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OBSERVATIONS

ON THE

CLINICAL HISTORY AND PATHOLOGY

OF ONE FORM OF

FATTY DEGENERATION OF THE HEART:

Being the Substance of a Paper read before the Brighton and Sussex Medico-Chirurgical Society, Oct. 4, 1849.

BY

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PART I.

Of fatty degeneration in general—primary and secondary. Fatty disease of the heart—writers on the subject—of three kinds—illustration of the second kind. The third kind the subject of this paper—its anatomical characters. Fatty degeneration a specific form of disease—its clinical history.

Fatty degeneration of the heart as a cause of sudden death—the compatibility of extensive disease with apparent health—manner of death—death by coagulation of the blood in the pulmonary artery; difference of the symptoms of, illustrated. Old coagula in the heart, connected with capillary phlebitis, only coincident with fatty degeneration of the heart. Death from valvular or fatty disease of the heart; points of resemblance between.

THERE are few subjects which have engaged the attention of pathologists of late years so much as fatty degeneration. It is a field in which the microscope has done good service in the hands of Mr. Gulliver, Mr. Bowman, Mr. Rainey, and Dr. Johnson, with many others. Mr. Paget has expressed so well the general principles of the process, in his Lectures on Atrophy (MED. GAZ., 1847, Lecture V. and VI.), as to render any lengthened exposition of them here quite superfluous. It will be sufficient merely to note the chief outlines, referring for further details to those elegant and instructive lectures to which the author of this paper must express his great obligations.

Fatty degeneration may occur under two conditions—viz. as a primary or a secondary affection. Primarily, it may occur in a part which is no longer wanted, as in the muscles of aged or crippled limbs, and in the structure of crippled organs. Such a change we see best in the bodies of some old people. The parts which are no longer wanted are not simply atrophied, but converted gradually into fat, and the blood apparently is so overloaded with this ingredient that it fails to remove the fat from those parts which are in earlier years naturally padded or distended with this substance.* So the body retains, in many respects, the form and plumpness of youth, but the texture is flabby, and the colour pale, for the want of blood and muscle to give it the hue and firmness of youthful life.

The other condition frequently connected with this peculiar form of atrophy, which may then be called secondary, is the previous occurrence of chronic inflammation in this part. Here, as in the former case, we must look to the general habit of the individual for some share of the explanation; but the fact of the one being occasionally ingrafted on the effects, or accompanying the progress of the other, rests on the best evidence. This is most commonly seen, as might be expected, in internal organs. Probably the same cause which has induced inflammation has itself, in a very large majority of cases, given the tendency to fatty degeneration of the particular organs.

Of the nature of this fatty degeneration, under whatever circumstances, there can be no doubt: it is essentially an atrophy, whereby the most highly organized elements of the body are replaced by one of the simplest. It is not that the substances are changed into fat, that is plainly impossible, but that the nutritive processes of muscle, or gland, or bone, no longer restore particle for particle whatever is lost by the daily use of the parts, but replace it with oil. Nutrition goes on in some sort, but the additions are of matters wholly unsuited to the office of those that they have replaced: they can neither move nor secrete, nor even mechanically support the weight of the body.

It is not proposed in the following pages to discuss the entire subject of fatty disease of the heart in general; for there is little to be added to what we already know of many parts of it. The records of medicine abound with instances of apparent conversion of more or less of the substance of the heart into adipose tissue; instances more striking in themselves, and better recorded, than any which the authorcould adduce.

The clinical history, too, of this class of cases, appears to be perfect; we know where to expect such degeneration, and in what way to dread its consequences; and if we have ceased to look for more exactly pathognomonic signs of its existence, it is because we know that it is not in the nature of the disease that there should be such.

* See Mr. R. W. Smith's two cases. Dublin Quarterly Journal, Vol. ix. p. 413.

The subject of the following remarks is, therefore, one form only of fatty degeneration, which, after some pains in the inquiry, I am content to believe is, if not exactly, as Rokitansky says, a form as yet unnoticed, at least a form which he was the first correctly to describe.

In the writings of Morgagni there are accounts of several fat hearts, but as the large size and abundant deposit of fat are the points particularly dwelt upon, these cases obviously will not do for the present purpose; for an excess of adipose tissue is far from being a common accompaniment of the particular form under consideration. The case detailed in Epist. xlv. §. 23, is the only one which I have met with, in looking through his great work, which seems with any high probability thus explicable. But the case is too long for insertion on the grounds only of probability.

It is not under the head of fatty degeneration of the heart that we are to expect to find illustrations of the present subject. Fat there is, but not of a nature to be detected by the unassisted eye. Much more might be expected from the examination of recorded cases of softening or discolouration of the heart. These were conditions familiar to Laennec, and well described by him. But the microscope was not then in daily use, as at present, and, without the microscope, this form of disease could scarcely have been separated from that form which has below been described as the second, and for whose accurate description we are indebted to Laennec. As a general expression, nothing could be more correct than Laennec's remarks (Auscultation Médiate, Tome iii. p. 223), where, after combating the notion that softening of the heart was necessarily an inflammatory condition, and to be treated by antiphlogistic means, he says—"To me softening of the heart appears an affection *sui generis*, the result of a disturbance of nutrition, through which the solid constituents of a tissue are diminished in proportion as the fluid or half-fluid constituents are increased." But he failed to perceive the whole connection between softening and fatty degeneration of the heart; for he says further on (op. cit. p. 226)—"There is in this case no evident perversion of nutrition, because there is no accidental [*i. e.* adventitious] product," which is contrary to the actual state of things under consideration.

The great French pathologists are not exactly agreed with Laennec; but their differences of opinion are not material to the present subject. Dr. Hope (On Diseases of the Heart, p. 332, third edition) follows Laennec's classification of white, yellow, and red softening, without connecting these conditions—or rather the second of them—with fatty degeneration. Dr. Joy (Lib. Pract. Med. Vol. iii. p. 365) expresses very plainly the possible connection between fatty degeneration and general softening of the heart, and speaks of them as probably explanatory of many a sudden death by syncope. Dr. Copland (Dict. Pract. Med. Vol. ii. p. 227) has given a very full bibliography of this subject; and a clear, concise statement of all which was at that period known of it will be found at page 216 of the same work. Little could be added to his description of what there will be occasion to speak of as the second form of fatty disease of the heart. But, upon a careful examination of several of the authorities referred to, there do not appear to be any cases certainly referable to the third form, the immediate subject of this paper. One case only by Mr. Adams (Dublin Hospital Reports, Vol. vi. p. 396) appears to belong here; for, besides the fat which had displaced the muscular tissue on the outside of the heart, "in both ventricles, even in the lining fibres, yellow spots, where fat had occupied the place of muscular structure, were to be observed. The muscular structure was soft and easily torn, and a section of it exhibited more the appearance of liver than of a heart" (Op. cit. pp. 398-9). But the clear, accurate manner in which this and other of the cases are detailed, almost forbids one to put any other interpretation upon them than what their authors have expressed.

In the absence, therefore, of any other cases which may with perfect certainty be quoted as the basis of an analysis, it appeared the best way to detail at length such as my own experience affords, rather than to adduce merely the inferences from them; though the security against error, or the means of correcting it, is purchased at the expense of great prolixity. I have only to observe on this subject, that my opinion as to the nature and importance of the disease rests on the facts placed before the reader, and that it is not to impress the importance of the change in every case, that every case has been adduced. Rather, seeing under what great variety of circumstances it may be found, I should hope that the same observations would lead him to the same conclusions as myself on the points hereafter to be investigated.

There are three forms in which fatty disease of the heart may occur:—

1st. In the first form the fat is accumulated in those parts where it is naturally deposited in the greatest abundance, as at the base of the heart. This is met with in those persons who have a general tendency to accumulate fat; and to this form, probably, most of what are called fat hearts belong.

It is certainly a condition of disease, but probably not an important one, and needs no additional illustration here.

2d. In the next form the fat collects chiefly about the apex of the heart (Laennec, Auscult. Médiate, Tome iii. p. 225). The deposit begins from without, and is not attended with so great an enlargement of the heart as in the form first described; for the fat is deposited at the expense of the muscular tissue—in its place, not merely laid upon it. Moreover, the degeneration is partial, and in patches, not general. This tendency to limit itself to particular spots is seen most characteristically in those cases where a single carnea columna is converted into adipose tissue, but still preserves the form of the muscular band whose place it has usurped. This form occurs in persons who have, as is popularly said, an unhealthy disposition to grow fat; in those where the fat has generally throughout the body, as in the heart, a tendency to accumulate in the place of, not in addition to, the proper structures. The disease is, in some sense, a degeneration in this form, but not perhaps so strictly as in the form remaining to be described, the proper subject of these remarks. To this second form belong nearly all the cases, as far as I am able to judge, described as examples of fatty disease of the heart. This fact in itself bespeaks the much greater importance of the second than of the first form; for there the change in the heart is commonly described as a morbid appearance among many others; here it is the change on which the interest of the case is made to turn.

Dr. Cheyne, and other Dublin practitioners, among whom the disease has excited a good deal of interest, have recorded cases of this description. But instead of repeating one of their well-known cases, the following, for which I am indebted to my friend Dr. Kirkes, will serve as an excellent illustration:—

Sudden death—fatty degeneration of the heart.

A German clock-maker, a powerful, well-built man, applied for admission at St. Bartholomew's Hospital, in August, 1847, and he looked so pale and ill that the only empty bed in the house was given him, though a fever patient had to be refused admission.

His friends said that he had always had remarkably good health, and that he was a man of great strength and powers of endurance. But within the last year he had been subject to occasional fits, during which he lost consciousness, fell, and struggled for a short while. He had one such fit ten days ago. His face was now pale; his expression languid; his gait tottering; pulse thrilling, occasionally irregular. He said he had no uneasiness in the chest, only he had wandering pains, with a sense of fainting and debility.

He was put to bed, and lay quiet and unnoticed that evening and night. Early on the following morning the nurse found him quite dead in his bed.

On examination of the body thirty-six hours after death,

The limbs were still rather rigid. A large quantity of fluid blood escaped from the divided cerebral vessels; the brain itself was healthy. Nothing particular was observed in the lungs, or in the abdominal viscera.

The heart was rather larger than natural: it was marked with two "white patches" externally; it felt like a loose bag, its walls being quite flaccid, and it was already partly decomposed, its walls interiorly being stained by the dark grumous blood which it contained. The right side of the heart was of the natural size, though the auricle was just then distended with blood. The left cavities were smaller than natural. On close examination, the heart appeared as if entirely overlaid with a layer of dirty yellowish fat, separated by a distinct boundary from the muscular substance of the organ; but the muscular tissue itself was pale, like dead leaves, contrasting forcibly with the bright red colour of his abdominal muscles.

The rapid decomposition which a portion of the heart set apart for the purpose underwent, vitiates any conclusions from the microscopic examination. A piece of abdominal muscle, from the same subject, had, in the same time, undergone no change.*

For the present, let this brief summary and this single example suffice for this very common yet very terrible form of the disease. Regarding it as the standard and recognised form, as it were, of fatty degeneration of the heart, it will be better to return to it hereafter, when the full exposi-

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* I am indebted to my brother for the particulars of the following case, which, however, want of microscopic examination prevents my numbering among the undoubted examples of this affection:—

A gentleman of about 40 years of age had always been fat and indolent. He bore the appearance of indolence to such a degree, that, as he walked along the streets, he used to be made the subject of practical jokes on that account, and to exertion of any kind he seemed to have an innate repugnance.

For the last few years of his life he had been resident in Australia. I am not not aware that he had been previously ill, he certainly had suffered no pains in the region of the heart, when one day he was taken as it were with the symptoms of death, and so died in about a quarter of an hour as he was moving about the room.

Nothing was found on examination of the body to explain these symptoms but the existence of a small fat heart.

tion of the characters of the form more immediately under consideration has supplied the means of comparing the two.

The third form of disease was first, as far as I am aware, described by Rokitansky, who speaks of it as affecting the heart by preference; then, next in order of frequency, the voluntary muscles, and sometimes, though very rarely, the involuntary muscles,—as, for instance, the coats of the gall-bladder.* The subject has been also investigated by Mr. Paget† with his usual ability. To his description of the morbid appearances I have nothing to add, and have only to offer my humble confirmation of the correctness of his remark that the fatty matter is partly within, and not wholly external to, the sarcolemma, as described by Rokitansky. But I cannot omit to acknowledge the extreme pleasure and advantage I have derived from the assistance of Mr. Paget, as well on the occasion of working out this part of the subject, as always on other occasions, whenever the direction of his pathological researches has happily coincided with mine.

To the unassisted eye, the muscular substance of a healthy heart presents characters distinguishing it from ordinary muscular tissue; for it is more compact and homogeneous, and not loosely divided into bundles of fibres, as is ordinary muscle. Under the microscope it also presents some striking differences, the transverse striæ being less distinctly marked, and the fibres having a singular granular appearance. It is very important to notice this normal difference at the outset; for the first step towards fatty degeneration consists in the loss of continuity of the transverse striæ, and in the increase of this granular marking of the fibres, which would seem to be in some degree their normal appearance.

This is the first step, and, as wholly undiscoverable by the naked eye, may often pass unnoticed, unless something in the symptoms, or some change in the general condition of the heart, call particular attention to that organ. Such conditions may be, a small, pale, flabby state of the heart, not inaptly compared to the colour of withered leaves, and to the feel of a moist glove. But such are not commonly the signs which call attention to the existence of this structural change: they are ordinarily much more obvious.

On opening a heart thus affected, the interior of the ventricles appears to be mottled over with buff-coloured spots of a singular zigzag form. The same may be noticed beneath the pericardium also; and in extreme cases the same appearance is found, on section, to pervade the whole thickness of the walls of the ventricles and of the carneæ columnæ. Of these latter the *musculi papillares* seem most liable to be affected. Not to say that this form of disease never occurs in the walls of the auricles,—at least, I have never seen it there.

Microscopic examination reveals the nature of these spots: they are not deposits, but distinctly degenerated muscular fibres; the outline, not merely of the masses, but of each single fibril, is accurately preserved. Instead, however, of transverse striæ and nuclei—the evidences of active vitality—there is little to be seen but a congeries of oil-globules. The whole history of the degeneration may be traced in one of these little spots. First, from the immediate neighbourhood of the spot we may obtain healthy muscular fibre; then the transverse

Fig. 1.

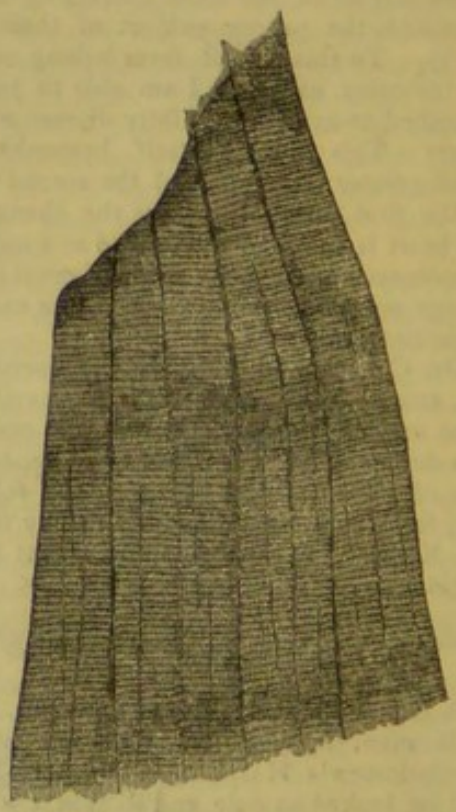

Fig. 1.—Healthy muscular fibres of the heart.

striæ become less distinct; they are rows of dots rather than continuous lines; then the intervals between the dots become wider, and the dots themselves run into longitudinal rather than transverse lines; and then all regularity is lost, and the dots appear to stud the surface all over, like the points on a bit of fish-skin. Probably long before this time the fibre has lost all its properties

* Rokitansky. Anat. Path. Bd. II. ss. 360, 459, III. s. 368; Budd, Dis. of the Liver, p. 191.

† Lectures on Nutrition, &c., London Med. Gaz. May 1847, Lect. VI.

Fig. 2.

Fig. 2.—Granular degeneration, with commencing fatty degeneration.

as a muscle: but there are further changes to observe; for now, mixed with these minute dots, are to be seen small oil-globules, which increase and coalesce till the fibril presents little else but a congeries of oil-drops contained within the sarcolemma.

Fig. 3.

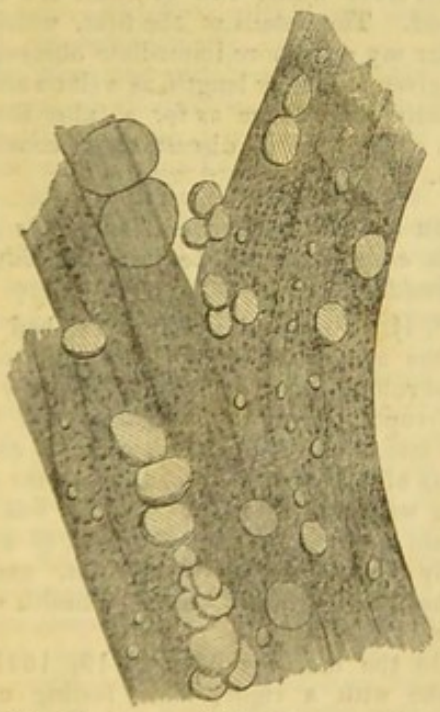

Fig. 3.—Extreme fatty degeneration; the fibres cleared by acetic acid.

This is not the only change which the fibres undergo; for, with whatever care they are disintegrated, they are found to be short, and as if unusually brittle,—a general condition which may, perhaps, be of more serious importance than the actual fatty degeneration of the organ.

Such are the most common features of the disease, and sufficiently obvious, when once noticed, to prevent their being readily overlooked afterwards. But we must not rely too exclusively upon them; for, as already observed, in the absence of these little spots marking the extreme degree of fatty degeneration in single points, the disease may have pervaded the whole substance of the heart; and the recognition of such a change will be difficult in exact proportion to its extent, and therefore its importance, from the want of healthy tissue wherewith to contrast the diseased fibres. And there is no solution for the difficulty except in the use of the microscope, whose information, should anything casually induce us to solicit it, on this subject at least is infallible.

It is perhaps almost needless to provide against such a misinterpretation as that this should be presumed to be an ordinary change of the heart. The following, however, has been chosen, in this view, as an illustration of what may be called fibrous degeneration of the heart, where, mixed with the atrophied muscular tissue, are the products of previous inflammation.

Angina pectoris—Sudden death—Aneurism of the left ventricle—fibrous degeneration of the heart.

A clergyman, aged 45, of sedentary studious habits, inclined to grow fat, though abstemious to a degree, for the last two years of his life was subject to paroxysms of pain in the region of the heart. The first paroxysm occurred on occasion of some mental anxiety; then he used to suffer them about once a month, and latterly almost daily.

There was no valvular murmur, only the heart's sounds were loud and ringing. He found more relief from constant mental employment, and from the practice of abstinence to a degree which most would call starvation, than from anything else. One morning he was found dead by the side of his bed.

On examination of the body after death, with Dr. Jeaffreson and Mr. R. Eyles, besides a few slight morbid changes irrelevant to the present subject,—

The heart was found large, weighing ℥xij.; there was a slight degree of general vascular injection of the pericardium, which contained about ℥ij. of clear fluid. The right side was healthy. The left valves were efficient, but thickened. The walls of the left auricle were thickened: the left

ventricle was rounded at the apex, which projected far beyond the right. In a space of about an inch in diameter, on the front of this ventricle, the walls were very thin, and could easily be made to bulge. The substance of the heart here was injected, but tough, and chiefly made up of cellular tissue. The carneæ columnæ hereabouts, and those leading to the right side of Lieutaud's valve, were pale and white, being composed in chief part of fibrous tissue, the fibres being long, straight, and parallel, mixed with only a few proper muscular fibrils. Elsewhere the fibres of the heart were long, straight, well coloured, and the transverse striæ well marked. There was no fatty degeneration anywhere.

The aorta was thick and uneven, with a few calcareous plates, and with the lining membrane removed in a few places.

In many respects, then, the anatomical history of fatty degeneration of the heart appears to be complete. We can trace it from its earliest beginnings to the entire disintegration of the muscular structure. It is a specific change with as distinctly marked characters as have any of those diseases of which we have cognizance by the eye or ear—tubercle or pneumonia, for instance. There are some points, however, which observation has yet to supply; for there are ulterior changes in the muscular structure of the heart—perforations, rupture, and apparent loss of substance,—which experience has not yet shown to be strictly the results of fatty degeneration, however probable the general circumstances may have made it that such will be found to be the case on more extended inquiry.*

The clinical history, however, on which these particular subjects trench has not yet been traced. It is in the attempt to elucidate this that the following cases have been detailed. There are two great points to be made out: first, the degree of importance to be attached to the disease; and, second, the causes on which it may be supposed to depend. The latter can only be investigated by an analysis, and it would be prejudging the question to use its presumed results as the basis of any classification. The former being, in our present state of knowledge of the subject at least, to be measured by the influence of the affection for life or death, it has appeared best to adopt this as a means of classification of the cases, which on this matter may well speak for themselves. There are, then, two great divisions, in one of which death has seemed more or less directly referable to this change in the structure of the heart; while in the other this structural change has taken place without the occurrence of any symptom referable thereto. This second larger division appears, for facility of reference, most properly to be again divisible into different sections, according as different organs have been affected coincidently with this structural change of the heart. From the more frequent coincidence of some of these diseases, it is hard to resist the conclusion that the connection is more than that of coincidence. For the digressions which occasionally interrupt the regular course of the narrative, I trust it is needless to offer any lengthened apology. Where not absolutely necessary to guard against any fallacy from misinterpretation of the particular case, the importance of their subject must be the plea for their admission.

Of the first class of cases—those, namely, where death has seemed to result more or less directly from this lesion of the heart,—there are six. The three first have already been published by Mr. Paget in his Lectures, from which two of them are transcribed. The details of the first, which was under my own more immediate observation, are given at greater length, as well on account of their importance as for a fuller illustration of the form of disease under consideration.

CASE I.—*Fatty degeneration of the heart in an apparently healthy individual—Sudden death.*

P. H., a medical practitioner, had lived to the age of 56 in the enjoyment of the most robust health, which had scarcely been interrupted, except by two attacks of fever, and latterly by the passage of renal calculi. Feats of strength and endurance for other men were his amusements. He was temperate and regular in his habits, of a most lively and cheerful disposition, and he looked the picture of the good health which he enjoyed.

On the morning of Feb. 19, 1847, he awoke with a rigor, and, feeling unwell during the ensuing day, he took some purgative medicine, and applied a few leeches to his head, on account of an uncomfortable feeling which he had there. He was a good

* On the controverted subject of the connection of rupture of the heart with the commonly known form of fatty degeneration, this would not be the place to speak had I anything to add to what is already known. References to this subject will be found in Bouillaud (des Maladies du Cœur, T. ii. p. 633), and Dr. Stroud's work (On the Physical Cause, &c. *passim*), as well as in all the common sources of information. My own very limited experience is entirely negative as concerns the form at present under examination; for in the only two cases of rupture of the heart which I have had occasion to examine this peculiar form did not occur. But the question waits further investigation in these two particulars,—Can rupture occur in a healthy heart, independent of external violence? and what is the essential nature of the disease which allows of such an occurrence?

deal sxhausted by the action of these remedies, and his friends, becoming alarmed, asked Dr. Burrows to see him on Feb. 20.

On this day he was very weak and low-spirited, breathing slowly, and sighing occasionally. He had pain and a little redness of the throat, and he himself apprehended that he had caught erysipelas of the fauces. Under the influence of a little wine and quinine he appeared better the next day; his pulse fell to 70, his spirits returned, but his weakness remained excessive. However, on the evening of Feb. 21, he rose and sat by the fire, talking cheerfully for some time. He had three or four hours' sound sleep during the night, and the improvement of the previous day was continued on the morning of Feb. 22.

About half-past 11 A.M. of this day his wife had left the room to seal a letter which she had been writing under his direction, and his servant had just finished straightening his bed, that he might sleep, when all of a sudden he pressed his head back on the pillow, his tongue slightly protruded from his mouth, his respiration became slow, and his face remained fixed. From the first his pulse had been imperceptible, and the heart's sounds were quite inaudible when examined within two minutes from that period. A little brandy was poured down his throat, but to no purpose: in less than five minutes he was dead.

The body was examined, with the utmost care, twenty-one hours and a half after death: it was still warm; the limbs were rigid; the blood was universally fluid.

The scalp was loaded with blood; the brain was generally rather softer than natural, otherwise healthy.

The posterior surface of the trunk and arms was of an intense purple, this colour terminating at an irregularly curved line which ran along the side of the thorax. The abdominal and thoracic integuments were much loaded with fat; the costal cartilages were firmly ossified. The pleuræ were free; the lungs healthy, save a slight degree of general emphysema, and a little œdema in parts.

The pericardium contained about one ounce of clear fluid. There was a "white patch" on the front of the right ventricle. The heart contained very little blood: as compared with the other organs it was small, and much fat was visible on its surface. There was a little opaque deposit in the substance of the mitral valve, and a little of the same round the attached edges of the aortic; but clearly the valves were efficient. The walls were pale, buff-coloured, and flabby; on the right they were very thin; on the left some patches of a more distinct buff colour could be seen, but no zigzag markings were seen anywhere.

Compared with a healthy heart the muscular structure appeared very pale, loose, and flabby, like wet buff leather. Under the microscope it appeared to be made up of—

1. Short broken fibres, with very indistinct transverse striæ, and beset with longitudinal rows of dots, some of which were longer than others, and some, especially towards the middle of the fibril, distinctly ascertained to be oil globules.

2. Masses of fat cells, and single fat cells intruding upon the muscular fibres.

3. Oil globules, floating loose, in great abundance.

The stomach was intensely injected about the cardiac end, elsewhere it was healthy. The lower end of the ileum contained four or five longitudinal ridges, of a pale brownish colour, bounding a fissure where the mucous membrane appeared to be wanting. The solitary glands were generally slightly enlarged; the mesenteric glands not notably so. The cæcum was healthy.

The liver was large; the gall-bladder contained two round, roughly polished calculi. The spleen was large, dark, and rotten. The kidneys were healthy, save a little injection of their pelves.

To express the care with which each organ of the body was examined, it is only necessary to say that the examination was made with a suspicion of the possibility that poison had been taken. But beyond the intense congestion of a patch of the mucous membrane of the stomach, there was no evidence of the local action of any poison; and this patch there can be little doubt arose from the brandy which was poured down his throat during the last few moments of life.

The disease under which he was suffering when he so suddenly died was supposed by himself to be an erysipelatous sore-throat, which was at that time prevalent. Generally, he was supposed to be in the early stage of continued fever. This supposition, however, furnishes no adequate explanation of the suddenness of his death,—a death so wholly unlike that which in rare instances cuts off patients in the first onset of the fevers of this country. It was not as if he had then first felt the attack of fever; this had taken place some days before, and he was rallying from it. There was no coma; nothing like slow poisoning of the blood; no appearance of struggling for life; the heart at once ceased to beat, and he was dead. It is open to question how far the morbid changes in the spleen and the ileum were due to the present or the previous attacks of fever respectively; but there can be no question that these were wholly inadequate to cause death at such a time and in such a manner.

Indeed, the explanation of the suddenness of the death, and of its peculiar mode, does not lie on the surface. The question, why did he die just then, is unanswerable, except by means of an assumption that the state of previous disease taxed the powers of the heart more than any mere physical exertion could do. Perhaps this is the correct explanation, connecting the failure of the heart *about* that time with the antecedent illness. The certain explanation must remain here, as, indeed, in nearly all other cases of death, from whatever cause, unattainable.

It is almost needless to do more than refer to the case of Lord George Bentinck, in so many points analogous to the above. The general features of the case are fresh in the minds of all, and their particular application to the present subject has been made by Mr. Corfe, in a paper published in the *Medical Times* (Dec. 9-16, 1848, pp. 142-157).

The real difficulty, however, does not appear to be so much why did the heart cease then, as why did it not cease long before. How could such a state of the most important organ of the body be compatible with apparent health? Probably there are few who will hesitate to place the anomaly in the moral—if I may use the expression—rather than in the physical heart of the individual. As physiologists, we do not need to be reminded of the almost inconceivable force of muscular contraction, to be satisfied that a very small number of fibres* will suffice for the labour which is ordinarily performed by a large mass, if so be only that their energy is proportionably increased. As physiologists, too, we may find many instances where an intensity of purpose has supplied such energy, and sustained the vital powers to the completion of that purpose, and where, with its cessation, life also has ceased. And this, not in man only, but in dumb animals who have died to do his pleasure or fulfil his wants. And the records of our own profession, the experience of each one of us, could supply countless instances more pleasing than these, where an unseen power from within has enabled feeble dying bodies to sustain great and long continued exertions, to which their physical powers have appeared quite inadequate.

Such—namely, increase of energy of the muscular fibres of the heart, to compensate for the inefficiency of part, or the increased demand on the whole—appears to be the true principle on which life continued, and even enjoyed, under these circumstances, is to be explained. And the principle is of common application, and to other diseases of the heart besides that at present under consideration.

The two following cases from Mr. Paget's Lectures (Med. Gaz. 1847, Lecture VI.), will illustrate this most important and terrible feature of the disease, the liability to induce sudden death. Mr. Paget says:—

"Case II.—I was requested to assist in the examination of the body of a woman, about 60 years old, who was wrongly supposed to have been killed by an overdose of morphia, given for the cure of some spasmodic pains to which she was subject. She had been a healthy person except for these pains, and except that for two or three years she had been growing fat. On the night of her death she took half a grain of acetate of morphia, which was twice as much as she was used to, but it did not produce deep sleep, and there was certain evidence that four hours after taking it she was awake, and talked sensibly with those around her; but after thus talking she went to sleep, and three hours afterwards was found dead. She had died in her sleep. On examination we found nothing whatever to account for death, except this fatty degeneration of the heart."

"Case III.—In the next case, a strong man, between 30 and 40 years old, addicted to very hard drinking, received a slight injury of the head. He paid no attention to it, and continued his work for four days; then, having headache, and feeling ill, he laid up, and left off all strong drink. In three days signs of delirium tremens commenced, and he was brought to the hospital. In the course of the second day after the beginning of these symptoms, while they were pursuing an ordinary course, he took 140 drops of laudanum, but no beer or spirits till in the evening, when, the opium seeming to have been sufficiently administered, brandy was given him and beef-tea. In about two hours he went to sleep, and he remained dozing and apparently improving in condition all night. But in the morning a new nurse came to him, who wished to change his bed-linen. For this purpose she took him out of bed, and sat him in a chair; but he had hardly been removed before he appeared to be dying, and he died before he could again be placed in bed. In his body the only changes were fatty degeneration of the heart and liver."

Mr. Paget adds—"Here, then, were three cases of sudden death, all the result of a similar disease of the heart, the existence of which had been completely unsuspected, and in all probability, at least in Case I., had not been attended with any signs that could

* Cruveilhier, Essai sur l'Anatomie Pathologique, tome i. p. 186, says that in a portion of muscle appearing to the naked eye totally converted into fat, and weighing thirteen drachms, one and a half drachms were composed of muscle, four grains of gelatin, all the rest of fat. Cyclop. Anat. Phys. Dr. Walshe, vol. iv. p. 96.

have attracted even scrupulous attention. The condition of the heart was alike in all, and very characteristic, though, indeed, to an unpractised eye it might seem unimportant."

Before detailing the three remaining cases of this division there is one point to be guarded. They are not put here simply because they died suddenly, and there was found fatty degeneration of their hearts; but because this sudden death was explicable on no other grounds, taking all things into consideration, than failure of the heart's action. And in their hearts was found what may go some way towards explaining that failure.

From the nature of the cases, coagulation of the blood in the pulmonary arteries might appear a very possible cause of so sudden death. The mode of death, however, was not exactly similar to what occurs under such circumstances, and, yet better evidence, no such coagula were found there after death. The point, however, is of such importance, of all the obscure causes of sudden death to determine which has happened in any particular case, that the following details will not be irrelevant. The case selected is the one which displays the symptoms of death from coagulation of the blood in the pulmonary artery in a higher degree than any other which my collection affords.

Paraplegia from disease of the vertebræ—Sudden death from coagulation of the blood in the pulmonary arteries.

Sarah Dillon, aged 20, Hope back ward, February 1847. A healthy-looking girl, of a fair complexion, who had been in the hospital a few weeks before with some anomalous symptoms which were not exactly made out at the time, was re-admitted with the same symptoms, and with great weakness of the lower extremities. She had complained at first of a feeling as if a bandage were drawn tightly round the lower part of the chest, and when, on her re-admission, her weakness drew particular attention to her spine, it was found that the middle dorsal vertebræ projected posteriorly, and were tender on pressure.

The notes of her case, extending from Dec. 18, 1847, to March 6, 1848, speak of little but constantly increasing weakness. By the end of January she had lost all power over her legs. On Feb. 9 she had very little, if any, sensibility of the feet, though pinching of the foot was immediately followed by retraction of the legs. Indeed, it was a great trouble to her that her legs were always doubling themselves up, and that she had to ask the nurse to come and straighten them. She had great difficulty in evacuating her bladder, or controlling the action of the bowels. On Feb. 19 it is noted that all her evacuations passed away unconsciously; she dreads being moved, and all her appearance of cheerfulness and health has passed away.

March 6.—She has been steadily getting worse, though she always says she is better; but her face is dusky, and her expression anxious. Last night she had a profuse perspiration. This morning she was quite easy, and unusually cheerful, while she was having her bed straightened. About a quarter of an hour after having been changed, she cried out suddenly, "Oh my breath!" and seemed much distressed, but was revived by a little ammonia. In a little while she seemed again to be sinking; complained of pain in the right arm, and kept swaying it in the air. She grew livid, and the sweat streamed down her; and still she kept crying out at intervals "Oh my breath!" till she died, in about twenty minutes from the time that she first called the sister's attention to "the queer feeling in her throat."

On examination of the body sixty hours after death, the limbs were still rather rigid; the blood was nearly all fluid. The brain and spinal cord were healthy, except the dorsal region of the latter, where it was closely pressed upon in front by a sharp ridge, over which it bent. This ridge was produced by the projection backwards of the intervertebral ligaments between the sixth, seventh, and eighth dorsal vertebræ. The body of the seventh was wholly, and that of the eighth partially, destroyed. There was about one ounce of pus in the right mass of dorsal muscles, in a cell, with smooth polished walls, about half an inch from the surface. This abscess was not evidently connected with diseased bone, but it passed between the spinous processes of the seventh and eighth dorsal vertebræ.

The heart was healthy; there were about two ounces of fluid in the pericardium.

The pleuræ were free, with four or five ounces of fluid in each cavity. A single, small, chalky tubercle existed in the apex of the left lung; otherwise the lungs appeared quite healthy. A large cylindrical coagulum, which had fallen out on the table, first called attention to the pulmonary arteries, which were all found more or less closely plugged by similar concretions. In the large trunks these coagula were hard, wrinkled, and of a dirty reddish-grey colour, but in the ramifications of the third order they were smooth, soft, and black. They were all free from adhesions to the coats of the vessels.

It would be out of place to digress any further on the various points of interest which this case presents, apart from that in illustration of which it has been adduced.

Nor on this need any more be said here,* than that whatever other fallacies may have interfered in the following cases, this at least has had no share in their misinterpretation.

CASE IV.—*Anasarca — Ascites — Sudden death with fatty degeneration of the heart.*

James Taylor, aged 34. Matthew front ward, October 1846. A plasterer's moulder, a singularly pale man, with dark hair, of notoriously intemperate habits, and having suffered from jaundice six months previously, was admitted about a month before his death with anasarca and ascites, and in a state of great debility.

It does not appear that much notice was taken of him, and I could not make out that his urine had ever been examined for albumen during his stay in the hospital; the only points which attracted attention being the occasional occurrence of diarrhœa, and latterly of vomiting. He sank gradually, always complaining of pains in the front of his chest. One day he complained much of a choking sensation, of which he tried to relieve himself by passing his finger down his throat. He then went to the water-closet, and was led back quietly to bed, where, half an hour afterwards, he was found sitting up in the bed-chair as usual, quite dead.

On examination of the body after death, the lungs were found very œdematous, and the bronchi full of froth. There were about fifteen ounces of reddish turbid serum in each pleura.

The heart was large, of a pale colour; the valves were healthy. The interior of both ventricles, and especially of the left, was studded with little zigzag lines of a buff colour, extending a tenth of an inch or more into the substance of the organ. Under the microscope these buff spots appeared to be composed of oil globules; in their immediate vicinity the muscular fibres were represented by granular cords, and at a little further distance the granules were placed in such order as to constitute striæ. Throughout, the fibres were short, and had a granular aspect.

The peritoneum contained about four pints of yellow, rather turbid serum. The hollow viscera were externally healthy; the mesentery was loaded with fat, the omentum not so.

The kidneys were lobulated, flabby, yellow looking, with abundant fine granular deposit. No fat could be detected in them by microscopic examination. The liver was about the natural size, but very heavy; the surface irregularly tuberculated, presenting broad white patches, through which little yellow granules rose up. On section it was found exceedingly hard, of the faintest buff yellow hue, with deeper coloured yellow spots (like those already noticed on the surface) which proved, under the microscope, to be collections of oil globules. The gall-bladder contained about one ounce of orange-coloured bile. The spleen was pale, its surface marked with old adhesions.

Compare this with the two next following cases.

CASE V.—*Anasarca — Pleural effusion— Sudden death, with fatty degeneration of the heart.*

Benjamin Goodwill, aged 45. John front ward, November 1845. A tailor, a man of temperate habits, and healthy till two months before his death, when he became subject to a dull aching pain behind the centre of the sternum, increased on exertion. For the first six weeks he managed to continue his work, but during the last fortnight he has been laid up by cough and dyspnœa, and for one week he has had general dropsy.

Now his face is dusky; skin warm; tongue clean and moist, but rather livid; pulse 120, feeble; bowels rather confined; respirations 36.

On *auscultation* the heart's sounds seem very distant, but healthy. In front there is some large crepitation in the right, and smaller in the left lung. Behind, there is good respiration in the upper half, feeble and mixed with crepitation (smaller and more abundant in the left lung) in the lower half. The right side is generally very dull to percussion posteriorly; the left is unequally so in different parts.

A small cupping between the shoulders, and the exhibition of gr. ¼ of elaterium, was ventured on, at the same time that he was supported by wine and beef-tea; and the next note states—

Nov. 14th.—The cupping was followed by no relief. He has had not more than two hours' sleep during the night. Face dusky; expression anxious; surface cold, and no pulse perceptible at the wrist. Three healthy solid evacuations from the bowels. On *auscultation* more air is heard to enter the chest in front.

He had a large blister-plaster applied to the front of his chest, and brandy, with stimulant diuretics, was ordered to be taken at short intervals.

15th.—No sleep; delirious during the night. The blister has not risen. Face dusky; expression anxious; surface cold; pulse hardly perceptible; bowels not open;

* See for further illustrations and observations, M. Baron, Arch. Gén. de Méd. Série iii., Tome ii., p. 1; Mr. Paget, Med.-Chir. Trans. 1844-45; D. Virchow, Traube Beiträge, Berlin 1846; and Lancet, 1848, p. 120.

urine scanty and turbid; respirations 54, with a mucous rattle in the throat.

Shortly after this note had been taken, he rose up suddenly in his bed, to go to the water-closet, but he fainted by the way, and died in the course of a few minutes.

The body was examined 44 hours after death.—The arachnoid was opake and thickened; the Pacchionian glands were especially large and numerous; the pia mater was healthy; the lining of the left lateral ventricle was granular; the rest of the brain healthy.

The right pleura contained about three pints of clear serum, the left somewhat less; there were a few thready adhesions on either side. The lungs were marked on the surface with abundant deposit of black pulmonary matter. Within, they were of a dirty grey colour, slightly œdematous, with emphysema of the apices. The bronchi were healthy; the pulmonary vessels were pervious throughout.

The heart was large and flabby, containing well-formed recent coagula in all its cavities. A coagulum, distinguished from these recent ones by its friable consistence, reticulated texture, and firmer adhesion to the walls, filled the appendix of the left auricle, taking the form of the cavity. This was as large as a horse chestnut. A few coagula of the same kind lay entangled beneath the carneæ columnæ, at the apex of the left ventricle, and three or four similar but smaller coagula, the largest not exceeding the size of a horse-bean, adhered like little tufts to the interior of the ascending aorta. The walls of three of the cavities were healthy, and the cavities themselves but just a little enlarged. The left ventricle only was considerably dilated; its walls were flabby and collapsed on section. Beneath the endocardium, and throughout the substance of its walls, were numerous pale yellow spots of the same nature as those described in the case last detailed.

The liver was small, but not otherwise notably diseased. The kidneys were small, but healthy, except the right, which exhibited on its posterior side, in some conical yellow masses, the change familiarly known as "capillary phlebitis." There was no deposit of any kind, fatty or otherwise, discoverable by the microscope in the kidneys.

Deferring, till the conclusion of the next case, to speak of the symptoms and morbid appearances which the two cases last detailed have in common, there are two points here which require a passing notice—namely, the nature of the coagula in the heart and aorta, and their connection with the peculiar change observed in the right kidney.

The coagula in the heart and aorta were all probably of the same kind, being what are commonly known as Laennec's globular vegetations.* The early history of the coagulum in the auricle was supplied by the little bean-like bodies entangled among the carneæ columnæ of the ventricle. For the illustration of the earlier history of these in the aorta, I am indebted to Mr. Courtenay, Surgeon to St. Luke's Workhouse. In a specimen brought thence to St. Bartholomew's, in February 1848, there were a number of little pedicled buttons, growing from the interior of the aorta, the coats of which, in more than one place, were cracked and perforated to some depth. From this condition it is but one step forwards to the formation of those tufts which were seen in the case last detailed; or backwards to the first deposition of the constituent particles of these growths on some rough or fissured point of the lining membrane of the aorta. Not, however, that any visible abrasion or other lesion of the lining membrane of the aorta is necessary for their origin, for an illustration of the earliest period of all which my notes supply describes the heart as large and dilated, the valves quite healthy, only the ascending aorta set with a few smooth round warty growths. There is no notice of any other lesion of the aorta.

The question of the nature of the patches of so-called capillary phlebitis is closely connected with the study of both the origin and the further progress of these changes in the heart and aorta. We are taught† to consider these patches as resulting from a local action, whereby the blood is coagulated and the circulation suspended in the parenchyma of an organ. And this may either arise spontaneously, or, as is more commonly the case, result from the direct transmission of molecules from the heart to the capillaries of the part. Not that the whole yellow mass is thus constituted, but that a few molecules induce the action which ensues in the formation of the mass. Clearly these masses might originate as possibly when the vegetations were first forming in the heart or aorta, or instead of their forming there at all, as when the vegetations or other growths rupture, and discharge their fluid contents into the current of the circulation.

This is not the place to discuss this interesting pathological question. The great point to be assured of at present is that these masses are not of a fatty nature. This much is quite certain on the faith of the microscope. But though this, the only point which concerns the present subject, might thus be met by a simple statement, the above digressions will not appear, in the sequel, to have been superfluous. For it is in cachectic subjects, with feeble circulation, that such masses are most likely to form in the heart;

* Laennec, Aus. Méd. Tome iii. p. 344; Rokitansky, Path. anat. Band ii. S. 475.

† Rokitansky, Path. anat. Band ii. S. 437, 680.

in the very persons, in fact, who are likely to be the subjects of fatty degeneration of this organ. As far, however, as their pathology is made out, the connection of capillary phlebitis and growths in the heart with fatty degeneration is only that of coincidence.

CASE VI.—*Bronchitis — Sudden death, with fatty degeneration of the heart.*

Thomas Bennington, aged 55. Luke back ward, February 1847. A large, full-made man, a porter, intemperate; healthy, save a liability to winter cough, till three weeks before admission, when he began to suffer from cough and dyspnœa, with œdema of the feet towards evening. Now his face is dusky, his expression anxious, and he cannot lie down in bed. Loud rhonchus and sibilus mask all the other sounds in his lungs. His urine is not albuminous.

He got a little better under the use of diuretics and stimulants; when awake he did not seem to suffer much, but his appearance of lividity and dyspnœa during sleep were truly horrible. On the fourth day after admission he had occasion to go to the water-closet: he returned safely, rolled himself up in his blankets, and was dead in a few minutes.

On examination of the body after death, the blood was all fluid. The brain was found healthy as far as it could be examined. The lungs were emphysematous and œdematous, and universally adherent; the bronchi dusky, and full of puriform mucus.

The heart was of the natural size; the valves all healthy; the right ventricle marked internally with buff-coloured zigzag lines. To this extent the muscular fibres, examined under the microscope, resembled irregularly granular cords, but there was no marked abundance of distinct fat or oil cells.

The kidneys were large, lobulated, and the cortex of the left ill defined. There was nothing abnormal to note in the condition of the other viscera.

Looking back to these six cases, designed to present the disease in its most formidable point of view,—they were none of them in perfect health: the first was scarcely escaped from the threatenings of some coming disease; the second was liable to pain, such as to require medical attention; the other four were actually suffering from the most serious disease, which it will be worth while to consider a little more in detail.

Clearly, had no marked change been found on examining the body of Case III., there would have yet been no hesitation in attributing his death to the same cause—syncope, the result of his being incautiously placed in the upright posture. It is right, however, to remark, that though this is an accident we should always be led to apprehend and provide against, from the loose flabby texture of the heart discoverable after death during delirium tremens, still, that fatty degeneration is not in these cases ordinarily found in conjunction with that soft flabby state.* Of five dissections after death, from simple uncomplicated delirium tremens, of which I have records, the heart is noticed to have been particularly soft and flabby in four, and in four to have presented numerous small ecchymoses beneath the investing or lining membrane. In one, the heart, though flabby, was not dilated like the rest, but small, and loaded with fat. Examination by the microscope failed, however, to detect the slightest evidence of fatty degeneration in its structure.

Between the three last cases (IV., V., VI.) there is a very close resemblance. The history is the same in all,—a more or less gradual breaking up of the health, with an ineffectual attempt to keep at work,—cough and dropsy,—a short stay in the hospital,—and, at last, sudden death, following the exertion of walking to the water-closet, apparently by syncope. A little inquiry would show that the general history of cases of valvular disease of the heart is very similar, especially as regards the somewhat singular mode of termination of the cases. The actual state of things at last, indeed, is much the same in both, though brought about by different means. In the one class the obstruction increases till the heart can no longer overcome it; in the other, the heart becomes less and less able to carry on the circulation against the natural resistance of the tissue, till, as in the former case, from whatever cause, it suddenly ceases to act.

How much of this series of symptoms preceding the fatal termination was justly referable to the condition of the heart, I would not pretend to determine. Frankly I would claim only the pain behind the sternum, and the sudden death, as the results of the heart disease. For all the rest, and perhaps for the pain, the co-existing disease supplied a most sufficient explanation. But without the change in the heart, the death by sudden syncope occurring in all three cases is not easily intelligible.

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* Dr. Watson says (Lectures, vol. i. p. 399, 1st Ed.) one morbid condition I have found constant in persons dead of delirium tremens—a remarkably soft, pale, and flabby state of the muscular tissue of the heart.

PART II.

Fatty degeneration of the heart, not directly connected with the mode of dissolution—as a morbid appearance following the occurrence of hæmorrhage—accompanying phthisis—its connection with phthisis. M. Bizot's observations—Coexistent with other disease of the heart—rarely with valvular disease—Instances to the contrary, how far explicable—Hypertrophy and atrophy of the heart are suited to the requirements of the individual—Fatty degeneration of the heart connected with granular disease of the kidneys—Induration of the heart—Fatty degeneration of the heart occurring under various conditions.

In the second division of cases, where there is no evidence to show that the heart disease was the cause of death, it must be regarded simply as a morbid appearance, for the existence of which some cause must be sought in the patient's previous history or particular condition. The groups into which the cases have been thrown, to facilitate the consideration of their chief particulars, must not be looked on, as already remarked, as always displaying this cause. In the first subdivision, however, the preceding circumstance, which was common to them all—namely, hæmorrhage,—has very fair claims to be considered as the cause of the change: so much so, that in this view Case VII. has been placed in this subdivision, as if owning an analogous cause, though the exact cause, after very careful inquiry, remained undiscovered. Rather, however, than class the case on that account with those where death resulted directly from the degeneration, it appears better to regard it as illustrating the effects of some unknown debilitating agency, such as was the hæmorrhage in the three cases with which it is associated. Of two possible errors this appears to be the least, and, as less tending to magnify the subject under consideration, on the present occasion, the safest.

Case VII.—*Marasmus—Fatty degeneration of the heart.*

Nicholas Hovenden, aged 40; Matthew back ward, July 1848; a letter sorter, temperate, and healthy till five weeks before admission, when he noticed that he was very pale; then he got weaker, his legs began to swell, and he had palpitation, and pain in the head on standing up; but he had no other pain, nor ever any cough or hæmoptysis, and he said that he lived in an open airy space, on sufficient food, and that he was not overworked.

It would be wearisome to tell how many times he was most carefully examined by the most competent physicians, in order to make out what was the matter with him. His urine varied in specific gravity from 1012 to 1015, and was not albuminous. The left lung was settled to admit air a little more freely than the right, but all were satisfied of the healthy condition of both of them; the heart's sounds also were healthy.

There was little to record, as he slowly sank during the next month, getting weaker, thinner, and more deadly pale, from day to day, and suffering occasionally from diarrhœa, but the evacuations were always found healthy. At last he always kept his bed, as he vomited when he stood up. The blood in the superficial veins appeared of a pink colour, not blue, as in a healthy person. He became a little jaundiced; he was incoherent and fanciful, and refused to take his medicine, and often his food. When he died, on Sept. 8, the case was as obscure as on his admission.

On examination of the body after death, both lungs were found œdematous, especially the right. The heart was of the natural size, pale and flabby, containing a little very pale fluid blood, with a few coagula in the large vessels. The valves were healthy. The right ventricle presented a few buff-coloured lines and spots; the left ventricle was uniformly dotted all over within with such spots, and the same change could be traced deep into the substance of the organ. These buff-coloured spots appeared, under the microscope, to consist of degenerated muscular fibrils. The sarcolemma of the altered fibrils which lay immediately beneath the lining membrane of the ventricle was filled with small oil-globules; those fibrils that were deeper seated were simply granular, and at last striæ appeared on the fibrils taken from near the outer surface.

The thoracic duct was pervious; the stomach and intestines were healthy; the mesenteric glands rather œdematous. The left kidney was paler and harder than the right; there was a little opaque deposit in each of them. Under the microscope the tubes of the left kidney appeared much convoluted, and some casts lay about the field; but there was no excess of fatty matter visible.

That there had been no hæmorrhage in this case was pretty well made out, and each probable cause of the emaciation and debility which suggested itself was carefully inquired into. More especially the state of health of all who lived with him was investigated, on the supposition that the marasmus might depend on some impurity of the food or water which he was in the habit of using. The poor man, so long as his intellect remained clear, used to share largely in the great interest which his condition excited, and all the information that was anyhow accessible was obtained, but all in vain: the case remains perfectly obscure, save for the light thrown upon it by the three following cases:—

CASE VIII.—*Epistaxis—Fatty degeneration of the heart.*

James Skinner, aged 8; Matthew front ward, March 1848; a little, pale, bloodless boy, who had suffered, for the last two months, pain in the limbs, and occasional profuse epistaxis. Latterly, as he became very weak, his legs used to swell at times.

He was under observation for about a fortnight, during which time the epistaxis recurred twice, and it was noticed on the last recurrence of the hæmorrhage, five days before his death, how pale the blood was. He appeared to sink from exhaustion; but just at last, having lain for the previous days in a very drowsy state, he complained of severe pain in the abdomen, and died in a fit of convulsions.

The body was examined 40 hours after death.—The membranes of the brain were exceedingly pale, contrasting strongly with a florid clot of blood, of about 3j. size, which lay in the cavity of the arachnoid, to the right side of the vertex; otherwise the brain was healthy. The lungs were œdematous, with a few purpurous spots scattered over the surface.

There were about two ounces of clear fluid in the pericardium. The heart was of the natural size, the outer surface covered with purpura spots, some of which appeared internally. The whole of both ventricles, both inside and outside, was marked over with numerous little buff-coloured zigzag lines, less thickly set on the right than on the left side, where they quite altered the appearance of the organ. The valves were healthy. Under the microscope the buff spots appeared to be made up of disorganized muscular fibrils, retaining the external form indeed, but the striæ being replaced by irregular rows of little granules of oily matter, and many loose oil-globules. The healthy contrasted very strongly with the diseased parts of the heart, in the absence of these oil-globules, and in the presence of the natural continuous transverse striæ instead of the granular dotting of the fibrils.

There was a large puckered cicatrix in the great curvature of the stomach. The liver displayed a few purpurous spots; the other organs were healthy.

The only circumstance requiring particular notice in this case seems to be the small effusion of blood into the arachnoid sac,—a less common, but more important, form of hæmorrhage of the anæmic than that which had produced here the purpura spots in the lungs and liver also. One cannot help mentally connecting this cerebral effusion with the liability to epistaxis.

CASE IX.—*Placenta prævia—Fatty degeneration of the heart.*

Clara Donoghue, aged 30, admitted into Ward VIII. of the Dublin Lying-in Hospital, Jan. 30, 1849; twelve hours in labour of her fifth child, the placenta presenting. A dark-looking woman, slightly made; in the seventh month of pregnancy. After labour pains had continued about six hours, the membranes ruptured, and with the waters came a dash of hæmorrhage. She had had a similar dash about the third month. There was another profuse flow of blood about two hours after admission, when the vagina was plugged, and the hæmorrhage ceased. In about twelve hours more the plug was expelled, and, on introducing the hand to remove the presenting placenta, the child's legs being felt, delivery was effected by turning, and extracting the child still-born, and subsequently the placenta also.

The notes of the case record the gradual sinking of this woman: they speak of odd wandering pains, and of frequent rigors. She had cough, and once herpes labialis appeared, but auscultation found nothing amiss in her chest. She lay in bed, deadly pale, very thankful for all attentions, but expressing herself as comfortable, and sleeping quietly to death, which came on February 19.

On examination of the body after death, the heart was found pale, marked outside with many buff-coloured zigzag lines over the right, and more thickly over the left ventricle, on the inner surface also of which

a few spots of the same kind were to be seen. A few small soft masses (Laennec's globular vegetations) lay at the apex of the left ventricle. Under the microscope these buff-coloured spots were distinctly seen to be composed of muscular fibrils degenerated into granular fatty matter. Elsewhere the muscular fibrils were quite healthy.

The liver contained a little more fatty matter than usual. The kidneys were pale; the left was in other respects healthy, but the right emulgent veins were obstructed by a large coagulum, coloured at the peripheral ends, but white where it joined on to a large, white, friable coagulum, which occupied the vena cava ascendens from the junction of the common iliac veins nearly to the point of its passage through the liver. The right femoral and uterine veins were also full of old coagulum, and there was a collection of pus about the right ovary, around which the fimbriated end of the corresponding Fallopian tube had contracted adhesions. In other respects the uterus and its appendages were quite healthy.

Assuming in this case the connection between the hæmorrhage and the degeneration of the heart, it seems more likely that the hæmorrhage at the third month was the first cause of this change, than the hæmorrhage accompanying delivery so short a time before death. Proof, indeed, is entirely wanting, but it seems most natural to refer the fatty degeneration of the heart to the earlier hæmorrhage, and to whatever other causes had reduced the patient to the delicate state in which she was on admission. The local and the general atrophy probably all owned the same cause.

One word more on a subject which has already been noticed. In a former page, the connection between capillary phlebitis and growths in the heart has been adverted to. The connection may now be followed out a little further; for here we have the cause, from a more remote point in the circulatory system, displaying its effects on the heart. There appears no reason to doubt that the uterine veins were the primary seat of the affection, which subsequently extended itself up the ascending cava, and thence, through the lungs, to the left ventricle. Why no traces of capillary phlebitis should have been discoverable in the lungs, and why the globular vegetations should have been found only in the left ventricle, it is not easy to explain; only it is rarely the case that these changes respectively are found in the lungs and on the right side of the heart. It is worth while to notice, again, while giving this additional instance of the liability of the same class of persons to fatty degeneration, and to these growths and capillary phlebitis, that there is no direct connection between the two forms of disease. As far as we may venture to express the sequence of the phenomena of disease in this patient, they were probably as follows:— She had hæmorrhage; thence came atrophy: she had hæmorrhage again, and with it local injury; thence, as in a person predisposed to disease, came phlebitis; and from the effects of phlebitis came the little growths in the heart, whose lodgement there was facilitated by the weakened action of the organ; and death came at last from exhaustion and debility, of which these morbid appearances were in some sort the anatomical expression.

CASE X.—*Hæmorrhage from the bowels—Phthisis—Fatty degeneration of the heart.*

John Kershaw, aged 44; Luke front ward, October 1845*: a policeman; temperate, large, well made: was admitted in a state of great anæmia,—flesh flabby, face dull, surface of the body quite blanched,—having had discharges of red coagulated blood from the bowels during the last three months. Pulse 100, soft, thready, with a little thrill; tongue moist, with a thick brown fur on the dorsum; elsewhere pale and smooth: urine not albuminous.

On *auscultation*, the heart's sounds were heard quite distinct, and unaccompanied by any abnormal murmur; beneath the right clavicle there was some smallish crepitation, with long expiration and pectoriloquy; and, on *percussion*, unnatural dulness.

He sank, after he had been twelve days in the hospital, from the effects of the previous hæmorrhage, little or no blood having passed during the time that he was under observation.

On examination of the body after death, the lungs were found generally slightly emphysematous, with little blood in front, but congested from gravitation behind. There was much pale grey tubercle scattered about, being in granular masses in the posterior part of the left lung, but more compact about the apices, especially about the right, which was so heavy as only just to float in water, and presented, besides, a small, lined, tubercular cavity.

The heart was large, of a pale colour, presenting many little, zig-zag, buff-coloured spots beneath the lining of the left ventricle. There were four pulmonary valves. In other respects the heart was healthy.

There was a large ulcer in the duodenum, apparently the source of the hæmorrhage. The kidneys were small, pale, hard, tough, breaking with an irregular fracture. The capsule was firmly adherent; there were a few small cysts on the surface, and, on sec-

* Lancet, 1846, p. 117.

tion the cortex appeared thin, and was marked with a scanty buff-coloured deposit.

The history of this class of cases is as distinctly marked as was that of the former: it is the gradual sinking to death after hæmorrhage. The history relates, however, almost entirely to the hæmorrhage, not to the change in the heart. If there is any thing in the progress of these cases that may be claimed as at all characteristic of the cardiac lesion, it is the absence of any attempt at rallying during the intervals of the hæmorrhage, as displayed in Cases VIII. and X. Regarding this degeneration as simple atrophy, it is very easy to understand how any great diminution of the quantity, and, at the same time, of the nutritive properties of the blood, might be followed by such a change. But for this specific form of atrophy there would appear to be something more requisite—some specific tendency in the individual; for neither hæmorrhage nor marasmus are frequently followed or accompanied by this change: out of a large number of cases of this nature, the four just detailed are all that I have met with bearing on the present subject.

The case last detailed introduces the next subdivision, including three cases where this degeneration of the heart has been coincident with phthisis. Two of the cases (XII. XIII.) have little interest beyond what they may possess as the basis of a general analysis. But the case next following (XI.) has been given rather more at length on account of its own interest, apart from the peculiar change of the heart under consideration.

CASE XI.—*Acute phthisis—fatty degeneration of the heart.*

Abigail Richmond, aged 15; Hope front ward, April 1847; a foundling, singularly small for her age, of gentle, childish manners, well cared for by her mistress. She had complained of a slight cough for about a fortnight, but nothing was thought of this till one day, on running to meet some friends, her distress of breathing became so great, that, by the advice of a physician who happened to see her at the time, she was sent to the hospital.

She remained under observation about a fortnight, till her death. During this time she had always a dusky look, a rapid pulse, and cough, with scanty expectoration. Auscultation and percussion, repeatedly and most carefully employed, elicited no signs whatever of disease, for the first ten days. Then, suddenly, a change came over her; she became more livid, and her dyspnœa was very urgent: she seemed almost dying. But from this condition she was as suddenly roused by the application of a blister-plaster over the sternum. The recovery, however, was only temporary: she relapsed, and died in about three days more. Only once could a little crepitation be heard in the front of the chest.

On examination of the body after death, besides some other appearances not connected with the present subject, the left lung was found free; the right pleura was universally firmly adherent, and a firm white substance lay in the angle between the ribs and the diaphragm (shrunken coagulable lymph). Both lungs were thickly set with little tubercles, about as large as poppy seeds, some larger, and of a less regularly circular form. None of them were softened; only from one of two large, white, cheesy, right-bronchial glands, did fluid exude on pressure. A few miliary tubercles also were found *beneath* the mucous membrane of the bronchi, when the bloody fluid with which they were filled had been sponged away.

The heart was small, and quite healthy, save a few zig-zag buff-coloured lines beneath the endocardium of the left ventricle, which, on examination, were found to consist of degenerated muscular fibrils. A few tubercles were found in the spleen and kidneys; a patch of "capillary phlebitis," certainly not tubercle, in the former, and a few sloughy patches in the pelves of the latter.

This little girl was so well cared for and attended to by her mistress, that the date of the commencement of the disease may be relied on as accurate, at least as near accuracy as we can hope ever to arrive. The case differs from the ordinary run of cases of acute phthisis, in the fact that the fatal termination was mainly due to bronchitis, not to pneumonia. In its extreme obscurity, however, it fully answered the description of cases of this rare form of disease. Indeed, there was a complete absence of all signs from which any diagnosis could be made. This form has been separately described by Dr. Stokes (On Diseases of the Chest, page 415), under the name of acute inflammatory tubercle, without suppuration, as a form in which we are to expect assistance from percussion only, none from auscultation. But the rapidity with which the disease ran on to a fatal termination was such, that at no time was enough tubercle deposited to impair the natural resonance on percussion. Even that source of information as to the nature of the disease was here closed.

CASE XII.—*Phthisis—fatty degeneration of the heart.*

John Fox, aged 15; Luke front ward, February 1847; a weaver, very small for his age, always delicate, was admitted in the

last stage of phthisis, and died in about three days after admission. His pulmonary symptoms dated about three years back. He had suffered acute rheumatism two years previously, which circumstance caused his heart to be carefully examined. The sounds were reported to be healthy.

On examination after death, the lungs were found full of tubercle in all stages; further advanced in the right than in the left lung.

The heart was of the natural size; the valves healthy. There were buff-coloured, zig-zag spots beneath the endocardium of the right ventricle.

There was nothing of particular relation to the present subject to notice in the healthy state of the other organs examined,—liver, spleen, kidneys, or ileum.

The next case, in the same way, presents nothing to notice beyond the coexistence of phthisis and fatty degeneration of the heart, unless it be the occasional occurrence of pain down the left arm and left side of the chest. But as the pain noticed in the previous cases was behind the sternum, and displayed no tendency to ramify, it seems more natural to attribute this single symptom to the pulmonary than to the slight cardiac disease.

CASE XIII.—*Phthisis—fatty degeneration of the heart and liver.*

William Cannon, aged 40; John front ward, September 1848; an intemperate man, who, for the last nine years, had suffered occasional severe pain in the left side of his chest, running down the left arm, came into the hospital to die of phthisis.

On examination of the body after death, besides tubercles of the lungs, extreme fatty degeneration of the liver, and a granular condition of the kidneys, with a little white, fatty deposit, he was found to have a pale, thin, flabby heart, marked on the interior of the right ventricle with a few buff-coloured, zig-zag lines. Under the microscope, the heart, in the vicinity of the zig-zag lines, appeared to have undergone distinct fatty degeneration.

The same remarks which have been made on the subject of general wasting as a cause of fatty degeneration of the heart, may be repeated here with regard to the wasting of phthisis. Without denying to hæmorrhage, or phthisis, or marasmus, their share in first causing atrophy, we want something more to explain why it took this peculiar form in the individual cases.

There are besides two other interesting questions for discussion—I. Can we attribute this change in the heart's structure to changes which phthisis, as a disease of the lungs, induces in the heart's actions? or II. Have we any just grounds for regarding this as one of the results of the specific tendency to fatty degeneration displayed in the organs of phthisical patients? The first of these questions will be better considered in the sequel. In answer to the second question, much may be gained from the labours of M. Bizot,* who has investigated this subject in the course of his elaborate inquiries. His conclusions may be generally stated thus :—

Fatty degeneration of the heart occurs more often in women than in men, and in fat than in thin subjects. In the hearts of male patients dead of phthisis, there was always a less amount of fat than is natural; but in women the amount of fat was rarely less, commonly equalling or very much exceeding the average of the healthy accumulation. Again, he found that men dying of phthisis had rarely fatty livers, while women were very liable to this change, particularly those that were emaciated, and among these fatty degeneration of the heart was often found. It must be observed that these remarks apply to degeneration of the muscular structure of the heart into, or displacement of it by, adipose tissue, and not to the particular form under consideration.

My own examinations of phthisical patients have been in a large proportion of male subjects; and my notes respecting the state of the heart do not generally speak of excess so much as of diminution of fat. So I cannot collate my own observations with those of M. Bizot, with regard to the form of fatty degeneration of which he speaks. But out of more than fifty cases of phthisis, dying as such, the present form has been found only in four cases (X., XI., XII., XIII.), three males and one female; and out of upwards of fifty dying with tubercles, or doubtful remains of tubercles, only in one (XIX.), also a male. Further, only one of these, a male subject (XIII.), had fatty degeneration of the liver. And before drawing any conclusions from the cases, it would be necessary to reduce their numbers still more by excluding Case X., as not fairly exemplifying the effects of phthisis.

The chief differences, then, between M. Bizot's results and mine, are that he finds the affection more common among women, and in connection with fatty liver and general emaciation, than I do. And it appears very probable that this is a correct statement of the difference of circumstances under which the two forms now under examination respectively occur. Regarding the disease under both these forms simply as atrophy, as already observed, there is no difficulty in connecting it with phthisis; and M. Bizot's researches add largely to what was already

* Mémoires de la Société Médicale d'Observation, Tome I. pp. 351, 356. Recherches sur le Cœur, &c.

known concerning the tendency of pulmonary phthisis to induce this particular modification of atrophy. But general experience does not warrant the belief that phthisis is preeminently the cause of fatty degeneration of the heart, even of the form to which M. Bizot's observations refer; for the most striking examples of this form of fatty degeneration of the heart occur in elderly people not phthisical. Perhaps his observations at pp. 356-7 illustrate the form which is the proper subject of this paper, though in the absence of microscopic examination they cannot be quoted as such. The fact of their happening in non-phthisical patients, however, certainly does not show them to have been rudimentary (Louis, Réch. sur la Phthisie, p. 121, 2d ed.), or less perfectly developed manifestations of the disease.*

Thus far there has been some pretext of a natural division, coinciding with the artificial classification of the cases, but henceforth there do not appear to be any grounds for considering the subdivisions as any thing but arbitrary. The next class consists of four cases where there existed disease of the heart or pericardium. Separately they have considerable interest; as a class, save in an exceptional view, almost none.

Case XIV.—*Disease of the tricuspid, mitral, and aortic valves, with fatty degeneration of the heart.*

Sophia Christopher, aged 50; Hope back ward, August 1848; a poor, industrious woman, mother of fourteen children, who had suffered for six years "from the heart," apparently with pain and palpitation, and for three years with anasarca. She came in four days before her death, with orthopnœa, anasarca, and ascites, suffering much, but so deaf that nothing could be elicited directly from her. But from repeated careful examination it appeared that there was no notable increase of the action of the heart, and there was only a suspicion of a murmur with the first sound at the apex. Her urine was not albuminous.

On examination of the body after death, the right side of the heart was found dilated; the tricuspid valves were uniformly white, thick, and rigid up to the edge, evidently not efficient. The left side was not notably dilated or hypertrophied; pale. The mitral orifice was reduced to a slit one inch long, the edges of which formed quite a tube, so much were they thickened. These edges were generally tough and leathery, but in parts, especially at the ends of the fissure, there were earthy patches. The chordæ tendineæ were short and thick, and gathered into bundles of parallel fibres. The lining of the left auricle was white and opaque, and in one point, over a flake of bone, quite wanting. The aortic valves were white, opaque, and a little wrinkled, but entire; only rigid, sticking out as round pouches into the stream, which they must evidently have much obstructed, while they must also have allowed of regurgitation. The aorta was much diseased, especially about the attachments of the aortic valves, which were lengthened and ossified. Under the microscope the fibres of the left ventricle appeared singularly granular, but not distinctly fatty. The transverse markings were much less distinct in the muscular fibrils taken from the left than in others taken from the right side of the heart.

The left pleura was free, the lung emphysematous; the right pleura universally adherent, the lung œdematous. The liver was small, weighing under lbij.; the kidneys hard, slightly granular, with the cortex indistinct. The spleen was also hard.

There is a point of great interest in this case. There was extreme disease of three sets of valves, without any valvular murmur. Now this circumstance is not very uncommon as concerns the left valves, and Dr. Blakiston (On Diseases of the Chest, p. 226) gives it as the result of his experience, that tricuspid disease rarely causes a valvular murmur. My own more limited experience would not quite admit so general a statement, but certainly in this patient there was an inefficient state of the tricuspid valve, and there was only a suspicion of a murmur. A much better test, however, than the ear, in a complicated case of long duration like the above, as to which valves do their duty, is the anatomical examination of the different cavities of the heart. Accordingly, we find that the right ventricle was hypertrophied, and its fibres in a state of active growth; while the left side was atrophied, and its elementary fibres degenerating.

There is nothing to detain us on the subject of the right side of the heart, where there was merely excessive action of the muscle to compensate for the imperfections of the valve. But why did not this same process take place on the left side? Probably the obstruction at the tricuspid orifice reduced the stream in which the left ventricle had to act, and relieved it of the *vis a tergo* through the pulmonary capillaries. The stream was too small either to cause a murmur or to tax the powers of the heart: and so the left ventricle shrunk back again to the requisite dimensions, and its supernumerary fibres became atrophied, some of them being in the process converted into fat. Let me not be misunderstood; when the

* Mr. Adams' case, already referred to, might be quoted in support of this objection.

stream is too small to produce a murmur, it does not follow that the labour of the heart is also diminished: quite the reverse; the bell may be silent either for want of the means to make the sound, the hammer, or for want of power to move the hammer, which has rusted in its place. So the murmur may cease when the whole quantity in the ventricle is all too small, or when the overloaded heart cannot send a stream large enough through the contracted orifice. The former condition appears to have obtained in this case.

It is possible to suggest other explanations, but the above appears the most probable. Extreme disease of the tricuspid valve is very rare; but the following case may be quoted in outline, in support of this view:—

Disease of the mitral and tricuspid valves—Death by exhaustion.

Jane Brooks, aged 42, first came under notice in 1846, when she had hemiplegia of the left side, of which, however, she got quite well. Seven months after, she was admitted after an attack of this nature:—She had turned pale, fallen, remained unconscious for half an hour, and then recovered without any paralysis. At this time she had a murmur with the systole, audible at the apex, and shorter at the base of the heart.

She came again under notice in September 1848, suffering from anasarca; and this time she came into the hospital to die. I never saw any one sink so quietly; but for her cold pulseless hand I could scarcely persuade myself that she was in the very act of dying.

On examination of the body after death, the frame was found generally healthy, only on the upper surface of the right corpus striatum, in front, was a yellow depressed space, about a quarter of an inch across, beneath which the cerebral substance was grey and soft. To the outside of this, and behind, two other similar spots were noticed, hard and yellow externally, grey and diffluent within.

The heart was large; it was chiefly the right side that was thus affected. The tricuspid valve was thickened and opaque, and a round edging encircled the orifice, running along the edges of contact, with a hard transparent granule projecting here and there at the salient angles. The mitral orifice was reduced to a narrow chink with rough, earthy, thickened edges.

The fact appears, from both of these cases, to be, that an obstruction at the right side of the heart diminishes the labour of the left ventricle. The two next cases, however, go far to vitiate the conclusions which this digression has been designed to guard.*

* Dr. Latham has recorded a case (Diseases of the Heart, Vol. ii., p. 40) where the appearances of the heart closely resemble that described in many of these cases as characteristic of the disease. "In its large carneæ columneæ were some peculiar deposits, giving them the appearance of grained oak." The case, on a careful examination, bears strongly in favour of the conclusion that this degeneration does not occur in hearts where the valves are inefficient,—a conclusion which, at first sight, it appears to contradict. But as the case was one of acute endocarditis, and there had formerly been acute pericarditis, there is a possibility that these might be recent or obsolete inflammatory deposits, and, as such, unconnected with the present subject.

CASE XV.—*Valvular disease—Fatty degeneration of the heart.*

Lætitia Dale, aged 40, August 24, 1848; Hope front ward; thin, with an anxious expression, suffering intense dyspnœa, her legs œdematous and vesicated in points. Pulse 96, soft, irregular. She said that she had never had acute rheumatism, but that fifteen years ago she was once much frightened, and fell, and that ever since that time she had suffered palpitation and dyspnœa, and for the last five years anasarca.

Her heart's action was very irregular, and there was extended dulness on percussion over the cardiac region. There was a systolic (and diastolic?) murmur most audible on the level of the fourth rib, not notably prolonged in any direction except towards the apex.

She was under observation about a fortnight, till her death; but there is no notice of any particular occurrence, beyond the continuance of her intense dyspnœa.

On examination of the body 33 hours after death, the lungs were found œdematous; the right pleura was adherent; the cavity of the left contained about a pint and a half of fluid.

The heart was large, weighing ℥xvj.; it was chiefly the auricles and right ventricle that were enlarged, being dilated, and their walls thickened. The valves were all healthy, except the mitral, which was rigid, much thickened, and the edges agglutinated, so as to form a fissure about an inch long, which was ragged, but not ossified at the extremities. The endocardium was continued over the thickened edges of the valve, which projected like a round moulding beyond the line of attachment of the chordæ tendineæ. Interlaced among the carneæ columnæ lay many bits of older coagulum than were found elsewhere; and when the stains of decomposition had been removed by maceration, it was evident that both ventricles were in an extreme state of fatty degeneration; buff-coloured spots, such as have been already described, appearing all over their surface.

The liver looked shriveled; it weighed lbijss., and was rather fatty. The spleen was small and hard. The kidneys were small; the cortex uneven, but well defined.

The stomach contained much dark mucus; the intestines were apparently healthy.

CASE XVI.—*Disease of the aortic and mitral valves—Fatty degeneration of the heart.*

William Jordan, aged 10; John front ward, December 1847; a pale, delicate little boy, who had suffered from dyspnœa all his life, had anasarca for a fortnight before admission. There was then a purring tremor to be felt in the second right intercostal space, close by the sternum; and a double murmur to be heard, loudest during the systole, and at the base of the heart. The heart's action was very irregular, beating in triplets, thus — 4, 2 : 4, 2 : 3, 1 : 4, 2 : 4, 2 : 3, 1—the length and intensity of the murmur being expressed by the numbers, and the two last beats of each triplet running into each other.

He continued under observation in the hospital till February 11, during which time the auscultation remained the same, and there was little change in his condition from day to day. The only circumstances which attracted much attention were his vomiting almost daily, about 5 P.M., and occasional attacks of intense dyspnœa about 7 P.M. The former nothing relieved, the latter yielded in a most striking manner to the action of a blister-plaster and diuretics.

He remained at home till the middle of May much the same, when his breathing became more difficult, and his cough increased. Suddenly, one morning in his distress, he rose out of bed—sat in a chair—then sank down, and died about two minutes after having been replaced in bed.

On examination of the thorax 36 *hours after death*, the pleuræ were found partially adherent about the base; the lungs were œdematous, and about their bases congested, as if from commencing pneumonia.

The pericardium contained two or three ounces of clear fluid. The heart was much dilated. The right valves were healthy: the mitral valve was a little thickened, but, though some of the chordæ tendineæ were ruptured, obviously efficient. There were only two aortic valves, imperfectly divided into three, almost destroyed by ragged ulceration. There were little zig-zag buff-coloured lines beneath the endocardium of both ventricles.

There was nothing so very peculiar in the mode of this little boy's death as to make one look for an explanation of it in the fatty disease of his heart. It is a very common thing (as already noticed) for sufferers from valvular disease to die thus suddenly after some little exertion. And more particularly children are liable to die thus, partly because their movements are more sudden and less under our control than are those of adults, and partly because the generally healthy state of their organs enables them to live under a condition of abiding disease greater than what an adult could bear. And so they are more liable to be suddenly cut off by some trifling accident, as was this little boy by the trifling exertion which his intense distress induced him to make.

The two last cases differ from all the others here adduced, in the fact that the valves were in a condition requiring increased action of the heart.* Strongly as they weigh against the conclusions that this degeneration is essentially an atrophy, it must be remarked at the same time that in the last case the heart was dilated rather than hypertrophied; in a state, namely, of passive, rather than of active resistance to the pressure of the circulation. With regard, however, to Case XV., I had rather leave it as an exception, than attempt to explain it on any grounds which, however plausible, should still be as unsatisfactory to others as to myself.

One more remark: fatty disease of the heart is generally believed to be closely connected, as a predisposing cause, with rupture of the organs. This case, however, must not stand as an illustration of this connection, for the ruptured ends of the chordæ tendineæ displayed, on the most careful examination, no evidences of fatty degeneration. Such changes in the fibrous textures of the heart my own experience would lead me to connect rather with that peculiar form of change of the valves which was here noticed.

CASE XVII.—*Encephaloid disease of the pericardium—Fatty degeneration of the heart.*†

James Cooper, aged 45; John back ward, May 1846; a navigator, intemperate, healthy till eighteen months ago, when he began to suffer from pain in the left hypochondrium. This pain has now moved into the epigastrium, and occurs in paroxysms. He has been getting gradually weaker, so that within the last five months he has given up work. Now his face is sallow, his expression anxious; tongue moist, clean, red; pulse 100, very feeble; bowels regular. His feet are a little swelled. Urine scanty, acid; spec. grav. 1023, not albuminous. On *auscultation* the heart's sounds are not clear, but they are not accompanied by any morbid sound.

He sank gradually; the immediate cause of his death appeared to be exhaustion from diarrhœa. He had found considerable relief to the epigastric pain, when in Guy's Hospital, from dry cupping; and during his

* Since writing the above, another similar case has come under my observation.

† Med. Chir. Trans. Vol. xxx., p. 46.

stay in St. Bartholomew's the nausea and vomiting, of which he for a while complained, were removed by a blister-plaster applied to the epigastrium. There was absolutely nothing else to notice in the progress of this very obscure case.

The body was examined 12 *hours after death.*—Passing over the lengthened details of the dissection relating to the state of recent disease of the lungs, and encephaloid disease of the pericardium, with doubtful scirrhus of the pylorus, the heart was found natural in size; there was an ordinary "white spot" at the apex; there were no traces of any malignant deposit in the tissue of the heart, but beneath the lining membrane, and also on section of the walls, appeared numerous buff-coloured zig-zag spots. The valves were all evidently quite efficient.

The other organs were healthy.

The first explanation of the fatty degeneration which suggests itself in this case is, that the heart had been stronger, to fit it for the requirements of the daily labour of a navigator. But when this daily labour was changed for a bed of sickness, the fibres immediately became atrophied and fatty. Another explanation would refer this fatty degeneration to the effects of old inflammation of the muscular structure of the heart; looking for its cause in the encephaloid masses of the pericardium, and for its symptoms in the wandering epigastric pains. Abstractedly there is no objection to eithe planation: both, indeed, may be correct. But while it is certain that the premises of the first conclusion existed, it is not certain that those of the second did.

It is possible that the connection between granular disease of the kidney and fatty degeneration of the heart does not appear from the details of dissections to be as close as it really is. For with granular disease of the kidney there is very often connected some great obstruction to the capillary circulation, the signs of which appear, before death, in dropsy and in an increased action of the heart. And after death, an hypertrophied left ventricle, with an aorta which it has twisted from side to side of the spine by its violent contractions, are traces of a state of things which could as little have allowed of atrophy as could the ventricle of a heart affected with valvular disease. This circumstance may prevent the change from manifesting itself: whether there be any strong tendency to such a change, there will be occasion to consider hereafter.

CASE XVIII.—*Anasarca—disease of the kidneys—Fatty degeneration of the heart.*

Catharine Knowles, aged 27; Mary back ward, April 1847; a needlewoman, temperate, and healthy till three years ago, when the catamenia ceased after exposure to cold. At that time her legs swelled, but the œdema seems to have disappeared, till eight months ago, when her legs began to swell again; and now she is generally œdematous, and has ascites also. She has pain in the right hypochondrium, cough, and dyspnœa.

Active purging by bitartrate of potash relieved the swelling, but she gradually sank about a fortnight after admission. It does not appear certain that her urine was ever examined.

On examination of the body after death, the lungs were found generally emphysematous, and œdematous in points.

The heart was small, the valves healthy; there were a great many little zig-zag buff-coloured lines beneath the lining of the right ventricle; none beneath that of the left.

The kidneys were large, rather uneven on the surface, the capsule not unnaturally adherent. The cortex was of a pale yellow, contrasting in this respect with the pink colour of the pyramids, and containing distinct small fatty deposits: [it is not mentioned in the original note in what structure these deposits had their seat].

The next case also presents the fatty degeneration limited to the right side of the heart. In this respect only, and in the fact of the kidneys being diseased, does it resemble the case last detailed. Two more dissimilar conditions of life can scarcely be conceived than those of a suburban actor, and an overworked London sempstress. The renal affection is one of the many diseases caused alike by dissipation and distress: the cause of the degeneration of the heart is not equally obvious.

CASE XIX.—*Anasarca—Albuminuria—Fatty degeneration of the heart.*

Robert Adams, aged 46; John front ward, April 1848: an actor, intemperate, but healthy till lately. He had rheumatic fever "some time ago," and since then has had palpitation on exertion. He had a severe cold six months ago, but the present symptoms do not date more than two months back.

He is a large, heavy man, face sallow, expression anxious; tongue furred on the dorsum, red at tip and edges; pulse 108, small, soft; bowels regular. He suffers from anasarca, with slight ascites. The heart's impulse is forcible to the right of the sternum and in the epigastrium, unaccompanied by any abnormal sound; urine, sp. gr. 1010, albuminous.

He was under observation for a week. The chief point of interest was the difficulty of administering opium to calm the delirium from which he suffered, and which appeared to have arisen from the want of his usual

allowance of spirits; for it was thought unsafe to use either opium or stimulants without first relieving the lungs and heart by cupping. He bore the necessary depletion well; but he became comatose, and so died after three days.

On examination of the body after death, the lungs were found œdematous.

The heart was very large, the walls stiff, like hard leather, and clouded yellow. Beneath the lining of the right ventricle were buff-coloured zigzag spots, and in some of the carneæ columnæ near the apex were little masses of adipose tissue. Under the microscope the appearances were very striking. In the hard stiff parts there were healthy muscular fibrils with well-marked striæ, but singularly twisted and convoluted. In other places the fibres were short, and apparently brittle, but in the buff-coloured parts the striæ were all gone, and numerous oil-globules floated across the field of the microscope.

A communication existed between the cavity of the stomach and the lesser cavity of the peritonæum, which latter exhibited marks of intense inflammation. On laying open the stomach, the perforation was found to be situated near the pylorus: it was about four lines in diameter, the mucous membrane being removed, however, to a much larger extent than the muscular or serous coats. There were also several ulcerated fissures in different parts of the mucous membrane of the stomach.

The liver was in a state of extreme fatty degeneration. The kidneys were of the natural size, hard, rough, marked with granular deposit. The cortex appeared, on section, almost entirely wanting.

Of the immediate cause of death in this case, the peritonitis, there is little to say: it can only be regarded as a morbid appearance discovered after death in a patient whose symptoms had given no reason to suspect the existence of such an occurrence as perforation of the stomach. The physicians of hospitals can tell of human beings brutalized by ignorance, or sensuality and drunkenness, till, by habitual neglect, they have become unable to express their own sensations; and when intoxicating spirits have ceased to ward off present suffering, or want prevents their obtaining them, they come into our large hospitals simply to die: they wish for nothing but to be left alone; for all they can tell of their sufferings, their treatment is like that of a dumb animal, and too often so is their death also. Such was this poor creature.

The muscular substance of the heart was in very striking contrast in different parts of the organ in this case. That the hard leathery condition noticed in some parts is not one of strength is certain; for it is acquired by the admixture of bundles of fibrous with the proper muscular tissue, which might, indeed, increase the passive, but not the active strength of a muscle. In my own experience it is not dilated ventricles that are usually the seats of this change; rather, it is found to coexist with universal adhesion of the pericardium when general, and, when local, either it has ensued upon endocarditis when affecting the musculi papillares, its common seat, or it occupies the circumference of aneurismal dilatations of the ventricles, as in the case already quoted. There appears, then, judging at least from the situations where it is most commonly met with, no reason to doubt the correctness of the common opinion which regards it as a result of chronic inflammation. The connection of this chronic inflammation with fatty degeneration of the heart, there will be an opportunity of considering hereafter in a more general point of view. On this the next case throws a little light.

Case XX.—*Anasarca—Fatty degeneration of the heart.*

Joseph Ganny, aged 59; Matthew front ward, January 1847: a watchmaker, of very intemperate habits, who for the last seven years had suffered from shortness of breath, with pain in the epigastrium, was admitted, complaining of inability to make any exertion; palpitation of the heart, and, within the last fortnight, anasarca. He was of a deadly pale aspect. Thirty years ago, after having been ill for three months, he suddenly expectorated about a teacupful of green fœtid pus. This was the end of that particular illness, but he continued to cough and spit occasionally throughout his life. He often spoke with his family about that single copious expectoration.

He remained in the hospital about a week, when, from the absence of all note to the contrary, he seems to have died quietly. All the anasarca had disappeared before death. It does not appear that any albumen was ever looked for in his urine.

On examination of the body after death, the right pleura was found universally adherent, the left free. The left lung contained ten or twelve pisiform bodies, laminated, of a cheesy consistence, and the bronchial glands at the root of the lung displayed the same change (obsolete tubercle). The bronchi were healthy: there was no puckering of the apex. The right lung was dark, tough, and condensed about the base, where, deep in the substance of the lung, was a cavity, with polished walls, of about the capacity of six drachms, containing a reddish fluid. This cavity was of an elongated form; the reticulated markings on its walls strongly resembled the same mark-

ings of the bronchial tubes, with whose lining membrane the lining of this cavity was continuous. On tracing the passages which opened obliquely into it they were found mostly to communicate with bronchi, —one, however, returned into the cavity.

The heart was large, its pericardial surface a little injected, displaying a few "white spots," and a little disease of the coats of the coronary vessels. The walls of the right ventricle were singularly hard; the carneæ columnæ were largely developed, and, on section, exhibited a yellow discolouration. The walls of the left ventricle were only a little thicker than those of the right, but much softer. On section, a mottled appearance was observable in the walls and the carneæ columnæ, as of a buff-coloured deposit. The valves were generally thickened, but apparently efficient. Under the microscope the discoloured parts of the heart were seen to be made up of irregular granular cords, with many loose oil-globules.

The liver was small, with hepatic venous congestion. The kidneys were hard and tough, the capsule rather more adherent than natural. The spleen was hard and dark, but of the natural size. The pancreas was singularly large and hard, but otherwise healthy. There was a small hard nodule beneath the serous coat of the stomach, and two or three small hard beads lay in the cellular tissue at the back of the duodenum.

The grounds on which this case is placed in the present class are not fully substantiated by the details; for it is not quite certain, however probable, that the dropsy in this case depended on disease of the kidneys. The most striking morbid appearance, apart from the subject under consideration, was the sac found at the base of the right lung, which was supposed to be the sac of an old pulmonary abscess: it might, indeed, have been an old tubercular excavation, or the remains of a sinus by which pus had discharged itself from the cavity of the pleura; but the results of the examination of the surrounding part of the lungs, and its situation in the lower lobe, did not favour either of these interpretations so much as that of its being an old pulmonary abscess the consequence of pleuro-pneumonia. From whatever cause the cavity may have been originally formed, the curious circumstance is, that it should have continued open for a period of thirty years.

The same induration of the muscular tissue of the heart as was spoken of in the last case was noticeable here also.* Here it was confined to one ventricle; the fatty degeneration, however, seemed to have gone on alike on both sides of the heart. Induration appears to have been the change to which all his organs were most liable: liver, spleen, pancreas, kidneys, and heart, were all alike either indurated or contracted, or both.

The next case also comes from the same large class which want and intemperance contribute to swell; but he died of an accident, so to say, before his organs were all fairly worn out: indeed, his heart was almost the only viscus which displayed evidences of the commencement of chronic disorganization.

CASE XXI.—*Pneumonia—Fatty degeneration of the heart.*

John Manning, aged 64; Matthew back ward, May 7, 1847: a lighterman, formerly very intemperate in beer, and so fat that he could not stoop to button his gaiters; but for the last two years he had been out of employ, and his legs became œdematous, and he had become much thinner; still he thought himself in good health, till, on May 3, he was taken suddenly ill, and he became dull and stupid, and unable to express himself. On May 7 he took three hours to walk up from Westminster to St. Bartholomew's (about two miles): he was taken into the hospital with the ordinary physical signs of pneumonia, cold and blue, but without any complaint of cough, and died in about four hours.

On examination of the body after death, the left lung presented the third and second stages of pneumonia, in the upper and lower lobes respectively, proceeding from above downwards. The right lung was emphysematous and œdematous.

The heart was large; all the valves were healthy, except the aortic valves, which were reticular and thickened, but apparently efficient. There were many buff-coloured zigzag lines beneath the endocardium on each side, most on the right. The aorta was extensively diseased, with bony flakes and atheromatous deposit.

The liver was healthy; the kidneys small,

* Dr. Fothergill details a case, apparently of this nature, of much interest in connection with the present subject (Medical Observations and Inquiries, vol. v. p. 252). A gentleman, aged 63, rather inclined to corpulency, after about four years' suffering of the ordinary symptoms of angina pectoris, fell down and expired immediately in a sudden and violent transport of anger. The body was examined by John Hunter, who says, "The heart, to external appearance, was also sound; but upon examination I found that its substance was paler than common, more of a ligamentous consistence, and in many parts of the left ventricle it was become almost white and hard, having just the appearance of a beginning ossification." The valves were apparently efficient: the aorta and coronary arteries were diseased.

hea'thy ; the spleen small, with a single pale spot of so-called "capillary phlebitis."

The explanation that must naturally suggest itself in this case is the same as that already applied to Case XVII.—namely, that the strength of the heart had latterly been diminishing, on account of the less amount of work required of it ; but this process had not gone on in the ordinary way, on account of some peculiarities in the individual. So, instead of the heart shrinking and remaining strong in proportion to its size, as in phthisis, cancer, and other emaciating diseases, the obsolete fibres underwent a different change, and were converted into fatty matter, maintaining the bulk, but adding nothing to the strength, of the heart. Probably the explanation in the two following cases is also the same ; the essential cause of the individual peculiarity is still open to inquiry.

CASE XXII.— *Sanguineous apoplexy — Fatty degeneration of the heart.*

—— Horner, aged 57 ; Luke front ward, July 1846 : a harness-maker, who had suffered for years with pain in the head, after three weeks' comparative affluence coming on a long season of want of work and food, fell down in an apoplectic fit. He lay in the hospital for eight days, sensible, but incapable of replying to questions. Then he became gradually comatose, and died in four hours.

On examination of the body twelve hours and a half after death, the lateral and fourth ventricles were found full of extravasated blood, by which the left optic thalamus and the septum lucidum had been torn down. The arteries at the base of the brain were much diseased. There was a little congestion, with œdema, of the lungs.

The heart was flabby, and rather large ; the valves thickened, but quite efficient ; the right side a little dilated, with some fatty degeneration. The aorta was roughened by disease.

The gall-bladder was full of cholesterine calculi. The left kidney contained several cysts full of a dirty chocolate-coloured fluid.

Sanguineous apoplexy is not a common accompaniment of fatty degeneration of the heart: death by coma, or with the symptoms of apoplexy, is much more common. Mr. Adams's case, already alluded to, and Dr. Cheyne's, both terminated this way ; in neither of them was there any cerebral effusion. Dr. Cheyne remarks on his case, in words applicable to all three, "Apoplexy in this case must have depended upon increased action of the vessels of the head. The heart itself was incapable of communicating much impetus to the circulating mass."*

The subject of the next following history, which concludes this series, differed in her habit of body most strikingly from all the rest. A better specimen of a thin wiry frame, as distinguished from the flabby texture of most of those of whom there has been occasion to speak hitherto, could scarcely have been found.

CASE XXIII.—*Fever fatal by exhaustion— Fatty degeneration of the heart.*

Mary Taylor, aged 64 ; Mary back ward, March 1848 : a thin and haggard, but active woman, long a nurse in the hospital, was taken in as a patient with a pain in the hypogastrium. As she was discharged on the relief of this complaint, she was taken as a temporary nurse to attend some cases of fever which had occurred in a surgical ward. For three weeks she kept her health, and then gave up work, more as if worn out than as suffering from any particular disease ; but in about ten days, three days after an increase of all her symptoms, a spotted rash came out all over her body. She had severe headache, but her mind was clear to the last. She had no diarrhœa, but simple exhaustion, with a fluttering pulse, hardly sustained by brandy, and finally ceasing about the end of the third week.

On examination of the body after death, the lungs were found congested, heavy, and soft, giving issue, on pressure at their bases, to a dirty grey fluid.

The heart presented buff-coloured spots beneath the left endocardium, such as have been already described in the other cases.

The intestinal canal was quite healthy, except the rectum, the lining membrane of which was thickened, puckered so as to make strictures in some parts and cellular dilatations in others, and slightly ulcerated.

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* Dublin Hospital Reports, vol. ii. p. 17.

PART III.

General analysis—nature of the cases—History of the disease, compared with that of the second kind of fatty heart—Pathology—Is this disease—I., *the result of general predisposition, as shown in the cellular tissue, the lungs, liver, or kidneys?*—II. *The direct effect of disease of some other organ, as the lungs?*—III. *A primary affection of the heart?*

General remarks—its connection with retarded convalescence—with angina pectoris.

Such, then, are the varied circumstances under which this form of disease may be met with, and such the materials from which its clinical history is to be collected. Clearly, not one of the heads under which the cases have been classed, can be considered as expressing the cause of the degeneration, except, perhaps, with very large qualifications, hæmorrhage. A general analysis promises better results: it will, at least, set forth in a concise form the general characters of the disease.

For this purpose, a tabular view of the chief particulars noticed in the cases detailed, is subjoined. And to these cases my friend and successor, Dr. Kirkes, has added those that have fallen under his observation in the same field—the pathological theatre of St. Bartholomew's Hospital. The entries in the table do not appear to require any particular explanation. They are arranged to elucidate two points, so far as they have not been considered in the narrative of the cases—1st, the general history of the affection; 2nd, its connection with any morbid process which may have left traces of its presence in any other organs.—(For Table, see following page.)

Of the 25 cases here referred to, 18 were male, and 7 female. Their age varied from 64 to 8, the average being 40·5, or excluding the four extremely young cases, much higher, 46 years. Of the habit of 9 of these no note has been taken; but of 9 it is recorded that they were full or fat, while 6 were notably thin, and extreme paleness was the chief characteristic of 2 others. Of the whole number, 10 were habitually intemperate. The most common complaints were exhaustion from hæmorrhage, or other cause, in 4; pulmonary phthisis, in 3; and anasarca, with or without an obvious cause, in 10 cases.

It may hence safely be inferred that this degeneration is a disease of advanced life, though its occurrence is not circumscribed within any very accurate limits of time. The subjects of the disease are not generally thin, but the reverse. They have not, however, a healthy fulness of body, and the pallor of some of them is very remarkable. Excluding the four children, the average number of intemperate subjects is perhaps not higher than among the subjects of any other chronic affection. The disease is found most commonly under circumstances of which anæmia and anasarca may express the types, where the circulation is lowered or obstructed; but there are numerous exceptions. Death in 6 cases out of 25 was sudden; in 5 of these almost momentary.

The same perhaps might be safely predicated of the subjects of what has been here called the second form of fatty degeneration of the heart: they are commonly past middle life, pale, cachectic, and fatty rather than what is usually understood by fat. But I think that symptoms of suffering directly referable to the heart are more commonly met with in this class, that a gouty disposition is more frequently to be traced, and that the external characters of the tendency to fatty degeneration are more marked. Dr. Latham tells me that it is not in those who have always been fat that he apprehends this morbid change, but where a man has been always thin, and in the decline of life becomes suddenly fat. There is reason also to think that the slowness of pulse which has been so often noticed in connection with fatty degeneration of the heart, is more particularly characteristic of this second form. At least it was not noticed in any of the cases above detailed.

Further than this the comparison of the two forms cannot safely be carried, for the information concerning each of them respectively is drawn from different classes. On the one hand the observations have been made by Mr. Paget, Dr. Kirkes, and myself, chiefly on the poor patients of Bartholomew's Hospital. Of the other kind, information has been derived from all the ordinary sources, including in the results of private practice, recorded or otherwise, a large proportion of the richer classes.

No.	Sex.	Age.	Appearance.	Habits.	Suffering at time of death from	Condition of the Heart.	Condition of the Lungs.	Condition of the Liver.	Condition of the Kidneys.	Condition of Various organs.
I.	M.	56	Fat.	Temperate.	Fever.	Double, fatty degeneration.	Emphysema, œdema.	Gall stones.	Healthy.	——
II.	F.	60	Do.	Do.	Ailing.	Not stated.	Healthy.	Healthy.	Do.	——
III.	M.	35	—	Drunken.	Delir. tremens.	Do.	Do.	Fatty.	Do.	——
IV.	M.	34	Pale.	Do.	Anasarca.	Double.	Œdema.	Do.	Granular.	——
V.	M.	45	—	Temperate.	Do.	Left, Laennec's growths.	Œdema, emph. pleurisy.	Small.	Cap. phlebitis.	Diseased aorta.
VI.	M.	55	Full.	Drunken.	Do. and bronchitis.	Right.	Œdema, emph. bronchitis.	Healthy.	Healthy ?	——
VII.	M.	40	Thin.	Temperate.	Unknown.	Double.	Œdema.	Do.	Granular.	——
VIII.	M.	8	Do.	Do.	Hæmorrhage.	Do.	Œdema, purpura.	Purpura.	Healthy.	Cereb. hæm. ulcer of stomach.
IX.	F.	30	Do.	Do.	Do.	Do. Laennec's growths.	Emphysema.	Healthy.	Renal phlebitis.	Phlebitis.
X.	M.	44	Full.	Do.	Do.	Left.	Emphysema, tubercles.	Do.	Granular.	Ulcer of duodenum.
XI.	F.	15	Fat.	Do.	Phthisis.	Do.	Tubercles.	Do.	Pyelitis.	Cap. phleb. of spleen.
XII.	M.	15	—	Do.	Do.	Right.	Do.	Do.	Healthy.	——
XIII.	M.	40	—	Drunken.	Do.	Do.	Do.	Fatty.	Fatty.	——
XIV.	F.	50	—	Temperate.	Anasarca, diseased heart.	Do. valvular disease.	Œdema, emphysema.	Healthy.	Granular.	Diseased aorta.
XV.	F.	40	Thin.	—	Do. do.	Double, valvular disease.	Œdema.	Fatty.	Healthy.	——
XVI.	M.	10	Do.	Do.	Do. do.	Do. do.	Do.	Not. ex$^{d.}$	Not examined.	——
XVII.	M.	45	Full.	Drunken.	Cancer.	Double, cancer of pericardium.	Pneumonia.	Healthy.	Healthy.	Scirrhus pylori.
XVIII.	F.	27	—	Temperate.	Anasarca.	Right.	Œdema, emphysema.	Do.	Granular.	——
XIX.	M.	46	Fat.	Drunken.	Do.	Do.	Œdema.	Fatty.	Do.	Ulcer of stomach.
XX.	M.	59	Pale.	Do.	Do.	Double.	Old tubercles, pulmonary abscess.	Healthy.	Do.	——
XXI.	M.	64	Full.	Do.	Pneumonia, anasarca.	Do.	Pneumonia.	Do.	Healthy.	Cap. phleb. spleen, dis. aorta.
XXII.	M.	57	—	Do.	Apoplexy.	Right.	Œdema.	Gall stones.	Cysts.	Dis. aorta, cerebral hæmorrhage.
XXIII.	F.	64	Thin.	Temperate.	Fever.	Left.	Pneumonia.	Healthy.	Healthy.	Ulcer of rectum.
XXIV.	M.	45	Full.	Drunken.	Aneurism.	Double, chiefly left.	Healthy.	Do.	Cysts.	——
XXV.	M.	39	—	—	Anasarca, ascites.	Left, old disease of endocardium.	Œdema. Pleural effusion	Cirrhosis.	Cap. phlebitis.	Diseased aorta.*

[* See note, p. 33.

Respecting the cause of the degeneration, the questions to be solved by morbid anatomy limit themselves to these three :—

1. Is this fatty degeneration of the heart only a local expression of a general morbid tendency, displayed after a similar manner throughout the body ?

2. Is it a disease of the heart itself, independent of any general cachexia, only connected with some particular condition of some other organ ?

3. Is it essentially and primarily a disease of the heart ?

I. The existence of a general tendency to fatty degeneration may be manifested in the ordinary seats of fatty accumulations, and in internal organs, as the heart, lungs, (Med. Chir. Trans. xxxi. p. 297) liver, and kidneys. On the meaning of the term fatty degeneration with regard to the heart and liver, there is no need to dwell. And the conclusions here drawn from the condition of the lungs are so unimportant, that it is not worth while to inquire how far fatty degeneration of the lungs be a correct expression of what is commonly termed emphysema. A few words, however, seem to be required on the subject of granular degeneration of the kidney.

There seems no reason to hold that the anatomical terms of fatty degeneration and granular degeneration of the kidneys are exactly commensurate. The one expresses a very common and the other a very rare condition of the organ, and between the extreme manifestation of each respectively, the difference is as great as exists between a fatty and a granular liver. But as in the liver so in the kidney, there are numerous intermediate anatomical changes connecting the two conditions, and, pathologically speaking, the connection is very close between them. The same observations which make me, in all diffidence, hesitate to adopt entire Dr. G. Johnson's explanation of the essential nature of the disease, have made me sensible of the general importance and correctness of the views he has so ingeniously put forward. It is in the belief of the pathological connection of the diseases of the kidney enumerated in the table that they have severally been adduced ; and that seeming in their existence to trace the effects of the same morbid tendency which had manifested itself in the heart, I have thought to find evidence of the general nature of the disease.

Of the 23 cases in which the exact seat of the fatty degeneration has been noted, in

11 cases.	1, 4, 7, 8, 9, 15, 16, 17, 20, 21, 24, . . .	both ventricles were fatty.
7 ,,	6, 12, 13, 14, 18, 19, 22, ...	the right ventricle only.
5 ,,	5, 10, 11, 23, 25, ...	the left ,, ,,

And the cases which displayed fatty degeneration, or disease analogous or tending to fatty degeneration of the liver, kidneys, or lungs, together with those where the general condition of the body in this respect was noticed, may be distributed thus :—

	6 cases.	7, 8, 9, 15, 16, 23, ...	were thin.
	9 ,,	1, 2, 6, 10, 11, 17, 19, 21, 24, ...	,, full or fat.
a	1 ,,	25, ...	had cirrhous liver.
a	4 ,,	3, 4, 13, 15, ...	,, fatty liver.
b	1 ,,	13, ...	,, fatty kidneys.
b	2 ,,	22, 24, ...	,, cysts of the kidneys.
b	7 ,,	4, 7, 10, 14, 18, 19, 20, ...	,, granular kidneys.
c	7 ,,	1, 5, 6, 9, 10, 14, 18, ...	,, pulm. emphysema.

Combining the results of these tables :—

Of the cases where both sides of the heart had undergone fatty degeneration—

4 cases.	1, 17, 19, 24, ...	were fat or full.
5 ,,	7, 8, 9, 15, 16, ...	,, thin.
2 ,,	4, 15, ...	are of class *a*.
4 ,,	4, 7, 20, 24, ...	,, ,, *b*.
2 ,,	1, 9, ...	,, ,, *c*.

Of those where the right ventricle only was degenerated—

2 cases.	6, 19, ...	were fat or full.
0 ,,	. . . . .	,, thin
1 ,,	13, ...	is of class *a*.
5 ,,	13, 14, 18, 19, 22, ...	,, ,, *b*.
3 ,,	6, 14, 18, ...	,, ,, *c*.

Of those where the left ventricle only was degenerated—

2 cases.	10, 11, ...	were fat or full.
1 ,,	23, ...	,, thin.
1 ,,	25, ...	is of class *a*.
1 ,,	10, ...	,, ,, *b*.
2 ,,	5, 10, ...	,, ,, *c*.

One would naturally expect to find that the cases where the fatty degeneration of the heart was double would be those where the disease depended on a general tendency of this nature, and where it would manifest itself in fatty degeneration of other parts. Such, however, does not appear to be the fact; for, in the first place, more of the double cases were thin than fat; and next, not merely the proportional, but the absolute number of cases belonging to the different classes is less among the double than among the right cases.

On the general fat or thin nature of the subjects perhaps too much stress should not be laid. The texture and appearance of the body generally are more important than the amount of fat, in this point of view. And this could scarcely be reached by analysis, being a matter of individual observation; to be undertaken, too, in all probability, with preconceived notions, rather than the subject of physical experiment.

The greater proportion of cases of fatty degeneration of the different organs among the right than among the double cases demands a closer investigation. Probably the same cause which reduces the amount of renal disease of this kind so low among the left cases, operates also to reduce its numbers among the double cases. And this cause, as already noticed, is probably the obstruction to the circulation, necessitating increased action and hypertrophy of the left ventricle, under the circumstances. Why the same cause, obstruction to the circulation by emphysema of the lungs, is followed by degeneration rather than hypertrophy of the right side of the heart, would be hard to say; but that so it should be agrees with the result of general observation, that emphysema is more commonly followed by dilatation than by hypertrophy of the heart. It is difficult to understand how, consistently with this explanation, fatty degeneration should occasionally limit itself to that cavity of the heart which, under the particular circumstances, would be the least likely to suffer it. For instance, admitting the correctness of the explanation generally, how comes the left ventricle ever to be solely affected when there is granular disease of the kidney; or the right ventricle only, in connection with emphysema? The difficulty must remain unsolved, in the absence of cases fairly to meet it by analysis. I think, however, that it is not strong enough to vitiate the above conclusion, that the existence of granular degeneration tends to protect the left ventricle of the heart from atrophy.

Of about 500 dissections after death of all diseases, of which I have more or less complete records, upwards of 110 presentsd emphysema of the lungs, and about 40 fatty degeneration of the liver. It follows, on comparing these numbers with those in the above table, that pulmonary emphysema is not much more commonly met with in cases of fatty degeneration of the heart than in other cases, while the reverse holds good for fatty degeneration of the liver.* With regard to the affection of the kidneys, those who have most studied the subject will most readily accept my profession of my utter inability to determine the exact number of cases which have had granular disease of the kidneys. In the absence of any simple criterion by which to analyse the whole series, I can only say that I believe the proportion of cases where the kidneys are affected in one or other of the manners described in the table, to all my cases, is about one in four or five. The proportion displayed above, of ten to twenty-five, points to a close connection which I believe to exist between the general cause of these conditions of the kidney and the degeneration of the heart. But nearer the truth than this general expression of my opinion I cannot approximate.

Five cases are noticed (V., XIV., XXI., XXII., XXV.) as having presented extreme disease of the aorta. My notes generally do not mention disease of the aorta unless of an extreme degree. The proportion presenting this degree in all diseases is less than one in fifteen; in this disease it is one in five cases. Regarding the morbid changes in the aorta as all of a similar nature, expressed by the term "atheromatous," this coincidence supplies another instance of the connection between fatty degeneration of the heart and that of other organs: in any case, of its connection with disease of the arteries.

II. The local changes whose connection as such with fatty degeneration of the heart it appears worth while to investigate, are those of the lungs and alimentary canal; emphysema and tubercle of the one, and ulceration of the other.

As far as we may conclude from the following low numbers, phthisis cannot be looked upon as exerting any influence in this point of view, and emphysema only a very feeble one. More generally expressed, it does not appear that obstructions to the pulmonary circulation determine the seat of the degeneration in cases otherwise predisposed to it.

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* It is a very interesting, though somewhat unsafe practice, to examine recorded cases of disease with the real or fancied light of recent investigations. The e der Dr. Latham published a paper on certain symptoms usually, but not always, denoting angina pectoris. (Trans. Coll. Phys., vol. iv., p. 278.) He quotes two cases of sudden death after such symptoms, where the liver was much enlarged. To this enlargement he attributed the symptoms. Is it not probable that the large liver was a fatty liver, accompanying fatty degeneration of the heart; and that in this last lay the cause of the pain, the slow pulse, and the paroxysms ending in sudden death?

7 cases.	1, 5, 6, 9, 10, 14, 18,	had emphysema.
4 ,,	10, 11, 12, 13,	,, phthisis.*

Of these 10 cases—

2	1, 9,	had degeneration of	both ventricles.
5	6, 12, 13, 14, 18,	,, ,,	the right.
3	5, 10, 11,	,, ,,	the left.

It would, perhaps, be most correct to consider ulceration of the stomach as another manifestation of the same general morbid tendency which had affected the heart, in however different a way. Let it be enough, in the absence of means of further tracing this connection, simply to express the frequency of the coincidence of the two forms of disease. After deaths from all causes, less than four per cent. presented ulceration (excluding follicular and cancerous ulcers) of the stomach or duodenum. Here three cases out of twenty-five (VIII., X., XIX.), or twelve per cent. were thus affected.†

III. Lastly, how far does morbid anatomy justify the claim of this disease of the heart to be considered as primary—as essentially a disease of the heart? On such grounds as have been discussed above, it would be rash to attempt to prove a negative; but it is evident that such a view derives no countenance from these cases. It is very unlikely that fatty degeneration to such a degree as to impair its functions should take place in the heart alone, which is of all organs, by the unceasing nature of its functions, the least liable to it. And the coincidence of fatty degeneration of other organs would destroy the claim of the degeneration of the heart to be considered in this light. Perhaps the distinction is scarcely just, which placing, under such circumstances, the cause of the fatty degeneration in the blood, and denying to the manifestation of its effects in the heart the claim of being primarily a disease of that organ, should allow that claim to inflammation of the muscular structure of the heart. This, however it may be classed, is a condition to which the incessant action of the heart renders it, beyond all muscles, peculiarly liable; and, as a consequence of this, I believe that fatty degeneration of the heart, unconnected with disease elsewhere, is to be especially looked for. The above table, however, supplies no such instance.

In one other manner might this fatty degeneration begin at the heart itself—namely, as a consequence of obstruction of the coronary vessels. It may be sufficient to remark that in extreme cases of this form of disease I have found the coronary vessels healthy, and have found them diseased where there is no record of the structure of the heart having been thus changed. M. Bizot* says that disease of the coronary arteries is much more common in men than in women, —the reverse, it will be recollected, of the comparative frequency of the occurrence of the form of fatty heart which he describes in the two sexes respectively. The inference is against disease of the coronary arteries having anything directly to do with fatty degeneration of the heart; but the probability, it must be confessed, of their direct connection is very great.

There are some points yet remaining, fitter perhaps for consideration on general grounds than by analysis. How are we to distinguish the existence of this disease during life? and how, when we have distinguished it, are we to prevent its further progress? With regard to the first question, the most extreme cases detailed may show that the diagnosis, on general or physical grounds, is almost impossible. One of them was a strong athletic man, a pattern of apparent health and strength, till within four days of his almost momentary decease; and several others had their hearts carefully examined during life, in the inquiry after cardiac disease, without anything abnormal having been noticed to arouse a suspicion of the existence of this particular disease. And, after this negative answer of the first question, it might seem useless to pursue any further the second, as to treatment. But we need not contentedly sit down and add this to our other medical problems which can only be solved by dissection, and have no interest to any one but the morbid anatomist; for though, abstractedly, we know so little about it, yet its bearing on other diseases is very important. And when its recognition becomes more general, with the more general use of the microscope, it may prove to be the key to much of cardiac pathology. Let us look at it, in conclusion, in this point of view.

We cannot, it is true, predict with certainty in any case that we shall find this lesion after death, but it is difficult for any

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* One case (XX.) has been omitted. There were only a few old chalky tubercles in his lungs, obviously insufficient to affect the condition of the heart in the present point of view.

† The connection of ulceration of the stomach and fatty degeneration of the heart through general cachexia is supported by Mr. Crisp's table of cases (*Lancet*, 1842-3, vol. ii. p. 639), showing the connection of this affection of the mucous membrane of the stomach with "a chlorotic condition of system."

* Op. cit. p. 384.

pathological observer not to be led to suspect the existence of a disease on the repetition of the same circumstances under which he has seen it occur previously.

In three of the cases above detailed (VII. VIII. IX.) of lingering illness, this condition of the heart has been met with in a striking degree—too striking to justify the unqualified inference that it resulted either from debility, or from inaction of the heart; for, if this were so, why should not the same changes be found more frequently under similar circumstances? These patients kept about, or lay in bed, slowly sinking, and nothing that was tried did them any good. Every organ was scrutinized, every rational suggestion (in one of them at least, where time allowed) was acted on, but all to no purpose: they quietly sank and died. Could one watch another similar case during life, and not suspect the existence of this lesion? Would not one be strengthened, by the examination of such another case after death, in the conviction that the state of the heart, or that which caused the state of the heart, had something to do with the inability to rally from the apparent disease?

Apart from all other considerations, this form of disease is, I think, less likely to occur, under such circumstances, in private than in hospital practice. For the treatment of acute disease, it is quite impossible to be better placed than as an inmate of one of our large hospitals: but during the dawn of convalescence, when little or nothing remains to be done, the contrast between private and hospital practice is very great; for hospital patients sadly miss all those kind attentions and encouragements which only friends can administer, and those only who have been sick can appreciate the comfort of. The suspicion of the existence of such a lesion should make us most assiduous, not only in the exhibition of tonics, more particularly in the form of iron, but also in the use of those mental appliances the want of which makes the spirits (may I say the heart?) flag, and allows the organs of active life to remain in a passive condition. This is common ground, on which the moralist and morbid anatomist might meet. A broken heart may not be so very incorrect an expression of the effects of intense grief, after all.

Another point to be considered in connection with this disease is the perplexed pathology of angina pectoris. The common cause of suffering and of death in this disease is perhaps correctly expressed by Dr. Latham and Dr. Heberden as spasm;* but the circumstances under which this spasm occurs are so various that we can scarcely consider any one of them as a very frequent or very influential cause. This uncertainty might well be shown by collating the diverse opinions of different physicians on this subject;* but it appears better to quote at length the experience of one physician who has seen occasion to follow diametrically opposite plans in different cases.

Dr. Latham says,† "There have been cases in which my treatment of angina pectoris, in the intervals of the paroxysms, has chiefly turned upon reducing the nutritious and stimulant quality of the patient's diet, abridging his animal food, and denying him wine and fermented drinks altogether. There was one case, and only one, in which I was driven to draw blood more than once from the arm—an unusual and a hard necessity! There have been more cases, on the other hand, in which the general habit of the patient has made me fearful of withdrawing support, and experience has shown me the need of supplying a well-regulated amount of stimulus in the shape of wine daily. The administration of steel in the intervals of the paroxysms has (I have convinced myself) in some instances been instrumental to their postponement."

My own experience on this subject is so limited that I forbear to urge it: I would content myself with comparing the inferences obtained from the facts adduced in this paper with the results of treatment which Dr. Latham has summarily expressed. How closely the two divisions of fatty degeneration of the heart appear to coincide with the two practical divisions of the patients! The wine and steel are for the soft, flabby, fatty hearts; the strict regimen and depletion for those where the fatty degeneration is secondary to inflammation.

The subject is too important, and bids too fair to have at least this view of it satisfactorily examined, that I should here endeavour to strain any facts in support of this explanation. It is true that fatty degeneration of the heart cannot have angina pectoris set down as one of its usual symptoms; for out of all the cases above detailed, not one, properly speaking, has presented it. But, on the other hand, it is impossible to read any collection of cases of angina pectoris without sharing in some degree Dr. Fothergill's opinion, without feeling how much further fatty degeneration goes to explain the symptoms than does any other morbid change usually found on dissection.

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* Latham on Diseases of the Heart, vol. ii. p. 386. Heberden, Comment. de Morb. Cur. § lxx. p. 275.

* See a most comprehensive essay by Dr. Forbes, Cyc. Pract. Med., Angina Pectoris, for a summary of all that is known on the subject; with the two lectures of Dr. Latham above referred to.

† Op. cit. p. 406.

[*Note to Table on page* 28.—Dr. Kirkes, who has kindly communicated to me the particulars of the two last cases, says:—The heart was small and flaccid: the muscular substance, especially of the left ventricle, had a dirty brown unhealthy aspect. Near the apex one part of the wall of the left ventricle felt peculiarly soft, and, when cut into, the muscular substance of that part was found to be changed into a tough yellowish substance, not unlike tubercle. Within, scarcely any part of the ventricle was found free from this morbid change, occurring in streaks or patches of a yellowish white colour. To this extent, under the microscope, the fibres of the heart presented a confused granular appearance, and the fibrils next the yellow markings had no transverse striæ. There was much oily matter hereabouts. The degeneration was so extreme that at first I thought there must be some tubercular or other deposit, but I satisfied myself that it was all fatty, and entirely confined to the left ventricle.]

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