On sour smelling perspiration in acute rheumatism, and its significance as a symptom / by Thomas Inman, M.D.

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ON SOUR SMELLING PERSPIRATION IN ACUTE RHEUMATISM, AND ITS SIGNIFICANCE AS A SYMPTOM.

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There is no symptom in acute rheumatism which strikes the senses of the physician more forcibly, than the copious sour smelling perspiration commonly attending it. Upon this single symptom certain theories respecting the disease have been built, and on these, again, distinct lines of practice have been adopted. One maintains that some poison is present in the blood, and that sweating is an effort of nature to eliminate it. To support this it is averred that the symptoms meliorate in direct proportion to the freedom of perspiration. Another goes still farther, and maintains that as the sour odour is due to lactic acid, so that acid must, in some way, be concerned with the poison to be eliminated; the theory is supposed to be demonstrably proved when, after this acid has been artificially introduced into the blood, cardiac imflammation frequently results.

As a natural consequence of these views, we have some authorities advocating an eliminant plan of treatment. They encourage perspiration, give a daily purge, and, if possible, get the kidneys to act freely. Others while not opposing strongly the first, consider it a better plan to endeavour to destroy or neutralize the acid in the blood. To effect this, alkalies are freely administered from the earliest stage of the disease up to the latest.

These are important theories to be built upon so few facts, and we shall do well if we inquire into the stability of the foundation ere we trust ourselves to the superstructure. We ask—

1. Is it a fact that all cases of acute rheumatism are attended with a sour smelling secretion from the skin?

2. Does the occurrence of profuse sour sweat relieve the other symptoms?

3. Are those cases the mildest in which the perspiration is the freest and sourest?

4. Is the sweat sour when first it is produced, or is its odour the result of decomposition?

5. Does a similar sour odour accompany perspiration in other diseases, and, if so, have they anything in common with acute rheumatism?

6. Why is the perspiration of a rheumatic patient more frequently sour than that of other people?

7. Are the results of the eliminant or alkaline plans of treatment so conspicuously successful, as to warrant us in taking them as corroborative of the theories upon which they are founded?

1. In answering this question we are met *in limine* by the fact, that some practitioners consider perspiration to be a necessary accompaniment of acute rheumatism, and refuse to acknowledge as such, any attack when it is absent. This, however, simply begs the question, and refuses to acknowledge a fact unless it squares with a theory. As Dr. Copland leaves out this symptom from his definition of the complaint, it is clearly non-essential, according to his judgment, and experience teaches us the same lesson. Perspiration is absent in about one-third of the cases which have come under my own notice, and has been excessive only in one-fourth. A sour odour is not perceptible unless the sweating is very considerable.

2 and 3. I have never yet seen the symptoms relieved by the occurrence of perspiration, or by its excess. So far from this being so, I have habitually noticed the reverse, and would say, after an experience extending over twenty years, that the patient improves in proportion as the skin becomes dry; that a return of perspiration is always accompanied by an aggravation of the other symptoms, and that those cases are the worst in which the sweating is most excessive in quantity and most acid in odour. With this experience it is impossible to regard the secretion as a salutary effort of nature to eliminate a poison, unless we are prepared to hold the paradox—"the more a deleterious matter is expelled the worse it is for the body." Of course it may be argued that an excess comes out because an excess is present within, as in confluent small-pox; but this argument fails when it is shown that the symptoms improve when the perspiration is checked, and nothing comes out at all.

4. In answering this question, we must first agree upon the evidence we should deem conclusive. It seems to me that if we can ascertain, 1. That the perspiration when it first appears is free from any unusual odour. 2. That no sour smell is noticed after a complete change of body linen and sheets, and

for a considerable period subsequently, we have *primâ facie* proof that it is not the natural perspiration alone that gives rise to the odour.

To illustrate our meaning, let us suppose that we visit a child who wets its bed every night, we smell a strong smell of hartshorn. If we wanted to know whether the child was in the habit of piddling ammonia, we should try and investigate the state of the urine when passed, or the condition of the linen on the morning after it had been changed. This has repeatedly been done, and every one now knows that the origin of the volatile alkali is from the *de*composition of the urine, and not from its primary state.

To determine whether there was any analogy between the alkaline smell of an urinous bed and the sour couch of a rheumatic patient, I have paid special attention to the smell of the latter's perspiration when first it has been formed, and have been unable to detect any sour odour, though this can readily be noticed in a few hours. I can best point this remark by narrating the case of a medical student recently under my care, who, with his attendant, paid special attendance to this subject at my request. Mr. R., who had twice before had acute rheumatism, asked me to attend him for severe pain in the chest and limbs. The skin was moist and perspiring, but there was no peculiar odour; next day there was excessive perspiration and a sour smell. The case threatened to be a bad one, the treatment consisted of lime juice and opium at night; the sour smell increased in intensity till the third day when it was imperceptible. This was accounted for by the whole of the body linen and the sheets having been changed. In two days it was as strong as ever. In two days more the perspiration had ceased, in ten days from the first attack the patient was convalescent, and has continued well ever since. I often cross-examined him upon the point, and he steadily assured me that the perspiration did not smell sour when first it appeared, nor until it had time to decompose.

5. Being now on the look out for other instances in which there was sour smelling sweat. I was referred to a patient of one of my colleagues, in the Royal Infirmary, suffering from pleurisy, in whom the perspiration was said to be as acid as ever it was in rheumatism. As I had previously noticed a similar phenomenon in phthisis, and other diseases of debility, I went to examine into the statement. There was no unusual smell to be detected, however, *for the patient had just changed all his body linen.* I have at the present time under my care at the Infirmary a young seaman, whose symptoms indicate incipient decline. He has already been two months in the

house, and has never had a symptom of rheumatism, acute or chronic; yet while he was confined to bed he exhaled as sour an odour as ever I smelt in the disease in question. This was accounted for by the fact, that he perspired very profusely, and was not sufficiently cleanly to change his clothes frequently.

Very recently, too, I have had the case of a young surgeon brought to my knowledge who was extremely uncomfortable about himself, for he perspired much, and noticed that the smell of the secretion was precisely similar to that attending rheumatism. With this idea he had been dosing himself with alkalies, and continued to do so until my friend, Dr. Rawdon, our junior house surgeon, told him of my views, and persuaded him that he had nothing to fear.

On the other hand, I have now a severe case of acute rheumatism, in which, though the perspiration has been copious, the smell has never been distinctly acid, and have recently had another in which there was neither perspiration nor odour.

On examining the literature of the subject we find that "lactic acid is more than usually abundant in the sweat of rheumatism and gout, and probably also uric and acetic acid; Dr. Prout detected the last in hectic fever, and both it and lactic acid may be present in the puerperal states of fever, and in erysipelas. Anselmino found free acetic acid in women during their confinement, and Stark an increase of lactic acid in scrofula, rickets, and several cutaneous eruptions."—Copland Art. "Symptomatology," sect. 30. I can remember to have noticed a sour smell like that in question, in the case of an elderly lady whose prominent symptom was excessive debility and profuse sweating.

On comparing together those diseases, then, in which sour smelling sweat is a common sign, we find that they have little in common except great poverty of the blood as regards globules, richness in fibrine, and constitutional debility. We do not commonly find that class of symptoms supposed to be due to the presence of lactic acid in the blood.

6. But the question still arises, how is it that this peculiar odour is more noticed in rheumatic fever than in any other disease. The answer is a very significant one; the assumed fact is untrue; or, if true, it has a ready explication. It is untrue, for we have the same smell in parturient women who have been excessively exhausted by their labour, who perspire profusely after it *and are too poorly to have their linen changed.* The explanation of the frequency of the smell in acute rheumatism is simply this, that when the sweating is abundant, the pain is always so severe that the patients cannot

endure the motion consequent upon an attempt to change their body linen, sheets, &c.

There is every reason to believe that the sour smell is the result of decomposition *after* the fluid has been secreted, and deposited outside the body, consequently no theory can fairly be founded upon the change as regards a poison being eliminated, and that poison being lactic acid. In fact we may compare for purposes of argument, incontinence of urine, and incontinence of perspiration one with the other. The two having much in common.*

7. Respecting the results of the eliminant and alkaline plan of treatment, I would say little, for the only experience I have of them has been gained by watching cases not under my own care, and by perusing records in various books and journals. After close attention to the subject, I have never been able to satisfy myself that the sour smell has diminished under the use of alkalies, so rapidly as it does when lime juice alone is employed.†

In the treatment of acute rheumatism it is necessary to see that the patient has good lime juice, and plenty of it. I had one case under my care for ten days before I found that the patient was taking a factitious liquid—acidified lemonade. She was getting worse during the whole of that time. I then got for her the pure material, and she left the house well in four days afterwards. I have now under care, too, a girl who did not improve in the smallest degree so long as she took only three ounces per day, but as soon as she took the quantity I originally ordered, and which I habitually use, viz., eight ounces per day, she improved rapidly. When first I began to use lime juice I had many such cases, quite sufficient to demonstrate that quantity as well as quality had to be regarded.

I have, in a vast majority of cases, found all the symptoms meliorate within two days after the lime juice has been given. But this conclusion was vitiated as an available stand point for argument, as soon as I ascertained that in some few instances the whole symptoms of acute rheumatism would subside rapidly without any special treatment whatever.

* I have already called the attention of the profession ("Foundation for New Theory, &c.," p. 292,) that debility has a direct tendency to increase secretions, including that of the skin; also that such secretions decompose more rapidly than others, and that they occasionally contain material not found in healthy ones. The facts above alluded to corroborate this view strongly. The white complexion, blanketty tongue, fibrinous blood, and the excessive weakness of acute rheumatic cases, all show how much debility is present; consequently, we can readily understand how it is that the sweating is the worst, and becomes the soonest sour in the most severe forms of the disease.

† I cannot help thinking that the alkaline plan of treatment is untenable until it is demonstrated that the salt supposed to be formed by the commingling of the acid in the blood and the alkali in the medicine is harmless. We know that the salts of arsenic are almost as deadly as the arsenious acid,—the iodides act much the same as iodine,—and cyanide of potassium, muriate of morphia, carbonate of lead, though salts, are all potent and energetic in operation, while all the salts of mercury act much in the same way. In like manner urate of potash may be as bad as uric acid, and lactate of soda as active as lactic acid.

On the other hand it is true, that the majority of cases evince no tendency to spontaneous cure, that lime juice alone does not always cure rapidly, and that patients do get well when taking diaphoretics, purgatives, and alkalies. The question, however, mainly resolves itself into one of time, and average; and if we find, as I have done, that the average duration of acute rheumatism under unlimited administration of lime juice, is about ten days, while the average duration of the cases treated on the eliminant or alkaline plan, exceeds that period considerably: we may conclude, that the success of the alkaline treatment cannot be adduced as a proof of the soundness of the theory on which it is based.

We conclude from the foregoing considerations, that the supposed facts on which the pathology of acute rheumatism has been built, require verification, and this cannot be effected without a careful elimination of all possible sources of error. If, after this examination, the original views are maintained, the next inquiry must be into the interpretation of nature's signs.

Until this has been done, we cannot logically show a warrant for belief in the current theories of the day respecting this disease.

Since writing the preceding, I have met with two other cases which seem strongly to bear out the view of this question which I have suggested. The first was in a gentleman, the subject of a mild attack of rheumatic fever, and in whom there was excessive perspiration only for a day or two, so long as he was unable to change his body clothes, the sour odour was such, that his wife was almost sickened by it, but when he was able to put off his old "Guernsey" and don another, the smell ceased. The lady remarking, that the smell seemed to be in the woollen and not in the skin.

The next case was that of an elderly man, very stout, but very active. I never saw him when he was not perspiring, and while he was sitting in my room he was constantly mopping his face and his bald head. He always had good health, and only came to consult me respecting his son. As he spoke I became conscious of a very peculiar odour about him, which for a long time I could not distinctly classify; on leaving the room, however, and returning suddenly, I recognised at once the sour odour which is so often spoken of as characteristic of rheumatic fever. The explanation of its existence here is easy. The man was always sweating, and rarely changed his body linen, and still more rarely changed his cloth clothes, consequently there was always about him a quantity of decomposing animal moisture.

The Œsophagus of the Ruminantia*.

By William Rutherford, M.D., Resident Surgeon, Royal Infirmary, Edinburgh; formerly Demonstrator of Anatomy at the Royal College of Surgeons, Edinburgh; President of the Royal Medical Society.

[Plate III.]

The muscular structure of the œsophagus of the Ruminantia seems to have been a subject which, nearly two centuries ago, attracted a considerable share of attention; for, in Peyer's work on the Ruminantia, the opinions of no fewer than twelve distinguished anatomists are quoted in reference to it.

In recent times it has not, so far as I have been able to ascertain, been re-examined, except perhaps by Mr. Spencer Cobbold (Todd's Cyclopædia of Anatomy and Physiology).

I have not been able, however, to meet with any description which at all approaches the truth. I will briefly allude to the opinions of previous authors before giving the results of my own investigations.

The first writers on this subject appear to have been Aponensis and Æmylianus, who said that the muscular fibres are arranged in two layers, the outer consisting of longitudinal, the inner of transverse fibres.

Aquapendentius and Guilandinus accepted this description; and Galen expressed the opinion, that while the food was swallowed by all the fibres, it was returned to the mouth through the action of the transverse ones only. On the other hand, Fabricius and Fallopius went so far as to say that the Ruminant's œsophagus contained no muscular fibres at all, but that it was composed of a peculiar tissue met with in no other part of the animal.

Stenson described the muscular fibres as forming double spirals, the bundles running spirally from one end of the œsophagus to the other, forming two layers, which interlace at two raphes; so that the same bundle, while running from one end to the other, lies alternately in the inner and outer layer.

* Being a portion of a Thesis for which a Gold Medal was awarded by the Senators of the University of Edinburgh at the Graduation in 1863. The Thesis was accompanied by numerous dissections and models, which were examined and approved of by Professor Goodsir and the other Members of the Medical Faculty, and are now in the University Museum.

Bartholinus and Grew's accounts somewhat resemble this; but they thought that there was an internal and also an external series of double spirals. Ducernus, Delphinus, and Willis adopted Stenson's description; and Peyer's account, though evidently intended to be original, does not differ from Stenson's, excepting that it is a more lucid and intelligible exposition of a mistaken notion as to the structure.

Monro (Secundus) has, in a thesis on dysphagia, mentioned the fact that the muscular fibres in the Ruminant's gullet cross each other like the lines of the letter X. But this was nothing new; it had been pointed out long before; and it is a fact which any one might perceive almost at a glance.

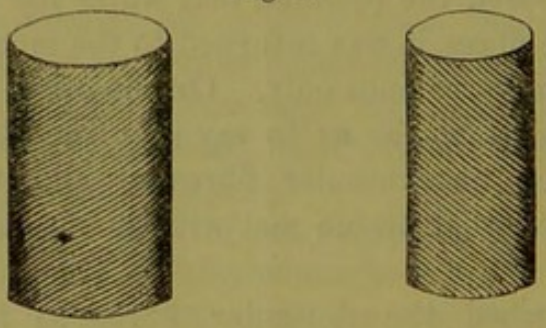
Dr. Spencer Cobbold's description* is the latest. He says that the muscular fibres are arranged in two layers, the *outer* "transversely circular," the *inner* "obliquely longitudinal."

These descriptions are all incorrect. Certainly Stenson's is nearer the truth than any of the others; but it is, nevertheless, wide of the mark.

After prolonged and careful dissection, I feel convinced that the following will be found to be the true description.

The muscular structure of the œsophagus consists of two layers of fibres running in an oblique direction. The fibres of both layers do not, however, run in the same direction, but cross each other like the letter X. The two layers are everywhere

Fig. 1.



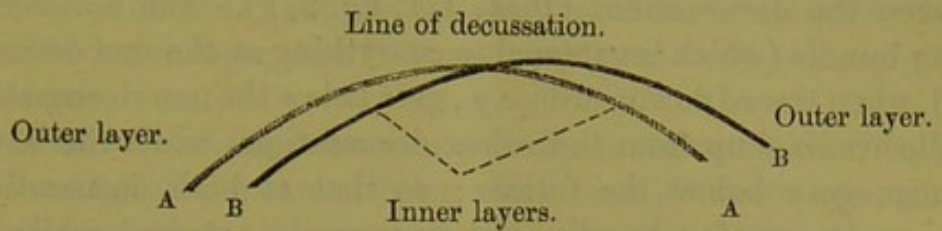
separable, except at two lines, which are exactly opposite each other, and run from one end of the gullet to the other, dividing it longitudinally into two symmetrical halves. (Plate III. fig. 2.)

These lines I shall call the *decussations*; for there the two layers intermingle, the inner layer of either side passing out to become the outer layer of the opposite side; that is to say, the inner layer of one side passes outward, to become the outer layer

* Todd's Cyclopædia of Anatomy and Physiology.

of the opposite side, and, in so doing, crosses and interdigitates with the inner layer of the other side as it runs outwards also.

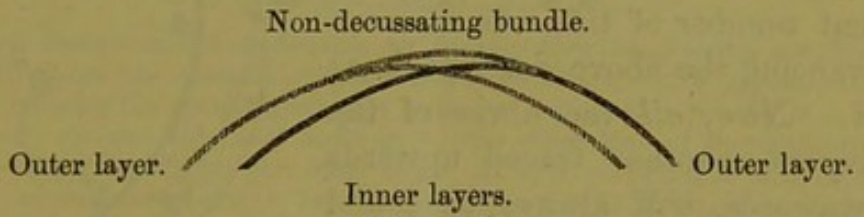
Fig. 2.



Inner layer (A) becomes the outer layer (A).
Inner layer (B) becomes the outer layer (B).

On examining one of the lines of decussation more minutely on both its outer and inner aspects (Plate III. figs. 1 & 3), it will be observed that, whereas internally the crossing is clear and distinct, externally it is obscure. This is caused by bundles of fibres of the outer layer, which, instead of dipping down to become internal, continue over the decussation to the outer layer of the other side (Plate III. fig. 1, A). These fibres, for the sake of convenience,

Fig. 3.



may be termed the non-decussating bundles—an incorrect term, however; for, if these bundles be traced round to the other line of decussation, they will *always* be found to decussate there, and pass into the inner layers. Further, the concavity of the non-decussating bundles (for they all run obliquely) (fig. 7) looks at the one decussation upwards, at the other downwards. At both decussations, *all* the fibres of the inner layers run into the outer layers; but *all* the fibres of the outer layers do not, in like manner, run into the inner layers, but the *half* of the fibres of the outer layer of one side runs *over* the decussation into the *outer* layer of the other side; and whether you trace these non-decussating bundles upwards from the one decussation, or downwards from the other, they will always be found to decussate at the decussation of the opposite side, so that they do not form a layer of *circular* fibres.

Fig. 4.

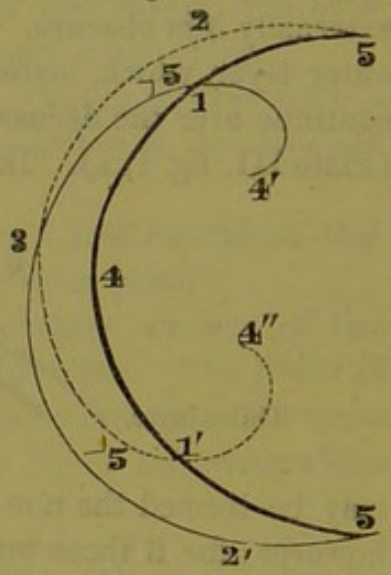
A bundle of fibres not decussating at one decussation, running round to the other (decussation), and there decussating.

The great difficulty in ascertaining the structure lies in tracing these non-decussating bundles from one decussation to the other; for an intermingling of the fibres of the outer layer takes place between the decussations (Plate III. fig. 2, F). The non-decussating bundle (which is external to everything at the one decussation), when traced downwards, *e.g.*, gets below the non-decussating bundle coming up from the other decussation; while the latter, in turn, gets below the former; so that at both decussations the non-decussating bundles are necessarily external, while the decussating bundles are internal (*vide* Fig. 7).

It would be an easy task to trace these bundles in the outer layer, did the fibres of both bundles not intermingle as they cross each other; but the bundles break up into their finest fibres, thereby rendering the interdigitation very minute and difficult to trace (Plate III. fig. 2, F). I have often failed to do so, but I have succeeded a sufficient number of times to warrant my advancing the above description as correct. Now, *all* the fibres of the inner layers, whether traced upwards or downwards, will always be found to decussate (except at the lower end of the œsophagus); and if they be traced onwards in the outer layers, they will be found to form the non-decussating bundles at the *next* decussation (*vide* Fig. 7, A′ & B′). This being true, the structure is simply this:—Each bundle of fibres forms a perfect loop, which crosses the œsophagus obliquely three times (Fig. 6). That is to say, a bundle of fibres (A, Fig. 6) in the outer layer, which runs over a decussation without decussating, when traced round to the other decussation (C) is there found to decussate and pass into the inner layer (F F), in which it crosses the gullet a second time and again decussates (D), passing into the outer layer (E E), in which it crosses the gullet for the third time, and forms at B the non-decussating bundle; so that the one *extremity* (so to speak) of the loop is *above*, on the one side of the œsophagus, while the other extremity is *below* and on the

Fig. 5.

Diagrammatic representation of the course of the fibres in one lateral half of the gullet, including both decussations.



1, 1′. The two lines of decussation.
2, 2′. Non-decussating bundles.
3. Crossing of the fibres forming the outer layer.
4, 4′, 4″. Inner layers.
5. Outer layers.

opposite side. The same bundle is twice in the external, but only once in the internal layer; hence it is that the outer layer contains twice as many fibres as the inner layer; it is not, however, twice as thick, because it contains much less cellular tissue than the inner layer, and is, in consequence, more compact than that layer.

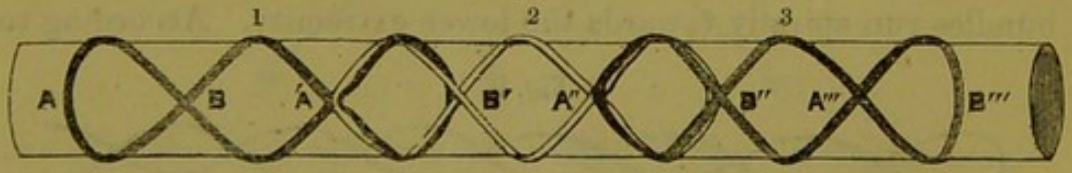
The fact which must more especially be borne in mind is, that the bundles of the inner layer, when traced either upwards or downwards, *always* decussate at the nearest decussation; and when traced onwards in the outer layers they form the non-decussating bundles at the next decussation. If this fact be for a moment thought over, it will be seen how impossible it is to draw from it any but the above conclusion as to structure, viz. that the bundles form perfect loops, crossing the gullet three times (Fig. 6). The manner in which the loops are linked

Fig. 6.

Drawing from a model made with an india-rubber band upon a cylinder of wood. It shows the essential element in the structure, viz. a continuous band of fibres crossing the œsophagus three times; it begins at A (which is a line of decussation) on one side, and ends at B (the other line of decussation) as a *non*-decussating bundle on the other side. At A, B, and E′, E, it is in the outer layer; it decussates at C and D, between which, F, F, it is in the inner layer; so that it is twice in the external, but only once in the internal layer: it decussates at both decussations C and D, and also forms non-decussating bundles at both A and B.

together, so as to form a continuous tube, is extremely ingenious. A diagram will, however, explain the arrangement better than any description (Fig. 7).

Fig. 7.



Drawing from a model made with three india-rubber bands instead of one, to show how the bands are connected one to another.

When the band 1 is decussating for the second time at A′, the band 2 begins as the non-decussating bundle, which, it will be observed, is external to the decussating bundles; as the second band runs round to the opposite decussation, B′, it gets in below the first band, which ends at B′ (external to the second bundle) as the *non*-decussating bundle. In the drawing, the first band has been folded round the second band between A′ and B′. The same arrangement as that described occurs when a third band is added (between A″ and B″), and so on.

The first turn of the first band (A) and the last turn of the third band (B‴) are necessarily incomplete, owing to the want of other bands. Between A and

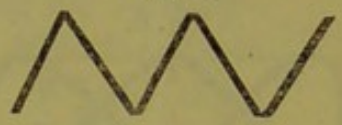
B, A′ and B′, A″ and B″, A‴ and B‴, the bands form the outer layer, which, however, is structurally complete only between A′ and B′, A″ and B″. The bands form the inner layer between B and A′, B′ and A″, B″ and A‴. The inner layers, unlike the outer, are, on the model, all perfect; it will be observed that they contain only single bands, and are, in consequence, only half as thick as the outer layers between A′ and B′, A″ and B″, which have double bands.

The whole could be converted into a solid tube by multiplying the bands, thereby filling up the open spaces.

Just before the gullet joins the stomach, the inner layer becomes very much thicker than the outer layer, owing to the presence of circular fibres, which probably act as a sphincter.

Two other theories suggested themselves to me in the course of my investigations:—First, that although a perfect loop, the bundle might be longer than I now suppose it to be; that is, instead of crossing the œsophagus only thrice, it might do so oftener, say five or seven times (Fig. 8). This I soon abandoned; for I

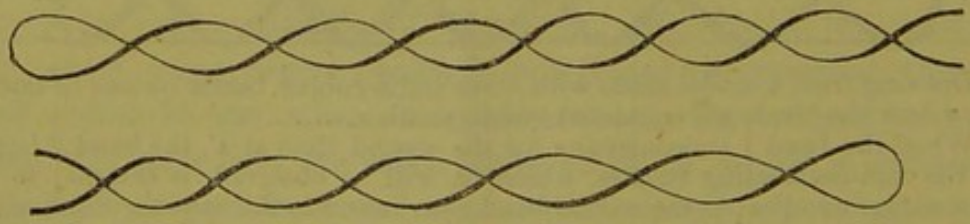
Fig. 8.



found that *all* the fibres of the inner layer, after passing into the outer, become non-decussating at the next decussation, which could not be the case if the loop crossed the gullet more than three times; and, moreover, I found that at a decussation the *non*-decussating bundle is as large as the decussating bundle, which could not be the case were this long-loop view correct.

The same facts overthrew the other theory which suggested itself to me, viz. that the fibres form long bundles, beginning by loops at the decussations—at one decussation the convexities of the loops pointing downwards, and the bundles running spirally to the upper extremity of the œsophagus, while at the other decussation the convexities of the loops point upwards, and the bundles run spirally towards the lower extremity. According to

Fig. 9.



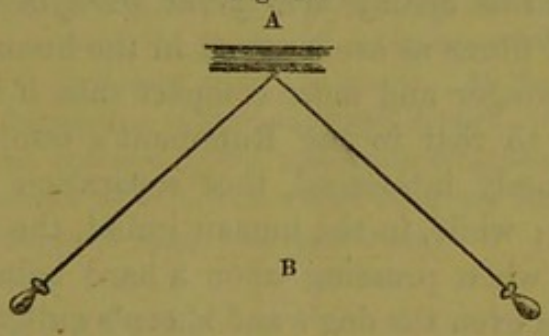
this view, the gullet would certainly have been of equal thickness throughout; but its truth is negatived by the same facts which oppose the long-loop view, viz. that the non-decussating bundles at the decussation are as large as the decussating bundles, and

all the fibres of the inner layer, when traced round in the outer layer, become non-decussating at the first decussation at which they arrive after having entered the outer layer*. I have calculated that, were this last theory true, the decussating bundle, at a decussation, would be ten times as large as the non-decussating; because a spiral running from the one end of the œsophagus to the other would cross it nine or ten times.

Now, what is the use of this remarkable and beautiful arrangement of the fibres? Is it to enable the animal to ruminate? It would be difficult to conceive why so elaborate and ingenious a structure should be required to bring the food *back* from the stomach; for the simpler human œsophagus, with its longitudinal and transverse layers, ought to be just as able to carry the food *up* from the stomach as to take it down; and that it is so is shown by the act of vomiting. And further, the idea that it is a special provision for rumination is proved to be erroneous by the fact (as I have ascertained) that the œsophagus of the dog has the same structure as that of the Ruminant. I have not examined the œsophagus of any of the other Carnivora; but the above is true of the dog's, at any rate.

The advantage gained by a structure such as this, seems to be, 1st, *rapidity of transmission*. A body at A, if pulled by two oblique strings, will be advanced to B more rapidly than if these strings were pulled directly before the body, supposing the power

Fig. 10.



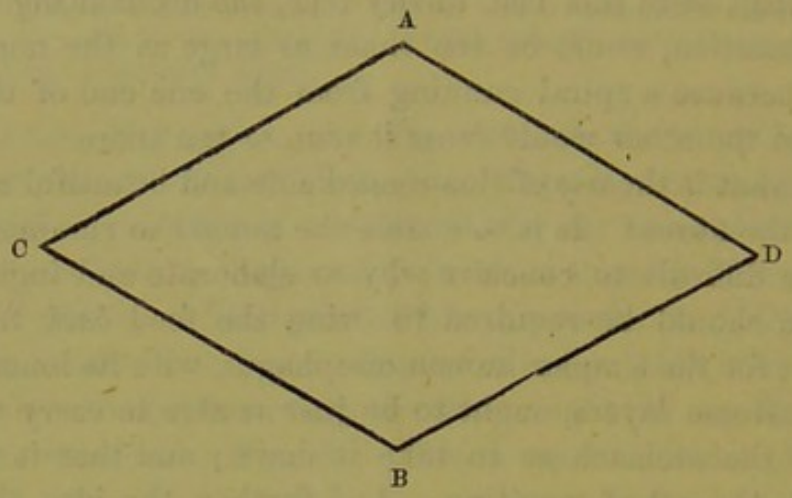
moves at the same rate in both cases. When the strings are oblique, however, although rapidity of transmission is gained, there is a loss of power.

A bundle of fibres of the inner and outer layers, when viewed in relation one to another, form a parallelogram (*vide* Fig. 11).

* I have frequently used the term "layer" both in the singular and plural numbers: *e. g.* the term "inner layers" refers to the internal layers of both of the symmetrical halves into which the œsophagus may be longitudinally divided, while "inner layer" refers to the internal layer of one of these halves.

During contraction, the points A and B, and also C and D, are approximated. By the approximation of A to B the gullet

Fig. 11.



is more rapidly *shortened*, and by the approximation of C to D it is more rapidly *constricted*, than is the human gullet by the action of the longitudinal and circular fibres; and thereby a bolus is carried more rapidly along the Ruminant's gullet than along Man's, or that of a Bird with its circular fibres only. This is a great advantage in the case of the Ruminant; for the slow passage of so much food backwards and forwards would very seriously interfere with respiration by pressing upon the trachea. But, on the other hand, why should the carnivorous animal have such an œsophagus? This fact renders necessary a second explanation, viz. *that by this arrangement great strength is gained.* The same number of fibres as are present in the human gullet would form a much stronger and more compact tube if interwoven in a manner similar to that in the Ruminant's œsophagus; for the fibres are so closely interlaced, that separation of them is extremely difficult; while, in the human gullet, the fibres are more apt to separate when pressing upon a hard bolus. And as the muscular tunic of even the dog's and sheep's gullet is thicker than that in the human subject, *strength*, in addition to rapidity of transmission, is certainly gained. I have often been astonished (before I knew the structure of its gullet) at the large masses of unchewed food which a dog can readily swallow.

I have examined the sheep's œsophagus more especially; for that of the ox, though having the same structure, is much more difficult to dissect on account of the greater brittleness of its fibres when boiled. I have not examined the gullet of any other Ruminant, as I have been unable to procure any other.

Finally, the Ruminant's œsophagus differs widely from the human as regards the microscopic character of its muscular fibre; for while in the human gullet there is a mixture of striped and unstriped fibres, in the Ruminant's they are all of the striped variety.

In conclusion, I have to acknowledge my obligations to my friend Mr. Deas, to whose kindness I owe the beautiful and very accurate drawings which illustrate this paper.

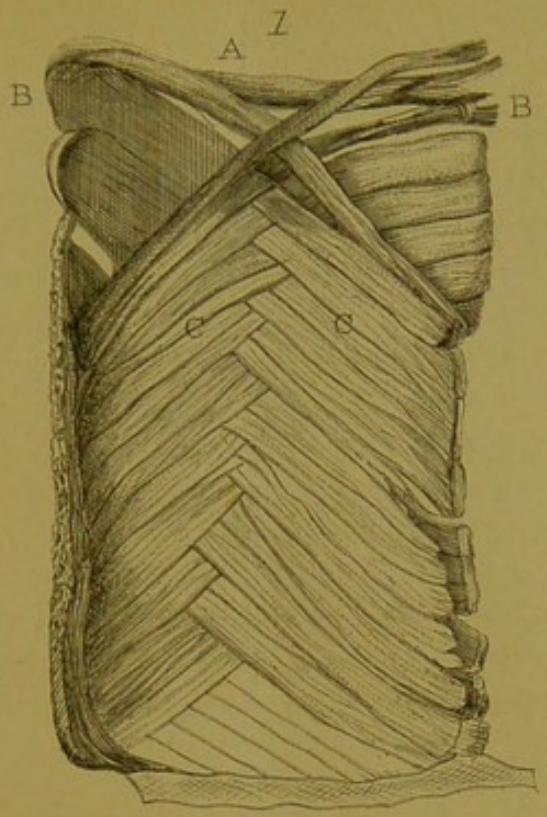
EXPLANATION OF THE PLATE.

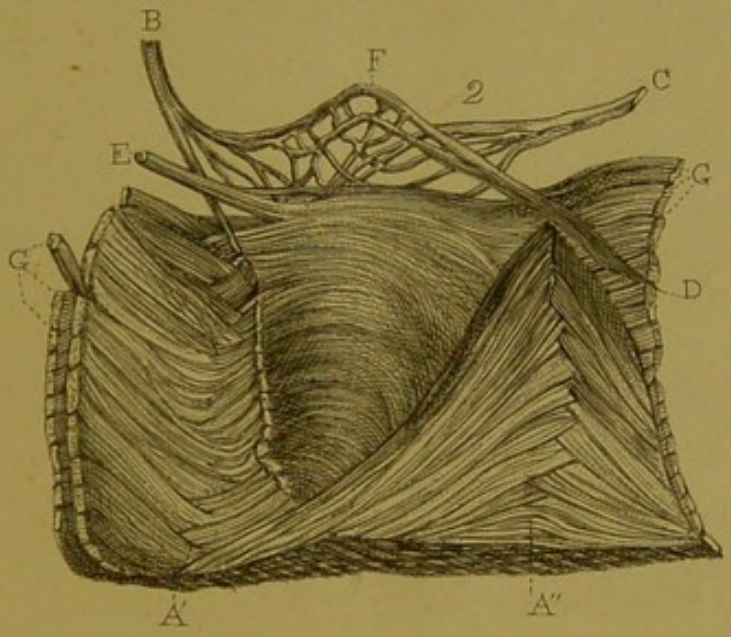
Plate III.

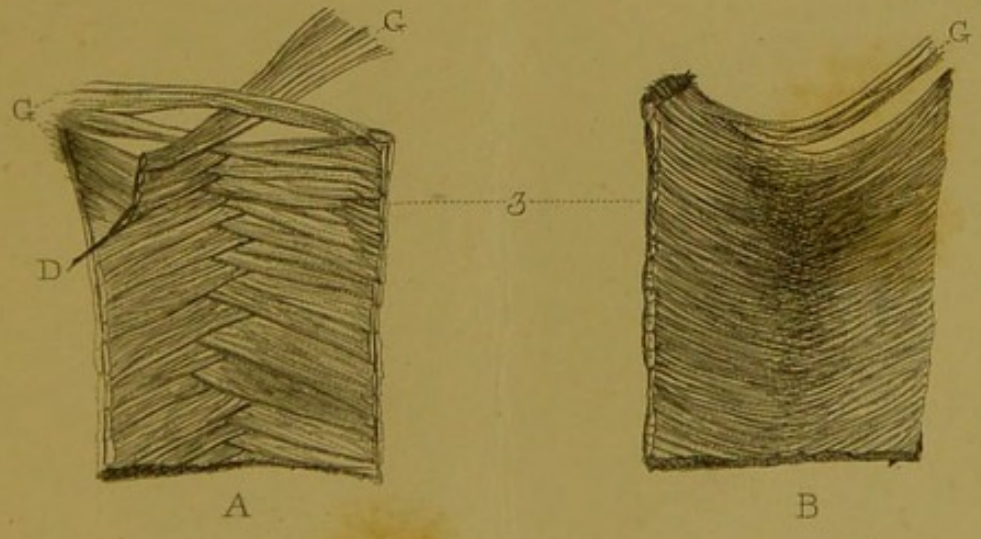
Fig. 1. Portion of sheep's gullet, twice the natural size (seen from within). The fibres of the internal layer, C C, are seen decussating and running into the external layer, B B; while at A is seen a *non*-decussating bundle passing outside the decussation, from the outer layer of the one side of the decussation to that of the other side.

Fig. 2. The entire circumference of a sheep's œsophagus, seen from within (natural size). It has been simply slit open and dissected. Above, A′, A″, are seen the two lines of decussation; B, a bundle of fibres which has not decussated at the decussation A′; and C, a similar bundle which has not decussated at A″. D, a bundle of fibres of the inner layer which decussates at the line of decussation A″; and E, a similar bundle which decussates at the line A′. D runs up to form B, and E goes to form C, both in the outer layer; below F they separate into their finest fibres and interdigitate one with another. G G, outer layer.

Fig. 3. Two portions of the œsophagus of a sheep (natural size), to show the difference between the inner and outer aspects of the line of decussation. In A it is seen from the inside. All the fibres decussate, so that the decussation is sharp and well defined; but on the outside, B, it is much obscured by the non-decussating fibres, which, instead of dipping down as in A, run across from one side of the line to the other. G G, outer layers. D, inner layer.


1
A
B
B
C
C


B
F
2
C
E
G
G
D
A'
A''


G
G
3
G
D
A
B