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Original Articles.

TUBERCULOSIS OF THE MYOCARDIUM.*

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PHILADELPHIA.

Previous to 1826 the existence of cardiac tuberculosis was denied. Tuberculosis of muscle was known to be rare and the heart, with its unceasing contractions, was considered immune. Again, the theory of the natural antagonism between cardiac disease and tuberculosis was almost universally accepted.

Laennec,[1] in 1826, in a "Treatise on Auscultation," affirmed the existence of cardiac tuberculosis, assigning to the heart the thirteenth rank among organs affected. He gave no instances. To Townsend[2] of Dublin belongs the honor of reporting, in 1832, the first case of myocardial tuberculosis. Others followed at infrequent intervals until in 1865 Haberling[3] was able to collect 12 cases, some, however, being of the pericardium only. Sänger,[4] in 1878, gathered and classified 22 cases, including 3 of his own and omitting several doubtful ones. This was thorough work and paved the way for all subsequent study of the subject.

In 1892 he was followed by Pollak,[5] and in 1895 Valentin[6] published his monograph—a critical summary of 36 reported cases. More or less extended articles by Mendez,[7] Labbi,[8] Barie,[9] Sangalli,[10] Kaufmann,[11] and others followed, to be summed up in an elaborate and valuable thesis by Fuchs[12] in 1898. This author collected 53 cases, including 2 of his own. The subsequent literature embraces articles by Eisenmeyer, Moser,[13] Crawford and other writers.

THE AUTHOR'S CASE.

Moser's personal observation with 5 cases he gathered from the literature, which are not included in Fuchs' list—3 being reported after his article was written—bring the total to 59. In a careful search of all available sources I have been able to add 12 cases exclusive of one of my own, making a total of 72. (See table.) My own case may be briefly reported:

A. T., aged 22 years, female, occupation clerk. The family history was good except for a slight tuberculous taint. The personal history includes the usual childhood diseases and a light attack of pleurisy at 17 years of age. One week prior to my first visit, March 20, 1888, the patient developed marked recurring chilly sensations lasting an entire day. These were accompanied by stitch-like pains in the left mammary and left axillary regions, by jerky respiratory movements and dyspnea. The temperature rose to 102 F. subsequently and then continued to range from this grade to 100.2. The physical signs were at first merely defective expansion and a friction-rub indicating plastic pleurisy, followed later by those of the serofibrinous variety. The apex beat of the heart was displaced and lost behind the sternum. The pleural sac was about two-thirds filled with exudate, which was freely movable on changing the patient's posture. There was nothing unusual presented by the case and the dyspnea was only moderately well marked, until the seventh day, when of a sudden the heart's action became distinctly weak and rapid, but there was no arrhythmia. At this time dyspnea was also more pronounced, and slight lividity of the mucous surfaces and finger tips was noticeable. These symptoms, however, passed away in the course of a few hours as the result of free stimulation. It was proposed to aspirate on the following day, but to my great amazement a member of the family called on me the next morning informing me that the patient had just died without the slightest warning and with startling suddenness. From all that I could gather, death probably occurred in syncope.

The autopsy was made twelve hours after death. Body well nourished, medium height. A flat percussion note over the left side from fourth rib to base of chest. On opening thorax nearly two liters of serofibrinous fluid, with only a moderate amount of fibrin, was found in left pleural sac; on the right side pleura adherent near to the base, otherwise apparently normal. Neither cultural nor inoculation tests were made of the exudate. No macroscopic changes were presented by the left pleural membranes and no bands of adhesion between the layers. The lower portion of the upper and the entire lower lobe were dark and atelectatic; near the lower border of the upper lobe and close to the pulmonary pleura there was a small caseous nodule, probably the primary focus. A similar small tuberculous area was situated in the upper lobe of the right lung, otherwise the lungs showed nothing abnormal.

The heart occupied a position within though near to the mid-sternal line; it had not been rotated on its own axis. The heart weighed 240 grams, hence was not hypertrophied. There was fibrous pericarditis present at the base of the heart and immediately beneath this area a single large caseous tubercle in the wall of the right auricle projecting slightly into its cavity and involving the entire thickness of it. The endocardium showed nothing pathologic on macroscopic examination. On account of the presence of the circumscribed pericarditis it was evident that the myocardial involvement was secondary and the result of extension by contiguity. The bronchial glands also were free from tubercular change. Briefly, the histologic peculiarities were those characterizing the tuberculous processes elsewhere: embryonal cells and giant cells with the presence on the interior of the latter of a few tubercle bacilli and caseous areas. An indefinite fibrous capsule envelopes the growth. Muscle fibers in the vicinity of the growth showed atrophy. The affected pericardium and the left auricle and ventricle were not examined microscopically. This is regrettable in the light of the recent microscopic studies of plastic pericarditis (vide infra).

Most of the important contributions to the subject of

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tuberculosis of the myocardium come from France and Germany, and much of the information is contained in monographs and dissertations, difficult, and in some places impossible to obtain. Hence, I believe that a brief, fairly comprehensive review of this interesting subject will be of some help and value.

PRIMARY AND SECONDARY FORMS.

Theoretically, tuberculosis of the myocardium may be primary or secondary. Practically, the existence of the primary form has yet to be satisfactorily demonstrated. Knopf and Demme[14] have claimed that their cases were of primary origin in the heart muscle, but their contention has not been generally accepted. In the former's case the patient died of acute miliary tuberculosis and there was nothing in the autopsy to show that the heart lesion was the primary one.

Demme's case showed at autopsy three small tubercles in the wall of the left ventricle and one in that of the right ventricle. No lesions were noted in other organs. Commenting on this case, Labbe observes that "the lack of histological examination, the insufficient search for tuberculosis in other organs and the well-known difficulty in discovering the mode of entrance of the bacillus of Koch, cast a doubt on the primary cardiac localization." All subsequent writers on the subject unite in declaring that the existence of primary tuberculosis of the myocardium remains to be proven.

A lesion of the heart wall, tubercular in nature, must therefore be regarded as practically always secondary to a focus in some other tissue. Later on this relationship will be considered in greater detail. A lesion with a total literature of 72 cases is far from a common one—far less so than pericardial tuberculosis. According to Valentin's table in 3,203 autopsied cases by different authors, myocardial tuberculosis was found in seven, a percentage of 0.21, or 2.1 per 1,000. Fuchs states that Kolb, searching Rokitansky's autopsy records for twenty years, 1840-1860, found no cases. Sangalli in 796 tubercular subjects during about 35 years reports two cases. In 285 similar autopsies by Letulle, Fuchs* found only one instance (reported by Pinn).

In marked contrast is the statement of Weigert[15] to the effect that in his experience the majority of victims of acute general miliary tuberculosis showed small discrete tubercles in different portions of the heart. Kaufmann, in reporting his case, states that he has often seen instances of the lesion, but until the present case had not considered the nodule of sufficient size to report. The actual fact lies between these extremes of view, probably nearer the opinion of Valentin. It is a noticeable fact that many more cases have been reported in late years than formerly; thus, from 1832 to 1890, nearly sixty years, 31 instances are recorded, while in the eleven years since the latter date, 41 cases are reported.

Formerly many nodules in the heart muscle were considered gummata, while in reality tubercular. For example, Virchow believed that most, if not all, of the so-called tubercles of the heart were of syphilitic nature. With more modern methods this confusion seldom occurs, and, of course, the presence on section of the tubercle bacilli renders the diagnosis absolute.

AGE, SEX AND PREVIOUS DISEASE.

This condition is relatively more frequent in youth. Fuchs, adding his statistics to those of Valentin, finds that out of 41 cases in which data are given nearly one-half are under 13 years.

Ages 0 to 15, 15 cases; 15 to 45, 16 cases; 45 to 60, 3 cases; 60 to 65, 2 cases; 71, 1 case; 72, 1 case.

If we consider in addition the 18 cases now recorded and not included in Fuchs' table, in 14 of which the age is given, we find:

Ages 0 to 15, 21 cases; 15 to 45, 24 cases; 45 to 60, 5 cases; 60 to 65, 3 cases; 71, 1 case; 72, 1 case.

These figures somewhat advance the average age, but still leave almost 40 per cent. of those affected under 15 years, while a little over 40 per cent. are between 15 and 45.

The relation between the question of age and that of causation will appear later, and it is not unimportant. In regard to sex there appears little difference. The question of trauma relative to the origin of this disease has received little attention and appears to need only scant mention. Valentin believed it might have some influence. Stoïcesco and Babès,[16] reporting their own case of a policeman who had probably been struck on the chest more or less often by reason of his occupation, lay considerable stress on the subject. Trauma may possibly play a rôle in the development of pericardial tuberculosis It may thus indirectly affect the heart itself, as myocardial tuberculosis may result from extension of similar involvement of the pericardium. A more direct influence may well be doubted, and, as Fuchs remarks, can not be affirmed on the basis of a single case.

The influence of pre-existing cardiac disease on the development of tuberculosis in the heart muscle has been little studied in the past. Sänger believed cardiac sclerosis to be a predisposing condition, but probably mistook an effect for a cause. Fibrous disease of the heart and arteries is usually a condition of later life than the period in which we have seen myocardial tuberculosis to be most common. The increase of connective tissue found more or less associated with this lesion is probably to a greater extent due to the effect of a toxin from the tubercle bacilli. Support is given this view by the analogous process seen in the heart as a result of the infectious fevers, intoxications, etc. It must not be forgotten also that fibrous tissue formation may indicate a healing or healed tubercular process in the heart as elsewhere.

THE SITES OF THE LESIONS.

While any part of the heart wall may be affected, certain sites are more common than others, varying with the character of the lesion and the mode of transmission. Lancereaux[17] and Valentin believed that the ventricles were more commonly involved and the latter found no instance recorded of involvement of auricles alone. In this he is supported by Labbe. Nathan-Larrier[18] and Fontoynont[19] both reported cases showing no ventricular involvement, but more extended study revealed their mistake.

In recent literature several observers report no growths in the ventricles with a nodule in the auricle. As in these cases no careful microscopic study is given of the ventricular wall, we must still assume the approximate correctness of Valentin's position. Certain it is, however, that oftener by far the largest part of the mass is found in the auricle, the growth in the ventricle being insignificant in comparison, e. g., Labbe, Fontoynont, Kaufmann, Eisenmeyer, my own case (Case 1), and many others.

While Valentin did not believe one side of the heart

* Following Fuchs, the case of Barie and two of Sangalli are included in the first series, as, though reported later, they were observed in 1850, 1873 and 1878, respectively.

to be affected more often than the other, Labbe took a different view. In this he is supported by the observations of Nathan-Larrier, Kaufmann, Fontoynont, as well as by the records of later cases, which I have investigated. We may safely say that the right auricle is affected more often than the left, while in regard to the ventricles the difference is less marked and is somewhat in favor of that of the left side. The influence exerted by the type of the lesion on the side affected is well shown in the summary by Heineman.

1. Acute general miliary tubercles:
   (*a*). Conus arteriosus of right side.
   (*b*). Great papillary.
   (*c*). Less often endocardium of ventricles.
2. Small nodular form, not general.
   Ventricles.
3. Large tubercles:
   (*a*). Auricles.
   (*b*). Junction of auricles and ventricles.
   (*c*). Ventricles often near septum.

Generally speaking, the small nodules are found usually in the ventricles and the large tubercles in the auricles, chiefly the right. The shape is only less variable than the size of the lesion, and both depend on the type and age of the growths. They range from the size of a pinhead in the miliary variety to that of a pigeon's egg (Fontoynont) or a hen's egg (Pollak) as seen in the large tubercles. Nathan-Larrier gives the dimensions of a caseous mass in his case as 8 cm. by 6 cm. by 5 cm.

The number of nodules is equally variable. A single tubercle may be present, even of very small size, or there may be many, large, small, or both. Usually there are several, two to five being perhaps a fair average. The greatest number recorded is 18 in a case of Fontoynont.

THE THREE VARIETIES.

The varieties of tuberculosis of the myocardium are three—large tubercles (or large and small nodular form), miliary form and the diffuse variety. I shall consider them in the order of their relative frequency.

1. *Large Tubercles.*—These may be single or multiple, pea to egg size. They may project beneath the endocardium or the pericardium or may be completely buried in the cardiac muscle, only discovered on section. In by far the largest number of reported cases they consist of firm, whitish masses, often with a softened, caseous center. Among the more recent ones are those of Pueschman, v. Genersich, Hoisholt,[21] Thiry, Eisenmeyer, Dunham, Wells and Hlava, not to mention many others in the literature.

2. *Miliary Variety.*—Here we find scattered small grayish, semi-transparent masses, often projecting as minute elevations under the endocardium. The latter is seldom affected. This variety is most common in the ventricles and shows the usual structure of the miliary tubercle elsewhere. It may be easily overlooked when situated in the depth of the heart wall. While less frequently seen than the large nodular form, it is far more common than the third variety.

3. *Diffuse Form or Tubercular Infiltration.*—There are a few instances recorded. It consists in a more or less general, diffuse tubercular infiltration of the cardiac muscle, generally that of a ventricle. Bret,[22] in 1893, reported the first case, followed in 1896 by the carefully studied cases of Labbe and Sangalli. The process is usually seen in connection with tuberculosis of the pericardium. The heart is enlarged, its walls thickened, firm and resistant to the knife. Microscopic examination shows near the pericardium separation of the muscle fibers by connective tissue of various degrees of firmness or by structureless, cheesy, poorly-staining material. In the middle of the myocardium is a layer of tubercular material in different stages, showing embryonal and giant cells and caseous masses. Still deeper, tubercular nodules are mixed in with areas of nearly normal muscle. In Labbe's case there was more tubercular than sound tissue in the part examined. Bacilli are present, many in Labbe's, few in Sangalli's case.

In this variety the cardiac muscle is more extensively altered than in the preceding ones. We find fragmentation, pigmentation and vacuolation of the fibers with atrophy and separation. In other places there is formation of intermuscular connective tissue and actual destruction of the original elements. In the nodular variety there is little alteration save atrophy of the muscle fiber, more or less limited to the immediate neighborhood of the tubercle, in Labbe's opinion, due to pressure. The actual loss of muscle substance here depends on the number of nodules.

A FOURTH VARIETY QUESTIONABLE.

A fourth variety mentioned by Brehmer[23] is admitted by some observers, but not generally accepted. It consists in a diffuse sclerosis of the heart muscle, a true tuberculous myocarditis, with excessive formation of fibrous tissue. Giant cells were found, and it is possible that the tubercular toxin can cause such a lesion without the immediate presence of the bacillus in the tissue. Still, a claim based on a single case in which the tubercle bacillus could not be found in the lesion can not be considered proven, and we should, for the present adhere to the usual threefold classification.

RARITY OF ULCERATION.

An interesting and important fact in connection with our subject is the rarity of ulceration. In fact, its occurrence was long denied by most competent students who rejected Murchison's instance reported in 1865. That ulceration does take place at times has, however, been more recently well established. Nathan-Larrier found erosion of the endocardium in the right auricle in his case, and believed it an example of secondary disease of the heart, with a subsequent infection of other organs by ulceration of the cardiac focus into the general circulation. He is supported by Letulle and Fuchs, both of whom examined his specimens. Mendez brings forward apparently good proof of the condition in his case.

Pueschman's specimen showed a caseous tubercle extending through the right auricular wall and capped with a thrombus containing enormous numbers of tubercle bacilli. The patient of v. Genersich had an erosion of the endocardium over a tubercle in the right auricle. There was a recent extensive miliary tuberculosis of the lungs, liver, spleen, etc. Borsch[24] reports a similar instance with the presence of Weil's symptom-complex as a result of an infection of the liver.

It is difficult to say why these tubercles often attain such a size, become so softened and yet so rarely discharge into the blood stream. If enough time is given for such growth, why not enough to reach the stage of ulceration? The explanation probably lies in another direction. From their situation in the substance of the heart muscle they are little exposed to secondary infection and resulting breaking down, such as occurs so often in organs more readily attacked by micro-organisms, for instance, the lungs.

The minute anatomy of the tubercular growth in the heart muscles reveals nothing unusual. The microscope

shows embryonal adjacent cells with areas of caseous material and more or less fibrous tissue. The tubercle bacillus is found practically always when sought with appropriate methods. It is present in small or large numbers, most often within the great cells.

DIAGNOSIS.

Differential pathologic diagnosis is not difficult with modern methods. Tumors show their individual peculiarities of structure and lack the presence of the bacillus. Actinomycosis, while presenting much resemblance to the tubercle in gross appearance, is rarely met with in this situation, and on section would show its characteristic structure. The presence of the ray fungus and the absence of the tubercle bacillus would establish the diagnosis.

Gummata simulate the large tubercle closely and, as mentioned, were formerly supposed to be the real lesion in the so-called tubercles of the myocardium. Virchow[25] held this view and laid stress on the presence of an envelope of fibrous tissue around the syphilitic growth. Fuchs has shown that such a fibrous capsule may rarely surround a tubercle. Under such circumstances, in the absence of tubercle bacilli, a diagnosis might be impossible without resort to animal inoculation.

COMPLICATIONS.

Quite a variety of morbid conditions have been observed as a result of the presence of tubercles in the heart muscle. Besides changes in the muscle fibers already described, hypertrophy and dilatation are frequent. Tuberculosis of the endocardium occurs, though rarely. In Townsend's case a large mass compressed the entrance of the pulmonary veins. Kaufmann notes narrowing of the orifice of the canal and pulmonary veins. Classen[26] reports narrowing of the superior vena cava to a mere slit. Frequently the cavity of the right auricle is almost obliterated by the size of the masses in its walls. Thrombi are seen occasionally on the heart wall, especially in the large tubercular form. Instances of this are seen in the reports of Pueschman, Moser and Wells.

The relation of myocardial tuberculosis to tuberculosis of other organs is an extensive and interesting one. As already stated, the lesion in the heart is always secondary to a focus in some other organ, at times found only after most thorough search. On the other hand, the growth in the cardiac muscle may in turn transmit infection to other portions of the body by rupture into the general circulation. Instances of this have already been cited.

Occupying one or the other of these relationships, practically all the organs of the body have been found affected. Lungs, pleura, mediastinal and mesenteric glands, liver, spleen, kidneys, intestines, joints, vertebræ and genito-urinary tract, all of these have been reported.

SOURCE OF INFECTION.

Accepting the fact that the heart is never primarily affected, how does the tubercle bacillus reach the cardiac muscle? Where is most often the primary localization and what the method of transmission to the heart? These questions have been a fertile field for discussion, and while there is now something of a crystallization of opinion on the subject, much room for investigation remains.

The current belief is expressed by Labbe when he says that the source of infection is found most often in the thorax, and that transmission to the heart generally occurs by way of the lymphatic system. In a large proportion of cases tubercular disease of the lungs and bronchial glands is present. We have seen how relatively frequent cardiac tuberculosis is in early life, and we know how general enlarged and tubercular bronchial glands are among children. Hence the theory that the most frequent source of infection is in the tubercular lymph glands of the bronchial and mediastinal groups. In a careful review of the 71 reported cases, I have found mention of enlarged and calcified or caseous bronchial glands in 29, or 40.8 per cent. In 40, or 56.3 per cent., this condition was not mentioned, though in one the cervical glands were affected and in 6 others general tuberculosis was present. In 2 cases only the glands were noted as free from disease, though in one of these also the cervical glands were involved. Considering only the cases in which the glandular condition is noted, 29 out of 31, or 93.5 per cent., are the seat of disease. These figures give much support to Labbe's theory.

From the bronchial glands the process extends to the mediastinal groups and thence to the heart, directly or quite commonly by way of the pericardium. According to Morris[27] the lymphatic vessels of the heart are in three sets, received by as many sets of glands. These glands, the anterior, superior and posterior mediastinal receive tributaries from the front, right side and left side of the heart respectively, in addition to the lymphatics of the pericardium. The anterior group sends its different vessels to the superior group and this in turn to the posterior. The latter communicates with the bronchial glands.

Gerrish[28] follows the same classification and states that the superior mediastinal nodes communicate with the bronchial and deep cervical groups and the posterior mediastinal freely with the bronchial. Thus there is an anatomic ground for the lymphatic theory of transmission. There is needed besides a reversal of the normal direction of the lymph stream. That this is possible has been shown by v. Recklinghausen.[29]

Infection through the agency of the pericardium is very frequent, the avenue of transmission being the same as for the heart directly. In the 72 cases I find adherent pericardium, total or partial, noted in 31 (including my own case), or 43.7 per cent.; also in 31, or 43.7 per cent., no mention, and no pericarditis in 10, or 14 per cent. Of the 31 cases of adherent pericardium, the process was evidently tubercular in 24, as shown by the presence in the adhesions of tubercles and caseous material. In view of the recent studies of Wells[30] and Reisman[31] showing that microscopic examination of what is apparently a mere fibrous pericarditis may reveal a tuberculosis, we may safely assume an even larger proportion to be cases of this nature. From the pericardium the process extends along the lymphatics directly to the heart muscle, most often that of the right auricle. In the 32 cases of this type the right auricle was the chief seat in 21. This accords well with greater age and intensity of the pericardial process at the base of the heart and the immediate relation of the right auricle in this situation.

In tuberculosis of the myocardium, as Tiessier and other writers have shown, associated lesions of the endocardium are rare. My own studies, however, have led me to conclude tentatively that "almost all of the rare instances of true tuberculosis of the endocardium are associated with other forms of cardiac tuberculosis, especially pericarditic."[32]

Infection by the blood has been urged by many ob-

servers as the mode of transmission in many if not all cases. I think we have seen good reasons for not accepting such an extreme view. There is, however, little doubt that such blood transmission of bacilli does occur in quite a proportion of cases. In cases in which an acute general tuberculosis exists and the heart lesion is of the miliary variety, this method is most probable. But where we find enormous caseous masses in the heart wall, can this theory hold? If the bacilli were circulating in the blood, derived from, let us say, a pulmonary focus, there would certainly be produced an acute generalized tuberculosis and the heart lesion would never have sufficient time to attain the stage of the large caseous tubercle. The situation of a nodule deep in the substance of the cardiac wall argues no more in favor of the blood than the lymphatic theory, although the contrary has been held. Direct extension from surrounding parts is seen at times, but is a less important and frequent mode than has been generally held.

It seems, then, probable that the mode of infection is most frequently from the bronchial glands by way of the lymphatic system, either directly to the heart or very often with the intervention of the pericardium. A certain but smaller proportion of cases are examples of infection by the blood, while a few are instances of extension by continuity.

TABLE OF THIRTEEN RECENT CASES COLLECTED BY THE AUTHOR.

Reporter.	Type of Lesion.	Probable Primary Focus.	Symptoms.	Remarks.
Pueschman[33]	Large tubercle.	Lungs and bronchial glands.		Adherent pericardium, secondary dissemination by rupture into right auricle
V. Genersich[34]	" "	Meninges or bronchial glands.		Adherent pericardium, tubercles, erosion of endocardium of right auricle and secondary dissemination.
Thiry[35]	Small tubercle.	Lungs and bronchial glands.		
Eisenmeyer[36]	*a* Large tubercle.		Heart dulness enlarged to right edge of sternum; 1st sound dull at all valves; 2d, pulmonic reduplicated; feeble mitral systolic and barely perceptible mitral diastolic murmurs.	Tubercular pericarditis; sudden collapse and death.
Eisenmeyer[37]	*b* " "		Heart sounds clear; attacks of collapse; quite well in interval; dyspnea and edema at end.	Tubercular pericarditis.
V. Bamberger[38]				" "
Dunham[39]	Small tubercle.	?		
Wells[40]	*a* Large tubercle.	Bronchial glands.		" "
Wells[41]	*b* " "	" "		Adherent pericardium.
Crawford[42]	Not classified.	?		
Hlava[43]	Large tubercle.			
Hartog[44]	" "			
Anders	" "	Pericardium (lungs)?	Pleurisy with effusion; heart sounds clear; one attack of collapse; next day sudden death.	Pericardium adherent at base; tuberculous areas in lungs.

THE SYMPTOMS.

Little is known of the clinical side of our subject. Among symptoms observed have been palpitation, feeble heart sounds, pericardial distress, diffuse pulsation, tumultuous and rapid heart action, fetal and gallop rhythm, rarely murmurs, sudden and recurring syncope, dyspnea, cyanosis, unconsciousness, general edema, sudden death. In all this list there is nothing specific, no single symptom or combination of symptoms that is not seen in functional and organic disease of the heart other than tubercular. Often the patient dies without a symptom that would attract special attention to the heart. In other cases lesions of the kidneys, liver, lungs, etc., easily suffice to explain the circulatory symptoms present and a heart lesion is never suspected. Fontoynont's patient, with a left-sided pleural effusion, died in an attack of asphyxia supposedly due to the pleuritic condition. Autopsy revealed extensive tubercles in the myocardium.

The history of my case, reported above, is identical. The tuberculous lesion of the myocardium, however, whilst marked, was apparently confined to the right auricle. It is well known that sudden death may occur in sero-fibrous pleurisy, especially in cases in which the heart is greatly dislocated. It has been ascribed to a variety of causes, such as embolism of the pulmonary artery, asphyxia, edema of the opposite lung and myocardial degeneration. The existence of tuberculosis of the myocardium, however, must be reckoned with as a factor in the causation of speedy death in pleural effusion. In Brosch's remarkable case, Weil's symptom-complex was present, due apparently, as shown, at autopsy, to miliary tuberculosis of the liver secondary to the myocardial condition. Other instances might be cited.

The diagnosis has never been suggested, still less made, during life. With autopsy findings before us it is easy to explain the symptoms usually noted. For part, the actual size, extent and pressure effects of the nodular growths are responsible; for the remainder we find a cause in the myocardial degeneration seen to a slight degree in the nodular type and to a greater extent in the diffuse infiltration.

Eisenmeyer believes that the diagnosis may occasionally be made by the presence in a victim of general tuberculosis of attacks of sudden, severe collapse, quickly passing and the detection of weak endocardial murmurs varying in phase and intensity. The short duration of the syncopal attacks and the comparative well-being in the interval, he explains on the ground that the heart muscle is not diffusely altered as in other myocardial lesions and the sound muscle present is able to quickly recover itself. It will be recollected that in my case symptoms of collapse lasting only a few hours occurred on the day preceding sudden death. That this is true except in the diffuse tubercular infiltration we have seen from the investigations of Mendez, Labbe and Sangalli. Along these lines, therefore, seems to lie our greatest hope for success in the future.

I would particularly emphasize the investigation, in a subject of general tuberculosis, of symptoms in any way referable to the heart, no matter if easily explained by coexisting disease of other organs. If the patient has a tubercular pericarditis, another extremely strong indication is present. Unfortunately, even if detected, the condition must ultimately be fatal. Treatment there is none so far as the cardiac tuberculosis *per se* is concerned. If, however, associated tuberculous processes are in evidence, the usual measures, both hygienic and medicinal, must be brought into prompt requisition.

CONCLUSIONS SUGGESTED.

1. Tuberculosis of the myocardium is more common than has been supposed.

2. At present writing not more than three pathologic varieties are justifiable.

3. It is practically always secondary to a lesion in some other situation, most commonly in the bronchial or mediastinal glands.

4. Transmission to the heart occurs most frequently by the lymph stream, less often by the blood current, and more rarely still as the result of extension by continuity.

5. Myocardial tuberculosis in a considerable proportion of cases is secondary to pericardial tuberculosis and the latter to disease of the bronchial glands.

6. The symptomatology is extremely variable and indefinite.

7. Diagnosis is excessively difficult, but is probably possible with great care and under favorable circumstances. In addition to the suspicious features that may be present the existence of generalized tuberculosis and pericardial tuberculosis, one or both, are essential to a diagnosis.

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Applicant's Right to Examine Papers.—The city solicitor of Washington, D. C., has, in an opinion given to the Board of Medical Supervisors, decided that an unsuccessful applicant for license to practice medicine has the right to inspect his papers afterward. The decision was the result of the refusal of the board to permit such inspection by a rejected candidate. —*New Orleans Med. and Surg. Jour.*

A CASE OF SCURVY, WITH UNUSUAL POVERTY OF THE BLOOD.*

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NARRATION OF CASE.

History.—W. G., white, single, age 32, bricklayer, was in the south of Cuba from August to December, helping in the erection of a mill. His condition when he went was good, as he had spent a vacation at the shore. He was a moderate user of alcoholic beverages, and never had any venereal disease. He was housed well enough, but the water was poor and the food distasteful in almost every respect. The beef that was killed in the morning was eaten for dinner and sometimes for breakfast. Vegetables were scarce, fruit apparently scarcer; and, as everything was cooked by a native cook in Spanish style, the patient could bring himself to eat hardly anything. There were forty men in the party and all but one were sick, usually with malaria or typhoid, although authoritative diagnoses are lacking, as the nearest physician was sixty miles away. The patient in question began to fail gradually, but at no time did he have fever, or was he sick enough to suspect either typhoid or malaria. He gradually lost strength, until finally, in desperation, he traveled the sixty miles to see the Spanish doctor, who told him that he had grave anemia and that his only hope lay in returning at once to the United States, which he did. He reached home very much exhausted, and slept the greater part of five days and nights, awakening only for water and a very little food. His relief was so great at getting home, and his fear of being pronounced tuberculous so genuine, that he steadily refused to see a physician until a few days before his death.

Examination.—On seeing the patient, the most noticeable thing was his breathlessness. It was bad when he lay quiet, but the slightest exertion increased it to a marked degree. The skin was dry and pale where it had been covered with clothing; elsewhere the decided sunburn made any detection of tints impossible. The scleræ were distinctly pearly, the emaciation was moderate, and there was no edema at all. The physical weakness was extreme, the mental apathy less so. There were a few petechiæ on the legs and thighs, but they developed less than forty-eight hours before death. There were no nodes or large subcutaneous ecchymoses anywhere. He said that he had never had a pain nor an ache during his sickness. The pulse was rapid—120 to 130 and above—and at times irregular. The temperature varied from 98 to 100, but it was never found above the latter. Anorexia was complete. The tongue was dirty, and the breath not very fetid. The teeth were good, and the gums not spongy, but of normal hardness and dark in hue. The saliva was not increased. There was an intermittent oozing of dark blood, finally forming a clot at the junction of teeth and gingival mucous membrane. Rarely would more than two teeth ooze at the same time, and the amount of blood was not large. This had probably been occurring intermittently for two weeks. There was a very slight epistaxis once or twice, but no hemoptysis, hematemesis, hematuria or melena after he came home, nor was there any previous history of these conditions. The bowels were obstinately constipated, and there was vomiting for a day, but this disappeared after the large amount of water he craved was reduced. There was a feeble expansile impulse over the precordia, but there was no friction nor thrill. On auscultation there were soft, low-pitched murmurs, heard best near the base, but also heard all over the precordia, and they showed no transmission. The lungs were normal, except for a few moist râles here and there, and the liver and spleen appeared normal. The urine was acid, had a specific gravity of 1016, contained no albumin and no sugar, and microscopically showed only a few uric-acid crystals.

Blood Examination.—This was made by my colleague, Dr. James R. Crawford, the instruments used being the Fleischl hemoglobinometer and the Thoma-Zeiss hemacytometer. The

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