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THE TONER LECTURES

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LECTURE V.

ON THE SURGICAL COMPLICATIONS AND SEQUELS OF THE CONTINUED FEVERS.

BY

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DELIVERED FEBRUARY 17, 1876.

WASHINGTON:

SMITHSONIAN INSTITUTION.

MARCH, 1877.

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SMITHSONIAN INSTITUTION,

Washington, April, 1877.

LECTURE V.

Delivered February 17, 1876.

ON THE SURGICAL COMPLICATIONS AND SEQUELS OF THE CONTINUED FEVERS.

By William W. Keen, M.D.,
of Philadelphia.

The province of the physician and that of the surgeon are, in general, sufficiently sharply defined and differentiated, yet they have many points of contact. While some diseases belong exclusively to the province of the one, and some to that of the other, other diseases may fall with equal propriety under the care of either practitioner. Still another class of cases, however, beginning in the domain of Medicine, may terminate in that of Surgery, and we may lack their complete history from the very fact of this division of their care and interest.

Among the diseases classed as strictly medical, none deserve the appellation more definitely than the continued fevers, and especially Typhus and Typhoid. Yet I hope to show that fevers are not infrequently the cause of the gravest and least expected surgical troubles, mention of which is generally omitted, even in our best text-books on medicine, still more rarely noticed in works on surgery, and where noticed, it is only with the greatest brevity.[1]

[1] "The cases of constitutional disease discovered by fever might serve to illustrate a large part of the convalescence of fever, a subject of the highest interest and full of promise of utility to one who will carefully study it. The sequelæ of scarlet fever are commonly enumerated; those of typhoid fever, especially those seen in surgical practice, are scarcely less numerous, but seem less known." Just as this is going to press, I find the above remarks by Sir James Paget, in his extremely interesting Clinical Lectures and Essays, London, 1875, p. 378.

My attention having been called to the matter, by several personal cases, I have been led to study the subject, and I desire now to record briefly the results of an extensive search of medical and surgical works as well as the records of individual cases, in the hope that by grouping together many isolated instances, I may be able to contribute somewhat to our exact knowledge of the surgical complications and sequels of the continued fevers, both as to their causes, the means of their early recognition, the best methods of treatment, and if possible that still higher object, the averting of dangers which otherwise may prove disastrous to health, and too often to life itself.

The exanthematous fevers are better known as causes of surgical troubles, and I have therefore carefully excluded them as well as a few cases following intermittent fevers.

I shall omit entirely all well-known results of a semi-surgical character, such as hemorrhage from the nose and bowels, peritonitis, with or without perforation, erysipelas, a not infrequent complication about the face, or when bedsores exist, and cases of thrombosis of the veins, which causes a variety of phlegmasia deserving a more extended study than it has yet received.[1] Bedsores and the ordinary abscesses are too well known to demand other than passing allusions. Much as I regret to do so, I must also omit, from want of time, the consideration of the forms of disease which especially interest the ophthalmic surgeon. Ulceration and perforation of the cornea are briefly, but I may say completely, treated by Trousseau.[2] Post-febrile ophthalmia or amaurosis, a peculiar retino-choroiditis which follows only relapsing fever, first described by Hewson in 1814, and Wallace in 1827, has been so carefully

[1] See Bibliography "Phlegmasia," where I have grouped the most important references.

[2] Clinique Méd. de l'Hôtel-Dieu, 2d ed., p. 271, and Gaz. des Hôp., 1856, 170.

studied of late by ophthalmic writers, as to leave but little further to be said, and I must refer those who desire to study it to the appended Bibliography.

I shall include only such cases as diseases of the joints, œdema glottidis and necrosis of the cartilages of the larynx, which often require tracheotomy, necrosis of the bones, gangrene of the extremities and other parts, fistulæ of various kinds, and the like.

The records of many cases are extremely imperfect, some in fact are mere allusions, and I have been compelled therefore, in tabulating them, to come as near the truth as may be. The frequency of many symptoms is therefore greater than appears from my tables. Especially is it difficult to discriminate between typhus and typhoid fevers as causes. The earlier cases, before the essential abdominal lesion of typhoid was recognized, are all classed as typhus, and even to-day many cases, especially in German books and journals, are briefly called "typhus," meaning "typhus abdominalis," *i. e.*, typhoid. If any error exist, therefore, it will consist in assigning too many cases to typhus proper, for I did not feel permitted to go back of the record unless plainly authorized to do so by the history or the post-mortem.

I. DISEASES OF THE JOINTS.

Two forms of disease of the joints are found, first a polyarticular inflammation, which may assume either a rheumatic or a pyemic form; and, secondly, a monarticular inflammation.

The rheumatic variety I shall at once dismiss. The pyemic form of inflammation is not very common, for Murchison, with his immense experience in the London Fever Hospital, has seen but one case. It follows the usual course of pyemia, both in its symptoms and its usually fatal issue. Huss and others have referred it to suppurative phlebitis from bedsores,

parotitis, etc., but it has been observed in cases in which no such complication existed. Pyemic arthritis, like the gangrene from pressure, parotitis, etc., is most apt to occur in severe cases, in which the blood change is at its maximum, and the "typhous crasis," as Stokes has expressed it, possibly becomes converted into pyemia.

It is, however, the monarticular form, which will most interest us. It affects the larger joints, such as the elbow and shoulder, the ankle and knee, but above all the hip. The pain is usually slight. The swelling is generally readily observed in all joints except the hip and the shoulder, where it is probably obscured by the muscular masses about these joints combined with the tardy increase in the swelling. Usually it arises spontaneously, but occasionally from periostitis or necrosis extending into the joint. It rarely produces suppurative or fistulous openings. The result is, therefore, generally a gradual return to usefulness, although in 3 cases I have found anchylosis. Of 43 cases, the lower extremities were affected in 39, the upper in only 7, 3 of the cases involving a joint in both, for occasionally two large joints are affected at once. Arthritis, therefore, resembles other surgical febrile affections, such as gangrene, necrosis, etc., in affecting mainly the lower extremities, as do also thrombosis and the ordinary œdema. The frequency of these joint troubles is not great. According to Güterbock, in a series of years in the Charité (Berlin) and the Hamburg Hospitals, not a case occurred, and in the Vienna General Hospital from 1868 to 1871 only 2 cases among 3130. Murchison does not even name this complication at all, nor do any other of our text-books, either on surgery or practice, except a few lines by Volkmann, in Pitha and Billroth's Handbuch. Güterbock and Hellwig are the only authors who have treated them at all fully. Yet that they are of great importance, and demand our utmost attention, will be

seen at once when we consider that of the 43 cases named, spontaneous dislocations occurred twenty-seven times in the hip, twice in the shoulder, and once in the knee.

These dislocations require more particular notice. From their similarity to febrile arthritis in the same and other joints their pathology seems clear, although—in singular contrast to the strangely fatal laryngeal stenosis I shall soon consider—not a single death has occurred, and therefore no post-mortem verification has been possible. They belong to the class of "Distension-luxations." That the cause is not the specific poison is evident, since similar results follow other and dissimilar diseases, such as locomotor ataxia, the exanthematous fevers, hemiplegia, sciatica, and rheumatism, as pointed out by Stanley in 1841.[1]

Usually in the period of convalescence, following, therefore, the prolonged exhaustion, there arises a subacute synovitis with a gradual serous distension of the capsular ligament, which, having reached a certain point, may slowly subside, and no further evil follow. In 3 cases, however, this burst externally, producing sinuses, but in none of them was the discharge purulent. The main result is a slow, generally unperceived, elongation of the ligaments, *e. g.* of the hip, with perhaps also a swelling of the so-called gland at the bottom of the acetabulum. This distension will spend its force mainly posteriorly, since the inverted Y ligament reënforces the capsular ligament in front. Given this condition, the slightest force will dislocate the head of the femur upwards and backwards on the dorsum of the ilium. In one case a fall to the floor produced it, in three others turning over in bed, and twice the lifting of the patient in the arms from one bed to another.

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[1] On dislocation espec. of the hip-joint. Med. Chir. Trans. xxiv. 123. See also Malgaigne, Fract. and Disloc., Paris, 1855, ii. pp. 218–226, 882–887.

But in all the other 21 cases no cause was assignable, and it is, therefore, likely that it was mere muscular contraction which becomes, at the time when these occur, more vigorous as health gradually returns. Seitz has recorded one of the most remarkable cases in which, from extensive bedsores, the abdominal decubitus was maintained for nearly a month, and he supposes that this was the immediate cause of the dislocation. But as in no other case is this posture noted, it cannot be regarded as correct. Indeed, if posture have any influence, as the dislocation is generally if not always iliac, the dorsal decubitus would be the most favorable for its production. In one of the shoulder cases a subcoracoid luxation was caused by the patient's assuming the erect posture. Gravity had here probably some influence, together with the muscular exertion. The dislocation of the knee also was posterior.

A remarkable case corroborative of the non-specific character of the lesion and the probable influence of gravity, I have lately seen in the service of Dr. Wm. G. Porter, at St. Mary's Hospital. The child was about two years of age, greatly exhausted from mal-nutrition, and for about six weeks was kept alive by inunctions of sweet oil, no other nourishment whatever being given. It had large abscesses in different parts of the body, and at present has necrosis of the left humerus. Early in the period of returning strength and before the necrosis appeared, spontaneous luxation of the left humerus into the axilla occurred. It was easily reduced by manipulation, and has not since recurred.

Typhoid was noted as the preceding fever in 15 and typhus in 7 of the hip cases. Sex has a marked predisposing influence in this, as we shall find in other diseases, for of 23 cases 15 were males and only 8 females. The age at which they occur is still more noteworthy: 15 were under 15 years, 6 from 15 to 20, 1 was 30, and 1 was 61 years old; that is, 21 out of 23 were under 20 years old. The analogy to coxalgia, it will be ob-

served, is, therefore, very marked. Usually they were single dislocations, 6 being on the right side and 6 on the left; but in 3 cases dislocation of *both* hips occurred.

From the apathetic condition of the patient in some cases, the subacute nature of the lesion, the absence or slightness of the pain, the masking of the swelling by even the wasted muscles about the joint, and, above all, the want of knowledge of cause or probability of the dislocation, and therefore the neglect to examine the parts thoroughly, it is not surprising that this threatening evil should have been often unobserved. In 9, that is, one-third of the cases, it is distinctly stated that the *actual dislocation* was the first fact observed, and in most of the others this is probably true.

The date at which the dislocation was, at least, observed, was generally after the third week. One case occurred in the first week, 4 in the second, and 9 in the fourth week or later, that is, during distinct convalescence. Pain was experienced in 13 cases. Usually, it was not severe, nor was it always strictly localized in the hip, but sometimes extended to the entire leg. In only 2 cases was it referred to the knee, thus differing markedly from the well-known coxalgic knee-pain. Swelling is only distinctly stated in 6 cases, though probably present here as in other joints, but either unobserved or often unrecorded in the brief statements I have often found. The variety of the dislocation is not named in 10, but as in all the other 17 it was iliac, there is good reason to believe that this is probably always the case. Shortening is recorded in 11 cases, and where the amount is named was generally one and a-half to two inches. In 5 cases the rotation was inward, in 2 outward, and in 2 of the 3 double dislocations both legs were rotated in the same direction, that is, right or left, thus producing a peculiar deformity when compared with the apparently reversely rotated body. The head of the bone in 4 cases was freely movable in all directions. This mobility of the head and the singular diver-

sity in the rotation of the limb, are additional reasons in favor of the distension theory of its pathology. Flexion and adduction, Dr. Sayre has shown to be the position of the limb which produces the greatest capacity of the capsular ligament of the hip, and we ought to see this position, therefore, as a rule in distension-luxations. But I only find two cases in which there were adduction and flexion. In the other cases the position is not stated, except in one in which the limb was extended.

As to treatment, reduction is generally easy when the luxation is discovered early, but if the discovery or treatment be tardy it is always difficult and often impossible. In 11 cases reduction was successfully accomplished seven times by manipulation, twice by extension, and twice by both means. In 8 cases reduction was not effected, and in 8 the result is not stated. Only two cases of recurrence of the luxation are noted, a rather surprising fact in view of the relaxation of the distended tissues; but its possibility should be borne in mind and guarded against by the same prophylactic means that I will name directly. No snap is heard on reduction, all tension and suction-power of the joint being lost. Even after reduction the leg may be somewhat longer than the other, owing, probably, to the distension, to the swollen articular gland, and possibly in old cases to interstitial changes in the neck of the femur.

The question of prophylaxis is perhaps the most important of all, and the indications are clear. First, a careful watching and repeated examination of the hip-joint, especially in children, to detect any effusion. If any exist, the position of the leg becomes of the greatest possible importance. As adduction and internal rotation favor spontaneous dislocation, the leg should be kept in abduction and external rotation. The first indication is easily fulfilled by two lateral sandbags which may be bridged across in front at intervals by a bandage, to keep the leg at rest between them, or by lateral splints. The foot may be kept in external rotation by bandages or adhesive plaster fastened to

the external sandbag or splint. If the effusion threaten to produce dislocation, it may well be a question whether aspiration would not afford a safe and efficient means of prophylaxis.

II. DISEASES OF THE BONES.

A popular name for necrosis is "fever sore," but, as Nathan Smith long since pointed out, more because it caused fever than because it was caused by fever. That it does follow fever and is caused by it is certainly true, but it is not a very frequent though a very important sequel. I have collected thus far 50 cases of necrosis proper following continued fevers, but among these are 19 reported by one single author—Whately—the histories of which are exceedingly brief and unsatisfactory. He states, indeed, that he has *seen* 30 cases—an incredible statement, I think, in view of the fact that from all other sources, after an extended search, I can only gather 31 more. "Fever" with him, however, may include a very wide range. One element of unavoidable uncertainty in the history is seen at once. The osseus disease usually falls under the eye of the surgeon at a period distinctly subsequent to the fever, and, knowing nothing personally as to the previous medical history, he must depend upon the statement of the patient—often a most unreliable means of information.

Two causes for such necroses and other forms of disease, such as periostitis and caries, are to be found: first, thrombosis, or in some cases possibly embolism; and secondly, absolute inanition or want of nutrition.

The role assigned of late to the marrow together with the spleen as a source of the red corpuscles, would seem to be confirmed by the similarity of the changes observed by Ponfick[1]

[1] Ueber die sympathischen Krankh. des Knochenmarks bei inneren Krankh. Virchow's Archiv, lvi 534. Cf. also Anatom. Studien über den Feb. Recurrens, Virch. Archiv, 1874, lx. 153.

and others in the spleen and marrow in typhoid. In later convalescence, or shortly after recovery, we find in the marrow many mother-cells holding numerous blood cells, enormous masses of large cells filled with pigment in complete analogy with the observed metamorphoses of extravasated blood. These are especially seen at the sides of the cavernous veins, and must retard still further a circulation already impaired in force by a weakened heart. Nutrition is here at its lowest ebb, and as the vessels, from the nature of the tissue in which they run, cannot enlarge in proportion to the needs of the circulation and are themselves more or less involved in fatty degeneration, we may readily understand how the lack of nutrition alone, as in Dr. Porter's case previously cited, would cause gangrene of the bone even more readily than in the soft parts in which we know it to be so common.

That the bones should suffer from vascular clots, and especially the bones of the lower extremities, where such clots are most frequent, as we shall see, in gangrene, is probable both from analogy and experiment, and from one case in which it has been actually observed in typhoid.[1] There is no reason to suppose, when thrombosis is so frequent elsewhere, that the bones would escape. Virchow has shown that in relapsing fever we frequently have infarctus in the marrow. Volkmann[2] gives an excellent case and illustration of necrosis of the tibia and talus from embolism, the result of endocarditis. We need a few similarly exact observations in cases of necrosis from fever, in which death or amputation affords the desired opportunity to settle the question positively; but generally the examination, if made at all, is of the most superficial character.

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[1] See Meusel's Case, p. 15.

[2] Pitha & Billroth's Handbuch, Bd. ii., Abth. ii., Lief. i., p. 287, and Langenbeck's Archiv, 1864, v. 330. See also Mollière, Lyon Méd., 1870, pp. 12, 149, 256; 1871, p. 38.

Hartmann[1] has shown, experimentally, that obliteration of the nutritious artery causes necrosis of the inner lamella of bone—a strong point it must be admitted in favor of Whately's theory that after fever the result is not ordinary necrosis but a central necrosis of the inner lamella which he limits to the tibia. Blocking of the veins is evidently not so dangerous in bones as blocking of the arteries, since the collateral venous circulation especially towards the extremities is abundant, while the collateral arterial circulation is scanty.

I have found 69 cases of diseases of bone following continued fevers. Of these, 50 were cases of necrosis, 12 of caries, 3 of periostitis, and 4 of indeterminate or doubtful nature. Three cases of necrosis following typhoid and smallpox I have excluded. Typhoid, as usual, claims the larger share, for of 41 cases 37 followed typhoid and only 4 followed typhus. Males also are in the preponderance, counting 38, to 14 females. Age has not a very marked influence, as 19 were under 20 years, 11 from 20 to 30, 11 from 30 to 40, and 5 over 40. Scarcely any region of the body escapes; 22 cases involved the head, 7 the trunk, 6 the upper extremities, and 42 the lower, a result strikingly in accord with the cases of arthritis and gangrene. In the head I have found 12 cases of necrosis of the alveoli and jaws. Among these perhaps the most remarkable, although somewhat doubtful, case is the one I saw in a soldier at Frederick, Maryland, in 1862, in which, after typhoid fever followed by pneumonia, the entire right upper jaw with a part of the palate bone and the intermaxillary bone necrosed and separated. The case is remarkable, both from its being a striking example of the limitation of disease by the embryonic development,[2] and also from the extraordinary series of ope-

[1] Nekrose herbeigeführt durch Verstopfung des Foram. nutrit. Virch. Archiv, viii. 114.

[2] H. Allen, Studies in the Facial Region, Phila., 1875, has specially called attention to this point.

rations subsequently done by Dr. Gurdon Buck, of New York,[1] to remedy the frightful deformity which had been produced. It is but proper to say that the man was reported to have taken about ʒij of various mercurials during his preceding illness; but from the facts I have stated, as well as his scanty history, I think it tolerably clear that the fever and not the mercury caused the necrosis. Mercury or syphilis complicated two or three of the other cases I have tabulated, but they were not, apparently at least, the cause of the trouble.

Mr. Salter[2] has pointed out the relation of alveolar necrosis to the eruptive fevers, especially scarlet fever, and believes that as these structures are dermal in character they partake with the skin in the eruptive mischief. While this relation remains undisturbed, yet I do not think the necrosis exists as a specific sequel of these fevers only. Of the 12 cases cited, 7 occurred as follows: one at 16, one at 12, and five at 10 years of age and under, that is, during the period of dental development and growth. That such cases are more frequent in the exanthemata is natural when we consider the relative infrequency of the continued fevers under 15 years of age.

The period at which these diseases of the bones arise varies greatly. Of 47 cases 10 arose in the first two weeks, 27 in from three to six weeks, and the remaining 10 followed often months after the fever. The earlier cases include probably, most of those from clots, and the later ones those arising from enfeebled nutrition, whose effects especially in structures which vary so slowly as the bones may readily extend over such long periods.

Especially does this enfeebled nutrition show itself in case where too early strain is put upon the parts and justifies the remark of Aitken that "no man can be considered fit for work

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[1] See Bibliog.

[2] Holmes's Syst. Surgery, 1st ed., vol. iv. p. 50.

or for general military service for three or four months after an attack of severe typhoid fever." The following case illustrates the wide-spread mischief that may follow in the osseous system when put to the test by labor, months and even years after such a fever.

H. W., a remarkably stout, healthy lad of 16, was attacked Dec. 17, 1871, with typhoid. He was delirious for four weeks, was in bed four months, and first got out of doors in May, 1872. Bedsores had formed, but they were kept in check by incessant care. In the fall of 1872, not yet being strong, he went to work at riveting in an iron works, which required him to stand and use a ten pound hammer, the main strain being naturally on the right arm and leg. His right arm soon began to swell, and finally four fistulous sinuses formed. After the removal or discharge of several pieces of bone, this arm recovered in about a year. Returning then to the same work, his health being still impaired, his right thigh began to trouble him, broke out, and healed several times, discharging several pieces of bone. He came under my care in July, 1875. He had then a scar and five open sinuses in the thigh, all leading in the direction of the bone, and in one, just above the knee, a fragment of dead bone an inch long was found. This sinus and a second just below the patella, an offshoot from it, threatened to invade the knee-joint. Meanwhile, in the fall of 1874, not having done any work on account of his right leg, the left thigh broke out, and a sinus in the direction of the bone was established, but no dead bone was ever actually found here. In January, 1875, an abscess also appeared in the left arm, and after the discharge of some bone finally healed. I enlarged all the existing sinuses in the right thigh, removed the dead bone, and after treating the case carefully for four months all the sinuses healed. A new one, however, has appeared of late in the right thigh, but no dead

bone is, as yet, to be found.[1] His health markedly improved early in 1875, and since my operation he has grown to be exceedingly robust and hearty again. His right knee, which was stiff from the sinuses among the muscles of the thigh and near the knee-joint, is now as mobile as ever, and he is at work with ease. The abscesses in the two arms were at or near the deltoid insertion, in the right leg, the earliest was just below the insertion of the glutæus maximus, and in the left near the lesser trochanter, all points at which muscular strain in standing and hammering would come.

The symptoms need scarcely be alluded to, for they are those of ordinary necrosis, although Whately endeavors to differentiate them. In 13 cases of necrosis of the long bones other than Whately's, in which the description enables me to judge, I find only 3 distinct cases of central necrosis,[2] and these differ in no especial manner from other cases. That it is limited to the tibia, as asserted by Whately, is disproved by the fact that of seventy-seven bones affected, the tibia was attacked only thirty times, including in these the 19 reported by Whately.

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[1] In Feb. 1876, it healed, broke out again in July, and did not heal until December, after a counter opening had been made. Since then he has been well (March, 1877).

[2] The third of these cases I have had in private practice while the MS. is passing through the press. A. W., a rather feeble girl, æt. 11, was taken sick with typhoid May 10, 1876. After three to four weeks in bed she began to walk, but soon had to stop on account of weakness, and especially of pain in her left tibia. After three weeks' poulticing it broke in two places, and has discharged ever since. I saw her first in December, 1876, and found two small sinuses which extended into the bone, but no dead bone had ever been discharged. After building up her general health by tonics and cod-liver oil, on February 17, 1877, I operated on the bone, using Esmarch's apparatus in the manner I have suggested (*Phila. Med. Times*, Sept. 26, 1874), and after making an opening into the medullary canal with the chisel and gouge, I removed a small, loose spicula of necrosed bone (central necrosis) seven-eighths of an inch long. At this date, March 5, 1877, she is doing well.

The results of necrosis vary with the situation. The ordinary sinuses etc., I need not mention further. If in the sacrum, coccyx, or innominate bone, perineal fistulæ may result, of which I have found 3 cases. If in the mastoid or petrous bone, the brain and its membranes may be involved. The following *resumé* of the case of Meusel is of especial interest, as it throws so much light on the cause of the necrosis, the clot in the meningea magna, and is as extraordinary for the audacity of the treatment as for the success of the result.

Fig. 1.

Necrosis of Frontal (A), Parietal (B), and greater wing of Sphenoid (C) bones, following typhoid. In B and C the middle meningeal artery and its branches are seen. Natural size. Meusel, Deutsche Klinik, 1872, p. 266.

A student in the gymnasium, æt. 19, at Easter in 1868, had an attack of typhoid fever, went home when convalescent, but did not improve, and suffered much from headache. In August, four months after the fever, he had a large fluctuating abscess over the right parietal region, which was opened, and

dead bone found. By Oct. 1, the bone was loose, and on the 5th, an incision of three inches was made and a loose piece of the frontal was removed. The rest of the dead bone was firm, but the incision was extended backwards till the whole of the necrosed portion was exposed. It was then carefully chiselled loose and separated at the squamous suture. At the anterior inferior angle the necrosis was there found to extend on the internal surface only; with a fine chisel this internal lamella, a piece $1\frac{1}{2} \times 2$ c. m., was chiselled away from the great wing of the sphenoid. In it was a groove in which lay the anterior branch of the middle meningeal, filled with the detritus of a clot. The whole piece was $5\frac{1}{2} \times 9$ c. m. The dura mater was but slightly injected. The scalp and the dura mater united and in fourteen days he was nearly well, having recommenced his Latin and Greek with the greatest zest eight days after the operation. Two small pieces of loose bone afterwards caused threatening symptoms, but improvement followed immediately upon their discharge. In March he was entirely well, and went to Göttingen to study philology at the University. Epileptiform attacks followed during 1869, but then disappeared, and had not reappeared in 1872.

As to treatment, the ordinary operation for the removal of necrosed bone is to be done at the proper time, especial care being taken to remove any small central sequestrum. Occasionally the disease of the bone may cause extensive disease in the soft parts, or may extend to a neighboring joint, though either complication is rare. Amputation then becomes imperative. Only four such amputations occurred in the cases reported; two died, one recovered, and one was under treatment. About the face not infrequently extensive plastic operations are required.

III. DISEASES OF THE LARYNX.

The laryngeal complications are noted very briefly by several medical writers, such as Murchison, Flint, Liebermeister, Griesinger, etc., but it is mainly the laryngeal ulcers themselves which are treated of, their surgical results being scarcely mentioned. Gross and Gray barely allude to typhoid as a cause of œdema glottidis. Even systematic writers on the larynx scarcely notice them. Gibb and Rühle refer to two or three cases. Cohen simply names fever. Türck gives, however, eight valuable cases. I have collected 169 cases, of which at least 67 (and probably many more) certainly involved the cartilages themselves.

The troubles which may demand surgical interference are all allied, and are the result of a low grade of inflammation. The entire respiratory mucous membrane (as is shown by the frequency of bronchitis) is in a more or less catarrhal condition like that of the bowels, and occasionally other mucous membranes such as those of the gall-bladder, urinary bladder, and vagina. It is not, therefore, a matter of surprise, that serious trouble should arise in the larynx, especially as slight variations in its mechanical condition gravely embarrass so vital a function as respiration.

Pathologically the troubles may be grouped into three varieties, viz., 1. Œdematous laryngitis. 2. Ulcerative laryngitis. 3. Laryngeal perichondritis. Practically it is often exceedingly difficult to separate these various forms even at the post-mortem, so far do they overlap each other. Œdema may exist alone or it may result from either of the others; ulceration may march steadily deeper until the cartilages are involved; or the perichondritis may produce an abscess which will burst, and so form an ulcer. How much more difficult, nay often impossible, then is it, to diagnosticate precisely the form of the disease, when, happily, the patient recovers. Dyspnœa,

suffocation—this is the one great overshadowing clinical fact which groups them all together whatever the form of the disease, or of the preceding fever.

That simple asthenic œdema may arise just as œdema of the lower extremities is not only probable, but has been positively observed by Emmet and Buck. It has also been observed after diarrhœa, bronchitis, and other diseases. But this is a much rarer form than those cases in which it is secondary to erysipelas, or parotitis, or laryngeal ulcers, often of small extent. I cannot help suspecting also very strongly that more careful future examinations will show in not a few cases that local venous thrombosis has been the cause of the œdema.

The other two forms especially merge into each other. Rokitansky believes that the ulcers are a peculiar form of typhus, the so-called laryngo-typhus. Others, and I certainly agree with them, do not believe that they are specific in their origin, but belong "to the common cortège of septic diseases" and other allied disorders in which the low grade of inflammation readily runs into ulceration, and even into local gangrene. How much influence local stasis of the blood or even clots in the vessels may have, has not been carefully investigated, but I believe them to be no unimportant factors.

These ulcers are sometimes very common. Thus Griesinger met with them in 31 out of 118 autopsies, Hoffmann in 28 out of 250, and Louis believes that "if found on the body of one who has died of an acute disease, they will establish with nearly perfect certainty and without going any further, that the affection is typhoid fever." At other times they are so rare that in nearly 13,000 typhus cases at the London Fever Hospital, Murchison records but 21 of laryngitis, of whom 8 died; and in typhoid he has only seen 3 or 4 cases.

Where the cartilages are involved, Moritz-Haller, and others, believe that it follows the ulceration which detroys the mucous membrane and eats down to the cartilages, while Sestier has

gone so far as to declare that the ulcers which accompany perichondritis are not primary but in all cases secondary. Both I believe are right, but both go too far. So far as the history and post-mortem appearances would enable me to judge, I have found in 20 cases that the perichondritis preceded the ulcers and caused them, while in 10 cases the ulceration had caused the perichondritis. In cases of perichondritis in which death takes place early, there is no opening in the mucous membrane, but a submucous abscess will be found surrounding the necrosed cartilage. If death takes place at a later date a small opening will exist, through which the probe will enter into a much larger cavity. In other cases the surface mischief will be by far the most widely spread, the ulcers being roughly conical, involving not only the mucous membrane, but eating deeply down to the cartilages. Similar necrosis of the nasal cartilages also sometimes results from fever.

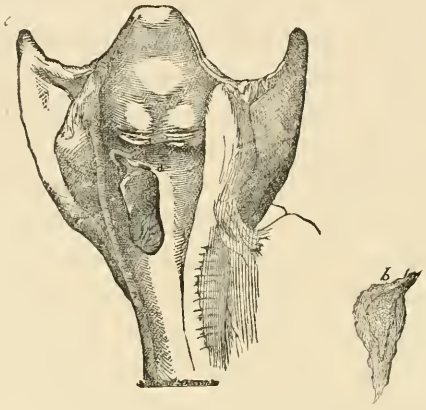
Those cases in which there is considerable cough, or the patient in his delirium has cried aloud, or sung much, or those in which, after distinct convalescence, there has been exposure to wet and cold, are predisposed to laryngeal troubles. They are exceedingly rare in children. In 94 cases in which the age is recorded, I have found but 6 under 15 years of age, 60 from 15 to 25, and 28 above 25 years. Sex is potent here as in the other diseases considered. Lisfranc thought them more common in women than in men, but of 110 cases, I find 86 in men and only 24 in women, or $3\frac{1}{2}$ to 1.

The cause of the stenosis is various. It may be, 1st, from œdema; 2d, the swelling produced by the abscess about the cartilage; 3d, the sides of the glottis may fall together if the cricoid be destroyed and in pieces; 4th, the permanent approximation of one, or more rarely of both vocal chords from destruction of the fixed points of origin of the muscles; 5th, as in two remarkable cases given by Hoffmann,[1] shreds of

[1] Op. cit., pp. 253 and 255.

sloughing tissue on which blood coagulates, may form a sort of polyp which suffocates the patient even in spite of tracheotomy.

Fig. 2.



Laryngeal Ulcer after Typhoid. Polypoid Hæmatoma hanging from it and causing death by suffocation after Tracheotomy. *a*, upper end of incision. The smaller cut, *b*, is a section of the Hæmatoma showing its two layers; the centre consisting of shreds of dead tissue hanging from the ulcer, and the outer layer of clotted blood. Hoffmann, Veränd der Organ. beim Abdom. Typhus, Taf. v. Fig. 16.

The seat of the stenosis is threefold. Most frequently (25 cases) it is supra-glottic, that is, in the epiglottis and ary-epiglottidean folds, especially where the œdema is primary, or where it is caused by ulceration, or arytenoid perichondritis. The next most frequent site is subglottic, *i. e.*, about the cricoid (22 cases). This is always, I believe, the result of cricoid necrosis or perichondritis. Russell reports 2 cases following typhus, which he regards as examples of Gibb's "subglottic œdema." That the second was a case of ulceration and perichondritis, is quite certain from the history, and most likely the other was too. The larynx was normal down to the chords, tracheotomy rescued both when suffocation was imminent, and both were followed by stricture, requiring the permanent use of the canula. The least frequent site of the œdema is in the

glottis proper, since œdema of the vocal chords is named but nine times.

The date of the development is generally here too, in the later fever, or more frequently in distinct convalescence. Of 102 cases only 4 occurred in the first week, 13 in the second, 19 in the third, and 66 from four weeks to two months. They follow typhoid far more frequently than typhus, in the proportion of 106 to 49, some of the latter being probably really typhoid, while 14 arose from other forms of continued fever.

The position of the ulcers in the larynx is noteworthy. Wherever they may be, from the arytenoid to the cricoid, they are almost invariably posterior. Rheiner[1] has shown that the posterior wall of the larynx is the richest in vessels, and that ossification begins here often as early as the twentieth year. Here, then, we should expect the most frequent inflammations and thrombosis of the smaller vessels; and when we add to this the effect of gravity, from the continuous dorsal position, and the mechanical effects of frequent use of the voice, and, therefore, repeated movement of the arytenoid cartilages in some delirious cases, we have a sufficient explanation of the phenomenon. Emphysema of the neck and trunk is an occasional result of such ulcers where they penetrate the mucous membrane. This was first pointed out by Wilks. Other cases are reported by Steiner and Loeschner. All three were children.

Necrosis of the cartilages is the most important form to recognize, in consequence of its excessive gravity; for, of 56 cases in which the result is given, 54 died. One recovered after tracheotomy,[2] and one without it.[3] We can scarcely agree with Türck, therefore, that the prognosis is "doubtful." The seat of the necrosis in the majority of the cases is the cricoid

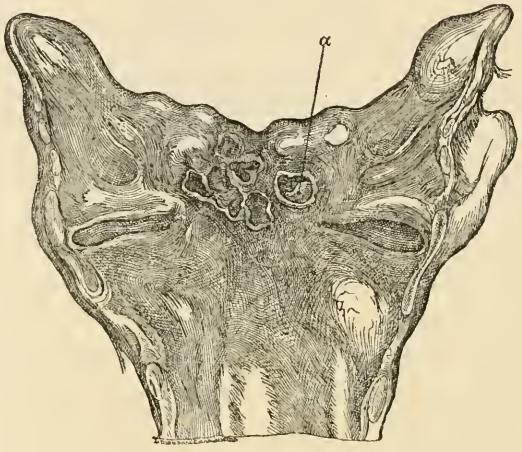
[1] Breitäge zur Histologie des Kehlkopfs; Würzburg, 1852.

[2] Türck, p. 223.

[3] Hérard, l'Union Méd., July 14, 1859, quoted by Trousseau, Clin. Med., Syd. Soc. Trans., 2d ed., vol. ii. p. 407.

(38 times), next the arytenoids (19 times), while the other cartilages were affected but 5 times. In 10 of the cases, the cricoid and arytenoid were involved simultaneously.

Fig. 3.



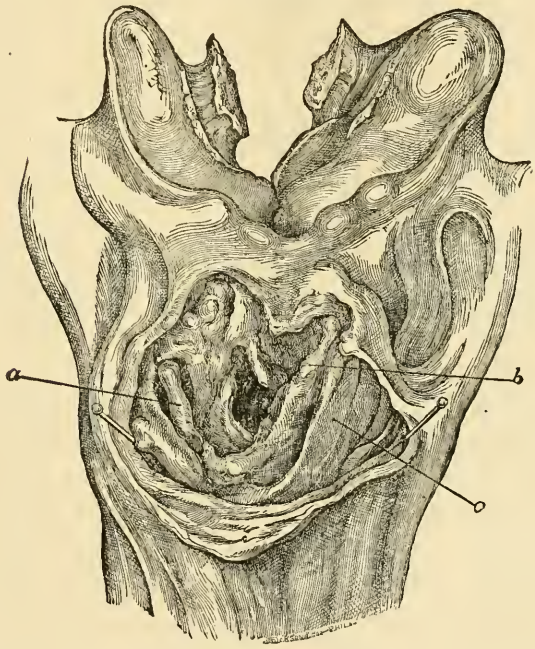
Perichondritis laryngea following typhoid. Ulcers on the posterior wall of the larynx. At *a*, a piece of the necrosed left arytenoid cartilage shows. Türck, Krankh. des Kehlkopfes, p. 216, fig. 78.

It is to be specially observed that probably a number of the cases which recovered were also really cases of perichondritis, of which the *positive* evidence was wanting. Thus, in 8 cases in which recovery followed tracheotomy, the patients spat up gangrenous or purulent matter, besides having other symptoms of cricoid necrosis; but as the expectoration of any pieces of necrosed cartilage was not positively observed, I have not included them in my statistics of necrosis. Were they included, the result, and especially the result after tracheotomy, would be far more favorable.

Whatever the origin of the necrosis, the cartilage is soon destroyed, either by molecular disintegration or is even broken in pieces. Sometimes it undergoes ossification; at others,

caseous degeneration. The articulations of the cartilages are also often destroyed, especially those of the cricoid, with the arytenoid or with the thyroid; and thus again voice is impaired. The function of the muscles, especially those which open the glottis, is impaired or destroyed, from direct implication of the muscles or from destruction of their cartilaginous attachments,

Fig. 4.

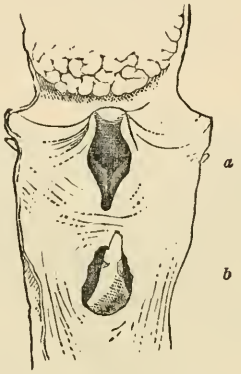


Perichondritis laryngea following typhoid; abscess opened from œsophagus and seen from behind. *a*, *b*, necrosed and partly destroyed cricoid cartilage; between *a* and *b* the dark spot indicates the point where the abscess communicated with the trachea; it did not communicate with the œsophagus; *c*, crico-aryten. post. muscle. Türck, Krankh. des Kehlkopfes, p. 218, fig. 80.

or of the articulation; and thus the vocal chord of that side is immobilized, and altered voice and stenosis of the larynx follow. If an abscess form about the cricoid, it may not only burst into

the larynx but also into the pharynx, and a fistulous opening be established by which food may enter the larynx and trachea.

Fig. 5.



Perichondritis after Typhoid. Opposite *a* the œdematous ary-epiglottidean folds are seen. Below the vocal chords the larynx was narrowed by thickening of the connective tissue. Opposite *b* a movable piece of the destroyed and broken cricoid, which has perforated the œsophagus, is seen.—Rühle, Kehlkopf-Krankh., p. 178 & Pl. I., Fig. 2.

This, Dittrich thinks, is the most frequent point of opening; but I have only found it recorded in six instances, though such an abscess would have burst here in several other cases had life been prolonged. An additional complication may follow, also in the pharynx. The cricoid presses upon its posterior wall, both from swelling and from gravity, and this pressure, together with the foul purulent discharge, may cause an ulcer; or, as in a case recorded by Armstrong, a retro-pharyngeal abscess. Hoffmann[1] records another case of retro-pharyngeal abscess extending from the base of the skull to the diaphragm, laying bare the left subclavian artery. This, however, did not result from a laryngeal necrosis.

The earliest symptom of grave laryngeal disease is usually

[1] Op. cit., p. 388.

an altered voice. I have only found it distinctly stated as unaltered in three cases. Generally it is hoarse, sometimes higher, but usually lower in tone, probably from involvement of the crico-thyroid muscle. Sometimes complete aphonia sets in. Stokes[1] has supposed the hoarseness and deafness both to be due to the muscular degeneration, to which I shall allude hereafter; but it is more likely to be due here, at least, to œdema and inflammatory swelling, and, at a later period, to the mechanical destruction of the parts involved.

Soon after this hoarseness is observed, the dyspnœa sets in. This is paroxysmal, the attacks being generally at night. With each succeeding attack the severity increases, until life is destroyed; but even the very first attack may be unexpected, sudden, and fatal. Especially does this seem to be the case in typhus, and in supra-glottic œdema. Emmet and others have recorded a number of cases, in which, even without the least previous dyspnœa, on simply assuming the erect posture sudden suffocation came on, and life was only saved by instantaneous laryngotomy. The delay of "a few minutes" caused death in one case which otherwise would probably have recovered. Most frequently the dyspnœa is inspiratory, but I cannot find that this is of any decided diagnostic value.

The expectoration, curiously enough, aids us but little; in but few cases did it attract sufficient attention even to be named; but if it be purulent or gangrenous, it should arouse instant attention. Pain and tenderness, though often masked by the mental condition, are generally present, especially in perichondritis. As we have the resisting vertebral column behind the cricoid, I would strongly urge that both by direct and lateral pressure on the cricoid, and also by sliding it sidewise, we may often elicit pain which might otherwise be overlooked. Lateral pressure will also often produce dyspnœa, or

[1] On Fevers, Phila., 1876, p. 105.

aggravate it, especially if the cricoid be broken, or nearly broken in two. Dysphagia is noted as present twenty-one times and absent five times, and is especially present in cricoid and arytenoid necrosis. Rarely is there any external swelling to attract attention, for I have only found it noted thrice. Nor can the pharynx be relied upon to warn us of impending evil, for in 16 cases in which its condition was observed, it was normal in 10 and inflamed in only 6.

Most of the cases occurred before the days of the laryngoscope, so that I have only thirteen such examinations from which to draw any inferences, seven of which are recorded by Türck. In several instances there was nothing whatever abnormal down to the vocal chords; and the swelling of a cricoid abscess on the posterior wall is not visible. But the usual facts observed, were the fixation of at least one chord in the middle line, diminished mobility of the other, swelling of the ary-epiglottidean folds, stenosis of the larynx increased by lateral pressure, a depression of the mucous membrane in case of destruction of the arytenoid, and sometimes the opening of an abscess, usually near the processus vocalis. A few such positive facts with any additional positive symptoms should lead to an equally positive treatment.

And first the mortality is so great that the treatment is of the greatest possible moment. Of 146 cases of all kinds of stenosis in which the result is recorded, 101 died and 45 recovered, a mortality of over 69 per cent., but not a surprising result when we add the laryngeal disease to the exhausting fever. But when we separate the cases operated upon from those in which no operation is named, or was certainly not done, the importance of the treatment becomes even more appreciable. Of the 76 cases not operated on, in which the result is stated (and, be it observed, I have included in these the cases of mere scarification), 17 recovered and 59 died, a mortality of over 77 per cent. Of the 70 cases operated

on by some form of bronchotomy, 28 recovered and 42 died, a mortality of 60 per cent. And when it is remembered that in two of the fatal cases the larynx was not opened, though tracheotomy was apparently performed; and a third, in full recovery thirteen days after the operation, on the removal of the canula, was suddenly suffocated before it could be replaced; and in another, the canula got displaced in front of the trachea; that in many, if not in most of the cases, the operation was deferred till the last possible—that is the most unfavorable—moment; that many cases that might have been rescued were plainly *allowed* to die from exhaustion, or even from positive suffocation, by timid doctors, in which the result could not have been worse had an operation been performed, the question of operation would seem to be decided.

Yet I would not be understood as an advocate of rash and indiscriminate bronchotomy. Its dangers are great, and not to be undervalued. The question, however, is often between a dangerous operation and a more dangerous refusal. In case the attack is sudden and severe, so that life is in immediate and positive peril, no question can arise as to the propriety of an operation, wanting which, the patient perishes on the spot, nor any question that crico-thyroid laryngotomy is the easiest, safest, speediest operation. Delay here means death. Of 14 such operations, 8, or 57 per cent., recovered, and in such cases as are recorded by Emmet and Anderson, no words can add force to the fact, that after life was apparently extinct, laryngotomy and artificial respiration saved 5, the delay of a few minutes resulting fatally in the 6th case. A cut throat here is not over-dangerous, and the operation is so simple, and may be so imperative that every medical man, as well as surgeon, should stand ready to do it in case of impending death.

But it is in the less suddenly threatening cases that judgments may differ, and here I hope to be able to assist in forming a decision. The moment, in a case of typhus or typhoid,

that hoarseness, aphonia, dyspnœa, or dysphagia sets in, the larynx should be examined with the utmost vigilance both from without and by the laryngoscope. If œdema or ulceration be found, but the danger be not as yet severe, leeches, iodine, ice in rubber bags, or possibly a blister may be used externally, and astringents, nitrate of silver, etc., internally, together with proper general treatment. In 1819, Lisfranc proposed scarification and operated succesfully in six non-febrile cases, and in 1847, Buck revived the practice and devised an appropriate knife. The laryngoscope has made such treatment far more certain, and it should first be tried in suitable cases.

But, should these means fail? Then an *early* rather than a late tracheotomy, for otherwise we add to the previous enfeebling disease a prolonged battle for breath, with its ensuing pulmonary congestion and general exhaustion. Especially is this to be commended in the peculiarly fatal necrosis of the cartilages. Of 67 positive cases, 41 of which occurred in men and only 10 in women, all died but 2, one with and one without an operation; 22 were operated on, 45 were not. But as before mentioned, 2 died from displacement of the canula, at least one of which had practically recovered; 8 cases of recovery after tracheotomy are not included, since, though the presumption in favor of necrosis is very strong, yet the evidence is not positive. Were only these 8 included, and it is certainly fair to do so, it would stand 30 operations and 10 recoveries, a mortality of 67 per cent., and 45 not operated and 1 recovery, a mortality of nearly 98 per cent.

Once, then, that perichondritis is established, death is almost unavoidable if no operation be done. If bronchotomy be resorted to, the chances are greatly improved. Indeed, it is probable that if the dangers which surround such cases be recognized in the future and a prompt attempt at relief be made, far more favorable results will be obtained. I would, therefore, join with Sestier in urging an immediate operation the moment

that perichondritis is recognized and serious dyspnœa sets in, without waiting for repeated attacks to exhaust the slight store of strength, all of which will be needed during the subsequent separation of the necrosed cartilage, and the sloughing of the soft parts.

In other cases than those of perichondritis such haste will not be necessary: yet if the respiratory murmur be progressively enfeebled; if pulmonary congestion set in; if the paroxysms of dyspnœa increase in frequency and severity, especially if orthopnœa arise; if the local disease be extensive or rapidly increasing; or if the general feebleness be so great that a little further interference with the respiration will destroy life; then no time is to be lost. Doubt is certain death.

What operation shall be performed? If an instant operation be needful, cricothyroid laryngotomy is the best; if time allow, tracheotomy. But if the cricoid be involved, with Beck, I would advise laryngo-tracheotomy, *i. e.*, tracheotomy prolonged through the cricoid; since it would allow readier access to the seat of the disease for the discharge of pus, the removal of any loose piece of cartilage, and the treatment of any ulcers or granulations. These I regard as greater advantages than the danger of possible collapse of the lateral halves of the cricoid, which is prevented in part by the canula, and would not impede the respiration even if it occurred. Unless necessary, it is best not to operate during a paroxysm; since the mechanical difficulties of the operation are then largely increased, and the danger of entrance of air into the veins is apparently much greater. In 36 operations for laryngeal angina, two such accidents occurred; whereas, in 245 non-anginose cases, not a single similar accident arose (Sestier).

Hemorrhage, as would be supposed from the condition of the blood, is sometimes a serious complication, both at the operation and subsequently, and caused death in three cases.

A curious and unexpected complication arose in two cases

reported by Mohr[1] and Laennec,[2] the knowledge of which should guard us against a similar error. The operation having been apparently achieved and the canula inserted, respiration was not bettered nor did air pass through the canula. In a few minutes the patients died suffocated. At the post-mortem it was found that the canula had entered, not the larynx, but the abscess around the cricoid. In Mohr's case the vertebræ were felt through the incision, and a probe and the canula moved freely about in what was naturally believed to be the trachea but proved to be the abscess cavity.[3]

In three cases of œdema the canula was removed in six, eight, and nine days respectively. But after such serious loss of substance and extensive organic mischief as are involved in the cases of perichondritis, it is not a matter of wonder that the stenosis of the larynx is generally permanent. In 17 cases of probable or actual perichondritis, the canula was removed in one case after seven months, but in the other 16 cases, when last seen, the patients were still wearing them. Once, after seven years' use, a piece of the canula wore away, broke off, and fell into the trachea, whence it was successfully removed by Albers. Busch, Russell, and others have attempted to dilate the stricture, both from above and below, but without any success.[4]

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[1] Casper's Wochenschr., 1842, p. 192. Also in Dittrich, Prag. Vierteljahr., 1850, iii. p. 129.

[2] Bayle, Nouveau Journ., t. iv. p. 37.

[3] Green, Brit. Med. Journ., Dec. 17, 1870, p. 649, and Marsh, St. Barth. Hosp. Reports, iii. 368, report cases (not following fevers, however) in which the tube was inserted in the cellular tissue in front of the trachea.

[4] By the courtesy of Drs. Otis and Woodward, of the Army Medical Museum, the casts and specimens of Dr. Buck's case, p. 18, and of several cases of laryngeal stenosis, were shown.

IV. GANGRENE.

The cases of gangrene may be divided into two classes: A, those from pressure; and B, cases of spontaneous gangrene.

A. Those from *pressure*, or the ordinary *bedsores*, are not peculiar to the continued fevers, as is well known, but arise from any prolonged debilitating disease or accident. They are more common, therefore, in typhoid fever than in typhus, on account of its greater duration. But they not infrequently follow typhus, if it be prolonged by any complication. The only points in addition to the greater danger of pyæmia, to which I desire to call attention, are as follows:—

First, as pointed out by Nélaton,[1] Blandin,[2] and others, if they penetrate deeply they may destroy the ligaments uniting the sacrum and coccyx, and so penetrate into the spinal canal and set up meningitis, etc. This complication, I believe, arises much more frequently from bed-sores, whatever their cause, than is generally known. Recently I have had two instances: one arising from a bedsore following confinement, the specimen which I show you, and which I owe to the courtesy of Dr. Schell, my colleague at St. Mary's Hospital; and the other in a boy who injured his knee and died some weeks after from tetanus induced probably by this complication. I have found 6 cases of tetanus recorded; 4 following typhoid and 2 typhus. Four of them were females. In one it was clearly caused by a bedsore.[3] Four of them died, but the two women who recovered had had menstrual irregularities, which probably caused the alleged tetanus.[4]

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[1] Path. Chir., Paris, 1844, i. 256–7.

[2] Anat. Top., 2me ed. p. 437. See also Charcot, Mal. du Syst. Nerveux, 2me ed., i. 89–90.

[3] Maclagan's.

[4] De Lauriere (see Bibl.) reports also a case of hydrophobia, which became ataxic, following a quotidian fever. He had been bitten by a

Secondly, large bedsores, as in two cases reported by Chenu in the Crimea,[1] may greatly hinder free motion of the legs and trunk by the extensive cicatrices.

Thirdly, the treatment first proposed, I believe, by Brown-Séquard, of ice poultices for fifteen minutes, followed by hot flaxseed poultices for two to three hours, often stimulates the most indolent bedsores to heal with surprising rapidity. During and since the civil war I have repeatedly and successfully tried this plan of treatment.

Sometimes gangrene results from the slighest pressure, as in a case reported by Stokes,[2] in which there were thirty such spots; two or three new ones appearing every morning, at points of such trifling pressure, as where the mammæ leaned on the arm, or one leg on another, and a black hand appearing where the face had rested on the hand. Strange to say, the woman recovered after a month's abdominal decubitus. For such cases Liebermeister recommends an almost continuous and complete bath, the body resting and reclining on sponges.

B. But the cases of so-called *spontaneous gangrene*, though less frequent, are of far greater interest from a surgical point of view. They vary greatly in frequency. Thus, neither Flint nor Trousseau ever saw a case; Nélaton does not name fever as a cause of gangrene; Murchison, though he has seen a few, does not cite a single English post-mortem. Yet Estlander reports 34 cases, and I have collected in all 113 cases. The frequency varies in proportion to the severity of the case and of the epidemic, and especially to the preceding conditions as to bodily nourishment, mental depression, and general mode of life. In former wars especially, from the time of Thucydides to that of Napoleon, fierce epidemics, especially of typhus, have

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healthy dog three months before. R. Reid (Pathol. and Treat. of Fever, Trans. Queen's Coll. Phys. Ireland, iii. 41) alludes to the similarity of hydrophobia and the excitable stage of fever.

[1] Rapport, pp. 520, 524. [2] On Fever, Phila., 1876, p. 210.

decimated armies and often displayed a most frightful tendency to gangrene. But of late, whether in civil or military practice, if we may judge from the scanty gleanings I have been able to obtain from the journals and the experience in our own civil war as well as in the late European wars, the condition of the sick has been so ameliorated that gangrene is happily a rare complication.

The history of the extraordinary series of cases reported by Estlander, well illustrates these predisposing causes. In Finland, a financial crisis and a series of bad harvests from 1862, were followed in 1865–7 by sporadic cases of fever, mainly typhoid; but from the thrifty habits of the people and governmental support, the epidemic was at first neither severe nor extensive. Then came the cold and rainy summer of 1867, followed by a very bad harvest. That winter, typhus raged in almost every household, so that often the well were not numerous enough to nurse the sick. The death-rate rose from 2.74 per cent. to 7.69 per cent. Instead of an annual increase in the population of over 15,000, it decreased, in 1868, nearly 94,000; and this in a population of less than 2,000,000. Of 105 doctors, 30 sickened and 8 died. Up to 1868 not a case of gangrene occurred, but in the first seven months of that year 28 cases occurred. Then came the bountiful harvest of 1868, and by August the epidemic had almost disappeared. Yet the lingering effects of the previous want were seen in six later cases of gangrene. But such an experience is altogether exceptional. From other writers I have rarely obtained more than two or three cases.

Estlander's 34 cases were all, except one, from typhus; but of the remaining 79 cases, 43 followed typhoid, 22 typhus. The influence of age is not very marked, as is seen in the fact that of 67 cases, 8 occurred before 15, 27 from 15 to 25, and 32 after 25 years of age. But sex, as usual, has a marked

determining influence. Of 81 cases, 56 were males and 25 females. This is the more curious when we consider that the number of deaths in men, and therefore presumably of cases, does not hold at all the same relation. In 1868, in Finland, 31,000 males and 28,000 females died; yet of 31 cases of gangrene of the legs, 25 were males, and only 6 females. The site of the gangrene is very suggestive also. In 5 cases it was in the ears, 10 in the nose, 27 in the face, neck, and trunk, 5 in the arms, 7 in the genitals, and 72 in the legs; that is, of 126 localities, in 77 it was in the extremities, and in 22 more, in other peripheral districts of the vascular system (ears, nose, genitals).

As far as the pathology of the cases is concerned, they may be divided into two classes: 1, those with a discoverable clot; and 2, those without such a clot. Murchison believes that all cases of spontaneous gangrene arise from arterial thrombosis, but the careful post-mortem examinations of Estlander and others show, that at least in the larger visible vessels, sometimes no such thrombus exists.

1. Those *with clot.* The cause of such clots, as Humphrey[1] and others have shown, is not the condition of the bloodvessels. But seldom have I found it stated, that the arterial walls were diseased; and when they were, it was presumably a secondary process, the result, and not the cause of the clot. Few, if any, pathologists will now attribute such results, with Bourgeois, to a metastasis, especially when arising in convalescence, as these so frequently do. Gigon has attributed them to chemical alterations in the blood, which give it an irritative character, and this, with friction at points of curvature, produces inflammation and coagulation. If so, a fair proportion of cases should be seen in the upper extremities, where the same irritating blood circulates and similar curves exist. How rare this is, we have already seen.

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[1] Brit. Med. Journ., 1859, p. 582.

Although the precise factors in determining the thrombosis in any individual case may be somewhat doubtful, as also why it is frequent in one epidemic and rare in another, apparently frequent in Germany and rare in Britain, and especially in the United States, yet three causes clearly exist which may vary *inter se* in producing the result: 1, the altered blood; 2, the weakened heart; and 3, the mechanical difficulties in carrying on the circulation, especially in distant parts.

That the blood is profoundly altered, and probably has an increased coagulability, is conceded. In some cases even air is found in the veins, as noted by Crisp[1] and Lebert.[2] That every such change, besides its depressing effect upon the nutrition, and therefore upon the vitality of the tissues themselves, would interfere more or less with its circulation, and consequently predispose to thrombosis, is most probable. But when we look at the seat of the cases of gangrene of both varieties which are under consideration, I think the conclusion is inevitable, that the last two causes are the more immediately determining factors.

The heart, as Stokes showed, is softened in its texture, and therefore weak. Hayem (see bibliog.) has shown that myocarditis is extremely frequent, and not rarely involves the endocardium. From the sixth to the fourteenth day is its weakest period, and not only is the general force of the circulation diminished at this time, but all the blood not being squeezed out of its cavity, clots may form in the heart, and then, or at a later period, when the heart regains somewhat of its force, be washed into the circulation and lodge as emboli. Such seems to have been the origin of the clot in a remarkable case related by Patry.[3] A decolorized adherent embolus was

[1] Dis. Bloodvessels, p. 18.

[2] Prag. Vierteljahr. 1858, i. 33. Moorehead, Trans. Med. and Phys. Soc., Bombay, 1843, p. 68, also reports a case.

[3] Archiv. Gén., 1863, i, 144.

found high up in the left external carotid, on which a secondary thrombus had formed nearly down to the bifurcation of the primitive carotid. Pain appeared from the jaw to the temple on the twentieth day of typhoid, two days later the ear was cold and violet, the artery pulseless, and the gangrene rapidly extended to the entire left side of the head and face, involving even the bones. I have found, however, but eight other cases in which the embolic nature of the primary obstruction was clear; but often the want of a minute examination of the clot renders the report useless; and sometimes probably the primary embolus is so overshadowed in size and importance by the secondary thrombus as to be overlooked.

The third factor, the mechanical difficulties of the distant circulation, combines almost inextricably with the weakened heart in producing the spontaneous coagula or thrombi. It is, nevertheless, clearly the principal factor in precipitating the gangrene in the lower extremities. Not only, however, are the inferior parts of the body thus involved in gangrene, but the frequency of venous thrombi, and the resulting phlegmasiæ in the same region, is a strong argument in the same direction. Bouchut[1] found in 51 cases of non-puerperal venous coagula, that 44 were situated in the pelvic, femoral, or tibial veins. I have memoranda of 63 cases of venous coagula following the continued fevers in which the site is stated. Only two cases involved the upper extremity alone, and were both followed by gangrene; one involved both the arm and leg; all the other 60 cases were limited to the lower extremities.[2] Both forms of coagula—the arterial and the venous—form most frequently, during or just after the period of greatest cardiac weakness—a weakness felt most at such distant points as the legs. Of 18

[1] Gaz. Méd. de Paris, 1845, p. 241.

[2] See a very interesting case which got well after a second attack of typhus thirty years later. Stokes on Fevers, Phila., 1876, p. 249 (republished in Med. News and Library).

arterial cases 12, and of 43 venous cases 24 occurred in the second and third weeks of the fever. Moreover the preceding circumstances, such as famine, individual poverty, and the deprivations of war, are such as impair the nutrition and the circulation in the peripheral districts of the body. The coagulation also takes place at points mechanically favorable to slowing of the currents, *e. g.*, the bifurcation of arteries and the valves in the veins.[1] In the veins at least, as described by Humphrey, the clots are sometimes laminated, the outer layers of decolorized fibrin, and therefore the oldest, and the centre, a bar-like recent coagulum of dark or black blood.

Once that the obstruction exists in the artery, it extends by additional coagulation, so that the collateral circulation may be widely and rapidly cut off. The progress of the clot can often be watched from day to day by the progressive annihilation of the pulse, first, for example in the tibial, then in the popliteal, then in the femoral or higher; and by the parallel progress of the gangrene. In cases of recovery this cessation of the pulsation and the hard tender cord in the course of the vessels are of course, the only, but sufficient proof of their occlusion. That gangrene follows so much more frequently in febrile thrombosis than after the traumatic thrombus which accompanies ligation, is not surprising, in view of the condition of the blood, the general enfeeblement, and the more widespread arrest of the collateral circulation. Yet, on the other hand, pyemia, which has so much to favor it, especially in the cases of venous thrombi, is a rare sequel. Even when it does follow, it is in most cases apparently the secondary result from the septic influences arising from the gangrenous parts.

The circulation in the artery being cut off, it is not strange that clots should follow in the veins, but even where both are

[1] See a carefully reported case of Phlegmasia by Cole, Med. Times and Gaz., 1875, i. 5.

obstructed, moist gangrene rarely follows. The foot especially generally mummifies. But this is not always the case. Occasionally the gangrene is moist from the beginning, from early obliteration of the vein, or having begun as dry gangrene, *e. g.* of the foot from a popliteal clot, suddenly both the femoral artery and vein may be obstructed, and a moist gangrene of the leg or thigh be added. Gangrene from venous obstruction alone is very rare. It is more apt to follow in the arm than the leg.[1]

Coagulation of the blood may also be caused occasionally by direct mechanical causes in fevers, as in a case given by Jaesche, in which a swollen gland surrounded the common iliac artery at its bifurcation and caused a clot, probably by direct pressure or by induced arteritis.

2. The second variety of spontaneous gangrene is that in which *no clot* apparently exists—certainly no such clot as is commonly designated either an embolus or a thrombus, that is, a local clot in an arterial trunk of some size which cuts off the circulation in the tissues supplied by its branches. But even in these cases I believe that the conditions affecting the circulation already so fully considered, will more readily and rationally explain the causation of the gangrene than any specific action of the indefinite though undoubted poison of the fever. Coagulation I believe still to be the cause, but not in the larger trunks. It begins rather as a blood stasis in the capillary circulation. The parts in which the often extensive coagulation takes place are at once struck with gangrene, and, as the blockaded vessels themselves are all involved in the general destruction of the gangrenous tissues, all evidence of the nature of the lesion is thus destroyed. This form of gangrene occurs generally in the nose, ears, penis, perineum,

[1] Hueter (Virchow's Archiv, xvii, 48) records, however, a very interesting case of gangrene of the right leg following a spontaneous clot without assignable cause, the vein wall being healthy.

labia, feet, and occasionally the fingers, that is, in parts supplied by no one large vascular trunk, but by many smaller branches; not only in parts distant from the centre, such as the feet, but in parts which lose their heat most readily by reason of their thinness and small size; and parts irritated, it may be by local discharges. Very probably, also, it may be due to the fatty degeneration of the smaller arteries as observed by Hoffman, Zenker, and Ponfick. Raymond (and following him, Fischer and Estlander) ascribes it to a spastic ischæmia, from contraction of the arterioles. The frequent bilateral or symmetrical character of this variety, to which Raymond has called especial attention, would indicate, at least, the probability of some such central cause. Whether, if it exist, it be the direct result of irritation of the nerve-centres, as is seen in the other nervous phenomena of fever, or whether it be a reflex spasm caused by the circulation of a deteriorated blood, similar to that to which Dr. George Johnson has attached so much importance in Bright's disease, we can at present only surmise.

The symptoms of gangrene are marked and characteristic. Towards the end of the fever, especially in the third week, or early in convalescence, as weakness is giving place to strength, and the brightest hopes of speedy recovery are cherished, sudden, severe, and persistent pain is felt. Usually it is at the seat of the impending gangrene, though not uncommonly at the clot itself, radiating thence to the periphery. In the lower extremities it is often felt in the ball of the great toe or in the heel. It is followed by numbness, coldness, loss of sensation, and sometimes of motion, and in a short time discoloration and all the other usual evidences of gangrene appear. Sometimes, but not usually, these local symptoms precede the pain. If the distal vessels be examined, the pulsation will be found feeble or utterly extinguished, while higher up at the seat of the obstruction they will be changed into moderately firm but

tender cords in which we may sometimes differentiate the artery from the vein—an important point in prognosis. Week by week, sometimes day by day, the progress of the coagulum may be traced upwards by the abolition of the pulsation and by the upward march of the gangrene. If old cicatrices exist they will be among the earliest parts to yield. Blebs may form in the early stages, but most frequently they will dry up and the parts will mummify, although, as already indicated, moist gangrene may supervene if a large clot form higher up or if the veins are extensively obliterated, thus involving great masses of moist tissue, such as the thigh, in sudden ruin.

Life may be rapidly destroyed, as in a case recorded by Barker and Cheyne,[1] in two and one half hours after gangrene began in the nose; but more commonly days or weeks will elapse during which nature as usual makes a powerful effort to rid herself of the dead parts by the establishment of a line of demarcation. On the establishment of this, the pain often ceases.

If recovery follow, the circulation is carried on by collateral branches, or in very rare cases the artery again becomes partially pervious.[2] This last result Humphrey has shown to be not infrequent in veins, and Pètres[3] has recently elucidated its mechanism through the extension and coalescence of the vasa vasorum.

In the variety of gangrene without a thrombus, the symptoms will vary somewhat. It is not so uniformly in the lower extremity, and is much more frequently symmetrical. If small in extent, pain is not apt to be a leading feature. The onset is often earlier, and from the nature of the case its progress is sharper and its limits much more quickly defined, so that usually, within a few days at least, the boundary of the gan-

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[1] Vol. i. p. 232. [2] Patry, Archiv. Gén., 1863, i. 136.
[3] Edinb. Journ., Aug. 1875, p. 175, from Le Progrès Méd.

grene is pronounced, since it does not progress with any gradually growing thrombus. For the same reason it less frequently returns in the stump after an amputation. Its area also is usually much less than those cases in which a thrombus exists, rarely extending in the leg beyond the foot or ankle; and it rarely involves surrounding parts to a large extent, if it occur in the nose, ear, genitals, etc. Sometimes, however, it may extend more widely, as in a case of typhus and starvation, mentioned by Lyons,[1] in which the patient walked to the workhouse, and on baring his chest the whole of the right side was "a dark, olive-green, jelly-like, tremulous mass." The abdominal wall is somtimes similarly involved. The probably irregular area in which the stasis of the blood will take place in this form, also accounts for the great irregularity generally seen in the line of demarcation; whereas, if a clot exists, it is apt to be fairly even. This sudden history is usually followed by a speedily decided issue. Death follows quickly, or reaction and recovery set in within a short time, instead of hanging in the balance for months.

The *results* of spontaneous gangrene vary much according to its situation and extent. In the extremities, if life be saved, the result is usually an amputation, either by nature or by the surgeon. In the nose, it may perforate the septum or destroy the entire organ to a greater or less extent. During some civil, as well as military, epidemics of typhus, this seems to have been a favorite spot for its beginning, so that the disease was popularly known early in this century as the "Blue Nose";[2] and inspired terror whenever it appeared. In 1834, Mauthner says, it was an extremely common result, seen in all the military hospitals, and "all hope was gone as soon as this dreadful symptom was seen." Another not infrequent form is

[1] On Fever, p. 191.

[2] See Mauthner, Kraft, Gutberlet, Wendelstädt, and Barker and Cheyne. i. 232.

noma, or cancrum oris. This is especially frequent in children and in the army. Murchison speaks of it in the Crimea, as frequent and invariably fatal; Chénu, however, in his report does not name it. Its ravages are extremely extensive, often involving even the bones.

The ear, also, and the eyelids are sometimes destroyed. From each of these, singly or all together, the most frightful deformities often follow, which require the utmost ingenuity in the plastic operations necessary to remedy them. In many cases the gangrene is local and subcutaneous, producing necrobiotic masses of tissue, which are, I believe, often, if not generally, the cause of the abscesses so commonly seen in all parts of the body. Sometimes even the mediastina are opened, the anterior from the chest wall, the posterior from the deep tissues of the neck;[1] unless, by a timely surgical operation, the danger be averted.

The male genitals are occasionally destroyed to a greater or less extent. Except the organic destruction, no special result follows, except, possibly, hemorrhage, for one case is recorded of death from a hemorrhage of ℥xxx from the scrotum.[2]

That the perineum and the female genitals are not more frequently the seat of gangrene, is rather surprising, when we consider the neglected condition of many of the patients and the constant soiling of the parts, as a result of unconscious and unavoidable discharges, especially in females. The troubles of the female generative organs are either distinct external gangrene, or gangrenous ulcers in the vagina. I have found 9 cases, 8 from typhoid and one from typhus; all in young persons from 17 to 27 years of age, except one of 34. In 6 of the cases there was gangrene of the labia, extending sometimes to the perineum and the thigh. At least one case was followed by

[1] See Bibliog. Fraentzel, Werner, and Hoffman, p. 388.

[2] Murchison, p. 194.

contraction of the vulva.[1] In another, reported by Guéneau de Mussy, there was complete occlusion of the vagina and menstrual retention, necessitating puncture, with a fatal result. The ulcers are generally on the posterior wall of the vagina, and in three cases recto-vaginal fistulæ have resulted. One is reported by Lebert, in which, when convalescent in the seventh week, chill, fever, and diarrhœa set in, and four weeks later the fistula was discovered by injection. It was situated in front of the hymen, and was as large as a 5-centime piece. A month later she died of pelvic peritonitis. A second is reported by Liebermeister.[2] It was caused by the sloughing of a large piece of the recto-vaginal septum, in mass. The large fistula thus produced healed without operation. The third case has been under my own observation, in St. Mary's Hospital, for three years past, and is the only case I have found of both recto-vaginal and vesico-vaginal fistulæ. Up to March, 1872, she was perfectly healthy, when, at the age of thirty-four, she had a severe attack of typhoid, for four months, following exhaustive nursing during her husband's fatal illness. About the fourth week the labia sloughed away to a large extent, and both water and feces passed by the vagina. In October 1872, she was admitted to the hospital, under the care of my colleague, Dr. Grove, having two large vesical openings (separated by a slight bridge of tissue), which destroyed the posterior part of the urethra and all the floor of the bladder up to the uterus; and one rectal opening an inch in diameter, one and a half inches above the anus. Dr. Grove operated on her three times unsuccessfully; once on the rectal opening by the rectum, when he divided the sphincter, and twice by the vagina. From Dec. 1873 to Dec. 1875, I have done nine operations. Thrice unsuccessfully I attacked the fistulæ proper, when, becoming convinced that the attempt

[1] Russell, Glasgow Med. Journ., 1864–5, xii. 165.

[2] Ziemssen's Cyc., Amer. ed., vol. i. p. 184.

to close them was hopeless, I proceeded to close the vagina. At first I attempted to preserve and utilize the remnant of the urethra, which gave me great trouble and necessitated several operations; but at the last operation, Dec. 28, 1875, I gave up the attempt, excised the useless urethra, and closed the entire vulval aperture by ten wire sutures. The operation has been a complete success. At present, after nearly seven weeks, she defecates, menstruates, and micturates entirely by the rectum, and without the slightest trouble. She rises usually once, sometimes twice, in the night, and micturates only five or six times during the day. My greatest fear has been that the softened feces would pass into the vagina or bladder and give trouble, but thus far at least, none has arisen, and she is happily rid of the annoyance of four years.[1] In the last four operations, instead of the usual sigmoid female catheter to empty the bladder, I inserted the curved branch of a pocket case male catheter into the vagina and bladder, by the recto-vaginal fistula, thus draining these cavities, while I drained the rectum below the eye of the catheter, by an ordinary drainage tube inserted into the rectum, lest the feces should be softened and then pass into the vagina. They answered admirably. The difficulty in obtaining a cure, I believe lay partly in the inherent difficulty of the case, and partly in her deteriorated health ever since the fever.

The perineum suffers mostly in males as 8 to 3, while in 2 cases the sex is not stated. Typhoid was the cause in 11, typhus in 2. Although not all cases of gangrene, they may be surgically grouped together, since all but one produced perineal fistulæ. The exception[2] was a case fatal from a large

[1] Soon after this was written, a small fistulous opening appeared in the cicatrix, caused probably by the feces. This healed after a thirteenth operation, and now (May, 1877) she has remained entirely well for over fifteen months. The rectum has answered perfectly both for the urine and the menstrual discharge, as stated above.

[2] De Change, Arch. Belg. de Méd. Mil., 1861, xxviii., 126.

abscess around the membranous urethra. Three fistulæ were caused by necrosis of the pelvic bones or sacrum, and nine by gangrenous ulcers, which sloughed not only externally, but in five, certainly communicated with the rectum, and probably did so in others. Except two cases of 21 and 22 years of age, they all occurred (when the age is stated) from 39 to 74 years of age, later in life than most of the other sequels. They arose from the third to the seventh week, that is, during distinct convalescence, and to this is probably due the fact that 10 recovered and 2 died, one from the peri-urethral abscess, the other from hemorrhage upon sloughing into the rectum.

The question of *treatment* of gangrene is, after all, the most important in a practical point of view, and is divided naturally into the preventive and remedial. The general supporting treatment of the disease is, of course, the most important preventive. Next, a careful and repeated examination of the body, especially the parts most likely to be attacked. If gangrene is specifically threatened, stimulation of the circulation, both at the centre and at the threatened spot, is imperative. To stimulate the centre, alcohol in liberal doses is the best remedy, and two extremely instructive cases are given by Stokes.[1] In one, "the surface was cold, and the pulse imperceptible. From the middle of the calf of each leg downwards over both feet, the surface was black, the skin hanging in loose wrinkles, giving an appearance as if the patient had on a pair of black socks." Sixteen ounces of brandy in the first eight hours saved his life. Digitalis might also possibly be used with advantage. The peripheral circulation must be stimulated by such means as will assist the threatened circulation by inducing alternate dilatation and contraction of the arterioles. Permanently wrapping up the part in cotton, and other similar means, will but assist permanent vascular dilatation and stasis.

[1] On Fever, Phila., 1876, p. 205.

The alternation of the two is the condition of health, and its artificial production will tend to restore healthful reaction.

Chapman's ice and hot-water bags to the spine, alternate heat and cold directly to the parts, with proper friction and stimulating liniments, at once commend themselves to us. The constant current battery also may prove an extremely useful aid, since it dilates the deep as well as the superficial vessels, and will aid the collateral circulation.

But suppose gangrene actually occurs, what then? Estlander gives most judicious counsel here. We must remember that good results follow both to life and limb *without* operation, especially if the gangrene be limited and the patient not too exhausted. We must not, therefore, be rash in our interference. If amputation has to be done, the question as to where it should be done, depends on the probable extent of the gangrene; as to when, on the line of demarcation. In the non-thrombotic cases, as the line of demarcation is usually established within two or three weeks, and the disease is not then likely to be progressive, the amputation may be done but little above or even through it. It is, therefore, usually best to wait for its formation. In the thrombotic cases, the clot and the gangrene *are* apt to be progressive. Until the line of demarcation forms, therefore, it is impossible to say precisely where the disease will stop. Yet we can gain some idea of the probabilities of the case from past experience.

If the clot extend no further than the popliteal, the limb may escape gangrene altogether, and if it follow, I have found it limited in 9 cases, to the foot 4 times and to the upper calf in 5; if the clot extend into the femoral, I find the gangrene extended to the upper calf in 6 and to the thigh in 4; if the clot extend above Poupart's ligament, I find in 10 cases it was limited to the foot in one, the calf in 3, and extended above the knee in 6. The results of amputation are good, giving 21 recoveries to 21 deaths, but the recoveries are largely after

amputations in the foot. Before demarcation was established, 5 out of 8 died; after demarcation, 12 out of 22, a mortality, respectively, of 63 and 55 per cent. As a rule, therefore, wait for the line of demarcation, but amputate soon after its appearance; but if danger of septic poisoning, or of speedy exhaustion should appear, amputate at once, at or above the probable limitation of the disease, which, if the femoral be free, will not be, in the majority of cases, above the tubercle of the tibia; but if the femoral be involved, amputation would probably be more dangerous than the expectant treatment. As dead parts slough, they should be removed to prevent septic poisoning. Fortunately there is but little danger of hemorrhage, either primary or secondary, in the thrombotic cases, since the arteries are all plugged securely.

Of course, the ordinary treatment of the gangrenous ulcers and abscesses, especially of the perineum and genitals, should be pursued, but I would especially urge the importance of free incision, especially in abscesses in the vicinity of the anus, and the use of detergent and stimulating washes in the vagina in case of sloughing of the labia, in order to prevent in both cases the establishment of fistulæ.

V. HÆMATOMATA.

The muscular system suffers, in typhus and typhoid fevers, in common with almost every other tissue of the body, undergoing a peculiar form of degeneration, resulting sometimes in rupture and the formation of hæmatomata. These, although not so strictly surgical as some of the other diseases noted, yet, as their proper surgical treatment is so important, I shall notice briefly.

Apparently, the first published case was observed by that shrewd surgeon Velpeau, in 1819, in the post-mortem examination of a soldier at Tunis. "Rupture of the muscles of the

belly," says he, "is not surprising. The organs become so fragile in advanced stages of putrid fever that their rupture is a phenomenon which is easily conceived when the patient in his delirium moves so irregularly." In 1844 Rokitansky noted their relation to typhoid. Virchow studied them also in 1857. In 1864, however, Zenker first studied the subject thoroughly. Since then, but especially within the last five years, they have been frequently observed or studied, mostly, however, from a pathological standpoint.

The muscular changes to be described are so frequent as to be almost an essential part, at least, of typhoid. Yet they are not peculiar to these fevers. They are said to have been met with in phthisis, scurvy, scarlet fever, cholera, pneumonia, dysentery, measles, tetanus, Bright's disease, cerebro-spinal meningitis, muscular traumatism, and I have seen a similar change in the muscles of the abdomen, in cases of large ovarian tumors.

There are two independent forms: 1, a granular degeneration of the muscular fibres, which is least frequent; and 2, a waxy change, which is by far the commonest. In the microscope the muscular tissue presents a glassy, translucent, slightly opalescent, shiny appearance, the fibres being swollen to even double their usual size, and changed to fragile cylinders. Sometimes the muscular tissue resembles even the flesh of fish. The nature of the change is as yet greatly disputed. Erb, Bernheim, and others attribute it simply to post-mortem imbibition; Hayem, to proliferation of the tunica intima, which, with granulo-fatty change in the arterial walls, produces an obstructive arteritis; Zenker ascribes the degeneration to the disturbance of a centre which regulates the nutrition of the muscles; Waldeyer, Hoffman, Ranvier, and Weihl believe that it is a coagulation of the myosin; and Liebermeister that it is due to the long-continued high temperature. Whatever the cause, the muscles become extremely

fragile, and when they are called into play by the distension from meteorism, by the efforts at coughing and other violent respiratory acts, by defecation, rising in bed, the movements of the legs, etc., they rupture with the greatest ease. Spasm or direct violence does not seem to have been noticed in any case. The arteries, which have also undergone an analogous change, are involved in this rupture, and muscular hemorrhages result. These assume three forms, according to their size and mechanical limitation: 1, ecchymoses; 2, diffuse infiltration into the muscular tissue, soaking it with blood; or, 3, distinct hæmatomata, the last being the most important and probably the most frequent. The effused clot, at first hard, well defined, and sharply limited, gradually softens and not infrequently suppurates, thus producing serious abscesses which, unless opened, may even burst into the peritoneal cavity.[1] Meanwhile the swollen muscular fibres gradually undergo re-absorption, until, finally, they disappear entirely, and a new formation of cells takes place in the perimysium, which, according to Hoffmann, first become spindle-shaped, then coalesce endwise with one another, and gradually assume the appearance of striated muscular fibre. Complete repair is then effected. The resemblance of these spindle-shaped cells, which are nascent muscular fibres, to the muscular fibre-cells is most striking, and seems to form a link connecting the two forms of muscular tissue, the striated and non-striated, such as I have long taught to be probable.

Almost all of the muscles may be thus invaded, but the favorite seats both for the degeneration and the hæmatomata are in the recti abdominis and the adductors of the thigh, then in the pectorals, and, as Hoffmann has noticed in 16 cases out of 22, in the diaphragm. The influence of the phrenic lesion in enfeebling the respiration is, perhaps, more

[1] Wenzel Gruber in Jacops' Thesis, p. 42.

serious than has been recognized. Zenker gives the adductors the first place, and Hoffmann reports the adductors involved in the degenerative changes in 75 out of 107, the recti in 87 out of 127. While this may be true of the degenerative process, yet the hæmatomata are certainly most frequent in the recti. Of sixty positions in cases I have collected from every side, they were in the recti in 27, and the "abdominal muscles" in 9, in the adductors but 5 times, and the upper extremities but twice.[1] If in the adductors, they may burrow so as even to strip off the periosteum from the bone. Hæmatomata are even found in the inter-ventricular septum of the heart itself.

Stokes suggests that febrile deafness and hoarseness may result from a similar degeneration of the muscles of the ear and the larynx. There are no post-mortem examinations on which to found such an hypothesis, and the fact that hoarseness and deafness are so often not seen, and that, as I have shown,[2] other and sufficient causes are found at least in the larynx, render the idea scarcely tenable.

Typhoid was the preceding fever in 44 out of 46 cases, but the severity of the fever seems to have but little influence. Nineteen out of 25 cases occurred from 15 to 25 years of age, and 22 were males as against 8 females. They rarely appear before the third week, since the muscular fragility is then at its height. Of 23 cases I find 19 arose in the third, fourth, and fifth weeks of the fever. Regeneration of the muscles usually begins at the third or fourth week, and is accomplished by the seventh, after which time they do not appear. Their period of development is therefore quite sharply defined by the anatomical history.

As in dislocation of the hip-joint, the symptoms are often

[1] In the recti they are, I believe, invariably below the navel, possibly on account of the absence of the support derived from the lineæ transversæ.

[2] Ante, p. 25.

nil. Indeed, of 47 cases, I find 10 were wholly unsuspected until revealed at the post-mortem. The position of the tumor accounts in part for this. Both in the thigh and in the abdominal wall they are almost always in the posterior part of the muscle—a position due probably, to the effect of gravity in the recumbent posture. Often, indeed, they are so deep as to extend to the pelvic and iliac muscles, and in two cases, under the serous coat of the bladder. Hence there is usually little or no discoloration of the skin, though Foucault reports a case with ecchymosis in the hypogastrium, extending later to the scrotum, thighs, and buttocks Swelling is only reported 13 times, and fluctuation but 10 times. Suppuration and softening are but rarely attended with any special fever. Pain is mentioned in 14 cases. Flexion of the legs to relax the abdominal wall, which we would suppose to be frequent, is named but once. The size of these blood tumors varies from that of a bean to that of an orange. If small, they may be obscured by meteorism; the symptoms then being so indecisive, as in many of these surgical sequels, the necessity for frequent and rigid physical examinations is at once apparent. If a sudden and fixed pain exist in the recti below the navel, or even if movements be only hindered or uneasy and painful, a close examination should be made, and if a tumor or only hardness be found, it should be carefully scrutinized, from day to day, especially for the pasty feel and other signs of œdema, and of fluctuation. The differential diagnosis is not usually very difficult. The most likely error, if in the rectus, is that of mistaking it for a distended bladder, but the catheter will at once unmask this error. If in the right iliac region, it may be mistaken for perityphlitis; or in the adductors, for a simple abscess; but, as in point of practice, the treatment of all three would be more or less similar, the error is of less moment than might be supposed. From aneurism, an abdominal tumor, and peritonitis, the differential diagnosis is sufficiently easy.

The *treatment* is important, especially when we consider the results. Of 13 cases opened by incision, only 2 died; of 34 in which there is no mention of an operation, all died. If small, they will either be overlooked until the post-mortem reveals their existence, or if recovery take place, absorption of the clot, and regeneration of the muscle will follow, independent of treatment. If large, every possible effort should be made by poultices, etc., to bring about early softening, and as soon as softened, they should be opened. That the aspirator may be of service, is probable, but as yet it is, I believe, untried.

VI. PAROTITIS.

Parotitis is occasionally an exceedingly important surgical complication, whose onset is always to be dreaded, lest it bring in other evils worse than itself. Murchison believes with Graves that the inflammation begins in the areolar tissue between the lobules of the gland itself, but Hoffmann has unquestionably shown that, at least in typhoid, the pancreas and all the salivary glands are in a state of rapid cell proliferation in nearly every case, and that parotitis proper is merely "an exaggeration of the changes that usually take place in this gland during typhoid fever, and bears the same relation to these changes that ulceration and perforation of the intestine do to the infiltration of the intestinal follicles." This exaggeration he believes to be due to the dense parotid fascia which compresses the gland. But this is not the only role this dense investing fascia plays. The compression of the swollen tissues not rarely produces gangrene, so that the entire gland may slough out in great masses like tow. In a case related to me by Dr. Grove, it involved both glands and proceeded so far that the fingers could almost meet behind the pharynx. The compression also is very favorable to thrombosis, which may extend to the brain by the diploic veins or even to the internal jugular itself. Necrosis and septicæmia not rarely

follow in its track. In two cases I have found facial palsy, from involvement of the seventh nerve. Facial deformity and anchylosis of the jaw are sometimes seen. In none does hemorrhage from the carotid appear to have followed.

The death-rate is largely increased in such cases, since of 352 cases, 125 died and 227 recovered, a mortality of nearly one-third. The sex is named in only 19 cases, of which 14 were males. Contrary to the fact in other complications, except in perineal fistulæ, this disease is most common after 30. Of 211 cases, the average age, according to Murchison, was $31\frac{1}{2}$. It is certainly very rare in children, for I have found but 2 cases under 15. Typhus was the preceding fever in 352 cases, and typhoid in only 26. Most cases do not go on to suppuration, for of 101, I find 40 suppurated and 61 did not. The abscesses generally discharge by one or often by several openings, the external meatus being frequently one of them. As Nélaton has pointed out, even where it has thus opened, if we would avoid burrowing and other subsequent troubles, we must open it still more freely, in order to divide the parotid fascia.

CONCLUSIONS.

If now, by way of review, we cast our eyes back over the general results of all the complications and sequels we have studied, we may arrive at some useful and important conclusions.[1]

1. Typhoid, probably from its usually longer duration, is by far the more prolific source of such surgical troubles except parotitis, especially when we consider that many cases tabulated as typhus are really typhoid. Of 433 cases, typhoid was the preceding fever in 252, typhus in 119, and other forms of continued fever in 62.

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[1] In this summary I have not included the cases of parotitis in the figures.

2. The surgical troubles to be apprehended in typhus are mainly restricted to gangrene and laryngeal stenosis, 103 out of the 119 cases being due to these two classes of disease, while typhoid bears in its train any and all of the forms of disease described.

3. The age is about the usual age of greatest frequence of these fevers.[1] From 15 to 25 years is by far the most frequent decade, counting 133 cases against 129 at all other periods of life. One singular exception is to be made, viz., the articular troubles, and especially dislocation of the hip, 21 out of 23 cases being under 20 years of age, of which 15 were in children under 15—in striking analogy to the frequency of coxalgia in children.

4. Sex is an unexpected and important factor in the predisposition to febrile surgical troubles. Of 303 cases in which the sex is named, 218 are males and 85 are females, or over two and a half to one. What is the normal proportion of the sexes in fever, it is difficult to determine. In nearly 6000 cases of typhoid, Murchison gives the proportions as precisely equal, and in over 18,000 cases of typhus the females were in a decided majority (8871–9267). Estlander's figures would give us a slight preponderance of males, while Liebermeister, in over 2000 cases of typhoid, gives 1300 males and 750 females.

Unfortunately, I omitted to tabulate the number of cases arising in military practice, which I am sure is not inconsiderable; but while this will account to some extent for the predominance of males, it could not be adduced in the cases of arthritis and dislocation, since most of the patients were children, yet the males were in the preponderance.

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[1] Leibermeister gives the ages in typhoid, as follows: 15–30, 1310; 30–71, 394, total 1704. None under 15 were admitted. In typhus, 15–30, 39 per cent.

Murchison gives in typhoid: 15–25, 2752; all other ages, 3159, total 5911; and in typhus, 15–25, 5332; all other ages, 12,806, total 18,138.

5. The period of development is not the initial period of the fever, but first, from its height to its close, that is, the complications, especially gangrene and stenosis of the larynx; and, secondly and most frequently, during convalescence, that is, the sequels. Of 240 cases, only 12 arose in the first week, 38 in the second, and 48 in the third, a total of 98. If we may assume that convalescence, on the average, begins at the end of the third week, then 142 occurred during convalescence, when health is apparently in the near future.

6. The lower half of the body is the especial seat of such surgical troubles. With the exception, of course, of the laryngeal cases and parotitis, of 307 cases 216 occurred in the pelvic region and legs, as against 91 in all other parts of the body. Moreover, the diseases attacking the upper half of the body are limited almost entirely to local gangrenes and caries and necrosis, and they are usually far less severe in type and more limited in extent than those in the lower half. Here, whole limbs are blighted by gangrene, here occur most of the dislocations, the hæmatomata, the fistulæ, here the severest necroses and largest abscesses, and were we to add the long catalogue of bedsores and phlegmasiæ, the preponderance of the lower half of the body in importance would be still further increased.

7. The *diagnosis* is, in general, moderately easy. The danger is not that difficulty of diagnosis may obscure the case, but that the diseases may be entirely overlooked. They occur most frequently in parts of the body covered by the bedclothes, parts which require time and trouble to expose and examine in the routine of an ordinary visit. Moreover, the patient is frequently so apathetic and insensible to pain, that he does not complain, or, if he do so, it is ascribed to the ordinary pains so frequent in the belly and legs in such fevers, or else to delirium itself.

Hence the most important hint I can give in the diagnosis—

and where indeed does the same rule not hold good?—is, that time and trouble *must* be taken, and that no patient, suffering from a continued fever, and especially from typhoid, should escape frequent, minute, complete, physical examinations, in which every part of the body from head to foot should be questioned. Especially should the physical condition of the larynx, the belly, the legs, and the toes, and in children, the hip-joint, be exactly ascertained. This should be done at least every second day, and that too, not only in severe, but in mild cases, and not only during the fever, but especially in early convalescence, for it is in just such mild and convalescent cases that the wariness of the doctor is the patient's surest reliance. Particularly should attention be paid to hoarseness or even the slightest change in the voice, and the larynx be examined at once with the greatest care from day to day, by the eye, the finger, and the laryngoscope, lest sudden œdema or the more insidious and more fatal necrosis of the cartilages be impending. The eye should seize upon any hindered movements, even without discomfort, and no complaint of pain should fall upon a deaf ear, especially if it be in the throat, the belly-wall, the buttock, the hip-joint, the legs, or the toes. True, it may mean nothing. It may be the vagary of a wandering mind. But it may also be, as we have seen, the herald of the gravest dangers whose attack may be entirely repelled or their force broken by heeding this timely warning.

8. The prognosis is naturally unfavorable, yet not to the extent we would suppose from the addition or sequence of such serious disease. Of 383 cases in which the result is named, 220 died and 163 recovered, a mortality of 57½ per cent.

9. Still more clearly I think, after such a review, do we see the powerful influence of mechanical causes as the proximate factors in the production of such troubles, working in conjunction with the profoundly vitiated blood. With the exception,

perhaps, of the almost constant muscular degeneration, and its not infrequent subsequent hæmatomata, these surgical results are not usually primary but secondary processes; not dependent directly on the fever-poison, but its indirect and often distant results; not constantly seen, but incidental, indeed, often rare; not parts of the fever, but its complications and sequels.

Pathologically all these results may be grouped into two categories, viz.: 1. Those in which a clot exists; 2. Those without any clot.

1. Those in which emboli of cardiac origin, or more frequently local thrombi exist, are unquestionably most of the cases of extensive gangrene and phlegmasia. In many other cases in which such a clot is at present unsuspected, I believe that more careful examination will reveal its presence in the smaller vessels, and prove that if venous, it may be a cause of œdema glottidis, and if arterial, of the local necrobiotic processes, which result in necrosis of the bones, and probably of the cartilages of the larynx, and gangrene of the soft parts with its abscesses, fistulæ, etc.

2. Those in which no clot exists, and yet œdema glottidis, dropsy, and dislocation of the hip, gangrene, ulcers, necroses, perichondritis, and other similar troubles occur. These are especially often ascribed to the fever-poison itself, acting locally and producing, for instance, the so called laryngo-typhus, the abscesses and ulcers in the skin and subcutaneous tissues etc., which are regarded as specific. While not denying this view outright, and especially in some cases, I feel still more strongly disposed to look upon them as allied disorders, the immediate results, as in the case of the pneumonia of fevers, of mechanical conditions, which produce a local stasis of the blood followed by œdema, low forms of inflammation or gangrene. True, these results of fever are most frequent in severe cases and severe epidemics, in which the poison

would be the most virulent, but it must also be remembered that such epidemics and such cases are themselves, as a rule, the result of exceptionally depressing pre-existing causes, such as famine and war, want and sorrow. Even simple inanition alone will produce identical results in many cases.

But it is especially when we consider the position of the troubles that this mechanical factor is apparent. Their posterior position, as is seen in the laryngeal ulcers, the perichondritis, the vaginal ulcers, the fistulæ, and in the hæmatomata the posterior surface of the recti and adductors, is most significant. Likewise is the fact that all such complications as we have seen, are especially frequent in the lower extremities, that is, in parts mechanically unfavorable to a ready return of the blood and eminently favorable, if not to thrombosis, at least to stasis.

10. The *treatment* must be bold, but not rash; conservative, but not timid.

Note.—After the portion on Diseases of the Joints was stereotyped, I received a letter from Dr. V. P. Gibney, of the Hospital for Ruptured and Crippled, New York City, giving the results in 860 cases of disease of the joints. The following is the only case which followed any continued fever, and it is not tabulated with the others.

"William H——, æt. 12, presented himself at the out-door department of the Hospital for Ruptured and Crippled, May 2, 1876. His general condition was good. The right hip was anchylosed with the thigh, abducted, semiflexed, and rotated inward, the trochanter carried upward, and the pelvis tilted to the right side. There was apparent shortening of the limb, but the real shortening was not ascertained. The thigh was atrophied three inches. Immense cicatrices of bedsores were found, one over each posterior superior spine of the ilium, one over the right natis, and one over each trochanter major, that over the right being the deeper, and covered by a scab one and three-fourths by one and a half inches.

"Prior to October, 1875, he was in perfect health, but was taken that month with typhoid fever, and lay very ill for six weeks, during which illness, the bedsores formed, and during convalescence the deformity at the hip was observed. This history I obtained from the mother, who was very intelligent. At the time I saw him, the disease was practically arrested."

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[1] In the preparation of this contribution to the Bibliography of the subject, I must acknowledge my very great indebtedness to the card catalogues of the National Medical Library, and of the private library of Dr. J. M. Toner, and my personal indebtedness for many facilities to both Drs. Toner and Billings.

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