the alkali is stopped, the carbon dioxide sinks again to normal limits. We have found this method of much assistance in diagnosis and consequently of great value in the institution of treatment.

A report upon further phases of intoxication will subsequently be made.

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Fibrinogen deficiency in hemophilia.

By ALFRED F. HESS, M.D.

[*From the Research Laboratory, Board of Health, New York City.*]

The amount of fibrinogen of the blood seems to vary within wide variations both in man and in animals. In hemophilia there have been some estimations of the percentage of fibrinogen based upon the amount of fibrin obtained after coagulation. However, these data are so divergent as to allow of no satisfactory deduction, quite apart from the fact that they give no information as to the quality of the fibrinogen.

In the estimations of fibrinogen here reported, a functional method has been made use of. Precipitated fibrinogen, made approximately according to the method of Hammarsten, has been added to the whole blood of cases of hemophilia, of purpura, and of normal adults and children. To ten drops of blood, one, two, and three drops of fibrinogen have been added; a fourth tube serving as a control. In this way we are able to ascertain whether the fibrinogen had a complementary action in hemophilia, as compared to the other cases, and also whether it brought the clotting time of the blood close to the normal. In all cases, the fibrinogen had been previously tested with calcium and found not to clot over night upon the addition of a few drops of a ½ per cent. solution of calcium chloride. In three cases of typical hereditary hemophilia, repeated tests showed that the addition of one drop of the fibrinogen solution to the whole blood markedly hastened the coagulation time. In one of these instances, a case of severe hemophilia, the clotting time was reduced by fibrinogen in four consecutive tests from 90 to 13 minutes, from 55 to 14 minutes,

from 106 to 87 minutes, and from 3 hours to 16 minutes. On the other hand, similar tests of three cases of purpura brought about no such result, the coagulation time remaining either the same or being delayed by the fibrinogen. This was true likewise when the fibrinogen was added to normal blood.

There is a significant difference between the clot formation of hemophiliac and normal blood. This can be clearly seen when we compare the clots of the colorless oxalated plasma. The normal clot shows a web composed of radiating threads of fibrin; the clot of typical hemophilia on the other hand is gelatinous and contains a basic material resembling powder rather than fiber.

In view of these results, it is concluded that there is a functional deficiency, qualitative or quantitative, of fibrinogen in hemophilia. This, however, does not seem to constitute the essential defect in this desease, for the addition of fibrinogen was frequently not able to bring the coagulation time to normal, nor does the local application of fibrinogen to the bleeding point bring about effective clotting. It is probable that a deficiency may be associated with other pathological conditions.

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Reflex vasodilation is not the cause of the collapsing pulse of aortic insufficiency.

By CARL J. WIGGERS.

[*From the Physiological Laboratory, Cornell University Medical College, New York City.*]

In 1908 Stewart[1] pointed out that the sudden rise and fall of the pulse and the low position of the dicrotic notch, in short the well-know "collapsing pulse" so frequently found in aortic insufficiency, could not be due to a regurgitation for (1) the rapid fall occurred before the dicrotic notch and hence during systole and (2) volume curves of the heart show that very little regurgitation takes place in experimental lesions. The changes were therefore attributed to a reflex vasodilation for (1) this would

[1] Stewart, *Archives of Internal Medicine*, 1908, I, 102.