I do not claim that these two things always coexist. I showed you, a moment ago, a primitive putrid osteo-myelitis with a non-putrid phlebitis. I shall show you, in other cases, putrid suppurative osteo-myelitis without any apparent phlebitis. The coincidence is nevertheless very frequent, and ought to be considered when the mode of development of purulent infection is discussed.

III. The third piece is the skullcap of a patient in whom we recognized the existence of a fracture of the parietal bone, together with a contused wound of the right side of the head.

Suppurative inflammation had invaded both the external wound and the bone, the parietal bone became denuded by one of those rapid disappearances of the periosteum which occur in acute suppurating osteitis, and the mechanism of which is not yet well known. On the 13th day chills commenced, then followed the cortege of the symptoms of purulent infection, and the patient died on the twenty-second day after the accident.

You know how frequent this kind of death is after compound fractures of the top of the skull. You will read of cases of abscess of the liver occurring after wounds of this kind, and the insufficient theories offered in explanation by J. L. Petit, Bertrandi, Quesnay.[1] Your books will show you that afterwards, according to the valuable works of Dance[2] upon suppurative phlebitis, and those of Breschet[3] upon the large venous canals of the diploë, the opinion was produced that the abscesses of the liver are the consequence of a consecutive infection or mixture of the blood with pus formed in the large bony veins of which I have just spoken. The latter become inflamed and suppurate, as do those of the soft parts, and allow a part of the pus, which is formed by the phlebitis in their interior, to enter into the circulation.

In this specimen you recognize the exactitude of the fact. One of the fragments into which I broke the bone with a hammer shows us an open vein of the diploë. This vein contains serous pus. Undoubtedly here was a suppurative phlebitis. Moreover we followed a veinule leading from the inner surface of the skull to the superior longitudinal sinus. This veinule and the sinus itself inclosed clots without apparent pus, so that we have a suppurative phlebitis of the diploë with a non-suppurative and simply adhesive phlebitis of the veins external to the bone. But I beg you to again notice here, two anatomo-physiological characteristics which escaped the attention of our predecessors. First, this pus which is in the vein of the diploë is serous, of a bad aspect and fetid odour. Secondly, it is not the vein alone which is suppurating; it is the entire diploë of the parietal bone. It is true that you do not see the pus flow; it is not abundant enough for that. But look more closely at this diploë, and compare it with that of the other parietal which I have also broken. The

[1] Mémoires et prix de l'Académie de Chirurgie, Paris, 1747–1797.

[2] Dance, Sur la Phlébite externe et la Phlébite en général. (Archives de Médecine, tome xviii. p. 286.)

[3] Breschet, Recherches Anatomiques, Physiologiques et Pathologiques, sur le Système Veineux, Paris, 1827–29, in folio.

colour is not the same; it is a dirty-gray in the first, and pink in the second. Nor are the contents of the cells the same; yellowish in many points, and blackish in others in the first, where it is composed of pus mixed here and there with blood; it is more purely red in the second, where it is composed of blood and fat. Nor is the odour the same. That of the first comes much nearer putrefaction than does that of the second. In short, here too is suppuration not only of the bony vein but of the whole diploë, and as the contents of the diploë are really analogous, in the normal state, to those of the medullary canal and of all the cavities of the long bones, I consider this also an acute suppurative osteo-myelitis; and on the other hand, as the pus is of bad quality and putrid, and as a fatal infection followed the diploic suppuration, I consider that this suppurative osteo-myelitis is putrid, and, for reasons which I shall hereafter give you, I place in this serious lesion the origin of the purulent infection which carried off the patient.

LECTURE XXVIII.

NECROSIS OF THE LONG BONES.

Its origin is most often traumatic in adults, spontaneous in children and adolescents—Obscurity of former descriptions—Too ready belief in a period of repair—Necrosis is a result of suppurative condensing osteitis, like the hyperostosis which accompanies it—Account of a necrosis of the humerus with invaginated sequestrum—Operation—Persistence of a long cavity in the humerus.

GENTLEMEN: I have often had occasion to show you patients upon whose limbs were fistulæ with longer or shorter suppurating tracts, which ended at denuded portions of the long bones, some of which portions were still immovable, others movable and about to be expelled. You have not forgotten, especially, the femurs, tibias, and humeruses which had been broken by balls, and which, after having been attacked by general suppurative osteitis, lost at different times mortified fragments, which we called *splinters* when they were small, *sequestra* when they were rather large. Nor have you forgotten those adolescents in whom I showed you the elimination of similar splinters and sequestra after spontaneous epiphysary osteitis. Finally, you know that we gave the name *necrosis* to the condition of mortified parts of the skeleton which are destined to be expelled. But you understood and have remembered that necrosis is an incident superadded to suppurating osteitis, when the latter is not putrid and fatal, and especially to general suppurating osteitis, that which occupies at the same time the periosteum, the parenchyma, and the deep parts of the bone, that is, the medullary canal, and the whole thickness of the cancellous tissue, when it is the extremity that is affected. Our authors, in describing necrosis, made a mistake in isolating this deep

suppurative osteitis, or osteo-myelitis, and making of it a special pathological entity. That is true and proper for the very thin flat bones, such as those of the palatal arch, and the turbinated bones. For them the phenomena of the suppurating osteitis are so slight, and the consequences of the loss of substance of the bone, which, moreover, are generally inseparable, are so predominant, that I understand the importance given to the phenomena mortification and elimination by a description which is confined almost exclusively to the necrosis.

But you may have noticed in the shaft of the long bones, in the cases of which I have just reminded you, that a great anatomo-physiological and clinical phenomenon, non-putrid suppurating osteitis, precedes the necrosis, and that another phenomenon, hyperostosis, accompanies and follows it. The necrosis consequently is a consecutive lesion, and, as it were, superadded to two others, suppuration and hypertrophy; and it belongs to a variety of osteitis which, to be well characterized, ought to be called *condensing and necrotic suppurating osteitis*. This connection of course is not sufficient for us to give up absolutely the description of necrosis. On the contrary, I consider this description necessary for those cases in which the disease has reached a period at which, the suppuration and hypertrophy no longer having any gravity or clinical interest, the mortification and elimination constitute the morbid condition, and alone call for the attention and intervention of the surgeon. But this too ready belief in the entity *necrosis* is based upon a physiological error which has greatly obscured the descriptions, and which it is time to correct. This error consisted in subordinating all the phenomena of the disease to a reparatory effort preceded by a destructive one. Read the works of Troja, Weidmann, Boyer, and all the contemporaneous French treatises upon this subject, you will see that their main object is to show how the bone is renewed, and to present its excess of volume as the result of a reparatory process supposed to be produced sometimes by the periosteum, sometimes by the marrow.

This opinion was based upon experiments made by Troja and Weidmann, experiments which consisted in destroying the periosteum, the marrow, or the nutritive artery of an animal's tibia, and noticing the anatomical phenomena which followed these lesions. These authors found that, after a certain length of time, the central part of the bone, which had been deprived of its nutritive materials, became mortified and surrounded by a bone which they supposed to be new, and to have been furnished by the uninjured periosteum. But to protect this interpretation from criticism, the destruction of the marrow should have been followed by the mortification of the whole thickness of the bone in those animals in which they found central necrosis. Now, this has not been observed, and it is very possible that a part of the thickness nourished by the periosteal and muscular vessels may have escaped the destruction, and that the new bone may have been furnished by this portion of preserved bone, and not by the periosteum. On the other hand, in the experiments in which they destroyed the periosteum, the mortification did not necessarily extend to the marrow, and they were not authorized to say that in such cases

it was this organ that had produced the new bone; for, as in the other case, it might have come from the part of the bone which remained alive.

I have never understood why they have so readily and so generally admitted the reproduction of the bones, either by a pretended medullary membrane, the non-existence of which I long ago demonstrated, or by the periosteum, and why they have had so much difficulty to admit that the bone itself, by its compact as well as by its cancellous tissue, should be able to grow, to vegetate, to produce, in a word, new ossification. In my opinion, it is sufficient to observe the clinical course of osteites, and a few specimens of pathological anatomy, in order to be convinced of two things: 1st. That bones can complete and repair themselves after spontaneous lesions, as I told you took place after fractures, by an augmentation of the nutritive movement in their frame itself, as well as by the hyperactivity of their envelope; 2d. That, moreover, in osteitis in man things take place differently than in experiments upon animals, and that observation of the facts, far from showing a period of repair consecutive to a period of elimination, tended rather to show that destruction was a salutary effort to rid of superfluous matter the bone, which had become too voluminous as a result of the hypertrophy and the osteitis which produced it.

To convince you of the correctness of these ideas, let me remind you of two pieces which I had occasion to show you last year, and which had been taken from two patients who had had for a long time before their death, the one a spontaneous osteitis of the femur, the other an osteitis consecutive to a fracture and the formation of a callus. I insisted upon the capital fact that, in one as in the other, there had been no bony suppuration. The osteitis had remained plastic, to use an expression which I have often employed; the periosteum and the medullary organ had been neither thinned nor hypertrophied. But I showed you in the sawn bone a compact tissue twice as thick as in the normal condition, and much more dense, and a cancellous tissue with smaller cells and stouter trabeculæ than usual. An excess of bony substance had evidently formed, and had increased the volume and the weight of the bone, and the object of this excess had not been to repair a loss of substance, for none had taken place. It was a simple product of the disease, that is, of the osteitis, and I showed you how just and useful was the expression of condensing osteitis invented by Gerdy. But in the cases of which I speak, the condensing osteitis had taken place without suppuration, it had been the plastic condensing osteitis.

Let us now return to the traumatic suppurating osteitis of adults, to the acute spontaneous suppurating osteitis of adolescents. What has the clinical and anatomo-pathological study of them shown us? The clinical study has shown us four things in the succession of observable phenomena.

1st. The destruction of the periosteum to a certain extent.

2d. Suppuration invading the surface, the whole thickness, and the medullary canal of the bone, taking, in short, the extension which it

has in the acute suppurating esteo-myelitis of adolescents, and accompanied, furthermore, by similar febrile phenomena.

3d. The increase of volume, quite similar to that which we found in the simple plastic and condensing osteitis.

4th. Afterwards, and long after this hypertrophy, an elimination of the necrosed portions, a single elimination in some patients, and a repeated one in others, with numerous variations in the volume of the sequestra and the interval of the eliminations, while the disease is prolonged for several months, and often for several years.

The anatomo-pathological study of the few pieces which I have had occasion to show you has shown us:—

1st. A first period, characterized by an injection of the periosteum at the points where it had not been destroyed, by a concomitant hyperæmia of the medullary organ, and finally by the dilatation of the Haversian canaliculi of the compact tissue which may be considered as a hyperæmia or injection of the compact tissue.

2d. A second period, in which the vascularized bone suppurates and increases notably in volume. Suppuration is found in the medullary canal and in all the canaliculi of the compact substance, canaliculi which are the principal theatre of the appreciable anatomical phenomena of inflammation of the bone. But at the same time certain points of the compact tissue, previously hypertrophied, lose the vascularization of which I spoke, and take a more or less eburnated aspect which is explained by a diminution of vitality following the augmentation which was indicated by the enlargement of the vascular canaliculi.

3d. A third period, in which the mortified portions are separated from the rest of the bone by a groove, at the bottom of which lies the pus whose formation coincides with the destruction of the bony tissue which is intermediate between that which is necrosed and that which remains alive. This period is more or less prolonged according as the sequestra are or are not invaginated, that is, surrounded by living bone, and according as the portions destined to die are more or less numerous, and succeed one another more or less rapidly.

4th. Finally, a fourth period, which always appears late, and which is characterized by the cicatrization of the fistulous openings, their adherence to the bone, and the preservation by the latter of a greater volume than normal.

What is to be remarked in this evolution is the formation, from the beginning and before the mortification is realized, of a hyperostosis exactly similar to that which takes place in the cases of non-suppurating osteitis, and in which the parenchyma of the compact tissue shares as well as the periosteum and the marrow. Now I cannot see in the increase of size a reparatory process destined to replace the mortified portions, since it commences before the necrosis, properly so-called. Between plastic condensing osteitis of the long bones and suppurating condensing osteitis, I find this difference, that the former takes place without necrosis, and that the second is easily accompanied by necrosis.

Why and how is this difference? The explanation is easy. I suppose that in suppurating osteitis of the compact tissue the inflammation is severe, and is followed by the obliteration of a certain number of vascular canaliculi by the deposit of too abundant bony layers, that this obliteration may produce mortification at the points where it takes place, and that the latter occurs particularly at the points where the periosteum has been destroyed by the excess of the inflammatory process, the bone being deprived at these points of a part of its means of nutrition, while at the same time those which remain are compressed and afterwards obliterated by the diminution of the calibre of their protecting canals. I do not know if my supposition is correct, but in any case, no one can give in the present state of the science an irreproachable explanation of necrosis in acute suppurating osteitis. The important thing, clinically speaking, is to recognize the phenomena, and not to allow ourselves to be turned from observation of them by the acceptation of theories which themselves are only presumptions, but which, instead of being accepted as such, pass, by being often repeated, for demonstrated truths.

In order not to obscure your ideas upon this subject I must add three more considerations:—

The first is, that, if suppurating osteitis of the long bones is often accompanied by necrosis, yet it is not inevitable, and you will sometimes see the first without the other.

The second is that the necrosis, that is, the mortified part, may be external or invaginated. The external one is unquestionably the most frequent, doubtless because, as I explained it, the previous destruction of the periosteum by an inflammatory or a gangrenous process contributes to the mortification. The invaginated form is found when, the periosteum not having been destroyed, chance or circumstances with which we are not acquainted cause the mortification to take place in the centre of the compact tissue or very near to the medullary canal. It is the custom to say in such cases that the old bone, or a part of the old bone, is inclosed in new bone. That is true in some exceptional cases, in those, for example, in which after an amputation the purulent osteo-myelitis has been followed by the mortification of the whole thickness of the bone to a certain height, and in which the remaining periosteum has furnished, under the influence of its excessive vitality, a new bony substance which forms in fact a real new bone. In most of the other cases the invaginated sequestrum is surrounded, not by a new bone, but by the part of the old bone which has not mortified, which is hypertrophied, and of which the hypertrophy, moreover, is not produced by the periosteum, since the latter is habitually destroyed over a certain extent. Nor is it produced by the marrow, for that too is sometimes destroyed, sometimes invaded itself by ossification, and because also it is too far from the sequestrum for us to admit the formation by it of such a thickness of bone as is sometimes seen.

In short, it is the same with the hypertrophy of necrotic suppurating osteitis as with that which accompanies the non-suppurating osteitis of adolescents. Strictly speaking it is not the result of a special reparatory process. It is due to the nature of the inflammation in the compact tissue. This nature doubtless intervenes when there is re-