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.

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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.

adequately account for such a pulse; (2) irritation of the aorta without insufficiency *apparently* caused a fall of diastolic without a corresponding fall in systolic pressure; and (3) when peripheral vessels were constricted by adrenalin during insufficiency the normal contour returned.

By producing *temporary valvular lesions*[2] while the aortic pressure curve was being recorded by an optical manometer, the following facts have been shown:—

1. When a lesion is suddenly produced the change in pulse occurs within the time interval of one heart beat, and when normal valve action is restored the normal type of curve *immediately* returns. The changes are too rapid to be attributed to a reflex vasodilation.

2. Irritation of the aorta never produces such a change. The records submitted by Stewart as evidence are clearly due to changes in heart rate.

3. The arterial pressure curve shows that while the pressure falls a little more rapidly in systole so that the pressure at the beginning of diastole is slightly lower, *the chief drop responsible for the large pulse and low diastolic pressure occurs during diastole.* The fact that the chief drop occurs before the dicrotic notch, when records are taken with the Hürthle manometer from animals or with a sphygmograph from patients, can be attributed to instrumental error.

4. These characteristic effects in the arterial pressure curves persist after a large part of the peripheral circulation is eliminated by clamping the thoracic aorta and, contrary to the observations of Stewart, the effect is intensified after adrenalin.

5. Changes in the arterial pulse similar to those found in animals can be obtained from an artificial circulation machine when the peripheral resistance remains unaltered.

These results indicate that the changes of pressure evidently responsible for the collapsing pulse, cannot be due to a reflex vasodilation but are due to a *regurgitation of pressure* into the ventricle, whether with or without an actual backflow of blood requires further investigation.

[2] Wiggers, Du Bois, PROC. OF SOC. FOR EXP. BIOLOGY AND MEDICINE, 1913, X, 87.