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## Fat emboli and shock.

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Among the many explanations as to the cause of the circulatory failure in clinical as well as experimental shock, the theory that it is caused by fat embolism has been recently suggested. At least two distinct views as to the manner in which fat emboli may produce the circulatory failure are held: According to Porter (1), as we understand his view, fat, injected intravenously or gaining access to the venous circulation after fractures or laceration of the subcutaneous tissues, *passes through the pulmonary vessels but lodges in the peripheral systemic vessels and thereby produces circulatory failure by some mechanism as yet not clearly explained.* According to Bissell (2) the circulatory failure of postoperative or traumatic shock is caused by the lodgement of fat emboli in the pulmonary vessels, making it synonymous with pulmonary embolism.

During the past ten weeks we have re-investigated the following questions: (1) Is the mechanism by which the circulation fails after intravenous injection of oil the same as that following operation? (2) Is circulatory failure following fat injection primarily due to emboli of the pulmonary or systemic vessels? (3) Do fatty emboli of the systemic vessels produce circulatory failure similar to that following operation and trauma? To do this the mean pressure in the carotid artery, pulmonary artery and the effective pressures in the left and right auricles were studied in naturally breathing animals.

Experiments showed that when the circulation fails during shock produced by exposing the intestines, the pressures in the systemic and pulmonary arteries fall and a *marked reduction* of the actual, as well as the effective venous pressures in the right auricle takes place. These dynamic changes, which we regard

[1] This research was carried out in collaboration with Miss A. Kuehner, Messrs. H. Belcher, H. Cooper, W. Dodd, R. Douglass, M. Holsted and J. Sutton, Jr.

as characteristic of shock, do not occur when oil as such or in emulsion is injected intravenously. The only similarity consists in the fact that the mean arterial pressure falls. The pressure in the left auricle falls, but in the pulmonary artery and right auricle rises markedly. This can be interpreted to mean only that the failure of the systemic pressure is due to pulmonary emboli, a fact verified by microscopical examination of the lungs by Professor Ewing. As the venous and pulmonary arterial pressure changes are just the reverse of those found in shock, it is preferable, for academic as well as for therapeutic reasons, to distinguish circulatory failure produced by fat emboli from that due to surgical shock.

Although pulmonary embolism is apparently primarily responsible for the failure of arterial pressure when fat is injected intravenously, the fact that some of the oil passes through the pulmonary vessels and produces emboli in the systemic circuit (Bissell (2); Warthin (3); preparations kindly examined by Professor Ewing) raises the question whether such systemic emboli may not produce circulatory failure similar to that found in shock. To test this possibility, oils as such and in emulsions were directly introduced into the arterial circulation via the left brachial artery, thus avoiding pulmonary emboli. The effects of such injections were variable, depending apparently on where the fat emboli lodged. In some experiments large quantities of oil could be injected without any apparent effect on the arterial pressure In one case 24 c.c. of oil in emulsion and 8 c.c. as neutral oil were injected without any effect on the arterial pressure. In other cases the injection of oil produced a slight rise of arterial pressure, after which the pressure gradually recovered to normal. In a third group of cases the arterial pressure after a slight initial rise fell rapidly and the animal died within a few minutes. Electrocardiograms showed that in these cases the heart was not fibrillating but that the impulses were distributed in a normal manner to the last. Death was evidently due to respiratory failure brought about by fat emboli of the medulla. The pressure in the right auricle rose markedly, due to the cardiac failure following asphyxia. In none of these cases did the circulation fail as it does in shock.

In many instances the intra-arterial injection of oil is followed by a pronounced elevation of arterial pressure. When this occurs the effective venous pressure remains unchanged as long as the heart rate is not modified to a pronounced degree. In some cases the elevation of arterial pressure is accompanied by a marked slowing of the heart, giving the curve an appearance very similar to that following the injection of pituitary extract. From the high level thus reached the pressure gradually returns to normal and, after an hour or so, may reach a level considerably below normal. So far as our observations have gone, however, the pressure never falls as low as 50 mm. of mercury, while the venous pressure is either unchanged or elevated. The animal died in several hours of respiratory failure or from a progressive slowing of the heart, the significance of which has not been determined. The venous pressure at this stage is elevated.

Since neither the intravenous nor the intra-arterial injection of fat produces changes in the dynamics of the entire circulation which are comparable to those found in shock following exposure of the intestine, the hypothesis that fatty emboli, either of the pulmonary or systemic vessels, is the cause of circulatory failure in surgical or traumatic shock is not corroborated by our experiments.

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## Lantern slide demonstration of the effect of magnesium sulphate upon tetanus.

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Four pictures were shown. The first picture was that of a dog five days after it had received tetanus toxin subcutaneously