Six patients, undergoing prolonged IPPV, were given single doses of pancuronium, followed some hours later by a series of incremental doses. Plasma pancuronium concentrations were determined at frequent intervals. In each instance an electrical analog model was used to construct a two-compartment open model for that patient which could be programmed to predict the effect of the ensuing increments (Hull and McLeod, 1976). Model predictions were then compared with the actual plasma concentrations. A good agreement showed that the model is likely to be valid, and as predicted, cumulation of pancuronium occurs after repeated doses.

REFERENCES

THE METABOLISM AND TOXICITY OF SODIUM NITROPRUSSIDE
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Sodium nitroprusside (SNP) breaks down in whole blood to yield free cyanide (HCN). The presence of sulphhydryl groups is probably necessary for this reaction both in plasma (Vesey, Cole and Simpson, 1976a) and in red cells. The decomposition of SNP may occur in the erythrocytes and necessitates the free transfer of the intact nitroprusside molecule across the red cell membrane (Smith and Kruszyna, 1974).

Two groups of anaesthetized dogs received either bolus doses of potassium cyanide 1.09 mg/kg body weight or bolus doses of SNP 1 mg/kg body weight. The increase and decrease of plasma and red cell cyanide concentrations in these two groups were compared in order to assess the rate of breakdown of SNP and the detoxication of HCN.

A third group of anaesthetized dogs received SNP 1.5 mg/kg body weight at a steady rate for 1 h, in order to simulate the effects in patients receiving our maximum recommended dose (Vesey, Cole and Simpson, 1976b). Measurements of arterial base deficit and plasma lactate and pyruvate concentrations were made also to test the reliability of acid-base status measurements as an index of the development of tissue hypoxia. Our results may be summarized as follows:

1. The increase in plasma cyanide concentrations occurs before the increase in RBC cyanide concentration.
2. Ultimately, red cell cyanide concentrations reach 50–100 times those in the plasma.
3. In the bolus-dosed animals KCN produced plasma HCN values 10 times those produced by SNP, whereas RBC concentrations were similar.
4. Peak thiocyanate concentrations of 40–60 µmol/litre were attained 2 h following injection or infusion.
5. In the bolus SNP and infused SNP groups of dogs the calculated mean percentage of cyanide in the nitroprusside converted to thiocyanate was 63 and 64.5%, whereas the value for the bolus KCN dogs was 82%.
6. The correlation between arterial base deficit and lactate concentrations during SNP infusion showed that the former was a reasonable indicator of the development of lactic acidosis, probably resulting from tissue hypoxia.

REFERENCES

ASSESSMENT OF LONG-TERM RECOVERY FROM SHORT DURATION ANAESTHESIA, USING THE CEREBRAL FUNCTION MONITOR

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Results from previous studies (Dubois and Geddes, 1977) have shown that patients who received single doses of thiopentone were impaired markedly on psychomotor tasks at 2 h after administration. The e.e.g. has been used also to assess the prolonged effects of anaesthetic drugs on the c.n.s. (Doenicke, Kugler and Laub, 1967), revealing that full recovery is delayed even longer. In the present study a variety of psychomotor tests were employed and the e.e.g. recorded. In addition to conventional electroencephalograph techniques, the Cerebral Function Monitor (CFM) was employed. This purpose-built device produces a readily interpretable tracing and has proved of value in a variety of acute clinical situations, for example, cardiopulmonary bypass (Schwartz et al., 1973). The aim of the present investigation was to determine the place of the CFM in the assessment of long-term recovery from short duration anaesthesia.

Eighteen female patients undergoing minor gynaecological surgery received an induction dose of methohexitone 2–3 mg/kg followed by one increment of 1/2 induction dose. On two occasions before the anaesthetic was given and seven times during the following 24 h, various tests were administered in a standard manner to minimize extraneous environmental factors. Clinical state was assessed, the CFM recorded and the response to six different psychomotor tests—"Maddox Wing", visual reaction timer, three tests of balance and "Memory Drum"—were determined. All the psychomotor tasks yielded numerical data.

Quantitative data were collected from the CFM using a digital chart reader and the paper tape obtained was subjected to statistical analysis. Six of the patients had conventional multi-channel e.e.g.'s recorded together with the CFM in order to compare the two types of tracing. The e.e.g.'s were rated visually, using a technique which gave numerical values for both the amount and duration of drowsiness and sleep.

Our results indicated that while clinical signs and performance tests returned to pre-anaesthetic levels 2 h following the injection of the anaesthetic drug, CFM tracings were still significantly different from those before injection (P < 0.05) at 8 h. In the six patients in whom the conventional e.e.g. was recorded, quantitative analysis