 1860
Meade, R. H., LSA 1835, FRCS 1845
Meldola, R., MRCS 1854, LSA 1860
Melhado, A. C. B., MRCS, LSA 1866
Meller, C. M., MRCS 1849, MD St. And. 1862
Menzies, J. J., MRCS 1859, LRCP Edin. 1860
Mercer, A. W., MRCS 1858, LSA 1859
Mercer, E., MRCS, LSA 1851
Merry, R. R., MRCS 1860, LRCP 1864
Metcalfe, E., MRCS 1841, FRCS 1859
Michell, G. A., MRCS, LSA 1835
Michell, S. V. P., LSA 1839, FRCS 1861
Mickle, D. K., LSA 1851, MRCS 1860
Middlemore, LSA 1825, FRCS 1843
Miles, C., MRCS, LSA 1848
Miles, H. C., MRCS 1854, LRCP Edin. 1859
Miles, J., LSA 1816, MD Erlang. 1837
Miller, C. M., MD St. And. 1862
Millington, LSA 1828
Milsome, J. R., MRCS 1862, MD St. And. 1862
Mitchell, S., LSA 1820, MRCS 1821
Mitchell, T. C., MRCS 1859, LSA 1860
Mitchinson, C. C., MRCS 1859, LSA 1860
Mitchinson, G., MRCS 1851, LRQCP Ireland 1859
Mitton, M. J., MRCS 1862
Moon, R., MRCS 1863
Monro, H., MD, MB Oxon. 1844, FRCP 1848
Montgomery, J. B., MD Glas. 1851, MRCS 1852
Moore, D. S., LSA 1841, MRCS 1842
Moore, E., MRCS, LSA 1851
Moore, E., LSA 1826, FRCS 1858
Moore, E. D., MRCS 1826, LRCP Edin. 1859
Moore, C. H., MRCS 1842, FRCS 1848
Moore, G. H., MRCS 1829, MD St. And. 1841
Moore, J., MRCS 1810
Moore, J., MRCS, LSA 1827
Moore, E., MD, LSA 1842, LRCP 1862
Moore, R. B., MRCS 1866
Moore, T., MRCS 1859, LRCP 1865
Moore, W., MRCS, LSA 1886
Moore, W. H., MRCS 1866
Moorhouse, J. W., MRCS 1852, LRCP Edin. 1864
Morris, E., MRCS 1854, LSA 1856
Morris, H., LSA 1831
Morris, R., MRCS 1857, LSA 1858
Morrish, T. F., MRCS, LSA 1861
Morse, A. C., MRCS 1844, LSA 1845
Mortimer, R., MRCS 1835, LRCP Edin. 1859
Moss, W. B., MRCS 1854
Mousley, G., LSA 1832, MRCS 1833
Moxon, J., MRCS 1857, LSA 1863
Mudge, B., MRCS 1863
Mudge, H., LSA 1828, MRCS 1829
Mullings, S. E., LSA 1854, LRCP E. 1860
Munday, C., MRCS 1847, LRCP Edin. 1859
Murrell, W., MRCS 1861
Muschamp, W. H , MRCS 1854
Myers, H. R., LM, LFPS 1859

Nash, W.L., MRCS 1863
Nathan, H. F., MRCS 1860
Neatby, T., MRCS 1861, MD St. And. 1861
Needham, F., MRCS 1858, MD St. And. 1862
Nelson, S., MRCS, LSA 1862
Ness, J., LSA 1826, FRCS 1860
New, H. S., MRCS 1835, LSA 1840
Newbold, E., MRCS 1835, LSA 1838
Newell, H. A., MRCS, LSA 1845
Newman, A., MRCS 1857, MB Oxon. 1859
Newman, G., MRCS 1835, LRCP Edin. 1859
Newman, H., MRCS 1841, LSA 1842

Newman, W., MRCS 1854, MD Lond. 1859
Newton, E., LSA 1838, FRCS 1846
Newton, L., MRCS, LSA 1839
Nicholls, L., MRCS 1856, LSA 1859
Nicholson, J., LSA 1839, FRCS 1857
Nicholson, W. H., MRCS 1858
Noad, G. W., MRCS 1862, MD St. And. 1862
Noble, T., LSA 1858, MD Aberdeen 1859
Norris, H., MRCS 1818, FRCS 1844
Norris, T. G., MRCS, LSA 1832
Norris, W., LSA 1816, MD St. And. 1823
North, W. D., MRCS 1862
Norton, W. A., MRCS 1862, MD St. And. 1862
Nunn, J. R., MRCS, LSA 1859

Oates, J. P., LSA 1829, MRCS 1842
Odell, T., MRCS, LSA 1851
Odling, W., FRS, MB Lond. 1851, FRCP 1859
Oldham, R., MRCS 1841, FRCS 1859
Oldman, J., MRCS, LSA 1858
Oliver, R., MRCS, LSA 1861
Oliver, W. H., MRCS, LSA 1836
Orford, W. C., MRCS 1847, LSA 1848
Oriel, F. H. C., LSA 1826, MRCS 1827
Ormerod, E. L., FRCP 1851, MD Cantab. 1851
Orton, G. H., MRCS 1863, LSA 1865
Orton, W., LSA 1832, MRCS 1833
Osborn, H., MRCS 1851, MRCP 1859
Osmond, T., MRCS, LSA 1863
Overton, J., LSA 1821, MRCS 1822
Owen, A. P., MRCS, LSA 1835
Owen, E. R., LSA 1834, FRCS 1857
Owen, R., FRS, MRCS 1826, FRCS 1843
Owen, R. J., MRCS, LSA 1861

Paget, G. E., MD Cantab. 1838, FRCP 1839
Paget, J., FRS, MRCS 1836, FRCS 1843
Paine, W., MRCS, LSA 1860
Palmer, C., MRCS 1846, MD St. And. 1854
Palmer, E., MRCS 1839, MD St. And. 1845
Parke, J. T., MRCS 1848, LSA 1850
Parker, D., MRCS 1860
Parker, L., MRCS 1828, FRCS 1843
Parker, R., MRCS, LSA 1859
Parker, S., LSA 1840, MRCS 1841
Parks, J., MRCS 1834
Parry, P., in practice prior to 1815
Parry, R., LSA 1829, MD Aberd. 1848
Parsley, W. H., LSA 1831, MRCS 1862
Parsons, E. K., MRCS 1843, LSA 1844
Parsons, F. H., MD, CM Glas. 1861
Parsons, J. St. J. G., FRCS 1858, LRCP 1859
Parsons, W. A., LSA 1826, MRCS 1828
Partridge, J. H., MRCS 1840, LSA 1841
Partridge, R., MRCS 1827, FRCS 1843
Patten, C. A., MRCS 1861, LSA 1865
Pattinson, H. B., MRCS, LSA 1866
Paull, J., MRCS 1866
Paxton, F. V., MB Oxon. 1864
Payne, G. S., LSA 1830, MRCS 1831
Payne, S., MRCS, LSA 1849
Pearce, F. D., LSA 1832, MRCS 1833
Pearce, F. D., jun., LSA 1862, MRCS 1863
Pearce, T., LSA 1825, MRCS 1826
Pearless, C. D., MRCS, LSA 1864
Pearse, W., LSA 1840, MRCS 1860
Pearse, W. D., MRCS, LSA 1836
Peatfield, T., MRCS 1863
Peel, R., MRCS 1861
Peirce, R. K., MRCS 1844, LM 1855
Pemberton, O., MRCS, LSA 1847
Penfold, H., MRCS, LSA 1850
Pennington, A., MRCS, LSA 1861
Pennington, F., MRCS 1854
Pennington, T., LSA 1828, MRCS 1829
Pepler, W. B., MRCS, LSA 1853
Perkin, R. C., MRCS 1850, LSA 1851
Perrin, W. J., LSA 1825, MRCS 1856
Perrot, H., MRCS 1856
Perry, C. H., LSA 1835, MRCS 1843
Perry, J. G., MRCS 1824, FRCS 1843
Perry, M., MRCS 1866
Pettifer, E. H., LSA 1861, MRCS 1862
Philbrick, S. A., MRCS, LSA 1830
Philbrick, T., LSA 1833, MD Giessen 1844, LRCP Edin. 1860
Phillips, E., MRCS 1839, MD Jena 1847
Phillips, G., FRCS 1855
Phillips, G. M., MRCS, LSA 1842
Phillips, J., LSA 1833, MRCS 1834
Phillips, J. W., LSA 1852, MRCS 1856
Philpott, H. G., MRCS 1853, LSA 1854
Picken, S., MRCS 1855, LSA 1858
Pickett, J., MRCS, LSA 1858
Pickford, J., MRCS 1828, MD Aberdeen 1852
Pierpoint, LSA 1855, MD St. And. 1862
Pilkington, T., MRCS, LSA 1863
Pilkington, W. H., MRCS 1845, LRCP 1861
Pinchard, B., MRCS 1843, MD Aberdeen 1859
Pinder, J. B., LSA 1855, MRCS 1856
Piper, C. C., MRCS 1848, LSA 1854
Pitt, E. G., LSA 1849, MD Aber. 1855
Place, J. L., MRCS, LSA 1866
Pocklington, E., MRCS 1860, LSA 1861
Pollard, J., MRCS, LSA 1854
Pollard, W., MRCS 1843, LSA 1844
Pollock, R. J., LSA 1826, FRCS 1852
Pooley, C., MRCS 1842, FRCS 1864
Porter, J. H., LSA 1827, M. D. Erlang. MRCP 1859
Potter, F. D., LSA 1832, MRCS 1835
Potter, T. H., LSA 1828, MRCS 1829
Potts, W. J., MRCS 1861
Powdrell, J., MRCS, LSA 1864
Powell, A., MRCS 1832
Powell, F., MRCS 1853, MB Lond. 1855
Powell, H., LSA 1831, MRCS 1832
Powell, H., MRCS 1839, LSA 1840
Powell, J. J., LSA 1830, MRCS 1834
Powell, J. T., MRCS 1854, MD St. And. 1862
Powell, L., MRCS 1864
Power, F. D., MRCS 1866
Power, H., MRCS 1851, FRCS 1854
Power, W. H., MRCS, LSA 1864
Powne, B. L., LSA 1864, MRCS 1865
Powne, W., MRCS, LSA 1856
Pratt, G., MRCS 1845
Price, J. L., MRCS 1864, LSA 1865

Price, R. C., MRCS, LSA 1832
Price, W., MRCS, LSA 1821
Prichard, A., LSA 1840, MD Berlin 1841
Prichard, H. P., LSA 1834, MRCS 1835
Prichard, W., MRCS 1838, LSA 1839
Pridham, J. W., MRCS 1860, LRCP 1862
Pridham, T. L., MRCS, LSA 1825
Prince, F. T., MRCS 1865, LSA 1866
Prince, T., LSA 1830, MRCS 1842
Pritchard, J. C., LSA 1826, MRCS 1843
Pritchard, J., MRCS, LSA 1849
Pritchett, G. M., MRCS 1839, LSA 1832
Pritchett, J. B., LSA, MRCS 1853
Proctor, A. G., LSA 1828, MRCS 1830
Proudlove, T. J., LSA 1834, LRCP 1859
Prowse, W., LSA 1848, MRCS 1849
Puddicombe, J. M., MRCS 1837, LSA 1838
Pugh, J. L. P., MRCS, LSA 1856
Pughe, H. R., MRCS, LSA 1857
Pullin, T. H. S., MRCS 1850, MD St. And. 1862
Pyne, R., LSA 1826, MRCS 1827

Quarrell, W., MRCS 1863
Quennell, J. C., MRCS, LSA 1859
Quick, J. R., MRCS, LSA 1836
Quick, J., MRCS, LSA 1866

Radcliffe, H. J., MRCS 1831, LSA 1830
Radley, W. H., MRCS, LSA 1857
Rae, Sir W. C. B., MRCS 1811, FRCS 1843
Randall, A. M., LSA 1821, MRCS 1822
Rainbird, H., MRCS 1856, LRCP Edin. 1860
Rainey, W. B., MRCS 1851, LRCP Edin. 1859
Randall, J., MB 1842, MD Lond. 1848
Randell, E. B., MRCS 1862
Raven, T. F., MRCS 1864
Ray, C., MRCS 1836, MD Pisa 1838
Raymond, H. S., MRCS, LSA 1853
Read, A., MRCS 1866
Read, C. G., LSA 1843, MRCS 1844
Reade, A. C., MRCS, LSA 1864
Ree, H. P., LSA 1839, FRCS 1858
Reece, R., LSA 1832, MRCS 1851
Reed, G., MRCS 1858, MD St. And. 1862
Reeve, J. F., MRCS 1812
Reeve, J. F., MRCS 1846, MD Aberd. 1857
Reid, J., MRCS 1843, LSA 1844
Reid, L. H., MRCS 1866
Rendall, C., LSA 1837
Rendell, R., MRCS, LSA 1832
Rendell, W. J., MRCS, LSA 1853
Rendle, J. D., MRCS 1846, MD St. And. 1862
Reynolds, T., MD Erlang. 1863
Reynolds, H. W., LSA 1822, MRCS 1843
Rhind, S., MRCS 1852, LRCP Edin. 1859
Rice, B., MRCS 1852, MB Lond. 1852
Richards, F. W., MB Lond. 1865, MRCS 1864
Richards, O., LSA 1834, MD Aberdeen FRCS 1848
Richardson, W. H., MRCS 1859, LSA 1860
Ridley, J. S., MRCS 1858, MD St. And. 1859
Rigden, G. C., MRCS, LSA 1839
Riley, H., LSA 1831, MRCS 1860
Riscoe, R. B., MRCS 1846
Robarts, H. P., LSA 1826, FRCS 1844
Roberts, J., MRCS 1834, MD Erlang. 1853
Roberts, R, LSA 1858
Roberts, T. E., MRCS 1864, MD Edin. 1864
Robinson, C., MRCS 1842, FRCS 1857
Robinson, C. R., MRCS 1846
Robinson, G., MRCS 1861, LM 1862
Robinson, H., MRCS 1861, LSA 1862
Robinson, S. H., MRCS 1862
Rogers, A., FRCS 1853
Rogers, B., MRCS 1857, MD St. And. 1862
Rogers, G. O., MRCS, LSA 1826
Rogers, H. C., MRCS, LSA 1865
Rogers, H., MRCS 1848, LDS 1860
Rogers, H. J., MRCS 1852, LRCP Ed. 1862
Rogers, T. A., MRCS 1847, LDS 1860
Rogers, T. L., MRCS 1853, MD St. And. 1857
Rogerson, G., MRCS, LSA 1827
Rogerson, J., LSA 1827
Rolleston, G., FRS, MB 1854, MD Oxon. 1857, FRCP 1859
Rolls, T., MRCS 1861
Roper, C. H., MRCS, LSA 1850
Roper, G. S., MRCS, LSA 1836
Roscow, P., MRCS, LSA 1845
Roskrudge, T. H. A., LSA 1838, MRCS 1839
Ross, F. D., MRCS 1858, LRCP Edin. 1860
Rouse, E., MRCS 1833, LSA 1836
Rowley, E. B., LSA 1825, MRCS 1826
Rowley, J., LSA 1834, MRCS 1837
Royds, W. A. S, MRCS 1866
Ruddock, E.H., MRCS 1863, MRCP Edin. 1865
Rudge, H., MD Erlang. 1814
Rummin, G., LSA 1859
Rumsey, C., LSA 1831, MRCS 1832
Rundle, H., MRCS, LSA 1866
Rusher, J. G., LSA 1841, MRCS 1842
Russell, G., MRCS 1852, LSA 1857
Russell, G. I., MRCS 1820, MD Aberd. FRCS 1854
Russell, G. I., jun., MRCS, LSA 1860
Russell, J., MRCS 1857
Rust, H. R. G., MRCS 1865
Rye, A. B., LSA 1836, FRCS 1852
Rymer, J., MRCS 1815

Sadler, M. T. jun., MRCS 1855, MD Lond. 1861
Sadler, S. E., MRCS 1832, FRCS 1862
Salmon, F., MRCS 1818, LSA 1817
Salter, J. R., MRCS, LSA 1852
Sandell, LSA 1830, MRCS 1831
Sanderson, H. J., MRCS 1842, MD St. And. 1853
Sandwith, H., MD St. And. 1834, FRCP 1859
Sandwith, H., MD St. And. 1850
Sandwith, T., MD Erlang. 1845
Sankey, F. H., LSA 1824, MRCS 1826
Sankey, W., MRCS 1810, FRCS 1844
Sankey, W., MRCS 1844
Sankey, W. H. O., MD Lond. 1850, FRCP
Sargent, D. W., LSA 1836, MRCS 1837
Saunders, E. D., MRCS 1828, LSA 1829
Saunders, G., MRCS 1845

Saville, R., LSA 1836, MD Erlang. 1855
Savory, C. T., MRCS 1862, MD St. And. 1862
Savory, J. E., MRCS, LSA 1854
Savory, J. T., LSA 1823, MRCS 1824
Savory, W. S., FRS, MRCS 1847, FRCS 1852
Sawer, T., MRCS 1837
Sawkins, J., MRCS 1835
Scholfield, H. D., MRCS 1842, MD Oxon. 1847
Schollick, T. J., MRCS, LSA 1860
Scobell, Rev. G., MRCS 1858
Scott, J., LSA 1833, FRCS 1854
Scott, R. J., MRCS, LSA 1844
Searle, G. C., LSA 1831
Sedgwick, H., MRCS 1866
Selwood, H. C., MRCS 1861, MD St. And. 1862
Selwood, J. H., LSA 1829, FRCS 1864
Senior, C., MRCS, LSA 1858
Sequeira, H. L., LSA 1852, MRCS 1859
Sewell, J. J., MRCS 1828, LSA 1847
Shackleford, G., LSA 1827
Sharpe, H. J., MRCS 1862
Sharpie H. W., LSA 1850, FRCS 1862
Sharples, W., LSA 1834, MRCS 1835
Shaw, C. E. M., MRCS 1863, LSA 1864
Shaw, G H., MRCS, LSA 1864
Shaw, H. E. F., MRCS 1844, LSA 1845
Sheehy, W. H., MRCS 1851, LRCP Edin. 1859
Shephard, F., MRCS 1855, LRCP Edin. 1860
Sheppard, J. B., MRCS, LSA 1849
Sheppard, W., MRCS 1843, LSA 1844
Sherwin, W. L., MRCS 1859, LSA 1860
Shine, W. L., MRCS 1859
Shipton, J. N., MRCS 1855
Shurlock, M., LSA 1836, MRCS 1837
Sicard, A. B., MRCS, LSA 1831
Simons, T. R., MRCS, LSA 1833
Simons, J. A., MRCS 1847, MD St. And. 1862
Simpson, C. M., MRCS 1862
Simpson, J. H., MRCS, LSA 1866
Simpson, R. P., MRCS, LSA 1865
Simpson, S. H., MRCS, LSA 1864
Simpson, W., MRCS 1863
Simpson, W. I., LSA 1823, MRCS 1824
Sison, E. N., MRCS 1842, LSA 1844
Skeate, E., MRCS, LSA 1834
Skeel, D., LSA 1827, MRCS 1829
Skeel, T., LSA 1837, MRCS 1838
Skelton, J., sen., MD Mass. 1855, LRCP Edin. 1864
Skey, F. C., FRS, MRCS 1822, FRCS 1843
Sladden, J. A., MRCS, LSA 1834
Slaytor, J. C., LSA 1836
Sleeman, P. R., MRCS 1839, FRCS 1854
Sloman, S. G., LSA 1837, MRCS 1839
Slyman, M. D., MRCS 1855, LSA 1858
Slyman, W., LSA 1828, FRCS 1855
Smart, T. W. W., MRCS 1827, MRCP 1860
Smee, A., MRCS 1840, FRS, FRCS 1855
Smee, A. H., MRCS 1866
Smith, C. E., MRCS 1858, MD St. And. 1859
Smith, E. R., MRCS 1864, LSA 1865
Smith, E., MRCS 1834, LSA 1835
Smith, F. M., MRCS 1851, MD St. And. 1857
Smith, H., MRCS 1864
Smith, H. S., MRCS 1837, FRCS 1843
Smith, J., LSA 1847, MRCS 1849
Smith, J. B., LRCS Edin. 1846, MRCS 1846
Smith, J. R., LSA 1824, MRCS 1859
Smith, J., LSA 1833, MD St. And. 1853
Smith, P., MD Aber. 1844, MRCP Lond. 1846
Smith, R. W., MRCS 1860, LRCP 1863
Smith, R., LSA 1828, MRCS 1829
Smith, T., MRCS 1854, FRCS 1858
Smith, W. A., MRCS, LSA 1856
Smith, W., MRCS, LSA 1834
Smith, W. J., MRCS, LSA 1857
Smith, W. A., MRCS 1856, MD St. And. 1857
Snook, J. W., MRCS, LSA 1864
Snowden, G. S., LSA 1829, FRCS 1852
Soame, C. B. H., MRCS, LSA 1854
Solly, S., MRCS 1828, FRCS 1843
Solomon, J. V., LSA 1838, FRCS 1854
Soper, J. H., MRCS 1855
Southey, R., MRCP 1860, MB Oxon. 1861
Southam, F., LSA 1841, MRCS 1842
Southam, G. T. M., MB Lond. 1863, MRCS 1862
Southam, T., LSA 1830, MRCS 1831
Southcomb, W. T., LSA 1824, MRCS 1825
Spackman, F. C., MRCS, LSA 1849
Spencer, E., in practice prior to 1815
Spencer, J. H., LSA 1835, MRCS 1836
Spicer, N. W., jun., MRCS 1853, LSA 1854
Spooner, E. O., LSA 1829, FRCS 1852
Spouncer, F. C., MRCS 1859, LSA 1860
Sprague, C. G., MRCS 1860
Spurway, C., MRCS 1863
Square, W., MRCS, LSA 1866
Square, W. J., MRCS, LSA 1835
Squire, A., LSA 1833, MRCS 1839
Stacpoole, G. C., MD Edin. 1845
Stafford, S. J. F., LSA 1858, MRCS 1851
Stallard, J. H., LSA 1842, MB Lond. 1858
Staning, R., MRCS 1837
Stanton, J., LSA 1832, MRCP 1859, MD Heidelb. 1842
Stanwell, W., MRCS, LSA 1855
Starke, A. G. H., MRCS 1859, MD St. And. 1861
Startin, J., LSA 1827, FRCS 1852
Starling, R. T., LSA 1824, FRCS 1856
Steel, C. D., MRCS 1836, FRCS 1859
Steel, J. S., MRCS 1843, MD Aberdeen 1854, FRCP Edin. 1855
Steele H. C. B., MRCS, LSA 1831
Stephenson, T. A., MRCS, LSA 1855
Stephenson, W., MRCS 1861, LSA 1862
Stevens, D. F., MRCS 1855, LSA 1856
Stevens, N. H., MRCS 1850
Stevenson, J. F., MD Edin. 1846, MRCS 1847
Stevenson, N., MRCS 1857, LDS 1862
Stewart, C., MRCS 1862
Stilwell, G., LSA 1827, MRCS 1828
Stilwell, H., MRCS 1857, MD Edin. 1857
Stilwell, J., LSA 1818, FRCS 1845
Stocker, G. S., MRCS 1831, LSA 1837
Stokoe, R., LSA 1828, MD St. And. 1862

Stone, D., LSA 1841, MRCS 1842
Stone, R. N., MRCS 1836, LRCP Edin. 1859
Stone, R. S., MRCS, LSA 1863
Stonehouse, C., LSA 1854, MRCS 1860
Stones, W. H., MRCS, LSA 1840
Storrs, R., MRCS 1856, LSA 1857
Stott, T. S., MRCS 1863, LSA 1864
Stowell, T., MRCS 1863, MD New York 1864
Stowers, N., MRCS 1838, LSA 1839
Stretton, S., MRCS 1854, LSA 1856
Stretton, W. H., MRCS 1851, LSA 1853
Strickland, E., MRCS, LSA 1860
Strutt, G. H., LSA 1860, MRCS 1861
Stubbs, H., LSA 1831, FRCS 1843
Sturkey, H. G., MRCS 1853, LRCP Edin. 1859
Stutter, F. A., MRCS 1853, MD St. And. 1860
Sugden, W., MRCS, LSA 1839
Summers, R. H., MRCS 1822
Sumner, W. A., MRCS, LSA 1842
Sutton, C. F., MRCS, LSA 1820
Sutton, F. J., LSA 1861, MRCS 1861
Sutton, H. G., MRCS, LSA 1860
Swales, E., LSA 1858, MRCS 1860
Swales, P., MRCS 1861, LSA 1862
Swayne, S. H., MRCS 1843, LSA 1844
Swift, G., LSA 1824, LRCP Edin. 1863
Sylvester, G. M., MRCS, LSA 1839
Sympson, T., MRCS 1847, LRCP Edin. 1860

Taaffe, R. P. B., MRCS 1852, MB 1863, MD Lond. 1865
Tait, E. W, LSA 1851, MRCS 1852
Tanner, R. C., MRCS 1861
Taplin, B. D., MRCS 1854, LRCP Edin. 1860
Taplin, T., MRCS, LSA 1824
Tarleton, J. H., MRCS 1865, LSA 1866
Tate, R., in practice prior to 1815
Taunton, G., MRCS 1848, LSA 1852
Taunton, T., MRCS, LSA 1835
Tayler, C., LSA 1837, MRCS 1838
Tayler, C., MRCS 1864
Tayler, G. C., MRCS 1866
Taylor, H., MRCS, LSA 1863
Taylor, J. E., MRCS 1845, LSA 1846
Taylor, J. S., LSA 1844, MD Aber. 1853
Taylor, T., MRCS 1842, LSA 1843
Taylor, T., MRCS 1818
Taylor, T., LSA 1821, FRCS 1861
Taylor, T., MRCS 1837
Taylor, W., MRCS, LSA 1830
Taltersall, W. J., MRCS, LSA 1866
Taynton, H., MRCS 1836, LSA 1839
Teare, T. M., jun., MD St. And., MRCS 1862
Tench, E. B., MRCS, LSA 1857
Terry, C., MRCS, LSA 1856
Terry, G., MRCS, LSA 1852
Terry, J., LSA 1825, MRCS 1826
Terry, J. J., LSA 1827
Thackwell, W., MRCS, LSA 1835
Theed, F., MRCS 1836, FRCS 1852
Thibon, J. W., MRCS 1864
Thomas, D. H., MRCS 1863, LSA 1865
Thomas, D. P., MRCS 1848, LSA 1851
Thomas, E., MRCS 1864
Thomas, J., MRCS 1836, LSA 1837
Thomas, J. B., MRCS 1864
Thomas, J., MRCS, LSA 1857
Thomas, J. H., MRCS 1850, MD Aberdeen 1860
Thomas, R., MRCS, LSA 1853
Thomas, R. H., MRCS, LSA 1853
Thomason, W. J, MRCS 1861, LSA 1860
Thompson, C. M., MRCS, LSA 1817
Thompson, C. R., MRCS 1850, LSA 1851
Thompson, C. T., MD St. And. 1855
Thompson, H., MRCS 1859, LSA 1860
Thompson, J. N., LSA 1824, MRCS 1825
Thompson, W. W., MRCS 1856, LSA 1857
Thomson, C. E., LSA 1828, FRCS 1863
Thorne, T. H., MRCS 1861
Thorne, R. T., MRCS 1863
Thornley, R. S., MRCS 1845, LSA 1846
Thurnall, W., MRCS, LSA 1838
Thursfield, R., LSA 1821, MRCS 1822
Thursfield, W., LSA 1824
Thurston, G. J., MRCS 1848, LRCP Edin. 1860
Tibbits, H., MRCS 1865, LRCP 1865
Tibbits, J., MRCS 1853, MD St. And. 1853
Tidboald, J. A., LSA 1827, MRCS 1828
Tindale, A. McI., MRCS, LSA 1865
Tinsley, W. W, MRCS, LSA 1850
Tomlinson, R. S., LSA 1827, MRCS 1828
Tompson, C. A. J., MRCS 1849, LSA 1858
Toogood, T. B., MRCS, LSA 1833
Toogood, J., FRCS 1843, LRCP 1844
Tothill, F. D., LSA 1833, FRCS 1864
Tracey, S. J., MRCS 1849, LRCP Edin. 1860
Trevan, F., LSA 1826, MRCS 1827
Trevan, M., LSA 1819
Trevan, M., MRCS 1863, LRCP 1861
Trollope, T. M. B., Cantab. 1854, MRCS 1856
Troughton, N., MRCS 1818, LSA 1819
Tuck, Rev. T. R., MRCS 1836
Tucker, R. P., MRCS 1834
Tuckwell, H. M., MRCS 1858, MD Oxon. 1863
Tuckwell, W. J. S., MRCS, LSA 1833
Tuke, D. H., MRCS 1852, MRCP 1860
Turner, C., MRCS 1853, LRCP Edin. 1860
Turner, C. C., MRCS, LSA 1841
Turner, E. W., MRCS, LSA 1856
Turner, J. W., LSA 1841, FRCS 1856
Turner, J., MRCS 1820, FRCS 1855
Turner, R., MRCS 1833, MD St. And. 1860
Turner, T., MRCS 1858, LRCP Edin. 1860
Turner, W., MB Lond. 1857, MRCS 1853, FRCS 1861
Turney, T. H., MRCS 1856, LSA 1857
Tuxford, J. E., sen., LSA 1824, LRCP Edin. 1859
Twigge, N. B., MRCS, LSA 1831
Twining, E., MRCS 1840, LSA 1841
Tyack, J., MRCS 1856
Tylecote, E., LSA 1826
Tylecote, E. T., MRCS 1855, MD Aber. 1860
Tyrer, R., MRCS 1843
Tyte, R. H., MRCS 1853

Upton, H. C., MRCS, LSA 1866
Upton, T. S., MRCS, LSA 1840

Vallack, A., MRCS, LSA 1836
Vallance, E., MRCS, LSA 1863
Vallance, J. T., LSA 1828, MD Erlang. 1844
Valpy, W. H., LFPS Glas. 1853, LM Glas.
Van, A. F., MRCS, LSA 1859
Vardy, J., MRCS 1842
Vaudin, C., MRCS 1853, LRCP Edin. 1860
Vaughan, W. E. W., MRCS, LSA 1853
Venn, T., MRCS, LSA 1848
Verey, G., MRCS 1859
Verity, A. B., MRCS 1840
Vernon, B. J., MRCS 1862, FRCS 1864
Vicary, C., MRCS. LSA 1845
Vicary, G. T., MRCS, LSA 1841
Vickers, T., LSA 1831, MRCS 1833
Vise, A. B., MRCS, LSA 1854
Vise, C., LSA 1826, MRCS 1827
Vise, E., MRCS 1841, LSA 1842
Vise, E. B., in practice prior to 1815
Voss, H. W., MRCS, LSA 1850

Wadams, E., MRCS 1843, LSA 1845
Waghorn, F., MRCS 1862, MD St. And. 1862
Wainwright, T., MRCS, LSA 1833
Walker, B., MRCS, LSA 1861
Walker, C., LSA 1846, MRCS 1848
Walker, C. E., MRCS 1866
Walker, G., MRCS, LSA 1825
Walker, G. A., LSA 1829, MRCS 1831
Walker, J. W., MRCS 1850, MB Lond. 1850
Walker, T., LSA 1834
Walker, W. H., MRCS 1860, LSA 1861
Wallace, A, MRCP 1857, MD Oxon. 1861
Wallis, G., MRCS 1857, LSA 1858
Walter, J., MRCS, LSA 1831
Walter, O. C., MRCS, LSA 1831
Walters, R. B., MRCS 1852, LSA 1853
Walton, H., FRCS 1848
Walton, J. S., MRCS 1835
Wane, W. J., MRCS 1865
Ward, J., MRCS, LSA 1835
Ward, S. M., MRCS 1860, MB Cantab. 1860
Ward, W. S., MRCS 1832
Ware, J., MRCS 1817
Ware, J. T., MRCS 1840, FRCS 1845
Ware, R., MRCS, LSA 1826
Waring, T. W., LSA 1833
Warner, J., MD St. And. 1862, MRCS 1860
Warner, T., LSA 1823, MRCS 1824
Warren, F. H., MRCS, LSA 1837
Warren, T. A, LSA 1838, MRCS 1843
Warry, E. T., LSA 1824, MD St. And. 1856
Warter, J. S., MD Edin. 1864, MRCP Lond. 1865
Warwick, J., FRCS 1859
Warwick, R. A., MRCS 1854, MD St. And. 1858
Waterland, H. J., MRCS 1847
Watkins, D. R., MRCS, LSA 1855
Watling, T. F., MRCS 1813
Watson, F. H., MRCS 1866
Watson, J., LSA 1824, MRCS 1825
Watson, R. M., MRCS, LSA 1827
Watson, Sir T., MD Cant. 1825, FRCP 1826
Watts, G., LSA 1852
Watts, G. H., MRCS 1850, LM Dub. 1852
Waugh, A., MRCS 1863, LRCP 1864
Waugh, J. W., MRCS 1840, MD St. And. 1856
Waylen, A. R, MRCS 1856, MD St. And. 1858
Waylen, C. W., MRCS, LSA 1861
Waylen, E., MRCS 1848
Waylen, G., LSA 1838, MRCS 1839
Waylen, W., LSA 1824, FRCS 1844
Wearing, A., MRCS, LSA 1864
Weatherly, F., MRCS, LSA 1841
Webb, G. F., MRCS, LSA 1865
Webb, H. S., LSA 1850, MRCS 1851
Webb, J. S., MRCS 1835, LSA 1836
Webb, J., MRCS 1839, FRCS 1856
Webb, R., MRCS, LSA 1854
Webb, T. F., MRCS 1835, LSA 1836
Webber, H. J., MRCS 1863
Webber, W., LSA 1821, FRCS 1859
Webster, G. E., MRCS, LSA 1851
Webster, W. H. B., LSA 1816
Weekes, G, LSA 1838, FRCS 1854
Weekes, J., MRCS 1849
Weekes, W. H. C., MRCS 1860, LSA 1862
Welbank, R, MRCS 1820, FRCS 1843
Wells, J. H., MRCS, LSA 1834
West, C., MD Berlin 1837, FRCP 1848
West, W. C., MRCS 1833, MRCP Edin. 1864, FRCS 1852
Wetherhead, T., MRCS 1838, FRCS 1856
Whateley, T., MRCS, LSA 1837
Wheeler, T. R., LSA 1842, MRCS 1843
Whidborne, G. F., LSA 1834, MD Heid. 1848
Whipple, C., MRCS 1863, LRCP 1864
Whitaker, E. F., MRCS, LSA 1824
Whitby, C. W., MRCS 1848, MB Aber. 1853
Whitcombe, E. B., LSA 1827, MRCS 1828
White, J. L., LSA 1836, FRCS 1845
White, R., MRCS, LSA 1816
Whitfield, R. G., LSA 1822
Whitling, H. T., MRCS 1854, LSA 1855
Whitney, W. U., MRCS, LSA 1835
Wicksteed, F. W., MRCS, LSA 1863
Wilcox, W., MRCS 1863
Wilding, R., LSA 1838, MRCS 1839
Wilkins, E. P., MRCS 1847, LRCP Edin. 1859
Wilkins, H., MRCS 1835, LSA 1836
Wilks, G. F., LSA 1832, MRCS 1833
Willett, A., MRCS 1859, FRCS 1862
Willett, C. V., MRCS, LSA 1861
Willey, T., MRCS 1844
William, J., LSA 1860, MD St. And. 1861
Williams, A. W., MRCS 1843, MD St. And 1847
Williams, C., MRCS 1857
Williams, E., MRCS 1834, MD St. And. 1859
Williams, E., MRCS 1804
Williams, E. H., MRCS, LSA 1861
Williams, E. E., MRCS, LSA 1860
Williams, H., MRCS 1815

Williams, T. M., MRCS 1853, LSA 1864
Williams, J., MRCS 1822, LSA 1823
Williams, J., MRCS 1841, FRCS 1859
Williams, J., in practice prior to 1815
Williams, J., MD St. And., MRCS 1860
Williams, N., in practice prior to 1815
Williams, O. W., MRCS 1820, MD Edin. 1840
Williams, P., MRCS, LSA 1856
Williams, R. P., LSA 1858
Williams, S. W. D., MRCS 1861, MD St. And 1862
Williams, T. E., MRCS, LSA 1863
Williams, W., MRCS 1840, MD St. And. 1842
Williams, W., LSA 1826
Williams, W., MRCS 1825, FRCS 1859
Williams, W., MRCS 1842
Willis, F., MB 1861, MD Oxon. 1864
Wills, C. J., MRCS 1864, LSA 1865
Wills, J. S., MRCS, LSA 1850
Wilson, E., FRS, MRCS 1831, FRCS 1843
Wilson, F., MRCS 1858, MD Heid. 1858
Wilson, H. G., MRCS 1866
Wilson, J. A., MRCS 1859
Wilson, T., MRCS 1836, MD St. And. 1848
Wilson, W. J., MRCS 1855, LRCP 1859
Wilton, F., MRCS 1855, LM 1856
Wilton, J. P., MRCS 1852, LSA 1854
Wilton, J. W., MRCS 1816, FRCS 1843
Wing, C., MRCS 1814
Wingate, R., MRCS 1858, LSA 1860
Winkfield, A., MRCS 1859, LSA 1860
Winkfield, W. B., MRCS 1858, LRCP 1864
Winstone, B., MRCS 1841, MD Aber. 1847
Winterbotham, L., MRCS, LSA 1856
Wingar, J., LSA 1826, MRCS 1827
Wise, R. S., MRCS 1840, MD St. And. 1842
Wiseman, W. W., MRCS, LSA 1833
Withers, F. O. B., MRCS 1859, LSA 1860
Witt, C., MRCS 1821, MRCP 1861
Witt, C., MRCS 1841
Witten, E. W., MRCS, LSA 1856
Wolferstan, S., LRCP 1864, MRCS 1864
Wolstenholme, J. H., MRCS 1851, LSA 1852
Wood, A. J., MRCS 1830, MD St. And. 1858
Wood, F., MRCS 1841, FRCS 1860
Wood, G. L., LSA 1818, MRCS 1819
Wood, J. Y., MRCS 1836, LSA 1837
Wood, T., MRCS, LSA 1827
Wood, T. C., MRCS 1840, MD Aber. 1846
Wood, W., MRCS, LSA 1857
Wood, W. J., MRCS 1858, LSA 1859
Woodhouse, J., LSA 1829, MD Aber. 1858
Woods, F. B., LSA 1830, MRCS 1856
Woods, F. H., MRCS 1859, LRCP Edin. 1860
Woodward, J. R., MRCS 1824
Wookey, J., MRCS 1858, LSA 1861
Woolhouse, H. H., MRCS 1849
Woollett, R. F., LSA 1838, MRCS 1839
Woolley, T. S., MRCS, LSA 1863
Worboys, F. T. S., MRCS, LSA 1862
Worger, T. H., MRCS, LSA 1864
World, R. R., LSA 1837, LRCP Edin. 1860
Wormald, T., MRCS 1824, FRCS 1843
Worship, J. L., MRCS, LSA 1847
Wotton, C., MRCS 1858, MD St. And. 1859
Wray, D. C., MRCS 1856, MD St. And. 1862
Wright, J., MRCS, LSA 1853
Wright, M., MRCS 1863, LSA 1865
Wright, T. G., LSA 1827, MRCS 1829
Wright, W. K., LSA 1832, FRCS 1852
Wrixon, J., MRCS 1842, LSA 1854
Wyer, O. F., MRCS 1858, LSA 1859
Wyman, G., LSA 1831, MRCS 1832
Wynne, J., MRCS, LSA 1834
Wynter, J. St. T., LSA 1830, MRCS 1858

Yarde, W., MRCS 1857, LSA 1858
Yarrow, G. E., MRCS, LSA 1863
Yates, J., MRCS, LSA 1846
Yates, W. H., MRCS 1851, LSA 1852
Yearsley, J., MRCS 1827, MD St. And. 1862
Young, R., MRCS 1818, MD Aberdeen 1851

*** In the above List the date of the earliest professional qualification is placed immediately after each name.

Gentlemen who desire their names to be added to this Register are requested to apply to the Honorary Secretary, who will be glad of any assistance in making it complete and correct.

EXAMINATIONS, 1866.

Senior Scholarship in Medicine, Surgery, and Midwifery—

1. W. Square
2. H. C. Upton

Senior Scholarship in Anatomy, Physiology, and Botany—

1. W. D. Butcher.
2. E. B. Crowfoot.

JUNIOR SCHOLARSHIP.

1. J. Rouch
2. J. H. Hendley
3. F. De H. Hall

Bentley Prize—

F. H. Haynes

Wix Prize—

T. Cole

PRACTICAL ANATOMY, SEN.

Foster Prize—W. W. Saul
2. L. Clapham
3. J. T. Hickman, J. Kirkman
5. J. T. Williams
6. J. O. Sankey
7. T. W. Lee

PRACTICAL ANATOMY, JUN.

Treasurer's Prize—A. F. Field.
2. *Examiner's Prize*—E. A. Brickwell.
3. T. H. Hendley, C. Bennett, J. T. Hartill
6. J. Rouch, F. J. Glencross, E. Groves

ST. BARTHOLOMEW'S HOSPITAL

AND

MEDICAL COLLEGE.

The WINTER SESSION will commence on October 1st, with an Introductory Address by Mr. SAVORY, at 5 o'clock P.M.

MEDICAL OFFICERS.

Consulting Physician—Dr. Burrows.
Physicians—Dr. Farre, Dr. Jeaffreson, Dr. Black, and Dr. Martin.
Consulting Surgeons—Mr. Skey and Mr. Lawrence.
Surgeons—Mr. Wormald, Mr. Paget, Mr. Coote, and Mr. Holden.
Assistant-Physicians—Dr. Edwards, Dr. Harris, Dr. Andrew, and Dr. Southey.
Assistant-Surgeons—Mr. Savory, Mr. Callender, Mr. T. Smith, and Mr. Willett.
Physician-Accoucheur—Dr. Greenhalgh.

LECTURES.

WINTER SESSION, Commencing October 1st, 1866.

Medicine—Dr. Black.
Clinical Medicine—Dr. Farre, Dr. Black, and Dr. Martin.
Surgery—Mr. Paget and Mr. Coote.
Clinical Surgery—Mr. Skey, Mr. Paget, Mr. Coote, and Mr. Holden.
Descriptive Anatomy—Mr. Holden and Mr. Callender.
Physiology and General Anatomy—Mr. Savory.
Chemistry—Dr. Odling.
Demonstrators of Anatomy—Mr. Smith and Mr. Baker.
Assistant Demonstrators of Anatomy—Mr. Vernon and Mr. Langton.
Demonstrator of Morbid Anatomy—Dr. Andrew.
Tutors—Dr. Duckworth, Mr. Baker, and Mr. Shepard.

SUMMER SESSION, commencing May 1st, 1867.

Materia Medica—Dr. Farre.
Botany—Rev. George Henslow.
Forensic Medicine—Dr. Edwards.
Midwifery—Dr. Greenhalgh.
Comparative Anatomy—Dr. Church.
Practical Chemistry—Dr. Odling.
Dental Surgery—Mr. Coleman.
Microscopic Demonstrations—Mr. Savory.
Demonstrators of Microscopic Anatomy—Dr. Southey and Mr. Vernon.
Demonstrators of Operative Surgery—Mr. Smith and Mr. Baker.
Vaccination—Mr. Wood.

Ophthalmic cases are admitted under the care of the surgeons.

The Hospital contains 650 beds—247 Medical and for the Diseases of Women; 322 Surgical and for Diseases of the Eye; and 81 for Syphilitic Cases. The number of patients exceeds 126,000 annually.

Collegiate Establishment.

Warden—Mr. Willett.

Students can reside within the Hospital walls, subject to the Collegiate regulations. Some of the Teachers connected with the Hospital also receive Students to reside with them.

Seven Scholarships, varying in value from £20 to £50, are awarded annually.

The Clinical Clerks, the Midwifery Assistant, and the Clerks to the Assistant-Physicians are appointed from the most diligent Students.

In accordance with the regulations of the College of Surgeons, Students have charge of patients under the supervision of the Assistant-Surgeons.

All Students preparing for their examinations are specially examined by the teachers of Anatomy or by the Tutors.

Further information may be obtained from Dr. Andrew, Mr. T. Smith, or Mr. Callender, at the Hospital.

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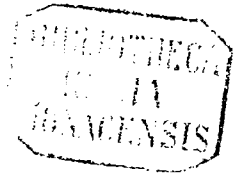

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