THE ANAESTHETIC PRESSURES OF CERTAIN FLUORINE-CONTAINING GASES

Sir,—If I understand them aright, Miller, Paton and Smith (Brit. J. Anaesth., 1967, 39, 910) have compared the narcotic potencies of certain gases on the basis of the inhaled pressures that produce a given effect. Such a basis is misleading, for it ignores the widely differing solubilities of the gases and therefore takes no account of the molar concentrations in the brain that are producing the effect. For example, they rate nitrogen at 35 atmospheres as equipotent with nitrous oxide at 1.5 atmospheres, suggesting that nitrous oxide is much the more powerful of the two anaesthetics. But nitrous oxide is 47 times more soluble in the brain than nitrogen. Therefore, at these respective pressures, the brain's molar concentration of nitrous oxide will be higher than the brain's molar concentration of nitrogen; and so, judged by the narcotic activity of their molecules, nitrogen is more potent than nitrous oxide. These authors have, in fact, used for their basis (in my terminology) conventional potency, when they should, I think, have been using intrinsic potency (Bourne, 1967).

If their basis for comparison is unsound, then their conclusions will also be fallacious.

J. G. BOURNE
Salisbury

REFERENCES


Sir,—Dr. Bourne (who kindly sent us a copy of his letter) begs the question by supposing that solubility in “brain” gives a reliable estimate of “intrinsic potency”. We would entirely agree in principle with a distinction between the operational potency of an anaesthetic (which anaesthetists, one presumes, are practically concerned) and its potency when taken up at the site of action critical for anaesthesia. The question we are trying to throw light on in the paper is the physicochemical nature of that site, and (to put our result in another way, using Dr. Bourne’s terminology) have concluded that if the site is lipid, with a solubility parameter of 8-9, then all the anaesthetics for which we could obtain suitable data have the same “intrinsic potency”. This is, of course, essentially what Overton and Meyer were arguing, much earlier. The difficulty about measurements of solubility in brain is that much of the dissolved gas will be taken up by tissues irrelevant to anaesthesia; and it is only too possible that they will swamp the uptake by the regions actually critical for anaesthesia. Of course, if we knew what the critical regions (molecules?) for anaesthesia were, and could measure solubility of anaesthetics in them, then it would be possible directly to test whether “intrinsic potency” is constant or not. But for the investigator, the boot is on the other leg; and we would argue that, in seeking to identify the critical regions, one approach would be to assume the hypothesis that so far fits the most data, and direct one’s search to lipid structures with the appropriate solubility properties.

K. W. MILLER
W. D. M. PATON
E. B. SMITH
Oxford

TAPERS—OLD AND NEW

Sir,—Whilst it is most desirable to change over to one standard of anaesthetic taper union, our experience of current conversions envisaged in your Editorial (Brit. J. Anaesth., 1967, 39, 611) falls short of the desired “complete removal of all the old fittings”. The service engineer has merely interposed adaptors at all unions in delivery line and circuit in accordance with his firm’s instructions. To avoid this source of leaks and instability, the two alternatives are to install only a new B.S. expiratory mount or to order replacements of sundry fittings where needed, for older anaesthetic apparatus. The latter involves removal of the size of taper and it is not surprising that manufacturers are often unable to supply the part needed for lack of a generally accepted nomenclature of the older taper unions.

There are only about six sizes of taper on anaesthetic equipment commonly supplied in the last twenty-five years, and if it were found possible to publish in this Journal a short description and name for each, this would prove of immense value to anaesthetists during the difficult period of transition. With the help of a micrometer to identify the size, one would be able to direct the suppliers’ attention to a long overdue, classified list of taper sizes.

R. J. STOUT
Gillingham

CLINICAL STUDIES OF INDUCTION AGENTS
XX: A METHOHEXITONE-PROPANIDID MIXTURE

(Brit. J. Anaesth., December 1967)

Sir,—May I bring your attention to the German translation on page 962, which, in my opinion, gives the wrong impression in the following sentence:


I feel that the following translation would be more accurate:

“Die Erwartung, dass dieses Gemisch weniger Nebenwirkungen auslösen würde als vergleichbare Dosen von Propanidid und Methohexiton alleine, wurde erfüllt.”

L. LEISTEN
Haywards Heath
FBA Pharmaceuticals Ltd.
MEASUREMENT OF BLOOD HALOTHANE CONCENTRATIONS BY GAS CHROMATOGRAPHY IN THE DOG AND MAN

F. W. Cervenko, B. R. S. McClements and J. P. Payne
Research Department of Anaesthetics, Royal College of Surgeons of England, London

A method for determining blood halothane concentrations has been evolved based on techniques described by Butler (1963) and Wolfson, Ciccarelli and Siker (1966). A gas chromatograph incorporating a flame ionization detector was constructed using air as the source of the flame oxygen. The stationary phase consisted of 35% W/W silicone oil M.S.550 on celite 44-60 mesh packed in a 4-ft. copper column of ½ inch internal diameter maintained at a temperature of 85°C. Nitrogen provided the moving phase at a flow rate of 60 ml/min.

Halothane was extracted from the blood sample by mixing approximately equal quantities of blood and carbon tetrachloride to which had been added a trace (6 μl./100 ml) of diethyl ether as an internal standard. Extraction was facilitated by agitation in a mechanical shaker and separation obtained by centrifuging. The injection of 3-5 μl. samples into the gas chromatograph produced sharp peaks with a total elution time of 6 min and a mean recovery of 94.5% from known samples.

The method has been applied to the study of blood halothane levels during closed-circle anaesthesia in the dog and man when the vaporizer is included in the circuit. Four unpremedicated dogs were anaesthetized with thiopentone and intubated. After cannulation of the aorta and the right heart to allow arterial and mixed venous sampling, anaesthesia was continued with closed-circle halothane. Respiration was first stimulated and then depressed; the maximum depression occurred between 5 and 16 min later, at which time the mean arterial concentration of halothane was 29.7 mg/100 ml (range 15.2 to 42.5 mg) and the mixed venous concentration 22.9 mg/100 ml (range 16.0-33.5 mg). On withdrawing the anaesthetic the arterial halothane level fell rapidly and after 8-20 min was slightly lower than the venous concentration.

Four patients undergoing major urological surgery were induced with thiopentone and intubated under routine anaesthesia. A gas chromatograph which lasted for 55-125 min, was continued with halothane in a closed-circle system. Arterial and venous blood samples were obtained at approximately 10-min intervals from catheters inserted percutaneously in the left radial artery and in a superficial vein on the left forearm. The maximum arterial concentration of halothane reached was 20 mg/100 ml, with a mean of 19.7 mg; the corresponding venous values were 17.0 mg and 15.7 mg/100 ml. On withdrawal of the halothane the "cross-over" effect between arterial and venous concentrations was seen within 15 min.

REFERENCES


CEREBRAL BLOOD FLOW, CEREBROSPINAL FLUID PRESSURE AND E.E.G. ACTIVITY DURING NEUROLEPTANALGESIA INDUCED WITH DEHYDROBENZPERIDOL AND PHENOPERIDINE

Institute of Neurological Sciences, Glasgow, and University Department of Anaesthesia, Western Infirmary, Glasgow

There are conflicting reports in the literature concerning the influence of neuroleptanalgesia on cerebral blood flow. Nilsson and Ingvar (1966), working with cats, reported that both phenoperidine and fentanyl increased the blood flow through the cerebral cortex while Kreuscher (1965) found that fentanyl reduced cerebral blood flow in dogs. The present studies were undertaken in order to determine the effect of dehydrobenzperidol and phenoperidine in clinical dosage on cerebral blood flow, cerebrospinal fluid pressure and e.e.g. activity in man.

Cerebral blood flow.

Cerebral blood flow (rCBF) was measured after the combined intravenous administration of dehydrobenzperidol 5 mg and phenoperidine 1.5 mg to 6 conscious patients undergoing carotid ligation. Blood flow was measured through the closed skull by recording the rate of clearance of 14C-Xenon after its injection into the internal carotid artery. In the present communication only the fast component of the clearance curve is reported so that the flow values are those of grey matter. The results are shown in table I, from which it will be seen that the mean flow, corrected to a PaO₂ of 40 mm Hg, was 87 ± 24 ml/100 g/min.

In the table this result is compared with the results of Ingvar et al. (1965) which were obtained by a similar flow measurement technique in conscious man without neuroleptanalgesia. It will be seen that dehydrobenzperidol-phenoperidine had no detectable effect on cerebral blood flow.

Cerebrospinal fluid pressure.

In 4 patients anaesthetized with nitrous oxide, oxygen and tubocurarine, cerebrospinal fluid pressure was studied prior to and following the combined intravenous administration of dehydrobenzperidol 5 mg and
The effect on the red cell circulation of replacement of operative blood loss with different fluids

M. L. Heath, D. Dunlap and M. D. Vickers

Royal Postgraduate Medical School and Department of Anaesthetics, Hammersmith Hospital, London

It has been suggested by Shoemaker and his colleagues (Shoemaker and Iida, 1962; Suzuki et al., 1965) that in shock states a proportion of the red cell volume may become sequestered from the circulation. They have demonstrated the apparent loss of red cells from the circulation during shock and their reappearance on recovery, and have also used low-molecular weight dextran (av. mol. wt. 40,000) to return these cells to the circulation.

It was decided to investigate this phenomenon as part of a project designed to study the effects of replacing operative blood loss with either blood or Macrodex (dextran, av. mol. wt. 70,000).

Blood loss expected to total more than 500 ml was replaced with blood or Macrodex. No patient received more than 1 litre of Macrodex so that blood loss in excess of 1 litre was then replaced by blood. Five patients received blood only, two had no replacement (low blood loss) and five patients received Macrodex, two receiving blood as well. Red cell volumes were measured on recovery from anaesthesia and the 2-hour sample was compared with the mean of the 10-25-minute samples.

Patients receiving only blood showed a significant increase in circulating red cells by 2 hours (P=0.019) while those receiving nothing or Macrodex did not (P=0.63)—see table I. Clinically, the Macrodex group appeared less vasoconstricted and blood sampling was easier in the immediate postoperative period than in the group receiving blood only.

Red cell volumes were measured by a modification of the standard "Cr tagging method. Six control patients were studied in duplicate. Following injection of tagged red cells dilution samples were drawn at 10, 15, 20 and 25 minutes and at 2 hours. No significant difference was found between the 2-hour sample and the mean of the 10-25 minute samples in any patient and the duplicate runs did not differ.

Twelve surgical patients had their blood loss measured by swab weighing and the anaesthetist made an estimate of total blood loss on the basis of this. Blood loss expected to total more than 500 ml was replaced with blood or Macrodex. The 2-hour sample was compared with the mean of the 10-25-minute samples.

No significant difference was found between the 2-hour sample and the mean of the 10-25 minute samples in any patient and the duplicate runs did not differ.

Electroencephalograms.

Records were obtained from the conscious patients in whom cerebral blood flow was measured. The administration of dehydrobenzperidol-phenoperidine resulted in some reduction of frequency in the alpha range with occasional slowing to the theta range. This is compatible with a normal sleep record.

THE EFFECT ON THE RED CELL CIRCULATION OF REPLACEMENT OF OPERATIVE BLOOD LOSS WITH DIFFERENT FLUIDS

M. L. Heath, D. Dunlap and M. D. Vickers

Royal Postgraduate Medical School and Department of Anaesthetics, Hammersmith Hospital, London

It has been suggested by Shoemaker and his colleagues (Shoemaker and Iida, 1962; Suzuki et al., 1965) that in shock states a proportion of the red cell volume may become sequestered from the circulation. They have demonstrated the apparent loss of red cells from the circulation during shock and their reappearance on recovery, and have also used low-molecular weight dextrans (av. mol. wt. 40,000) to return these cells to the circulation.

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BRITISH JOURNAL OF ANAESTHESIA

Mean results for rCBF (fast component) in conscious man without neuroleptanalgesia (Ingvar et al., 1965) and following the intravenous administration of phenoperidine 1.5 mg and dehydrobenzperidol 5 mg (present series). In both groups flows have been corrected to a Pa02 of 40 mm Hg.

| Neuroleptanalgesia (n=6) | 87 ± 24 |
| No drugs (n=7) (Ingvar et al.) | 80.5 ± 14.8 |

TABLE I

Mean results for rCBF (fast component) in conscious man without neuroleptanalgesia (Ingvar et al., 1965) and following the intravenous administration of phenoperidine 1.5 mg and dehydrobenzperidol 5 mg (present series). In both groups flows have been corrected to a Pa02 of 40 mm Hg.

| Neuroleptanalgesia (n=6) | 87 ± 24 |
| No drugs (n=7) (Ingvar et al.) | 80.5 ± 14.8 |

REFERENCES


THE EFFECT ON THE RED CELL CIRCULATION OF REPLACEMENT OF OPERATIVE BLOOD LOSS WITH DIFFERENT FLUIDS

M. L. Heath, D. Dunlap and M. D. Vickers

Royal Postgraduate Medical School and Department of Anaesthetics, Hammersmith Hospital, London

It has been suggested by Shoemaker and his colleagues (Shoemaker and Iida, 1962; Suzuki et al., 1965) that in shock states a proportion of the red cell volume may become sequestered from the circulation. They have demonstrated the apparent loss of red cells from the circulation during shock and their reappearance on recovery, and have also used low-molecular weight dextran (av. mol. wt. 40,000) to return these cells to the circulation.

It was decided to investigate this phenomenon as part of a project designed to study the effects of replacing operative blood loss with either blood or Macrodex (dextran, av. mol. wt. 70,000).

In no patient did the 10, 15, 20 and 25-minute samples show a trend towards greater dilution, suggesting that mixing of red cells in the circulation at that time was complete but in the blood-replaced group a proportion of red cells rejoined the functional circulation between 25 minutes and 2 hours.

REFERENCES


EFFECT OF HALOTHANE ON TETRAHYMENA PYRIFORMIS

J. F. Nunn, K. L. Dixon and J. R. Moore
Department of Anaesthesia, University of Leeds

The ciliate Tetrahymena pyriformis is a convenient model for study of metabolism at the cellular level. It may be grown in axenic culture in nutrient media and has available a wide range of metabolic pathways including those of the Krebs cycle. Cell division is mitotic, in logarithmic phase, the doubling time is of the order of 3 hours at room temperature.

The organism has, in the past, received little attention as a cell model for study of the effects of anaesthesia, and we are reporting a pilot study carried out to assess its usefulness for investigation of the effects on motility, oxygen consumption and division rate. To date only halothane has been used and this has been vaporized to produce a saturated concentration with two fritted bubblers in series and the effluent gas diluted to produce the required concentrations. Gas flow rates were measured with bubble flowmeters and concentrations were determined from vapour pressure tables.

Although the organism continued to swim at control speed for 10 minutes after exposure to 100% nitrogen, its velocity was rapidly reduced in accord with the concentration of halothane, complete arrest occurring at a concentration of about 4%. Recovery of most organisms was complete within 2 minutes but some Tetrahymena did not recover motility after withdrawal of the stronger concentrations. Confirmation was also obtained of the observation of Seifriz (1941) that anaesthesia could produce reversible interference with protoplasmic streaming in the slime mould Physarum polycephalum. Oxygen consumption was reduced in parallel with motility, being about 30% of control value with 4% halothane. Cell division was also inhibited by halothane and 50% reduction was obtained at a concentration within the range 1-1.5%. Complete arrest of division occurred at about 3% halothane, a concentration which caused arrest of germination of mustard seedlings. Germination was resumed when the halothane was withdrawn.

ACKNOWLEDGEMENTS

K.L.D. and J.R.M. supported by a grant from the Medical Research Council.

We are indebted to Mr. J. B. Alexander for supply of a culture of Tetrahymena pyriformis (Strain S) and to Dr. C. J. P. La Touche for the Physarum polycephalum.

REFERENCES


STUDIES MADE IN CATS OF SOME CARDIOVASCULAR EFFECTS OF A NEW GASEOUS ANAESTHETIC—TEFLURANE

G. W. Black, R. S. J. Clarke, P. J. Howard and H. McCullough

Department of Anaesthetics, The Queen’s University of Belfast, Northern Ireland

Teflurane is a non-explosive gas, one of a series of halogenated hydrocarbons developed by Abbott Laboratories in the United States of America. The present investigation was designed to study changes in arterial pressure and heart rate and rhythm during anaesthesia; also the relationship of catecholamine liberation and acid-base balance to the production of cardiac arrhythmias was examined. The cat was chosen as being suitable for the production of “spontaneous” arrhythmias, although it suffers from the disadvantage of being very labile in terms of sympathetic nervous activity and acid-base state. In order to make the data of more practical interest a comparable study of cyclopropane and halothane was undertaken. Teflurane is approximately equipotent with cyclopropane, concentrations of 25–50% being required for induction and 10–25% for maintenance.

Basal anaesthesia was induced with intraperitoneal chloralose (50 mg/kg) and following intubation the trachea 66% N, in O, was administered via a T-piece system. The femoral artery was then cannulated. Control observations of blood pressure, e.c.g. and heart rate were made and arterial samples were taken for catecholamine and acid-base determination. The anaesthetic to be studied was then given in the chosen concentration for 30 minutes, at the end of which time further observations were made. In most cases the effects of a different anaesthetic were studied following a second control period of 30 minutes.

During the inhalation of teflurane ventricular ectopic contractions frequently occurred when concentrations greater than 10% were inhaled, whereas a concentration of at least 25% cyclopropane was required to initiate cardiac irregularities. Arrhythmias were observed during halothane anaesthesia when the inspired concentration was 2% or more. Like halothane, but unlike cyclopropane, teflurane caused a profound fall in arterial pressure. Even the lower concentrations of teflurane and cyclopropane caused a severe respiratory acidosis while halothane had little effect on Pco2.

In the control state the standard bicarbonate was low, figures ranging from 15 to 21 m.equiv./L. By human standards this is a severe metabolic acidosis but it appears to be a constant finding in cats. All three anaesthetic agents caused a further but slight fall in the level of bicarbonate. Analysis of plasma catecholamine concentrations showed that teflurane and halothane produced substantially smaller increases in circulating noradrenaline than cyclopropane.

In summary, teflurane is a gaseous anaesthetic of similar potency to cyclopropane. It is more prone to produce cardiac irregularities than the older agent but, like it, causes profound respiratory depression. On the other hand, its other cardiovascular effects resemble those of halothane. It causes little increase in plasma noradrenaline levels and even more circulatory depression than halothane.
PHARMACOLOGICAL OBSERVATIONS ON THE INTERACTION BETWEEN MONOAMINE OXIDASE INHIBITORS AND NARCOTIC ANALGESICS IN ANIMALS

K. J. ROGERS AND J. A. THORNTON
Departments of Pharmacology and Anaesthetics, University of Sheffield

It is well known that abnormal responses to therapeutic doses of pethidine, and possibly other potent analgesics, may occur in patients receiving monoamine oxidase (m.a.o.) inhibitors. The present work was undertaken to investigate the possible mechanisms involved in this drug-drug interaction.

In acute toxicity studies the LD50 of morphine, pethidine, phenazocine and pentazocine were determined in normal mice, and in animals pretreated with either iproniazid (500 mg/kg) or tranylcypromine (15 mg/kg). It was found that the two m.a.o. inhibitors increased the toxicity of the analgesic drugs to approximately the same degree (table I), indicating that the more recently introduced narcotic agents, phenazocine and pentazocine, carry the same risk in subjects receiving m.a.o. inhibitors as do pethidine and morphine. It has been suggested that this increase in toxicity may be due to a reduction in the rate of breakdown of the analgesic drug. However, the blood levels of pentazocine in mice pretreated with tranylcypromine (at a time when the toxic symptoms of pentazocine were at their maximum) did not differ significantly from the levels in mice not receiving the m.a.o. inhibitor. Furthermore, examination of the brain and liver m.a.o. activity showed that there was no correlation between the increase in analgesic drug toxicity and the decrease in enzyme activity.

<table>
<thead>
<tr>
<th>Percentage increase in toxicity of analgesic drug</th>
<th>Tranylcypromine pretreated</th>
<th>Iproniazid pretreated</th>
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<tbody>
<tr>
<td>Pethidine</td>
<td>53%</td>
<td>56%</td>
</tr>
<tr>
<td>Morphine</td>
<td>42%</td>
<td>48%</td>
</tr>
<tr>
<td>Phenazocine*</td>
<td>76%</td>
<td>33%</td>
</tr>
<tr>
<td>Pentazocine</td>
<td>44%</td>
<td>45%</td>
</tr>
</tbody>
</table>

*Tranylcypromine (15 mg/kg i.p.) injected 4 hours before the analgesic drug.
Iproniazid (500 mg/kg i.p.) injected 6 hours before the analgesic drug.
* Injection vehicle dimethyl sulphoxide.

Finally, a study was made of the relationship between the change in pethidine toxicity and the levels of noradrenaline, dopamine and 5-hydroxytryptamine in mouse brain. The time course of the changes in the concentration of brain amines and also the amine changes with increasing dosage of tranylcypromine, indicated that the increased pethidine toxicity in mice was related to elevated levels of cerebral 5-hydroxytryptamine.

In summary, the results suggest that the abnormal pharmacological responses produced by potent analgesics in combination with m.a.o. inhibitors are not due to a decelerated metabolism of the analgesic drug, but involve a reaction in the central nervous system which is related to an increased concentration of 5-hydroxytryptamine in the brain.

POSTOPERATIVE LARYNGEAL INCOMPETENCE

P. J. TOMLIN
Department of Anaesthetics, University of Birmingham

In an investigation into the aetiology of postoperative atelectasis patients were given a radiopaque dye to swallow after they had apparently fully recovered from the anaesthetic. There were 56 patients in this series, of whom 53 had had simple non-abdominal surface operations. The neohydriol swallow film was taken 1-3 hours after discontinuing the anaesthetic. Chest radiographs were taken pre-operatively, at the time of the swallow, and on the day after operation. The films were reviewed independently by two radiologists.

The films show that 12 of the 56 patients had radiological evidence of atelectasis and that 6 of these patients had also inhaled some neohydriol.

Statistical evaluation failed to show any single factor of anaesthetic technique or dosage to be responsible for the atelectasis. However, operations, even very simple operations like breast biopsy, around the chest and neck areas had a significantly higher evidence of atelectasis than had operations on more peripheral areas.

It is concluded that following apparently full recovery from an anaesthetic the larynx may be incompetent for some time afterwards, and so patients remain at risk of inhalational atelectasis should any liquid material be in the pharynx. This failure of return of the laryngeal closure reflex after anaesthesia could well be a major factor in the aetiology of postoperative pulmonary complications.

EFFECTS OF PROPRANOLOL IN HYPOTHERMIC DOGS

D. J. F. MACDONALD*
Department of Surgery, Manchester University

Using a gated sine-wave electromagnetic flowmeter, the effect of propranolol on dogs at different temperatures was studied.

Method.

Flow-probes were implanted at the root of the aorta in mongrel dogs which were then allowed to recover. The experiments were carried out 3 to 10 days after thoracotomy.

* Present address: Department of Anaesthetics, Glasgow Royal Infirmary.
Anaesthesia was induced with thiopentone 20 mg/kg and muscle relaxation was achieved with n-allyl nor-torxiferine 0.2 mg/kg with 0.1 mg/kg repeated hourly. The dogs were intubated and ventilated with 0.5% halothane in oxygen by a Starling pump set to give a Pao2 of 40 mm Hg at 37°C. Halothane was discontinued at an oesophageal temperature of 28°C. Propranolol was given at temperatures of 35 to 38°C: two of these were anaesthetized and two conscious, and all four were in a state of respiratory alkalosis and metabolic acidosis comparable to those at low temperatures. All dogs were in sinus rhythm. All measurements were made either immediately before or between 6 and 12 minutes after intravenous administration of propranolol.

Findings.
Heart rate, cardiac output and maximum acceleration (see below) fell 45–50% with temperature and showed a further fall after propranolol. Stroke volume showed no consistent change due to hypothermia or to propranolol, nor did mean arterial pressure.

The fall in heart rate produced by propranolol was smaller at low temperatures than at normal temperatures. In one dog at 33°C the heart rate, initially only 47 beats/min, fell to 42 beats/min, suggesting that the smaller fall in heart rate was due to the lower initial heart rate rather than to temperature per se.

When the fall in heart rate was considered as a percentage change, propranolol produced a significantly greater fall in heart rate at low temperatures than at normothermia (P<0.05).

Cardiac output fell significantly when propranolol was administered at normothermic temperature and also fell significantly with hypothermia. When propranolol was administered at low temperatures a further reduction in cardiac output occurred in every animal except one. Owing to this one exception, the difference in cardiac output with propranolol during hypothermia was not significant.

Maximum acceleration of blood (measured by the tangent of the angle of upstroke of the flow trace) showed a significantly greater depression by propranolol at low temperature than at normal temperature (P<0.02).

The results are summarized in table I.

<table>
<thead>
<tr>
<th>Table I Percentage changes after propranolol.</th>
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<tr>
<td></td>
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<tr>
<td>35–38°C</td>
</tr>
<tr>
<td>Heart rate</td>
</tr>
<tr>
<td>Cardiac output</td>
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<tr>
<td>Maximum acceleration</td>
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</tbody>
</table>

CALIBRATION OF P02 ELECTRODE WITH GLYCEROL

G. H. HULANDS,* J. PANDAY AND G. M. PATerson
Department of Anaesthesia, University of Leeds

It is well established in polarography that a difference exists between the P02 of gas and the indicated P02 of a liquid equilibrated with the same gas: in this study the symbol o is introduced to represent the ratio. This difference is probably due to the development of tension gradients between the bulk of the sample and the tip of the cathode. The persistence of this discrepancy, despite attempts to remove the gradients, has led to difficulty in calibration of the polarograph and from this difficulty three methods of calibration have emerged.

The first uses gas-equilibrated blood. This is theoretically correct, but practical problems militate against its routine use in the clinical laboratory. The second uses air, and then applies a constant correction factor but, in this study, the blood o varied daily and therefore the application of such a constant factor could result in large errors. The third method uses gas-equilibrated water. This is a compromise, as water o lies between unity and that for blood.

As these methods seemed unsatisfactory, the possibilities of using glycerol/water mixtures for calibration of the polarograph were investigated. Preliminary studies along these lines had been carried out by Nunn and Casselle in 1959 (unpublished). Normal human blood and various concentrations of glycerol were equilibrated with gas mixtures containing 8%, 21% or 97% oxygen: P02 values were then determined with a Radiometer electrode.

A comparison was made between 0 values for water, blood and 30% glycerol. When the 0 values for water were plotted against those for blood the points were generally distributed below the line of identity; however, those for 30% glycerol against blood were distributed along this line.

In this study a normal 0 value appeared to be <1.07, a greater value suggesting an unstable system. This can be easily detected by initially calculating 0 from the indicated P02 of air and glycerol equilibrated with air, a rising or high 0 being indicative of a malfunctioning electrode. Following calibration with 30% glycerol, the indicated P02 of a blood sample would also be the true P02 of that sample without the application of any correction factor. This appears to be true over the whole range of P02 we studied, even if the value for 0 should be high.

* Member of the Medical Research Council Scientific Staff.

LACTATE AND PYRUVATE CHANGES IN NEUROSURGICAL OPERATIONS WITH AND WITHOUT HYPOTENSION

A. R. HUNTER
Department of Anaesthetics, University of Manchester

The blood pressure needs to be reduced to some 50 mm Hg for operations on intracranial aneurysms. Such a degree of hypotension might be expected to cause some failure of tissue perfusion. In order to
TABLE I

<table>
<thead>
<tr>
<th>Cases</th>
<th>Excess lactate (m.mole/l.)</th>
<th>L/P ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>After induction</td>
<td>End of operation</td>
</tr>
<tr>
<td>Aneurysms 11</td>
<td>0.1 ± 9.9</td>
<td>22.3 ± 10.6</td>
</tr>
<tr>
<td>Controls 20</td>
<td>-6.9 ± 10.8</td>
<td>21.6 ± 7.2</td>
</tr>
</tbody>
</table>

evaluate this the serum lactate and pyruvate were determined immediately after the induction of anaesthesia and at the conclusion of the operation. The methods of anaesthesia for the aneurysm cases involved nitrous oxide, oxygen and halothane with spontaneous respiration for the induction of hypothermia. Thereafter controlled ventilation with nitrous oxide, oxygen and curarization was employed as detailed elsewhere (Hunter, 1964). This latter technique was employed throughout anaesthesia in the control cases where no hypotension was induced. The excess lactate was calculated according to the formula of Huckabee (1958) and in view of the doubts of others (Harris, Bateman and Gloster, 1962) concerning this calculation the results were also expressed as lactate/pyruvate ratios. Results are shown in table I. They do not indicate any failure of tissue perfusion during hypotension.

REFERENCES


FIRST INTERNATIONAL SYMPOSIUM ON DETECTION OF CANCER

SPA (BELGIUM): SEPTEMBER 26-29, 1968

Chairman Dr. Henri Ramioul, Civil Hospital of Verviers.

The aim of this Symposium is to discuss the different problems of the detection of cancer.

Programme Detection of gynaecological cancer.
Detection of breast cancer.
Detection of digestive cancer.
Detection of pulmonary cancer.

Round tables Psychological and sociological aspects of the detection of cancer.
Economic aspects of the detection of cancer.
Deontological aspects of the detection of cancer.
General results of the detection.

Scientific exhibition. Recreational programme.

Registrations Secrétariat Général, Quai du Barbou, 4, Liège (Belgium).

Further particulars: The Secretary of the Organizing Committee, Doctor Albert Liegeois, Civil Hospital of and in Verviers.