Inaugural dissertation on enlargement of the heart : submitted to the medical faculty of the university of Edinburgh in conformity with the rules for graduation by authority of the very reverend principal Baird and with the sanction of the senatus academicus / by William Robertson, Surgeon ; candidate for the degree of doctor in medicine.

ON

ENLARGEMENT OF THE HEART.

E. Forbes Esq
from the Author

INAUGURAL DISSERTATION

ON

NLARGEMENT OF THE HEART.

SUBMITTED TO

The Medical Faculty of the University of Edinburgh,

IN CONFORMITY WITH THE RULES FOR GRADUATION,

BY AUTHORITY OF

THE VERY REVEREND PRINCIPAL BAIRD,

AND WITH THE SANCTION OF

THE SENATUS ACADEMICUS.

BY

WILLIAM ROBERTSON, SURGEON,

CANDIDATE FOR THE

DEGREE OF DOCTOR IN MEDICINE.

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TO

HIS GRACE

THE DUKE OF ROXBURGHE,

&c., &c., &c.,

THIS ESSAY

IS DEDICATED WITH EVERY SENTIMENT OF RESPECT AND GRATITUDE FOR

EARLY AND CONTINUED KINDNESS,

BY

THE AUTHOR.

TO

THOMAS STEWART TRAILL, M.D., F.R.S.E.,

PROFESSOR OF MEDICAL JURISPRUDENCE IN THE

UNIVERSITY OF EDINBURGH,

&c., &c., &c.,

IN GRATITUDE FOR HIS VALUABLE INSTRUCTIONS,

AND IN

ACKNOWLEDGEMENT OF HIS UNIFORM KINDNESS,

THIS ESSAY

IS RESPECTFULLY INSCRIBED

BY

THE AUTHOR.

ON

ENLARGEMENT OF THE HEART.

No form of disease which affects the heart is more familiar to the medical observer than Hypertrophy. The frequency of its occurrence, the fatal issue to which it too often leads, and the circumstance of its being in some measure within the reach of his remedies,* combine to give an interest to its pathology.

The remarks contained in the following pages are designed to include the consideration of two morbid conditions of the heart, viz. Hypertrophy and Dilatation. From the similarity of the causes which produce these morbid states, and from the frequency with which both occur in the same heart, it is inferred, that we can best illus-

* Alison's Elements of Pathology, p. 577.

trate the pathology of each affection by considering both together, and that no complete account of the one can be given without embracing some notice of the other.

It is my intention, in the first place, to enumerate and define the different varieties and combinations of disease included under the title of this dissertation. The causes, symptoms, and diagnosis will be next considered, and, in conclusion, the general principles of treatment will be briefly alluded to. A few cases which have lately fallen under my observation while I attended the Royal Infirmary will be introduced where they seem to illustrate the subject, or give occasion for remark. The pathological conditions which it is proposed to consider, (although not embracing all the divisions established by authors) are the following:—1. Hypertrophy. 2. Dilatation. 3. Hypertrophy with Dilatation. 4. Hypertrophy with Contraction. The first division (Hypertrophy, or increase of nutrition,) implies simply a thickening of the muscular walls of one or more of the cavities of the heart. This thickening takes place by an actual increase of muscular substance, and, probably, consists essentially in an interstitial deposition of muscular fibres. Hypertrophy may affect a cavity of the heart either generally or partially; hence a cavity

is sometimes found to have its parietes thickened and somewhat indurated at one part, while other points have undergone no such change, and may even have become softened and attenuated.* Lænnec, in his great work on Auscultation, states, that in the parietes of a heart affected with hypertrophy, besides the increase of thickness, an unusual degree of firmness and intensity of colour may be observed. Simple hypertrophy seems to be one of the most common species of enlargement of the heart, although certainly not so often met with by itself as in combination with the next variety.

Dilatation signifies increase of the capacity of a cavity. Like hytertrophy, dilatation may be general or partial, in the latter case constituting true aneurism of the heart. In all cases of pure dilatation, the muscular walls of the cavity are flabby, often pale, and necessarily more or less attenuated: for if they exceed, or even maintain their natural thickness, the case must be referred to the next and most usual variety.

Hypertrophy with dilatation is a term used to describe the state of a cavity when simultaneously affected by both the preceding varieties. The appearances, which hearts so affected present, are

* Andral Anat. Path. vol. i. p. 305. Lænnec, vol. ii. p. 500: Paris, 1826.

very variable, according as the hypertrophy or dilatation is the more conspicuous.

It is by no means uncommon, to meet with cases presenting hypertrophy of one part of the heart, and dilatation of another;* but of course, such cases cannot be included with propriety under this head of the subject. Of all the cavities of the heart, none is so prone to disease as the left ventricle; and, accordingly, it is here that hypertrophy and dilatation are most frequently found. The right ventricle is more frequently found dilated than hypertrophied, and the same may be said of the auricles. Andral† doubts whether the auricles (particularly the left one which seems least prone to disease)‡ have ever been found hypertrophied, except where there coexisted disease of the ventricles. Dilatation of the auricles is comparatively frequent, and a remarkable case of this lesion, which occurred in the Royal Infirmary,§ will be hereafter introduced.

Hypertrophy, with contraction or diminution of the cavity, was a form of disease known to Morgagni and Corvisart; but it is to Lænnec, Bouilland, and Bertin, that we are indebted for a

* Lænnec, vol. ii. p. 517.

† Anat. Path. vol. ii. p. 305.

‡ Corvisart sur les maladies du Cœur, p. 120.

§ See Case IV. of Table, and Case V. in the text.

full account of it. The last named author, in particular, has bestowed much attention on this affection, and has named it Concentric Hypertrophy. The appearances observed in a case of this kind are the following:—The bulk of the heart, or portion affected, may remain unaltered, be rather contracted, or considerably augmented. The muscular substance feels firm, and is of unusual thickness; while the cavities are diminished, and in some instances, could scarcely hold an almond. Bouilland asserts, that these appearances are most frequently observed in the right ventricle, and says, that in one case of concentric hypertrophy, he found the walls of this cavity to measure eighteen lines in thickness. Cruveillhier seems to have been the first to question the propriety of regarding concentric hypertrophy as a disease. He expresses it as his opinion, that the diminution of capacity with thickening of the parietes, is entirely an accidental post mortem appearance. He accounts for it, by supposing the heart in the full vigour of contraction at the moment of death. He instances several cases of malefactors executed by the guillotine, in whose hearts he observed this peculiar contraction. Similar appearances were observed by Mr. Jackson in the bodies of those who died of cholera at Paris. Dr. Budd is the most recent writer on this subject. In his

paper,* he has given several cases of apoplexy, in which concentric hypertrophy was found after death. In these cases, there existed none of those valvular derangements which so frequently produce hypertrophy; there had been no irregularity or intermission of the pulse during life, no anasarca, no symptom of pulmonary congestion; and yet upon dissection, while the right ventricle seemed of average capacity, the left was very much contracted, in one case almost obliterated. Now, it seems hardly possible for such a condition of the heart to exist, for any space of time, without causing a most serious obstruction to the circulation. Besides, Dr. Budd found, that by dilatation with the fingers, or by maceration for a few days, he could restore the heart to its ordinary capacity and proportions; and, in one opportunity which occurred to me, his statement was fully verified. Hope† believes, that concentric hypertrophy may cause anasarca and dyspnœa, and there seems every reason that these symptoms should be observed: for in cases of contraction of the left auriculo-ventricular orifice they are always present; and concentric hypertrophy of the left ventricle would cause a similar retardation of the circulation. We are not, however, informed, that Dr.

* London Medico-Chirurgical Transactions, 2d S. v. iii. p. 316.

† Hope on the Heart, p. 209.

Hope's cases were uncomplicated with other disease, which might have caused similar symptoms. May not the cases of supposed concentric hypertrophy have been merely instances of the ordinary forms of hypertrophy? and may not the contracted state of the cavities after death be accounted for, on the supposition of life having become suddenly extinct, while the heart was acting violently? "To be justified in declaring the existence of concentric hypertrophy," says Dr. Elliotson,* "not only should the parietes be thick, but the bulk natural." A heart, with parietes and cavities of more than ordinary dimensions, might not exceed, when in a state of contraction, the bulk of a healthy heart, consequently this rule of Dr. Elliotson's cannot be applied.

In Bouillaud's case of enormous thickening of the parietes of the right ventricle, (a part of the heart by no means prone to such disease), it is not improbable that some dilatation had existed during life. As a congenital malformation, there is little doubt that concentric hypertrophy has occurred, and Dr. Budd gives several cases to prove it; in these it was observed in the right ventricle only. After attentively considering the arguments brought forward by Dr. Budd, I am led to

* On recent improvements in Diagnosis, p. 25.

the following conclusions. That, as a congenital malformation, concentric hypertrophy has been proved occasionally to occur ;—that it is still doubtful, whether it be a disease to which the adult body is subject ;—that in most cases where a post mortem shows the lesion, it is merely an accidental appearance, caused by the heart having contracted on its contents, as seems fully established by the absence, during life, of any symptoms of obstructed circulation. Analogy is opposed to the recognition of this disease ; no hollow muscle in the body presents such appearances unless when in a state of contraction, as, for instance, the intestinal canal when empty, and uterus after the expulsion of its contents. "The increased thickness," Dr. Budd also observes, "of the muscular parietes of the left ventricle, in cases where its capacity is vastly diminished, even in relation to its discharging orifice, is rendered improbable also, by reflecting on the mechanical considerations by which the almost constant occurrence of hypertrophy in cases of dilatation is explained."

It is a singular circumstance, that the proportionate thickness of the ventricles of the heart varies at the different periods of life. Thus, in the prime of life, the thickness of the right is to that of the left, as one is to two ; in infancy and

old age, the proportion is as one to three or four.* Andral, after making these statements, remarks, that though in the two last periods, the proportionate thickness of the left ventricle be nearly equal, nothing can be more different than the character of the pulse, and concludes, that the force and hardness of the pulse cannot be dependant exclusively on the degree of the thickness of the left ventricle.

The causes of the several forms of enlarged heart must next be considered; and here, perhaps, a case may serve to illustrate a part of the subject.

Case I.—The patient, Mary Elmslie, aged 24, a servant, and unmarried, was admitted to the Infirmary on the 7th November, 1838, and was placed under the care of Dr. Graham. Upon admission, she stated that, eight years before, she had an attack of acute rheumatism, and six years afterwards, "an inflammation in the left side." Towards the end of October, after unusual exposure to cold, she had rigors and other febrile symptoms. On the 2d November, was seized with pain extending from the occiput along the spine, and a "stitch" under the lower part of the sternum, preventing full inspiration; was bled,

* Andral Path. Anat. vol. ii. p. 303.

and the symptoms abated after the operation; soon afterwards became very sick, and vomited much bitter matter. When first seen in the hospital, "she appeared of sanguine temperament, her face was flushed, she had no local pain,—complained of nausea on raising her head, of frequent bitter vomiting, and of a cough, which was not urgent, but accompanied by copious expectoration of tough mucus. The respiration was natural, the tongue coated with a thick whitish fur, the bowels regular, much thirst and anorexia for the preceding eight days, and little sleep obtained for the same period. Thorax sounded well on percussion, except in the cardiac region, where some dulness was observed. Respiratory murmur every where audible. Heart's action very violent and diffuse. A tremor communicated to the finger when applied over the apex. Loud rough bruit with the first sound, best heard a little to the left of the sternum—over the cartilage of the fourth rib. P. 128, intermitting slightly, and irregular in force."

The treatment consisted in leeching, together with the exhibition of digitalis, colchicum, and, latterly, of mercury, which was given freely, in order to affect the system rapidly. This, however, it was found impossible to do, probably on account of some diarrhœa excited by the previous use of

colchicum. Notwithstanding the administration of the acetate of lead and opium, the purging continued, delirium was occasionally observed, the respiration became affected, and on the 18th November, at 11, A. M. she died.

Sectio. 20th November.—" Numerous adhesions, both new and old, between the left lung and the pericardium. The heart inseparably united to the pericardium, throughout its whole surface, by old and firm adhesions. Left ventricle slightly hypertrophied. Mitral valve shortened and thickened in both its segments; left auriculo-ventricular orifice contracted; when viewed from the auricle, its margins seemed thickened, and presented an irregular waved line. Further examination was not allowed."

That, after attacks of acute rheumatism, enlargement of the heart is peculiarly liable to occur, is a fact generally admitted; but pathologists differ, as to the manner in which rheumatic disease acts in producing this enlargement. It is stated by Hope, Copeland, and others, that hypertrophy is liable to take place after attacks of acute rheumatism, without any inflammatory action, either in the pericardium or internal membrane of the heart having intervened.* In many cases, however, as in that given above, traces of

* Dictionary of Practical Medicine, Heart.

pericarditis are found upon dissection. The obstacle to the movements of the heart, occasioned by adhesion of the pericardium, has been supposed by some, to be the sole cause of hypertrophy in these instances.* A case is given by Andral,† in which death took place, obviously from a difficulty at the heart in the transmission of blood, and in which, it appeared upon dissection, that adhesion of the pericardium had taken place, while no other morbid appearance was observed. Now, if, as Andral believes, death took place in this instance, from the heart being "compressed and embarrassed in its movements, by the solid envelope of lymph which everywhere surrounded it," there seems no reason to doubt, that, had the patient lived longer, the necessity for increased propelling power, in order to overcome the obstacle to the circulation, might have induced hypertrophy, just as valvular disease is frequently found to do. Cruveillhier thinks, that too much importance has been attached to adhesion of the percardium as a cause of hypertrophy, and remarks, that he has known cases where this adhesion existed along with diseases calculated to impede the free transmission of blood through the heart, and even hints,

* Hope, p. 138. Alison, p. 579.

† Andral Clinique Medicale, by Spillau, p. 231.

that such diseases will be always found to co-exist.* Andral has another and more theoretical way of accounting for enlargement of the heart after pericarditis. A previous inflammatory condition of the pericardium, he conceives, operates in this manner, in accordance with a law of pathology, viz.—"That wherever a muscular membrane contributes to form the parietes of a cavity, we constantly find hypertrophy of that membrane produced, by an antecedent hyperæmia of the mucous or other membrane lining the interior of the cavity."† In another work of M. Andral's, I find the following words, "In every part, where the muscles of organic life are in contact with an inflamed membrane, whether mucous or serous, they have a remarkable tendency to become the seat of a more active nutrition, to become in fact hypertrophied. This is very manifest, with respect to the fleshy tunic of the stomach, intestines, and bladder, in cases of chronic inflammation."‡ The case of Elmsley seems to possess some interest, when viewed in connexion with this subject. The adhesion of the pericardium, in her case, must have been of at least two years standing, and when

* Cruveillhier Anat. Pathologique, vol. ii., p. 72-3.

† Path. Anat., t. ii., p. 317.

‡ Clinique Medicale, p. 237.

we consider the active life of the patient up to the date of her admission into the hospital, it is astonishing that the bulk of the heart was so little altered. According to Dr. Hope, no affection of the heart produces fatal effects more rapidly than adherent pericardium.*

Another obstacle to the circulation through the heart in Elmsley's case, was the contracted and rigid state of the left auriculo-ventricular orifice. Although the left ventricle was the only part of the organ which was hypertrophied, it is by no means impossible that the impediment to the flow of blood from the left auricle into the ventricle, may have caused the increase in bulk, by acting back through the pulmonic circulation, in the manner described by Hope,† or by permitting regurgitation during the ventricular systole. That some regurgitation took place during life, is rendered probable, by the circumstance of bruit being heard during the systole, over the mitral valve. Certain it is, that no cause of hypertrophy is more common or efficient, than diseased conditions of the valves of the heart. Before entering more fully upon this subject, a case may be given by way of illustration.

* Hope on Heart, p. 367.

† Hope, Op. Cit., p. 199.

Case II.—Alexander Galloway, aged nineteen, a tailor, was admitted under Dr. Christison's care, on the 4th July 1838. On admission, he had a variety of symptoms indicating heart disease, viz. dyspnœa, palpitation, throbbing in the vessels of the neck, starting from sleep, &c. The apex of the heart was felt to pulsate below the sixth rib. The impulse was preternaturely strong and diffuse; it was felt in the epigastrium. There was an unusual extent of dulness on percussion in the præcordial region, and bruit was distinctly heard with both the heart's sounds. The patient had been long a martyr to rheumatism. The treatment consisted at first of moderate blood-letting and leeching; a seton was for some days kept inserted over the heart. An attack of acute rheumatism, which he took while in the hospital, yielded to the employment of blood-letting and sudorifics. About the beginning of August, his cardiac symptoms became greatly aggravated, and he died on the 6th August.

Sectio, 8th August, ℥iv. of a straw-coloured fluid in the pericardium. Some tolerably firm lymph adhering to the auricles, and near the apex. All the cavities dilated, and more or less thickened, especially the left ventricle. Aortic and mitral valves thickened, shortened, and insufficient. The inner surface of the left ventricle

was very white, apparently from thickening of its lining membrane. Lungs gorged with blood and serum. The weight of the heart, when all the blood was squeezed out of it, was ℥xxii.

This case embraces several points of interest. The peculiar appearance of the cavity of the left ventricle renders it probable, that an inflammatory process had at one time being going on there, and that the thickened and shortened state of the aortic and mitral valves, was the effect of the contraction of a portion of coagulable lymph, thrown out upon these valves, during the course of the inflammation. Many similar instances of contraction of these valves have fallen under the notice of Andral, and he has given a valuable record of his reasons, for believing inflammation to be the primary disease in such cases.* Another interesting feature in the case of Galloway, is the example which it presents of "active aneurism of the heart," caused undoubtedly by disease of the valves: and it becomes necessary to consider, how these valvular derangements operate in producing enlargement. A valve, which has suffered from inflammation, may offer a double obstacle to the circulation: by its contraction, it presents an obstacle in an orifice through which blood

* Andral Clinique, p. 239, and Appendix to Cullen, by Gregory.

must pass, and by its rigidity and consequent patescence, it permits regurgitation at the moment when it ought to prevent it, thus offering an additional impediment to the circulation of the blood. Hypertrophy, in such circumstances, may be accounted for, on the general principle, that when resistance is habitually offered to the contraction of a muscle, that muscle endeavours to react against the impediment, and becomes hypertrophied. The occurrence of dilatation admits of a similar explanation. The aortic valves, we shall suppose, are shortened and thickened, and consequently a difficulty in the transmission of blood into the aorta results. The effect is, that as a constant pressure is kept up against the parietes of the left ventricle, if these are not able to react against the obstruction by becoming hypertrophied, dilatation is the natural consequence. "When, along with increased action and increased bulk of the heart, there is a full quantity of blood in the body, the cavities of the heart are usually found dilated, at the same time that their parietes are thickened; but when the quantity of blood in the body is much diminished, the tonic contraction of the thickened fibres of the heart, which is no longer distended, occasions a diminution in the size of its cavities."* So, in a system

* Alison's Pathology, p. 563.

debilitated and destitute of tone, a distending force will tend rather to the production of dilatation than to hypertrophy of the heart.

If cases of congenital malformation be excepted, the causes of enlargement of the heart may be reduced to the following, viz.: Whatever tends materially to obstruct the circulation, whether pulmonic or systemic. To illustrate this position, a few of these causes may be enumerated. In some instances the aorta is found to be of small calibre, without any morbid contraction or loss of elasticity, but apparently from an arrested development. A much more frequent morbid appearance is a dilated, rough, and inelastic condition of the aorta and its great branches. The roughness is observed in all degrees, from the slightest cartilaginous thickening to a copious deposit of an ossific character. The last variety has been termed "bony" deposit, incorrectly however, as no anatomical evidence of its osseous nature has been adduced, and "calcareous" would probably be a more proper epithet. A well marked case of this kind occurred in the Infirmary, in November 1838.

Case III.—The patient, Alexander Campbell, aged 60, had, in addition to enlargement of the right side of the heart, (consequent on emphysema

pulmonum, asthma, and bronchitis,) hypertrophy of the left ventricle, and this was believed to depend more upon a dilated aorta, studded with calcareous deposit, than upon a small amount of disease in the aortic semilunar valves, which was also discovered upon dissection. Dilatation of the aorta is the first step to the formation of an aneurism, which, *a fortiori*, may be a cause, and a most irremediable one, of the production of hypertrophy. Partial aneurisms of the heart itself have been observed, sometimes in connexion with enlargement of the organ. A number of such cases have been published in the London Medico-chirurgical transactions, by Mr. Thurnam. In a subject brought to one of the dissecting-rooms in town, I had an opportunity of seeing an aneurism at the very origin of the aorta, which had caused death by bursting into the pericardium. The left ventricle was hypertrophied in this instance, but the state of the valves I cannot recollect. Another case of aneurism of the ascending aorta, communicating with the pulmonary artery, and connected with enlargement of the heart, will be found in the appendix.* It were superfluous, after the remarks made upon the case of Gallo-

* See Case XV. Hodge, in Table.

way, to advert more particularly to disease of the valves. I may merely mention, that, when enlargement of the right ventricle occurs in consequence of obstruction at an orifice, that obstruction is most commonly found at the left auriculo-ventricular orifice; and that, in such circumstances, pulmonary apoplexy is very liable to take place. In one fatal case, however, in the Infirmary, this winter, it was shewn by dissection, that the enlargement of the right side of the heart depended upon a very diseased state of the semilunar valves at the origin of the pulmonary artery. Chronic bronchitis, especially when complicated with emphysema pulmonum and asthma, leads, in a great many instances, to a dilated or hypertrophied state of the right side of the heart. This has already been incidentally mentioned in the case of Campbell, and many other instances might be given. I shall select one of several that were treated in the clinical wards of the Infirmary this session.

Case IV.—Thomas Chisholm, aged 22, a patient of Dr. Alison's presented the following symptoms, cough, dyspnœa, muco-purulent sputa, livor of face and extremities. Chest resonant on percussion. Subcrepitant râle, and resonance of

the voice in several points of the upper part of right side of chest. Strong pulsation in epigastric region.

A post mortem examination displayed emphysema of both lungs, with dilated bronchial tubes, chiefly on the right side. The right ventricle was dilated, and its parietes were firmer than usual.

Other diseases which prevent a free circulation through the lungs produce similar effects, as repeated attacks of pleuritis and pneumonia, and especially empyema. In some of these cases, however, it becomes difficult to assign a sufficient reason for the enlargement of the heart, and to determine whether the disease of the heart owes its origin to the pulmonary affection, or whether the pulmonary disease is not merely rendered unusually obstinate by being complicated with disease in the heart of previous origin. It is probably from the obstruction to the pulmonary circulation which their occupations present, that divers, sawyers, smiths, &c. are so subject to cardiac disease. Hereditary predisposition, habitual and severe exertion, intemperance, and mental excitement, certainly assist in producing such affections. It seems, also, not improbable, that the formidable fits of palpitation so often observed in dyspeptics, and particularly in hysterical females, may sometimes lead ultimately to organic disease.

The frequent disturbance thus offered to the equable flow of the blood, and the inordinate action of the heart, offer conditions not unfavourable to the production of hypertrophy.

The next division of the subject includes the symptoms and diagnosis. The ordinary symptoms may be first enumerated, and the physical signs afforded by auscultation and percussion afterwards discussed.

Of the local ordinary symptoms of enlarged heart, palpitation is the most obvious. It is not, however, uniformly present, and it is often unheeded by the patient, particularly when his disease is of long standing. In most cases of hypertrophy, it is easily detected by the medical attendant, as it is sometimes so violent as to be perceptible at the distance of several feet, and even to raise the bed-clothes when the patient is in the horizontal posture. After any exertion, as walking up stairs, it is, of course, aggravated. Palpitation may, however, be present,—may cause the utmost annoyance and alarm to the patient,—may even exist along with many of the most usual symptoms of enlarged heart, and yet the viscus may be perfectly healthy in structure. When, however, it occurs in connexion with disease, although modified in degree by the ordinary exciting causes, it is *constantly* present, or at least

more so than in purely nervous cases, in which mental agitation excites it powerfully, and in which complete remissions take place. Upon the whole, palpitation, unless its exact seat be attended to, (as will be explained when treating of the physical signs,) is of little value as a means of diagnosis.

Neither are the indications afforded by the pulse free from fallacy. Frequently it is felt to be hard, full, and incompressible, and often indicates, in such cases, that rigid and dilated state of the aorta, with which, as has been already remarked, hypertrophy is so often associated. In other cases, a small, feeble, intermitting pulse is found connected with a contraction of the left auriculo-ventricular orifice, a lesion generally followed by enlargement of the right cavities of the heart, and frequently by similar disease on the left side. I shall here insert a case of narrowing of the left auriculo-ventricular orifice, which was the cause of enlargement in all the cavities of the heart.

Case V.—Robert Clarke, aged 40, a patient in the male clinical ward, on the 15th November 1838, presented the following symptoms. Erisipelas of the head and face of several days standing, general œdema, bronchitis, (to which he had

long been subject), dyspnœa, drowsiness. Pulse extremely irregular in force and frequency, generally very small. Percussion dull over the sternum. Impulse of the heart very diffuse. Sounds confused and without bruit. The jugular veins pulsate, and when the patient coughs, they become remarkably turgid. He died in a few days, and on the 20th November the post mortem examination took place.

ʒxxx of a straw coloured fluid were found in the pericardium. To various parts of the membrane, there adhered shreds of pretty firm lymph. Enormous dilatation of the right auricle had taken place, and a very large clot of decolorized blood was found in it. Similar clots adhered to the musculi pectinati in the auricle, and to the chordae tendineæ in the right ventricle. The right auriculo-ventricular opening was very large, and the tricuspid valve was shortened in all its segments. Hypertrophy, but no dilatation of the right ventricle. Left auricle much dilated, but not by any means to such an extent as the auricle on the right side. The flaps of the mitral valve were shortened and thickened, so as to narrow considerably the left auriculo-ventricular orifice. No dilatation of the left ventricle, which, however, was preternaturally thick

and firm in its parietes, and contained some coagula. Semilunar valves at the origin of the aorta and pulmonary artery healthy. Liver large and indurated.

In a case like this, some irregularity of the pulse was easily accounted for; but irregularity or intermission of the pulse cannot be considered as pathognomonic of any form of diseased heart. It is well known, that the pulse may present the most marked irregularity, in cases where the heart is in its normal condition. Thus, in affections of the brain, in fever, and even in a state of health, the pulse has often been observed to be remarkably slow. In three cases of individuals, whose pulses averaged 40 in a minute, I was unable to detect any symptom of organic disease. The intermittent pulse is met with also in persons apparently in good health,—more frequently in the subjects of dyspepsia, hysteria, and hypochondriasis.

There are many other symptoms referable to the circulation, which may be mentioned here. The chief of these are, throbbing sensations in the vessels of the head and neck, vertigo, tinnitus aurium, muscae volitantes, and momentary loss of vision. That pain which has been termed Angina pectoris may be present, but seldom in the very aggravated form described by authors.

It is of most frequent occurrence, according to Dr. Elliotson, when the pericardium is the seat of inflammation ; others have endeavoured to connect it with an ossified state of the coronary vessels of the heart. It is no doubt observed in some cases of these affections, but is at best an inconstant symptom,* and is as often met with in cases of enlarged heart as in either of them. A tendency to syncope is one of the most alarming symptoms with which disease of the heart can be complicated; many cases might be cited, in which a person labouring under disease of the heart has been cut off suddenly by syncope, from which he could not be resuscitated. Dyspnœa, (one of the commonest symptoms of the diseases under consideration,) may depend upon various circumstances, such as, 1. Upon organic disease of the lungs, as Emphysema or Bronchitis; 2d, Upon effusion of blood into the pulmonary tissue, as in Apoplexia Pulmonum; 3dly, On effusion into the pleural cavities and pericardium, as in Hydrothorax; 4thly, On true Œdema Pulmonum; 5thly, and perhaps most frequently, on the collection of frothy serum in the air-cells and bronchi consequent upon congestion.

* Appendix to Cullen's Practice of Physic by Gregory, page 334.

Anasarca and ascites are often symptomatic of enlarged heart. The ascites is usually caused by an obstruction to the portal circulation, hence disease of the liver frequently co-exists. The anasarca seems to be an effort of nature to relieve the distended capillaries; it usually commences at the feet, and by degrees extends upwards, infiltrating the loose cellular tissue sometimes to a frightful degree. The granular disease of the kidney often co-exists, and, in such cases, it is often difficult, but happily quite unimportant, to determine from which cause the anasarca proceeds. This complication, however, must contribute to render the prognosis unfavourable. Livor of the face is often observed, when an obstacle to the free transmission of blood through the right side of the heart is present; and when the jugular veins are turgid and pulsate, especially if, at the same time, we can feel the systolic impulse in the Epigastrium, there are good grounds for inferring, that the right side of the heart is enlarged. To similar causes, many cases of hæmorrhagies, as epistaxis, hæmatemesis, purpura, &c. owe their origin. In every case of cerebral apoplexy, the state of the heart ought to be investigated, for, in a large number of such cases, hypertrophy of the left ventricle, with or without dilatation,

has been observed. A great number of the cases recorded by Andral presented this lesion; and a very remarkable instance of the sort was seen on the post mortem examination of a girl, in the Edinburgh Infirmary, this winter. Another patient named Mrs. Rodney, who died in the hospital last year of dropsy and dilated heart, had, while in the house, a paralytic stroke, probably from a like cause. Pneumonia, bronchitis, and enlargement of the liver, although not strictly speaking symptoms, are such common complications, that they should lead the practitioner to examine the cardiac region with some care. A symptom, complained of by several patients, whom I have had an opportunity of questioning, and who presented unequivocal evidence of hypertrophied heart, is, the expectoration of a small quantity of blood in the *morning* occasionally, or, as one expressed it, *finding blood in the mouth upon wakening.*

Having concluded the above very imperfect sketch of the ordinary symptoms in cases of enlarged heart, I shall next attempt a brief outline of the signs afforded by the means of physical diagnosis. When the hand is laid upon the præcordial region of a person affected with enlargement of the heart, the following circumstances may be observed:—1st, The hand may

be repelled by the stroke of the heart, with preternatural force, and with a decided thrusting sensation. 2d, The apex may be felt *punctuate* below the sixth rib. 3d, The action of the heart may be felt over a larger space of the chest than is natural: it may be strongest under the lower end of the sternum, and even extend to the epigastric region. 4th, Instead of the strong and "jogging" beat, an undulatory tumbling motion may be communicated to the hand,—a motion which does not raise it decidedly from the chest. 5th, The impulse may be so weak as to be hardly perceptible. 6th, There may be a double or even a triple impulse. 7th, That peculiar thrill, which was so appropriately termed by Lænnec, "Fremissement cataire," may be distinguished. Upon placing a finger over the situation of the carotid and subclavian arteries, these vessels will frequently be found to bound during the ventricular systole, to feel large and jarring, and even to communicate a distinct "fremissement." Many of these signs are also appreciable by means of the stethoscope, but not being proper auscultatory signs, I prefer the arrangement of them under the head of *palpation*.*

By the use of the stethoscope, the following

* Piorry.

conditions of the heart's sounds may be detected:—1stly, The sounds may be either prolonged or shortened. 2dly, The sounds may be heard over an unusual extent of the chest. 3dly, The first sound may be long and loud. 4thly, It may be short and flapping,* like the normal second sound. 5thly, An abnormal sound or bruit may accompany or replace the first sound. 6thly, The second sound may be accompanied or replaced by a bruit,—and this bruit may be prolonged, so as to fill up the interval which, in ordinary circumstances, is interposed between the periods of the second and first sounds. Percussion may elicit a dull sound over a large space in the præcordial region, indicating effusion into the pericardium, or the size of the heart itself.

These being the chief physical signs of enlarged heart, let us consider them *seriatim*, and briefly state the pathological conditions necessary for the production of individual symptoms. The phenomenon of increased impulse is often associated with a thickened state of the parietes of the heart. When preternatural impulse is felt to the left of the sternum, hypertrophy of the left ventricle is inferred;—when under the

* Hope.

lower end of the sternum, and in the epigastrium, it is symptomatic of disease in the right ventricle. The jogging resisting character of the impulse, is ascribed by Hope and others, to the comparatively slow and forcible contraction of a hypertrophied ventricle. The punctuate pulsation of the apex below the 6th rib in a well-formed chest, is probably the most unequivocal symptom of enlargement of the heart; but even this is not free from fallacy. During a deep inspiration, Dr. Williams has remarked, that the apex of a healthy heart beats in the 6th intercostal space. Besides, in cases of dilatation, it is sometimes difficult to ascertain the precise position of the apex, owing to the feebleness of the impulse. An emphysematous lung may project over the heart, so as to prevent the apex being felt, and a hypertrophied lung may produce the same effect. Neither must we conclude, that because the apex is not felt lower in the chest than natural, considerable enlargement cannot have taken place, for an enlargement of the liver may have prevented the apex from descending, and forced it aside. A case occurred in Dr. Alison's ward this winter, which may serve to illustrate these remarks. The patient, named George Dear, died, and, on dissection, presented enlargement of the liver, together

with great dilatation and some hypertrophy of the left ventricle; thus explaining, what had been remarked during life, viz. that although other symptoms left no doubt of the existence of considerable cardiac disease, yet the apex continued to pulsate in the 5th intercostal space. A patient named Ainslie, who died under Dr. Christison's care, had similar symptoms, and presented similar appearances on dissection. Empyema, and pneumo-thorax may affect the position of the heart. In the case of Robert Mackay, a patient of Dr. Traill's, in the Infirmary, who died of pneumo-thorax and acute pleuritis of the left side, consequent upon the bursting of a tubercular cavern, the apex was forced to the right of the sternum. The undulating tumbling motion, felt by the hand, is most frequently observed when dilatation exists without hypertrophy. A double or even triple impulse is mentioned by authors, and is frequently met with. Sometimes it depends upon the "back-stroke*" or return of the heart to its position during the diastole of the ventricles. In other instances there is a kind of fluttering irregularity in the heart's contraction, which seems to give the double or triple impulse. This symptom occurred

* Hope.

in Clarke,* who had disease of both auriculo-ventricular orifices, and is well-marked at the time I write in a girl in Dr. Christison's ward, named Helen Duncan, who has all the symptoms of an enlarged heart, consequent on a contracted state of the left auriculo-ventricular orifice.

"Fremissement cataire" is so similar in its mechanical cause to "bruit de sufflet," that I shall postpone further mention of it, till the sounds of the heart are treated of.

The great impulse of the arteries at the root of the neck, commonly indicates dilatation of the aorta and its large branches, frequently combined with enlargement of the left ventricle;—when taken by itself, however, it is not a symptom to be much depended upon.

The prolongation of the first sound may depend upon an hypertrophied condition of the ventricles, especially if at the same time there exist a contraction at the origin of the aorta or pulmonary artery. The second sound can hardly be prolonged, without losing its character; in cases, however, in which the aortic valves permit regurgitation during the ventricular diastole, and more especially when an auriculo-ventricular orifice is contracted, the second sound is apt to be accom-

* See Page 32.

panied or replaced by a prolonged bruit. The extent of the thorax, over which the heart's action is audible, varies so remarkably in the state of health, that it is difficult to assign normal limits to the sounds. In hysteria, in dyspepsia, in pneumonia with condensation, and in pleuritis with effusion, the sounds of the heart are sometimes audible over the whole chest. It cannot, however, be questioned, that when the heart is enlarged, its sounds are most commonly heard over a preternatural space, and that the position of this space varies with the part of the heart affected. Thus, in a case of hypertrophy and dilatation of the right ventricle, the extension of the sounds is usually in the direction of the epigastrium, and right side of the chest. When the first sound is loud, and accompanied by a smart impulse, we are inclined to infer, that hypertrophy and dilatation are conjoined. When the systole of the ventricles conveys a short and flapping sound with a feeble impulse, dilatation simply is believed to exist. It has been stated, that the sound in cases of dilatation is louder than natural, but I am convinced, that in more than one case of simple dilatation which I have examined, this statement could not be verified. Dr. Hope describes the character of the first sound in most cases of simple dilatation, as "short, clear, and

flapping," omitting the term "loud," which other writers have perhaps too hastily applied.

The abnormal sounds observed in many cases of enlarged heart next demand attention; and it is proposed to enter upon this subject, with a few general observations upon the mechanical cause of "bruit de sufflet" and "fremissement cataire." Bruit is so often observed in cases where no organic lesion exists, that its value as a sign of disease becomes questionable, and it seems to be only when taken in conjunction with other symptoms that it can materially aid our diagnosis. How is bruit produced? After considering the theories proposed by Lænnec, Bouillaud, Hope, and others, for the explanation of this phenomenon, with some care; I cannot resist the conclusion, that the theory proposed by Dr. Corrigan of Dublin, is the most satisfactory one that has appeared. I may say the *only* satisfactory one, for by this theory, all the circumstances under which bruit has been observed are explicable; and I may refer to the paper in the Dublin Journal* for a refutation of all preceding theories on this subject. Two circumstances must be combined, according to Dr. Corrigan, in order to produce bruit or fremissement; there must be a current

* Dublin Journal, (Medico-Chirurg.) Vol. X.

of blood, and that current must enter a cavity, the parietes of which are in a state of diminished tension. One difficulty which this theory obviates, and which is fatal to some of the others which have been proposed, is, the occasional occurrence of "active aneurism" of (we shall say) the left ventricle, without murmuring accompanying the systole. This state of matters is precisely equivalent to contraction of the aorta at its origin, and, according to Bouillaud's theory, ought certainly to give rise to bruit. But, upon Dr. Corrigan's theory, the fact is easily explained, —the aortic valves being sound, the cavity of the aorta is never partially emptied by regurgitation, and, consequently, its coats are never thrown into that state of diminished tension which is necessary for the production of a murmur. But there are cases of "active aneurism" in which neither sound is altered, and such cases seem rather inexplicable upon Dr. Corrigan's theory. During the ventricular diastole, which must take place to a larger extent, in proportion to the dilatation of the cavities which exists, blood must be sucked in through the mitral orifice with unusual rapidity, and the current thus produced must impinge upon the parietes of a cavity in a state of diminished tension. Here we have both the postulates of Dr. Corrigan present, and must

make some supposition in order to account for the absence of murmur. In defence of the theory, I would suggest, either that the ventricle does not in such cases empty itself completely during its systole, and, consequently, that during the diastole no more than the usual quantity of blood passes through the auriculo-ventricular orifice,—or that this orifice becomes enlarged in proportion to the size attained by the ventricle, and that the increased quantity of blood which is sent through at each diastole, is thereby prevented from being thrown into current-like motion.

In the class of cases to which this dissertation is restricted, bruit is not a constant symptom. It is most frequently present in those cases of enlargement which depend upon valvular disease, and, as a general rule, is best heard over the affected valve. When the semilunar valves of the aorta are diseased, a murmur commonly accompanies the first sound,—if they permit considerable regurgitation, there is often a murmur with the second sound, and an appreciable interval between the pulsations of the heart and those of the radial artery, a fact first noticed by Dr. Henderson. When a diseased state of the pulmonary semilunar valves has induced hypertrophy of the right side of the heart, the diagnosis might be facilitated by attending to the following circum-

stances :—1st, The pulsation under the sternum and in the epigastrium. 2d, Probably, pulsation in the external jugular veins. 3d, Bruit of a *superficial* character, according to Hope. 4th, The non-transmission of bruit or fremissement along the great arteries of the systemic circulation. 5th, The absence of symptoms of diseased aorta, and of enlargement of the left side of the heart. When chronic disease of the lungs has induced even a considerable degree of dilatation of the right cavities of the heart, it is but seldom that bruit is perceptible. It were needless to dwell at any length upon the different conditions of the system which occasion bruit, while the heart remains healthy in structure. The phenomenon is frequent in hysterical females, in some dyspeptics, and in individuals whose constitutions have been weakened by copious blood-letting, by inanition, by fever, or other protracted illness. Neither does it seem to be of any practical importance to distinguish the different varieties of morbid murmur by such names as " bruit" " de soufflet," " de scie," " de râpe," " du diable," &c., which merely indicate slight differences in the character of the sound, not in its cause. The only variety on which I shall make an observation, is the curious musical sound or " chant des artères." The causes producing it are probably similar to the causes of

the ordinary "bellows murmur," but it is a much rarer phenomenon. One well-marked case was that of an individual treated by Dr. Alison in the Infirmary about 18 months ago, who left the hospital at his own desire. Another man, named Prichard, under the care of Dr. Shortt in the Infirmary, had this symptom formerly, but it has now disappeared, and given place to the ordinary bruit.

Percussion affords physical signs of some value in cases of diseased heart. An unusual extent of dulness in the cardiac region may indicate effusion of fluid into the pericardium, or enlargement of the heart itself. Enlargement does not, however, uniformly occasion dulness in the cardiac region, for an emphysematous lung sometimes overlaps the heart, and by its clear sound on percussion, masks the dull one, which, but for its interposition, would be elicited. Deception may also be caused by a condensation of the pulmonary tissue in the neighbourhood of the heart, which has the double effect of causing dulness on percussion, and of transmitting readily the sound and impulse of the heart.

Bouillaud has observed, that in cases of hypertrophy, the cardiac region sometimes becomes protruded, but it is probably only in extreme cases of what has been termed the "bullock heart," that such a symptom has been noticed. At all events, of a considerable number of patients whom I have

had an opportunity of examining, and who obviously had enlargement of the heart, none have presented that prominence of the cardiac region described by Bouillaud.

In considering the treatment of these affections, little can be said as to means of *cure*. It is scarcely possible to resist the impression, that many reported cases of cures effected when the heart has been much enlarged, are mere instances of mistaken diagnosis. However, Lænnec* has recorded his success in a remarkable case of active aneurism of the heart, by means of a steady perseverance in the treatment of Valsalva and Albertini; and states his conviction, that he has cured at least a dozen more by the same means.

Valvular disease, although distinctly indicated, ought not, according to Lænnec, to prevent the practitioner from pushing the bloodletting and starvation to the utmost limit that nature can bear. In cases of "passive aneurism," he does not recommend bloodletting, but advises the employment of bitter tonics, chalybeates, antispasmodics, and digitalis. Hope is sparing in the use of bloodletting, which he recommends to the extent of from ℥iv to ℥viij at long intervals, in cases of hypertrophy; he strongly advises the use of diu-

* Auscultation Mediate, t. ii. p. 739.

retics, even when no dropsical swellings are present, with the intention of diminishing the quantity of blood in the system, without impairing its nutritive quality. With this view, he prescribes digitalis in preference to other diuretics. M. Bouillaud strongly advocates the employment of the digitalis to allay the inordinate action of the heart, and particularly lauds the endermic method of administering it. There seems reason to believe, that this remedy has been too indiscriminately used in diseases of the heart. Where dilatation and valvular disease exist, its exhibition must often be prejudicial. The indication, in such cases, is to give force to the circulating powers, which is surely more likely to be effected by the removal of local congestions, and employment of tonics, than by inducing the sedative and dangerous effects of digitalis. The hydrocyanic acid has been recommended as a sedative, in cases where the palpitation is violent, particularly by Elliotson, but Lænnec puts little faith in its reputed virtues. The same observations that were made on digitalis, apply with equal force to the employment of this and other powerful sedatives in cases of "passive aneurism." Colchicum, mercurials, and hydragogue cathartics, have been severally recommended by high anthorities. Counter irritation

by means of blisters and issues, has been long the common practice. All authors agree, that the state of the digestive organs must be particularly attended to. A light diet, and abstinence from stimulating liquids is in general indispensable. Flatulence must be guarded against, and the action of the bowels promoted by the use of mild laxatives.

I shall conclude the subject of treatment, with a short sketch of what ought to be done in uncomplicated cases of hypertrophy, and of dilatation.

In a pure case of hypertrophy, the employment of bleeding or cupping in the manner recommended by Hope, of a light and easily digested diet, of laxatives, of diuretics (of which digitalis is perhaps the best), constitutes the chief part of the physician's duty. But much must be done by the patient; he must abstain from violent exertion both of body and mind; avoid exposure to cold and wet, and all those causes which he has previously found to aggravate his uneasy feelings, or to induce palpitation.

In a pure case of dilatation, general bloodletting is improper, except there be manifest plethora of the system, or some inflammatory affection demand its employment. The same attention to the general health, the same prophylactic treatment must be had recourse to here, as in hy-

pertrophy. When it is considered proper to give digitalis, its dangerous effects may be partially counteracted, without any detriment to its diuretic properties, by combining it with some tonic, as the sulphate of quinine, or some preparation of iron. In is needless to add, that anasarca, ascites, pulmonic affections, &c. when they occur in the course of this disease, must be combated by the usual remedies; and as the subject of this dissertation does not embrace the treatment of such affections, it is sufficient to mention their existence, as rendering the prognosis unfavourable.

And now, in conclusion, let us enquire how far treatment is likely to be successful. Many have expressed an opinion, that disease of the heart is beyond the reach of the physician's art, and necessarily fatal; but it is the medical man's duty to use his utmost endeavour, at least to palliate the urgency of symptoms, and where there is hope of success, to attempt a *cure*. There is no reason to despair in uncomplicated cases of hypertrophy, and we have the highest authority for asserting, that in such cases, a cure may sometimes be accomplished.* Where, however, hypertrophy is complicated with dilatation or valvular disease,

* See Page 40, and Auscultation Mediate, loco citato.

our hopes of a cure must of course be greatly diminished; but even in these less tractable cases, the art of the physician may do much—the progress of the disease may, in some instances, be arrested—and, in most, retarded,—the troublesome symptoms may be alleviated—and years often added to the probable life of the patient.

Yet it must not be forgotten, that in every case the chances are greatly against recovery; and while we hope that more efficient means of treatment may be discovered, we must assent to the general proposition, "That of all the "diseases to which the human body is subject, "there are none more unmanageable than the or-"ganic diseases of the Heart."

TABLE OF CASES.

No.	Name.	Age.	Date.	Pulse.	Physical Signs.	General Symptoms.	Post Mortem.
1.	Alexander Campbell.	60.	Nov. 1838.	96, large, irregular.	Cardiac region dull. Double bruit over sternum. Apex below sixth rib. Impulse strong, and felt in Epigastrium, carotids, and subclavians.	Dyspnœa, anasarca, livor.	Fluid in pericardium. General enlargement, but especially hypertrophy of left ventricle. Aorta dilated. Valves of do. slightly diseased.
2.	Margaret Rodney.	50.	Jan. 1838.	Feeble, intermitting.	Apex low. Action of heart diffuse, feeble. First sound flapping.	Pneumonia, anasarca, dyspnœa, &c.	Dilatation of left cavities and of aorta, without thickening. Valves sound.
3.	William Macleod.	40.	Dec. 1838.	Quick, regular.	All the signs of Phthisis. None of Morbus cordis.	Those of Phthisis.	Concentric hypertrophy of left ventricle. Cavity easily restored by pressure.
4.	Robert Clarke.	40.	Nov. 1838.	Irregular and intermitting.	Lower part of chest dull. Impulse diffuse, felt in Epigastrium, and to right of sternum. Jugulars pulsate. Sounds confused. No bruit.	Liver, dyspnœa, bronchitis.	℥xxx. fluid in pericardium. Great dilatation of right auricle. Hypertrophy of the left ventricle—less of the right do. Mitral and Tricuspid valves diseased.
5.	Mary Elmslie.	24.	Nov. 1838.	130, hard, irregular.	Cardiac region dull. First sound rough. Violent impulse.	Cough, pain in cardiac region, dyspnœa.	Pericardium adherent. Old and recent pericarditis. Left ventricle thickened. Left auriculo-ventricular orifice contracted and patulous.
6.	Alexander Galloway.	19.	July 1838.	Strong, jarring, regular.	Cardiac region very dull. Impulse violent, diffuse, felt in Epigastrium. Both sounds accompanied by bruit, loudest over upper sternum. Interval observed.	Rheumatism, pericarditis, dyspnœa, hæmoptysis, palpitation.	℥iv. fluid in pericardium, some adhering lymph. All the cavities, but especially the left ventricle, dilated and hypertrophied. Aortic and mitral valves insufficient. Membrane lining left ventricle white and thickened. Heart weighed ℥xxii.
7.	Jane Slater.	22.	Dec. 1838.	Regular, 90.	Chest unusually clear on percussion. Apex low. Impulse strong in Epigastrium, and veins of neck are very turgid. No bruit. Sounds loud.	Palpitation, dyspnœa, livor, emphysema pulmonum apparently.	℥ij. in pericardium. Both ventricles hypertrophied, particularly the right one, which, as well as right auricle, was dilated. Mitral valve, with excrescences, but sufficient.
8.	George Reid.	38.	Dec. 1838.	Strong, jarring, regular.	Cardiac region dull. Double sawing sound. Impulse very violent below sixth rib, in Epigastrium, carotids, and subclavians. Veins of neck pulsate.	Cough, dyspnœa, slight œdema of feet. Died very suddenly.	Both ventricles dilated, and hypertrophied, especially the left one. Valves of aorta insufficient and puckered. Aorta and large vessels dilated and roughened.
9.	George Dear.	38.	Dec. 1838.	Large, regular, jarring.	Dulness in cardiac region. Impulse strong. Apex obscure. Some interval. Second sound with faint bruit. First sound short and flapping.	Rheumatism, dyspnœa, cough, anasarca.	Pericardium contained much fluid. Heart, particularly left ventricle, much dilated, and moderately hypertrophied. Aorta rough, its valves insufficient. Mitral valve rough. Liver large.
10.	Mary Ainslie.	50.	March 1839.	Irregular, about 100.	Cardiac dulness. Bruit and fremissement with first sound. Impulse diffuse, rather violent in carotids and subclavians.	Dropsy, livor, cough, and bloody sputa. Died very suddenly.	℥ij. fluid in pericardium. Ventricles dilated, the left also hypertrophied. Aortic valves insufficient, but slightly so. Left auriculo-ventricular orifice contracted.
11.	James Prichard.	32.	Jan. 1839.	60, large, jarring.	Apex low. Cardiac dulness. Some interval. See-saw bruit over the aortic valves. Pulsation strong in cardiac region, and in great vessels.	Old rheumatism, dropsy, dyspnœa. Died very suddenly.	Heart of enormous size, all the cavities being dilated and hypertrophied. Aorta rough and dilated. Valves of aorta permitted much regurgitation.
12.	John Stewart.	36. ?	June 1839.	Large, jarring, regular.	Extensive cardiac dulness. Apex very low. Much and diffuse impulse, and fremissement. Carotids and subclavians pulsate very violently. Interval. See-saw bruit over sternum.	Œdema, dyspnœa, morbus renum. Died gradually exhausted.	℥viii. in pericardium. Heart enormous. Right side dilated, the ventricle thickened. Left ventricle much dilated and thickened. Aortic valves puckered and insufficient.
13.	Margaret Walker.	13.	April 1839.	130, strong, regular.	Dulness over heart. Action violent, tumultuous. Frottement very distinct. Bruit with first sound over aortic valves.	Pericarditis, cough, dyspnœa, pain in cardiac region.	Old and recent pericarditis. ℥iv. of fluid in pericardium. Both ventricles dilated, and the left hypertrophied. Mitral valve thickened. Aortic orifice small.
14.	Lydia Maslind.	28.	April 1839.	Large, strong, regular.	Dulness. Apex low. Double bruit over upper sternum. Impulse diffuse, violent, felt in Epigastrium. An interval observed.	Old rheumatism, extreme dyspnœa, occipital headache, œdema. Died suddenly.	Dilatation of all the cavities. Right do. hypertrophied. Aorta rough. Aortic valves rough and insufficient.
15.	John Hodge.	41.	May 1839.	Irregular in force.	Interval readily observed. Pulsation felt in Epigastrium. Much dulness over heart. Double bruit over sternum. Impulse moderate.	Dyspnœa, livor, anasarca, faintings, palpitation. Died rather suddenly by asphyxia.	Aneurism of ascending aorta of small size, communicating with pulmonary artery. Insufficiency of aortic valves. Right ventricle hypertrophied and dilated. Right auricle and left ventricle dilated, and slightly thickened. ℥iiij. in pericardium.