Original Articles.

OBSERVATIONS ON HEART DISEASE.¹

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It is the function of the heart to move a certain quantity of blood in the right direction against a certain resistance. The healthy heart does this within wide limits of resistance and of mass, and as happens with many other important organs it is undoubtedly endowed with powers very considerably in excess of the average and usual demand upon it.

The movement depends upon the myocardium, understanding by this word not only the muscular substance, but the complex of nerves and ganglia situated in close connection with the muscular valves, and the resistance upon the arteries, and mass and stimulating and regulating its action.

The direction of the flow depends upon the sometimes in part upon the valves.

In the point of view of diagnosis and pathology it is very obvious why, when methods bidding fair to clear up so much that had been obscure and indefinite were applied to the study of heart diseases, the lesions of the valves, which could be seen after death and heard during life and easily understood, should absorb almost the whole interest of the diagnostician, and that this interest should have only slowly yielded, as it became clearer how much these gross defects could be compensated and made comparatively harmless on the one hand, and counterfeited on the other by those changes in the propelling power which are less accessible to any form of observation.

It is within the easy recollection of many of us (perhaps we need not go beyond our own case books for instances) when prognosis was based upon the observations of cardiac murmurs almost exclusively, as indicating valvular disease, and as a consequence was often unwarrantably serious either from actual mistakes in diagnosis or from a failure to recognize the modifying effects of the other factors. I was, however, surprised to be told after the symposium of Feb. 4 that even now that much depreciated individual the "average practitioner" was supposed to be still in bondage to the old ideas, notwithstanding the large amount of literature in monographs and articles which has now been showered upon him for many years, and the teachings of textbooks as old at least as Flint's "Practice."

The exact diagnosis of the condition of the heart muscle is certainly not so easy, but its importance has not been undervalued of late years. But even if absolute precision is unattainable, an estimate of the relations between the amount of work to be done and the power of the heart to do it may often be made to an extent sufficient for purposes of prognosis and therapeutics.

The variations in the load which the heart has to work under arise to some extent from the mass

of the blood, that is, as to anemia and plethora, but much more — we may say for practical purposes in most cases entirely — upon the demands of the voluntary muscular system for its supply of oxygenated blood, and upon the resistance offered to its distribution by the contractions and dilatation of the vessels under the influence of the vasomotor system.

The constant resistance — that is, the resistance above the normal — may come from a chronic contraction of the arterioles, the theoretical precursor of organic disease, or from the developed arteriosclerosis, and from imperfection of the valves.

When the heart is able readily to adjust itself to widely varying demands without distress, it is functionally healthy. When it cannot furnish all the blood that is called for without suffering, or at any rate without continuing to suffer after the strain is over, or when, after the strain has ceased to exist, it fails to return to its former condition, it is not healthy, whether we suppose that any structural change would be discoverable or not.

It seems at once that the line between the healthily acting and the inefficiently acting heart cannot be a sharp one but is represented by a margin of varying width, which may be called the margin of adjustability or safety, having upon the one side the maximum of endurance called for by the extremes of intense and prolonged muscular exertion and on the other a minimum just sufficient to cover the demands of a carefully regulated life with as slight as possible variation from uniformity of muscular or emotional exertion.

The width of this margin, or perhaps more correctly the position of its lower limit, it is of the utmost importance to estimate correctly in diagnosis, to be able to tell a patient what he may or may not do.

There is one way to tell with a good deal of precision, and that is by experiment; but it is one to be tried with caution, and the patient should not be placed in a position where he cannot retreat before carrying it too far. It is in a methodical and careful manner of trying it, so that it may be stopped before it becomes dangerous, it seems to me, that the chief advantage of the method of Oertel consists. It is the object of therapeutics to place, so far as possible, the working capacity of the heart well above all probable demands upon it, and this may be accomplished to some extent upon either side. Diminution of resistance, when it depends upon organic narrowing of an orifice or rigidity of the great arteries, is of course out of the question, but when it is the result of too great tonicities of the arterioles it is not altogether out of reach of eliminatives like cathartics and alkalies, of diet, or of vasomotor paralysants, as nitroglycerin and other nitrites.

Even the upper limit of this margin may be transgressed in the way of violent long-continued exertion, especially when combined with under-feeding, or when of such a character or exerted under such circumstances as to require the fixation of the thoracic muscles and to impede respiration. This has been pointed out by Allbutt, DaCosta and

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others in soldiers and heavy laborers. The results of this transgression may be permanent or at the best recovered from slowly and imperfectly. There is evidently here an extreme exhaustion, which is in rare cases a paralysis but more frequently a paresis with loss of muscular tone although not completely of muscular contractility; but there is no reason to suppose that it is connected until after a considerable time with any lesion detectable by the usual means of observation.

It is, however, chiefly in the increase of the power of the muscle, and in so regulating it that it may be expended with the greatest economy, that the most important results are to be obtained. This means on the one hand proper nutrition and a combination of rest and exercise suited to the peculiarities of each case, and on the other the use of cardiac regulators and stimulants, as digitalis and its congeners, and of strychnine. The myocardium, if of healthy structure, can, like any other muscle, gain somewhat in energy by judicious exercise, and also, like other muscles, can be so thoroughly exhausted by overstrain as to be practically paralyzed. The difference between them is that while the voluntary muscle can be usefully exercised at long intervals of hours or days, with intervals during which it is called upon to expend little or no energy, the heart must get its training while it is constantly at work, although with a minimum or average load, yet never without any load at all. The heart must receive the benefit of exercise not at intervals of hours or even minutes but at intervals of a second or usually less, and then as an addition to an average which may be near the maximum capacity,—in many cases we cannot tell just how dangerously near.

The observation of the pulse, of the venous circulation, of localized congestion and edema, is the real guide as to how efficiently the heart is acting. If the work is not done thoroughly, easily, and without disturbance, or if a little extra call for exertion shows that the margin of adjustability is a very narrow one, it is then the part of the physical examination to distribute the difficulty properly between the two factors—propelling force and resistance.

The mistake may be, and undoubtedly has been, frequently made—and it is really a very natural one—of attaching the greatest importance to that which furnishes the most definite and clearly marked signs; that is, valvular disease; and when these signs or their counterfeits are almost accidentally discovered, of drawing from them alone the meaning they can justly carry only when in combination with others less prominent.

The pulse was more carefully studied by our grandfathers than by ourselves, but its characters, even when distinct, are not always easy to describe. Perhaps it was for this reason its study has been allowed to fall into the background.

The sphygmograph, although it is true that it requires care in its application, that the greater or less thickness and elasticity of the superjacent tissues obscure its indications of arterial pressure,

and that tracings from the same pulse may be made to vary very widely from each other by changes in pressure, in position, or in the weight of the levers, and has, consequently, not unnaturally been depreciated by the physiologists, to whom of course it cannot yield results comparing in numerical precision with those of the manometer, is not unworthy the attention of the clinician. It is of much more value in determining the flow and resistance in the arteries, than it is in interpreting the peculiarities of the heart's impulse, with the valvular lesions; but that it does demonstrate with considerable accuracy actual facts of varying pressure may be shown by comparing an average tracing with that simplest of all sphygmograms—the marks made on a strip of moving paper by a jet of blood from an artery or even a trocar thrust into the ventricle itself.

But it is probably inferior in some respects, beside that of convenience, to the trained finger, which can better take in at one time all the components that go to make up the character of the pulse. But I say advisedly *trained* finger, and for the training of the fingers there is nothing better than a constant use of them side by side with the sphygmograph, for I suppose sight will always be a more definite and accurate sense than feeling. I think it is Vierordt who winds up his remarks upon the sphygmograph, of similar tenor to my own, by saying that it “teaches the student what he ought to feel.” The fingers should determine how large and how elastic is the artery, how full it is filled by each beat of the heart, how long the blood is retained there without running back through the aortic valves, how rapidly does it escape from the arterioles, how much pressure is required to extinguish the pulse. The sphygmograph cannot do all these things at once, but it can do some things that the finger does not (see Hayden). It shows a diastolic not perceptible at least to the average finger, it deciphers some forms of irregularity better than the finger does, and besides these it records what it does find for exhibition, which is clearer than any description, and for future reference. The relation between a sphygmogram and the palpation of the pulse is somewhat like that between an instantaneous photograph and the eye. Nobody ever knew before Muybridge what shapes a galloping horse got himself into, but the artist and the prospective buyer still prefer for their purposes their own eyes and hands to the camera. There is no form of pulse evident either to the finger or to the sphygmograph which is pathognomonic of any form of cardiac degeneration. It may indicate want of tension or extreme feebleness, but a tracing is of value only in connection with other symptoms. Feebleness of the pulse, in the absence of aortic stenosis or other obstruction, or of mitral regurgitation which allows the force of the ventricle to be expended in the wrong direction, means of course feebleness of the contraction of the heart, but no tracing is evidence of this unless we know what pressure was used in taking it, and whether it is the best possible tracing with the most ad-

vantageous pressure. Of this condition of the pulse and heart, especially if extreme, the educated finger is a better standard than the sphygmograph.

Is it possible by any combination of symptoms to diagnosticate actual degeneration of the cardiac muscle? It seems to me not with absolute certainty, but under many circumstances with a degree of probability entirely sufficient for practical purposes. It is one of the common remarks that fatty degeneration is sometimes suspected for the first time upon the autopsy table, but the question at once arises, what were the opportunities for suspecting? I must doubt if a muscle which has begun, even to a very small extent or degree, to show a degeneration visible to the microscope could have retained its full efficiency or have had more than a very narrow margin of adjustability or safety.

Such a muscle might of course have been perfectly able to respond without trouble to the average demand upon it, and this average may have been at no time sufficiently exceeded to show how near the danger line was until it was fatally passed, but such an even tenor of life is not likely to be more than a rare exception.

Quain, whose paper,² now 50 years old, has been but little added to or improved, says:

"It should be stated that many cases are recorded in which patients have died, and in whose hearts this condition has been unexpectedly found. Some of these cases are said to have been in the enjoyment of perfect health, others have been ailing, but not in connection with the heart; others ailing likewise, and the heart being suspected has been examined, and failed to give evidence of the existence of the disease, which post-mortem examination has subsequently shown to be present. I have not met with such cases. On the contrary, even in some instances in which the individual dying was said to be in good health, I have found on inquiry that symptoms, more or less marked, had existed, and might have been previously recognized if attention had been called to them."

If the heart is weak to a degree not accounted for by its dilatation, if it fails to improve with rest and treatment, if the sounds are sharp and snappy as if destitute of the muscular element and dependent solely upon the closure of the valves, if the impulse, instead of being either limited to a small spot or strong and heaving, be a sort of wave passing over a considerable area or, as Leyden says, a "tremor," the grounds for suspecting actual degeneration are very strong. The presence or absence of a systolic murmur at the time of examination is not conclusive either way, but the history of a murmur may be very significant. The slowed pulse of obesity (Kisch), between 50 and 60, is not dangerous. The somewhat slowed pulse is not of great diagnostic significance. It is said to be quite frequently met with in the fattily overloaded heart in corpulent people.

The extremely slow pulse, the "permanent slow pulse" of some authors; that is, below 40, has been

by some considered as closely connected with fatty degeneration; but although the coincidence is too common to be altogether accidental, it is by no means invariable, and cannot be conclusive for diagnosis. There have been many cases where careful examination has failed to find any fatty degeneration. Is it of great importance to distinguish between a simple dilated heart and a fatty dilated heart?

Acute dilatation is, as Dr. Prince has shown, a physiological adjustment. When too great or too long continued it becomes pathological, and fatty degeneration marks further progress in the same direction; but there is no proof that dilatation is always capable of recovery, any more than that fatty degeneration never is.

Murmurs disappear, the area of the heart's dullness may diminish, and patients recover from conditions which, when they go a little further, are well known to be accompanied by extreme fatty degeneration.

A heart may be slowly losing reserve force; its store of energy, or, more correctly, its ability to restore the amount of energy as fast as it is expended, may be gradually diminishing; that is, the margin of safety may be growing slowly narrower, and yet, if never called upon for more than the expenditure of the usual or average amount, will give no very obvious or urgent sign, perhaps under specially favorable circumstances even none at all.

If, however, the call comes for an increase of exertion—perhaps extreme, perhaps only slight—demanding an expenditure greater than the margin, whatever it may be, the organ can no longer respond, or does so with difficulty and distress, and we have an overstrain resulting in an attack of cardiac dyspnea or a sudden death. If not fatal it may result in a partial recovery or a permanent increase of the disability. The prognosis may depend very largely upon causes within the heart itself, like a valvular lesion which has been comparatively unobtrusive, or upon general conditions, like an attack of acute disease or the slow impairment of nutrition by alcohol. The pathologists can certainly tell us of many cases where death has been rapid and, so far as their data can decide, unexplained, as well as of others where signs have existed so slight that they have been neglected or misinterpreted until death has come too late to tell us what they mean, and also of others where the symptoms have been extreme and pointing with the utmost definiteness to the heart, in all of which no lesion can be detected in the course of an ordinary post-mortem examination, no matter how skilfully and carefully conducted. They find, if not absolutely nothing, no more than a little arteriosclerosis or a trace of fatty degeneration, such as may often be seen in cases dying of entirely different diseases.

As pathological anatomy sheds little light, we have certainly the right to compare such cases as to symptoms and manner of death to others where we can find a lesion. In aortic stenosis and regurgitation, for instance, we may have a train of events like what has just been described. Here

² *Medico-chirurgical Transactions*, xxxii.

also the margin of safety has been narrowed. The heart cannot do an increased amount of work, not because the muscular force is diminished,—on the contrary it may have been, under exercise, considerably increased,—but because the obstacles have been becoming disproportionately greater. The margin has been narrowed from the other side.

Now, with all due respect to the board of health, it appears to me that no more properly descriptive phrase can be found for this state of things than the objectionable “heart failure,”—objectionable only because it is so easy to misapply.

In the latter case—that of valvular disease—we describe the causative lesion because it can be seen and felt, and it is of course understood that the myocardium was not strong enough to carry so heavy a load. In the former the patient dies in the same way, and the last link in the chain of causation is the same; but as we cannot yet put our finger on the lesion, we are obliged to give a name to the functional result. What we want to know in such cases is, why has the heart failed to do what it should do?

Shall we always be obliged to rely entirely upon symptoms as we do now in describing and classifying what we call the functional diseases of the heart? It would certainly be strange in these times, when new and delicate laboratory methods are throwing the light of pathological anatomy upon so many conditions of the nervous system hitherto supposed to be beyond its province, if some of the clear and definite groups of cardiac symptoms did not yield their secrets to the microscope.

Hardly any department of physiology has been more thoroughly and fruitfully studied than the innervation of the heart, and yet one of the most recent, most learned and most concise statements, by one who has himself contributed largely to its original investigation, acknowledges that many points are yet far from decided. And pathology is far behind this point.

The relations of the vagus and accelerator nerves, presenting the most complete and typical example of stimulus and inhibition, have often been called upon to explain irregularities in the cardiac rhythm. The vagus has also been called the trophic nerve of the heart, but I am not aware that any distinct connection has been traced between its affections and the demonstrable degenerations of the heart muscle.

There are two very distinct groups of cases in which the rhythm of the heart's beats appears very closely connected with nervous influence, but in which anatomy has given no demonstration.

In one of these, Graves' or Basedow's disease, the connection is so intimately involved with the action of some peculiar autogenetic substance that it is perhaps hardly reasonable to expect anatomical changes. At any rate the chemical side of the question appears likely to be cleared up first, and will then throw light upon the structural.

In the other an extremely slow pulse associated with epileptiform or syncopic paroxysms has not as yet been placed on a thoroughly satisfactory

basis of pathological anatomy, nor any constant relationship established with changes in the nutrition of the muscle.

A slow pulse is not one of the usual accompaniments of any recognized form of cardiac degeneration and, although it is true that in a large number of cases, where the slow pulse has been observed, there have been found arterial and muscular degenerations to a greater or less extent, yet these conditions are altogether too common and the symptom too rare not to make it evident that another cause, either in conjunction or separately, must be at work. There are also too many well-marked and most typical cases in which practically nothing has been found to be neglected. The supposition is a very obvious and reasonable one, that we are dealing with some kind of nervous phenomenon. A stimulation or irritation of the vagus is naturally the first thought and is easily applied clinically, having in its support a considerable number of cases of slow pulse where anatomical conditions, producing irritation of the vagus nucleus in the medulla, were found. One of the most decisive of these is the celebrated and oft-quoted one of Holberton,³ where a man whose pulse had previously been 60 had a fall which produced some injury to his neck. He had fainting fits, his pulse became habitually slower than before, and at times went down even as low as 7. Five years after the accident he died. The heart was large and weak. The medulla oblongata was small and extremely firm. The occipital foramen scarcely admitted the little finger, from enlargement of the odontoid process and the thickened ligaments anteriorly. This was before the days of section cutting and of staining, and the condition of the vagus nucleus is only to be surmised. There have been cases similar to this but none quite so clear and definite.

There are, however, left many and in fact the majority of typical cases, where such an explanation, if urged at all, must rest entirely upon conjecture.

In a case reported by Prentiss, which I had the opportunity of examining both before and after death, sections made at the level of the vagus nucleus failed to show any lesion. The slight enlargements upon the course of the cardiac branches of the vagus were probably not of importance although not examined microscopically.

It would of course be perfectly possible that the irritation might take place at the other end of the cardiac nerves; that is, the inhibitory ganglia of the heart itself; but observation on the effects of atropine seem to put aside this hypothesis very distinctly. It is well known that this alkaloid paralyzes the vagus completely, including its cardiac end-organs, thus completely destroying its influence. Now if atropine be given in cases where the pulse is known to be slowed by central irritation, as for instance in some cerebral diseases, it quickens it materially; but in cases of the kind

³ Medico-chirurgical Transactions, xxiv.

we are considering it produces no effect at all or only an insignificant one.

The nervous centres of the acceleratory system are those to which, finding nothing satisfactory elsewhere, we should next turn for an explanation. Theoretically a degeneration in them would produce the symptoms we are seeking to account for, but for very obvious reasons actual observation has furnished little basis for the theory.

The cardiac ganglia of the sympathetic, in particular the united inferior cervical and first thoracic, might well have been the seat of changes; but, so far as I am aware, none have been found. Certainly few, if any, observations have been published. I was unable in Prentiss' case to find in them any increase of connective tissue or atrophy of the cells.

As to the intrinsic ganglia of the heart situated chiefly in the auricular septum, they have been extensively and carefully examined for pathological purposes but, with very few exceptions, without reference to cases of this kind. In a great many cases where degeneration of these ganglia have been found, the pulse has been rapid. These ganglia are by no means easy to find and are surely not likely to be the objects of examination in the gross at any ordinary post-mortem. For this reason I am inclined to be extremely skeptical as to a single case of permanent slow pulse, in which it is stated, in an incidental sort of way, that there was found "atheromatous degeneration of the aorta and of all the large arteries, fatty degeneration of the heart and a change of the heart ganglia, which were enlarged, hard, and a part of them the seat of calcareous degeneration."

Several series of observations of these ganglia in fever and in organic disease of the heart have been published, but in none, with one exception, having reference to permanent slow pulse.

Even this case⁴ was not exactly of the class I am speaking of, but of mitral stenosis with pulse beating half as fast as the heart. Even with this allowance, however, the heart was slow, and the examination, which was made by cutting serial sections through the part of the auricular septum which contains a great many small ganglia and staining them by modern methods, was an interesting one. The upshot of it was, however, that the writer was not sure that the Nissl sections represented anything more than post-mortem changes, or at the very utmost such as may be found in temporary changes of activity in other nerve cells as in the well-known experiments of Hodge and others. An apparent shrinkage of the cells he also supposed to be post-mortem, since there were no traces of edema or thickening of the capsules to account for it.

The suggestion made by Jacobi, that the difference between the cases of fatty degeneration with a very slow pulse, of which there are some, and those with quick and weak ones, of which there are more, consists in the involvement of the cardiac ganglia in the same process which affects the muscle and very frequently the coronaries in both,

is a very plausible one and has some clinical evidence in its favor, but does not as yet rest on an assured anatomical basis. Although recent embryological observations show that the intrinsic cardiac ganglia take their origin from the sensitive ganglia of the cord, and are hence themselves to be looked upon as sensitive and having nothing to do with the muscular action of the heart, yet there is too much physiological evidence of their connection to allow them so easily to be laid on one side in discussing this subject. The accelerator or augmentor nerves of the heart join the cardiac plexuses through the ganglia of the sympathetic, even if not connected with the cells in passing among them. It seems to me that the ganglia, if not strictly motor, stand in somewhat the same relation to the heart that the ophthalmic ganglion, for instance, does to the iris, a sort of local reflex centre; or as the plexuses in the walls of the intestines do to the muscular walls, carrying on the regular succession of contractions producing the peristaltic movements. They receive the stimulus of distension by the blood and convert it into the motor impulse which produces the successive contractions of the auricles and ventricles, co-ordinating them so as to carry on the progressive forward movement of the blood.

THE PUPIL IN GENERAL DISEASE.

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For the recognition and correct interpretation of pupillary symptoms it is necessary that there be a clear understanding of the normal actions of the pupil, the mechanism of those actions, the proper method of testing them, and the variations within the normal. It must be borne in mind, too, that abnormal pupils are often caused by pathological lesions local to the eye.

The normal movements of the pupil are reflex and associated. The reflex movement is contraction under the influence of light falling on the retina and dilatation on the removal of the light. The associated action is the contraction in the accommodative effort, the focusing from a distant to a near object, and in the converging movement of the eyes in fixing that object.

The nerves governing the iris are the third and the sympathetic. The stimulus of light falling upon the retina is transmitted through the optic nerves and tracts branching by some path to the pupil-contracting nucleus of the third nerve in the floor of the third ventricle and from here out to the iris. The associated contraction is brought about by the coincident action of this centre and those for convergence and accommodation which are near it. The active pupil-dilating centre is the so-called ciliospinal centre in the sympathetic system in the lower cervical cord. It is considered that the normal moderate dilation of the pupils is dependent on the constant psychical and sensitive stimuli transmitted through this nerve to the iris,—the sympathetic reflex.

⁴ Jona. Riv. Ven. d. Sc. Med., 1898.

¹ Read before the Massachusetts Medical Society, June 11, 1901.