tions of the fracture, I accept them as such, and do not claim to be able to avoid them. These irregular calluses, moreover, cause very little inconvenience; on the one hand, they do not interfere in any way with the functions of the limb, and on the other hand, they diminish with time because the point of the upper fragment is absorbed little by little. That which remains would be disagreeable only in case the patient were a young lady and compelled to appear in society with bare shoulders.

To avoid criticism, and to protect his responsibility, the surgeon should, in such a case, give the preference to Desault's bandage, the application of which, because it requires minute care, excuses the imperfection of the result. Indeed he might, in imitation of the fact cited by Mayor,[1] propose manual retention, that is, retention with one hand left permanently upon the fracture, as the only means of certainly obtaining a cure without deformity.

I have a last remark to make upon the treatment of fractures of the clavicle. It is not necessary to leave this bone immovable for a long time; twenty to twenty-five days are sufficient for an adult, fifteen to twenty days for a child.

At the end of this time it is necessary to examine carefully the condition of the bone, and allow movement if mobility and crepitation are no longer found. In those cases where the functions of the limb are slow to become re established, it is due most often to this, that, the apparatus having been kept on too long, the articulations, and especially the small ones of the hand, have taken on, in consequence of the immobility, a greater rigidity than they would otherwise have done.

[1] Mayor, Chirurgie simplifiée, tome ii.

PART III.

TRAUMATIC OSTEITIS AND NECROSIS.

LECTURE XXVII.

TRAUMATIC OSTEITIS OF LONG BONES.

Exposed wounds of the bones—Acute osteo-myelitis, suppurating, and putrid—Its relations with septicæmia (traumatic fever and purulent infection)—Its anatomical characteristics—Its coincidence with simple phlebitis and putrid phlebitis.

GENTLEMEN: I gladly take the opportunity which is offered to-day to show you the pieces coming from three patients who have succumbed, one of them to traumatic fever, the other two to purulent infection, after injuries which had placed the bones in contact with the air and had exposed them to suppurating traumatic osteitis.

I shall speak to you on some other occasion[1] of the relation which exists between acute suppurating osteitis and these two dangerous diseases which it often engenders: traumatic fever and purulent infection or pyæmia.

To-day I leave the latter aside to call your attention solely to the first, the osteitis, which you will not find described in our classical authors with all the details which it deserves.

I. Notice first these two tibias: they belong to that one of our patients who was admitted six days ago for fracture of the middle portion of the right leg complicated by a quite large contused wound. The skin was not gangrenous, the wound was covered with blood clots, exudations, and small superficial eschars, which are seldom lacking in the first period, that during which suppuration of the contused wound is preparing. At the same time an abundant and fetid liquid escaped from the superficial and deep layers. Twenty-four hours after the admission of the patient a burning fever came on, with 125 to 140 pulsations, the temperature rising to 105° in the evening, and varying between 103° and 104° in the morning. Then, after two days, delirium set in, so that the patient had to be tied in his bed; then the abdomen swelled, and at the end of six days, during which the leg had notably increased in size, and the wound had not ceased to furnish an abundant and reddish fetid discharge of which I shall speak in a moment, death occurred.

The fever was not preceded by a chill.

[1] See Lecture xxix.

At the autopsy we found none of the visceral lesions of purulent infection. Furthermore, we found no appreciable lesion, either of the brain, or of the throax or abdomen. The only thing which attracted our attention was the enlargement and friability of the spleen, and the same distension of the intestines which we had remarked during the last days of the patient's life. It is evident, then, that the patient has succumbed to one of these febrile affections without appreciable lesion, upon which we can discuss for a long time without coming to a conclusion, or at least without convincing those who demand visible, material demonstrations in support of the explanation of the death. Without spending any time in this discussion I shall content myself with telling you that in my opinion, according to the negative results of the autopsy, the patient succumbed to what we have called, since Dupuytren,[1] *intense traumatic fever*, and to what we can consider as one of the varieties of traumatic septicæmia, that is, acute and primitive septicæmia.

But let us return to the examination of the bones. This is the tibia of the uninjured side. Externally it shows nothing peculiar. But I have broken it with a hammer to see the inside; you there find the medullary substance of the shaft and that of the cancellous tissue of the extremities with its reddish-yellow colour. Its injection is more marked than in many subjects, and might be considered hyperæmic. But you must remember that nothing varies in different subjects so much as do the proportions of the vascular and fatty parts of the marrow of the bones. Here, the vascular element is greatly developed; but you find nevertheless the yellow colour of the fat which crushes easily under the finger, but is not fluid; when rubbed upon paper, as I rub it now, it leaves large greasy spots. Its appearance is the same throughout; you find in it no effusions of blood, no plastic deposits, and, above all, you do not find the fetid odour which recalls that of putrefaction or maceration of the bones.

Compare this with the other tibia, the one which has been broken, and broken by an indirect cause (the patient had fallen while running, and the upper fragment had pierced the skin). Between the two principal fragments, for the fracture is slightly comminuted, you see softened blood clots which break under the fingers and give a very fetid odour; in the medullary canal of these fragments you find a substance which is much darker than that of the opposite side, is softer, and, above all, has a fetid odour. This substance shows in only a few small places the yellow colour of fat, and it greases the paper much less. At two points you find whitish deposits which are also fetid, and are composed of inflammatory products. These lesions extend about an inch beyond the solution of continuity, and on the upper fragment are continued as far as the cancellous tissue of the extremity.

If you saw the marrow of this side alone you might fail, as happened to many of our predecessors, to appreciate what I consider the very considerable morbid condition which exists. But if you

[1] Dupuytren, Leçons orales, tome vi.

notice the differences between it and the opposite side, you see that this medullary substance is profoundly altered, and that its alterations consist especially in the intimate combination of the fat and the albuminoid substance with blood poured out by the torn vessels, a disappearance, either by absorption or by escape to the outside, of a part of this fat, and finally a putrid decomposition, as well of the infiltrated blood as of the marrow itself and the exudated products. Something has taken place here which is analogous to that which occurs in a wound of the soft parts, and which always takes place in the first period of contused wounds which are preparing to suppurate. The tissues exposed to the air by the accident inflame, become partly gangrenous and putrid. But here the putrefaction has attacked the fatty parts at the same time with the blood inclosed in an almost incompressible cavity, into which the air penetrated easily as soon as a part of the liquid contents escaped. Notice, on the one hand, that this putridity has invaded that one of the fatty tissues of the body which is normally the best protected and most hidden, and which, for this reason perhaps, supports less easily than any other the consequences of exposure to the air.

Even if it should not be admitted that this lesion has the gravity which I attribute to it in considering it the starting-point of the putrid absorption which causes the septicæmia, you will admit that this decomposition of fat and blood at the bottom of a bony cavity ought to be mentioned.

Our predecessors paid no attention to it, and the modern writers who have the merit of having attached their names to the description of osteo-myelitis, especially Raynaud,[1] Chassaignac,[2] Th. Vallette,[3] and Jules Roux,[4] omitted to describe the first period of this osteo-myelitis.

They spoke especially of the period of suppuration. It is true that they said that before the suppuration there was hyperæmia with exudations, but they did not describe this form, at the same time exudative and putrid, which is not yet gangrene, but is very near it, and is even sometimes gangrene in places. It is the possible but not inevitable consequence of compound fractures, and I consider it as one of the forms of osteo-myelitis. I call it *putrid osteo-myelitis* preceding suppuration, or primitive. Taking into account the propagation of the medullitis along the medullary canal, I might even call it *putrid and diffuse osteo-myelitis.*

I looked to see if the neighbouring veins were affected. I could find nothing in the nutritive vein at its point of emergence from the nutritive foramen. But the posterior tibial and the popliteal veins were obliterated by clots, without admixture of serosity or of pus, and without the fluidity and the bad odour of coagulated blood. I did not

[1] Raynaud; De l'Inflammation du Tissu médullaire des Os longs (Archives Générales de Médecine, 1831, tome xxvi. p. 161).
[2] Chassaignac; Mémoire sur l'Ostéo-myélite (Gazette Médicale, 1854).
[3] Th. Vallette; Gazette des Hôpitaux, 1855, p. 594.
[4] J. Roux; De l'Ostéo-myélite et des Amputations secondaries à la suite des coups de feu. (Mém. de l'Acad. de Médecine, 1860, tome xxiv. p. 539, avec plate.)

find the internal membrane of the vein thickened, and I cannot say that this vein was actually inflamed; I might then use the term spontaneous coagulation or thrombosis, accompanying putrid osteo-myelitis. I shall often have occasion to point out to you the frequency of the coincidence of these venous lesions with osteo-myelitis, and to discuss the nature of the first. To-day, I content myself with telling you that if it is allowable to doubt, in this and in similar cases, the reality of the inflammation of the surface of the vein, there are, on the other hand, reasons to admit this inflammation; and, as the blood contained in the veins thus affected presents capital differences, according to whether it has retained or lost its chemical qualities by decomposition, according also to whether it alone fills the vein or is mixed with pus, which can be attributed to nothing else than a phlebitis, I continue to admit phlebitis in cases of this kind. I only say that here the phlebitis is coagulating and not putrid.

II. Examine now this upper half of a right femur.

It comes from a patient 32 years old, whose thigh I amputated eighteen days ago, for a white swelling of the knee. He was carried off by a purulent infection, of which the first chill occurred on the tenth day, and which had been preceded by an intense traumatic fever.

You know that we found metastatic abscesses in both lungs, but to-day I wish you to study especially the lesions of this femur.

You notice, first, that the periosteum has entirely disappeared for a distance of about an inch. What has become of it? Has it been destroyed by gangrene? Has it been absorbed? It is very difficult to give a satisfactory answer to these questions, because we do not see the succession of the phenomena upon the living patient. We perform an operation; we leave the bone covered by the soft parts; and we carefully avoid raising the latter every day to see what is taking place upon the former. And, when we have the opportunity to examine it, either during life or after death, we no longer find the periosteal covering, and we cannot know how it disappeared; but remember that Reynaud[1] has pointed out the remarkable peculiarity that, in such cases, the marrow inflames and suppurates to a height which is about the same as that of the destruction of the periosteum.

We have broken the femur and found the marrow altered a little higher than the periosteum. The alteration consists, as you see, in a diminution of volume, which has left a gap and, consequently, a place for the air; a grayish putrilaginous softening; and an absolute impossibility to discover the normal anatomical characteristics of the medullary substance; here and there very fluid pus; but above all, a fetid odour, as well of the putrilaginous as of the liquid part. I look to see if the Haversian canaliculi also include this broken-down matter and fetid pus; it is not very certain, but that is undoubtedly due to the smallness of the parts; for, by using a glass, I can see in the small open cavity of the canaliculi, a grayish liquid which looks like serous pus; and I do not find the reddish colour which indicates

[1] Reynaud; loc. cit.

the presence of bloodvessels. Most of these vessels seem to have disappeared, and, as the different sections of the compact tissue are less red than those of the other femur, I conclude that this one, although still living, had nevertheless lost a part of its means of nutrition, and, calling to mind those patients who, after suppuration of the bone, have had consecutive necrosis, I find here the first period of a necrosis which would inevitably have taken place if the fatal pyæmia had not intervened.

The chief point in this specimen is the profound alteration of the marrow, its transformation into dirty broken-down matter, and its fetid suppuration; lesions which are doubtless due, in part, to the nature of the inflammation, and mainly to the decomposing influence upon this inflamed marrow of the outer air within the gaping cavity of the medullary canal; and, as the walls of the Haversian canaliculi are also rigid and gaping, I find in it another reason for believing that putrid suppuration in them coincides with that which takes place in the central canal.

However that may be, I see there the second degree of the disease of which I showed you the first degree upon the preceding specimen. It is still a putrid osteo-myelitis, but the putridity no longer affects the infiltrated and effused blood; it is that of the pus and of the remains of the profoundly altered marrow. It is, in a word, consecutive putrid osteo-myelitis, or, if you prefer, putrid suppurative osteo-myelitis.

Remember this fact and its coincidence with purulent infection, for, when I shall discuss the pathogeny of the latter in your presence, I shall refer to it in support of the opinion which I shall then express.

A final remark: the crural vein, which I here show you, is filled with softened clots, and here and there contains fetid pus. Its internal membrane is slightly thickened and friable; its cellular coat is also thickened. These are certainly the anatomical characteristics of phlebitis, and since the matter contained in the vessel has undergone putrid decomposition, like that of the marrow, I conclude that this is a putrid phlebitis. I look in vain for direct vascular communication between the marrow and the crural vein. On the one hand, the amputation having been made above the nutritive foramen, I cannot find the nutritive vein, the only one large enough to be easily dissected out. On the other hand, the other veins, which might perhaps have established the communication, are too small to be perceived, and many of them have doubtless disappeared in consequence of the beginning of the mortification of which the marrow and the compact substance itself were the seat.

You see that I am again, as I was a moment ago, struck with the coincidence of the osteo-myelitis and the phlebitis; I am preoccupied with the possibility of a pathogenic relation between them; I should like to know if the suppurative phlebitis is developed independently, or if it owes its origin to the passage into the veins of putrid matter coming from the marrow through the veinules leading from the bones. But I have no proof, and can only insist upon the coincidence of the putrid phlebitis with the putrid osteo-myelitis.

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