NOTES AND OBSERVATIONS ON DISEASES OF THE HEART AND LUNGS.

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[*Continued from page 8 of number for Jan. 6th.*]

Besides the instances of debility of impulse, with an abnormal increase of dulness, previously referred to, there is yet another class, of apparently similar cases, to be considered. This class comprises those cases whereby an abnormal dulness is induced by physical complications external, but contiguous, to the heart—such as morbid deposits in the pericardium and pleura, the occurrence of tumours, etc. In these cases, though there be a diminished impulse associated with an abnormal dulness on percussion, it does not necessarily follow there is disease in the heart itself, or even that there is really a failure of impulse; it may be only masked. It is, therefore, necessary to ascertain if any of these sources of interference with the due appreciation of the normal impulse exist. In cases of pericardial effusion, though the heart's impulse be really not impaired, it may be so masked as in some cases to be scarcely appreciable, or even to be rendered completely and entirely imperceptible. This occurs more especially in the course of passive effusions rather than from effusions caused by active inflammation.

In these cases of pericardial effusion, the general indications of this diseased condition must be sought for. The cardiac region may be arched forwards, and the intercostal spaces be obliterated by the bulging outwards of the integuments, and perhaps the characteristics of an œdema may be presented. The apex-beat, slight though it be, may be seen raised so as to range with a line even with the nipple. The hand laid over the surface discovers little or no movement; if there be any, it is weak and of varying force, but not undulatory. The undulatory movement is rather the accompaniment of dilatation of the cavities of the heart itself, especially of the right ventricle, when occurring without effusion.

The dulness on percussion is very much more marked in these cases of effusion than in the cases previously referred to. It is also widened in area, and may even range above the right rib. It has, as Dr. Walshe points out, a tolerably uniform pyramidal shape; its base ranging with the sixth, rarely with the seventh, rib, and extending its apex upwards, according to the extent of the fluid. At the apex the sounds are often nearly, if not entirely, imperceptible. Should the first sound be heard, it will be weak and uncertain, with a muffled character, and anticipates the feeble indication of the impulse. At the base, the sounds, though they be faint, are audible and distinguishable: the first sound is here certainly louder than it is over the apex or the ventricle; the second sound is audible through the course of the arch of the aorta. Besides the above, there may also be the more specific indications of the existence of serous inflammation of the pericardium, by the presence of friction-sounds, and of the existence of fluid by the presence of ægophony at the edge of the dulness. The impulse of the heart may also be obscured by an empyema, by pleuritic effusions, by deposits (solid and fluid) in the mediastinum, and by other contiguous tumours; but these are all causes which obviously present themselves for observation and consideration on account of their own specific lesions, rather than in relation to the accompanying condition of a diminished impulse.

We will now consider the existence of a diminished impulse under entirely different conditions. An impulse, feeble and very limited in extent, sometimes exists with an evidently very deficient area of percussion-dulness, both superficial and deep, and this evidently without any emphysema or other cause to interfere with the correctly ascertaining that the size of the heart is abnormally diminished. The cardiac sounds are everywhere at a minimum; but, if heard, may be a little sharper in tone than natural; the pulse is small and deficient in force. This state of things indicates a condition of muscular atrophy. If there be frequency and some palpitation in the heart's action, it is probably due to the atrophy being caused by, or associated with, a generally depraved condition of the blood, as in tubercular degenerations or carcinoma. In these cases the heart is not only diminished in size, but its fibres are degenerated, being pale, soft, and deficient in firmness. This may be simply caused by an anæmic state of the viscus, but it is more commonly due to a fatty degeneration of the fibres, and which is evidenced, not by the deposit of separate fatty masses, but by the heart exhibiting both on its external and internal surfaces the appearance of buffy spots; and, as Dr. Quain summarily states, "paleness, softness, peculiar mottling and friability of texture". This fatty degeneration not only causes weakness, by its metamorphosis of fibre into fat, but may also, in some rare instances, cause diminution in bulk.

This state of atrophy may be simple, as seen in phthisis and carcinoma, or in diseases where there is a general wasting of the muscular structure of the body; or it may be due to the specific wasting associated with pericardial adhesions—occurring not infrequently after the acute form of pericarditis; or it may be due to the specific metamorphosis of the fibres. These conditions of the heart, now weakened in power and diminished in bulk, offer important and fatal indications.

It is not easy in these several cases of anæmia, of simple muscular atrophy, or of fatty metamorphosis, to discriminate one from the other solely by the physical phenomena exhibited by the heart. We must seek to determine the precise form of affection rather from the general symptoms; but these, with their etiology, had better be referred to when considering the morbid states of the heart in the category of structural diseases next to be described, which comprises the morbid structural changes specified above, but unaccompanied by any diminution in the heart's bulk.

This category includes the very important range of cases, as regards diagnosis, in which the heart, though the impulse be feeble, is not enlarged, nor involved in any surrounding or contiguous dulness, in which the indications are that the heart, though weak, is in fact of the normal size. These cases may be due to structural, or only to purely dynamic, causes. Between these two classes of cases it will be well to discriminate, and then to find out in the latter where we may anticipate neither danger nor cause for alarm; *i.e.*, the existence of a normal condition, or of functional debility only. The character of the impulse, when the heart is feeble and small in size, has just been described. Should this character be well marked, though there be no indications of a want of proper bulk, and there be, in addition, an impulse always reduced in strength, and only very rarely exhibiting a visible *ictus*, and this rather having the appearance of an undulation than of a beat; while the sounds, the first being sharp and flapping, the second very thin, are generally very weak and toneless, and entirely free from any complication of murmur; with a pulse irregular in force or rhythm, but for the most part slow, we may surmise there is muscular weakness. Should the pulse be rather fast than otherwise, this weakness is then probably due to a state of anæmia. If, under these circumstances, the impulse of the heart, though generally feeble, be occasionally characterised by a forcible beat, it is very probable these conditions are associated with a concentric hypertrophy of weakened tissue. Should we, however, find these specific indications of muscular or anæmic weakness greatly exaggerated, so that there is a variable impulse, always feeble, but with the character of a fluttering unsteadiness, indicative of great variability both in force and rhythm—at times even jerking and abrupt—and with the heart, as it were, largely projected forwards; the first sound flapping, short, weak, and toneless, and then obtuse and dull; the second weak and thin, no murmur, no jugular pulsation; the pulse at the same time weak and irregular, with loss of power, and little or no tendency to general or local arterial or venous congestion: if with all these the apex-beat, though difficult to define, be yet more distinct than in the cases of muscular or anæmic weakness, we may infer the presence of debility from fatty metamorphosis.

The general symptoms characterising the two categories of disease above referred to—viz., debility of impulse, with diminished or with normal size—are those of physical weakness and easily induced breathlessness. In muscular atrophy, the feeling of weakness is immediately experienced on the exercise of any undue exertion; and the patient, for the most part, shows a disinclination to submit himself to the test. In anæmia, there is a more general depression of vital force, and the breathlessness is more marked and more persistent than in simple muscular weakness. It is essentially a disease of young life, and associated with the nervous and hysterical temperaments, and is met with more commonly in females than in males. By far the greater proportion, however, of these cases of weak impulse are due to a fatty degeneration or metamorphosis of the muscular fibres; and this, unlike that of anæmia, is rather a disease of males than of females, and in them occurring at a more advanced period of life—being rarely met with in those under forty years of age. It is less a disease of the labouring classes than of the well-fed. This, probably, is to be explained by the heart in the former, if predisposed to this form of degeneration, early taking on the character of hypertrophy or dilatation; but in whatever class it may occur there will be found associated with it a pale flabby skin, indicative of the constitutional tendency. When a state of fatty metamorphosis of the heart exists, the countenance is usually pale and sallow; sometimes the lips are livid, generally deficient in the natural colour; the tissues are soft, the manner is languid, the temper is apt to be dejected, and there is a sadness and melancholy foreboding not commonly met with in many other affections of the heart; muscular power is deficient and soon exhausted, and the effort to exertion is not very promptly aroused; there is evidently a depression of vital force; the appetite is feeble, digestion is weak and flatulent. In advanced cases,

there is marked uneasiness across the loins, and the feet and ankles show slight œdema; cardiac uneasiness, at times passing into pain, often supervenes, with occasional sensations of a palpitation, or rather flutter; and if this increase, often accompanied by a choking feeling, somewhat of the nature of a globus hystericus. The respiration is irregular, often hurried, and frequently sighing. When the right ventricle is prominently the seat of weakness, the respiration may present peculiar characters, as described by Dr. Cheyne (*Dublin Hospital Reports*, vol. ii, p. 217), in which a protracted period of apnœa is slowly recovered from to be followed by a few hurried respirations. The apnœa, as shown by Dr. Reid, is associated with an increased cardiac action, as evidenced by acceleration of the pulse, followed by remissions during the hurry of the breathing. In the case described by Dr. Cheyne, the several phenomena occupied about a minute, and occurred in a case of advanced disease. It is probably a symptom solely due to very advanced disease. In extreme cases the memory often is notably impaired; and mental efforts not only exhaust but induce irritability, and are not without risk to life. The tendency to feel faint becomes more marked, and is often accompanied with a distressing and alarming vertigo, and this vertigo sometimes heralds a more decided disturbance of the nervous system, so that something of the nature of a convulsion may take place. It does not present the features of an epileptic convulsion, but rather of a violent spasmodic struggle for life.

It has been advanced by Mr. Canton, that the appearance of the arcus senilis is concomitant with, and as such to be considered a symptom of, fatty metamorphosis of the heart. My own observation does not lead me to confirm this view. I have seen many cases of fatty degeneration where it has not occurred, while in many in which it has been strongly marked there has obviously not been the least tendency to a fatty metamorphosis.

On comparing these general symptoms of an anæmic or fatty heart accompanied with an enfeebled impulse, with those that characterise weakness of impulse accompanied by evidences of dilatation, it will be seen that they are in many respects similar; still there are differences. In the anæmic and small fatty degenerated heart there is less evidence of distant local congestion; the lungs and the portal system are not so prone to be loaded, and hæmorrhages rarely or never occur; and œdema is only rarely, if at all, met with. The difficulties of breathing are also less the result of congestion than of the obviously nervous debility. There is also a more extreme state of general physical weakness, and this gives a character to the occasional sensations of faintness and the less marked convulsive character of the attacks that occasionally supervene upon the occurrence of vertigo, and in the entire absence of valvular murmurs.

The proximate cause of the whole of the symptoms which characterise this class of heart-disease must be found in deficiency of dynamic power, so that the heart is unable to effect other than a feebly expressed contraction on the blood which it has to propel ordinarily, and an utter incapacity to do anything beyond this without great general distress. If it be called on to do this excessive work, its action immediately becomes hurried and irregular, and with the result of acquiring a still greater incapacity than belonged to it in its previously quiescent state. The remote cause is evidently deficiency and alteration of structure, an attenuated or degenerated condition of the muscular fibres.

The consideration of these anæmic, enfeebled, or degenerated forms of heart-disease is not only interesting but important, from their association with pre-existent diseases, and also as influencing other and more serious lesions in the heart itself. Doubtless, each of these forms of disease may be fatal in themselves; but, for the most part, they are to be considered, in the former case, as aiding in the general depreciation of the powers of life, and, in the latter, as the forerunners of those other more advanced conditions of disease, as dilatation, hypertrophy, and perhaps valvular inefficiency. Simple muscular debility is more apt to pass into excentric hypertrophy, while fatty metamorphosis rather determines to simple, if not to concentric, hypertrophy. Both of these lesions are, for the most part, slow in their progress, and in the course of events, may often threaten the termination of life; still, under favourable circumstances, they are consistent with prolonged life. For the most part muscular debility, as it proceeds to excentric dilatation, leads to visceral congestions, which apparently kill, rather than the original heart-disease; but, in the case of fatty metamorphosis, death is, for the most part, determined by dynamic failure, or even by rupture, of the heart itself.

It is obvious, from the nature of the symptoms, and the anatomical condition of the heart which produces them, that the prophylactic management of these cases mainly consists in quiet, in the avoidance of all excitement, bodily and mental, while the system generally is sought to be improved and invigorated by fresh air and a generous diet.

The medical treatment, as indicated by the evident feebleness of the general system, and of the heart in particular, finds employment in light preparations of iron, in antispasmodics, and stimulants; the occasional use of mineral acids with digitalis is often most beneficial; and, at times, sedatives, in the forms of opium, hyoscyamus, hydrocyanic acid, and chloroform, are useful. The associated attacks of vertigo or faintness must be met by the stronger restoratives of æther, ammonia, or brandy.

These symptoms of a deficient impulse without evidence of any increased size in the volume of the heart, occasionally develop themselves in an active form, and suddenly; sometimes, as it were, idiopathically or as symptomatic of an inflammatory condition of the substance of the heart, or by way of a metastasis, or during the course of other diseases. Illustrations of these several sources of a deficient impulse are, occasionally, to be observed in the course of certain forms of idiopathic febrile conditions of the heart itself, in pericarditis, in gouty metastasis, and as a concomitant of typhus, typhoid, and scarlet fevers.

The physical examination of the heart in these cases reveals, perhaps, little more than the negative evidence of a deficient impulse, while the valvular sounds are often very indistinct, or are merged into one feeble sharp sound; the only peculiarity being that, during the frequently recurring paroxysms of cardiac distress, which for the most part accompany these affections, there is an increase of the urgent symptoms, both impulse and sounds being almost or entirely suspended. This is peculiarly the case in idiopathic inflammations, and in gouty or other metastases. In these the frequent recurrence of paroxysms of varying duration and intensity is peculiarly a characteristic. In fevers this is also appreciable, but not to so marked an extent, as the remissions are not so readily recognisable.

The rhythm of the heart's action in these cases is generally, but not always, accelerated. In some cases of idiopathic inflammation, and more especially where there is a gouty metastasis, it is occasionally slower than natural. The pulse is invariably small and thin.

In the examination of these latter cases by the stethoscope, an obvious uneasiness is often induced when pressure is made over the region of the heart. There are reasons, however, for concluding that pain is not experienced in the heart itself, but in the surrounding tissues, which, sympathising in the disordered state of this organ, thus are prompted to protect it, and render immediately obvious the necessity for its being unmolested and kept quiet.

The general symptoms in these cases of asthenic inflammation, whether idiopathic or due to a gouty metastasis, are characterised by præcordial anxiety; an uneasiness rather than pain; the complexion assumes a sordid or livid aspect, with occasional flushings. The countenance has a varying expression of distress; there is a small, thin, and subdued pulse, with restlessness both of mind and body, more especially of the latter; excessive faintness is induced in any than the recumbent position, and yet a restlessness induces the patient to seek change from it. To the patient these symptoms excite sensations of anxious alarm; to the experienced observer they do not fail to indicate the reality of the danger, and that the result may too often be fatal, and that rapidly, occurring often, as it were, suddenly, or on some very slight exertion.

The physical condition, which is the immediate cause of these symptoms, is probably due to a sudden nervous depression in the vital powers of the heart, caused by the asthenic forms of inflammation, to which, whether idiopathically or by metastasis, it has become subject. Probably no very notable changes in its structure can be detected; but should the fatal termination be postponed, evidences of a softened structure are usually to be observed.

When these symptoms present themselves in typhus and typhoid fever, they usually occur early after fever has fully developed itself, becoming appreciable, about the sixth day, by a sudden and notable prostration of power. The loss of impulse, on physical examination, is first to be detected at the apex (Stokes, p. 377) and to the left side. Sometimes the impulse becomes inappreciable everywhere; occasionally, but not always, the valvular sounds are also inaudible. In favourable cases, after the expiration of eight days from the time these symptoms have set in,—*i.e.*, the fourteenth of the fever—the heart shows indications of rallying, by a return of the impulse, and then of the valvular sounds. Recovery is always first indicated by a restoration of the impulse; it is the first to fail, and the first to be restored. The pulse, during this period, is always small, weak, and greatly accelerated.

In all these cases the tonicity of the heart's power is rapidly lessened, or even destroyed; and this is due to softening of its fibre; so much so, as in some cases, to disintegration of its structure. No alteration in volume takes place, but the left ventricle, which is the essential seat of the diseased condition, becomes livid in hue, soft in texture, and with so little power of resistance as to receive the impression of the finger pressed upon it as if œdematous; in parts all trace of muscular fibre is obliterated, and a dark homogeneous-like looking structure takes its place, resembling somewhat the cortical portion of the kidney.

The treatment of the above two classes of cases is not identical. In continued fever, when the powers of the heart are thus impaired, sustained stimulation may be useful, but certainly not always so; for, frequently, notwithstanding the prevailing reliance on stimulants in these cases, they are not only uncongenial to the patient, but evidently foment the disease by adding to an injurious irritability. Milk, light animal drinks, etc., are more often suitable. Still some use of stimulants is, for the most part, required. In the cases of idiopathic asthenic inflammation of the heart, and in metastasis from gout and other diseases, stimulants are not well borne, in fact their exhibition, more often than otherwise, both induces repetition of the paroxysms and an increase in the severity of the symptoms that characterise them. Sedatives, alkalis, with light drinks, appear the more appropriate remedies.

[*To be continued.*]

ON THE NUTRITION OF MUSCULAR AND PULMONARY TISSUE IN HEALTH AND IN PHTHISIS.*

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On the Nutrition of Muscular Tissue in Phthisis.

On considering the state of emaciation, and consequently the deficient nutrition, constantly met with in advanced phthisis, I had been prepared for a result, from the analysis of the flesh of consumptive subjects, very different from that obtained from the investigation of the healthy tissue. Such, however, was not the case; and, although there was some difference found in the absolute composition of the two kinds of muscular tissue, still the numerical relations were very much alike, the phosphoric acid and potash effete in flesh after death from consumption bearing the same relative proportion as they do in pyrophosphate of potash.

The following is a tabular statement of one of my analyses of human muscular tissue after death from phthisis. Two other analyses have yielded similar results.

On 200 Grammes Human Muscular Tissue, after Death from Phthisis.

	Class I. Muscular Tissue proper.	Class II. Nutritive Material.	Class III. Effete Material.
Albumen	20.520	4.770	2.700
Phosphoric acid	0.160	0.037	0.318 }
Potash	0.135	0.008	0.418 }
Soda	(0.021)	(0.005)	0.389—total.

On 200 *grammes* Flesh.		Found.	Theory.
Water	166.5	Phosphoric acid	43.2 .... 43 } Pyroph. of
Fat	3.64	Potash	56.8 .... 57 } potash.

It will be observed in this table, that the amount of phosphoric acid and potash of the effete material again occurs in the proportions necessary for the formation of a pyrophosphate, although the absolute quantity of these substances present is less than in the case of healthy flesh. There is also less albumen, phosphoric acid, and potash, in the tissue proper and nutritive material, than in the corresponding classes of constituents of healthy muscle; the difference for 200 *grammes* of flesh being made up by an increase of water, chlorine, and soda, or chloride of sodium. The excess of water, however, is but slight, amounting only to 6 per cent. of the tissue. With respect to the state of the water in muscles after death from consumption, I have observed that, as a rule, they show a certain degree of dampness, and are sometimes quite wet, instead of exhibiting the dry appearance of healthy muscular tissue; and from this circumstance it appears to me as if the water of the muscles of consumptive subjects had lost to a certain extent its colloid attraction for the solid portion of the tissue. The increase of chloride of sodium might partly account for this, as I have observed that, should a solution of this substance, of a certain strength, be added to a jelly of isinglass, it causes the jelly to soften down and become nearly quite fluid. The mean quantity of chloride of sodium in 200 *grammes* of muscular tissue (from eleven analyses) of consumptive subjects amounted to 0.634 *gramme;* the whole of the chlorine being considered as combined with sodium (which is a probable assumption); while the mean quantity of chloride of sodium in 200 *grammes* of muscular tissue from healthy flesh (from eleven analyses) amounted only to 0.275 *gramme* (one analysis of human flesh yielded 0.300 *gramme* of chloride of sodium)—showing that muscular tissue after death from phthisis contains more than twice the proportion of salt with which it is supplied in health.* This fact is remarkable, appearing to show that the force which in health retains chloride of sodium in blood, or checks its passage into flesh, is weakened in phthisis; and therefore, in accordance with the high degree of diffusibility of common salt, it finds its way from blood into flesh apparently in virtue of a physical property kept in abeyance during health.

Taking into account the whole result of the analysis of muscular tissue in health and after death from consumption, we find its colloid condition to be lessened in several respects in the present disease; and this appears to account to a certain extent for its deficient nutrition. A somewhat similar conclusion was obtained respecting pulmonary tissue.

Composition and Nutrition of Tubercular Pulmonary Tissue (consolidated and softening).

On considering a portion of a tubercular lung partly consolidated and partly undergoing the process of softening, it appears at first sight impossible to obtain for this mass of diseased tissue anything like a fixed chemical composition. However, by means of the method of analysis which I had applied to the investigation of the constitution and composition of muscle and healthy pulmonary tissue, I succeeded in obtaining what may be considered as the true chemical constitution of tubercular lungs in the above condition. The results have been entered into the following table, which shows the mean composition of three different samples of material analysed, taken from three different subjects.

Mean Composition of Consolidated (Cheesy) and Softening Tubercular Pulmonary Tissue (mean of three Analyses) on 200 grammes.

	Class I. Material (insoluble and colloid.)	Class II. Nutritive Material.	Class III. Effete Material.
Albumen	15.720	7.060	2.388
Phosphoric acid	0.289	0.130	0.276
Potash	0.027	0.012	0.302
Chlorine	——	——	0.452
Soda	(0.052)	(0.023)	0.544—total.

Water	On 200 *grammes*,	165 *grammes*.	
Fat	,,	,,	3.91 ,,

The proportions for the phosphoric acid and potash effete were per cent.:

	I.	II.	III.	Mean.	Theory.
Phosphoric acid	44.7	46.8	50.8	47.7	43 } Pyroph. of
Potash	55.3	53.2	49.2	52.3	57 } potash.
	100.0	100.0	100.0	100.0	100

Hence, in tubercular pulmonary tissue, the proportions of phosphoric acid and potash *effete* are not far from those which a pyrophosphate would require; and it may be concluded that a process of nutrition does indeed take place in tubercular matter; or, in other words, that tubercular matter is undergoing waste and renewal. This accounts for the fact that tubercular lungs are seldom or never found to emit a smell of putrefaction when examined soon enough after death. The composition of this material points to a deficient colloid condition, from a slight increase in the proportion of water, and from a low proportion of the colloid constituents—albumen, phosphoric acid, and potash; while that of the crystalloids chlorine and sodium remains much the same as in health. The most obvious indication, however, of a loss of colloid state, is the physical condition of the mass, which, on being minced, becomes transformed into a wet paste, wanting in consistence, and in which the water has clearly very little colloid attraction for the solid material; and it is interesting to observe that this wet paste contains but very little more water (3.5 per cent.) than healthy tissue.

Death from consumption, except when caused by direct asphyxia, which is seldom the case, is therefore apparently the result of a diseased state of the nutrition of the muscles and lungs, and probably also of the other tissues, which is due to a gradual loss of their colloid condition.

This change in the properties of tissues in consumption may be con-

* Concluded from page 152 of number for February 10th.
This communication may be considered as the abstract of two papers. One of them was read to the Royal Society last April; and the other, at the meeting of the British Association at Edinburgh in August of last year.

* There is more soda present in muscular tissue than is required for the formation of chloride of sodium with the chlorine. The determinations yielded on 200 *grammes*.

	Mean in health.	Mean in phthisis.
Chlorine	0.167	0.385
Soda	0.239	0.446