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PUTREFACTIVE PROCESSES IN RELATION TO
DISEASE.¹

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IN introducing this subject I wish to point out that I have not selected it because I possess any claim to a special acquaintance with it, but rather because I consider that it is one of the highest importance to the practitioner; neither do I lay claim to the possession of any special knowledge of bacteriology, for such a claim can only be made by those who have made the matter a special study. But, notwithstanding the truth of this, we are all in a position to take advantage of the results of the labours of bacteriologists, and to apply the knowledge so obtained to the explanation of phenomena met with in every-day practice.

In dealing with the subject I shall not confine myself to the consideration of putrefactive processes arising in connection with the body alone, but shall also deal, to some extent, with the effects in the system of the products of putrefaction generated outside the body, and introduced thereto by ingestion and inhalation.

As preliminary to the discussion of the subject it may be as well to notice the terms which have from time-to-time been employed in designating diseased processes dependent for their production upon putrefaction.

Probably one of the oldest of these terms is *blood-poisoning* (or, more technically, *toxæmia*, *ichorrhæmia*); and, while this is, on the whole, the simplest and most easily understood, it is too deficient in

¹ A paper read before the Midland Counties' Veterinary Medical Association, August 1892.

precision for us to accept it as an absolutely satisfactory term. *Septicæmia* had its origin in the supposition that the blood was actually in a putrid condition (hæmatosepsis), a condition, I need scarcely say, incompatible with the continuance or maintenance of life. In a similar manner the term *putrid* or *septic fever* (*typhohæmia* of Piorry, *hæmatosymosis* in sheep of Simonds) has been employed, and, while it is correct as implying that a form of fever is induced by the introduction of putrefactive products into the blood, it is incorrect as implying that the blood itself is engaged in the putrefactive process.

Pyæmia, again, was long used indifferently with septicæmia, but a sharp line of distinction has for long been drawn between the two conditions; notwithstanding this, however, the two processes are, as I shall hereafter show, often associated.

In 1878 Koch drew a distinction between cases of disease due to the introduction into the blood of the products of putrefaction, and these due to the organisms of putrefaction themselves, distinguishing the former condition as *septic intoxication*, the latter as *septic infection*; and this distinction was acknowledged to be, in the main, correct by the Committee of the Pathological Society of London, which undertook an investigation into the subject in the early part of 1880.

At the present day the term *putrid intoxication* (*sapremia*) is more in favour, but it is a distinction without a difference, and it will, I suppose, like the old term *putrid infection*, have its day; whatever terms we use, they have all, up to a certain point, the same significance.

For the purposes of this paper it is necessary that we should consider briefly the two terms putrid or septic intoxication, and putrid infection. In the former the products only of putrefactive fermentation are absorbed into the blood, and they do not and can not increase therein; the pathological results depend entirely on the quantity (dose) absorbed, and there is no re-production in the blood of the toxic agent; consequently, the blood of the victim, unless supercharged therewith, is not infective to another animal. In putrid infection the organisms of putrefaction gain access to the bloodstream; and not only do they gain access thereto—they either multiply there, or obtain lodgment in some material or tissue contiguous to the lumen of a vessel or the interior of the heart; consequently, the question of dose is of minor importance, and the blood is infective when introduced into the circulation of other animals. The question as to whether the latter process ever occurs in actual practice will be more profitably considered after the question of putrefactive organisms has been discussed.

For a very long period a definite term was applied to those organisms which were believed to possess a monopoly in producing putrefactive fermentation of organic (animal) matter, and that term was "bacterium termo." On this point we cannot do better than quote the remarks of Fraenkel.¹ After remarking that the name bacterium termo was first given by Dujardin and Ehrenberg and accepted by Cohn, he goes on to say, "But such conclusions cannot stand before the requirements of more recent times. They want precisely that proof which at the present day every bacteriological fact must be able to show; they have not passed the ordeal of pure culture and the application of modern methods of examination."

¹ Text-Book of Bacteriology, p. 188.

"If these methods be employed upon the material which is found in the observation of putrefaction, the single bacterium termo resolves itself into a number of the most diverse forms. A simple experiment will suffice to illustrate this, superficially at least. Take a drop of any putrefying liquid, put it into gelatine, and spread it over a glass plate; it will always be found that quite a number of different colonies appear, which, of course, must be carefully examined as to their appearance and behaviour."

"Moreover, the process of putrefaction was attributed, by a very high authority (Pasteur), chiefly to the action of strictly anaërobic bacteria, while the micro-organism described as bacterium termo was certainly not anaërobic."

"Bacterium termo is for us, therefore, an abstract idea, and does not apply to any one species. It should be banished from our nomenclature until some well-defined species is found that performs a particularly important and conspicuous part in the process of putrefaction, and to which we might then justly give the name "bacterium termo" as a title of honour."

Fraenkel goes on to refer to certain species of bacteria described by Hauser under the name of "proteus," and which Hauser believed to be important exciters of decomposition in organic substances containing albumen; but, he observes, "the results which he (Hauser) has made known are not yet so fully established as to be regarded in the light of indisputable fact."

Three species of proteus were described by Hauser, and of these the proteus vulgaris seems to have attracted the greatest attention, as it has been shown by Foà and Bonome that not only does it produce a toxic principle (*neurin*), but that by the use of this agent they succeeded in rendering animals immune to the effects of proteus cultures.

To the three species of proteus described by Hauser there has been added, according to Fraenkel, two others, viz., proteus hominis and proteus capsulatus; and in referring to this fact Fraenkel makes a most important statement which bears not only on the question of the possibility of ordinary non-pathogenic organisms contracting pathogenic attributes, but also on the question of the organisms of putrefaction becoming parasitic in the living body. Referring to the last two mentioned species Fraenkel says, "they seem to be inhabitants of the intestinal canal which are harmless under ordinary circumstances, and only under special conditions—for example when the power of resistance in the tissues is diminished—penetrate into the latter, multiply in the blood, and so take a pathogenic character, which is clearly perceptible also in experiments performed on animals."

The sentence just quoted is pregnant with a meaning of the highest importance to us, and opens up the question as to whether, and if so, under what circumstances, organisms ordinarily innocuous in their character became nocuous. I should not have time to enter into a lengthy discussion as to the possibility or impossibility of such transmutations taking place; fortunately, the matter is simplified for us by the acceptance of the doctrine first enunciated by Pasteur, viz., that certain virulent organisms can be rendered non-virulent, and that their lost virulence can again be restored; it is, in brief, only a question of cultivation, and, so far as the animal body is concerned, of accidental opportunity. The lower organisms are, like their higher prototypes

in vegetation, capable of modification in a variety of ways. We are all conversant with the fact that in horticulture and in agriculture different forms of cultivation favour the growth of diverse plants; nay, more, that in the adoption of new methods of cultivation and of manuring new forms (or rather forms strange to the particular soil) of vegetable life spring up and flourish. The seeds have been undoubtedly in existence, but there has been wanting something necessary to convert their potential into actual energy—and so it may well be with the micro-organisms of disease. Again quoting Fraenkel, “the line which is drawn between the *pathogenic* and the *non-pathogenic* bacteria is by no means so clear as might at first be thought.

A not inconsiderable number of micro-organisms are already known which generally show quite a harmless character, but which under some circumstances may become pathogenic; and many pathogenic species can be induced, on certain terms, to lay aside their noxious qualities and pass over into the ranks of innocuous bacteria.”

Now a few words as to the actual probabilities of such organisms as those under discussion gaining access to the blood and multiplying therein.

It is an acknowledged fact that under certain circumstances, as, for example, when brought into contact with membranes in whose epithelial armour a breach has, by some means, been effected, organisms of putrefaction gain access to the circulation, but if the animal whose blood is so invaded is in vigorous healthy condition they are destroyed by the colytic action of the blood, or got rid of by the excretory organs; thus, they have been found in the milk of milch animals, and also in the urine after experimental introduction; but it is not inconceivable or improbable that under certain conditions, which will be presently considered, the organisms of putrefaction preserve not only the power of reproduction but also the power of exerting their virulent attributes when introduced into the circulation. That they may find their way into the blood, and that they will either be destroyed therein or be expelled therefrom goes without saying, but what proof have we that they may become pathogenic there?

Many years ago Professor Law of Cornell University claimed to have demonstrated in the milk of cows suffering from a low form of fever an organism identical with one found by him in the water of the well from which they drank, and he was able to reproduce or arrest this fever at will by simply withholding or allowing access of the animals to the particular well. Cheyne (p. 31 *Suppurative and Septic Diseases*), referring to Ogston's statement that in septicæmia in man micrococci are present in the blood, says, that this statement has been confirmed by Von Eiselsberg, “who examined the blood of almost all the cases in Billroth's Clinique which were suffering from septic fever, and was able to demonstrate the presence of certain organisms therein.” Even Cohnheim tacitly acknowledges that such organisms may play an important part in the production of infective processes, for he says (p. 584, section II, *The Pathology of Nutrition*, Syd. Soc.'s Publications), “Still less open to objection seem to me the experiments of Koch, who succeeded in distinguishing perfectly the effects of putrid intoxication from those of infection with simultaneous introduction of

organisms simply by reducing the dose of putrid fluid to a minimum. As might be anticipated, the course of the parasitic infective disease was only very slightly influenced by diminishing the quantity of virus; while whatever effects were due to the dissolved poison, *i.e.*, the intoxication, altogether failed to appear, as soon as the amount of the poison fell below a certain limit." Friedberger and Fröhner (*Pathologie et Therapeutique* translated into French by Cadiot and Ries and re-translated into English at my request by my colleague Mr Mettam) say, "There is no essential difference between pyæmia and septicæmia; in the latter, as in the former, the blood-stream is infected by pyogenic germs," and again, "Septicæmiæ are generally infectious diseases in which there does not exist metastatic abscesses or secondary purulent centres." Now! although there exists, legitimately, a doubt as to the power of multiplication in the blood-stream of the organisms of putrefaction, there exists no doubt as to their power of producing putrefactive changes in organs whose vitality has been lowered or arrested by depriving them of nourishment, as *e.g.*, by arresting the circulation locally.

Many years ago, Chauveau published the results of experiments carried out by him on the testicle of the goat. He showed that if the spermatic artery be tied subcutaneously the organ dies but it does not putrefy; but if, previous to the ligaturing of the artery, putrefactive matter be introduced into the general circulation putrefaction of the mortified organ results. Cornil induced a septic nephritis by ligaturing the renal arteries for some hours, and then, after removing the ligature, introducing septic matter into the blood-stream; and Haubner has shown the same thing in reference to the fundus of the bladder.

While much that I have here said in reference to the existence of an actual parasitic infection, may be open to the charge that it is of a speculative character, the same cannot be said in reference to the general aspects of the question, *e.g.*, the action of predisposing influences in favouring the production of putrefactive processes, or of the poisonous action of their products, such as general surroundings, lowered constitutional vitality, mixed infection, and functional or organic disease of important organs; nor can there be any doubt as to the fact that in the vast majority of cases the source of these processes is, primarily, in connection with some dead or dying tissue; that the infection is, in a sense, of local origin, and that the actual materials engaged in producing the clinical phenomena met with are toxines; and, further, that while there may be some doubt as to the actual existence of a general septic malady of a parasitic nature (*i.e.*, excluding such specific diseases as Koch's malignant œdema, Davaines septicæmia, etc.) there can, again, be no doubt of the fact that organisms of putrefaction may be carried by the circulation to dead or damaged tissue within the body, and there become a source of continuous infection; or that they may be carried by emboli which are also charged with the products of putrefactive organisms, and that these emboli may be arrested in one or more of the vessels and induce therein acute inflammation, in the products of which the organisms multiply and become an internal source of general infection.

Some of these aspects of the subject can be considered conjointly, others must be considered separately. The effects of lowered vitality, whether as the result of previous infection or otherwise, are seen in

many ways in general practice. Thus, ewes, improperly fed, as on turnips alone, or exposed to the vicissitudes of the weather or to the effects of insanitary conditions, are not only subject to putrefactive endo-metritis and putrefactive mammitis, but their lambs become the subject of putrefactive omphalitis and its results; and this tendency is at once done away with by introducing a complete change of management. A simple stomatitis in the dog becomes a gangrenous affection when this animal is subjected to the effects of lowering influences. A benign pneumonia in a weakened constitution becomes a malignant malady under the influence of the exhalations from sewage matter. Lambs die in hundreds in the castration season when the barometrical conditions are of a particularly lowering character. Simple catarrh in all animals becomes a malignant (putrefactive) catarrh when the atmosphere they breathe is charged with the products of decomposition, as, for example—cattle carried in the holds of ships during the prevalence of stormy weather, when the ventilation is interfered with. Calves confined in insanitary houses when suffering from diarrhœa are attacked by septic gastro-enteritis; parturient cows subjected to the lowering influences of fatigue (induced by travelling), exposure, and improper feeding develop—very frequently—septic endometritis followed by putrid pneumonia. A simple mammitis in the cow under lowering influences becomes a septic mammitis, *e.g.*, when that animal is confined in filthy byres; wounds and bruises which, under ordinary circumstances would, clinically, be of little importance become the seat of septic processes when animals are subjected to the lowering influences of fatigue and a foul atmosphere; *e.g.*, in horses fatigued by travelling long distances, and then conveyed in the holds of steamers in stormy weather, a simple bruise of the skin and subcutaneous cellular tissue is followed by septic cellulitis, and probably by septic intoxication and death; and if severe bruises are inflicted on the limbs or bodies of horses when labouring under the influence of the more severe forms of influenza and strangles, the probability is that the bruised tissues will become the seat of destructive putrefactive processes which may ultimately kill the victims; and cattle suffering from the effects of foot-and-mouth disease die from septic processes if they are subjected to such influences as those under consideration, as do dogs suffering from distemper or variola when exposed to the effects of insanitary conditions. These practical facts are emphasised by the experimental work of pathologists, which furnishes numerous instances of induced susceptibility in animals naturally immune to certain maladies by artificially lowering their vitality, or by setting up artificial forms of disease.

Not only do lowering or relaxing agencies influence the production of general or systemic putrefactive processes, but they also hasten the advent of such processes locally. And I can give no better example of this than that furnished by an accidental inoculation from which I myself suffered in my student days.

On the occasion in question I had wounded the index finger of my right hand by a spicule of bone produced by sawing through the pubis in order to make a dissection of the urinary organs, and during the course of the dissection, the bladder, which contained a quantity of putrefying urine, was cut into, and its contents escaped and flowed over the cut surface of the bone. For 48 hours no evil effect resulted from the

inoculation, but on the third day I walked some distance into the city for the purpose of obtaining a hot bath; as I returned home it suddenly dawned upon my mind that my hand appeared to my senses to have become as heavy as lead, and on my arrival at home I found the finger presenting all the symptoms of an acute infective inflammation. Here, then, was an inoculation with infective matter, followed by a period of incubation, and this again by a rapid invasion of characteristic symptoms, suddenly induced by the influence of relaxing agencies.

The question of *mixed infection* has, to some extent, been dealt with in the immediately foregoing remarks, but, in view of the fact that it has a wider bearing than this, it is necessary that I should consider it a little more in detail, especially as it refers more to the excretions of micro-organisms than to the micro-organisms themselves, and more, perhaps, to micro-organisms that grow outside the body than to those which multiply within it.

Fraenkel (p. 123) says "Under some circumstances, the excretions of different micro-organisms, which separately are harmless, may unite and develop poisonous qualities. Thus, for instance, we know from the researches of Roger that so harmless a bacterium as micrococcus prodigiosus, which alone is almost entirely innocuous, can, in conjunction with another bacterium which is also non-pathogenic for the animal in question, become a cause of disease."

Cheyne (*Suppurative and Septic Diseases*, p. 82) says, "In some instances it appears that a mixed infection is more dangerous than infection with one species of organism." And Lauder Brunton in his article on ptomaines (*Croonian Lectures, Brit. Med. Journal*, June 15 and 22, 1889) says, "In the same way, it seems not unlikely that cases of acute poisoning by game may be due to the accidental presence of more than one kind of bacillus, leading to the formation of specially poisonous products. More especially is this likely to be the case if one of them is a pathogenic microbe, which has already produced disease in the beast or bird yielding the meat, and if a certain amount of decomposition (putrefaction I suppose) of its tissues has arisen before its death." The last remark is, I need scarcely say, of especial importance in connection with the question of the putrefying flesh or tissues of animals that have suffered from any form of bacterial disease. As to mixed infection by the products of individual micro-organisms, I need only make two references. Thus, Fraenkel (p. 122) says, "The different bacteria do not, as a rule form only one definite substance, but always produce several, which by their common joint effects produce the pathological complex of symptoms which we regard the micro-organisms in question as answerable for;" and Woodhead in the address on "Bacteriology" delivered by him at the recent meeting of the Brit. Med. Association lays especial emphasis on the fact that particular micro-organisms possess the property of producing a variety of processes which go to make the sum of the clinical phenomena of particular forms of disease.

Both functional and organic diseases of important organs exercise a material influence upon the production of disease by micro-organisms. It must be self-evident that if the purifying action of the lungs is interfered with, materials which would otherwise be rendered harmless are not only retained in the blood but also themselves retain their

noxious properties, and that if the excreting organs, especially the kidneys and bowels, are inactive, there is no expulsion of deleterious matter from the system by these channels; but, personally, I attach greater importance to the liver, in this connection than I do to any other organ, especially in reference to the absorption of putrefactive products from the intestines, against which the liver, if active, largely protects the system.

Dr Lauder Brunton (pp. 349-50 *Materia Medica*) says, "The liver therefore seems to have a most important function in destroying the poisonous properties of peptones and perhaps other substances produced during digestion," and after referring to the fact that the poisonous action of conia, cobra poison, and hyoscyamin is materially diminished after they pass through the liver, he assumes (p. 351) that poisons may be excreted along with the bile and in this way be prevented from reaching the general circulation, and that this organ has the power of destroying poisons or even removing them temporarily from the circulation by merely storing them for a time.

In reference to the connection existing between dead or dying animal tissue and the initiation of putrefactive processes, it is a generally accepted theory that whenever any organic tissue is deprived of its vitality it is liable to undergo putrefactive fermentation; this doctrine is only true under certain conditions, and most importantly of all it is in the main true in those cases where such tissue is exposed to the contact with atmospheric air. But while we acknowledge the literal accuracy of this theory, we find certain exceptions to it, *e.g.* in the dead tissue of the lung in tubercular necrosis, in the necrosed tissue of necrotic stomatitis in calves, in the necrosed lung tissue in pleuro-pneumonia, in the sloughing tissues of the limb in purpura hæmorrhagica, in the destroyed tissues of the face, tongue and jaw in actinomycosis, in the necrosed tissue (core) in boils, and in the destroyed tissues of the tail, pelvis, and surrounding parts in inoculation for pleuro-pneumonia, and in any of the tissues in dry mortification. Various influences are in existence here to prevent putrefaction, *e.g.* plugging of blood-vessels and lymphatics, drying and consolidation of tissue; but in some cases, at least, the manufacture, by the micro-organisms which have given rise to the destructive processes, of a disinfecting or an asepticising principle is the means by which putrefaction is prevented or arrested. I have elsewhere pointed out that in this theory may lie the explanation of the fact that a dead pleuro-pneumonia lung does not undergo putrefaction even though air may gain access to the cavity in which it is contained, and I may emphasise it still further by directing your attention to the fact (which I have also placed in record), that no matter how much tissue may be destroyed by irrigation with iodine no putrefaction ever takes place, nor is there any danger of absorption of putrefactive matter after the separation of the slough; the latter being accounted for, however, by the further fact that whenever tissue is destroyed by iodine active granulation takes place in the surrounding healthy tissues before the process of separation of the slough commences. No better example of the difference which arises from the admission or non-admission of air can be given than that with which we are furnished in the relative fate of an unborn dead foetus to which air has gained access and one to which no such access has been

possible. In the former case the body undergoes putrefactive dissolution ; in the latter it becomes converted into a dry inodorous distorted skeleton clothed in skin—*mummified*.

With the exceptions above quoted, it may, I repeat, be laid down as a law that whenever dead or dying tissue, or organic exudations, as lymph, blood, or pus, are attached to or lodge in contact with living animal tissue, or are retained in natural cavities, they serve as a nidus for the development of micro-organisms of certain species, and undergo putrefactive fermentation, as a result of which various principles of a poisonous character are elaborated, become absorbed into the system through the medium of the lymphatics and veins, and produce *septic intoxication*. These poisonous principles—variously spoken of as ptomaines, toxines, toxic-albumens, enzymes, etc.—are of the nature, both chemically and physiologically, of alkaloids, and behave exactly like them when introduced into the system, or even, in some cases, when brought into contact locally with living organic tissue.

As early as 1814 such a substance was obtained by Burrows from putrefying fish. In 1820 Kerner obtained an alkaloid from decomposing albumen the action of which resembles that of atropine. In 1856 Panum described a poisonous principle which came to be recognised as *sepsin*. For many years the term *cadaverine* has been applied to poisonous matter developed in dead bodies, just as the term *putrescine* has been applied to similar matter produced in putrefying albuminoid bodies ; at the present day definite terms have been applied to such alkaloids, *e.g.* *neurin* and *hydrated neurin*, whose action is similar to that of curare ; *muscarin* from poisonous mushrooms and putrefying fish, having an identical action with that of eserine ; *mytilotoxine*, from decomposing mussels ; and *tyrotoxine* from putrid cheese, and, under certain conditions, from milk.

The analogy in action between the poisonous vegetable and animal alkaloids can be shown in every-day practice.

In or about the year 1858 my preceptor, Mr Kettle of Market Drayton, was asked to attend a horse to which had been administered the products of 2 ounces of tobacco as a remedy for worms ; that horse evinced at the outset the symptoms of laminitis, this was followed in succession by enteritis and pneumonia, and reverted finally to laminitis—metastatic inflammation we called it. I have attended mares after foaling in which we had, primarily, a putrefactive endometritis or a putrid decomposition of placenta or of blood clot, followed, seriatim, by laminitis, mammitis, enteritis, and pneumonia ; here also we spoke of the process as a metastatic one in the past, and it is a very convenient term for general use even now. The supposed translation of the morbid action is due to the continued absorption of poisonous alkaloids from the original source, the inflammatory action becoming exhausted in turn in each separate organ attacked.

Again, we are all acquainted with the poisonous effects of wheat on the horse, and of acorns and some forms of distillery dregs or over-ripe rye-grass on cattle, when these alimentary matters are ingested in undue quantities or in unfit condition for digestion. They are each in themselves harmless in small quantities ; but, in large, they induce indigestion, and, as a result of this, fermentation processes are set up and poisonous ferments are formed ; and, in the case of wheat poisoning in the horse, you have often a train of clinical phenomenon identical

with that resulting from toxæmia, barring only the lesions peculiar to the latter; in the case of acorn poisoning you have an acute gastro-enteritis associated with great general constitutional disturbance; and in the case of dreg and over-ripe grass you have indigestion and other forms of stomach derangement (so-called Stomach Staggers), and, in addition to gastric disturbance, you have marked cerebro-spinal and general nervous disturbance.

In reference to the similarity in local action between vegetable and putrefactive alkaloids, I will only observe that it has been for long known, that conine and aconitine, particularly the former, when injected subcutaneously produce a violently destructive inflammation ("Caustic Effect"—Woodhead), equal in intensity, though of a somewhat different character, with that resulting from the action of some of the putrefactive toxines.

Now, as to the conveyance of the organisms of putrefaction to damaged tissues within the body, and to the effects of infective embolism or thrombosis. It must be acknowledged at the outset that the latter takes place much more frequently than the former, and as it is the more important condition of the two I shall direct attention more particularly to it.

As the result of numerous experiments, it has been established that foreign particles of a mere septic character may be introduced into the circulation without leading to the production of anything more than a limited necrosis in the tissues of some organ or part, or the formation of simple abscesses, but that if these foreign particles are saturated with putrefying fluids an infarction is produced whose characteristics are distinctive; and, more, that the area of the destroyed tissues (providing always that the blood-vessels are not destroyed or blocked) becomes a focus of putrefactive infection to the rest of the circulation; we have, in other words, putrid intoxication or septic infection set up. Such an occurrence results more particularly from the conveyance of emboli from broken up thrombi in the uterine sinuses in cases of septic endo-metritis, from the portal vein in septic phlebitis, and in any part where the veins are abundant and their walls comparatively thin, as in the keratogenous structure of the foot and the cellular tissue of the coronet. Thrombi formed in any of the veins or even in the heart may become the seat of putrefactive processes by the conveyance to them of the organisms of putrefaction from some infective source. Two instances, indeed, of putrefactive cardiac thrombosis have come under my observation in cows.

Having considered the methods by which putrefactive processes are set up within or about the body, we must next consider the clinical phenomena resulting from, and dependant upon, such processes.

These phenomena are manifested much more pronouncedly in some animals than in others; and in the horse, perhaps, more than in any other species.

Fever may or may not be present—it depends upon the stage of the malady at which we see our patient, the intensity of the infective processes, the absence or presence of complications, and the nature of the organs involved; it is always less in septic intoxication than it is in septic infection. In all cases, lassitude, depression, inappetence, arrest of the normal functions, suppression or perversion of the secretions, emaciation, and rapid sinking of the eye in the orbital

cavity are common phenomena ; but, perhaps, one of the best of the initial phenomena, especially in septic infection, is rigors or shivering. In the horse we have usually a foul mouth, a dirty reddish-yellow colour of the conjunctiva, yawning and gaping, drooping head and ears, indifference to surrounding objects, a rapid weak pulse, and, as time goes on, diarrhoea—the discharges being of a dirty brown colour and evolving a very foetid odour, tympany of the bowels and, it may be, colic ; scanty, high-coloured, urine ; and, as dissolution approaches, stupefaction, coma, convulsions or delirium ; relaxation of the sphincter ani with expulsion of foetid flatus at each expiration ; coldness of the extremities with rapid lowering of the internal temperature. If blood is withdrawn, it is dark in colour and somewhat tarry in consistence. In the most malignant forms death takes place too rapidly to allow of the production of some of these clinical signs. In the more sub-acute cases the animal rapidly emaciates, and the long hair of the mane and tail becomes loose in its follicles.

In cattle, while on the whole the clinical signs are much the same as in the horse, they are less in degree. The milk should in all cases in milch cows be examined, as there may be bacteria present in it, as in Professor Law's case, or it may possess a putrefactive odour, as was the case in a cow suffering from septic peritonitis which came under my observation some time ago. In cows suffering from putrid parturient intoxication, there is a tendency to circumambulation and to boring of the head into a corner or against some fixed object ; there is also spasmodic movement of the hind limbs and staggering gait. These symptoms are frequently confounded with those of milk-fever.

I have only detected a putrefactive odour given off from the bodies of cattle, or exhaled with the expired air, in two diseases, viz., in malignant catarrh and in cattle plague ; and, in the case of the sheep, in malignant catarrh and in sheep-pox.

It is in the dog, perhaps, that we get the most pronounced symptoms of septicæmia, as in addition to the symptoms observed in the horse we have *vomition*, the vomit being bile-stained and sometimes bloody, and the urine icteric ; there is enlargement of the liver, a dirty chocolate hue of the fæces, a foul odour in the breath, and sordes ; but, most important of all, an intensely hyperæmic condition of the gums commencing as a definite line at the junction of the gum and the alveolus, and extending gradually and evenly over the buccal membrane.

As in other important diseases, so here, the animal isolates itself, seeks warmth, and coils itself up in a listless mass.

In septic intoxication, as, for example, after distemper, the delusions, delirium, and viciousness sometimes simulate the conditions seen in rabies, and especially is this the case when the limbs become eczematous and the animal gnaws and destroys them.

In pregnant animals, especially in the advanced stages, abortion frequently takes place.

If the lungs are the seat of septic processes, the breath contracts a foul and somewhat characteristic odour, and I would here direct attention to a fact which has frequently come under my notice, viz., that if the change is confined to one lung the odour is detected only or mainly in the current of expired air from the corresponding nostril ; there may be a foul discharge from one or both nostrils if the disease

is of the catarrhal form, or there may be hæmoptysis in a greater or less degree—the blood being black in colour and expelled more particularly after coughing.

If we have septic gastritis, vomition in the dog and pig is constant; if muco-enteritis, we have in addition to the foul diarrhœic discharges, tenesmus, subdued colicky pains, and tympany, and, in the cow, moaning.

If we have septic endometritis, we find that there is more or less straining followed by the discharge of foul stinking sanio-purulent matter, with debris of blood clots and placenta; and colicky pains, which, in the mare, may be mistaken for those of ordinary colic, especially as there are also uneasy movements of the hind limbs.

If we have septic (malignant) nasal catarrh there is a bloody muco-purulent nasal discharge and the formation of large ulcers, having a foul aspect, in the Schneiderian membrane, and, in the horse, enlargement of the submaxillary lymphatic glands. This latter circumstance, coupled with the nasal ulceration, may lead to the supposition that the animal is suffering from acute glanders, but ozæna is never present in that disease unless the destructive process extends to the nasal bones, or unless there is hæmorrhage into one or more of the sinuses, and subsequent decomposition of the effused blood.

Local signs of putrefactive inflammation.—In no animal do we obtain a better idea of these than we do in the lamb when that animal is suffering from septic omphalitis.

Here, we have a white skin which under the influence of septic inflammation rapidly becomes red, bluish, or purple in colour, tumefied and softened—these conditions extending by continuity in a forward direction to the sternum, and backwards to the pubis and the inner aspect of the thighs, and ultimately involving the larger joints. The wound in the umbilicus gapes, and discharges a sero-sanguineous fluid which rapidly contracts a putrefactive odour, and which is, as shown by the aid of the microscope, charged with bacteria.

In the case of the udder, we find, that, in addition to the usual physical signs of catarrhal mammitis, we get discoloration of the skin and œdema of the surrounding tissues, and, if fluid is withdrawn from the teats it is found to be a mixture of altered milk, serum, and of blood; and it, also, is charged with bacteria and gives off a foul odour. Frequently the local lesion assumes the character of erysipelas.

After castration we find that the scrotal sac contains a quantity of grumous blood, having a sour odour at the outset, but rapidly becoming foul; the skin assumes a purple hue, loses its normal heat, and ultimately presents the usual conditions seen in gangrene from any cause.

In septic cellulitis the most notable feature of the process at the outset is its diffusive tendency; after a time the skin becomes boggy to the feel and pseudo-abscesses form, which, when lanced, are found to be collections of grumous blood with or without an admixture of pus, or with or without fœtor, and, in muscular parts of the limbs, destruction of the muscle tissue followed by sloughing. Sloughing indeed is the gravest condition here, as we have always to dread destruction of joint capsules and extension into joints; moreover, the process sometimes steals a march upon us, and, instead of extending

up the limb from the foot, for instance, by continuity, it seems to become arrested at a given point, but makes its appearance higher up, as in the pectoral muscles and surrounding tissues.

I had one case (originally a tramp of the coronet) in which the whole of the right pectoral muscles became gangrenous and were removed *en masse* by myself (surgically); but the tissues from below the knee to the elbow were perfectly healthy. This case probably arose from arrest of septic emboli in the vessels; it certainly was not the result of extension of septic phlebitis, and a parallel condition is often seen in the tails of cattle after inoculation with the virus of pleuropneumonia, the secondary lesion first making its appearance at the upper part of the tail, the tissues intervening between it and the primary lesion remaining unaffected.

Post-mortem Lesions.—The muscular tissue of the body of animals suffering from putrefactive disease is usually of a dark colour, soft in consistence, and soapy to the touch; it rapidly undergoes decomposition, and there is frequently sero-sanguineous intercellular effusion and effusion into cavities. In the most malignant cases there may be intra- and inter-muscular hæmorrhage, which, in the case of septicæmia in ewes, is usually localised in the muscles of the quarters. The flesh when heated may evolve a putrefactive odour; in all cases it is absolutely dangerous as an article of diet.

The blood is dark in colour, coagulates imperfectly, and may have a putrefactive odour, but this can only occur in cases where there is absorption of foul gases from some septic organ or cavity, such as the peritoneum; or, where a putrefactive clot is formed in one of the large vessels or in the heart; putrefaction of the general mass of the blood could only occur as a result of necræmia, and it is patent that this would be incompatible with the continuance of life. The most important change observable in the blood, however, is that which occurs in the red cells and in their contents.

The former become disintegrated, the latter dissolved in the blood plasma, which, in consequence, stains any white material over or on to which it flows.

The colouring matter, especially in the large vessels, may form crystals, but I have seen this in cases of relapsing fever in the horse produced by exposure to the exhalations of foul gases from cesspools, quite independently of the usual *ante-mortem* or *post-mortem* concomitants of septicæmia.

The liver is enlarged, congested, and more or less softened, as is also the spleen; the lymphatic glands are enlarged, softened, congested, and hæmorrhagic. The heart muscle is usually pale, soft and friable; rarely congested or hæmorrhagic. There is very frequently sub-endocardial ecchymosis, especially on the left side, with extravasation into the connective tissue investment of the large blood-vessels. The endocardium, particularly of the right heart, may be stained of a deep red hue. The glands are mere masses of hæmorrhagic pulp. If brain or spinal symptoms have existed during life, these organs will be found watery and softened, and, it may be, dark in colour, their meninges intensely congested, and their blood-vessels charged with blood. The serous cavities may contain an excess of the normal fluid, which is frequently turbid; and in cases of localisation of the septic inflammation in serous membranes

they are found intensely hyperæmic, thickened, and frequently coated with lymph which is, in some cases, intensely infective. The mucous membranes are congested, or, when the lesion is localised therein, they are softened, thickened, intensely congested or even hæmorrhagic; they may be eroded, ulcerated or sloughing, the slough being of a dirty greyish or brown colour, and the edges of the surrounding tissue markedly hyperæmic. In the case of the bowels it will often be found that where patches of gangrene have been produced, contiguous portions of bowel have become involved in the destructive process.

In the lungs, the character of the lesions depends upon the exact form of the inflammatory process. If it is of a croupous character we have the usual hepatisation of the lobules, but the consolidated tissue is not so firm as in the case of ordinary pneumonia; there is, instead of a plastic exudation, a quantity of fluid, sometimes blood-stained, in the interlobular tissue, and, if the animal has lived for a sufficiently long period, the involved parts are surrounded by a grey zone of coagulated fibrin and necrosed connective tissue. If it is of a catarrhal character, we find the bronchial membrane either congested or gangrenous, of a deep red, green, or black colour, and the tubes filled with a non-cohesive clot of fibrin of a dirty hue, or with sanguinous pus; the alveoli contains small masses of pus or broken up epithelial products presenting the appearance of grey points, or masses of caseous matter; the veins are packed with semi-solid thrombi, and their internal tunic is usually deeply stained, of a red or black hue; the interlobular tissue is charged with serum.

In the case of embolic pneumonia, the lesion is circumscribed and more or less wedge-shaped; the type is that of a croupous pneumonia, and the involved area is demarcated by a zone of coagulative necrosis. In all forms the lesion is distinguishable from that occurring in ordinary pneumonias by the presence of a putrefactive odour more or less pronounced, and, in the catarrhal forms, certainly, by the presence of bacteria of putrefaction. In the uterus, we find, in addition to the conditions common to mucous membranes, that the venous sinuses are plugged with semi-coagulated blood, and the connective tissue is charged with serum. The cotyledons (in cotyledonous animals) are softened, hæmorrhagic, and enlarged and, later on, atrophied, disintegrated, and of a dirty yellow colour. In old sub-acute cases the cotyledons have disappeared, and their site is marked by a light coloured smooth spot. The mucous membrane is of a dark colour—pigmented. The cavity of the uterus may contain a quantity of foetid chocolate-coloured fluid, or it may contain masses of inspissated pus. Septic cellulitis is marked not only by the universal staining of the tissue and infiltration with sanio-purulent matter, but, more importantly, by the presence of soft, black, coagula of blood in the veins, the tunica intima of which is usually of a dark colour.

Recent abscesses of putrefactive origin are characterised by the caseous nature of their contents, the putrefactive odour emanating therefrom, and the intensely hyperæmic condition of the vessels of the surrounding tissues; old abscesses by the thickness of the abscess wall, the pigmented condition of its internal lining, by its dirty greyish colour and foetid contents. Bone may be either of a black or greyish-black colour; it always evolves an intensely penetrating

sulphurous odour, and when silver is brought into contact with its fluids it becomes tarnished.

All putrefactive tissues give off a more or less fœtid odour owing to the fact that sulphuretted hydrogen is one of the chief gaseous products of putrefactive fermentation, but—and this is an important point—other materials, as indol, skatol, and phenol, of the aromatic group of chemical products, are sometimes formed, and especially, I think, in putrefactive hepatic processes. In such instances the odour is less fœtid, though it may be more pronounced and penetrating. In the case of pregnant animals the tissues and the more vascular organs of the fœtus may be the seat of extravasation of blood more or less pronounced, and bacteria may be detected in the blood. I am well aware that the latter part of this statement may be received by some with hesitation, in view of the belief, so long held by pathologists, that organisms could not pass from the mother to the fœtus. Long before pathologists acknowledged the probability of such transmission taking place I had demonstrated the fact that anthrax bacilli sometimes pass from the dam (in the case of the ewe) to the fœtus. Even now some of those who do acknowledge that inter-placental infection may take place hold that for its production laceration of placental tissue or of placental vessels must first ensue.

The therapeutic aspect is one of the most important in connection with putrefactive processes affecting the animal body.

Locally, all our energies must be directed to the arrest of the extension of the process, and to the prevention of the absorption of putrefactive products. For the disinfection of cavities, solutions of mercuric chloride, phenol, calcium chloride (too often overlooked), permanganate of potash, and iodine are the most reliable agents; but in the case of the uterus it is necessary to utter a word of warning in connection with the amount of force employed in the injection. I have seen at least one case in which the fluid injected found its way into the abdominal cavity *via* the Fallopian tubes. In connection with this matter it may be useful to give a brief summary of an article on "Intra-uterine and Intra-venous Injection of Toxic Agents," by M. Bouchet (*Recueil de Vet. Med.* 30, 5, 92) in which he states that M. Tarnier had observed poisonous symptoms and syncope after intra-uterine injections of:

Corrosive Sublim.		1 p. 5,000.
Acid Phenique.		20 p. 1,000.
Cupri. Sulph.		5 p. 1,000.

He also studied the action of antiseptics when injected into veins:

Acid Phenique.		20 p. 1,000 ; syncope and grave symptoms — recovery.
Cupri. Sulph. and Ferri.		
Sulph.		5 p. 1,000 ; syncope and death.
Corrosive Sublim.		1 p. 5,000 ; no syncope but death by mercurial intoxication.

Pot. Permang. 1 p. 2,000 ; no syncope, no accidents, no appreciable diminution in number of red blood corpuscles.

He concludes that for intra-uterine injection :

Acid Phenique, Cupri. Sulph., and Biniodide of Mercury are dangerous.

Corrosive Sublimate—less dangerous.

Pot. Permang. and Microcidine—not dangerous.

Iodine and mercuric chloride are the most effectual local disinfectants ; but it is necessary that their entrance into the tissues should be facilitated by deep puncture, incision, or irrigation, taking care, in the case of incision, not to wound blood-vessels or healthy tissue.

In all cases where blood coagulæ have formed in cavities or in wounds, they should be carefully removed, or, if this is impracticable, asepticated ; but in the case of extravasation of blood into the subcutaneous tissue as the result of a bruise, great care should be exercised to prevent the ingress of organisms if the blood (or fluid) is removed surgically before plastic inflammation has been set up or granulations have formed.

In the case of the respiratory tract disinfection should be carried out by means of sulphurous acid gas, chlorine, the vapour of phenol, creasote, turpentine, terebene, or iodine ; *par excellence* the two latter—terebene in recent, iodine in advanced, cases.

For the purpose of circumscribing the lesion in septic cellulitis and septic dermatitis there is no plan more effectual than the one I always adopt, viz., the painting of the surrounding skin with strong tincture of iodine or pure phenol, or a mixture of these ; or, the application of a blister to the skin around or above the area of putrefaction.

Constitutionally, it is questionable if we possess any absolutely reliable agent in assisting us to arrest putrefactive processes. Arsenic, quinine, and salicylic preparations are the most reliable ; salol probably ranks next in importance to arsenic and quinine. The difficulty in choosing remedial agents wherewith to combat the effects of septic processes lies in the fact that we have not only to discover an agent (a microbicide) capable of destroying the micro-organisms themselves, but also an agent (an anti-toxine) capable of neutralising their poisonous products. One agent, termed microcidine, has been prepared and used for the former purpose, and the partial success which has followed the use of tuberculin should stimulate pathologists and chemists to continue more earnestly their investigations in this direction. If we could with greater impunity and greater ease have recourse to intra-venous injection we should possess much more powerful means of dealing with septic processes than we now do. The intra-venous injection of iodine, for example, would be extremely valuable if once the proper dose in which to inject it were determined. Nutrient food, port wine, eggs, milk, spirituous liquors, ammonia, æther, and iron should all be pressed into our service when the condition of our patient indicates the necessity for their use.

As gastro-intestinal disinfectants none are superior to, or even comparable with, iodine, phenol, charcoal, hypo-sulphite of soda, sulpho-carbolate of soda, and camphor.

As an anti-convulsive in septic intoxication, chloral surpasses all other drugs, and in addition to its calmative effects on the nervous system, it exercises a most important influence on the colouring matter of the blood. It may be beneficially combined with potassic bromide.

Prior to 1872 (see *Veterinarian*, September 1872) I was in the habit of administering chloral hydrate chiefly as an anti-convulsive and anti-spasmodic, but owing to the perusal of an article on the effects of chloral hydrate on the blood by Thos. Shearman Ralph, M.R.C.V.S., Eng., (*Veterinarian*, February 1872) I was led to make some experiments in this direction, and as the outcome of these I discovered that by administering such drugs as prussic acid, and along with them, or immediately after, chloral hydrate, I could obtain various forms of crystals in the blood of the living animal; and not only so, but that the chemical effect on the blood was so remarkable that in the skin of white dogs a series of chameleon-like changes in the hue of that structure ensued; and further, I proved that chloral hydrate is the most reliable (chemical) antidote to prussic acid. Consequent on the results of these experiments, I began to administer chloral hydrate in blood diseases, such as septicæmia, and more recently in affections due, in my opinion, to the absorption of ptomaines, *e.g.*, azoturia, the congestive forms of milk-fever, and so-called stomach staggers.

In concluding this paper I desire to impress upon all the imperative necessity which exists for the adoption of strictly antiseptic precautions, and for observing absolute cleanliness in the performance of all operations; and still further, for protecting all bruised skin and exposed surfaces from the atmosphere and from contact with putrefying fluids, or gases of putrefaction; particularly so in cases in which there is reason to fear that the constitutional condition of the patient is unsatisfactory.

EXPERIMENTS WITH MALLEIN.¹

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(Translated by Professor A. E. Mettam, M.R.C.V.S.)

MALLEIN is a glycerine extract of the bacillus of glanders; it was prepared for the first time by the Russian Military Veterinarian Kalning, of Dorpat.

The first experiments made in Russia and in Germany showed at once that mallein could be used for the diagnosis of glanders. Its action upon animals affected with glanders is identical with that of tuberculin on tubercular subjects; it brings about a marked elevation of temperature. In France, the very important experiments of M. Nocard confirmed the specific action of mallein, and at the same time specified the methods to be adopted in its use. Since the publication of these details, all animals glandered or suspicious,

¹ Translated from the *Revue Vétérinaire*, September 1892.