CARDIAC ARREST TEMPERATURE: THE EFFECT OF ETHYL ALCOHOL AND CARBON DIOXIDE ON BABOONS

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SUMMARY

The effect of high inspired carbon dioxide concentrations and i.v. ethyl alcohol on the temperature at which cardiac arrest occurred was studied in chacma baboons. Alcohol-treated baboons developed cardiac arrest at a temperature of 24.3 °C (SD ± 0.85) and control animals at 27 °C (SD ± 1.30). There were no signs of cardiac failure before the occurrence of ventricular fibrillation.

Previous studies (White and Nowell, 1965; Duthie and White, 1977) have supported the view that ethyl alcohol protects the rat heart from the effects of hypothermia, with the result that cardiac arrest occurs at a lower temperature than normal. It has been shown also that high inspired carbon dioxide concentrations augment the protective effect of ethanol on the rat heart, and allow still lower temperatures to be achieved before the occurrence of cardiac arrest (Duthie and White, 1977).

This study was designed to investigate the effect of high blood concentrations of ethanol and high inspired carbon dioxide concentrations on the cardiac arrest temperature of larger mammals. The chacma baboon was selected as the experimental animal. A pilot study on 10 animals suggested the use of an ethanol (absolute alcohol) dosage of 4 ml/kg body weight and an inspired carbon dioxide concentration of 10% in oxygen.

METHODS

Fourteen male chacma baboons were studied. These were all mature adults, well nourished and free from any obvious disease. They were allocated randomly to a test or a control group.

Anaesthesia was induced with ketamine hydrochloride 10 mg/kg body weight i.m., the weight of the animal being estimated by eye. Following loss of consciousness, the baboon was weighed and a cuffed tube was inserted into the trachea following the administration of alcuronium 0.2 mg/kg i.v. The animal was ventilated with oxygen using a Starling Pump at a frequency of 36/min and the tidal volume was adjusted to maintain an end-tidal carbon dioxide concentration of 5%. Anaesthesia was maintained with sodium thiopentone 0.1 mg/kg body weight i.v. administered at 30-min intervals.

Cardiac temperature was measured by a thermocouple probe inserted into the lower third of the oesophagus (Whitby and Dunkin, 1968, 1969, 1971). The probe was connected to an Ellab thermometer with a range of 0–50 °C.

Systemic arterial pressure was measured with a short cannula inserted into the femoral artery and connected to a Statham 23 DP pressure transducer calibrated previously against a mercury manometer. Central venous pressure was measured with a cannula inserted through the axillary vein into the right atrium and connected to a Statham 23 BB pressure transducer. The arterial and venous pressures were displayed on an oscilloscope screen and recorded by a potentiometric recorder. The e.c.g. was recorded by means of needle electrodes attached to the limbs and displayed on the oscilloscope.

Gas was sampled continuously through a 19-gauge needle inserted into the lumen of the endotracheal tube and the carbon dioxide concentration was measured using a Beckman infra-red carbon dioxide analyser (Model LB-1), calibrated previously against known carbon dioxide concentrations.

In the test group of animals, venous blood samples were taken at various intervals and at the moment of cardiac arrest. Blood alcohol concentrations were measured by spectrophotometry. Alcohol infusions were started 30 min after the injection of ketamine and completed 60 min later. The dose of 4 ml/kg body weight was diluted to a 20% solution in normal saline. In the control group, the baboons received an infusion of normal saline, 20 ml/kg body weight over the same period of time. These infusions were made into the femoral vein.
One hour after the induction of anaesthesia, the animal was placed in a bath containing ice-cold water. At the same time in the test group, carbon dioxide was added to the inspired mixture until the end-tidal carbon dioxide concentration was 10%. This concentration was maintained during cooling, which was continued until cardiac arrest occurred. No attempt was made to resuscitate the animals. The ventilation of both the control and the test animals was maintained at the setting which produced an end-tidal carbon dioxide concentration of 5% at 37 °C, this having been defined as "normal" ventilation during hypothermia by Severinghaus (1959).

RESULTS
The infusion of ethanol in combination with a high inspired carbon dioxide concentration resulted in the survival of baboons to lower temperatures than occurred in the control animals (table I). None of the test group developed cardiac arrest at a temperature greater than 25 °C, whereas 29 °C would have been the lowest temperature to which it would have been safe to cool the controls. Ventricular fibrillation was the terminal event in every experiment.

Blood samples were taken immediately after the cessation of the infusion of alcohol and at intervals throughout the experiment. In each instance the blood alcohol concentration remained almost constant, and there was little decrease resulting from metabolism. The concentrations in the samples taken at the moment of death are shown in table II.

A comparison of systemic arterial pressure at 30 °C and immediately before cardiac arrest (table III) shows evidence of only moderate decreases attributable to cooling. However, the pressures were markedly lower in the alcohol treated compared with the control animals.

Central venous pressure remained constant during the experiment and in only one animal was there a significant increase before cardiac arrest (table III).

In the test animals bradycardia was marked whilst in the control group only minor decreases in heart rate were noted.

End-tidal carbon dioxide concentration was maintained at 10% in the test group by the addition of carbon dioxide to the inspired mixture. In the control group which received "normal" ventilation, the concentrations decreased progressively as cooling occurred and the final measurement varied from 2.2 to 3.7%.

There was no significant difference between the rates of cooling from 35 to 30 °C in the two groups (table IV). However, the rate of cooling correlated significantly in each group with the weight of the animals. For the alcohol group $r = +0.7064$ and for the control $r = +0.8330$, $P<0.05$ (1-tailed test). However, the rate of cooling allowing for weight may be different between the two groups. Figure 1 shows that the regression slope for the control group ($b = +0.2704$) is in fact steeper than for the test group ($b = 0.1744$) with these small samples, but this
difference does not attain statistical significance ($t_{10} = 0.8579$).

**Table IV.** Time to cool from 35 to 30 °C in alcohol-treated (test) and untreated (control) baboons

<table>
<thead>
<tr>
<th>Baboon number</th>
<th>Weight (kg)</th>
<th>Time (min)</th>
<th>Baboon number</th>
<th>Weight (kg)</th>
<th>Time (min)</th>
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<tbody>
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<td>15.10</td>
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</table>

**Fig. 1.** Relationship between body weight and rate of cooling.

**DISCUSSION**

Our results have shown that baboons with high blood alcohol concentrations and high inspired carbon dioxide concentrations could be cooled to lower temperatures than untreated animals before the occurrence of ventricular fibrillation.

The highest alcohol concentration measured in venous blood taken immediately following ventricular fibrillation was 131.5 mmol/litre. In man a blood alcohol concentration in excess of 87 mmol/litre indicates a state of advanced intoxication (Goodman and Gillman, 1970a), and Dundee (1970) showed that sleep can occur following the rapid i.v. infusion of a solution of alcohol to produce a venous blood concentration of 43.5 mmol/litre. In our study the impression was not gained that the high blood alcohol concentrations contributed in any way to the death of a baboon. This view is supported by the work of MacGregor, Schonbaum and Bigelow (1964) who found, in the dog, that a concentration of 325.9 mmol/litre was necessary to produce death when ventilation was supported, and that in all instances, cardiac arrest occurred in asystole.

Both alcohol (Goodman and Gillman, 1970b) and hypercapnia (Nunn, 1962) increase skin blood flow, and therefore increase the rate of heat exchange between an animal and its environment. Therefore one might have expected a higher rate of cooling in the alcohol-treated animals than in the controls (Sellick, 1957). This did not occur, possibly as a result of the insulating effect of the animals' fur. However, there does not seem to exist any controlled comparison of the rates of cooling of peripherally dilated and constricted animals, and though apparently illogical, the conclusion drawn from this study is that there is no difference between the two.

The changes in the cardiovascular system are in agreement with those reported by other observers. As expected, there was a progressive decrease in both heart rate and arterial pressure during cooling, but the decreases were more pronounced in the alcohol-treated animals. None of the baboons in the trial received atropine, although previous work (Hegnauer, Schriber and Haterius, 1950; Cookson and di Palma, 1955) indicates that this would not have reversed the bradycardia occurring below 30 °C.

In common with the findings of McMillan and others (1957), no significant increase in central venous pressure was seen in either group of animals, and this suggests that the heart performed efficiently up to the moment of cardiac arrest. The decrease in arterial pressure was probably a result of an increase in vascular capacity resulting from vasodilatation unless there was a loss in circulating blood volume. Swan and others (1953) found a small decrease in blood volume in dogs cooled to 20–25 °C, and Suzuki (1965) observed an increase in the haematocrit during hypothermia. However, these changes were small and should not account for the maintenance of a normal central venous pressure co-existing with a significant degree of cardiac failure.

This work suggests that the use of a high inspired carbon dioxide concentration and i.v. ethyl alcohol may permit the use of lower temperatures during surface cooling than are considered safe at present.
ACKNOWLEDGEMENTS

We wish to thank Dr W. Castle for statistical advice, and Miss F. Ramsay for secretarial help.

REFERENCES


TEMPERATURE AU MOMENT DE L'ARRET CARDIAQUE: EFFET DE L'ALCOOL ETHYLIQUE ET DE L'ACIDE CARBONIQUE SUR LES BABOUINS

RESUME

On a fait une étude sur l'effet des fortes concentrations d'acide carbonique inspiré et des injections intraveineuses d'alcool éthylique sur la température à laquelle se produit l'arrêt cardiaque chez les babouins. Les babouins traités à l'alcool ont subi un arrêt cardiaque à la température de 24,3°C (déviation standard ± 0,85) et les animaux témoins à 27 °C (déviation standard ± 1,30). Il n'y a eu aucun signe d'insuffisance cardiaque avant que se produise une fibrillation ventriculaire.

HERZSTILLSTANDS-TEMPERATUR: DIE WIRKUNG VON ÄTHYLALKOHOL UND KÖHLENSTÖRZÜSD AUF PAVIANE

ZUSAMMENFASSUNG

Die Wirkung von eingestemmten hohen Kohlendioxyc-Konzentrationen und von i.v. verabreichtem Äthylalkohol auf die Temperatur, bei der der Herzstillstand eintritt, wurde bei Chacma-Pavianen studiert. Mit Alkohol behandelte Paviane entwickelten Herzstillstand bei einer Temperatur von 24,3 °C (±0,85), die Kontrolltiere bei 27 °C (±1,30). Vor dem Auftreten eines ventrikulären Flimmerns gab es keine Anzeichen eines Herzversagens.

PARO CARDIACO: EFECTOS DEL ALCOHOL ETILICO Y BIOXIDO DE CARBONO EN EL MANDRIL

SUMARIO

Se ha estudiado el efecto de altas concentraciones inspiradas de dióxido de carbono y de alcohol etílico i.v. sobre la temperatura a la que se produjo paro cardiaco en el mandril chacma. Los mandriles tratados con alcohol desarrollaron paro cardiaco a una temperatura de 24,3 °C (DT ± 0,85) y los animales testigos a 27 °C (DT ± 1,30). No hubo signos de fallo cardiaco antes de producirse fibrilación ventricular.