anæsthesia and subsequently to the operation, diacetic acid was found to be present before operation, and only a few of these developed severe symptoms. Indeed, it has been demonstrated that mere change of dietary, restriction (starvation) or improved feeding, is associated with diacetonuria. The pathology of the condition is certainly obscure, and not a few instances of septic changes in the organs have been advanced as cases of 'delayed chloroform poisoning.' Opie, indeed, has produced fatty changes similar to what is described as 'delayed chloroform poisoning,' by means of bacteria (*B. coli* and *Streptococcus pyogenes*). On the other hand, Leonard Guthrie and others have certainly demonstrated that the lesions—destruction of the glycogenic function of the liver, degeneration of the parenchymatous tissues of many organs, and marked fatty degeneration in the organs of the alimentary tract—occur without the incidence of sepsis, syphilis, or other distinctive disease. Association with cyclic vomiting has been suggested, but although probable, it cannot be considered proved. Most authorities agree that the perversion of the hepatic function leads to failure of metabolism of carbohydrate foods, consequent upon which is destruction of glycogen and some damage to the tissues, with liberation of fatty acids. The condition is, however, uncommon, and even those who have had wide hospital experience have seen few cases, unless we group all patients who incur prolonged post-operation vomiting as suffering from the appalling perversion of metabolism witnessed in acidosis. That the anæsthetic is but one factor in producing this toxæmia is obvious, but that it may act in this way is equally certain. Experiments on the lower animals have demonstrated that repeated inhalation of anæsthetics, and excessive strength of their vapours, as well as accumulation of the anæsthetic in the tissues, produce destructive tissue effects quite similar to those described above. The association of this toxæmia with diabetes and glycosuria is more than probable.

To estimate the prognosis is difficult, since we cannot be sure that children with normal urine will not subsequently develop acidosis; but when they are weakly, have suffered from cyclic vomiting, are febrile, and presumably suffer from sepsis, it is certainly grave unless the anæsthetic can be postponed for a few days and the carbohydrate deficiency counteracted by rectal injections of dextrose or glucose. Such rectal feeding (glucose ℥j in saline ℥vj), given every three or four hours, is excellent both before the anæsthesia and as soon as the vomiting appears. It has been suggested that the intravenous infusion of saline containing 6 per cent of dextrose offers even a better means of supplying the requirements of the organism. Whether the commonly adopted method of introducing large quantities of alkalies is of much value is open to question: it does no harm, but at best it is only treating a symptom, and not removing the cause of the pathological perversion of metabolism. As has been stated above, diacetic acid is too commonly present in the urine of all patients for its discovery before operation to veto the employment of an anæsthetic, although