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THE CIRCULATORY SYSTEM IN TUBERCULOSIS.*

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In order to understand how the circulatory system is affected in tuberculosis it is necessary, first, to understand normal conditions, and second, to understand what there is in tuberculosis which disturbs this normal action. The circulatory system provides the water for the body cells: 1, in which they live; 2, from which they take up the substances which are necessary to their life and activity, and 3, into which they give off products which have a regulatory effect upon the organism and deleterious products which result from their activity.

The body cells are capable of taking up and giving off water. This water content is subject to change from time to time under varying conditions, which depend upon their acid base balance. This varies with many pathological states which are met in practice, such as toxemia, anaphylaxis, asphyxia, and anesthesia. Any change either toward greater or lesser alkalinity on their part increases their water absorbing power and correspondingly decreases the relative amount of free water in the tissues. The amount of water that can be absorbed beyond the normal, as given by Fischer (1) is surprising. He says that even the difference in absorption of water by the red and white corpuscles in venous blood under the influence of carbonic acid concentration as compared with arterial blood amounts to from five to fifteen per cent.

Any marked variation in water content is accompanied by corresponding variation in function,

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but just what effect this exerts upon the individual we do not yet fully understand. A slight variation in the acid base balance of the cells in the nature of an increased acid content is expressed as fatigue; a marked variation is at least accompanied by such gross disturbance in physiological balance as is represented by malaise, lack of endurance, loss of strength, loss of appetite, digestive disturbance, aching, rise of temperature, and, if the brain cells are involved, delirium and coma. Therefore, it should be a part of clinical study to determine what factors disturb the acid base balance of the body cells and the free water balance of the body, and what part they play in the production of symptoms.

The heart and bloodvessels form the system which keeps the body cells supplied with water, and if the cells are to work to best advantage this function must be carried on regardless of the changes which occur in the water containing power of the body cells themselves and regardless of the amount of free water in the body. It will be readily understood that anything which disturbs the acid base balance of the cells and changes the amount of free water in the body or reduces the functioning power of the heart or alters the texture, elasticity or size of the vessels, will interfere with the efficiency of the circulation. In tuberculosis there are conditions which are inimical to circulatory equilibrium, which produce their effects in each of these ways.

In discussing a disease with so many and so varied manifestations as tuberculosis, it is necessary to make plain that it causes changes in the organism which vary from disturbances in equilibrium scarcely perceptible to those which impair the action of every system. It is further necessary to realize that the circulatory disturbances will vary accordingly.

Tuberculosis is an infectious disease and when active gives origin to toxins which gain access to the blood stream and produce their deleterious effects on the body as a whole. Toxins show a particular

affinity for the nervous system and through their action on the vegetative system affect every tissue of the body. One effect of toxic action is to make the tissues more acid. As a result of this they bind more than the normal amount of water, and are disturbed in the normal performance of their function. The heart and bloodvessels bear their share of this injury. Not only is the amount of free water reduced by this reduction in normal alkalinity, but, if the reduction be very marked, the function of the cells is disturbed and existence of the body as a living unit is threatened.

Conditions are often met in tuberculosis, particularly in the advanced phases of the disease, in which circulatory efficiency is disturbed by alterations in the character of the circulatory fluid and in the efficiency of action of the body cells themselves. All changes of this kind throw extra burdens upon the circulatory apparatus at a time when the efficiency of the heart and vessels is reduced because of other handicaps which will be mentioned later.

The mechanism of the circulatory apparatus is interfered with in many ways which are evident to clinical observers. In discussing the competency of the heart and bloodvesesls to meet the circulatory needs in tuberculosis, we have very different conditions to describe in early and in late tuberculosis. At first the heart is fully competent to cope with all factors which interfere with it. The toxic effects are slight and whatever reflex disturbances are present are unable to make serious impression; consequently, except under undue stress, the heart is able to maintain its physiological balance. In more advanced tuberculosis, the physiological reserve is used up and the heart functionates at a disadvantage.

The heart undergoes degenerative changes as toxemia becomes chronic; and the patient approaches an extensive disease which is accompanied by many conditions which call for increased circulatory demands, with a heart which has been deprived of more or less of its reserve strength.

Toxins act upon the nervous and endocrine systems as a whole but cause peripheral effects which are expressive of stimulation of the sympathetic nerves and sympathicotropic glands of internal secretion. This tends to accelerate the heart action and to disturb the vasomotor equilibrium.

Pulmonary tuberculosis and pleurisy both reduce the mechanical action of the diaphragm. The diaphragm is the most important muscle of respiration. It has a pistonlike action and when it contracts (descends) it enlarges the thoracic and reduces the abdominal area. The effect of this is to dilate the lungs and the right auricle thus favoring the ingress of air to the lungs and blood to the heart. At the same time there is also an active force exerted upon the great blood containing reservoirs of the abdomen by the compressing action of the diaphragm, by means of which the blood is literally squeezed out, as described by Wenckebach, (2) like water from a sponge. This aspiration of blood into the right heart by the inspiratory act is one of the important extracardiac mechanisms by which the circulation is maintained.

Decrease in the inspiratory act is recognizable in very early cases of pulmonary tuberculosis, because the strong crura of the diaphragm are innervated by the phrenic nerves and through them are thrown into spasm when the pulmonary parenchyma is inflamed. In advanced cases this effect is more marked and more easily recognized. In many cases of pulmonary tuberculosis, pleural adhesions, particularly at the base, are also present as another factor in producing the same effect. The lesion in the lung itself by decreasing the elasticity of the tissue also reduces the normal inspiratory excursion.

As a result of all of these factors the blood pressure is low, the arteries of the body are relatively empty, and the veins are relatively full. Most patients who are even moderately advanced in the disease show some cyanosis. In many cases of far advanced tuberculosis large areas of pulmonary ves-

sels are obliterated. One would naturally think that this would throw a tremendous overload on the heart and, in fact, that has been the teaching; but experiments have been performed on animals in which one half of the pulmonary area has been removed without showing any lowering of the systemic blood pressure. The remaining vessels dilated to such an extent that the circulation was carried on with no recognized embarrassment. It is also a common experience today in inducing artificial pneumothorax to collapse an entire lung. This may be done without producing any serious circulatory embarrassment. While these facts show that the heart is able to withstand whatever extra burden is thrown upon it by this procedure, yet it would not be safe to advance them as arguments to prove that no extra load is thrown on the heart by destroying large areas of pulmonary vessels. These observations must be interpreted rather as showing that, in instances where no untoward effects appear, the heart is able to carry on its function in spite of the decrease in vascular area.

The heart performs its function best when lying freely in the pericardium in its normal bed between the two lungs; however, if any considerable amount of lung tissue is destroyed in one lung, either by cavity formation or fibrous changes, that lung contracts and becomes smaller. The opposite lung takes upon itself a state of compensatory enlargement. The result is that the heart is disturbed in its relationships to the surrounding tissue. The contracting lung often withdraws that portion which is normally interposed between the heart and the chest wall and the enlarging lung on the other hand increases correspondingly in volume that portion of its tissue which lies between the heart and the chest wall.

Another condition which militates against its normal working is the shifting of the heart to the right or to the left with the pathological changes which occur in the pulmonary tissue. Sometimes the heart, following a contracted right lung, will shift entirely

to the right of the median line, and more often following a contracted left lung will shift to the left. In some cases the entire organ lies considerably beyond the left border of the sternum. As the heart shifts its position it is compelled to functionate at a corresponding disadvantage.

Pleuropericardial adhesions also interpose obstacles to normal action. In instances where the heart is markedly shifted to one or the other side the pericardial space is encroached upon since the sac cannot move *in toto* because of its attachments. Instead of having the heart moving freely and unrestrictedly in the pericardial sac, we have it pushed over to one or the other side of the pericardium while the other is encroached upon by the compensatorily enlarged lung. In other instances we find an adherent pericardium which embarrasses the heart in its rhythmical contraction and expansion.

In pulmonary tuberculosis the innervation of the heart and its endocrine control too are disturbed. During toxemia the action of the sympathetic nerves and such sympathicotropic internal secretions as that from the adrenal body and the thyroid increase the tendency to accelerate the heart beat. This is also favored by the shifting of the acid base balance (acidosis) which results from the toxins as well as many other causes. On the other hand, the pulmonary vagus of the parasympathetic system is stimulated and sends reflex impulses to the heart which attempt to slow the contractions. The resultant rate depends upon the relative strength of these opposing stimuli.

In this brief discussion I have attempted to show a few of the complex factors which are operating to disturb the normal circulatory efficiency in pulmonary tuberculosis and from this we may draw conclusions how to aid the circulatory mechanism in its service to the organism during the time that the body is combatting the disease. It is evident from this description that there is practically no therapeutic problem as far as the heart is concerned

in early clinical tuberculosis, but that the problem grows and becomes a serious one as the disease advances with its prolonged toxic action and its tissue destruction. When we say, then, that early tuberculosis is practically always curable, the cure of the patient at this favorable time should be the answer to the heart problem. But it will not be, for cases of advanced tuberculosis will continue to be found which we as physicians will be called upon to treat.

The particular group of symptoms which destroy the tuberculous patient are those which are due to the toxins. As a part of the general action of toxins there is a disturbance in integrity and functional activity of all body cells. The heart and bloodvessels share this injury and it is probable that this proves to be the one greatest factor in interfering with the normal working of the circulatory mechanism. It is reinforced, however, by many others which increase its difficulties.

Therapeutic measures in advanced tuberculosis should be directed first toward warding off toxic effects and second toward improving circulatory efficiency when it is disturbed. Needless to say rest is the most effective of all measures of relief which can be directed toward the heart. This is evident from the fact that it requires six to eight times as much blood for muscles in action as it does for muscles at rest, and that the lungs are called upon to furnish six to eight times as much oxygen to provide the increased energy. Translating heart contractions into terms of work the heart does an amount of work daily equivalent to raising a ton weight one hundred and twenty-two feet. Exercise increases this and active tuberculosis interferes with the normal circulatory mechanism and forces the heart to work at a disadvantage. When the disease is active rest should always be prescribed for safeguarding the heart as well as for its beneficial effect upon the disease as a whole.

Since the main heart condition met in tuberculosis which is directly traceable to the disease is of the

degenerative type there is little in the way of therapy that can be applied when once the organ begins to show signs of weakening. Strain upon the heart muscle usually manifests itself by vague symptoms. The patient begins to show signs of nervousness, irritability, anxiety, insomnia, lagging appetite, and usually disturbed digestion. Rarely is pain present, although it may be at times. This condition should be met by rest, mental and physical. Bromides and opiates will aid in bringing about both, although the latter should be discontinued as soon as the critical condition is past.

Digitalis in large doses, twenty to thirty drops of tincture three times a day, should be administered for its effect in slowing and strengthening the contraction and thus favoring the filling of the coronary arteries and improving the heart's nutrition. Digitalis should not be given in small doses, but in sufficient amount to produce slowing of the pulse or nausea within a very short time. Intravenous preparations are even better than those given by the mouth. If the heart passes the crisis digitalis should be kept up for some time. I usually give it for a few days, then discontinue for a few days and resume again. Little help should be expected, however, when frank dilatation comes on as a result of the disease, for then therapeutic measures are too late. The heart has fought its battle and lost.

One must always bear in mind that conditions of toxemia are accompanied by an increase in acid radicles in the body cells and a decrease in free water in the body. While the degree of acidosis may not be severe, yet, lasting as it is apt to do over a long period of time, it produces its deleterious effect upon the organism as a whole. It may be combated by the administration of alkalies. It is my custom to combat this condition by the use of bicarbonate of sodium. I administer about ninety grains a day for a period of several weeks, then discontinue for a week or two and resume again. It should be accompanied by large quantities of water, so as to

keep an ample free water supply for the body cells. Or it may be used by the Murphy drip, employing a three per cent. solution and continuing three to four hours a day for two or three weeks or more at a time.

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