charge of which I had been placed at the beginning of my surgical career, after the bloody outbreak in June, 1848, proved to me that suppurative phlebitis was absent much more often in patients affected with purulent infection than the works of Dance, Cruveilhier, and P. Bérard had led me to believe. And by suppurative phlebitis I mean that of the large veins which run through the soft parts of a limb that has been amputated, or has received a comminuted fracture. On examining the bones of those who had succumbed after multiple suppurations of the skeleton and soft parts, I found, it is true, suppuration and putrid changes in the medullary substance, and I at first supposed that, according to the theory warmly taught by Blandin, the pus, instead of forming in the large veins just mentioned, had started in those of the marrow, and that thus a phlebitis of the bones had been the starting point of the purulent infection.

Seeking next upon the cadaver for the proof of this phlebitis of the bones, I found in many cases the nutrient vein filled with pus. But in many others I found nothing of the kind. I was unable to isolate the veinules of the marrow itself sufficiently to determine the presence of the pus which would demonstrate the existence of phlebitis.

I was, therefore, forced to recognize that if phlebitis of the bones may in certain cases, and when phlebitis of the large veins of the soft parts is absent, be invoked as the cause of purulent infection it cannot be demonstrated anatomically.

But while making these searches, I was struck with the profound alteration of the medullary substance, in which I vainly sought for suppurating veins. I saw that it was gangrenous, mixed with altered blood and decomposed pus, and that it had the odour of putrefaction. I then began to ask myself if these products of putrid osteo-myelitis of which I have already spoken to you (page 214) might not pass into the circulation without being carried there by the pus.

For such a thing to be possible, it was necessary that the surface of the wound and that of the bone should be capable of absorption. Bonnet of Lyons[1] had admitted the first. The teachings of physiology also justified the belief. Nevertheless, I studied this question by experiments upon men and living animals. In 1854 and 1855, I placed a great many times pads soaked with a ten per cent. solution of the iodide of potassium upon wounds at different periods of their course, and half an hour or an hour afterwards I easily detected the iodine in the urine or salivæ by the aid of starch, the mixture of which with these liquids gave the blue colour of the iodide of starch. I have already spoken to you (page 228) of these experiments, and of another which I made upon dogs three different times, and which consisted in forcing into the medullary canal of the femur, by means of a syringe and a tube provided with a screw, which I fixed in one of the walls of this canal, a little of the same solution of iodine. A faucet placed at one of the ends of the tube enabled me to retain the liquid, and to keep it from spreading over the soft parts. In each case I found the iodine in the animal's urine about three-quarters of an hour afterwards.

[1] Bonnet, loc. cit.

In my opinion, therefore, it could not be doubted that the surface of a wound and that of the medullary canal were capable of absorption. A new demonstration of the former has likewise been furnished by M. Demarquay in a paper read before the Académie de Médecine in 1867,[1] and upon which I made a report.[2]

Having acquired these two facts, the presence of putrid substances upon wounds and in the medullary canal, and the possibility of absorption, I continued to examine the large veins in the bodies of those who died of purulent infection, and the interior of the bones which had been attacked by acute suppuration, and I felt more and more convinced that it was not simply the passage of the pus of suppurating veins into the blood which occasioned the infection in question, that if this passage took place consecutively to phlebitis, the pus of the veins probably swept along with itself putrid matter, coming either from the wound or from the bone, and that finally the passage of these putrid substances might take place and cause the affection without the veins sharing in the suppuration, and without the pus serving as vehicle. I developed this opinion in my work upon V-shaped fractures.[3]

But I did not stop at purulent infection. In the same article, as I have already told you (see page 229), I did not hesitate to assign a poisoning as the cause of grave traumatic fever; and as that occurs especially in cases in which the large bones are the seat of suppurative inflammation together with a concomitant wound, I thought that the poison in such a case also was formed out of the medullary substance exposed to the air, and seriously inflamed in consequence of the accident. I further expressed the opinion that other febrile surgical affections, "such for example as that seen after the opening of an abscess by congestion,"[4] after incisions and operations upon the urethra, after the opening of hæmatoceles with thickened walls, that which characterizes erysipelas, and lastly, that called puerperal fever" can be explained by an analogous poisoning.

You see, gentlemen, I also lacked only the word *septicæmia* to sum up and make intelligible my conception of the pathogeny of purulent infection, as well as of the other forms of febrile affections that may complicate wounds at the different stages of their evolution. I admit that I was wrong in not making use of this word which has the advantage of expressing very well the general doctrine I adopted, but I did not think of it.

Since that time I have not failed to expound in my pathological and clinical lectures these ideas of the method of development of traumatic fever and purulent infection, and some years afterwards I saw with satisfaction the confirmation by German authors, by means of experiments upon animals, of the views which I had uttered re-

[1] Demarquay, De l'Absorption sur les Plaies (Bull. de l'Académie de Médecine, Paris, 1866–67, tome xxxii. p. 157, and Mémoires de l'Acad. de Méd. 1867–68, tome xxviii. p. 424.)

[2] Gosselin, Bull. de l'Acad. de Méd. 1866–1867, tome xxxii. p. 930.

[3] Gosselin, loc. cit.

[4] A collection of pus which has migrated under the influence of gravity or other mechanical cause from the point at which it was formed.—Trans.

garding traumatic fever, and those which my compatriots and myself had held upon purulent infection properly so-called. These authors did not quote us, and seemed to think that they were the sole inventors of the doctrine of septicæmia. They fell into this error, doubtless, because they were not familiar with what had been done here. The thing which astonishes me, that against which I protested before the Académie de Médecine,[1] the 27th of March, 1871, is the readiness with which French authors, forgetting in their turn what has been done amongst us to establish this doctrine, have not feared to give it the name of German doctrine. According to what I have just told you the Germans did not invent this doctrine, they only strengthened it, fortified it, and popularized it. Neither did they invent the word septicæmia, which for more than twenty years has had a place in the nomenclature of our learned compatriot Piorry.

And now, gentlemen, after all the researches I have made, after the fresh study of the subject into which I was necessarily led by the long discussion in the Académie de Médecine, I adopt and I offer you the following theory in explanation of purulent infection. This grave disease is composed mainly of two things:—

1st. A set of clinical symptoms which we can classify under the name of fever.

2d. Multiple anatomical lesions the chief of which are the so-called metastatic abscesses.

Let us examine these two points successively.

1st. I consider the fever as the result of a poisoning by toxic materials formed on the surface and especially in the deep parts of wounds.

These toxic materials are often transported by the large veins, within which they are mixed either with pus or with blood. I do not believe that simple non-putrid pus causes the disease in question. If the pus passes into the blood it produces it only because it is mingled with deleterious substances formed either by the decomposition of the pus within a vein which has become the seat of a putrid suppurative phlebitis, or by the decomposition of other parts of the wound, the products of this decomposition being capable of causing suppuration by their passage into the large veins, at the same time that they infect the organism. In my opinion thus there are several gates by which the toxic substances which cause the infection can enter: first the large veins containing altered pus and communicating with the general circulation by some collateral branch, the flow through which has not been obstructed by a clot; next, the veinules which absorb and transport putrid materials without their being arrested in the large veins and mixed there with pus or coagulated blood; and lastly the lymphatics which may likewise convey these poisons without themselves receiving any injury or any irritation from contact with them which might make them suppurate as the veins do. For, notice it well, gentlemen, the lymphatics and the veins act in three ways in the development of purulent infection:

[1] Bull. de l'Académie de Médecine, 1871, tome xxxvi. p. 182.

sometimes they are merely passages and undergo no change, sometimes their internal surface inflames and suppurates in consequence of the contact of the poisons which pass over it; sometimes they take on suppurative inflammation before this passage and as a result of their participation in the inflammatory process which invades all the constituent parts of the solution of continuity, and the pus which has formed within them becoming putrid furnishes the deleterious material which the collateral branches then carry into the general circulation. Thus we explain all the facts which occur since we have recognized the relation between phlebitis and purulent infection, to wit: sometimes the coincidence of the suppurative phlebitis with this disease, sometimes, and more rarely, the coincidence of suppurative lymphangitis, sometimes, on the contrary, the absence of pus in these same vessels, or at least in those of them which anatomical investigation allows us to trace and to observe.

Now, whence came the poisons and how are they formed? I have told you that possibly they may come from pus previously formed within the veins and the lymphatics, and changed either by contact with the air or by the unwholesome nature of the morbid process which gave rise to it. But they often come from the suppurating wound and are carried into the lymphatic and venous trunks by the capillary vessels which have absorbed them on its surface. Their origin then is either in the decomposition of the pus, or of the blood, or of the suppurating and gangrenous medullary substance, or of the gangrenous and broken-down soft parts, or several of these together. This decomposition is still the consequence either of contact with the air, or of the malignity of the suppurative inflammation; but the conditions in which the disease is most frequently developed compel us to attribute most influence to the air. For you know, gentlemen, that the suppurating wounds which are most often complicated with pyæmia are the deep ones, that is, they are those in which, notwithstanding the surgeon's efforts, the pus is retained between the muscular layers, in which, furthermore, the sloughs of the aponeuroses, tendons, and muscles are slow to be eliminated, and remain so long as their separation has not taken place in the depths of the wound.

This forced sojourn of poorly vitalized or quite dead tissues in the midst of soft parts which at the same time admit the air, leads to the decay—that is, a putrid or septic decomposition similar to putrefaction in the open air—of the soft parts which are entirely separated from the organism. You see how much more favourably constituted in this respect superficial wounds are; the pus flows away easily, the eschars are thin and quickly eliminated, and, if necessary, we can ourselves remove them. The prolonged sojourn which is favourable to decay and to the formation of toxic substances does not exist here.

And let us not forget that among the deep wounds the ones which give rise most often to this affection are those at the bottom of which large bones take part in the suppuration.

In fact, before the development of suppurative inflammation, and during the first few days which follow it, something analogous to what we see upon the soft parts takes place within the bones. In

the former there is no suppuration without a certain amount of alteration of the blood remaining on the surface of the wound, and without the production of eschars and of exudations likewise destined to mortification and elimination. When the wound is deep these phenomena appear throughout its whole extent, consequently within the medullary canal, and probably also in the canaliculi of the bone. Suppuration is preceded and accompanied within these canals by gangrene and exudations whose products are altered by contact with the air which has penetrated and become confined there.

Putrid and toxic products, therefore, are formed in every suppurating wound. But when the wound is superficial they are less abundant and remain for a shorter time, and consequently are less liable to be absorbed than when it is deep, or where a broken or divided bone lying at its bottom takes part in the suppuration. In this latter case there are moreover the toxic products formed by the medullary fat, products which may be dangerous in themselves, but which are rendered especially so by their prolonged retention in an open cavity. This, I think, explains why purulent infection is much more common when suppuration of the soft parts is accompanied by suppuration of a bone than when it is not so accompanied.

In short, gentlemen, according to the theory which I propound to you the dangerous alteration of the blood which produces the disease is not due exclusively to pus; it is due to numerous putrid substances, of which some have their origin in the pus, and the others in the decomposed blood, the eschars, and the exudative detritus of the putrid and gangrenous marrow.

But here three objections are made. I do not hesitate to meet and answer them.

They say to me: You attribute a rôle to gangrene and to the detritus which it furnishes for absorption, and yet you are the first to recognize, and you often teach, that destruction of soft parts (normal tissues and tumours) by caustics which produce eschars is almost never followed by purulent infection. That is true, but notice the differences: when we use caustics their first effect is to destroy the lymphatics and bloodvessels, and to cause their obliteration by the coagulation of the blood and lymph, and by primitive adhesion by means of plastic lymph; the eschar is produced immediately, or at least is completed very rapidly, and when decomposition sets in, when suppuration and elimination begin, all communication has long since ceased between the mortified part and the adjoining living tissue. Moreover there is no blood to putrefy, no exudation deprived of vitality, no serosity liable to decomposition; in a word, there are none of the many sources of poison which we find in wounds. The eschars of the latter form much more slowly; during several days they have still enough vitality to carry on exchanges with the living parts, and to communicate to them the putrid substances they have produced; the adjoining vessels are not yet obliterated, they too share in the unhealthy inflammation which invades the entire solution of continuity; and, lastly, there are, together with the eschars, those other

liquid and fatty products which suffer change by contact with the air, and which are not found upon parts destroyed rapidly by caustics.

The second objection is this: You regard as one of the factors the suppuration of the bone and especially of the marrow, and yet you show us every day patients suffering with suppurative osteitis, in the affections generally known as *caries* and *necrosis*, who are not attacked by purulent infection. They live months or years with this suppuration of the bones and die from other diseases. That is all very true; but notice again that it is not the suppuration alone that I regard as a factor, but also the destructions and mortifications that accompany its beginning, and which you do not have when the osteitis advances slowly towards suppuration and when the latter is not carried on in contact with the air. When these ossifluent abscesses of caries and necrosis open and the air penetrates into the cavity, it finds pus and nothing but pus there. It may still cause this pus to putrefy. When the cavity is deep and extensive, as in abscess by congestion, this putrefaction may become the cause of another variety of septicæmia, that known by the names of putrid infection, hecticity, hectic fever. But the air does not meet, as it does in wounds, with those mutiple elements of which I have spoken, blood, fat, mortified exudations; now, it is this multiplicity of elements which gives rise to the septic products capable of engendering pyæmia. In short, a great distinction must be drawn, in this respect, between acute suppurative osteitis and chronic suppurative osteitis. The first produces, together with pus, putrid substances which the latter does not.

Furthermore, we must draw a distinction between the acute suppurative osteitis which is set up without preliminary solution of continuity, and that which occurs after such solution of continuity. The former sometimes causes purulent infection, as I told you when speaking of the spontaneous acute osteitis of adolescents; but the latter is much more liable to do so, though not always and inevitably.

Remember that the essential condition of the production of purulent infection is that the acute suppurative osteo-myelitis should become putrid. Now this does not always happen, and, as I have often told you, our treatment should be directed towards, and should sometimes succeed in, preventing it.

The third objection is as follows: You speak to us of poisons coming from the blood, from serosities, exudations, pus, eschars, and mortified marrow; but now which one of all these furnishes the real poison of purulent infection? And furthermore, when you explain traumatic fever you still take your poisons from the same sources. Are they then the same in both cases? On this point, gentlemen, I do not conceal my embarrassment and my doubts. If I could isolate the organic poisons of a wounded patient, if I could watch their formation, I would tell you exactly where they arise, whether they result from the union of molecules coming from all the altered parts of the wound, or whether they come mainly from certain ones of them. I would tell you also in what the pathogeny of traumatic fever differs from that of purulent infection. But none of these points can be made out by me. I see an important effect produced—a dangerous

fever. I see, as a plausible explanation of this effect, putrid substances and their absorption. But I can go no further, and that is why I told you at the beginning that, whatever we might do, we always brought up in this research against something that was inexplicable and hypothetical.

I presume that the poison of purulent infection has a complex origin, and that the molecules furnished by the putrescent marrow of the bones play a large part in it. I presume that the poison formed during the first few days, that which occasions traumatic fever, is different from that which is developed later and which gives rise to purulent infection. I recognize that according as the symptoms of the presence of the former have been more marked, so is the latter more likely to be produced. I understand how distinguished surgeons, M. Billroth, in Germany, M. Verneuil, in France, seeing so often grave traumatic fever precede and in some sort prepare the way for purulent infection, have been led to think that these two affections are but one and the same, and that pyæmia is the second degree of a septicæmia of which traumatic fever is the first. But upon this point we are all unable to prove anything. We are obliged to confine ourselves to suppositions, to ask indulgence for our theory in consideration of the excellent points it furnishes for prophylaxis as I shall try to show you in another lecture.

2d. Let us now see how the multiple suppurations, often internal, sometimes external, the so-called metastatic abscesses, can be explained by the theory of septicæmia.

Here again I shall be compelled to admit that a rigorous explanation is nearly impossible.

I have already spoken of the original theory, the deposit in all parts of the body of pus taken up or absorbed from the surface of the wound, and you know the reasons why we could not adopt it.

The first was that it is difficult to believe in an absorption sufficiently abundant to supply the numerous and sometimes large collections which we find, especially those which form within the synovial and serous cavities.

Next, the theory being true, the microscope ought to show large quantities of leucocytes in the blood of pyæmic patients; but, as I told you, the blood contains only the usual number. And finally, since the facts have led us to the opinion that it is not pus but invisible and intangible poisons which cause the infection, we cannot admit that the pus is all formed in the blood and has only to be deposited in the organs.

I spoke also of the suppurative capillary phlebitis admitted by Dance and Cruveilhier as a means in the formation of metastatic abscesses. These authors thought that the blood, by admixture with pus, became irritant, phlogogenic, as it is termed to-day, and that, passing into the venous capillaries of certain viscera, especially those of the liver and lung, it produced in them a suppurative inflammation similar to that caused by mercury and other foreign bodies when injected into the veins, as in Cruveilhier's and Darcet's experiments.

I should be willing to accept this interpretation if the metastatic abscess developed everywhere in the same manner, that is, if it passed everywhere through a first stage characterized by the black spot resembling an ecchymosis, and which might possibly be attributed to the stoppage of clots in the inflamed veins. But this first stage is seen only in the lungs, sometimes in the spleen. We do not find it in the liver, where the abscess seems to begin by a yellow spot which is not blood, and which is not pus either. Still less do we find it in the serous and synovial membranes and the muscular interstices, where the pus forms very rapidly without being preceded by any appreciable lesion.

The same objections can be made to Darcet's fibrinous and Virchow's sanguineous embolus. You may possibly accept it for the lung, but you need another explanation for the liver, the serous and synovial membranes, and the muscular interstices.

In presence of these varieties in formation, as shown by anatomical investigation, there is only one thing to be said, and that is that as soon as the blood has become altered by its infection, and the fever has declared itself, the whole economy becomes apt for suppuration. So long as there is no poisoning, the suppuration remains local, and all the efforts of the organism are turned to the process of repair, of which the regular secretion of pus is an essential condition. As soon as poisoning has occurred, the pyogenic aptitude is disarranged; it becomes generalized, and the organism makes pus, at the expense of the altered blood, everywhere except at the point where it first prepared to make it.

LECTURE XXXI.

ETIOLOGY OF SURGICAL SEPTICÆMIA.

General etiology of traumatic fever and purulent infection. 1st. Local or anatomical causes. 2d. Individual general causes—Influence of age, sex, temperament, alcoholic habits, moral emotions, physical suffering. 3d. Atmospheric general causes, vitiation of the air by crowding—Possible absorption of miasms by the wound and by the respiratory organs.

GENTLEMEN: I gave you in a former lecture my opinion upon the mode of development of purulent infection. But that lesson would remain sterile if I did not try to show you how, on the one side, the tangible causes of the disease accord with this pathogeny; and how, on the other, the knowledge which we have of the relations between the etiology and the pathogeny leads to therapeutical and prophylactic notions. But, as a close connection exists between traumatic fever and purulent infection, and since the prophylactic measures