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A CASE OF FIBROID DISEASE OF THE HEART.

WITH OBSERVATIONS UPON THE GENERAL PATHOLOGY OF FIBROSIS.

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[*A Paper read before the Medico-Chirurgical Society of Montreal.*]

GENTLEMEN,—As this Society combines the features of a clinical and pathological, as well as of a medico-chirurgical and obstetrical society, it may not be out of place to bring before its members the clinical and pathological facts of a case of Fibroid Transformation of the Heart which not long ago has been under my care. Although not a very rare affection, it is a comparatively infrequent one, so that private practitioners have not many opportunities of seeing an example of the disease, which, moreover, is usually, as it was in this very instance, mistaken for fatty degeneration of the heart.

Mr. H. H. W., aged 60, of sedentary habits for the last 15 years, but previously a very active merchant and politician, consulted me first on the 11th September, 1874, respecting an attack he had had for the first time a night or two before. About bedtime he was suddenly seized with urgent dyspnœa, threatening suffocation, as it appeared to him; an incessant cough followed promptly, and in 15 or 20 minutes he expectorated large quantities of very frothy white mucus, and the orthopnœa disappeared.

His complexion was rather waxy-looking, features anxious, and his general appearance suggested failing health.

On enquiry, I learned that over 30 years before he had had a cough, attended with other symptoms of such import, that his physician had ordered a sea voyage, which he took, and returned in a few months quite recovered; that with that exception he had never had any serious illness, and, especially, had not had rheumatic fever; that about 8 years ago, after walking half-a-mile up a slight incline to the St. Anne's station, he experienced for the first time great dyspnœa, with distress at the precordia, and from that time felt it necessary to take his time in walking, so that he occasionally missed the train, although he had arranged his breakfast hour so as to secure sufficient time for his morning walk to the station. Upon another occasion, two years later, when walking up Beaver Hall Hill, he was obliged to stop for some time from dyspnœa and precordial distress. His then medical attendant advised him to avoid going up hills and fast walking, advice which he has since then observed.

On careful examination of the heart at the time of his first visit, I found the impulse and sounds feeble, and a faint systolic murmur confined to the region of the left apex; made a record of "weak heart," and prescribed treatment in accordance with that view. The sudden attacks of dyspnœa above described recurred several times during '75 and '76, and he had on two or three occasions attacks of what seemed to be mild bronchitis. His breathing continued to be somewhat short in walking, and he used a lift in going to the upper rooms of the building in which his office was.

Re-examined in October, '75; the signs of "weak heart" were observed. In March, '76, the nitrite of amyl was prescribed to relieve short attacks of dyspnœa and cardiac distress; and quinine, with nux vomica, were given. In May, '76, he consulted me for a cough which had recently supervened, and took with benefit the hypophosphite of soda, ammonia and lactucarium. He had been free from the sudden attacks of orthopnœa for a considerable time, but not in other respects as he had been, and I regarded him as the subject of fatty degeneration of the heart.

He returned to the city from his summer residence (in Lachine) on the 17th September, '76, and sent for me on the 18th. His condition was then as follows: Marked œdema of both legs to knees; none of scrotum; moderate ascites; physical signs of effusion in the lower half of the right pleura; not a sign of any in the left; cardiac impulse feeble, apex beat just within line of nipple at 6th rib; superficial cardiac dulness somewhat increased to left; cardiac sounds feeble, and *free* from murmur. Complexion very waxy and sallow; pulse very compressible, of medium volume, and at times intermitting; one or two beats in the minute omitted. He says that his ankles have been swelling for several weeks past, that his breathing has been gradually becoming more difficult, and he has been inclined to sleep chiefly upon the right side. He has not had a sudden attack of pain in it, or any illness suggestive of pleurisy. Urine rather scanty, very high colored, and depositing purplish-pink lithates abundantly; contains moderate amount of albumen. The diagnosis then made was "right hydro-thorax, ascites and anasarca, probably due to weak, fatty heart; no valvular lesion present."

The treatment employed consisted of digitalis, in combination with saline diuretics, and these agents were varied from time to time. The most important vegetable and saline remedies of the class all being tried in their turn, without any marked effect on the urine. Occasional hydrogogues (elaterium, resin of scammony and the acid tartrate of potash, the compound powder of jalap, etc.) were given as the anasarca and pleuritic effusion increased. A generous diet, perchloride of iron and quinine, belladonna and quinine were also employed perseveringly from time to time. Under these measures the ascites remained stationary, but the anasarca and hydrothorax increased, the line of dulness reaching the 2nd rib, and with the assistance of Dr. Campbell (who had on a previous occasion, in consultation with me, confirmed the above diagnosis), I removed from the right pleura, with the aspirator, 3 pints of transparent citron-colored serum. We thought it prudent, mainly on account of the weak heart, not to exceed that amount at the first tapping. A good deal of pain set in that night in the right back, in the neighbor-

hood of the puncture, attended with a feeling of great constriction across the chest, and for three days he did not feel at all as comfortable as he had felt before the operation.

The reduction of the pleuritic effusion was not followed by an increased secretion of urine, although his thirst was very much augmented, nor by any improvement in the color of the skin, which had been somewhat icteroid for several weeks.

Although the jugular veins were habitually distended, and visibly pulsated, and filled up from below when compressed by the finger, at no time could a tricuspid murmur be heard; nor was the slight murmur formerly heard in the "mitral area" perceptible after he came under observation with the dropsy. His pulse generally ranged about 90, although towards the close it reached 108 and 118. Its volume continued fair, and its rhythm was but occasionally disturbed by an intermission or two in a minute.

On the 26th the fluid in right pleura again was on a level with the second rib; no effusion could be detected in the left chest; the œdema in the back and lower extremities was very considerable, and a couple of acupunctures in either ankle were made, and flannel rollers applied. The fluid escaped in moderate quantities; he soon became unusually drowsy and much weaker; his mind wandered, and he died exhausted three days afterwards. No redness of the skin at the site of puncture had supervened.

Dr. Osler conducted the autopsy. Considerable œdema of thighs and legs remaining. Subicteroid color of surface.

Thorax.—Drew off about 7 or 8 pints citron-colored transparent serum from right pleural cavity through a trocar. Right pleura is deeply injected, and some shreds and flakes of *recent* lymph are found at the posterior and inferior regions, as well as two or three broad bands of recent false membrane dividing the pleural cavity into loculi. No fluid in left pleura. A very old adhesion at base of left lung. Left lung presents very slight puckering and fibroid thickening of pleura at extreme apex. The right apex is exempt. A peculiarity of right upper lobe is that it is *bifid* vertically, the smaller lobe being posterior and

external to the larger. Whole right lung except about the upper fourth is carnified. Pericardium contains 2 or 3 ozs. transparent citron-colored serum; substance is unusually thick and resisting. All heart's cavities distended wfth blood, as well as the great veins. White patch on *front of left ventricle towards the apex*. Some white coagula closely adherent to lining of right appendix auriculæ. Rest of blood in auricle and right ventricle black and coagulated. Colorless clot in pulmonary artery. Tricuspid orifice very large—5½ inches in circumference. Tricuspid and pulmonary segments normal. Coagula in some of the pulmonary veins and left auricle. Besides large soft black clots in left ventricle, a layer of yellow stratified fibrin about ¾ inch thick adheres to lining of left ventricle and covers about the inferior two-thirds of its anterior wall, whence it extends somewhat over the septum. On section, about the internal half of the wall of the ventricle at the site of the adhering fibrin, has lost its muscular appearance and color, and is anæmic, of pale grey hue, and tough, crunching under the knife. The outer half of the wall appears to be normal, and the whole thickness of the ventricle above this grey area is of healthy appearance. The columnæ carneæ at their basic attachments appear whitish, firm and fibroid on section. The entire thickness of the septum ventriculorum is on section greyish white, firm and tough—fibroid looking. Mitral valve healthy. Aortic segments thickened at their base, and a narrow calcareous ring at the insertion of one of them. Their free margins normal and the valve competent. Aorta from heart to iliacs studded with atheromatous patches.

Abdomen.—About two pints transparent serum without flocculi in peritoneum. Spleen very firm, cuts crisply and with considerable resistance. Liver has very few irregularities on its surface, is tough and firm—very congested and nutmegged. No calculi in gall bladder. Both kidneys of normal size, thick, firm, coarse on section, and congested venously; their capsules above average thickness, but peel off without tearing cortex. Intestines appear healthy.

Brain not examined.

Dr. Osler kindly examined the diseased heart muscle. Microscopical examination of the portion involving the septum near the apex showed it to be made up of ordinary connective tissue fibres, arranged in wavy bundles, with very few corpuscles or fibre cells. In the greater part of the area no trace of muscle substance is found, but at the margins the new growth is seen penetrating between the muscle fibres, some of which are completely cut off and isolated. The endocardium over the spot is thickened. Many of the muscle fibres in contiguous parts presented brown granules and fatty molecules.

There are several points of interest in this case, upon some of which I will offer a few remarks.

The opinion formed of the nature of the case when it first came under my observation was weakness of the heart—probably from fatty degeneration—an opinion based upon the following combination of clinical facts:—Weak impulse and sounds of the heart, without evidences of decided dilatation of the ventricles; occurrence for the first time eight years previously of symptoms like those of angina while walking up a slight incline, and ever after a sense of dyspnœa when walking; recurrence of similar symptoms of angina two years after the first seizure, and under like circumstances; sudden attacks of severe orthopnœa, terminating in expectoration of frothy mucus, yet not due to spasmodic asthma, nor to marked valvular or aortic disease, nor to dilatation of the heart; and a peculiarly sallow complexion and anxious and sad expression of countenance. It is true there was a faint systolic murmur in the mitral area, but the other accompaniments and sequences of valvular disease of at least eight years' standing (which there must have been to account for the features of the case) were wanting, and there was no history of an attack of acute rheumatism. The probability, then, was largely in favor of the existence of fatty degeneration of the heart, for the symptoms and signs which obtained are amongst the most reliable and constant of those met with in that affection. However, I am of opinion that the diagnosis of fatty degeneration of the heart just named cannot itself be pronounced with the scientific certi-

tude that affections of the valves and the more common alterations of the walls of the heart may be. The diagnosis must very often be one of probability rather than of certainty. The case under review proved to be one of fibroid disease, an affection which can seldom, if ever, be diagnosticated from the former alteration, and indeed is very apt to be discovered only after death. It has no constant group of symptoms or signs.

One great English authority, Walshe, speaking of cirrhosis of the walls of the ventricles, states that "it is not revealed by either symptoms or signs, unless it have proceeded to such lengths as to have induced aneurismal pouching," etc. (Disease of the Heart, 4th edition, p. 359.) Friedreich says that in extensive fibroid degeneration of the left ventricle "we have most frequently observed phenomena explicable by an insufficiency of contractile power in the heart"; and further on remarks that "it is easy to see that these phenomena do not in any manner permit it to be concluded with probability that a chronic myocarditis (*i.e.*, fibroid degeneration) exists," etc.

To quote anothor recent authority. Prof. Schrœtter observes: "It would only be possible to diagnosticate chronic myocarditis when we could with certainty exclude all other diseases of the heart which cause the same or similar symptoms, and especially fatty degeneration; this we could scarcely succeed in doing." (Ziemssen's Cycloped. Pract. Med., Vol. VI., p. 242.) It will probably be admitted that I have cited sufficient authority to prove the difficulty, if not impossibility, of recognizing fibroid degeneration of the heart during life, and two of our latest English authors, Balfour and Hayden, might be adduced on the same side.

Fibroid degeneration of the heart occurs as a localized and as a general alteration of the heart.

(*a*) The localized form obtains most frequently in the papillary muscles of the left ventricle, usually along with chronic disease of the mitral valve, and this is the only form that has frequently come under my own observation.

(*b*) It is more rarely found affecting circumscribed portions of the walls of the left ventricle, and manifests a singular and ill

understood preference for the *anterior* wall in the neighborhood of the apex and the adjoining septum, as in the case now under consideration. It may be, as some morbid anatomists suppose, that in both these varieties the process begins in the *endocardium* lining the valves in one case, and the ventricular walls in the other, or in the pericardium, whence it extends to the musculature of the heart and its connective tissue. But not unfrequently the reverse appears to be true, viz., that the disease begins in the myocardium and thence extends to the endo- or the pericardium.

(*c*) As regards the frequency with which the respective sides of the heart suffer, authorities differ—Wilks maintaining that, combined with hypertrophy, it is far more common in the right than in the left ventricle, while Friedreich, Green and others allege that it is more frequent in the left, and that when present in the right, the same change, but in more marked degree, also exists in the left heart and septum. When the degeneration co-exists with chronic pericarditis, it is said by some to thus implicate both sides of the heart.

(*d*) A still more rare form of localized fibroid transformation presents itself as an annular induration of the upper part of the *conus arteriæ pulmonalis*, which produces a real cardiac stenosis. This form, very rare in adults, is more frequently met with in early fœtal life.

II. The *generalized* fibroid transformation, in which all the walls of the heart are the seat of excessive development of fibroid tissue, was described by Sir Wm. Jenner as occurring together with hypertrophy of the musculature of the heart, when the substance of the organ is the seat of gradually developed, long continued and intermitting congestion—conditions so frequently present in chronic valvular disease. (*Med. Chir. Trans.*, vol. XVIII., 1860, p. 199 *et seq.*) This form has not met with universal acceptance, however, although I think I may say that we are familiar with it here.

The *nature* of the morbid process which determines fibroid transformation of the heart is an unsettled question. The prevailing opinion, however, is that it is a *chronic* myocarditis, origi-

nating in an endo- or a peri-carditis, which extends to the myocardium, and many, especially German and French writers, describe the affection under the title Chronic Myocarditis.

Until pathologists can agree as to what constitutes inflammation, and can point out a reliable means of distinguishing the local changes essential to chronic inflammation from other nutritional proceedings such as the reparative process, hypertrophy, the development of some tumors, etc, it will be difficult to decide whether fibroid transformation is (1st) truly an inflammatory process, or (2nd) a primary hypertrophy of the connective tissue, or (3rd) a secondary and more degenerate modification of nutrition in which, while the secreting structure of glands (liver or kidney for example) or the contractile substance of muscle, wastes and perishes, the connective tissue of the wasting organs, receiving more than its normal proportion of nutriment, experiences a hyper-nutrition. Probably all three views are correct, but they require renewed study and investigation.

Permit me to offer some observations in illustration of the three several modes of fibrosis, or fibroid disease, that I just indicated, but in the reverse order.

1st, *Fibrosis secondary to degeneration of secreting or contractile tissue.*

In some examples of cirrhosis of the *liver*, atrophy of the secreting substance is a very early alteration, and Beale has taught that such atrophy is the primary change, the increased development of fibrous tissue a secondary and very insignificant alteration; and Johnson holds very similar views respecting the origin of the contracted *kidney*.

Another example of this variety of fibroid transformation occurs in muscles the contractility of which has been impaired by some form of paralysis. As the muscular elements disappear a fibrous tissue replaces them. Dr. Beale, indeed, appears to regard the process as one of degeneration, analogous to fatty degeneration. He says:—"The contractile material of the muscles has degenerated into fibrous tissue." (*Med. T. & Gaz.*, vol. I., p. 295, 1869.) And if recent investigations justify the statement that the epithelial cells of the

liver (Hamilton) and kidney (Saundby), and even of the mamma (Creighton), may proliferate and be converted into connective tissue, that is a strong analogical argument in favor of the transformation of muscle cells into connective tissue.

May not some cases of localized fibroid degeneration of the heart depend upon some disturbance in the innervation of the organ equivalent to that productive of paralysis in voluntary muscles? Such disturbance leading to trophic changes in which the sarcous elements are replaced by, if not transformed into, fibrous tissue? Why should not powerful mental emotion or violent muscular effort now and then damage the nervous ganglia of the heart as they do the cerebral ganglia?

In support of the idea that local disturbances of innervation may bring about local hyper-growth of tissue, I would cite the opinion of that acute observer, Dr. T. Addis Emmet, that uterine fibrous tumours are common in female celibates, because they have no "outlet for the nervous force which is being constantly directed to their wombs." (Emmet's Principles and Practice of Gynæcology, 2nd Ed., p. 84.)

Other instances of local fibroid disease of the heart may originate in a local atrophy of muscular fibre induced by a patch of pseudo-membrane, the product of a bygone pericarditis; hyper-growth of the connective tissue following closely upon degeneration of the contractile sarcous elements.

Finally in this connection, as Beale's researches show that the quantity of connective tissue in muscles bears some proportion to age, so that in old age much of the muscular tissue is replaced by fibrous material, it is highly probable that the heart, in common with other organs and other muscles, often suffers in its nutrition, so that its sarcous elements waste more or less, and are replaced by connective tissue as a contingency of advanced life.

2nd. *Primary Fibrosis from Hypertrophy.*

I have suggested that so called fibroid degeneration may sometimes be a primary or at least concurrent hypertrophy of connective tissue, constituting the first great step in the morbid process.

In pseudo-muscular hypertrophic paralysis, hyperplasia of

the connective tissue of the muscle is a very early change, and if not antecedent to, is at least concurrent with, more or less hypertrophy of the muscles.

Such appears to be the correct view, especially of the fibroid transformations that occur as consequences of passive congestion resulting from valvular diseases, and are met with in the heart itself, in the kidneys, liver and other organs. And under this heading belongs the diffused "fibroid transformation" of the heart already mentioned. The morbid process appears to be neither atrophic nor inflammatory, but a real hypertrophy. And "areolar hyperplasia" of the uterus probably often results from similar congestion of that organ, a consequence, it may be, of various degrees of displacement or flexion of the organ itself, or of valvular disease. My friend Dr. Gaillard Thomas has in this connection long ago advocated the distinction that ought to be drawn between chronic inflammation and interstitial hyperplasia.

The *large fibroid kidney* (Klebs' "Cyanotic Induration") affords an excellent example of primary hypertrophy of the connective tissue of an organ. Take the same process in the heart, it occurs independently of inflammation, and as a consequence of slowly developed and long continued congestion of the kidney. But some of the ablest modern authorities, Grainger Stewart, Liebermeister, and quite recently the lamented Bartels, have referred the *chronic contracted* or *red granular kidney* to primary *hypertrophy* of the connective tissue of the organ, which, as a general rule, has *not been preceded by inflammation.*

The very latest investigations of Saundby and of Greenfield (*Lancet*, 27th Mar., '80, p. 490) go to prove that the initial step in the production of this form of fibroid kidney is the formation of connective tissue by proliferation of the epithelium covering the glomeruli (Greenfield), or of that lining the tubules and the glomeruli, and covering the vessels (Saundby). This view of the origin of granular kidney, in my opinion, favors the theory that the process is rather simply hyperplasia than inflammatory; but be that as it may, it is another example of primary overgrowth of connective tissue in an organ.

An interesting variety of this primary hyperplasia of the connective tissue of organs is afforded now and then in Rickets. Dr Dickinson has found that the enlargement of the spleen and liver, occasionally observed in that constitutional affection, is largely due to an hypertrophy of the fibroid tissue of these viscera.

The "simple" form of "grey degeneration" so frequently observed in the posterior columns of the spinal cord affords an excellent example of hypertrophy of the connective tissue of an organ independently of inflammation, although it is not yet settled whether the atrophy of the nerve fibres precedes or follows the overgrowth of the neuroglia. Rindfleisch, however, sustains the latter view.

One of the most important varieties of primary hypertrophy of connective tissue is that in late years specially studied by Sir Wm. Gull and Dr. Sutton, and called by them arterio-capillary fibrosis. They have endeavored to establish the existence after middle life of a cachexia which manifests itself in a tendency to hypertrophy of connective tissue, which first involves the arterioles of the entire vascular system, and may then develop fibroid changes in one or more of the other organs, kidney, liver, brain, cord, stomach, heart, etc.

It is true that some of the ablest pathologists do not believe in the fibroid diathesis, but all admit that an excessive growth of fibroid tissue frequently occurs in the outer tissues of the smaller arteries after middle life, as had been previously pointed out by Charcot and Bouchard under the name of Sclerous Arteritis.

I know of no sufficient reason, however, for regarding the process as inflammatory; and if Mohammed is correct in referring the fibroid thickening of the arterioles to increased arterial tension, then why not recognize in the increased growth of the connective tissue of the over-worked vessels simply a physiological process analogous to that which causes muscle to grow when exercised. Why suppose inflammation necessary or even favorable to the process? It appears to be highly probable that some cases of local fibrosis of the heart and other organs begins in this hypertrophy of the sheaths of the blood vessels,

and extends thence to the connective tissue between and around the muscular fibres, and ultimately causes their atrophy. (See Rindfleisch's Path. Hist., p. 129.)

3rd. *Fibrosis of Inflammatory origin.*

But, thirdly, the new fibroid tissue is no doubt sometimes of inflammatory origin.

Such is doubtless the nature of the tracts of fibroid tissue met with in tubercle in the lungs for example; so a peribronchitis appears now and then to extend to the interstitial pulmonary tissue and originate a fibroid induration of the parenchyma, and it is highly probable, and is generally held, that an endo- or a pericarditis may sometimes extend to the intermuscular connective tissue of the heart and induce an active proliferation and increased development thereof.

In addition to those more or less extensive productions of fibrous tissue so familiar as a sequence of *inflammation* of the serous membranes, may also be mentioned the scleroderma of adults, the spurious cheloid which occasionally succeeds burns, small-pox, etc., the masses of cicatrix-like tissue resulting from syphilitic disease of the liver, lungs, skin and other organs.

Amongst the most important examples of localized development of fibrous tissue under the inflammatory nisus is that afforded by syphilitic arteritis—a process which is not only responsible for many instances of inflammation, of thrombosis, and of softening of the great nerve centres, but, according to Mr. Welch, assistant professor of pathology at Netley (*Med Chir. Trans.*, Vol. LIX., p. 73), for 50 to 66 per cent. of the aortic aneurisms occurring in the British army. It has, in my opinion, been satisfactorily proven that syphilis is a cause of fibroid disease of the heart, as advocated by Wilks, Moxon, and others, but Hilton Fagge has shown that it is at most a very unusual cause (*Path. Trans.*, Vol. XXXV., p. 95 *et seq.*) It did not exist in my patient.

One of the most interesting examples of development of connective tissue in an organ by inflammation, and the last that I will adduce, is afforded by scarlatinal nephritis, in which it is now known that an interstitial growth of embryonic tissue takes place in the parenchyma of the kidneys, which may even fibrillate.

Reverting from these observations upon the general pathology of *fibroid* hypertrophy to the special example under consideration, I would call attention to the circumstance that while the area of fibroid degeneration in the wall of the left ventricle corresponded accurately to the region in which the endocardium was opaque and covered by a firmly-adhering layer of fibrin, the degeneration also involved the ventricular septum, although no fibrinous deposits lay upon the endocardial lining of that part. And in a case related by Dr. Bristowe (*Path. Trans.*, Vol. VI.) it is mentioned that the patches of endocardial thickening did not "usually" correspond with the patches of fibroid degeneration. These cases appear to show that if the localized variety of fibroid degeneration of the heart is sometimes a consequence of disease of the endocardium, it may also *begin* in the myocardium itself and extend or not to the endocardium, and that, consequently, even when the alteration in the endocardium corresponds in site with that in the myocardium, we are not therefore to infer that that the endocardial disease caused the myocardial.

It might be urged that the co-existence of extensive atheromatous degeneration in the course of the aorta, which is generally regarded as a consequence of chronic endo-arteritis, renders it probable that the fibroid degeneration of the heart was an *effect* of a chronic endocarditis, and such is not an improbable view. But it must not be overlooked that a general tendency to hyperplasia of the connective tissue of organs also existed, witness the firmness and toughness of the spleen, liver and kidneys, as well as of portions of the musculature of the heart, spoken of in the record of the autopsy. And it can hardly be affirmed that it has been proved that the changes in the arterial system which result in atheroma are truly imflammatory, and not simply degenerative.

The co-existence in this case of fibroid changes in many organs appears to point to some general cause, as systemic congestion from tricuspid incompetency (which did not exist) or as the "arterio-capillary" fibrosis of Gull and Sutton. If the former had been the cause of the fibroid transformation of the cardiac muscle, one would have expected it to have been dif-

fused throughout the heart and not localized. On the other hand, it would be begging the question to assume that the cardiac change was part of an arterio-capillary fibrosis, a manifestation of the so-called "fibroid diathesis," as the smaller arteries and arterioles were not examined. However, it must be very rarely, even in that diathesis, that cardiac fibrosis is accompanied, as in this instance, by fibroid induration of the liver, spleen and kidneys—for Hilton Fagge "could find hardly a single example" of such co-existence in any of his own cases of cardiac fibrosis or among those recorded in the Transactions of the Pathological Society up to 1874.

My own view is that the cardiac alterations long preceded those observed in the liver, spleen and kidneys, and that the latter originated in systemic congestion produced by the cardiac disease, especially the tricuspid incompetence.

In looking over some of the records of the past on this topic, I met with a case related by Dr. Whipham which resembles in many respects the case under discussion. (*Lancet*, vol. 1., 1873, p. 25.) In that, an old blood clot adhered to the endocardial lining of the wall of the left ventricle and septum, the endocardium was thickened and the muscular tissue in a state of fibroid degeneration. The author attributes the lesion of the endocardium and of the wall of the heart to the irritation caused by the adhering blood clot, and claims to have first pointed out such a mode of causation of alterations in the heart's substance. In my opinion, the correct explanation of Dr. Whipham's case, as well as of my own, is that a diseased condition of the endocardium preceded and induced a deposition of the fibrin of the blood upon the arterial part. Such a view appears to be more in accordance with known pathological laws than that which supposes the precipitation of fibrin upon a healthy endocardial surface.

There are some interesting features also in the clinical history of the case under review, but I have occupied more than sufficient of the Society's time, and will only mention (1) the suddenness with which the first signs of failing heart-power set in; (2) the disappearance of a systolic murmur of organic

origin, and its absence notwithstanding the presence of a thick layer of fibrin on the lining of the left ventricle; (3) the existence of marked tricuspid dilatation without mitral stenosis or incompetence and without emphysema or bronchitis; (4) the difficulty of satisfactorily explaining in this instance the occurrence of the dilatation of the tricuspid orifice; (5) the supervention of pleuritis after aspiration of the pleural cavity; and lastly (6), the singular character of the sudden attacks suffered by the patient of dyspnœa and cough, followed promptly by copious expectoration of a frothy mucus, and immediate relief.

Hospital Reports.

MEDICAL AND SURGICAL CASES OCCURRING IN THE PRACTICE OF THE MONTREAL GENERAL HOSPITAL.

MEDICAL CASES UNDER CARE OF DR. OSLER.

(*Continued from page* 114.)

CASE X.—*Anomalous Case of Pyæmia.—Suppuration about tissues in left inferior carotid triangle; Pyæmic abscess beneath tensor vaginæ femoris; Pyæmic infarcts in the lungs; Septic Pleurisy.*

REPORTED BY MR. J. B. HARVIE, OF OTTAWA.

James C., aged 33, mechanic, a large, powerfully-built man, admitted June 8th, complaining of pain in the front of the left shoulder and shortness of breath. Family history good; personal history satisfactory. Has been a ship carpenter; is temperate. Present illness began on June 5th with pain about the left shoulder and fever. Had no chill. Swelling came on above and below left clavicle, and there was pain on outer side of right hip.

Condition on admission.—As he lies in bed, chief features noticeable are: Suffusion of face, lips slightly livid; respirations 30 per minute, and are short, the inspiratory act is caught suddenly, and expiration does not follow for a second or two. *Chest* movement is slight. An extensive swelling is seen in the upper part of the left side extending from the root of the neck, obliterating the supra and infra-clavicular spaces, and extending

as far as the shoulder and anterior axillary fold, while below it reaches nearly to the nipple. To the right it is bounded by the mid-sternal line. Just over the 1st rib the swelling is most intense, and the skin here is reddened. It pits on pressure, does not fluctuate or crepitate, and is not very painful to the touch. Shoulder joint not swollen; glands in axilla not enlarged. The left arm and hand is colder than the right, and look a little livid. The veins are also more plainly visible, particularly the cephalic. On *percussion*, clear note, except over region of swelling on left side. On *auscultation*, breathing weak over left infra-clavicular and mammary regions; otherwise appears normal. *Heart's* apex cannot be felt; diminished area of dulness; sounds normal. Nothing special detected in examination of abdominal organs. *Liver* and *spleen* not enlarged. Complains of pain in the right hypochondriac region, and there is both pain and tenderness over right iliac crest. Tongue slightly coated. No vomiting. 10 *p.m.*—Pulse 136, small; respirations 60; temperature, under tongue, 101·5°; temperature on swelling in left mammary region, 99·1°: on opposite side, 97·8°. Is quite sensible. Has no cough. Pain has continued in situation above noted.

9*th.*—Passed a bad night, in spite of morphia. General condition same as yesterday. Temperature 98°; pulse, 132; respirations 46. Swelling in left lower cervical and mammary regions is unchanged. Has been perspiring profusely; no cough. On examination of chest, expansion is deficient on right side, and there is slight dulness in lower mammary region of this side. On auscultation, fine crepitant râles in this region. Behind there is no evident dulness on right side, but a loud friction sound is heard in infra-scapular region. Tactile fremitus not increased. Evening temperature, 100·2°.

10*th.*—Patient passed a bad night; respiration very hurried, and constant pain in neck and outer part of right ilium. Is much weaker, and face is becoming cyanotic. Pulse almost imperceptible—135 in the minute; respirations 55, and becoming gasping in character; temperature 100°. Râles abundant on right lower mammary region. Was not examined behind.