Macfarlane and Currie (1943) described a case occurring in a woman aged 22 who died of fatal pyrexia following a blood transfusion. Rinvik (1941-2) described two cases in infants between 2 and 3 months who were kept alive for long periods by blood transfusions. Robson and Sweeney (1948) described a case of red-cell aplasia arising in a child of 18 months.

Smith (1949) refers to the condition as chronic congenital aregenerative anaemia. He postulates prolonged depression of the red-cell elements of the marrow by an antibody formed during foetal life. This antibody is thought to be due to ABO incompatibility between mother and child—for example, the infant's group being A and the mother's O. The history of jaundice shortly after birth would make this theory attractive in our case, but the patient's being Group O militated against the hypothesis. Further test showed no incompatibility between the child's cells and the mother's serum.

I am indebted to Dr. R. D. C. Johnstone for permission to publish this case, and to Dr. F. Graham Lescher for his advice and encouragement.

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Immediate Death after Intravenous Injection of Iodoxyl

Death following the intravenous injection of contrast media for pyelography is very rare. When it does occur it may be immediate or delayed, the immediate death being due to reaction to the drug, and the delayed death being due to pre-existing severe renal damage, which may or may not be aggravated by the injected substance. All immediate deaths hitherto reported in the literature have followed the injection of diodone (3:5-di-iodo-4-pyridone-N-acetic acid and diethanolamine), and none so far have been reported after the administration of iodoxyl (disodium-N-methyl-3:5-di-iodo-4-pyridoxyl-2:6-dicarboxylate). The following case, therefore, is of interest in that a fatal anaphylactic type of reaction occurred after the injection of this drug.

CASE REPORT

A technical mechanical engineer aged 53 was admitted to hospital on September 8, 1951, for investigation of diarrhoea, vomiting, and swelling of the left leg. These symptoms had been present for three weeks. There was nothing relevant in the past history: his general health had been good and his weight was steady.

Physical examination revealed a large fixed mass in the pelvis and gross oedema of the left leg, extending to the groin. There were no other abnormal physical signs. His blood pressure was 150/90, and Hb 92% Haldane (13.6 g.%). Urine: reaction acid, no albumin or sugar, culture sterile. Blood urea, 15 mg. per 100 ml.; serum protein, 6.48 g.%.

Sigmoidoscopy under anaesthesia revealed no growth. It seemed probable that the oedema was due to pressure of the mass on the vessels of the left leg, so intravenous pyelography was ordered to exclude ureteric obstruction by the mass.

On September 13 20 ml. of iodoxyl solution ("pyelectan") was injected intravenously. The solution had been warmed to 37° C. and was given slowly (three to four minutes). During the injection the patient was asked whether he felt any ill effects, particularly pain in the arm, which often

occurs with this preparation. The complete absence of any local reaction was remarked upon at the time. As the radiographer was about to take the five-minute picture she noticed that he failed to co-operate. He complained of dyspnoea, a tight feeling in the chest, and a desire to defaecate.

On examination he was dyspnoeic and his pulse was weak, fast, and irregular. Nikethamide, 3.5 ml., was given intravenously at once, with slight improvement, and he was returned to the ward immediately. By the time he arrived in bed (10 minutes after the injection) he had become intensely dyspnoeic and moist sounds could be heard all over the lung fields. He began to expectorate a large amount of frothy sputum. His pulse was now imperceptible and there was marked peripheral circulatory failure, with deep cyanosis. Atropine, 1/33 gr. (2 mg.), and morphine, 1/16 gr. (4 mg.), were given intramuscularly.

In spite of all efforts at resuscitation (oxygen, artificial respiration, and aspiration of the pharynx) his condition deteriorated rapidly and he died three-quarters of an hour from the time of the injection.

Post-mortem Report.—A well-nourished man. No external injuries. Heart: muscle rather flabby and fatty, otherwise normal. Lungs: acute oedema present; all air passages full of pale frothy fluid. Liver: slightly congested and slightly fatty. The spleen and kidneys were congested. The bladder was normal. In the pelvis was a small collection—a few drachms—of thick yellow pus arising from a perforated diverticulum of the rectum. There was an area of induration around the bowel, but the mucosa was normal. The stomach and intestines were otherwise normal. Oedema of the left leg was present, but no thrombosis was found. *Cause of death:* acute pulmonary oedema and myocardial degeneration accelerated by perforation of diverticulum of the rectum.

COMMENT

The empty ampoule and the remaining full ampoule of the box were returned to the manufacturers for analysis, and were found to be pharmacologically pure.

Pendergrass *et al.* (1942), in their survey of 661,800 pyelographic examinations, record 10 immediate deaths—all following the administration of diodone. Of these, three presented a clinical picture and necropsy findings very similar to the above case.

The cause of the reaction is obscure, but it is now generally agreed that it is not due to the iodine in the compound, but to some other part of the molecule. Thus, Gautot (1950) suggests that these are anaphylactic reactions, and that the sensitizing agent is diethanolamine in the case of diodone. He points out that this substance is used in the manufacture of many toilet and household preparations, and that from these sensitivity is acquired.

No reliable test for sensitivity has so far been discovered, and the prevention of an occasional very severe or even fatal reaction in a healthy patient with normal renal function seems to be impossible at present.

If the condition is recognized at once adrenaline should be given. However, in the present case it was not until the patient had developed acute pulmonary oedema that the true nature of the condition was recognized; at first, either a coronary occlusion or a pulmonary embolus appeared more likely. Even if treatment is given promptly, as it was in most of the cases so far reported, it seems to have little or no effect upon the course of the illness in the fatal case.

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