death by Drs. Henderson, Mundell, and myself. The body was well nourished; very pale; the abdomen only was examined; there was about half an inch of fat on the abdominal walls, and the omentum and mesentery were loaded with soft fat; the stomach was disturbed with gas and fluid, pale but healthy; the intestines contracted and pale; the liver small, slightly fatty, but not otherwise diseased; the gall bladder distended; the spleen normal in appearance, rather small; the right kidney was normal in size and appearance; the left kidney about twice the natural size; the pelvis filled with soft fat that nearly obliterated its cavity; nearly the whole of the medullary and the greater part of the cortical portion was infiltrated with fat, by which the characteristic structure appeared to be destroyed and replaced, a few small patches only retaining the normal appearance. Owing to the objections of friends, we were unable to take it away for microscopical examination. The left ureter was normal in calibre and appearance, except that a short distance from the kidney it bifurcated and was double for four or five inches, when it joined again and entered the bladder single; the bladder was moderately distended with pale urine, the mucous membrane lining it being pale, but otherwise healthy. The patient evidently died of some other disease, and, though the left kidney was found enlarged to double its normal size, yet the writers agree that the changes noticed were insufficient to account for the phenomena. Indeed the majority of writers on the subject seem to be agreed that the original source of the discharge of blood-stained urine is not to be found in the kidney, but is to be sought either in the nervous system or in a pathological condition of the blood itself, and this view is borne out by the peculiar phenomena attendant on the occurrence of the paroxysm, these being generally brought on by exposure to cold, and commencing with a sensation of chilliness. This initiatory chill, however, seems to me to be no more than the consequence of the sudden escape of the contents of the blood corpuscles, and is paralleled by the chilly sensation that attends a sudden and copious flow of blood under any circumstances. I have just said that the majority of writers (as far as I have been able to find) regard the dissolution of blood corpuscles as occurring independently of any kidney lesion, but there are some exceptions to this view. Dr. Stephen Mackenzie, in his elaborate article on this disease published in the *Lancet* in 1884, says that he formerly thought that the kidney was the seat of the change, and that Murri held the same view but had discarded it, and he himself in the light of recent investigations felt it necessary to reconsider the question. Sir W. Gull believes there is reason for thinking that a blow or injury to the loins may be the cause of this affection, and cites a case in point; and Roberts, both in his work on urinary diseases, and in the article on the subject in Reynolds' System of Medicine, whilst admitting that injury may cause hæmaturia or even hæmatinuria, would hesitate in the absence of further evidence to accept injury as a cause of paroxysmal hæmatinuria. He regards the pathology as obscure, but it is clear that the kidneys themselves are affected, the symptoms pointing to sudden but transitory congestion of the renal capillaries, with escape of their contents, but without rupture of their walls. Into the relation that the nervous system bears to the attacks I do not propose to enter; this part of the subject has been carefully investigated and as thoroughly discussed as the present state of knowledge will allow by many, notably by Dr. Stephen Mackenzie, in the article already referred to, and also more recently in a very interesting paper by Drs. Bristowe and Copeman, published in the *Lancet* last year, recording a series of experiments performed by themselves on a patient under their care. My chief object in relating this case is to show that in some instances, at any rate, the disease may be dependent upon kidney lesion, and also to point out that owing to the infrequency with which it proves fatal, and the consequent paucity of *post mortem* examinations, the assumption that is made by most observers that the blood change takes place elsewhere than in the kidneys, and that such structural disease as has been noticed is insufficient to account for it, is based rather upon theoretical grounds than upon actual observation. It seems probable that there are at least two distinct causes for the affection; that just as jaundice may be of hepatogenous or hæmatogenous origin, so paroxysmal hæmaturia may be of