

**On intravascular clotting / by L.C. Wooldridge ; communicated by Professor Burdon Sanderson.**

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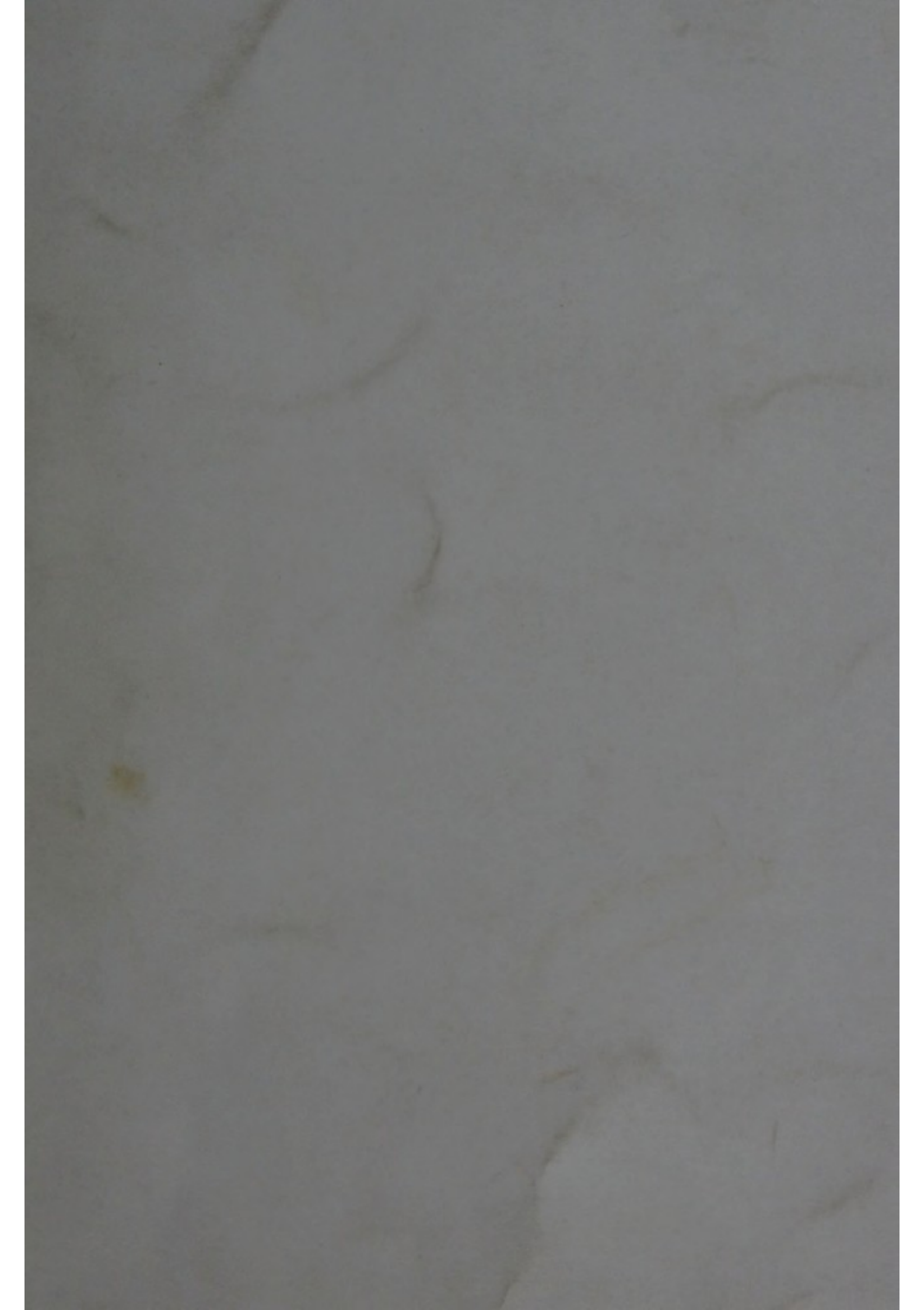
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“On Intravascular Clotting.” By L. C. WOOLDRIDGE, M.B., D.Sc., Demonstrator of Physiology in Guy’s Hospital (from the Brown Institution). Communicated by Professor BURDON SANDERSON, F.R.S. Received January 21, 1886.

Notwithstanding all the work that has been done on the subject of the coagulation of the blood, the definite results which have been obtained as to intravascular clotting are extremely scanty.

I think most physiologists will agree with me in the statement, that no method is known by which one can, at will, produce a complete fibrinous coagulation in the vessels of a living animal. I have found such a method, and one which appears to be infallible in its action.

I have succeeded in obtaining from the testis and thymus gland of the calf, a substance presenting the characters of a proteid, the injection of which in sufficient quantity into the veins of an animal, will cause instant death, owing to widespread intravascular clotting.

In its preparation I proceed in the following manner:—The organ having been finely minced, is mixed with a large quantity of distilled water and allowed to stand for some hours. The liquid is then strained off and subjected to the action of a centrifugal machine so long as any deposit is separated from it. The clear liquid is then made strongly acid with acetic acid, whereupon a bulky precipitate appears, which is collected by the centrifugal machine, and well washed with water acidified with acetic acid.

If this precipitate is dissolved in alkaline salt solution and injected into the circulation, it produces intravascular clotting. If the quantity injected is considerable (1 to 2 grams), it causes instant death in a dog of moderate size with complete thrombosis of the vena porta and its branches. Clots are also found in the right side of the heart and in the pulmonary artery. In a rabbit I found that the injection of 1 gram caused death before the injection was completed. Here there was thrombosis of the portal vein, iliac and renal veins, and of the vena cava and aorta, and clots in both sides of the heart.

When death occurs, the blood which flows from a cut artery fails



to coagulate, and when the quantity injected is insufficient to kill, the blood (drawn off after injection) may remain uncoagulated for some days. In either case coagulation of shed blood may be induced by the addition to it of the liquid which has been injected. It therefore appears that the agent which brings about coagulation, *intra venas*, must disappear in the act of coagulation. The shed blood contains only a minute trace of fibrin ferment.

The acetic acid precipitate is soluble\* in 0.5 per cent. HCl solution. On digesting this solution at 37°, after the addition of pepsine, a part of it is converted into peptone, but a precipitate appears in the process which is permanent. When the digestive products (peptone and precipitate), having been rendered alkaline, are injected into the circulation, no effect is produced.† There is neither intravascular coagulation, nor is the blood deprived of its power of coagulation; but if fresh acetic acid precipitate be added to the liquid, both effects follow injection. Consequently, the failure of effect when the products of digestion are injected alone, is not due to presence of pepsine or peptone. I have ascertained that the acetic acid precipitate does not cause coagulation of dilute magnesium sulphate plasma, which coagulates readily on the addition of fibrin ferment. The agent, therefore, in producing intravascular coagulation cannot be identified with that body.

\* As casein is "soluble" in milk.

† The total quantity of peptone is very small.



