TABLE 2. Maternal and neonatal plasma 11-hydroxycorticoids 'Cortisol' concentrations ($\mu\text{g}/100\text{ ml}$)

	Age (days)	Number of cases	Range in plasma concentrations		
			Basal	Post-Synacthen	Increment
Normal Infants	1	25	11-77	40-122	12-88
	3	25	6-29	29-102	15-87
	5-7	20	6-31	21-122	7-83
Maternal					
Third trimester		12	≥ 52	—	—
Fourth day					
Post-natal normal vaginal delivery		8	≥ 55	—	—
Post-natal Caesarean section		4	≥ 73	—	—

be greater than normal would also result in temporary beta-cell hyperplasia in the infant. The neonatal hypoglycaemia and adrenocortical insufficiency already demonstrated would be not unexpected. So too, the premature labour could possibly be explained by the increased corticotrophin production which has been noted to have such an action in sheep although similar evidence in human subjects is less well documented.

There is, however, some doubt as to the origin of the possible excess corticotrophin in the mother. Ectopic corticotrophin-producing tumours are associated with clinical features which were absent in this case, and the response following radiotherapy is usually poor. Another explanation in the present case is that the stress caused by the bronchial carcinoma induced an increase in the output of normal pituitary corticotrophin. This would also account for the observations and would perhaps be easier to reconcile with the restoration of clinical and biochemical normality following treatment. In any event, the patient is being seen at regular intervals, morning and evening plasma cortisol concentrations being monitored in an attempt to detect a

possible recurrence of the condition. Estimations of plasma corticotrophin will be performed if elevated cortisol values are detected.

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Recovery from severe paraquat poisoning

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Summary

Recovery from poisoning with the herbicide, paraquat (20% concentrate—'Gramoxone') is rare (Malone *et al.*, 1971). A patient is reported who survived despite

ingesting an apparently fatal dose. Treatment consisted of forced diuresis and a single haemodialysis. Oxygen, steroids and immunosuppressives were not given.

Case report

A 28-year-old peat worker accidentally took from a lemonade bottle a large mouthful of 'Gramoxone' on an empty stomach. He noted the bitter taste and spat out what was left in his mouth. He did not vomit. Over the next 48 hr he developed a sore throat and severe dysphagia.

On admission, 52 hr after ingestion, there was superficial ulceration of the soft palate and pharynx. Scattered rhonchi were heard on auscultation. The previous medical history was unremarkable. He smoked twenty cigarettes a day.

Investigations. The abnormal laboratory findings were a urinary paraquat level of 6800 $\mu\text{g}/100\text{ ml}$; Pao_2 75 mmHg (normal 85–95); D_LCO 26 ml/mmHg/min (predicted normal 33); $\text{FEV}_1\%$ 68.5 (predicted normal 83); blood urea 68 mg/100 ml; GFR 60 ml/min; excretion of renal tubular cells 350,000/hr (normal 14,000–57,000); urinary lysozyme 2.5 $\mu\text{g}/\text{ml}$ (normal 0.63 ± 0.43). The chest X-ray showed increased vascular markings. (Fig. 1).

Treatment. Forced diuresis was begun with 200 ml of 20% mannitol and a high fluid intake, mainly oral, and output (10–24 l/day) was maintained throughout the admission. A 5-hr haemodialysis was carried out 67 hr after admission, but no paraquat was recovered from concentrates of the dialysing fluid. Oxygen was withheld, although the Pao_2 fell to 66 mmHg on the sixth day after ingestion. D_LCO did not fall below 25 ml/mmHg/min (day 10).

The renal tubular cell excretion was maximum on day 11 (735,000/hr). There was proteinuria (100 mg/100 ml), but no glycosuria, phosphaturia or aminoaciduria.

Hepatic damage developed and the bromsulph-

thalein retention at 45 min was maximal on day 14 (37%). Serial electrocardiograms showed the development of marked ST segment depression, particularly over the left ventricle, maximal at day 14.

At the time of discharge, 26 days after admission, the PaO_2 , D_LCO , $\text{FEV}_1\%$, blood urea, tubular cell excretion, and bromsulphthalein retention had returned to normal. The electrocardiogram changes were improving. Three months after discharge the patient remains well.

Discussion

On admission the initial findings of pulmonary, gastro-intestinal and renal damage were typical of paraquat poisoning and suggested a fatal outcome (Malone *et al.*, 1971). Following admission hepatic and myocardial damage developed. Urinary paraquat levels more than 48 hr after ingestion cannot be used with confidence to assess prognosis but a concentration in excess of 200 $\mu\text{g}/100\text{ ml}$ at this time is indicative of serious poisoning (Ferguson & Litchfield, 1971).

Gastric lavage and enemas and purgation were not performed, nor the administration of bentonite, kaolin, etc., because of the delay before the patient was seen. Forced diuresis, up to 24 per day, was maintained because, although in animals the excretion of the fraction of an oral dose of paraquat absorbed is largely complete within 48 hr, in man the excretion may continue for days (Grundies, Kolmar & Bennhold, 1971). Survival from poisoning with 20% concentrate is rare but three cases have been reported from Germany treated with forced diuresis and steroids (Weidenbach, 1969; Grabensee *et al.*, 1971; Grundies *et al.*, 1971). The rationale for the use of steroids or immunosuppressives, also recently suggested (Fennelly, Fitzgerald & Fitzgerald, 1971) is not clear, and they were not used in this case. Haemodialysis was carried out as a prophylactic measure in an attempt to remove paraquat despite the lack of evidence that it is of value and no paraquat was recovered from concentrates of the dialysing fluid.

It has recently been suggested that oxygen should not be given because the pulmonary toxicity of paraquat may be enhanced (Flenley, 1971). This patient was not seen until 52 hr after ingestion of paraquat, but despite progressive hypoxaemia and a reduced transfer factor, oxygen was not administered. The typical severe pulmonary changes of paraquat poisoning did not develop although the renal dysfunction persisted and hepatic and myocardial damage developed. Whether the atypical course of the pulmonary features was due to the lack of oxygen therapy, or to the intensive fluid therapy, or to some other unidentified factor is not established.

FIG. 1. Chest X-Ray—increased vascular markings.

Paraquat has been described as a 'hit and run' poison (Barnes, 1968), but in view of recent reports of the apparent value of forced diuresis, the severity of pulmonary fibrosis and the prognosis in paraquat poisoning would appear to depend not only on the quantity ingested but also on the rate of removal. This case shows that even when a delay occurs before the patient is seen that the outcome of poisoning with 20% paraquat is not inevitably fatal.

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Primary haemorrhagic thrombocythaemia with Philadelphia chromosome

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Summary

A case of primary haemorrhagic thrombocythaemia with the Ph¹ chromosome is described and its relation with myeloproliferative disorders is discussed.

PERSISTENT and marked increase of platelet count may be associated with polycythaemia vera, agnogenic myeloid metaplasia (Linman & Bethell, 1957), chronic myeloid leukaemia (Minot & Buckman, 1925), after splenectomy (Hardisty & Wolff 1955), and sometimes with no obvious cause (Ozer *et al.*, 1960), called primary haemorrhagic thrombocythaemia.

The Philadelphia (Ph¹) chromosome was discovered by Nowell & Hungerford (1960) in patients with chronic granulocytic leukaemia. Since then the Ph¹ chromosome has been demonstrated in patients who initially presented as polycythaemia, in eosinophilic leukaemia (Gruenwald *et al.*, 1965) and in primary haemorrhagic thrombocythaemia (Tough *et al.*, 1963; Dougan, Woodliff & Onesti, 1967).

I present here a further case of primary haemorrhagic thrombocythaemia with the Ph¹ chromosome.

Case report

A thinly-built married female aged 75 years was admitted to hospital in May 1970 for pain in the upper abdomen, particularly in left hypochondrium, bruises on the legs, a history of haematemesis, moderate anaemia and weight loss. The liver and spleen were not palpable but there was some tenderness in the splenic region. There was a past history of similar recurrent attacks of abdominal pain mainly in the splenic region and repeated gastro-intestinal haemorrhage for many years.

Investigations in a different hospital in 1959 revealed hypochromic anaemia with haemoglobin 10.5 g/100 ml, and normal white cell count, but a platelet count was not performed. Various biochemical investigations and radiological investigations including barium studies of the gastrointestinal tract at that time did not reveal any