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A CLINICAL LECTURE

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DISEASE OF THE HEART
AND LUNGS,

WITH SPECIAL REFERENCE TO

PHYSICAL DIAGNOSIS.

By STEPHEN S. BURT, M.D.,

PROFESSOR OF PHYSICAL DIAGNOSIS IN THE NEW YORK POST-GRADUATE
MEDICAL SCHOOL AND HOSPITAL.



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A CLINICAL LECTURE ON
DISEASES OF THE HEART AND LUNGS,

With Special Reference to Physical Diagnosis.

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While directing our attention more especially to the physical diagnosis of disease of the heart and lungs, we are constantly reminded of the intimate relations that exist between them and the remaining organs of the body. A study of these relations forces upon us the conviction that only by thorough knowledge of disease in general may we hope to arrive at anything like precision in the diagnosis of special subjects.

He is indeed a poor practitioner who allows himself to be so carried away by some absorbing specialty as to lose his interest in the general health of the body. But what shall we say, on the other hand, of the physician who believes physical exploration unnecessary, and goes his way, having prescribed for symptoms only?

To the general practitioner an understanding of the methods of physical diagnosis has now become a necessity, and one that is well recognized. It is expected by his patient and demanded by the profession, so universally, indeed, that some are forced to go through a sort of mummery for its moral effect upon the patient, when in reality there is little if any true perception of what the examination reveals. Fortunately, however, this class is in the minority. There is a desire prevalent in the profession, as shown by the increasing numbers at our clinical schools, to grasp the subject of physical

diagnosis, and ere long no doctor will think of prescribing for a cough until he has discovered the cause thereof. No practitioner will mistake a functional for an organic disease of the heart; nor will he make life miserable to a patient just because an organic murmur *has* been found. He will be capable of explaining that life may continue in comparative comfort so long as compensatory hypertrophy lasts. Furthermore, he will be able not only to recognize failing compensation, but also to ward off its fatal effects, maybe for years, by judicious treatment, and finally he will be less likely to interfere when nature does not call for aid—an achievement perhaps one of the greatest in medicine.

Now, let us turn to the study of diseases of the chest as they are presented, not in books, but at the clinic. It is one thing to learn the symptoms and physical signs of disease, and quite another to recognize them in the patient. Here lies the difference in medical men. Book knowledge is important, but some never get beyond it.

The first patient that it is our privilege to examine gives a good family history. She is thirty years of age, has not had rheumatism, nor any severe illness. She complains chiefly of loss of appetite, headache, and flatulence. Upon exertion and on exposure to cold she has a slight cough. You observe that it is not until the leading question has been asked that she tells of any shortness of breath on exertion. Her symptoms are mainly those of dyspepsia. But these disorders of the digestive tract that receive the name of dyspepsia are not always so innocent as they might seem. Not infrequently a grave kidney lesion may be found lurking in the background, and then again the heart may be at fault. A careful examination has excluded the probability of a

kidney complication in this case. We will now investigate the heart. Upon inspection, it is seen that the apical impulse is somewhat to the left and below its normal position. The impulse, on palpation, is found to be slightly increased in force. Percussion does not give a marked increase in the cardiac area. Auscultation, however, reveals a soft, blowing systolic murmur, extending to the left, with point of maximum intensity in the mitral area. Mitral insufficiency is the lesion, and for it the heart muscles fairly compensate. There is neither œdema nor cyanosis.

This is a very instructive case, illustrating as it does one of the secondary symptoms of heart disease—namely, flatulent dyspepsia. Long before serious engorgement becomes apparent there is this slight alteration in the circulation which results in defective digestion. And, too, a little unusual exertion or some slight exposure of the neck and shoulders produces a dry cough. Besides, if you closely watch these patients you will see that there is dyspnoea, not upon ordinary exertion, so long as it is confined to a plain surface, but during an ascent of a very slight elevation, or on any unusual exercise. As a result also of this, the bright-red color of the lips is seen to change to a much darker hue. I have had just such an instance under observation for the past four years. The patient was treated for simple dyspepsia, with very indifferent results, her heart lesion being unrecognized. But when the real cause of her trouble became known she made rapid improvement. A pill containing a grain each of digitalis, iron, and quinine, in addition to the stomach mixture, with, now and then, medicine for the intestines, comprised most of the treatment. The slight cough, which is provoked by a similar condition in the pulmonary circulation, is prevented

by taking only moderate exercise and by keeping the superficial circulation active with sufficient clothing.

There is still another very interesting example of venous congestion resulting from heart disease that occurs to me. A patient who was completely prostrated each month by menorrhagia came for treatment, but her errand was as fruitless as it had been elsewhere until I discovered that she had a stenosis of the mitral orifice. No lesion could be found as a local cause. Concluding, therefore, that the excessive hæmorrhage was due to venous stasis, I put her upon large doses of the infusion of digitalis during that period, and the treatment proved most efficacious.

On the other hand, it is a matter of frequent remark, the number of patients that come firmly convinced they are suffering from an organic disease of the heart, because of the pain and palpitation that often attends dyspepsia. A physical examination enables the physician to dispel all these fears, while properly directed remedies will remove the cause, quiet the heart, and quell the pain.

Here we have exemplified the interdependence of viscera, and, at the same time, the importance of not attending to one at the neglect of another part of the body.

We now have a patient on whom the ravages of time and disease have set their stamp. It is seen, by pitting on pressure, that his ankles are both œdematous. His face, marked with fine red streaks, is pale, and his lips are slightly cyanotic. The pulse is irregular and small. He tells us that exertion produces shortness of breath, and that upon two occasions he has had hæmoptysis. Hence there is evidence of an increased venous and a diminished arterial pressure. By palpitation we find the

apex of the heart a little to the left of its normal position, with its impulse fairly strong, while epigastric pulsation is forcible. Upon auscultation there is accentuation of the pulmonary second sound, and just over the apex can be heard a systolic murmur. This murmur is not heard in the back; neither is it carried to the left nor to the right. It is the indication of mitral regurgitation, but there is probably very little regurgitation as compared with the amount of obstruction at this orifice.

Delafield says: "The same lesion frequently produces both stenosis and insufficiency of a valve." And here we have the physical signs that most commonly proclaim this condition.

Such a murmur is much oftener present than the auricular systolic, or so-called mitral presystolic, murmur with this lesion. A feeble mitral systolic murmur, due to a weak ventricle, is also confined to the area of the apex, but that is not the case in hand. Exceptionally, a systolic murmur of the kind we have here gives place temporarily to a presystolic murmur. Obstruction of the mitral orifice produces an accentuation of the pulmonary second sound. This is brought about in two ways--first, by pulmonary engorgement and consequent hypertrophy of the right ventricle, and, second, by the diminution of the aortic second sound from decreased arterial pressure in the general circulation.

When hypertrophy of the left auricle is sufficiently in excess of dilatation to follow up the at first passive flow of blood by a firm contraction, in completion of auricular systole, we may observe the so-called mitral presystolic murmur. But under other conditions of stenosis, unless the valve is closed during ventricular systole, such a systolic murmur will be produced as we find here. The case is of further interest in that we are

able to restore the arterial circulation, sufficiently at least to do away with the dropsy. In order to accomplish this we must increase the quality of his blood by food, rest, and tonic medicines. By the addition of digitalis, compensation will be re-established, and thus nature assisted by a timely and not unintelligent interference.

The history and symptoms of patient number three are of a cough, attended at one time by white frothy sputa, at another by muco-purulent expectoration. This began with an hæmoptysis early in the spring. She has night sweats, and believes she is losing flesh and strength. A brother and a sister died of consumption. We are led naturally to suspect incipient phthisis; but the diagnosis of the early part of the first stage, by physical signs, is not always an easy matter; and it is quite beyond the reach of one who has not given some special attention to the subject. We know that fremitus is more marked at the right apex in health than at the left, that the pitch is higher on percussion, that expiration is higher in pitch and longer in duration upon auscultation, and also that vocal resonance is exaggerated. With these signs at the left apex we should be almost certain of phthisis. How, then, are we to determine whether there is phthisis upon the right side? In the first place, the disparity seems to be greater than is found ordinarily in health; and, secondly, there is evidence of circumscribed bronchitis, shown by the localized subcrepitant râles. Besides, there are a few crackling and crepitant râles, indicating some slight co-existing pleurisy and pneumonia.

These adventitious signs are confirmatory evidence, and, taken in connection with an elevation of temperature, complete the diagnosis.

The advantage of detecting the presence of phthisis at an early stage is very great, for that is the time in which judicious treatment is productive of the best results. While, unfortunately, the greater number do not, still it is a well-established fact that patients do recover from phthisis. We find this demonstrated in autopsies, when death has taken place from other causes, by the presence of cicatrices or encapsulated cretaceous remains of old phthisis. It is also within the experience of many of us to have watched the progress toward recovery. Localized pneumonia undergoes resolution, circumscribed bronchitis disappears, and with them all decisive evidence of pulmonary phthisis.

The next patient comes with history of a cough which has lasted nearly two years, associated with night sweats, loss of flesh and strength, but no hæmoptysis. His father and a brother died of what he thinks was consumption. Upon inspection, we see that he is much emaciated, especially about the chest. Under both clavicles there is depression, the retraction being more marked upon the right side. On palpitation, we find fremitus exaggerated upon the right side, and his respirations are twenty-four a minute. Light percussion shows dullness in the upper part of the right infra-clavicular region and over the upper half of the left side of the chest, while forcible percussion brings out cracked-pot resonance from the left infra-clavicular region. Auscultation reveals bronchial breathing and bronchophony over the right, with amphoric respiration and whispering pectoriloquy in front upon the left side, while behind on the left side are large and small bubbling râles. Thus we have an example of the beginning of pulmonary phthisis at the same time with one approaching its end. It is but ill-conceived advice that sends a patient with

lungs in this advanced stage of destruction from home and friends, to find discomfort and finally death among strangers in a strange land; and it would seem that a knowledge of the physical signs of disease should enable physicians to avoid doing this thing.

There are a few cases, to be sure, where phthisis advances to the stage of excavation and remains stationary, the patient practically recovering, but this is rather exceptional. If the physician can decide that the phthisis is non-progressive, and finds the pulse good and the general condition of his patient fair, he may give a guarded favorable prognosis, and possibly allow him to try a change of scene and climate.

To return, then, to our opening proposition, we see in all these cases that a comprehensive knowledge of disease is quite indispensable to a specific understanding of the malady under which each patient labors. And without physical exploration there is no certainty, for different diseases have so many symptoms in common that dependence upon symptoms alone is often misleading. By a thorough examination of each case the physician exhausts all possible causes of any given complaint. He begins to know definitely the matter in hand. He knows what is not as well as what is before him. He can direct his remedies to the true seat of the disorder, and he is not under the necessity of trying what may prove an ill-judged experiment, while without this examination his diagnosis is more or less guesswork. He may be right, for a guess has always one chance of being right; but he will often be wrong, and wrong, too, from avoidable causes.

I cannot, therefore, urge upon the student too forcibly the importance of a thorough familiarity with physical diagnosis. He will not fail to be convinced of its supreme value in all cases of thoracic disease.



