

The application of positive intra-thoracic pressure caused a transient slight rise in blood pressure of about 4 s duration followed by a steep fall to less than 50 mmHg. When the intrathoracic pressure was released a further slight fall occurred for 4 s followed by a prompt recovery with overshoot in fit, lightly anaesthetized patients. The time taken for the systolic pressure to reach the previous diastolic level equalled that needed for recovery. In poor risk patients, in those deeply anaesthetized with halothane and in those given certain autonomic drugs recovery time was increased by a factor of up to 10 and the overshoot was modified or abolished.

Experience has shown that under certain conditions the stress could constitute a danger to the patient. Accordingly work is proceeding in an attempt to shorten the duration of the stress but still be able to predict the future course of the system.

During the manœuvre the pulse rate changed little, the right atrial pressure rose by up to 30 cm H<sub>2</sub>O and the plethysmograph pressures reflected the atrial pressure.

The manœuvre has the potential of being a repeatable non-invasive monitoring technique without the need for calibration.

#### **The effect of ingested alcohol on hand blood flow in resting man**

S. J. CARTER, C. E. HOPE\* and J. P. PAYNE, *Research Department of Anaesthetics, Royal College of Surgeons of England, London, W.C.2*

The relationship between blood alcohol concentration and hand blood flow was studied in fourteen healthy volunteers of both sexes with a mean age of 25 years (range 19 to 37 years). Twelve of the subjects were studied twice at intervals of 2 to 8 weeks. All studies were carried out with the volunteers fasted and resting. Hand blood flow was determined by water displacement venous occlusion plethysmography. For the duration of the experiment the temperature of the plethysmograph, the environmental temperature and the humidity remained constant. Plethysmograms were collected in batches of six at 5 min intervals throughout the study. A suitably programmed digital computer calculated individual blood flows and the mean value for each batch, and plotted the means as a function of time. At the end of each collection a blood sample was obtained from an indwelling catheter inserted percutaneously into a superficial vein on the dorsum of the opposite hand. The concentration of alcohol was determined by a modification (Payne, Foster, Hill & Wood, 1967) of the gas chromatographic technique devised by Curry, Walker & Simpson (1966). After a 30 min control period during which resting blood-flow values were determined for each subject, the volunteer drank in 2 to 3 min 100 ml 70% proof whisky diluted with 100 ml water.

The mean ( $\pm$ S.E.M.) resting blood flow for the group was  $(8.29 \pm 4.74 \text{ ml}/100 \text{ ml})/\text{min}$  derived from forty-two individual measurements on each subject. During the period of drinking the blood flows fell so sharply in every subject that measurement was impossible; thereafter they rose steadily to reach a maximum mean value of  $(17.36 \pm 10 \text{ ml}/100 \text{ ml})/\text{min}$  in  $33 \pm 15 \text{ min}$ . In twelve of the fourteen volunteers the maximum blood flow coincided almost exactly with the peak alcohol concentration in the blood. The mean value for the peak blood alcohol concentration of  $89.76 \pm 40 \text{ mg}/100 \text{ ml}$  was reached in  $34 \pm 13.6 \text{ min}$ .

A characteristic feature of this study was the fact that although the pattern of response differed widely from one individual to another, for example the peak alcohol

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**

J. A. GIL-RODRIGUEZ\*, J. P. PAYNE and D. R. POTTER, *Research Department of Anaesthetics, Royal College of Surgeons of England and St. Peter's Group of Hospitals, London W.C.2*

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,  $\text{PaO}_2$ ,  $\text{Paco}_2$  and halothane concentrations.

The rapid injection of atropine (0.6 mg intravenously) was followed in all patients by a highly significant increase ( $P < 0.0005$ ) 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 ( $P < 0.05$ ). 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-