In sharp contrast to the above findings during the convulsions of insulin shock, a very marked increase in the serum phosphate and serum potassium has been found during strychnine convulsions in rabbits, in which condition, as is well known, an extreme destruction of muscle glycogen occurs.

In seeking an explanation of the above phenomena, attention is drawn to the recent work of Embden, Meyerhof, A. V. Hill, and others, which indicates that a hexose diphosphate is an intermediary between glycogen and lactic acid in the contractile process in the muscles. It is suggested that an analogous phosphate compound is formed during the process of storage of glycogen and that insulin causes or accelerates its synthesis. This would account for the disappearance of phosphate into the tissues during the period in which insulin is acting, and the subsequent increased excretion of phosphate may be due to the further conversion into glycogen of the hexose portion of the hexose diphosphate, thus leaving the excess of phosphate available for excretion. The massive, rapid breaking down of the phosphate compound during the tetanic convulsions of strychnine, would further account for the appearance of inorganic phosphate (as well as lactic acid) in the blood stream, as has actually here been shown to occur. The shift in concentration of the potassium indicates the formation of a monopotassium salt.

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An electrocardiographic sign in pericardial effusion.

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The electrocardiogram as ordinarily taken is a record of the differences of potential occurring between various parts of the body remote from the heart. While these differences of potential can be shown to be due to a primary electrical effect in the heart muscle itself, it is evident that they also depend on the nature of the body tissue as a conducting medium. Thus, if the body were composed of a non-conducting material, it is obvious that the primary cardiac electrical effect would not give rise to differences of potential in remote parts, while, on the other hand, if the body possessed the property of a good conductor, such as copper, it can be shown that the difference of potential between parts remote from the heart would be infinitesimal.

These considerations led us to believe that the presence of a large effusion surrounding the heart, especially a large pericardial effusion, might manifest itself by a lowering of the voltage of the electrocardiogram. This belief has been reinforced by our observation of seven cases of low voltage associated with large pericardial or pleuropericardial effusions. In some of these cases the markedly low voltage of the electrocardiogram led to a suspicion of effusion, which was later confirmed by X-ray examination, aspiration or autopsy finding.

The characteristic finding in the electrocardiogram is a decided lowering of voltage of the main deflection in all three leads. This is not necessarily associated with any constant alteration in the shape of the various waves, although it is quite conceivable that the effusion may also alter the electrical axis of the heart in some cases, or cause other changes in the propagation of the electrical disturbance.

It is possible that fairly large effusions may fail to effect the voltage of the electrocardiogram appreciably in certain cases. It is also quite true that some patients have electrocardiograms with low voltage not associated with any effusion, *e. g.*, cases with myodegeneration. However the occurrence of the electrocardiographic finding in fairly constant association with an effusion and the fact that such a relation is to a certain extent predictable in advance, renders it significant and suggests further clinical and experimental observation.