of this kind, and I have shown you that when patients have occasionally escaped suppuration of the bone after gunshot wounds, this fortunate result could not be attributed to our method of dressing the wound, but was due simply to the idiosyncrasy of the patient, the immobilization of the limb, and abstention from irritating explorations.

Finally, do not be surprised that I do not speak of curative treatment; there is none that has much influence upon this dangerous affection. Derivation towards the alimentary canal by means of laxatives is always indicated, also alcoholic stimulation, and even sulphate of quinine and tannin as antiseptics. You have seen me employ these, but your observations and mine have shown that they were not very efficacious.

LECTURE XXX.

PURULENT INFECTION OR PYÆMIA.

Two cases of purulent infection or pyæmia, one following gunshot fracture of the thigh, the other, gunshot fracture of the leg—Anatomical characters and pathogeny of this disease.

GENTLEMEN: We have recently lost two of our patients who were suffering from gunshot wounds. One had had the femur, the other the tibia broken by a ball. In each case the fracture was near the middle of the bone and moderately comminuted. They suppurated; the patients, who were both young but much broken by exposure to cold, forced marches, and loss of sleep, had intense traumatic fever from the beginning, and, in one case on the ninth, in the other on the eleventh day, had an initial violent chill which lasted twenty or thirty minutes, and was followed by great acceleration of the pulse. The tongue became dry, the skin clayey, and then subicteric in color. The chill was repeated once or twice each day at irregular intervals, the strength grew less, slight delirium, diarrhœa, and abdominal tympanites set in. Meanwhile, the suppuration diminished, the pus became thinner, and had that fetid odor which you have heard me compare to that of a mouse. Finally, death took place on the twelfth day in one case, on the fifteenth in the other.

The autopsies were made, and I now show you some of the specimens taken from the bodies.

I. The principal lesions were found in the chest, abdomen, some of the joints, and the broken bones.

A. *In the chest*, each pleural cavity contained a notable quantity of serosity, together with soft false membranes lining the parietal pleura and the lungs, especially at the base and the lower lobes.

After having taken out the lungs and removed the false membranes, I examined the upper and middle lobes, without finding anything worth mention in them. Then, taking hold of the lower lobes, in which lesions are most frequently found in cases of this kind, I felt, in the tissue of the lung along the outline of the base and behind, several hard lumps about as large as peas, over some of which the surface of the lung was of a deeper colour than elsewhere, whilst over others the colour was yellowish. On cutting into these different points, we found different appearances. I here show you two of them, in which the surface of section is black, and from which I can scrape or squeeze a thick sticky liquid which is nothing but blood. But this blood does not flow away freely; after the scraping and squeezing there is still enough left to keep the colour dark and to give the pulmonary parenchyma a firmer consistency at these points than elsewhere.

Here are two other spots, in which you find the centre of the section yellow and the outer part of the same deeper colour as before. The yellow centres yield, when pressed and scraped, a small quantity of liquid which to the naked eye seems to be pus, and in which the microscope shows us purulent globules; but this pus does not flow away in sufficient quantity to leave a cavity behind. In addition to this rather scanty infiltrated liquid, there is a yellow substance, probably plastic matter, united very intimately with the parenchyma of the lung.

Lastly, I show you three other spots over which the surface itself of the lung was yellow. On cutting into them you see real pus flow, yellow and creamy like wholesome pus. After its escape there remains a cavity, here as large as a large pea, there as large as a hazel-nut, the inner surface of which is still lined with a rather adherent yellowish exudation. But the red and yellow centres have disappeared, and with them all that remained of the parenchyma.

You see there, gentlemen, the three stages of what are called metastatic abscesses of the lungs; the brown foci belong to the first, those that are gray in the centre and brown at the periphery to the second, and the purulent collections to the third. The anatomical characters during the first two stages differ from those of ordinary phlegmonous abscesses. In the first, for example, instead of a simple hyperæmia with infiltration of serosity, it seems that we have an ecchymosis, that is, a flow of blood from torn capillaries, and at the same time a thickening of this blood and an intimate union of its coagulum with the infiltrated portion of the parenchyma of the lung.

Dance[1] and Cruveilhier,[2] however, explained these brown foci in another way. They attributed them to small blood clots formed within the capillaries of the lungs in consequence of the development of a capillary phlebitis.

Virchow[3] and the German authors afterwards adopted this explana-

[1] Dance, Article Abces metastatiques, in the Dict. de Méd., in 30 vols. Paris, 1832.
[2] Cruveilhier, Article Phlébite in the Dict. de Médecine et Chirurgie pratiques, in 15 volumes.
[3] Virchow, Pathologie Cellulaire, Paris, 1868, 3d edition.

tion of the formation of the brown foci by clots, but they added that these clots, instead of being formed locally, as Dance and Cruveilhier thought, came from a distance; that they were embolic clots formed in the affected veins near the wound, swept along in the current of the circulation, and stopped in the capillaries of the lungs; and they invented the word infarctus, which bears the signification of an obstruction of the capillaries by fixed, but imported, clots.

I wish I could show you which of these two theories, that of ecchymotic infiltration, or that of embolic clot, is the right one; but I cannot do so.

I see the thick blood intimately mingled with the parenchyma of the lungs, but I cannot make out whether it is contained within the capillaries or whether it is outside of them, and seductive as the theory of embolism may be, I do not find evidence sufficient to make me consider it irrefutable, as most authors do.

I can understand as easily the possibility of an ecchymosis analogous to that which we see formed in the lungs after the ingestion of narcotic and narcotico acrid poisons. I shall tell you in a moment that I consider purulent infection as a septicæmia, as a poisoning; it is possible that the poison acts upon the lungs like those I have just mentioned, that is, that it gives the blood qualities which are irritating and corrosive for certain vascular walls, hence its escape and infiltration. It is certainly difficult to understand why this corrosive action should be exerted upon the lungs and liver more than upon the other organs, why in the lungs themselves the vessels of the base and those of the superficial layers of this base are more often and more easily torn than those of the upper lobe and of the deep portions of the lung.

This difficulty is, moreover, only the prelude of many others which the study of this singular affection will offer us. You will see at every moment that I shall be at a loss how to explain the various phenomena which characterize it.

Look, for example, at these yellow foci of the second stage. By what are they formed? Probably by an exudation of plastic matter at the centre of the red spot. But whence comes this exudation? Is it, as the name would indicate, a new formation substituted for the blood, which, originally arrested, would then be reabsorbed? Is it not rather a transformation of this blood? I cannot give you a satisfactory solution of this question.

And then these cavities of the third stage, how are they formed? Is it again by a new formation, which would presuppose the absorption of the exudation just as its deposit presupposed the absorption of the blood; or might it perchance be a transformation of the original exudation into pus? These are obscure questions, which I ask and do not answer, although I do not hide from you that I incline rather towards the theory of substitution than towards that of transformation.

B. *In the abdomen*, we found the spleen larger and more friable than usual, and filled with very thick black blood. On the convex surface of the liver we found two yellow spots of about the size of a dime;

on cutting them they proved to be formed of a concrete semi-solid substance, resembling that of the second stage of pulmonary metastatic abscesses, and which seemed to us to be likewise formed of plastic material mingled intimately with the tissue of the liver. Furthermore, on cutting more deeply, we found two cavities within its parenchyma, containing thick, yellow, creamy pus. Were these abscesses preceded by a plastic deposit analogous to that which we found on the surface of the liver? It is very probable, although in cases of pyæmia we find metastatic abscesses of the liver more often in the condition of collection than in that of infiltration. You notice here what is generally the case, that there are no brown spots similar to those which, in the lungs, characterize the first stage of metastatic abscesses. The infiltration of blood, if it precedes, must then disappear very rapidly. But as I have never met with it, even in cases in which death took place very early, I believe that it is ordinarily absent, as is also the case in the muscular interstices and the articulations, and that consequently the embolic clots to which so much importance is attached in Virchow's theory must not be considered as inevitably preceding the formation of pus in abscesses of this kind. So long as we do not find sanguinolent foci in the liver we are not justified in believing in a preliminary morbid condition, with or without rupture, of a certain number of capillaries.

C. We opened the right scapulo-humeral articulation of the first patient, who had complained of pain there, and we found in it a considerable amount of pus. I called upon you to notice: 1st. That this pus also was thick, creamy, and presented, consequently, the characteristics of what we term wholesome or laudable pus; 2d. That the synovial membrane, notwithstanding this abundance of pus, presented neither ecchymoses similar to those which we found in the lungs nor the infiltrations, nor even the redness, nor the thickening which in articular abscesses of different origin indicate the existence of a synovitis ending in suppuration. In like manner, as you saw no traces of hepatitis below and about the abscesses in the liver, so you see here no traces of synovitis.

The same thing is seen in the cases in which we find metastatic abscesses among the muscles. There are collections of pus, but about them we find neither injection nor serous infiltration, nor any of the anatomical characters which belong to a phlegmon preceding an abscess.

In a word, purulent collections without preliminary inflammation; that is the most striking point in these so-called metastatic abscesses of purulent infection; for I cannot consider as belonging to inflammation, properly so-called, the lesions of the first two stages of the pulmonary metastatic abscess. These lesions are unusual, bizarre, if you choose, but they are not those of an ordinary phlegmasia.

D. Here now are the femur and the tibia, which were broken by the balls. Both offer the multiple fragments of comminuted fractures, and these fragments lay in the fetid and blackish pus which communicated with the exterior by the openings of the perforating course followed by the projectile. The periosteum still remains on some of

the fragments, but is lacking for about an inch on the two principal ones, the upper and lower pieces.

If you examine the outer surface of the compact tissue of these fragments, you will find there a little reddening, and that enlargement of the Haversian canals which was pointed out by Gerdy[1] as one of the characters of osteitis of the compact tissue. Neither upon this outer face, nor upon the fractured surfaces of the fragments, nor upon what remains of the periosteum, do we find any appearance of the process of consolidation. It is evident that the purulent secretion has taken the place of the secretion and subsequent transformations of the plastic lymph, which, at the stage of injury, if there had been no suppurating wound, would have supplied the cartilaginous callus. If the patient had lived, the granulations of all the surfaces of fracture would have undergone this transformation, as I have previously had occasion to explain it to you (see p. 76 et seq.).

I call your attention particularly to the condition of the medullary tissue of the two bones. In order to understand it properly, I have had recourse to two measures: I have broken the upper and lower fragments of each of the fractured bones with a hammer, and I have taken out and broken in the same way the corresponding bones (femur and tibia) of the other side. I wished to show you the interior of the medullary canals, and to enable you to compare the appearance of the healthy with that of the affected side. What do you see on the healthy side? The medullary canal, and the meshes of the spongy tissue which are continuous with it at the ends of the bone, are filled with rather firm fat, very yellow in one case, pinkish, and at the same time a little more diffluent, in the other. The predominant fact, notwithstanding the differences in appearance and consistency (differences which are very common, and by no means imply an abnormal condition),[2] is the fat. Look now at the fractured bones: at the line of division there is no longer any fatty marrow; in its place is a substance red in some places, gray in others, blackish here and there, rather firm, with a fetid odour, not looking at all like fat, hardly greasing paper when rubbed upon it, and apparently composed of an exudation mingled with what remains of the marrow. Scraping it with a scalpel removes a puriform substance. At a little distance from the line of division, we find here and there collections of pus, instead of the thick substance infiltrated with pus of which I have just spoken, and that continues for about one and a half inches beyond the point of fracture. It is only after reaching the spongy tissue near the extremities that we find normal medullary fat without admixture of pus. There is then in the medullary canal a mixture of plastic deposits, of purulent infiltration and collections, and of gangrene here and there, with diminution and total disappearance in places of the normal fat. This is somewhat similar to diffuse phlegmon with sloughs of the soft parts.

[1] Gerdy, Maladies des Organes du Mouvement, p. 155, Paris, 1855.

[2] The marrow of the bones is always more vascular and more diffluent in the child and adolescent than in the adult and aged, and, in this respect, there are many individual varieties among adults; sometimes the marrow is more, sometimes less, vascular, and these differences are not due to appreciable pathological causes.

The lesion which you find here is that which on other occasions I have shown you under the name of putrid and diffuse osteo-myelitis (see p. 215 et seq.).

E. *Veins.*—We examined the femoral vein near the point of fracture of the femur, and found it filled with black uncoagulated blood, without any sign of pus, and without the changes in the lining membrane which we often see in phlebitis. We also examined the nutrient vein at the nutritious foramen, and found that it also contained no pus and no blood clot. Consequently, in this case, the purulent infection could not be attributed to a suppurative phlebitis. We examined the tibial veins in the other case; one of them contained soft and badly smelling blood clots without pus; the others were permeable. In this also there was no suppurative phlebitis.

II. I am naturally led by what has preceded to ask the pathogenic question: What is the cause of purulent infection, and what relations exist between these visceral, articular, and muscular abscesses, and the suppurating wound which preceded and doubtless caused them? That is a question which has occupied surgeons since the end of the last century, and upon the answer to which they have not been able to agree, for the very simple reason that, in this malady, as in many others, such as most of the contagious diseases, a moment comes when appreciable phenomena are wanting, and when we are obliged to substitute an hypothesis for them, which some are willing to accept, but which the others criticize or reject, asking for a demonstration which no one can give them. The best plan, perhaps, in the presence of this difficulty would be to adopt no theory, and to wait until we should possess one established upon solid bases.

I am not quite ready to adopt this plan, for a reason which I gave to the Académie de Médecine at the time of the discussion on purulent infection,[1] that is, because we have efforts to make to preserve the wounded and those upon whom we operate from this dangerous affection, and it is very difficult to advance safely in the search for prophylactic measures if we are not guided by some idea of the pathogeny.

We can group in three main classes the opinions which have been held upon the mode of development of purulent infection.

1st. *Metastasis and Absorption of Pus.*—The authors who first noticed the connection between internal abscesses and external suppurations, especially Van Swieten, J. L. Petit, and Morgagni, spoke of the transfer of the pus from the wound to the viscera, but without explaining the manner in which this transfer was effected.

Afterwards Velpeau[2] and Marechal[3] were more explicit, and expressed the opinion that the pus of a wound could be absorbed by the open veins, pass thus into the circulation, be deposited in certain viscera, and there form the abscesses known as metastatic. This doctrine rested upon two incontestable facts: the presence of pus in the

[1] Discussion sur l'Infection purulente (Bulletin de l'Académie de Médecine, March 27th and August 16th, 1871, tome xxxvi. pp. 182 and 620).

[2] Velpeau, Graduation Thesis, 1823, and several articles in the Archives Générales de Médecine, in 1824, 1826, and 1827.

[3] Maréchal, Graduation Thesis, 1828.

veins adjoining the wound in many cases of amputation, and the presence of pus in the parenchyma of the lung, liver, and brain. But the absorption of pus by venous trunks opening on the surface of a wound was a pure hypothesis. That venous capillaries are able to absorb liquid substances in contact with them is incontestable, but it was difficult to make a cautious physiologist admit that divided, gaping vessels could do the same. Again, by admitting for the formation of metastatic abscesses the transfer and the deposit of pus in the internal organs, they placed themselves in contradiction with the facts, since we have a first and even a second stage in which is found, at the place which would afterwards be occupied by the abscess, an infiltration first of blood and then of plastic material. Nevertheless the theory was accepted, and reigned for a certain number of years under the name, which you still hear from the mouth of old physicians, of purulent absorption.

2d. *Theory of Phlebitis.*—Dance[1] taught, and after him Cruveilhier, Blandin, and P. Bérard developed, another doctrine, in which the mixture of pus with the blood still appeared as the main explanation of the disease, but in which phlebitis was made to play a great part, as well in the origin of the pus as in the mode of formation of the metastatic abscesses. Dance had discovered in many puerperal women this fact, recognized afterwards by surgeons in patients who had undergone amputation, that the venous trunks of the affected parts, we may say of the wound, were inflamed and suppurating, and that the pus often extended to the neighbourhood of a collateral branch opening into the affected vein. He inferred that the stream of blood through this collateral might carry away the pus and pour it into the general circulation. This pus once in the blood would mingle with it so thoroughly that it could not afterwards be separated from it. But it would alter it and render it so irritating that its passage into certain capillaries, notably those of the viscera, would lead to new phlebites (capillary phlebitis) characterized first by the formation of small clots, and then by the production of pus which would be the consequence of the irritation caused by the clots.

Although based upon an undeniable fact, the presence of pus in the veins, yet this theory was frequently found in contradiction with the facts, and still left a large part to hypothesis. It first supposed that in every case the veins corresponding to the wound which was the starting point of the infection had suppurated; but Darcet, Sedillot, Tessier, in works which I shall presently mention, had been led to modify Dance's and Blandin's theory, because they had often sought for pus in the veins without finding it.

I myself, when examining the bodies of patients who had died of purulent infection, have often carefully dissected the veins of the region of the injury or operation, within which the disease must certainly have had its origin, and I have found no pus in them. The theory of phlebitis supposed moreover that this pus found an easy

[1] Dance, De la Phlébite uterine et de la Phlébite en Général (Archives Générales de Médecine, 1828-29).

passage into the neighbouring veins. But Tessier[1] proved clearly that the inflamed veins contained blood clots as well as pus, and that in many cases these clots lay above the pus, and were so adherent that they must have opposed its migration.

Finally, this theory would lead us to expect that the pus globules that had passed into the blood could be detected in it by the aid of the microscope. But examination of the blood of pyæmic patients has shown that it does not contain more than the usual number of leucocytes.

As for a capillary phlebitis preceding the formation of the metastatic abscesses, that might possibly be admitted for the lungs, but it could not for the liver, the synovial membranes, or the muscular interstices, in which we do not find the purulent collections preceded by those blood clots which may be attributed to coagulation in the veinules.

To these serious objections it must be added that the theory of phlebitis had the disadvantage of leading to no prophylactic measures. Admitting that the disease is engendered by pus in the veins, we can suggest nothing to prevent the production of this result. Surgeons could only bow and wait. You will see that the final theory of which I have yet to speak, the one which I have long accepted, purely hypothetical as it still may be, has the advantage of inviting and justifying prophylactic measures the efficaciousness of which has been proved by experience.

3d. *Doctrine of Septicæmia.*—It was after the objections made by Tessier to the doctrine of phlebitis as the starting point of purulent infection, that the first works appeared in France upon this theory, which in the beginning received no special name, and to which we have since given that of *septicæmia*. This theory explains purulent infection by the absorption and introduction into the blood of invisible and intangible putrid or septic materials produced by the blood, serosity, gangrenous tissues, and mortified inflammatory exudations which are found on the surface of wounds during the early weeks of suppuration and sometimes later.

This theory, generally attributed, even by French authors, to German physicians, had in reality its origin among us. To assure yourselves of this fact you have only to follow the chronological sequence of works published upon this subject.

Darcet is the first, so far as I know, who, without using the word *septicæmia*, formulated in France the doctrine in question. But his work was based upon facts and ideas which were related not to purulent infection itself, but to other diseases which are closely allied to it by their nature and gravity, and which, in fact, belong to the great morbid group now called septicæmia.

Thus Gaspard and Magendie published, in 1823,[2] a series of experiments showing that the injection of putrid matter, notably of pus and urine, into the veins of animals produced artificial fevers similar to putrid and typhoid fevers.

[1] J. P. Tessier, Journal l'Experience, 1838.
[2] Gaspard and Magendie, Journal de Physiologie de Magendie, 1823.

Bouillaud also, in a very remarkable article upon phlebitis, published in 1825,[1] pointed out the frequent coincidence in human beings of phlebitis with symptoms analogous to those of putrid or typhoid fever. He recalled Gaspard and Magendie's experiments, and spoke of pus formed by phlebitis and poured into the general circulation. But on the one hand he occupied himself especially with putrefied pus as the cause of the trouble, and assimilated it to the putrid matter used by Gaspard and Magendie, and, on the other hand, he explained the development of fevers by the passage of deleterious substances into the blood, without paying special attention to the one which has since been known as purulent infection.

Thus, too, Bonuet of Lyons, in 1837,[2] studying the changes of pus due to contact with the air, showed that in consequence of the habitual presence of sulphur in pus the usual products of its decomposition were sulphuretted hydrogen and sulphydrate of ammonia, and that these essentially deleterious substances, when once formed, could be absorbed by wounds and give rise to febrile complications, to which he gave no special name.

Lastly, Prof. Bérard also, long before the publication of his masterly article in the *Dictionnaire* in 30 volumes,[3] taught that a distinction was to be made between purulent infection, which he attributed, as did Dance and Blandin, to the passage of pus into the blood, and another variety of poisoning which occurred later, and which was due to the passage into the blood of the materials formed by the decomposition of the pus, which had been so well studied by Bonnet. Bérard gave the name of *putrid infection* to this malady, similar in its origin to purulent infection, but differing from it by the later period of its development, by its symptoms, its anatomical lesions, and its prognosis. If P. Bérard had applied the same opinion to the development of traumatic fever, and if he had employed the expression septicæmia to indicate these various morbid conditions due to the absorption of deleterious substances differing themselves according to the moment of their formation, he would have completed the edifice whose entire construction has been very improperly attributed to our German contemporaries.

The ground had thus been prepared by previous works when Félix Darcet,[4] leaving the rather vague generalities in which Gaspard, Magendie, and Mons. Bouillaud had remained, and leaving aside also P. Bérard's putrid infection, came to give an explanation, analogous to that of the others, of purulent infection properly so called.

This author reported a series of experiments which showed, according to him, that the decomposition of pus in contact with the air gave rise to two products: the one a subtile intangible poison, which, after having been absorbed, and thus distributed over the entire economy, would produce the phenomena of fever; the other less toxic, solid,

[1] Bouillaud, Sur le Phlébite (Revue Médicale, 1825).
[2] Bonnet, Mémoire sur la Composition et l'Absorption du Pus (Gazette Médicale, 1837, p. 593).
[3] P. Bérard, Dictionnaire de Médecine, article Pus, 1842.
[4] Darcet, Recherches sur les Abcès Multiples et sur les Accidents qu' Amène la Présence du Pus dans le Système Vasculaire (Inaugural Thesis, Paris, 1842).

divided into very fine particles, which could pass through vessels of a certain size, but were promptly stopped in the capillaries, especially in those of the lungs, and might, by acting as irritating foreign bodies there, lead to the formation of metastatic abscesses.

I am glad to recall this opinion, for you see in it the germ of the one uttered by Virchow fifteen years afterwards. The latter author attributes metastatic abscesses to the arrest in the pulmonary capillaries of small so-called embolic clots coming from the veins of the injured region. According to Darcet, also, fibrinous particles were arrested in the capillaries, but he supposed them to come from the pus and not from the blood.

After Darcet, M. Sédillot[1] wrote that purulent infection was not due simply to the passage of pus as such into the blood, but to the passage of putrid substances absorbed from the surface of a wound, and resulting from the ulcerative and gangrenous destruction of the parts in which suppuration was impending. According to this eminent surgeon, in short, the deleterious principles which cause the disease come especially from the mortified and sloughing portion, which we always find upon wounds at the beginning of suppuration.

Then M. Alphonse Guérin,[2] in 1847, likewise opposed Dance's theory, and sought to prove the correctness of the opinion which he afterwards defended warmly before the Académie de Médecine in 1871,[3] that purulent infection is due to the passage into the blood of special miasms scattered through the air, which fall upon wounds and are absorbed through them. According to M. A. Guérin the miasms in question are found especially in hospital wards, in those in which cases of purulent infection are already present, and in all crowded, badly ventilated places, and thus he explains the greater frequency of the disease in hospitals than in private practice, in the city than in the country.

Notice in passing, that this theory, without being strictly proved, had at least the advantage of causing the adoption of the great prophylactic measure, free ventilation and renewal of the air about those who have been wounded or operated upon.

Similar ideas, so far at least as the explanation of the disease by a sort of poisoning is concerned, were maintained by Jules Guérin[4] and Maisonneuve, who, admitting like Darcet and Sédillot the decomposition of the pus, blood, and mortified tissues on the surface of a wound, explained purulent infection by the absorption of these deleterious substances, whose passage into the blood may take place with the pus, but also without it.

I myself had been familiar for several years with these ideas. The autopsies of a certain number of wounded and amputated whom it was my lot to treat in one of the services of St. Louis Hospital, in

[1] Sédillot, De l'Infection Purulente (Annales de la Chirurgie Française et Etrangère, 1843, tome vii. p. 128), and De l'Infection Purulente ou Pyoémie, Paris, 1849.
[2] Alph. Guérin, Thèses de Paris, 1847.
[3] Alph. Guérin, Discours sur l'Infection Purulente (Bull. de l'Académie de Médecine, 1871, tome xxxvi. pp. 202 and 307).
[4] J. Guérin, Bull. de l'Acad. de Médecine, 1871, tome xxxvi. p. 332.