sequelæ; as an example, although both impede the circulation through the liver to an equal degree, yet the same result from this obstruction is not witnessed, ascites is not produced by yellow atrophy but is an unfailing consequence of cirrhosis. This last fact is not easily explained, unless it be assumed there are more elements concerned in the induction of the dropsy, than that popularly accredited. If a mechanical obstacle be competent to bring about a certain effect, when created by one method, it should be equally operative when brought about in any other method. By the agency, however, of concomitant causes the supervention of varying events may be understood. Atrophy of the liver alone will not produce ascites, but with the assistance of cotemporaneous lesions it may readily do so. Thus the seat of cirrhosis is the membrane that surrounds the portal canals, and forms the capsule of the organ, and this is continuous with the peritoneum; any irritation, therefore, originating in the former may by simple extension, be participated in by the latter and hence ascites may be more or less due to excited action, propagated to the peritoneum from the liver. But in yellow atrophy there can be no such diffusion for the disease is confined to the hepatic cells, and does not implicate either Glisson's capsule or the peritoneal envelope, so that it is not afforded any direct communication with the peritoneum.

Jaundice always attends yellow atrophy; it exhibits no peculiarities whereby it can be distinguished from a similar symptom, induced by a less formidable cause; and its duration is variable, for it may be limited to a few days, as in the above case, or be protracted over several weeks. Its occurrence is not readily understood, for by applying the generally received doctrine of jaundice to the present case, many of the phenomena of the latter are not intelligible—thus, 1. The existence of the disorder in the absence of any obstruction to the circulation of bile from the liver or gall bladder to the intestine, as demonstrated at the post mortem. 2. The continuance of biliary secretion as shewn in the full condition of the gall bladder during the formation and augmentation of the jaundice. Both of which positions contradict the presumption, that the jaundice had arisen either from retention of bile in the liver, or from its non secretion by this organ. 3. The jaundice not having been intensified in appearance, prior to or simultaneous with the supervention of head symptoms, as should have been the case, were these symptoms due to an increase in the previous amount of bile in the blood. 4. The condition of the urine, proving that the kidneys had not ceased to extrude bile. 5. The deep yellowishness of the tissues universally, which could only have been caused by pigment derived from the blood. The two circumstances last mentioned, chiefly disprove that there had been any failure, before death, in the elimination of bile from the blood. 6. The apparent performance of