

ART. XV.—*Contributions to the Pathology of the Heart.* By

BENJAMIN GEORGE M'DOWEL, A.B., M.D., one of the Physicians to the Whitworth and Hardwicke Hospital, Lecturer on Anatomy and Physiology in the Carmichael (formerly the Richmond Hospital) School of Anatomy, Medicine, and Surgery, &c. &c.

THE following observations are derived from an analysis of numerous cases of cardiac disease treated by the writer in hospital practice during the last few years, and which were carefully recorded as they came under observation.

It is not the object of this communication to enter upon the subject of heart disease in general, but rather to direct attention to the records of some cases which present in themselves peculiar features, and to allude to such general matters only, relating to the pathology and diagnosis of cardiac disease, as the writer feels he is enabled to do by the materials at his disposal.

Were the differential diagnosis and pathology of cardiac diseases clearly established, such a communication as the present might well be deemed superfluous, but as our knowledge of this class of diseases is comparatively recent and as yet imperfect, it seems desirable, as opportunities occur, to advance that knowledge by the results of clinical observation.

Organic diseases of the heart have been considered under two heads, as they engage:—1. Its valvular structures; and 2. Its muscular tissue. This classification has been found no less useful in practice than convenient in the study of morbid appearances. True it is that lesions of both structures often co-exist, or even spring up together; but, on the other hand, in many instances they exist separately, or even where combined, they not unfrequently hold to each other the relation of cause and effect.

The valvular diseases of the heart have received by far the largest share of attention, and hence they are by many too ex-

clusively regarded as the sole cause, both of the symptoms and of the fatal result. Yet the morbid changes which affect the muscular tissue of the heart are no less deserving of our attention, as they assist us to explain the laws which regulate the fatality of heart diseases, as well as to understand better the differential characters of these affections.

At the present day it is comparatively easy to distinguish the several valvular diseases from each other. The chief difficulty seems to be in establishing the differential diagnosis in certain cases, between organic and non-organic diseases; and again (in cases of organic disease), in discriminating those in which there is valvular lesion from others, in which with healthy valves the muscular tissue and cavities of the heart have undergone morbid changes. To illustrate some of the structural changes which belong to this latter class is the principal object of the present communication.

SECTION I.—THE EFFECTS OF EXCESSIVE DILATATION OF THE HEART, ESPECIALLY OF THE VENTRICLES, WITH SOFTENING OF ITS TISSUE, AS EXEMPLIFIED BY CASES WHERE THIS MORBID CHANGE EXISTED, UNCOMPLICATED WITH VALVULAR DISEASE.

CASE I.—*Passive Dilatation of the Ventricles. Enlargement of the Heart and softening of its muscular tissue. No Valvular Disease. Signs of obstructed Circulation.*

Mary Byrne, aged 33, was admitted into the Whitworth Hospital under my care, October 12, 1846. She states that she has laboured under some of the symptoms of disease of the heart for the last fifteen months: palpitation was first complained of. Five weeks later she had an attack of hemoptysis which continued for two days. Six weeks prior to her admission into hospital, dropsical effusions became developed: her face and ankles first swelled, and subsequently the abdomen. She never at any time laboured under rheumatism. On her admission general anasarca and ascites, to a slight extent, were present; extreme distention of the legs by serous effusion ren-

dered her very helpless; while a frequent harassing cough, with scanty expectoration, increased much her sufferings; there was extreme dyspnœa occurring in paroxysms, and amounting to orthopnœa; the face was bloated, and so darkly congested as to be almost livid; the superficial veins of the neck were turgid; whilst the extremities were constantly cold and mottled. The urine was small in quantity and high-coloured, specific gravity 1016, not containing albumen. The digestive organs were much deranged

The chest generally yielded a dull sound, owing to anasarca of its parietes; cardiac region extensively dull; heart's impulse weak as contrasted with the apparent amount of cardiac enlargement; heart's action extremely irregular; second cardiac sound normal and distinct, whilst the first sound was accompanied or replaced by an evident murmur, heard most loudly over the heart's apex, but lost along the sternum. No *frémissement*. Radial pulse extremely small, weak, and intermittent, 108 in the minute. The signs of universal bronchitis with pulmonary congestion were present.

No treatment could be expected to have much influence under such unfavourable circumstances. Diuretics, purgatives, and diffusible stimulants, were employed with some benefit; subsequently blue pill in minute doses, with digitalis and squill was prescribed. On the 21st it was reported that much relief had been experienced, but all improvement ceased here, and her most unfavourable symptoms became more developed. With increasing dropsy there was increased dyspnœa, and death was impending from apnœa, when coma suddenly set in, and she died November 4th.

Post-mortem Examination.—The lungs were gorged with blood, but were not structurally altered; the cavity of each pleura contained about eight ounces of straw-coloured serum; the pericardial cavity half-an-ounce only. The heart was greatly enlarged; when washed out it weighed thirteen ounces; the left ventricle was extremely dilated; its walls were not

hypertrophied, and therefore bore no just proportion to the size of the cavity (dilatation was the predominant change, and hence the size and weight of the heart were not duly proportional, as they must be when hypertrophy predominates). The carnae columnæ of the mitral valve were considerably enlarged; the mitral valve itself was healthy, except a small cartilaginous nodule, not larger than a grain of small shot, on its anterior flap; the left auriculo-ventricular opening was somewhat dilated, and consequently the normal relation as to size between the opening and its valve no longer existed; the left auricle also was more capacious than usual; the sigmoid valves of the aorta, the lining membrane of that vessel, and the valves of the right side of the heart, were perfectly healthy. The same changes were found in the right cavities, but in a much slighter degree. The substance of the heart itself was altered in appearance and consistence; its fibres were paler than usual, being of a yellowish-brown colour, whilst their cohesion was diminished; the muscular tissue was manifestly softened. The liver and kidneys were engorged, but were otherwise healthy^a.

CASE II.—*Passive Dilatation of the Ventricles. Dilatation of the Left Auriculo-ventricular Opening. Enlargement and Softening of the Heart. No Valvular Disease. Signs of Obstructed Circulation.*

Isabella St. John, aged 63, admitted into the Whitworth Hospital under my care, December 11, 1847. On admission, in this case as in the preceding one, the symptoms were essentially those which are usually attributed to obstructive valvular disease. In addition to palpitations, distressing dyspnœa, and breathlessness,—the usual accompaniment of confirmed cardiac disease,—lividity of the face, remarkable turgescence of the jugular veins, and anasarca to an extreme degree, were present, and evi-

^a For the report of this case I am indebted to Mr. Robert Cryan, who was my clinical clerk in 1846.

dently pointed to an obstructed circulation. The symptoms of cardiac disease had first appeared a year previously, and since then had gradually become more developed. Dropsy had been but lately superadded; she had never had rheumatism in any form; hemoptysis had occurred on more than one occasion. The cardiac signs consisted in considerable increase of the natural amount of dulness, a *bruit* at the apex with the first sound, a faint impulse, and an irregularly acting heart. The radial pulse was equally intermittent with the heart, and extremely weak.

It is unnecessary to follow this case through all its details from day to day. The distressing symptoms which have been enumerated admitted of no benefit from treatment. The dyspnoea increased, whilst the limbs continued to enlarge, until at last they became enormously distended. The circulation then grew more languid, and livid vesicles appeared on the legs as the forerunners of gangrene, when death put a period to her sufferings.

Post-mortem Examination, January 26, 1848.—The body was of great size from serous infiltration, as also from an excessive deposit of fat. The heart was large, flabby, and softened. Its left cavities were very much enlarged, without any increase in the thickness of their walls. The left auriculo-ventricular opening was remarkably dilated, and allowed five fingers to pass with ease. The venous system was universally gorged with blood, which was yet fluid, but presented no traces of any admixture with oil. The valves, without exception, were perfectly healthy.

In the two cases which have now been detailed, the cardiac lesions were essentially the same. In both there was an enlarged, softened, and dilated heart, with ventricles so much expanded, that their cavities had become disproportioned to their propelling power. These changes occurred independently of any valvular disease; for in Case 1., the small spot of thickening, no larger than a grain of shot, cannot be considered to have inter-

fered with the functions of the mitral valve. The left auriculo-ventricular opening in both instances was dilated, so that although the mitral valve was perfect in point of structure, it was, nevertheless, quite inadequate to guard an orifice, dilated, as in Case II., to at least twice its usual dimensions. In both cases consequently there was regurgitation through the left auriculo-ventricular opening,—of which the *bruit* heard over the apex of the heart was significant,—and a languid systemic, together with an obstructed pulmonary circulation. Hence, in obedience to a well-known law, that morbid changes in the heart are propagated in a direction contrary to the course of the circulation, there resulted, engorgement of the lungs, enlargement of the left cavities of the heart, venous obstruction, and plethora, and lastly, general dropsy. As hepatic obstruction was but a part of the obstruction of the general circulation, we find that ascites occurred subsequently to anasarca, and existed to a comparatively slight degree. The prominent symptoms in these two cases were in all material points the same: they were also almost identical with those which characterize a contracted condition of the left auriculo-ventricular opening.

The following arrangement enables us to compare advantageously the prominent symptoms observed in Cases I. and II., with those which denote left auriculo-ventricular contraction:

<i>Contraction of the left auriculo-ventricular opening.</i>	<i>Passive dilatation of the ventricles; softening of the Heart.</i>
--	--

—CASES I. and II.

An irregularly acting heart.	An irregularly acting heart.
An intermittent, feeble, and rapid pulse, not synchronous with the heart's impulse.	An intermittent, feeble, and rapid pulse.
Dyspnœa, hæmoptysis, and pulmonary congestion.	Dyspnœa, hæmoptysis, and pulmonary congestion.

Jugular pulsation and turgescence. Jugular turgescence.

Cerebral symptoms.

Death by coma in Case I.

General dropsy.

General dropsy.

Jaundice (occasionally).

Jaundice (not observed).

It is to be observed, that "the want of correspondence" between the heart's action and the radial pulse, a symptom so much insisted on by Mr. Adams in contraction of the left auriculo-ventricular opening, did not exist in the cases of dilated ventricles, whilst the strong impulse of the heart in the former affection contrasts remarkably with the feebleness of its action in the latter.

Were the first-mentioned sign (want of synchronism between the impulse of the heart and the radial pulse) an *invariable*, and therefore a pathognomonic sign of a contracted mitral opening, the elements for establishing a differential diagnosis might be therein found, but it has occurred to the writer to meet with several cases in which this symptom was wholly wanting, and yet mitral contraction was proved to have existed on examination after death^a. Probably it may be a constant

^a A brief abstract is here given of three cases in which the pulse was regular, and synchronous with the heart's impulse, whilst, on examination after death, mitral narrowing was proved to have existed.

I.—Celia Long, aged 80, a patient of mine in the Whitworth Hospital, died January 28th, 1848. Cardiac disease supervened on rheumatism. Symptoms during life:—A very small and rapid but regular pulse; disproportionately strong action of the heart; bronchitis; dropsy.

Autopsy.—Left auriculo-ventricular opening so contracted as to admit the point of the little finger only; mitral valve opaque and rigid; left auricle greatly dilated and hypertrophied; aortic orifice contracted likewise.

II.—John Casey, aged 19. Whitworth Hospital. Died January 25th, 1850. Never had rheumatism. Symptoms during life:—Dyspnoea; bronchitis; hemoptysis; pulse small but regular; wild delirium occurred suddenly on the 24th, followed by profuse hemoptysis, which continued for twenty-four hours, when death occurred.

Autopsy.—Mitral valve rigid from bony deposits, opening contracted, and with the valve representing a bony thimble, terminating inferiorly in a narrow chink,

symptom in the advanced stages of the disease, when the narrowing has become so great that the ventricle sometimes contracts on an almost empty cavity.

The disproportion between the force of the heart's action and that of the radial pulse is a more valuable, because it is a more constant, indication of contracted mitral orifice. But, apart from other symptoms, this cannot be regarded as a diagnostic sign, since it may be present under other and different morbid conditions.

The difficulty of distinguishing between these affections will be further increased when it is considered that the other symptoms which have been in the table assigned to contraction of the mitral orifice are not necessarily connected with this condition exclusively. For example, hœmoptysis may depend on passive dilatation without softening, as in Case v., or on aortic patency and dilatation, as in Cases vi. and vii. Jugular turgescence indicates engorgement of the right side of the heart; jugular pulsation, regurgitation through the right auriculo-ventricular opening; and these phenomena denote mitral contraction only, inasmuch as that lesion generally produces those changes in the right cavities of the heart on which the symptoms in question depend. Cerebral symptoms have been very generally referred to a contracted condition of the left auriculo-ventricular opening. No doubt apoplectic seizures do often occur in the course of this disease^a, and not unfrequently

though admitting the point of the finger superiorly; the left ventricle was the only part of the heart which retained its natural appearance; the other cavities were all very much dilated; the walls of the left auricle were immensely thickened. The lungs were gorged with fluid blood.

III.—Jane Carroll, aged 16. Whitworth Hospital. September 16th, 1846. Had acute rheumatism one year previously. Symptoms:—Anasarca; bronchitis; frequent hœmoptysis; livid lips; pulse 120, very small but regular; contraction of mitral valve diagnosed. Six months afterwards she died in Dr. Banks' ward, and on examination a contracted condition of the mitral opening was found to exist.

^a *Vide* Mr. Adams "on Diseases of the Heart," *Dublin Hospital Reports*, vol. iv. p. 416; and Cases i. and ii.

prove the immediate cause of the fatal result. But, at the same time, cerebral lesions may take place in any morbid condition of the heart which enfeebles the general or systemic circulation. The apoplectic death of Case I. has already illustrated this point; and in Cases IV. and VIII., to be hereafter detailed, cerebral symptoms will be found associated with dilated cavities and with mitral patency*. Neither does jaundice essentially belong to mitral contraction: in Case V. it will be found associated with aortic patency; and besides, contrary, perhaps, to what might *a priori* be expected, jaundice so rarely depends on heart disease, that its occasional development can scarcely be considered of much weight as an element of diagnosis.

If these remarks have conveyed the impression that the symptoms of passive dilatation with softening of the heart may readily be confounded with those of contraction of the mitral orifice,—the signs furnished by auscultation and percussion in these two classes of cases will scarcely clear up the difficulty. These signs are very similar in both classes. In Cases I. and II. there was detected by percussion an enlarged heart, whilst by auscultation a bellows murmur was revealed over the region of the apex, systolic as to the time of its occurrence, and single. Similar are the phenomena which are generally met with in practice, or described by authors, as associated with contraction of the left auriculo-ventricular opening.

It is stated by some authorities, that in mitral contraction a double *bruit* may be heard, one murmur being produced by the obstacle in the way of the blood entering the ventricle, the other by its subsequent regurgitation. The writer has never verified this observation, although he has examined a great many cases with reference to this particular point. It has oc-

* *Vide* Dr. Law's Observations on Disease of the Brain dependent on Disease of the Heart, Dublin Journal, vol. xvii. 1840.

curred to him, and not unfrequently, to meet with cases in which a double *bruit* was audible at the apex of the heart; but in all of them he could satisfy himself that one of the murmurs, at least, was propagated thither from some other locality.

Whilst the physical signs just enumerated are those which usually indicate mitral contraction, it must be clearly understood that their absence does not necessarily indicate valvular soundness. In many cases of mitral contraction no abnormal sound whatever indicates the lesion, and the diagnosis must then rest on general symptoms. The presence or absence of *bruit* does not appear to the writer to depend on the extent to which mitral contraction has advanced, as do the other symptoms. The *bruit* is chiefly due to regurgitation from the left ventricle into the left auricle. And as contraction of the orifice may occur without any impairment of the functions of the mitral valve, there may be obstruction and no regurgitation. In such cases there will consequently be no *bruit*. This was observed in another case^a.

Mitral narrowing, it would thus appear, may occur under at least two forms. In one, the morbid change affects the orifice chiefly, which is then reduced more or less in size, until it represents a "semilunar fissure," with the concavity turned towards the aorta^b. In the other, the valve is likewise engaged, and becomes puckered, thickened, and shortened, whilst the opening is contracted; and not unfrequently both opening and

^a As this case will not appear in the present paper, the following brief abstract of the morbid appearances is here introduced, in order to preserve the connexion of the subject.

Catherine Leonard, aged 40. Contraction of the left auriculo-ventricular opening. Admitted November 5th, 1851.

Post-mortem Examination, November 21st. — Heart much enlarged; right cavities greatly increased in size; in the left chambers hypertrophy predominated; the left auriculo-ventricular opening was considerably diminished in size; *but the leaves of the mitral valve were unaltered, and consequently quite capable of performing their functions.*

^b *Vide* Mr. Adams' truthful description, *loc. cit.* p. 433.

valve are converted into an appearance like that of a bony thimble, the point projecting into the ventricle, and presenting a small, unyielding aperture, which not only obstructs the entrance of the blood, but allows of its partial regurgitation. But another circumstance may influence the existence or absence of *bruit* in mitral narrowing, viz., the amount of regurgitation. In those cases of valvular imperfection in which a considerable amount of fluid is transmitted with force through an altered opening, the murmur which results is always well developed, as, for example, the diastolic murmur of aortic patency, and the prolonged systolic murmur which accompanies mitral inadequacy. Where contraction of the left auriculo-ventricular opening is established, the tendency of the disease is to diminish the amount of blood entering the left ventricle, and in an equal degree the amount and force of its regurgitation into the left auricle (where the lesion is such as to admit of regurgitation). Now, this cause operates more and more as the narrowing goes on, until finally, when the opening is so much diminished that the ventricle can only get a "precarious supply of blood" (Adams) the amount of regurgitation must be extremely small, or, perhaps, only occasional. In accordance with these views, it is in the advanced stage of mitral contraction that a *bruit* is most frequently wanting^a.

It would appear, therefore, from the details of these Cases (I. and II.) that passive dilatation of the ventricles of the heart (with which dilatation of the left auriculo-ventricular opening is generally associated), and softening of their muscular walls, will be attended with a group of symptoms and of physical signs which are almost identical with those which usually de-

^a Mr. O'Ferrall has clearly shown that the progress of mitral contraction may cause the disappearance of an originally well-developed murmur. He explains this occurrence by supposing that the shortened valve becomes again adequate to its task in consequence of progressive contraction.—*Vide* Mr. O'Ferrall's "Clinical Researches on Diseases of the Heart," Dublin Journal, First Series, vol. xxiii. 1843.

note a contracted condition of the left auriculo-ventricular opening.

SECTION II.—PASSIVE DILATATION AND ENLARGEMENT OF THE HEART, DEPENDING ON DISEASE OF THE COATS OF THE AORTA, AND PRODUCING THE SIGNS OF AN OBSTRUCTED CIRCULATION.

CASE III.—*Extreme Dilatation and Enlargement of the Heart. Atheromatous Degeneration of the Aorta. No Valvular Disease. Signs of obstructive Heart Disease.*

Timothy Houston, aged 61, was admitted into the Whitworth Hospital, under my care, January 6, 1852. He had laboured for several years under the symptoms of heart disease, but dropsical symptoms had only lately appeared. A year previously he had incomplete paralysis of the left arm and leg, for which he was in the Whitworth Hospital, in Dr. Gordon's ward, and which was removed by treatment. Increasing dyspnœa and anasarca were of late the symptoms of which he chiefly complained.

On admission he was universally dropsical. Ascites existed to a slight degree only. The lungs were congested, and cough was very troublesome. Breathlessness and dyspnœa were constantly present, but at times there were paroxysms of difficult breathing, with palpitations, which rendered the horizontal position insupportable.

The pulse was small in volume, and regular. The heart was manifestly enlarged, and that to a considerable degree. Its impulse was quite out of proportion to the smallness of the pulse. The sounds of the heart had a muffled character, and were difficult to analyze; but no abnormal *bruit* could at any time be detected. The jugular veins were turgid. The face was invariably pale.

Soon after his admission this patient was attacked with hemoptysis. The blood was florid, and was expectorated in considerable quantity, mixed with thin, frothy mucus. The

signs which indicated pulmonary apoplexy were these :—A sharp, crepitating *râle* over the lower lobes of both lungs posteriorly, with absolute dulness on percussion, but without the bronchophony or bronchophonia which would accompany the same degree of loss of sonoriety, in a case of pneumonic consolidation. Moderate venesection, at this crisis, was well borne, and gave great relief. The pulmonary hemorrhage continued for a fortnight, and then gradually ceased. But whilst the crepitus disappeared, the healthy resonance of the chest was not restored.

From this period the paroxysmal attacks of dyspnœa became more periodic, and assumed all the features of distressing angina. This painful complication continued to the close of life. The lower extremities had, by this time, become so distended, that a few punctures were made with a small needle: a copious flow of serum was the result, by which the anasarcaous swellings were greatly diminished. Towards the close of life there was less suffering than might have been anticipated, for a drowsy condition became manifested, accompanied by decided imbecility of mind. He now almost ceased to suffer, and dosed away nearly all his time, either in an easy chair, or propped up sitting in bed. It was surprising how long life was protracted in this condition. For upwards of three weeks this state of stupor continued, when complete coma occurred, followed, after a few days, by death.

Post-mortem Examination, May 1st.—The lungs were rather small in bulk, although they were emphysematous. Their lower lobes had been the seat of pulmonary apoplexy. On incising them, several large, dark-coloured masses appeared, which were evidently old coagula. The intervening portions of pulmonary tissue were somewhat condensed, whilst the remainder of the lungs was essentially healthy.

The heart was very much enlarged: it extended greatly beyond its normal limits. When washed out, and separated from all its connexions, it still weighed twenty-eight ounces.

In form it was not much altered, except that its apex was more rounded than usual. There was hypertrophy, with excessive enlargement of the ventricles, especially of the left. The muscular tissue had its natural firmness. The valves were perfectly free from disease, and the most rigid examination failed to detect the slightest imperfection in them*. The carnae columnæ were not hypertrophied (as they are generally observed to be in cases where regurgitation has been allowed). The aorta, throughout almost its entire extent, was brittle, from extensive atheromatous depositions, and its elasticity, from this cause, was sensibly diminished. There were no evidences of recent inflammation of the aorta.

CASE IV.—*Extreme enlargement of the Heart. Dilatation of the Ventricles. Signs of obstructed circulation. No Valvular disease, but extensive atheromatous degeneration of the Aorta.*

Very similar to the preceding was the case of a man named John Clarke, aged 60, who was under observation for a short time in the latter end of August, 1851. He was admitted into the Whitworth Hospital labouring under ascites and anasarca, accompanied by extreme difficulty of breathing. His other symptoms were as follows:—His face was bloated and

* The valves were severally tested as to their adequacy in the following manner:—The pipe from a water cistern was introduced into the aorta and secured to it by a ligature. On allowing a full stream of water to flow into the artery, it was found that none of the fluid passed the sigmoid valves. Their competency to prevent regurgitation was therefore manifest. To test the mitral valve the aortic sigmoid flaps were broken down, and the aorta secured on the pipe as before; the water was now allowed to rush into the left ventricle, but though the mitral flaps were floated up, and even made to bulge upwards, owing to the pressure to which they were subjected, yet none of the fluid passed into the left auricle. The mitral valve was thus shown to be perfect. The integrity of the valves on the right side was similarly tested, and with the same result.

This method, which, with reference to the aortic valves, the writer first saw adopted by Dr. Corrigan, is so easy of application that it may be employed whenever it is desirable to determine with certainty the adequacy of the valvular apparatus of the heart.

congested; there was much dyspnœa, with the signs of œdema of the lungs; the pulse was small, weak, but regular. The emphysematous condition of the lungs interfered somewhat with auscultation of the heart, but it was satisfactorily ascertained that, though the impulse of the heart was strong, yet no abnormal sound existed. The urine was highly albuminous, with a diminished specific gravity. He died soon after coming under observation.

Post-mortem Examination.—Some clear serum was found in the pleural cavities; the lungs were congested and emphysematous; the heart was enormously enlarged; externally it presented the appearances which are generally impressed on the organ by aortic patency; there were the same elongation and increased development of the left ventricle. Impressed with this idea, the aortic valves were tested in the manner described in the last case, but no regurgitation was allowed. On opening the heart, not only the aortic, but all the valves, were found to be perfectly normal. The enlargement of the heart was chiefly due to dilatation with hypertrophy. The left ventricle was principally affected. The walls of this cavity were much thicker than natural, but the size of the cavity was out of proportion even to the thickened walls; dilatation, therefore, predominated. The right ventricle presented the same changes, but in a slighter degree. The tissue of the heart was of healthy consistence. The aorta was extensively diseased; its interior presented almost universally the yellow discoloration produced by atheromatous depositions in the sub-serous tissue. In some places the inner coat was replaced by large patches of bone. In this case, as in the former one, fat was deposited in considerable quantities, not only around internal organs, but also in the subcutaneous tissue. The kidneys were greatly enlarged, and smooth externally, whilst a section exhibited the "fatty degeneration" in an advanced stage of development.

These cases very clearly illustrate the influence of dilata-

tion of the heart as a direct source of obstruction to the circulation, since in both of them the signs of obstruction were very fully developed, and dilatation of the ventricles was the only lesion found.

Cases I. and II. were less conclusive on this point, as in them there was softening in addition to dilatation.

Dilatation of any of the cavities of the heart implies that the amount of pressure exercised on the inner surface of such cavity must have been relatively greater than the power of resistance of its walls. In Cases I. and II., independently of any obstruction to the circulation, the muscular tissue was softened; the muscular power was consequently defective; the pressure from within, although not more than normal, overcame the resistance from without, and dilatation was the result. Many doubt that any considerable degree of enlargement of the heart, can occur independently of valvular disease or of pulmonary obstruction. In the four cases which have now been detailed, however, it existed to a very marked degree, and in the form of dilatation with hypertrophy. In Cases I. and II., as has been already stated, the enlargement of the heart depended on softening of its tissue. In Cases III. and IV. no cause for enlargement was found within the heart itself, but extensive disease existed in the tunics of the aorta, which, it is more than probable, is a sufficient cause for cardiac hypertrophy in general.

How far this condition is to be recognised as capable of producing in all cases the fatal train of morbid changes which has just been described, the writer is unable to affirm, but as it seems almost certain that a direct connexion existed between the pathological conditions and the symptoms recorded in Cases III. and IV., a few observations on the subject may not be inappropriate.

The elasticity of the larger arterial trunks is, by modern physiologists at least, acknowledged to be an efficient auxiliary in carrying on the circulation of the blood. "The elastic arterial wall, stretched by the contraction of the heart, re-acts with

a power which approximates more closely to that by which it was dilated, according as the arterial tissue is more or less elastic. The arteries are thus made to contract upon their contained blood, and to drive it onwards, or from the heart, and backwards, or to the heart. Its course in the latter direction is speedily checked by the sudden and forcible closure of the aortic valves under the pressure of the regurgitating current. Therefore, the great mass of blood rushes onwards towards the capillary system, propelled first by the heart's impulse; and, secondly, by the elastic re-action of the arterial walls"^a.

If the elasticity of the aorta which is here stated to be effective in moving the blood be much impaired, as occurred in Cases III. and IV., we may suppose that the heart is called upon to make unwonted exertions to overcome the *vis inertiae* of the fluid lying in that vessel. An impediment to the circulation will thus be established, and hypertrophy and enlargement of the heart will result. As the obstruction from the cause assigned becomes greater, the muscular parietes become proportionally thicker. But at last hypertrophy finds its limit, whilst enlargement of the cavities is still progressive. Thus dilatation predominates, and the symptoms of obstructed circulation become fully developed. For when once established, excessive dilatation becomes a direct cause of obstruction. Though the dilated cavities contain much blood, they are able to receive or to expel but little, the heart becomes embarrassed and oppressed, especially when the circulation is hurried; it beats with increased rapidity to compensate for diminished power, and hence arise palpitations with irregularity of action, dyspnoea, hemoptysis, œdema and congestion of the lungs, dropsy, anasarca, and perhaps an interruption of the functions of the brain.

Thus it is, that an excessively dilated heart, by its inefficiency to unload its chambers, produces symptoms similar to those which are the result of obstructive valvular disease.

^a Todd and Bowman's *Physiological Anatomy*, Part iv. p. 351.

SECTION III.—INFLUENCE OF DILATATION OF THE CHAMBERS OF THE HEART AS A COMPLICATION OF VALVULAR DISEASE.

Since dilatation of the ventricles uncomplicated by any other cardiac lesion may produce the fatal results just mentioned, it will be readily admitted that dilatation may prove a most serious complication in cases of pre-existing valvular disease.

Much variety is observed in the duration of different cases of valvular disease. Some run on to a fatal termination in a very short space of time; others, on the contrary, live for many years, suffering, no doubt, from various distressing symptoms, yet at intervals in the enjoyment of tolerably good health. The difference in the amount of valvular disease in any two cases will rarely suffice to explain this circumstance; for in the case soonest fatal, there may have been the least amount of valvular derangement. The accidental lesions to which the subjects of organic heart disease are so liable, such as pneumonia, bronchitis, or fresh endocardial inflammation, will, no doubt, account for the fatal issue at an early period in many instances. But the great difference in the duration of life in all cases will be more obviously and more easily explained, if it shall appear that, in all organic diseases of the valves or apertures of the heart, the urgency of the symptoms is not so much proportional to the mechanical derangement, as to the changes produced thereby in the capacity or the muscular development of the several cavities.

Again, we are familiar with the fact, that an identity of symptoms is often met with in the advanced stages of different valvular diseases. This apparent anomaly would be easily explained if it should appear that there is a liability for all forms of valvular disease to be ultimately complicated with the same structural changes of the muscular tissue.

Hypertrophy of the muscular parietes of the cavities of the heart is the inevitable result of mechanical obstruction, and owing to it, in such cases, a more equable circulation is main-

tained than could otherwise be hoped for. But with this there is also a tendency to enlargement of cavities, which, so long as it is accompanied by a proportional amount of hypertrophy, may not be productive of inconvenience. As soon, however, as the increased bulk of the muscular wall ceases to be proportional to the increased capacity of the cavity, whenever in fact dilatation is predominant, the cavity enlarging, as it were, at the expense of its walls, then a great aggravation of symptoms is the result. Where the original disease has been valvular inadequacy, the signs of obstruction are superadded, and where the primary lesion has been essentially an obstructive one, the symptoms depending on obstruction all become exasperated.

The following Cases are illustrative of these remarks:

CASE V.—*Aortic regurgitant Disease. Excessive Dilatation, with Hypertrophy of the Left Ventricle. Mitral Valve healthy. Signs of obstructed Circulation. Pulmonary Apoplexy, &c.*

Margaret Foy, aged 42, a servant, was admitted into the Whitworth Hospital under my care, February 27, 1850. She had acute rheumatism four years ago, which affected all her joints, and from which she suffered for seven weeks. Last Christmas she caught cold, and then first suffered from palpitation of the heart, and from difficulty of breathing, with severe cough. These symptoms becoming more aggravated, she was compelled to apply for hospital relief.

On admission she had much cough, and the breathlessness of cardiac disease. The least exertion brought on violent palpitation. There was dyspnoea, which was intermittent, usually coming on at night, after she had slept for a little. Sometimes the paroxysm would last all night. During its continuance she was invariably obliged to sit up in bed.

On examining the chest, the heart's action was felt over a large extent of surface, which, with increase of dulness, indicated considerable enlargement of the organ. The usual signs of aortic patency were fully developed. The larger arterial

trunks throbbed violently, and their course was distinctly visible as each vessel was thrown up out of its bed at every beat of the heart. The pulse was jerking and large. A distinct double bellows murmur was heard along the sternal region, but its greatest intensity was over the valves of the aorta, and opposite to where that vessel makes its first curve. The diastolic portion of the murmur was the most intense. Aortic regurgitant disease, with considerable enlargement of the heart, was diagnosed.

Leeches were applied from time to time over the region of the heart, and on one occasion six ounces of blood were abstracted from the arm with marked relief. Morphia and the tincture of lobelia, given at bed-time, materially diminished the severity of the paroxysms of dyspnœa. Ten days afterwards pneumonia attacked the apex of the right lung; it was subdued by appropriate treatment, but from this period the paroxysms of dyspnœa recurred more frequently and with greater urgency. They now assumed much of the characters of angina.

March 15th. Jaundice appeared, and the sputa were tinged with blood. The feet now became anasarious. Diuretics and diffusible stimulants were directed. For the next fortnight the symptoms underwent but little change, the attacks of angina were occasionally very severe, but the lobelia seldom failed to procure relief. The anasarca increased.

29th. A severe paroxysm of dyspnœa occurred this morning, accompanied by profuse hemoptysis. Free cupping to the chest; diffusible stimulants.

30th. The hemorrhage continues, but the dyspnœa is less urgent.

April 2nd. Dyspnœa of the most urgent kind is present. The face is pale, and the pulse failing. No treatment now afforded even the slightest relief. She continued to suffer intensely, and died after a painful struggle on the morning of the 3rd.

Post-mortem Examination.—Heart greatly enlarged; eccen-

tric hypertrophy of the left ventricle. The capacity of this chamber was enormously increased; the chambers of the right side were also enlarged, and the right auriculo-ventricular opening dilated. The aorta was dilated at and above its origin, its lining membrane was opaque, yellowish, and roughened by fibrinous deposits. The sigmoid valves were of normal size, but thickened, and inadequate to close the mouth of the dilated vessel, as was proved by experiment. The mitral valve was perfect, but the serous membrane which covered it, as well as the endocardial lining of the left auricle, was of a yellowish colour. The lungs presented a remarkable specimen of pulmonary apoplexy. Cirrhosis of the liver and granular degeneration of the kidney likewise existed.

CASE VI.—*Aortic Patency. Aortitis. Dilatation of the left Chambers of the Heart, and of the left Auriculo-Ventricular Opening. Signs of Obstruction.*

Michael Connor, aged 30, a porter, was admitted into the Whitworth Hospital, under my care, December 12th, 1850. Three years previously this patient laboured under rheumatic fever, for which he had been treated by Dr. Corrigan. He recovered perfectly, and remained well until the month of May (seven months before admission), when the first symptoms of heart disease were observed. On admission palpitation of the heart, and breathlessness, were present, whenever he exerted himself even slightly; in addition to which he complained of dyspnœa at night, which used to occur suddenly during sleep, and was paroxysmal. His feet were anasarcous.

The following physical signs existed at this period. There were extensive cardiac dulness, and a double bellows murmur over the base of the heart and along the sternum. Over the apex of the heart, which had passed considerably towards the left side, a single loud systolic *bruit* was evident.

In a fortnight this patient left the hospital much relieved. He was re-admitted January 4th, with all his former dis-

troubling symptoms much aggravated. The dyspnoea was most urgent, and the fits of angina at night threatened, at each paroxysm, to terminate his existence. Mr. Hill, the clinical clerk, reporting his condition in one of these seizures, stated, that he found him "labouring under the most intense dyspnoea, which had come on with extreme suddenness; bathed in perspiration; and breathing with the utmost difficulty. He was supported in a sitting posture, with his shoulders raised. The *alæ nasi* were dilated; the lips blue; the pulse weak, small, and compressible. He complained of excessive pain in the sternal region, and of coldness of his feet and legs."

These paroxysms generally lasted each for three or four hours. The dropsical symptoms had increased, and the signs of pulmonary congestion were very evident.

January 7th.—Breathing very difficult; hands and feet cold; the pulse 132, weak and thready. Pressure on the epigastrium could not be borne. The lower extremities were much swollen, and fluctuation was distinct in the abdomen; the physical signs, as before enumerated, were unaltered. He died two days afterwards.

Post-mortem Examination.—The heart was greatly enlarged; the right cavities were simply dilated. The left presented dilatation with hypertrophy,—but dilatation was the predominant change. Tissue of the heart much softened, so as to be readily torn or broken down. Left auriculo-ventricular opening much dilated. The left auricle, as has been stated, was very capacious, besides which its lining membrane presented a well-marked yellow colour, such as generally indicates that regurgitation has been freely allowed. The mitral valve was healthy. The aortic valves were thickened and shortened, so as to have been altogether incapable of meeting. Aortitis existed to an extraordinary degree; the lining membrane of the aorta was swollen, and presented a villous appearance, with the colour of scarlet cloth; it was likewise soft and pulpy to the touch.

In the lungs blood was found freely extravasated, and the

vessels were yet greatly gorged. The liver presented the "nutmeg" appearance.

CASE VII.—*Aortic Patency. Dilatation of the Left Chambers of the Heart, and of the Left Auriculo-Ventricular Opening. Signs of Obstruction. Pulmonary Apoplexy, &c.*

John King, aged 30, was admitted into the Whitworth hospital, under my care, June 13, 1852. He had been complaining for five or six months of vague feelings of ill health, but had no symptoms to lead him to imagine that he had heart disease, until very lately, when his feet became swelled, and his breathing difficult.

On admission there were present general anasarca, and slight ascites; the face was bloated, and the conjunctivæ injected. The breathing was difficult. There was general bronchitis, with congestion of the lower lobes of the lungs. The liver also was enlarged, and projected three fingers' breadth below the ribs. The pulse was thrilling, full but soft; much softer than in cases of uncomplicated aortic patency. Visible pulsation of the larger arteries was observed, and turgescence of the jugular veins.

The heart beat over a large surface, but its impulse was not immoderate. The cardiac region was extremely dull, and a loud double *bruit*—a *bruit de scie*—heard over a large surface, but with greatest intensity over the sternum, indicated aortic regurgitation. The urine was scanty, and not albuminous.

Local depletion, diuretics, and diuretic purgatives were prescribed with much advantage, but the dropsical symptoms could not be wholly got rid of. No material change otherwise occurred until July 20, when he began to spit up blood. A frightful paroxysm of dyspnœa occurred that night, which obliged him to stand for hours at an open window. Next day he was greatly sunk, and partially jaundiced. The extremities were cold and greatly swollen. The liver was tumid and tender. Hemoptysis continued.

21st. The dyspnœa during the night was extreme, and

occurred in paroxysms. This morning he was quite unable to lie down. He sank rapidly, and died that night during a violent convulsion.

Post-mortem Examination.—The heart was greatly enlarged; weight twenty-five ounces; the aorta was dilated; the sigmoid valves thickened, and their pliancy diminished; but their inadequacy was mainly owing to dilatation of the vessel. There were patches of lymph in the left auricle, and also in the left ventricle. The latter cavity was much enlarged, and its walls hypertrophied. The mitral orifice was greatly dilated. The mitral valve was healthy. The tissue of the heart was abnormally soft; right cavities enlarged, without corresponding hypertrophy; right auriculo-ventricular opening greatly dilated.

Blood was extravasated in small quantity under the external serous membrane of the ventricles from the rupture of a small venous branch. There were the traces of old pericarditis, which had not terminated in adhesion, for lymph in patches of considerable size was found on the exterior of the auricles. Pulmonary apoplexy existed in the right lung, and pneumonia in the left.

In the three cases last detailed, the primary lesion was valvular inadequacy, in which the signs of an obstructed circulation do not necessarily exist; yet dropsy, engorgement of the right side of the heart, pulmonary hemorrhage, and hepatic congestion, were, in these instances, as fully developed as they would be in cases of obstructive valvular disease. To what, then, are we to refer these symptoms, since the mechanical derangement of the valves is insufficient to account for them? That they depended chiefly, if not altogether, on excessive dilatation of the ventricles, with which softening was combined in Case VII., is more than probable; and the writer is the more inclined to adopt this opinion from having, in so many instances, found dilatation alone, that is, uncomplicated with valvular disease, capable of developing all these symptoms in

the highest degree. (Cases I., II., III., and IV. were examples of this.)

These cases of aortic inadequacy were, towards their close, accompanied by symptoms usually found where contraction of the left auriculo-ventricular opening has existed. This illustrates the observation already made, "that *dissimilar valvular diseases* are liable to be attended ultimately with the *same symptoms*," because there is, in all of them, a tendency to the development of the same changes in the muscular tissue of the ventricles.

In the subjects of aortic patency, death generally results from a failure of the muscular power of the heart; and the great principle to be observed in their treatment, as has been clearly pointed out by Dr. Corrigan, is to seek to avert, by such stimulants as may be suitable to each individual case, this tendency of the disease, whilst at the same time local congestions are to be relieved by suitable depletion. But where aortic patency is complicated with excessive dilatation, it would appear, from the preceding cases, that death is rather the result of apnœa. Angina, more or less modified, was present in all these cases. In Case VI. aortitis existed in a well-marked degree; but in Cases V. and VII. extreme dilatation of the heart was the only probable source of this distressing complication.

Herein we see contrasted the effects of hypertrophy and of passive dilatation. In aortic patency, the hypertrophy of the left ventricle, so invariably associated with that lesion, has been well called "conservative"^a; for so long as no farther

^a By this expression, as I believe, is generally understood that, as the function of the aortic valves is annulled by their inability or insufficiency to meet behind the blood, the left ventricle is called upon not only to propel the blood by its systolic contraction, but, during the diastole of the heart, to support the column of blood in the aorta as the valves do in health. Hence hypertrophy is developed. But I apprehend it may be understood to mean more than this. When the aortic valves have become inadequate the influence of the elastic power of the aorta in propelling the mass of blood is more or less diminished, because the resistance of the valves behind

change occurs, little distress, comparatively speaking, is experienced; but as dilatation supervenes, the force of contraction of the ventricle is gradually weakened, the benefits of augmented muscular power are counteracted, and an obstructed circulation, with all the symptoms which invariably attend it, are ushered in.

As aortic patency, when uncomplicated, produces no obstruction to the circulation, so, in like manner, of mitral patency—mitral regurgitant disease. As a sequel of rheumatic endocarditis, mitral patency is very common. It may exist for years without producing much inconvenience. To illustrate the phenomena of mitral regurgitation, and to contrast them with the symptoms developed at a later period, when dilatation has been superadded, the following case, at present under observation, may be briefly narrated:—

CASE VIII.—*Mitral Patency. Hypertrophy of the Heart. Signs of Obstruction wanting.*

Maria Benson, aged 15, was admitted into the Whitworth Hospital under my care, June 28, 1852. After a severe wetting in September last this young girl was attacked with acute rheumatism in a very severe form. For four weeks she was confined to bed; after this, and even when able to walk about, she was not free from a certain amount of swelling and deformity of the joints; in December the disease was yet lingering in a subacute form. Under the use of the iodide of potassium the rheumatic affection was completely removed, and she quickly regained health and strength. At this period she became liable to palpitation of the heart, and was conscious of the unusually violent action of that organ. Being of an extremely uncom-

is essential to enable the elastic pressure of the artery to urge the fluid onwards (*vide* page 368). Hypertrophy of the left ventricle, therefore, is necessary to compensate for the loss of this important auxiliary in moving the blood. For this twofold reason the hypertrophy which attends aortic patency is always excessive, and as it enables the circulation to be carried on with more efficiency than otherwise, it has been aptly termed "conservative."

plaining disposition, she did not mention this for some months afterwards, when she grew alarmed at the continued force of the heart's action. At present she has all the appearances of robust health, being large and well formed for her age; her appetite is good; she sleeps well; she makes no complaint except of the force with which the heart beats, and suffers from palpitations only on making any unwonted exertion.

The impulse of the heart is very strong and can be felt over a large space. The parietes of the chest are visibly displaced by the violent propulsion of the organ; there is an increase in the extent of cardiac dulness, indicating considerable enlargement of the heart; the force of the impulse denotes that this is due to hypertrophy; a loud prolonged systolic bellows murmur heard over the region of the apex, which lies to the left of its usual position, denotes free mitral regurgitation; over the sternal region the sounds of the heart are heard unaccompanied by any abnormal murmur; between the spine and the vertebral border of the left scapula a single *bruit* can be distinguished. The pulse is quick, ranging from 110 to 120; its volume is disproportionate to the force of the heart's impulse. There has been no pulmonary hemorrhage or œdema of the feet. The signs of obstruction are wholly wanting.

July 20th. This case has now been for about three weeks under treatment. The force and rapidity of the heart's action have been considerably diminished by the careful use of digitalis, with mild aperients, and by maintaining mind and body free, as far as possible, from any over-excitement.

Cases such as the preceding will be familiar to every practitioner as a sequence of acute rheumatic endocarditis. The symptoms present a striking contrast to those which are developed when excessive dilatation of the ventricles has been superadded, which still further corroborates the remarks which have been already made on the influence of dilatation, especially when softening is superadded, in causing obstruction of the circulation.

The following case from Dr. Hope's work furnishes this contrast^a.

CASE IX.—*Mitral Regurgitation. Dilatation with Hypertrophy. Signs of Obstruction.*

“ Elizabeth Dennis, aged 50. Admitted into St. George's Infirmary under Sir J. Clarke, December 9th, 1830, affected with *all the symptoms of organic disease of the heart in their most severe form*. Has been affected with ascites and anasarca. Bellows murmur accompanying the first sound below the middle of the heart, but not in the region of the aortic valves; impulse strong. Pulse irregular, unequal, and extremely feeble, later than the ventricular systole.

“ *Autopsy*.—Hypertrophy and dilatation of the heart; all the valves healthy except the mitral, the free margin of which was thickened by fibro-cartilage, and the chordæ tendineæ were shortened in such a manner as not to allow the layers of the valve to come into apposition, hence a space judged to be about as large as a finger was left, through which regurgitation would take place.”

The next case not only illustrates the influence of dilatation in producing the phenomena of obstruction, but also the effects of mitral regurgitation, and of a dilated heart, on the nervous centres.

CASE X.—*Mitral Regurgitation. Dilatation of the Left Auriculo-ventricular Opening. Rupture of the Chordæ Tendineæ. Dilatation of the Heart. Signs of Obstruction. Softening of the Brain.*

Anne Connor, aged 30, was admitted into the Whitworth Hospital, under the care of Dr. Banks, November 10, 1848.

For six months previously the unequivocal symptoms of heart disease had existed. Menstruation had been irregular for three months. Since then she frequently complained of a sensation of weight in her head, dimness of sight, with great

^a Dr. Hope on Diseases of the Heart. Third Edition, p. 573.

depression of spirits, to which was superadded an uncertain and tottering gait. Symptoms of hemiplegia then became developed. On awaking one morning her tongue felt swollen, and she was unable to articulate distinctly. In twenty-four hours there was complete paralysis of the left side.

This patient left the hospital in a fortnight, and was readmitted three weeks afterwards, under my care. The prominent symptoms in the case remained unaltered. The left side was hemiplegic, without any diminution of the ordinary tactile sensibility of the surface. General dropsy had supervened. There was dyspnœa from congestion of the lungs; the face was bloated; the pulse small and frequent. She died two days after her return to hospital.

Autopsy^a.—The heart was considerably enlarged, especially its systemic portion; the left auricle presented a remarkably capacious cavity, with thickened walls; in its interior was found distinct evidence of previous endocardial inflammation, for on the free surface of its lining membrane a patch of rough, granular lymph was deposited. This extended into the left auriculo-ventricular opening, and implicated the mitral valve.

This valve was found thickened, its edges rough and irregular, whilst several of its chordæ tendineæ were ruptured.

The left auriculo-ventricular opening was rather larger than natural; the left ventricle presented a dilated cavity, whilst its walls were diminished in thickness; the aortic valves were healthy.

In the heart it appeared, therefore, that every condition existed to allow of regurgitation through the left auriculo-ventricular opening. The lungs were very much congested, but were otherwise healthy.

The morbid condition of the brain was exactly limited to the corpus striatum on the right side, the interior of which was extensively softened. The remainder of the brain was perfectly healthy, and no traces of inflammatory action, either

^a Published in Reports of the Pathological Society, January 13th, 1849.

of the brain or of its membranes, could be detected (*exsanguineous ramollissement*).

In this case, death occurred so soon after admission that there were not opportunities sufficient for carefully analyzing the physical signs. The symptoms of a lesion of the nervous system were predominant, but from the history of the case, which stated the previous existence of the symptoms of heart disease, together with the existence of general dropsy, and a loud bellows murmur over the heart, the conclusion was arrived at, that the heart was the organ primarily engaged, and that the lesion of the brain was consecutive to, and depended on, it.

This case furnishes another instance of the phenomena of an obstructed circulation as associated with dilatation of the left chambers of the heart, and of the left auriculo-ventricular opening.

It has been stated that cerebral lesions, in cardiac disease, are not peculiar to mitral contraction. In the case last recorded, extensive softening of the brain accompanied an opposite condition of the heart. But the explanation is not difficult. "The imperfect condition of the mitral valve (the result of endocarditis and subsequent rupture of some of its tendinous cords) induced 'permanent patency' of the left auriculo-ventricular opening. The lungs, no longer protected by the mitral valve, as they are in health, were gorged by the reflux blood of the left ventricle, whilst the system at large was proportionally deprived of its due supply of arterial blood: and in this instance, as in other similar ones, as also in cases of large aortic aneurism, some part of the grey substance of the brain, which so especially requires a large amount of red blood for the due performance of its functions, was found in a state of *ramollissement*. A contracted state of the left auriculo-ventricular opening, by obstructing the flow of the blood into the left ventricle, would induce the same morbid changes in the brain equally with a permanently patent condition of the same opening; for in each case the effect is the same—a dimi-

nution in the energy and efficiency of the systemic circulation"^a.

In those cases, and they are of very frequent occurrence, in which cardiac and renal disease co-exist, there is an obvious and a direct cause for a diminution in tone of the muscular fibre. It is now universally admitted, that with degeneration of the kidneys there is a diseased condition of blood (it matters not with reference to the present question, whether this depravation of the fluids *preceded*, or was the *consequence* of the renal unsoundness), and when the elements of nutrition are diseased, the heart, for obvious reasons, must be early affected. In some of the preceding cases disease of the kidneys existed, and, no doubt, exerted a direct influence on the cardiac affection (Cases iv., v.). It is unnecessary, however, that I should do more than allude to this subject here; it has been fully illustrated by the genius and researches of Dr. Bright, who first opened up an extensive field of inquiry, which has since been extended by the labours of many talented observers.

I have thus dwelt much on dilatation of the heart as a direct cause of obstruction to the circulation. It occurs under a great number of circumstances, which, if traced back to their primary source, may be reduced, probably, to three varieties:—

1. Mechanical obstruction may be the proximate cause of dilatation, which is then generally combined with hypertrophy. The several forms of valvular disease furnish numerous examples of this class (Cases v., vi., vii.), to which must be added, obstructions in the aorta. A loss of elasticity from disease of the coats of this vessel I have shown, in two instances, to be capable of producing dilatation of the heart equally with valvular disease (Cases iii. and iv.).

2. The muscular tissue may yield to the pressure from within, owing to inflammation of the substance of the heart, either acute or chronic: carditis is a generally admitted cause of softening.

^a Pathological Reports, as cited above.

3. Dilatation may exist, and yet be less a local disease than the result of deficient or depraved nutrition, as occurs in the different forms of "Bright's disease" of the kidney. In such cases, valvular disease is often co-existent; but, on the other hand, there may be no other evidence of disease in the heart than excessive enlargement. Dilatation, which will then be the *primary cardiac* affection, is, after all, in such instances, but *secondary to renal disorganization*.

The following are some of the more important inferences deducible from the preceding observations:—

1. Excessive dilatation of the ventricles of the heart is a direct and an efficient cause of obstruction of the circulation.

2. It contrasts, in this respect, with hypertrophy, the benefits of which latter condition in valvular disease are often subsequently counteracted by progressive dilatation.

3. Dilatation may exist independently of diseased valves, and may produce the general symptoms of obstructive valvular disease, or when accompanied by softening, it may develop the signs which specially indicate mitral contraction.

4. Dilatation may occur as a complication of all forms of valvular disease, modifying their signs, and producing an identity in the final symptoms of dissimilar diseases.

5. Hence, in valvular diseases not essentially obstructive, when dilatation is superadded, the signs of obstruction become developed. The occurrence of pulmonary apoplexy, and other phenomena of an obstructed circulation in aortic patency, may thus in general be explained.

6. Atheromatous disease of the aorta causes obstruction from the loss of elasticity of the vessel. Enlargement of the heart may thus be induced, which, as dilatation becomes established, proves fatal, though the valves of the heart are free from disease.