

that vessel. The laryngeal affection caused the true nature of the disease to be suspected during life, although no corroborative evidence existed.

ON THE CLOSURE OF ARTERIES AT THEIR ORIGIN; AND ON SOME MORBID CHANGES OF THE HEART.

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At the kind invitation of my friend Dr. Ridge, I am again induced to occupy the attention of my fellow members of the Harveian Society; and, on the present occasion, I am indebted to him for the suggestion of the subject on which I am about to speak.

In the Hunterian Oration, which Dr. Ridge lately delivered, he has given a most valuable and elaborate analysis of the record of the symptoms, death, and *post mortem* examination, of the great anatomist in whose honour the oration was pronounced. Angina pectoris and the pathological changes upon which it depends, form, of course, a prominent part of this interesting discourse. Nevertheless, Dr. Ridge felt himself so restricted by the time reasonably to be occupied in the delivery, that he was precluded from stating much which was present in his mind to offer. Amongst the points thus omitted, was the *modus operandi* of the closure of the coronary arteries. Knowing this was a subject to which I some years since paid distinct attention, he proposed it to me as one on which I might write a short article, to be laid before you. I do so with the more pleasure, as it recalls to my recollection two important communications made to this society at a time when I was a more frequent attendant at your meetings than I have lately had the advantage of being. I allude to the cases of obliteration of the coronary arteries, presented by Dr. Ridge himself, and to the experiments of another distinguished member, John Erichsen, in which the coronary arteries were tied at the suggestion of Professor Sharpey, in both of which papers the importance of these arteries to the performance of the heart's functions was clearly shown.

In the cases related by Dr. Ridge in particular, I noticed at the time, as I believe he had also done himself, the operation of a principle which I had taught when I was in the practice of delivering lectures on morbid anatomy at Guy's Hospital. My attention to this point in the pathology of arteries was first excited on the occasion of the late Chas. A. Key operating on a patient affected with aneurism of the arteria innominata.

He applied the ligature to the right carotid, and the patient rather suddenly expired a few hours after. On examination, the left carotid, though apparently healthy, and of normal size, was found to communicate with the arch of the aorta by an aperture scarcely large enough to admit the passage of a small ordinary probe. This conformation of the artery, which very satisfactorily accounted for the patient's death, by the deficient supply of blood which it caused the brain to receive, appeared at the time to be regarded merely as an untoward accidental coincidence.

In reflecting upon it, however, I quickly perceived that it was only the natural consequence of a pathological principle of very general application, and on which I had laid particular stress when treating of the serous membranes, which, on many accounts, afford the best field for the study of morbid processes in their simplest as well as their most evident forms. The principle to which I refer, is the contraction of the more or less plastic deposit resulting from inflammation. The deposit beneath the lining membrane of an artery forms no exception to this rule, but the contraction is probably the more marked in proportion as the deposit possesses the plastic character. It is extremely probable that the aneurismatic dilation takes place most readily at the very time at which the inflammatory

process is producing the deposit, the artery then being in a more yielding state.

The branches given off from the dilated vessel do not necessarily increase with the trunk from which they proceed, although this is sometimes the case, for which exemption an explanation may be given, which I shall presently offer. When the dilation of the trunk has acquired a considerable size, we might expect to find the branch having a funnel-shaped mouth; which, however, I do not remember ever to have seen, unless the branch also has been diseased.

Let us now consider what must be the effect produced upon the mouth of the branch when the adventitious deposit has arrived at that stage at which its contraction takes place.

We know that this contractile force is very great, inasmuch, that muscular effort is incapable of resisting it; as in the case of false membranes on the pleura costalis and diaphragm, and in the contraction of the cicatrices of burns. In the case of a dilated diseased artery the force of the heart in propelling the blood may for a time resist this contraction; but it must ultimately fail, and the contraction take place, more especially if the natural decline of the patient's strength or the course of treatment employed should have the effect of diminishing the force of the heart and the volume of the blood. Now, if by this contraction an area of an inch in diameter around the mouth of an arterial branch be reduced by only one-third of its diameter, it must have the effect of greatly diminishing or closing the mouth of the branch. When this idea had struck me, from seeing the specimen taken from C. Aston Key's patient, I sought its confirmation in other analogous specimens, and found that my views were completely corroborated.

It must not be supposed that the process which I have described invariably takes place. As I have already observed, the branches may partake of the disease of the trunk, and, like it, become dilated and somewhat funnel-shaped, in which case they may participate in the contraction also without being obliterated.

I have said that the integrity of the branches originating from a diseased trunk has its analogy in the phenomena presented by the serous membranes. I allude to the abrupt and defined termination of inflammation in these membranes, which is sometimes seen where a sudden change of direction takes place: as, for example, in the pleura pulmonalis, in which it often ceases where the pleura passes from the external surface into the interlobular fissure, and also in many instances in which the peritoneum changes its direction.

Even where the branch may not participate in the disease of the trunk, its closure or contraction may be prevented by a further stage, into which the adventitious deposit may pass. Neither the atheromatous nor the bony deposit, so commonly met with beneath the lining membrane in diseased arteries, is likely to undergo the process of contraction. It is, however, to this latter change to which I more particularly allude, as the comparatively salutary barrier by which nature obviates the inconveniences which must follow the obliteration of arterial branches at their mouths; and I have in my mind's eye preparations, in which this provision for the preservation of the circulation is strikingly exhibited in the human subject, and also in the horse, in which an aortic aneurism has become completely ossified, forming a permanent cavity of bone, with patent orifices for its branches.

Although by this provision the closure of the branches given off from a diseased trunk may be prevented from taking place through the contraction of the deposit situated beneath the lining membrane, it is quite possible for the obstruction of the blood from entering the branch to be brought about by another morbid process taking its rise at this stage of the derangement. The lining membrane may give way and expose the osseous, or rather earthy, matter to immediate contact with the blood, which then begins to deposit phosphate of lime, or other earthy salts, upon the earthy surface so exposed. Such deposi-

tions assume the form of irregular earthy excrescences, having somewhat of a minute cauliflower appearance. I have no doubt that a comparatively small artery might be so closed, since I have seen the aorta itself all but closed in two places by earthy growths of this character, apparently deposited in the first instance upon the exposed bony surfaces of a deposit formed beneath the lining membrane; and I cannot doubt that a production of precisely the same character within the semilunar valves must have been competent to prevent the blood from entering the arteries, though, I confess, I was not sufficiently alive to the importance of this inquiry when such cases came under my examination.

This production of earthy concretions formed directly from the blood, offers a striking contrast with the collection of matter of similar composition which we find taking place in the interior of veins, and constituting those bodies which we call phlebotomites. In the one case, the earthy salts are separated directly from the blood by a sort of crystallisation, whilst in the other they are instances of the much more ordinary process by which, in accordance with what appears the prevalence of an organic law, the earthy salts are separated beneath an investing membrane, by means of endosmosis. This law seems to be essentially called into operation in the normal production of bone, in the ossification of callus after fracture, in the ossification of the pulps of the teeth, and in other cases, both physiological and pathological.

The atheromatous deposit beneath the lining membrane of arteries, though apparently unsusceptible of contraction, may effect the obstruction or even the obliteration of the branches of arteries simply by its accumulation. This appears to have been the case in the example recorded by Dr. Gull, in the first volume of the third series of *Guy's Hospital Reports*; it is also met with in the arteries of the brain, where it is not an unfrequent cause of cerebral apoplexy.

The causes which I have hitherto mentioned are of a more or less chronic character; and I must not omit to notice one to be considered as acute, and which it would probably have been more in order to have described in the first instance, since it is very likely that it exists as an early stage of the contracted, the atheromatous, and the bony deposits: I mean that form of arteritis in which the lining membrane is raised by a more or less coloured semi-transparent effusion. This condition, which is met with about the valves of the heart in rheumatic and other forms of endocarditis, is not unfrequently associated with the adhesion of small portions of fibrin separated from the blood and adherent to the unattached surface. It is not always easy to account for its existence in the aorta and other arteries; yet it is sometimes evidently produced by local causes: as, for example, where an elongated semilunar valve, or an adventitious structure attached to it, has obviously acted as a mechanical irritant; or where a valve has been ruptured from some extraordinary effort; or when inflammation has occurred as an extension of endocarditis. A good example of this form of arteritis is described and figured in a paper by Dr. Corrigan, in the *Dublin Medical Journal*. It is obvious that this affection, when occurring about the orifice of an arterial branch, must greatly contract or altogether close it.

Although the foregoing observations may possess some interest in themselves, as exhibiting the stages and results of some pathological processes, they would scarcely warrant my occupying your time to listen to them, were they not susceptible of being connected with practical considerations.

When we reflect that endocarditis is by no means a very rare affection, and that either as an extension of endocarditis or as a primary affection, the first part of the aorta is frequently a subject of disease, we may reasonably suppose that the coronary arteries are more exposed to the closure of their orifices than perhaps any other artery in the body, and yet it is extremely probable that this affection has been very generally overlooked. I suspect that I may

have frequently made this omission myself, for I do not remember that I ever even sought for this important organic lesion, before I heard the detail of Dr. Ridge's cases; and since that time, my opportunities have been comparatively rare.

I cannot pretend to offer an opinion whether it may be one office of the foramina Thebesii to provide against these obstructions by supplying the muscular structure of the heart with blood through another channel. I had intended to have made this question a subject of inquiry before meeting you this evening, but I have been prevented from doing so.

The continuance of life is so essentially connected with the incessant performance of the heart's function, that we might naturally be led to suppose that its muscular structure would be very little liable to disease; and such I believe to be the case to a greater degree than any other texture of the body possessed of an equal degree of organisation and activity of function. It is, nevertheless, like them, liable to atrophy and hypertrophy, to various forms of degeneration, and also to be the seat of strumous and cancerous deposits; and it may also be encroached upon by parasitical animals. I have myself seen the acephalocyst hydatid lodged beneath the close pericardium in the human subject, and numerous cysticerci embedded in the muscular substance in the heart of a bear. Of all these derangements, the degenerations of the muscular structure are probably the most frequent and the most important; and yet it may safely be said, that they have been the last to receive careful investigation; and to no one are we more indebted for researches connected with this subject than to our fellow member, Dr. Richard Quain. I think it highly probable that the fatty degeneration of the heart, which Dr. Quain has so successfully studied, is in a large number of cases a consequence of the diminished supply of blood furnished to the heart, occasioned by the contraction or closure of the origins of the coronary arteries, resulting from disease of the aorta, in one or other of the modes which I have been describing this evening.

Amongst the records of some hundreds of inspections, made many years since, and of course before the degenerations in question had been recognised, I have described various appearances in the muscular structure which I now confidently believe to have been produced by different stages of these degenerations. They consisted of different shades of colour, for the most part paler and browner than the healthy heart; differences of texture, as for example, close and cheesy, or waxy, or more or less soft and lacerable; or, on the contrary, more or less firm and resistant. As they were not made the subject of either the microscopical or the chemical examination, which I am now sensible they should have received, they merit no more than a passing notice of the fact; yet, at the same time, I may mention that these appearances repeatedly arrested my attention, in consequence of their having concurred with breast-pang or disturbance of the heart's action, without valvular disease or ossification of the coronary arteries, to account for the symptoms.

These perceptible alterations of structure likewise claimed my attention from their situation. They were, for the most part, neither common to the whole substance of the heart, nor irregularly interspersed through it; but, if my recollection does not mislead me, were chiefly situated in the substance of the ventricles, about equally distant from their internal and external surfaces—a fact which might possibly be explained, if we were better acquainted with the course taken by the muscular fibres of the heart, and with the distinct sets to which they might be reduced.

This subject was long since studied by Lower; and when I was a student in Edinburgh, the late Dr. Duncan, jun., took it up with great assiduity for a time. It may, I believe, be safely said, that this subject generally receives less attention than it merits.

[To be continued.]