**On insufficiency of the aortic valves in connection with sudden death, with notes historical and critical.**

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ON

# INSUFFICIENCY

OF THE

# AORTIC VALVES

IN

# CONNEXION WITH SUDDEN DEATH;

WITH

## NOTES HISTORICAL AND CRITICAL.

BY

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# INSUFFICIENCY OF THE AORTIC VALVES,

## &c. &c.

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### PART I.

#### HISTORIC LITERATURE.

"Nescire quid antea quam natus sis, acciderit, id est semper esse puerum."—CICERO.

IT is proposed, in the present sketch, to trace, from the time of Harvey, the successive steps by which our own refined knowledge of the disease in question has been attained. Such an outline ought not to be without interest, at least, for those more immediately concerned in this branch of Pathology. It does not, however, appear that much inquiry has been made, of late, in this direction.

It is clear that Insufficiency of the Aortic Valves could find no place in Pathology, prior to the discovery of the circulation of the blood. A correct appreciation of the mechanism of this process, and of the methods of Physical Diagnosis, are the indispensable pre-requisites for a scientific knowledge of the diseases of the heart. Without adequate ideas of the former, not even a centralization of the signs of such diseases was possible; and, in default of the latter, no differential diagnosis was attainable. But no sooner was the circulation of the blood a truth of demonstration, than Physiological Pathology soon achieved the richest conquests in cardiac diseases.

We owe to our distinguished countryman, Mayow, the philosophic exposition of the common law which governs the changes sustained by the heart, consecutive to structural derangements of its valvular apparatus*—the law, as it is now termed, of Retro-dilatation and Hypertrophy.

It is to be regretted that later authorities should have been so unmindful of the observations of this most original physiologist. He thus describes the pathologic sequence in

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* Mayow's work bears date, Oxon. 1669. Some have attributed the views taken by Mayow to Blancard; but, according to both Morgagni and Haller, Blancard was frequently guilty of the grossest plagiarism.

a case of obstruction of the left auriculo-ventricular orifice* :—

"A young man, æt. twenty, of delicate constitution, had suffered during several years from a palpitation of the heart, so violent, that the ribs were forced outwards, and decided bulging was visible in the left præcordial region. On placing the hand over this situation, the heart was felt to elevate, forcibly, the chest wall. Notwithstanding this, the beat of the arteries of the wrist was unusually weak and small; and to this fact I can bear personal testimony. In course of time the patient became breathless on exertion, and fainted from the extreme violence of the palpitation. At last, after a long journey in a carriage, he was seized with a paroxysm of unusual violence, accompanied by frequent syncope and coldness of the extremities. And thus he died. Upon examining the body, the heart was enlarged, particularly the right ventricle, which was much larger than usual, and full of coagulated blood. The muscular walls, moreover, were unusually thick and strong. Besides this, the pulmonary artery and veins were distended by grumous blood; but the vein † which empties itself into the left ventricle was so nearly closed by a cartilaginous growth, that scarcely any blood could pass into the ventricle. Nor, can it be doubted that the obstruction of the pulmonary vein was the cause not only of the palpitation but, also, of all the other phenomena described. For, inasmuch as the blood, on account of the obstruction, could not pass freely into the left ventricle, it necessarily happened that the vessels of the lungs, and, also, the right ventricle, were distended with blood; as a consequence the heart, particularly the right ventricle, would have to contract more violently, in order that it might, as far as possible, propel the blood through the lungs, on to the left ventricle. This, again, explains why the walls of the right ventricle were so strong and dense, since this chamber, being submitted to more violent action, would be enlarged beyond the rest." ‡

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* Mayow acknowledges himself indebted to his friend, T. Millington, for the particulars of the case.

† Left auriculo-ventricular orifice.

‡ "Portal, Anatomie Medicale," 1803, Tome iii., p. 90, has also well explained the law of retro-dilatation. He states that the cause of dilatation of the heart frequently resides in the lungs. In such a condition, the pulmonary artery not being able freely to deliver its blood, this fluid is retained in the right ventricle, and dilates it the more readily, as its walls are naturally thinner than those of the left ventricle. Again, the blood not being able to escape from the right ventricle, the right auricle cannot empty into it, being

With respect to Insufficiency of the Aortic Valves, the first clearly described case that I have met with is by one of the greatest masters of Cardiac Pathology of the seventeenth century—Raimond Vieussens.

In his introductory remarks to this case, Vieussens observes: "I am aware that some anatomists have found the trunk of the aorta ossified, both in men and animals; but I have never either read or heard of changes of structure of the sigmoid valves of this vessel.* I have, nevertheless, once found these valves ossified, as is shown from the following observations:—A man, æt. thirty, of melancholic temperament, subject for a long period to attacks of epilepsy, was seized, in the course of the year 1695, with so violent a paroxysm, as to cause alarm lest some vessel should rupture in the brain. However, on being brought to the hospital the attack so soon abated, that, if not quite convalescent, he was, at all events, out of immediate danger. Nevertheless, on my visit to the hospital, I examined the case. His expression was dull; face, pale and bloated;

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already full of the blood it receives from the cavæ, the auricle, consequently, dilates, and occasionally equals in size an infant's head.

In many autopsies, the right cavities of the heart have been found greatly dilated, without those of the left side in any way participating. On the other hand, there are very few recorded examples in which these last-named cavities were enlarged, without the right side being more or less implicated. This difference generally depends upon the situation of the obstacle to the circulation. Should it be situate in the pulmonary semilunar valves, for instance, or in the trunk itself of the pulmonary artery, dilatation of the right ventricle ensues, in consequence of the impediment offered to the onward passage of the blood. But the left auricle and ventricle, contract, rather than dilate. But, if the impediment to the circulation is situate at either the mouth or other portion of the aorta, or branches of this vessel, more or less distant, then the left ventricle becomes dilated by the blood thus impeded in its exit; the left auricle becomes full and distended; the pulmonary veins cannot empty themselves of the blood sent by the pulmonary artery; they, consequently, the right ventricle as well as the right auricle, and perhaps, also, the caval veins, undergo successive dilatation. Generally the walls of dilated hearts are softened, although at the same time often thickened, not merely with respect to their cellular tissue, but as to their actual sarcous element.

See Vieussens, Traité du Cœur, ch. 16, 1715, for a similar chain of reasoning. Also Treviranus, Biologie. Band iv, p. 231.

* It is singular that the same remark should have been made by Lancisi, the great cotemporary of Vieussens. He does not appear, however, to have had any insight into the pathology of aortic insufficiency. The following passage is from his work, "De Subitaniis Mortibus," Ed. 1706, p. 100, s. vii.:—"Igitur, ad posterioris animadversionem transeuntes, ingenue fatemur, hoc sarcomatum vitium in tot observatis cadaveribus nunquàm alias nobis fuisse compertum, hoc adhuc ex aliorum lectione cognitum; utcumque viderimus hujusmodi valvulas cartilagineas, osseas, exesas, phlogosi affectas, aut cum sanguiferis varicosis." It is, however, quite certain that diseases of the valves had been described by several authors before either Vieussens or Lancisi. Vide Morgagni, Letter 23, a. 10.

pulse, very full, very frequent, very hard, somewhat unequal, and so strong that the arteries in both arms conveyed to my finger the sensation of a tense and strongly vibrating cord. A pulse of this kind, such as I have never before observed, and trust never again to encounter, impressed me with the conviction that the patient must suffer from violent palpitation of the heart. This opinion proved correct; for, upon questioning him, he informed me that for some considerable time he could neither lie upon the sides or back, unless the head were greatly elevated, being prevented by the violent palpitation of the heart. He added, moreover, that when he tried to lie upon either side, especially upon the left, it seemed to him as if the strokes of a hammer fell upon his ribs. After the examination, I observed to the surrounding physicians and students, that a polypus of considerable size had formed in the left auricle, but that the left ventricle was free. But, in consequence of the absence of dyspnea, together with the character of the pulse, that the passage of blood from the right to the left heart was free and unobstructed, which never happened if a polypus of any size had formed in either of the ventricles. I also stated, moreover, that independently of the polypus, there was something unusual in the case, concerning which I could form no accurate opinion, but which the death of the patient would soon unravel. My prognosis was justified, for the patient died in three days after this investigation.

"*Autopsy.*—A polypus was found in the right auricle, the left ventricle was dilated to an extraordinary degree, and the ascending aorta was thickened, hard, and cartilaginous. The sigmoid valves were serrated on their unattached borders, resembling, in shape, the teeth of a saw, and, in consistence, stone or gypsum.

"*Remarks.*—In consequence of the rigid state of the aortic semilunar valves, very powerful action of the left ventricle was necessary to propel the blood into the aorta; and, since these valves were serrated and torn at their free border, they could no longer close: consequently, at each systole of the aorta, blood was forced back into the cavity of the ventricle. There existed, therefore, a derangement of the circulation, produced by the rigidity and calcification of the aortic valves, which had caused the increased action of the heart."

In the year 1749, Senac gave to the world his classical Monograph upon the diseases of the heart. It is literally, as has been said of it, "*opus magni sudoris.*"

This work contains the results of a life of almost undivided labour in the cause. Writings which obtained the unqualified commendation of Morgagni and Haller, need no further recommendation to the modern investigator of the history of Cardiac Pathology.

Senac was clearly cognisant both of obstructive and regurgitant valvular disease, and of the influence these conditions exercise both upon the chambers of the heart and the arterial pulse. But, his knowledge, of *obstructive* disease was, apparently, restricted to the arterial sigmoid valves, and that of *regurgitant* disease to the mitral valves.* He mentions, however, two cases; one from a great authority on diseases of the heart, Ballonius; the other from Malpighi, which would seem to be connected with aortic Insufficiency, from dilatation of the aortic mouth.

In the case of Ballonius, a man aged twenty-two was long troubled with palpitation. There was visible beating of all the arteries. He died suddenly after swallowing a bole of cassia, which was supposed to have caused his death. The true cause, however, of this unexpected event, resided in the aorta and heart, the cavities of which were extremely dilated.

The case narrated by Malphigi runs as follows:—

"A man, in whom the pulse was strong and tense, and into whose arteries the blood was projected with violence, had the left ventricle so large that it could have contained a heart of moderate size. The area of the aorta was three inches, and its internal surface thick set with bony scales, and the walls very solid."

The great author of the Letters on the "Seat and Causes of Diseases," has devoted several of them to the consideration of the diseases of the heart. With few exceptions, these letters contain all that is known, even now, with regard to Cardiac Pathology. The reflections that Morgagni has made upon the subject of aortic Insufficiency in connection with sudden death, are so judicious that they apply equally at present as when written. The following case is given in the twenty-seventh letter:—

"12. A young man, of an excellent habit and proper

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* Tome ii, liv. iv, ch. ix. When the valves of the aorta are ossified, the blood is impeded in its exit; the pulse is small, and the heart liable to palpitation. But, when the auriculo-ventricular valves are ossified, and incapable of closure, as in the case narrated, what derangements disturb the action of the heart? The blood must reflow into the auricles during the contraction of the ventricles. Less blood passes into the aorta, and the pulse, consequently, is but little dilated: but if the auricle is distended at the time the ventricle contracts, palpitation necessarily occurs.

conformation of body, and of a good stature, having been long troubled with a difficulty of breathing, was relieved by blood bursting forth, every now and then, from the nostrils. But, after he had begun to be deficient in this discharge, he happened to take a long journey from Trent to Padua, which he performed, partly on foot and partly on horseback, within the space of two days. Immediately after coming off his journey, and while he was stooping to his portmanteau, which was laid on the ground, he fell down dead.

The thorax being cut into on the day following, the vessels of the head and the neck were previously observed to be very turgid with blood. But when the cavity was opened there appeared to be no extravasation in it, nor were the lungs seen to adhere, anywhere, to the pleura. Both the lobes of this viscus, however, were livid, from the blood stagnating in them; and they were small besides, from the vast magnitude of the heart, which (when the pericardium was cut into, where there was no small quantity of reddish serum) appeared to be even bigger than that of a bullock. And, indeed, the auricles, and the right ventricle, were bigger than usual; yet, the greatest bigness was observed to be in the left ventricle. Nor, was this owing to the parietes, which were not thicker than usual; but to the cavity of the ventricle being dilated to such a degree that nothing could be conceived as being more so, and not only filled with a quantity of black blood (that had not formed itself into polypus concretions, though it was somewhat collected into grumous coagula), like the other cavities of the heart, but also distended therewith. Besides these appearances, the semilunar valves, which lie at the orifice of it, were not bony, indeed, but hard; and what immediately occurred to the eyes, very small, for they were contracted and corrugated. But the great artery, although it was not larger than it naturally is, yet in proportion as it receded from the heart, so much the thinner than usual were its coats. Nor was its internal surface entirely free from longitudinal furrows, notwithstanding they were somewhat obscure. However, in the heart and the whole thorax, there was nothing besides that was not natural. . . . . .

"14. You will, perhaps, ask why, as all the four cavities of the heart were dilated in the young man in question, the dilatation of the left ventricle was, nevertheless, the greatest. Without doubt, because the dilatation of this ventricle had given rise to the dilatation of the other ventricle and the two auricles; that is, by admitting a less quantity of blood

than it ought, for the reason which I have just now given you; and, in consequence of this obstruction, by retarding the motion of the blood in the left auricle, in the lungs, in the right ventricle and its adjoining auricle.

"But why was the left ventricle the first of all to be dilated? Why? Certainly, because the semilunar valves, whatever the cause of this circumstance might be, having been contracted and corrugated, could not properly expand themselves so as to prevent the blood being, in part, sent back into the ventricle from whence it came, during the contraction of the aorta; which part of the blood would, perhaps, have been less, if the coats of the more distant parts of the aorta had been able to drive on towards the veins the proper quantity of blood which it had received; but this, the thinness of these coats—that is, the decreased number of their fleshy and elastic fibres—did not permit."

In the 23rd Letter, also, Art. 12, he again alludes to obstructive and regurgitant disease of the aortic valves, and shows most clearly how the lungs and right ventricle, at times, become implicated:—

"Wherefore, in the woman also spoken of by me, besides the aorta being here and there bony, or inclining to a bony state, the valves of it are, also, to be attended to. For, as one of these was bony, and the others indurated, so being, of consequence, less yielding to the blood, they might increase the obstacles to its exit, and, on the other hand, not sufficiently prevent its return, when soon after repulsed by the great artery: so that, as some portion of it returned into the left ventricle of the heart, when the ventricle ought to receive the blood that was coming in from the lungs, it would necessarily happen, that the returning portion, as well as the portion which had not been extruded just before, must occupy some part of that space which, from the design of nature, was entirely due to the blood that was coming in from the lungs; which circumstance, finally, could not but overload both the lungs and the heart, and compel the latter to throw out, every now and then, with a great impetus, the blood that stagnated in it.

Christian Theophilus *Selle*, a physician of celebrity, published in 1790 his "Observations on Medicine," or rather, in part, a translation of the work of Brocklesby. That he saw the pathological bearing of the disease, is clear, from the detail of his 24th observation, "*Ossification of the Valves of the Aorta.*"

"In 1781, a young man of robust constitution, ætat. seventeen or eighteen, was brought to the "*Maison de Charité.*

"I observed a considerable pulsation in all his arteries. He had violent palpitation of the heart, and a pulse singularly large, quick, and hard, although quite regular. He could get no rest, from the difficulty in lying down.

"All the other functions were in their natural state.

"I had no doubt of organic disease, and reasonably presumed that an aneurism existed, which would, sooner or later, burst and cause death.

"I tried to diminish the mass of the blood by bleeding, but this did not at all lessen the pulsation of the arteries, neither were saline nor anodyne medicines of greater efficacy. A leuco-phlegmatic condition ensued, and death soon followed. Upon opening the body, I was greatly astonished to find no disease in the vessels. All the viscera were equally sound, but the valves of the heart were ossified and immobile. They were greatly retracted, and in such a way, that the blood ejected by the heart could readily pass; but, as a consequence of such retraction, it would naturally reflow, and occasion this disorder of the arterial system."

The writings of Laennec show no familiarity with the disease in question. Although well acquainted with the changes of the Aortic Sigmoid Valves, which obstruct the onward current of the blood,* he does not appear to have been aware that these might, also, permit a regurgitant one. It is the more singular that the marvellous sagacity of Laennec should have failed him here, inasmuch as, he has even shown how the valves were, at times, curled round upon themselves.†

It may, perhaps, be matter of question, whether he did not occasionally confound aortic Insufficiency with what he designated "Spasm of the Arteries, with murmur and fremitus."‡ His distinguished editor, Andral, gives him no

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* Traitè de l'Auscultation. 1837.

† Op. cit., tome iii, p. 264-9.

‡ Op. cit., p. 513-15. In a large number of cases, in which slightly marked bellows murmur exists in some arteries, the radial pulse conveys a peculiar fremitus, a kind of vibration exactly resembling that of a tense metallic cord, which, after being made to vibrate, is touched with the tip of the finger. This character of the pulse is, probably, that observed by Corvisart in cases of ossification of the mitral valve where fremitus exists over the cardiac region. It would appear to be a simple diminution of this latter phenomenon.... [This view of Laennec, appears to be similar to that of Bertin.—J. C.]

credit for knowledge of the affection, but admits it to be of later growth.*

Bertin, whose collected writings upon the *Diseases of the Heart* were edited by Bouillaud and published in 1824, has devoted a most valuable section to the Diseases of the Aorta and Sigmoid Valves, consequent upon inflammation.

This distinguished pathologist, however, was so completely fettered by the dogma of *Frank*, respecting the common occurrence of arteritis, that he has actually (obs. xxvii) detailed a case of Aortic Insufficiency, from dilatation of the mouth of the Aorta, and overlooked it, in consequence of preconceived views. It is headed :—" *Thinning, with Dilatation of the Walls of the left Ventricle ; Inflammation and Ossification of the Aorta, &c.* Anne Berger, huxtress, ætat. forty-seven, had suffered for about two years from spasms and other anomalous symptoms, which were supposed to be of a nervous character, dependent upon the ' Critical Period.' She was treated by various practitioners, who all regarded her complaint as ' nervous,' and prescribed accordingly, calmatives and anti-spasmodics, but without success. On her admission into the Hospital Cochin, the eighteenth prairial, an. 11 :—She complained of great dyspnœa. Each moment she was in dread of suffocation, and obliged to keep constantly in a sitting position ; she felt, she stated, as if something rose in her throat and threatened to choke her. She complained of a sensation of heat about her head ; her legs were slightly swollen. All the arteries sensible to the touch, appeared more dilated than natural : their pulsations were vigorous and accelerated, those of the carotids were very visible, the pulsation of the arch of the aorta occasioned a kind of elevation at the epi-sternal notch. The cubital arteries pulsated violently, and the patient said that she also felt internal pulsations. In constant dread of death, she yielded to despair, and became unconnected in her ideas. The cardiac movements were accelerated, but otherwise exhibited nothing peculiar. All the symptoms became augmented, and death took place on the 27th of the same month amid violent suffering.

*Autopsy*, twenty-six hours after death. Face tumid ; surface of the body marbled, presenting numerous livid patches of considerable extent. The left pleura contained a small quantity of reddish fluid. Heart larger than natural. Right auricle considerably distended. Nothing particular

* Op. cit., tome iii, p. 283.

in either right ventricle or in pulmonary artery. Left auricle normal; but the left ventricle was, at least, as big again as it should have been, and its walls were sensibly thinned. The great sinus of the aorta was very developed; the walls of the artery were hard and thickened in many places. The lining membrane was inflamed from its origin to the common iliacs, as also that of the Carotid Arteries and Sigmoid Valves.* Independently of this, throughout the entire extent of the vessels, small, white and hard tubercles were observed.

The remaining arteries were sound. Nothing particular was observed with regard to the abdominal organs."

The following is the reasoning of Bertin respecting this case:—"This observation offers us, in all the plenitude of their energy, the principal signs of arterial inflammation. These signs, indeed, consisted essentially in augmented action of the arterial system, in pulsations active, strong, vehement, and vibrating, such as were observed in our patient. The pulsations were so violent at the episternal notch, as to induce suspicion of aneurism of the arch of the aorta.

"Such violence in the arterial dilatations and contractions, is the more remarkable, inasmuch as it was associated with thinning and dilatation of the left ventricle, a circumstance unfavourable in a two-fold sense, to the force and vigour of the pulse. We are, consequently, compelled to admit a proper contractile force in the arteries, and an action, to a certain extent, independent of that of the heart." †

In the Edinburgh Med. Chirurg. Transactions, Vol. I, 1824, Dr. Abercrombie has, with his usual accuracy of detail, described a case of Aortic Insufficiency, with the *post mortem* appearances; but he has utterly failed in seizing the pathology of the disease.

"To these desultory remarks, I shall only add one case of active aneurism of the left ventricle, accompanied by an appearance which is very uncommon.

"Case 21. A man, aged about forty, received a severe injury of the left side of the thorax, by a fall from a horse. He soon recovered from the immediate effects of it, but was from that time affected by a train of obscure symptoms in the thorax, which, after a considerable time began to assume the characters of an affection of the heart. There was ob-

* This appearance was, doubtless, caused by simple imbibition.—J. C.

† Inflammation de l'Aorte, p. 35.

scure deep-seated pain, with occasional attacks of dyspnœa, and a remarkably strong, but regular, pulsation of the whole arterial system, particularly a peculiar and strong jarring of the carotids and subclavians; the pulse generally about 120. The action of the heart was rather stronger and more extended than natural, but by no means corresponding with the remarkable strength of the arterial pulsations, upon which large and repeated blood-letting made almost no impression. His breathing became more and more difficult, with extensive anasarca, and he died about five months after the period of the fall.

Dissection.—The left ventricle was nearly twice the capacity of the right, and in its substance much thickened, and very firm; the columnæ carneæ were much enlarged. One of the semilunar valves presented the appearance of a ring, its body being perforated by an irregular opening, which occupied the greater part of it, and gave an appearance as if it had been torn from its attachment along its base, remaining attached only by the two angles. The other valves were healthy. The right ventricle appeared somewhat enlarged; and the right auricle was nearly twice its natural size, and very thin.

"The prominent symptom in this case was the peculiarly strong pulsation of the arterial system, especially of the large arteries about the neck. The pulsation of the heart did not by any means correspond with it, and, indeed, was much less remarkable than in some of the preceding cases, in which the ventricle was enlarged without thickening. It is also worthy of observation that a strong and extended pulsation may exist without enlargement."

In the London Medical Gazette for the year 1829, Dr. Hodgkin published his observations upon one form of Disease of the Aortic Valves, permitting reflux into the cavity of the left ventricle, and termed by him "Retroversion of the Valves of the Aorta."

These papers are very valuable, more particularly in an anatomical sense. It is the first example of a series of cases brought forward to show the influence of diseased conditions of the Aortic Valves, admitting reflux, upon the left chamber of the heart.

The anatomical description is given with Dr. Hodgkin's characteristic accuracy in every particular. With regard to the constitutional and physical signs, in this early stage of auscultation, much was not to be expected. In one case, however (that of Dr. Cox,) one of simple Aortic In-

Insufficiency, the double murmur was particularly noticed, but without allusion to its site, or explanation given of its cause. So far from this, Dr. Hodgkin remarks, "The contractions of the ventricles were marked by strong impulse, and a constant *bruit de scie*, which presented this peculiarity, that it was double, attending the systole as well as diastole, but not exciting the idea of being at all connected with the auricles. Yet, by a singular coincidence, at the end of this paper, when again adverting to this case, Dr. Hodgkin seems to have divined the physical cause of the double murmur he heard, for he writes, "It would still appear, that, in the majority of instances there is no *bruit de scie* accompanying retroversion of the valves. The peculiar character of this sound in the case of Dr. Cox is well worthy of attention, as connected with some of the appearances noticed in the inspection. It was repeatedly observed to offer a double or spondaic character, the one part marking the systole, and the other the diastole of the ventricle. The spots of partial thickening on the interior, both of the ventricle and of the aorta, and which had evidently been occasioned by the contact of the elongated and much retroverted valve, sufficiently prove that the blood on the left side had been subjected to two motions, the one progressive and the other retrograde, in both of which it might easily give rise to some sound as it passed the elongated valve." Dr. Hodgkin states that in these cases the force of the pulse does not correspond to the heart's beat. The violent beating of the carotids, and the thrill of the pulse are, also, recorded. The distinction, too, between the generally undisturbed rhythm of the pulse in Aortic compared with Mitral valve diseases, is expressly stated. Dr. Hodgkin appears to have been taught by his experience that in this particular class of cases, "depletion seems rather to aggravate than to relieve the distress of the patient."

These Memoirs contain the germs of our present knowledge with regard to both physical signs and treatment. Indeed, had Dr. Hodgkin but traced the physical signs with the same success that he did their anatomical cause, the next acute observer had been completely anticipated.

From this date until the year 1832, no additional observations were recorded illustrating the Pathology of *Insufficiency of the Aortic Valves*. Throughout the entire historic period of the disease, isolated cases only had occurred, and these so

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* Edin. Med. and Surg. Journal. 1832.

widely scattered through the general literature as to have eluded any attempt to classify, and deduce from them, any general pathologic principles. But, in the year named, a living pathologist of eminence, Dr. Corrigan,* published his well-known Memoir. He may be considered, in the strictest sense, the discoverer of the malady; the very first physician to correlate the signs and morbid changes. It is true, that objection might be taken to some of his positions, and, perhaps, from more extended experience, he might himself be disposed to modify some points of his doctrine. I would particularize the assertion that the cardiac impulse is unchanged; again, that collapsing vessels are a *sine quâ non*, as it were, of the disease; and, also, the statement of the non-occurrence of pulmonary congestion. In one other sense I regard Dr. Corrigan's paper as incomplete, viz: in not only entirely overlooking the accident of sudden death in these cases, but even implying an immunity from such event. This contingency, now well ascertained, is most important in a prognostic sense. Still, despite these (except in the last named sense) minor matters, Dr. Corrigan's paper is indispensable to the student of Aortic Disease. Indeed, its value, in a Therapeutic sense, is immense. He looked at the disease with a vision clouded neither by authority, nor prejudice, but rather as a physiological pathologist, watching the efforts Nature herself made to mitigate the evil. He was thus enabled to rescue the treatment from blind routine, and to place it on so firm a basis that it never has or could be again abandoned, without manifest disadvantage and even danger.

Dr. Hope* had noticed the "jerking pulse" in Insufficiency of the Aortic Valves, as early as 1831, and, in the third edition of his Work states, moreover, that the murmur in this disease "is louder and more superficial, opposite to, and above the aortic valves than about the apex of the heart, by which it is distinguished from a murmur in the auricular valves with the second sound." Dr. Hope's observations connected with the pathology of Aortic Valvular Insufficiency are very brief, but he must have the credit of being the first to indicate the general principle that the murmur was engendered at the immediate site of, and by, the changed valves themselves. This was a great advance in Physical Diagnosis, inasmuch as murmur, replacing in part, or entirely, the second sound at the base of the heart, and *a fortiori* if of musical character is, when present, really pathogno-

* Diseases of the Heart, p. 380. 1839.

monic; the exceptions being so rare that they may, in a practical sense, be wholly disregarded.

On the 6th of June, 1834, Dr. Guyot presented to the Faculty of Medicine of Paris his admirable thesis, *On Insufficiency of the Sigmoid Aortic Valves.* He has clearly shown, how considerably the condition of the aortic valves, modifies many of the signs enumerated by Dr. Corrigan. They may, for example, yield readily to the systolic contraction of the heart, and yet be widely patent during its diastole. In such a case, the phenomena of collapsing vessels would be strongly marked; or, on the other hand, the valves may have the power of motion so limited, consequent upon calcification of their tissue, that they are almost motionless in the stream, unable to be thrown open sufficiently wide for the outward gush, or to shut close enough to act as a floodgate to the refluent current. Here, double murmur is met with, showing the conditions alluded to.

Two years later, Dr. Charcelay defended his thesis on the same subject at the Faculty. It is well worthy perusal, and contains much interesting detail connected with the various morbid conditions of the valves in their causal relation to the disease in question. He accepts the physical signs already current.

Almost the last contribution to the literature of Aortic Insufficiency, superadding any physical sign, is from the pen of Dr. Henderson.* The paper is a most valuable one. It clearly and correctly points out how some of the signs enumerated by Dr. Corrigan may be, indeed often are, at times wanting, at all events in the fulness implied in Dr. Corrigan's paper. To supply such defect in the symptomatology of the affection, Dr. Henderson is of opinion that he has discovered a sign of occasional importance: "a greatly increased interval between the systole of the heart, and the pulse of the remote arteries, such as the radial." The question is, has clinical enquiry proved such condition an essential or only accident of Aortic Insufficiency? I believe the latter, and that some degenerative change in the walls of the heart, is additionally present in the cases presenting such phenomenon. I have examined, carefully, a large number of cases of the disease without discovering the connection. Dr. Henderson's statement, that the tortuousness of the arteries becomes more remarkable as the disease continues, requires, so far as I can judge, some qualification. I believe this only happens in cases of marked atheromatous degeneration.

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* Edin. Med. and Surgical Journal, Oct., 1837.

Although, since this period, many esteemed contributions have been made, they have rather been in the way of confirmation than extension of the signs described, and more particularly with reference to the occasional results of the disease.

There is, however, one other addition to the physical signs to be mentioned, and that is the phenomenon of the so-called "sounding pulse" of Bamberger. He states that, in health, two sounds are heard, on auscultation, in the carotid and subclavian arteries, but are lost in those more remote, as the brachial, radial, crural, etc. In Insufficiency of the Aortic valves, these relations no longer obtain. Not only in the last named arteries, but even in those of still smaller calibre, as those forming the palmar and pedal arches, a sound is audible during their diastole, or, more correctly speaking, a short, almost noiseless shock, nearly resembling that produced by a fillip on the nose. This constitutes a fourth peculiarity of the pulse, very rarely observed under other circumstances, and then only in the larger arteries, as the brachial, crural. The cause depends upon the stronger tension of the elastic arterial coats, and therefore becomes less distinct as the elasticity of the arteries diminishes, until, in extreme degrees of rigidity, it may wholly disappear. To appreciate this phenomenon clearly, the stethoscope or naked ear must be lightly placed over the site, since pressure generates a hissing murmur without significance, as it is heard even in the normal state. If Insufficiency and particularly the secondary Hypertrophy of the heart are not marked, or great debility exist, the sounding character of the pulse may fail.*"

This phenomenon must not, I suppose, be confounded with the musical sound, at times, transmitted from actual disease of the valves themselves.

Thus far it has been my endeavour to chronicle the efforts made towards perfecting the Pathology and Diagnosis of Insufficiency of the Aortic Valves. As happens with each Discovery, it was seen, at first, how isolated the cases were, and how long and difficult was the task of generalization. This process was, perhaps, mainly owing to the more rigorous cultivation of morbid anatomy in connection with improved methods of physical research. The motto adopted by *Piorry* was never better exemplified. "*Pouvoir explorer est une grande partie de l'art.*" The early masters, Vieussens,

* Lehrbuch der Krankheiten des Herzens. 1857.

Lancisi, Albertini, Valsalva, Senac, and Morgagni were conversant with Inspection and Palpation only. By Inspection must be understood not merely the amount of visible cardiac impulse, or the phenomena appertaining to the arteries and veins, but also the precordical bulging caused by enlargement of the heart. Under Palpation, also, is included both the force of the heart's impulse and the peculiarity of the arterial beat. Later, however (1763) Physical Exploration acquired an addition of the greatest value, by the introduction of Percussion, by Auenbrugger. The heart could now, within certain limits, be made the subject of actual measurement. "Speaking of the signs of enlargement of this organ," he says, "Signum pathognomonicum hujus mali est, quod locus, ubi cor situm obtinet, percussus in magnâ circumferentiâ, carnis percussæ sonitum exactè referat." To the great commentator of Auenbrugger, Corvisart, Science owes the detection of another important sign, developed by the heart and arteries, namely, the tactile phenomenon known as *fremitus*, or thrill.* It was, however, the peculiar privilege of Laennec, by the invention of the Stethoscope, to give the finishing stroke to the sense-edifice of Cardiac Diagnosis.

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* It would, perhaps, be more correct to say that Corvisart first gave precision to this sign, as significant of disease of the mitral orifice. Selle (opt. cit.) had already described it. Speaking of a case he saw in 1782, he writes, "She suffered unceasingly from such violent palpitation, that each beat of the heart was visible when the chest was exposed, and, on palpation, a kind of oscillation was present, which was attended by *bruissement* [fremitus.] I thought an aneurism existed."

END OF PART I.

## PART II.

The following observations concern some points of the affection known as "Insufficiency of the Aortic Valves," together with the influence such condition exerts upon the chambers of the heart, and, more particularly, its connection with sudden death—an occasional accident of the disease which merits far greater attention than it has hitherto received.* Indeed, the omission of this accident is almost the only one of importance in the well-known memoir of Dr. Corrigan—a memoir to which we are all so largely indebted.

Before discussing, however, the pathology of the affection, I would invite a reconsideration of the physiology of the circulation of the blood in the coronary vessels of the heart, inasmuch as our right comprehension of the accident alluded to may greatly depend upon the views held as to the time and manner in which the arterial blood enters the coronary arteries. Upon this point, even in the present as in past time, complete unanimity has not prevailed.†

Perhaps, the very large majority believe, with Haller, that the coronary arteries are filled by the heart's systole, and, of course, during the dilatation of the aorta. But, some few, and I include myself among the number, think it not improbable that the arteries are filled by the systole of the aorta, and, consequently, during a period corresponding to the heart's diastole. So far as my knowledge extends, Boerhaave first advocated this view, in his Physiological lectures. Others, to reconcile the difficulty, have suggested that the arteries receive blood during the acts of both systole and diastole.

The chief arguments against the injection of the arteries during cardiac systole, may be thus stated;—first, the retrograde course taken, and angle formed, by the arteries: second, the position of these vessels in relation to the sigmoid valves;‡ and last, the apparent want of adaptation of

* The memoirs of Aran and Mauriac should be consulted. The last named author has given a statistic of the cases of death which have occurred in aortic insufficiency.

+ Even at a recent period, the subject has been discussed with unusual acrimony by Brücke and Hyrtl.

‡ For the state of opinion on these points at the time of the early Masters, who, by the way, paid quite as much attention to them as ourselves, see Senac (opt. cit.) liv. i. ch. v. tome i.

the time, as respects the nutrition of the cardiac muscle. There is experimental proof that, during the period of systole, no blood enters the parenchyma of the organ; the observations of Harvey, showing that, in some animals, during this act, the cardiac wall, while it is most dense, is at the same time most pallid: semitransparent, and only darkens during diastole, when the tissue is again pervious to the blood.

It is, I think, no valid objection to this view, that Haller should have observed blood forced out of the arteries during systole, inasmuch as this act would naturally give rise to reflux of a given portion of blood from the interstitial branches into the primary trunks, and thus explain the phenomenon witnessed by this great observer, upon section of these vessels. It is clear, that the tissue is rendered pallid from the reflux of the arterial and onward passage of the venous blood.

The facts favouring the hypothesis, that the coronary arteries may be filled during the systole of the aorta and dilatation of the heart, are: first, the unique course and condition of the coronary arteries, placed with their mouths in direct contiguity to the aortic flooring, and, in their course from the aorta, taking a direction easily to be injected by the blood after its shock against, and reaction from, the closed sigmoid valves. It is, also, during the relaxed condition of the cardiac walls, that the interstitial vessels are most readily filled, and thus, probably, the subsequent act of contraction, in part at least, determined. Experiment additionally testifies that the coronary arteries, when injected with blood, immediately stimulate the heart to contraction.*

It would certainly be unsafe to draw rigorous conclusions from such coarse experiments as we are permitted to institute on the dead subject, with regard to the systolic injection of the coronary arteries: the process of ventricular contraction is so essentially vital. But we can, with far less risk of error, experimentalize upon the aorta and its valves. Here the mechanism, aiding the general circulation, is of an almost purely physical character. When the blood tension is greatest on the ventricular wall, the pressure tends to raise the aortic valves. When, on the contrary, the tension preponderates on the side of the arterial walls, the valves are

* It is not from any single fact, but from the collocation of many, that the argument gains in po nt of probability. One series of phenomena, apparently trivial, when in juxtaposition w.th others, not unfrequently assumes a high degree of importance.

closed. If the aorta and its valves are healthy, and freed from clot, and the coronary arteries dissected and divided, water, poured in the common trunk and submitted to gentle pressure downwards, most readily passes by the divided coronary vessels.

In further support of this position, it was found, by the philosopher *Bernouilli*, that if fluid was injected into a tube of given *calibre*, it would pass with facility into secondary tubes, the direction of which was in harmony with that of the main branch; but if, on the contrary, the direction of the secondary tubes was opposed to that of the trunk, not only would the onward current be prevented passing therein, but that fluid would actually be drawn up into the latter from a reservoir below.

It now remains to be shewn how far the view I advocate can be made to harmonize, in some essential particulars, with the Pathology of the disease in question.

The ventricles of the heart, in common with every other organ of the body, require some respite, however short, from active labour. Such respite is afforded by the Semilunar valves which, by a mechanism purely physical, resist the downward pressure of the columns of blood completed, by the last contraction of the ventricle. Thus is the heart, as it were, momentarily relieved of the principal portion of its burden. In a healthy physiological condition, such relation is maintained between the power of the ventricles and the resistance to be overcome, that they are enabled at their next contraction to force these flood-gates barred by backward blood, and thus to maintain the balance of the greater and lesser circuits. But should these gates refuse to open, or become too narrow ever again to close, then the heart, baffled by the unusual, though intermitting effort, or exhausted by its necessary constancy, may gradually sustain such injury, both in its vital and physical endowments, as is no longer compatible with health or even with life. It is, however, more particularly in reference to insufficient closure that the present remarks are intended to apply, and it is my aim to show, moreover, how such faultiness in the valvular mechanism may furnish the conditions under which sudden death by Cardiac Syncope occasionally occurs.

It will be found both practical and convenient to divide Insufficiency of the Aortic Valves into three stages:—the Incipient, or Irritative; the Physiological, or confirmed; and the Degenerative, inasmuch as it is the Ventricle which plays the leading part in, at least, the two latter stages of the affection. The first stage, when the direct result of Rheumatic

valvular aortitis, so far as I have observed, appears to be one simply characterised by the signs of general Cardiac excitement, and in no wise susceptible of Physical Differential diagnosis, from Mitral disease, or even nascent Pericarditis.* Increased, often irregular, impulse, obscured sounds, and, later, slight systolic murmur, are alone observed. It is true that in pericarditis, when either a friction or leather-creak sound is audible, or the percussion area becomes suddenly and greatly enlarged, there could be little chance of diagnostic error. But it is found, clinically, that pericarditis in its developmental stage, at times occurs without the stethoscopic signs, and certainly without any greater extent of dulness than would consort with that caused by a heart enlarged by distension of its cavity and congestion of its walls. The *second stage* is, as is well known, characterised by the striking phenomena of greatly increased impulse of the heart and throbbing or collapsing of the superficial arteries. Here the Physiological Hypertrophy of the chamber, consisting of a real addition of healthy, contractile tissue, is a genuine compensation—an instinctive accommodation of Nature, under injury sustained. Let us consider how impossible it would be for the circulation to continue if the strength of the left ventricle were unchanged and any great amount of reflux occurred. For the requirements of the Economy there must be as much blood sent into the aorta *plus* the quantity which reflows. To make this provision, is the final cause of the hypertrophy of the left chamber. The amount of collapse of the vessels, practically speaking, measures, as I believe, the extent of patency of the Aortic valves. This second stage, may at times, persist for years without passing its original limits.

It is in the early period of the third, or Degenerative stage, that we are to anticipate the supervention of the accidents or complications of the affection. I wish, here, merely to allude to such as involve the Lungs, Right heart, and aortic trunk. There are two modes by which the lungs and Right Ventricle become implicated in this affection. One, almost purely mechanical; the other, vital. The former acts

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* In this stage, if endocarditis is the originating cause, the valves are *tumid* from subserous effusion. This condition, while muffling the sounds, may really prove for a time an additional barrier to regurgitation. Dr. Richardson's experiments in this particular are most interesting. It is in a subsequent stage, when the plastic matter unabsorbed is gradually undergoing contraction, that the phenomena of regurgitation become as gradually developed. Regurgitation may also occur from vegetations on the free edges.

in the following manner. When the dilated hypertrophy of the left Ventricle has attained such magnitude as to merit the epithet of "cor bovinum," it occasionally encroaches so much upon the area of the left lung, as to cause considerable condensation of its tissue. Such condensation, by obstructing the pulmonary circulation, tends directly to the production of dyspnœa, catarrh, hæmoptysis, and almost necessarily, as in Mitral disease, to dilatation of the right side of the heart.

To the second, or vital, mode I shall shortly allude.

The aortic trunk, also, sustains direct injury. The constant over-force of the ventricle tends, first, to the dilatation of this discharging tube, and eventually, at times, to its entire loss of elasticity and atheromatous degeneration.

These conditions, in their turn, react injuriously upon the chamber. The impaired elasticity, by obstructing the onward current, furnishes its contingent to the Hypertrophy, and, in the Degenerative stage, weighs heavily on the failing power of the heart. Morbid anatomy has recently brought to light the strict correlation between Hypertrophy of a Ventricle, and atheroma of the trunk in immediate connection with it. Even in hypertrophy of the right Ventricle, it is frequently found that the pulmonary artery has undergone atheromatous degeneration.

Such change in the aorta very often extends to the mouth of the coronary arteries, modifying the nutrition of the cardiac walls, and tending to still further results.* I cannot too emphatically redirect attention to the diseased condition of the aorta and destruction of its elasticity, as an almost invariable sequence of disease of the aortic valves.

This undeniable fact I regard of cardinal importance, inasmuch as such incapacity of reaction of the aorta must, if the view I advocate of the time of injection of the coronary arteries, be correct, prevent either a constant or sufficient repletion of these nutrient vessels, and thus directly favour cardiac mischief. Here, I apprehend, we but trace the operation of a recognised law in pathology—that in proportion as an organ is over-worked and under-fed, degeneration of structure is sooner or later of almost inevitable necessity.

No affection could more strikingly exemplify the fatal motto of Corvisart—"*hæret lateri lethalis arundo.*" From the constant overstrain of the ventricle; the perverted nu-

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* If the natural supply of blood is cut off from a muscular organ, paralysis will as speedily and certainly follow, as if all communication with the brain were interrupted.

trition of the cardiac walls; and from the cachexia, general and local, induced by disturbed balance of the circulation, the left ventricle, eventually, undergoes an irreparable change of a fibroid or, more commonly, of a fatty nature. The extreme limit of the third, or Degenerative stage, is then attained.* The Ventricle, no longer capable of efficient contraction, still further yields from the blood-pressure on its walls, and secondary incompetency of the Mitral valve occurs. This enlarged capacity of the left chamber must never be lost sight of in Insufficiency of the Aortic Valves. It is here that it nearly attains its *maximum*. It is the direct cause of the cyanotic signs, and, remotely, furnishes a condition for sudden death. The marked throbbing, too, of the heart and arteries gradually lessens, only to be reproduced at intervals. This fact is often noticed even by the non-professional observers of the case.

In consequence of the incompetency of the Mitral valve, just named, being superadded to the diseased walls, there is a transference of the objective signs from the left to the right side of the heart, and now all those phenomena, at times, gradually appear, common to the closing stage of heart disease. This is, perhaps, the more ordinary clinical history. But there remains the consideration of the mechanism of those accidents, so to speak, by which life is, occasionally, suddenly cut short. Leaving among the outstanding points, death by embolia, or plug detached from the parts immediately diseased, sudden death in Aortic Insufficiency is almost invariably syncopal, or blended with more or less of asphyxia. Two causes exist for the occurrence of this accident; the one of extrinsic, the other of intrinsic origin. The extrinsic cause is sudden pericardial effusion, induced by the cardiac venous congestion from the diminished *vis a tergo*, owing to insufficient blood tension of the coronary arteries. The intrinsic cause, I assume to be as follows, although it is difficult to state the precise time or invariable exciting cause. It may be mental shock, bodily pain, effort, or general exhaustion. But, granting the degeneration of the enormously dilated left ventricle, whenever, from the influence of these exciting causes, singly or concurrently, the exhausted heart permits the diastole to be prolonged and the *hiatus* at the aortic mouth is sufficiently large, the systemic blood flows unimpeded into the left chamber, which acts as a vast reser-

* Degeneration of the tissue, whether fibroid or fatty, when extensive, must equally involve both capillaries and nerves.

voir.* Thus is the general system, and, particularly the brain, robbed of its needful stimulus. The *point d'appui* for the backward blood being lost, and the elasticity of the aorta gone, the coronary arteries, on the theory given, remain unfilled. Add to this, the disposition to paralysis of the left ventricle from the enormous blood-pressure on its walls, and we have the sum of the conditions under which death occurs, by what is known as Cardiac Syncope.

The physical signs of Aortic Insufficiency are so well known, and for the most part so readily detected, that detail is superfluous. It is only with regard to the significance of one or two of them that I would ask the reader's consideration. First, it is matter of clinical experience that, occasionally, in cases of defective aortic valves, the first sound of the heart is absent, although the mitral valve is in its physiological condition. Now what explanation is to be given of a fact beyond dispute, accepting the common theory—that the first sound of the heart is caused by the closure of the auriculo-ventricular valves? What can be the relation existing between a defective aortic flooring, and the extinction of the first sound of the heart? I think I am correct in stating, that Dr. Traube, of Berlin, is the only clinical observer who has given a pathological explanation of this fact. His view is so original and, I think, so important, that I may perhaps be excused its introduction here.

"In cases of well-marked Insufficiency of the Aortic Valves, I have," says he, "frequently found a more or less decided flattening and elongation of the papillary muscles of the left ventricle, without any appreciable alteration of their volume. This condition stands in singular contrast with the marked Hypertrophy which the dilated ventricle undergoes; and it is the more remarkable as, in cases of dilatation and hypertrophy of the left ventricle, without affection of the aortic valves, the papillary muscles are, as usual, round and correspondingly hypertrophous. Upon a transverse section of these muscles, there is, almost constantly, observed a number of irregular white *striæ*, within which, as is shown by microscopic observation, the muscular structure is replaced by fibrous tissue. In the majority of instances there is no warranty for the notion of inflammatory exudation, since neither in the endocardium of the degenerated papillary muscles, nor on the wall of the ventricle, is such fibrous tissue observed. The

* Vesalius records an instance in which the left ventricle contained two pounds of blood.

change, consequently, appears to be essentially different from that of ordinary inflammation. The elongation and flattening of the papillary muscles, indicate, therefore, that these structures were exposed to a permanent *extension* in the direction of their vertical, and to a permanent abnormal *pressure* in the direction of their transverse diameters. Indeed, from observation of the phenomena occurring in cases of marked Insufficiency of the aortic valves, during the period of diastole, in the left ventricle, the conditions are discerned which have thus affected the papillary muscles. In such cases, the relaxed ventricle is supplied from a double source. It receives, simultaneously, blood both from the left auricle and from the aorta. As a consequence of this abnormally large addition, its walls must soon acquire a tension greater than the pressure under which the blood flows from the auricle. A current is consequently originated with a direction from the ventricle towards the auricle, and which effects the closure of the mitral valve.

"The mitral valve closed, the contents of the ventricle thus cut off from the auricle, are submitted for a period to the pressure equal to that which presses upon the blood in the aorta. The blood from this latter tube would continue to be poured into the ventricle to distend it further, until finally the tension of its walls is sufficiently great to maintain the balance of the pressure in the aortic system, or at least, until the moment preceding the ensuing systole. That this *series* of results do actually follow, two facts concur to prove. One is, that in Insufficiency of the aortic valves despite the integrity of the mitral valve, the systolic sound at the apex, so frequently fails; and the other, certainly a rare one, is, that in insufficiency of the aortic valves a loud diastolic sound is audible at the heart's apex, but at no other point of the cardiac region. The condition of the papillary muscles, during these phenomena, admits of the following explanation. From the moment that the mitral valve is closed, their points of insertion into the cardiac wall, in consequence of the increasing distension and tension of the Ventricle, become, continually, further removed from the point at which their tendons unite with the curtain of the valves; whilst the bellies of these muscles become more and more strongly pressed. If we reflect, on the one hand, upon the slight comparative elasticity of the living muscle, and, on the other, on the unusual amount of the forces acting upon it, it is obvious that the frequent recurrence of these actions for years with each diastole must, finally, produce permanent

disturbance of the sarcous elements from their natural equilibrium, and, consequently, a permanent elongation and flattening of the belly of the muscle.

"But it is not less important to know that, under such conditions, the nutrition of the muscles also, undergoes a change.

"The molecular interchange between the contents of the capillaries and the cardiac muscular tissue, and, as matter of course, in those portions of it which constitute the papillary muscles, occurs, almost exclusively, during diastole, since, during contraction of the muscle, the contents of the capillaries must be almost exclusively forced into the veins. But under the influence of the unusual pressure to which the papillary muscles are subjected, in cases of insufficent aortic valves, the repletion of the capillary vessels cannot be normal, even during diastole; they must, in fact, be in part narrowed and in part pressed, even to the annihilation of their area. The necessary consequence of this condition is, as may be imagined, a disturbance of molecular interchange, in other words, a faulty nutrition of the muscular tissue. That muscular, under abnormal conditions, can immediately degenerate into fibroid tissue, is proved beyond doubt in the recent work of *Billroth*."

But, while the closure sound of the Mitral valve may, even in a physiological condition of this structure, become extinct from the cause assigned by Traube, we must not forget that in aortic Insufficiency the first sound of the heart may also become modified from a direct pathological cause: that is, by the continuous extension of the atheromatous process from the aorta to one curtain of the mitral valve; a fact explained by the Descriptive Anatomy of the structures.

I would next venture to criticise the ordinary teaching, that the Differential Diagnosis of aortic regurgitant murmur may be, at least in part, established by the clear click of the valves of the Pulmonary artery, auscultated to the left of the Sternum. I am far from asserting that this precept is not occasionally correct; but, so far as my opportunities of observation extend, I should, in the majority of cases, deny the validity of this proposition. When the diastolic murmur is at all intense, I believe that the sound of the pulmonary artery is always more or less obscured, and, in the case of Musical Murmur, *a fortiori*, completely so. Indeed, if we bear in mind the anatomical condition existant, and consider how readily vibrations are excited in contiguous and homogeneous structures, the difficulty of conceiving the reverse is obvious. The last point for question, relates to the significance of a

simple Diastolic Murmur, if at all well marked at, or even a little beyond, the left apex. Such point has before been hinted at in Traube's remarks, and I have, on more than one occasion, after death verified its connection with simple Aortic disease. Such a murmur may practically (for the exceptions are so few) be regarded as the physical expression of Aortic Insufficiency, and this, the more certainly, if percussion signs of enlargement of the Right Ventricle and general Venous stasis, are absent.

But, with regard to the Semeiotic importance of simple murmur, whether of limited or of wider range, we must be cautious not to allow it to transcend its just value. I would venture the opinion that such murmur indicates the kind, but not the degree of the lesion. This latter condition is measured, as before stated, by the amount of collapse of the vessels. Moderate obstruction at the aortic mouth is to be considered a most important kind of compensation for Aortic Insufficiency. An immobile, or still healthy valve, mingling its proper sound with the murmur, arrests and breaks the refluent stream, and thus directly tends to prevent collapse, as also that worst evil of the disease—Dilatation.

It is not, perhaps, saying too much, that the management of these cases is not unfrequently, even now, matter of mere routine.

To treat a diseased heart, not only with ordinary success, but even with ordinary safety, it is not enough that the practitioner be thoroughly conversant with the workings of Nature to compensate for injury done to its valvular apparatus, or even, when her resources are exhausted, with the simultaneous or successive steps that lead to death. Perhaps the larger and more valuable part of his knowledge is derived from the clinical study of the Congestion of the heart, as also of the Congestions and the vicarious action of the other large organs;—now acting as safety valves by increased secretion; and now again, as a consequence of such Congestion being inadequate to the task of eliminating refuse material, thus giving rise to a chronic asphyxiating process, and eventually entailing degeneration of their proper structures. The laws, too, of the reciprocal influence of the various organs, are of the greatest importance to a right appreciation of the use of remedial agents. It is astonishing in cases of long-standing disease of the valves associated with a weakened heart, how slight a cause will terminate life. Ordinary catarrh, congestion of the coronary veins, may determine,

respectively, pulmonary œdema or effusion into the Pericardium, and it requires but a small quantity of such effusion to exhaust the already weakened heart.

In the incipient stage of aortic valvular disease, the most important question that Therapeutics can ask of Pathology, is that which regards the causation of the affection. If this is the direct result of Rheumatic fever, those agents are of course indicated which experience has invested with a specific action over the rheumatic poison, and, subsequently the long continued use of moderate counterirritation, with the internal use of iodide of potassium, alone or in combination, with a view to the absorption of the exudation matter. Simultaneously with these means, the avoidance of the ordinary causes of cardiac excitement is to be sedulously cared for. It is certain that valvular disease of the aorta, occurring as stated, is amenable to treatment and occasionally so far modified as to leave no signs of either general or local disorder. On the other hand, when Atheroma is probably causative of the affection, treatment is, for the most part, abortive.

It is ever to be remembered that, in this incipient stage, the phenomena of cardiac excitement are, for the most part, but the reflex of the obstructed blood current. So that here we treat the valve disease, *qua* valve; while, in the later stages, with permanent valvular obstruction, it is to the chambers of the heart that our remedial measures are to be addressed. In the second, or confirmed stage of Insufficiency of the aortic valves, the one most frequently presented to our notice, and characterized by so many striking and familiar phenomena, medical treatment is, strictly speaking, absolutely contraindicated. This stage may, and often does, endure for years with but little appreciable encroachment on the vital powers, unless any of the accidents of the affection, induced by some excess, should suddenly supervene. Our care should simply be to enforce, so far as is practicable, the avoidance of all the mental and physical influences likely to embarrass the heart's action. Attention to diet is of extreme importance. There is no cause of cardiac suffering, or even death, more common than improper diet, in the Organic diseases of the heart. The restricted use of fluids is important, to prevent the *plethora ad molem;* since we have seen that, in many cases of advanced disease, all the great eliminating organs are congested and inadequate to the extra stress, so that the vessels are kept preternaturally full.

In this stage, digitalis, at times, that true opiate of the heart, as Bouillaud terms it, and often so indispensable in Mitral valve disease, both for the purpose of equalizing the heart's contractions and, for prolonging the period of diastole, to allow sufficient blood gradually to flow into the left chamber for the requirements of the general system, is, in Aortic Insufficiency, most dangerous; inasmuch as to prolong diastole, is to favour the worst result of Aortic Insufficiency—Dilatation of the systemic Ventricle. In the third, or Degenerative stage, it has been already seen how the characteristic throbbing of the heart and arteries gradually lessens. We would now gladly restore this apparently over-action of the left heart, which formed the great barrier to the oppression and effusions that now threaten. But it is precisely at this juncture,—amid all these accidents,—where remedies are most wanted, they are least to be found. It is true, that if life were imperilled by the imminence of Cardiac Syncope, we might, perchance, if present, successfully interfere by the recumbent position; the use of external and internal excitants, and, beyond all, by the sudden application of cold to the region of the heart. But under the more ordinary circumstances, however, the remedies in use, as iron, zinc, or strychnine, must fail, for they cannot rechange fibroid, or fatty tissue, into healthy muscle. Science knows no agent that can restore vital contractility to a structure which has lost, from disease, its originally inherent power; more especially to one so immediately essential to the maintenance of life, as that which constructs the left Ventricle of the heart.

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