concentrations in blood range from 24 to 206 mg/100 ml, for any one subject the response was reasonably reproducible. Of the twelve subjects who were investigated twice ten showed such reproducibility. Indeed one volunteer who was studied a third time as part of another investigation had peak alcohol concentrations in his blood of 82, 70 and 77 mg/100 ml reached in 55, 60 and 60 min respectively. The corresponding blood flow values were (23, 21 and 20 ml/100 ml)/min. The interval between the first and last study was 10 months.

REFERENCES

CURRY, A. S., WALKER, G. W. & SIMPSON, G. S. (1966). Determination of ethanol in blood by gas chromatography. *Analyst*, **91**, 742–743.

PAYNE, J. P., FOSTER, D. V., HILL, D. W. & WOOD, G. D. L. (1967). Observations on interpretation of blood alcohol levels derived from analysis of urine. *Br. med. J.*, **2**, 819–823.

Some cardiovascular effects of intravenous atropine in patients anaesthetized with halothane

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The effect of atropine on heart-rate, rhythm and blood pressure was observed in twenty patients (average age 56 years; range 23 to 87) anaesthetized with 2 to 4% halothane in oxygen. Anaesthesia was induced with 5% thiopentone (250–500 mg) after premedication with pentobarbitone (200 mg intramuscularly). When indicated suxamethonium (40 to 100 mg intravenously) was used for intubation.

The pattern of the e.c.g. waveform was followed with a non-conventional limb lead, displayed on a multi-channel oscilloscope and charted on a Mingograph 81 recorder. The blood pressure was measured either with a Von Recklinghausen oscillotonometer or from a polyethylene catheter inserted percutaneously into a radial artery at the wrist. In ten of these patients from whom consent for the study had been obtained cardiac output determinations and right atrial pressure measurements were also made. For these measurements a polyethylene catheter 60 cm long was inserted percutaneously into a superficial vein on the forearm and threaded into the right heart. Arterial blood samples were withdrawn at intervals for the measurement of pH, Pa_{O_2}, Pa_{CO_2} and halothane concentrations.

The rapid injection of atropine (0·6 mg intravenously) was followed in all patients by a highly significant increase ($P<0{\cdot}0005$) in heart-rate which rose from a mean value of 61 to 108 beats/min and by the development of nodal tachycardia in five patients, pulsus bigeminus in four, and multifocal ventricular extrasystoles in six. The average time taken to produce these effects was 45 s (range 31 to 61). The mean arterial blood pressure rose from a mean value of 61 to 85 mmHg at the same time.

Atropine also caused an increase in cardiac output in each of the ten patients in whom measurements were made. The mean value rose from 3·88 to 5·62 l./min ($P<0{\cdot}05$). This increase was associated with a 29% decrease in the appearance time, a 33% reduction in the mean circulation time, a 20% fall in systemic vascular resistance and an 18% increase in left ventricular work. The mean halothane concentration in blood was unchanged over the period of the investigation, but in one patient a fall in concentration from 19·7 mg to 9·9 mg/100 ml blood probably influ-

enced the marked increase in output from 1·91 to 6·79 l./min. No arterial oxygen tension value fell below normal during the study and no significant changes were noted in pH nor in the arterial carbon dioxide tension.

In three patients the onset of nodal rhythm was associated with an immediate increase in right atrial pressure from 3 to 5 cm H_2O together with a slight but definite fall in arterial blood pressure. The return of sinus rhythm restored these variables to their previous values.

Practolol in the management of cardiac dysrhythmias in patients anaesthetized with halothane

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Disturbances of rate and rhythm were studied in twenty-one patients (average age 62 years, range 35 to 81) anaesthetized with halothane after premedication with pentobarbitone (200 mg intramuscularly) for a variety of surgical procedures. Induction was with 5% thiopentone (250–400 mg intravenously); when intubation was necessary suxamethonium (40–100 mg intravenously) was used, thereafter the patients breathed spontaneously. Cardiac rhythm was followed by a single non-conventional e.c.g. lead displayed on a suitable oscilloscope and charted on a Mingograph 81 recorder. Blood pressure was measured intermittently in seven patients with a Von Recklinghausen oscillotonometer and continuously in fourteen from a catheter inserted percutaneously into a radial artery. Arterial blood samples were withdrawn before and after treatment with practolol for the measurement of oxygen and carbon dioxide tensions.

The development of sinus tachycardia (four patients), nodal rhythm (four), pulsus bigeminus (five) and multifocal ventricular extrasystoles (eight) was treated with practolol (4 mg intravenously); almost invariably normal rate and rhythm was restored but in three patients a second injection was required; the mean recovery time was 39 s (range 8 to 65). The heart rate was slowed significantly ($P<0.0005$) from a mean of 108 to 85 beats/min but the blood pressure was unchanged. At the onset of the dysrhythmia no arterial oxygen tension was below normal (mean $\pm$ S.E.M. $Pa_{O_2}=227\pm83$ mmHg, range 95 to 370/mmHg) but most carbon dioxide tensions were high ($Pa_{CO_2}=62\pm15$ mmHg, range 41 to 89 mmHg). In eighteen of the twenty-one patients the onset of the cardiac disturbances was preceded by the injection of atropine (0·6 mg intravenously). In the remaining three patients not given atropine a significant fall in blood pressure ($P<0.0005$) associated with a marked reduction in heart rate followed the administration of practolol. In one patient the heart rate dropped markedly from 108 to 36 beats/min and the blood pressure fell from 115/80 to 65/40 mmHg; atropine (0·6 mg intravenously) abolished the bradycardia and restored the blood pressure.

In seven patients from whom consent for the investigation had previously been obtained, the effect of practolol on cardiac output and on the right atrial pressure was studied. No significant change in cardiac output could be detected but a 15% increase in the appearance time of the dye was noted. In no patient did the stroke volume fall and in five it improved (mean increase 26·7%). The right atrial pressure was unchanged.