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The Effect of Alteration of Inspired Oxygen Concentration on Jugular-bulb Oxygen Tension during Deliberate Hypotension

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Jugular-bulb oxygen tension or saturation has been considered a reliable index of the adequacy of cerebral oxygenation.[1-6] Consequently, jugular-bulb oxygen tension (Pv_{O_2}) has been monitored in patients undergoing carotid reconstructive operations.[1-4] Provided adequate arterial oxygenation is maintained, factors influencing Pv_{O_2} include[1-4] cerebral blood flow, cerebral oxygen consumption and the presence of cerebral arteriovenous shunting.[7]

During deliberate hypotension, compensatory mechanisms come into play to maintain cerebral oxygenation.[8-10] Cerebral vasodilatation occurs, and the rate of oxygen extraction is increased. As a result, Pv_{O_2} tends to decrease during deliberate hypotension.[5, 9] However, the lowest Pv_{O_2} measured in one series was 27 mm Hg despite profound hypotension, indicating that cerebrovascular compensation was adequate under the circumstances.[8]

The purpose of this study was to investigate the effect of alteration of the inspired oxygen concentration (100 per cent *vs.* 40 per cent) on Pv_{O_2} during deliberate hypotension.

METHODS

Fifteen patients between the ages of 36 and 71 years, undergoing radical surgical operations on the neck, were chosen for the study. Initially, anesthesia was maintained at a light level, using halothane in oxygen (eight patients) and halothane–60 per cent nitrous oxide in oxygen (seven patients). *d*-Tubocurarine was injected in intermittent doses as required. Respiration was controlled throughout the procedure using an Air-Shields ventilator/ventimeter at rates between 6 and 10/min and tidal volumes between 600 and 900 ml. Airway pressure was allowed to fall to ambient levels between inflations.

After a steady state had been obtained, pentolinium tartrate (Ansolysen) was administered. Tilting allowed the blood pressure to fall gradually to the desired level. The average decrease in systolic pressure, measured with an oscillometer, was 49 per cent. The inspired halothane concentration was then readjusted using a calibrated Fluotec Mark II vaporizer so that a stable level of hypotension and a relatively bloodless field were obtained.

When the internal jugular vein had been exposed, a 22-gauge needle was introduced in the upper part of the vein and directed toward the bulb. Jugular and arterial blood samples were obtained simultaneously in heparinized syringes. The inspired oxygen concentration was then altered from 40 per cent to 100 per cent (seven patients) or from 100 per cent to 40 per cent (eight patients) by discontinuing or adding nitrous oxide, respectively. FI_{O_2} was measured with a Beckman oxygen analyzer. After a ten-minute period, another set of blood samples was obtained. Inspired halothane concentration (0.4 to 0.7

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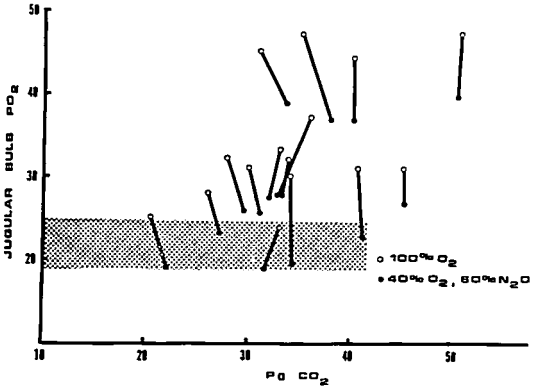

FIG. 1. Relationship of Pa_{CO_2} to Pv_{O_2} during deliberate hypotension. Lines connect observations in the individual patients during administration of 100 per cent oxygen and 60 per cent nitrous oxide in oxygen at the same halothane concentrations (0.4 to 0.7 per cent). An increase in Pv_{O_2} occurred in each patient with the administration of 100 per cent oxygen. Shaded area represents the critical range of Pv_{O_2}.

per cent) and arterial blood pressure were unaltered during the study period. To minimize changes in arterial carbon dioxide tensions and airway pressures, tidal volumes and respiratory rates were kept constant.

Blood gases were analyzed immediately by the following methods: *p*H with the Radiometer glass electrodes; P_{CO_2} with the Severinghaus electrode; P_{O_2} with the Clark electrode. Oxygen contents in venous and arterial blood were calculated from the equation [11]:

$$\text{Oxygen content} = (\text{Hb concentration} \times 1.34) + (Pa_{O_2} \times 0.0031)$$

RESULTS

The average Pv_{O_2} during administration of 100 per cent oxygen was 34.3 mm Hg (range 23.5–47), while the average Pv_{O_2} with 40 per cent oxygen–60 per cent nitrous oxide was 27.8 mm Hg (range 18.5–40). In every patient there was an increase in Pv_{O_2} when 100 per cent oxygen was substituted for 40 per cent oxygen (fig. 1). Pv_{O_2} values tended to be elevated in patients who had high Pa_{CO_2} levels.

The mean increase in calculated arterial oxygen content following administration of 100 per cent oxygen was 1.05 ml/100 ml blood, while the mean increase in calculated jugular venous oxygen content was 2.02 ml /100 ml (table 1). The mean arteriovenous oxygen difference was 6.98 ml with FI_{O_2} 1.0 and 7.95 ml with FI_{O_2} 0.4.

DISCUSSION

Measurement of jugular venous oxygen tension or saturation serves as a sufficient index of overall cerebral oxygenation even though it cannot distinguish between variations in oxygen consumption or alterations in cerebral blood flow.[1-4] On the other hand, Pv_{O_2} may not be indicative of regional or focal oxygenation to certain critically important small areas of the brain.[7] Moreover, Pv_{O_2} may be misleading in the presence of anomalies of cerebral venous drainage and arteriovenous shunting such as might occur about a healing brain infarct.[7] The selection of critical levels by different authors is based on observations that manifestations of cerebral dysfunction may occur at these levels. Since a critical level cannot be outlined with great accuracy at all times, a critical range is preferred.[12]

In our data, Pv_{O_2} values during deliberate hypotension were lower than those reported by Eckenhoff *et al.*, who found that despite a mean brachial arterial pressure as low as 32 mm Hg with the patient tilted head-up, the lowest Pv_{O_2} measured was 27 mm Hg. The difference may be attributed to differences in Pa_{CO_2} levels and depths of halothane anesthesia. However, in their data, much

lower Pv_{O_2} values were found when airway pressures had been increased.[8]

A linear relationship between Pa_{CO_2} and Pv_{O_2} is apparent in our data, which is in agreement with other studies.[1, 2, 4, 5, 8] This indicates that the increase in Pa_{CO_2} continues to exert its effect on the cerebral blood flow during deliberate hypotension with a head-up tilt.[8]

It is evident that the use of 100 per cent oxygen instead of 60 per cent nitrous oxide in oxygen at the same halothane concentration is accompanied by a significantly higher Pv_{O_2}. Some low Pv_{O_2} values, which fell within the critical range during 40 per cent oxygen administration, rose to safe levels when 100 per cent oxygen was given (fig. 1). The increase in jugular-bulb oxygen saturation would be greater than the increase in Pv_{O_2} since most of values lie at the lower part of the oxygen-dissociation curve.

It is unlikely that the increase in the available oxygen to the brain following administration of 100 per cent oxygen is the sole factor responsible for the increase in Pv_{O_2}, since the mean increase in calculated arterial oxygen content is only about half the observed increase in calculated venous oxygen content (table 1). Elimination of nitrous oxide concomitant with 100 per cent oxygen administration seems to contribute to the elevation in Pv_{O_2}. It has been shown in dogs that during light halothane anesthesia administration of nitrous oxide results in an increase in cerebral oxygen consumption.[13] Although such an increase is not of great magnitude under normal circumstances, it may be important during deliberate hypotension. The stimulating effect of nitrous oxide on the cerebral metabolism is less at deeper levels of halothane anesthesia.[13]

Administration of halothane in 100 per cent oxygen during deliberate hypotension seems to be beneficial. Not only does it increase the oxygen available to the brain, it also helps to reduce the cerebral oxygen consumption indirectly by avoiding the use of nitrous oxide. It is unlikely that this high oxygen concentration will have any deleterious effects on the lungs, since its use is limited to a few hours.

The case against induced hypotension with the patient tilted head-up rests mainly on the danger of inadequate cerebral oxygenation.[9] Together with other precautions, such as avoidance of hypocarbia, aiming at a hypotensive level consistent with the patient's general conditions, detection of warning signs

TABLE 1. Calculated Arterial and Venous Oxygen Contents with F_{IO_2} 1.0 and F_{IO_2} 0.4 in 15 Patients during Deliberate Hypotension

	Calculated Arterial Oxygen*		Calculated Venous Oxygen†	
	F_{IO_2} 1.0	F_{IO_2} 0.4	F_{IO_2} 1.0	F_{IO_2} 0.4
Patient 1	16.341	15.468	8.047	3.878
Patient 2	17.866	16.728	10.401	8.913
Patient 3	18.021	16.9	12.454	10.655
Patient 4	16.866	16.156	9.504	7.827
Patient 5	16.919	15.835	9.778	6.807
Patient 6	14.877	12.938	6.638	4.088
Patient 7	14.741	13.856	7.412	4.573
Patient 8	19.323	17.466	10.648	8.576
Patient 9	17.928	16.918	12.875	11.681
Patient 10	16.742	15.71	12.311	10.708
Patient 11	20.701	19.945	15.723	14.063
Patient 12	15.766	14.898	7.886	6.957
Patient 13	14.446	13.793	8.752	6.622
Patient 14	12.939	11.748	4.435	2.580
Patient 15	15.938	15.298	7.737	6.374

* Mean increase in calculated arterial oxygen content = 1.050 ml/100 ml blood.
† Mean increase in calculated venous oxygen content = 2.019 ml/100 ml blood.

indicative of cerebral hypoxia, and avoiding sudden and prolonged hypotension, the use of halothane in 100 per cent oxygen undoubtedly will enhance the safety of the technique.

SUMMARY

The effects of alteration of inspired oxygen concentration on jugular-bulb oxygen tension during deliberate hypotension induced by the combination of pentolinium, halothane and the head-up tilt position were studied in 15 patients. Increasing the oxygen concentration to 100 per cent by eliminating nitrous oxide consistently increased jugular-bulb oxygen tension. Possible mechanisms include increase in oxygen available to the brain and reduction of cerebral oxygen consumption. It is suggested that the administration of 100 per cent oxygen during induced hypotension may contribute to the safety of the technique.

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Intraoperative Doppler Blood Pressure Measurements in Infants

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Intraoperative and postoperative blood pressure monitoring in the neonate and small infant remains a problem. In a previous report we described our experience with use of the Doppler ultrasound technique to monitor blood pressure in adults.[1] Mention was made of the potential usefulness of this method for small infants. The method has been described.[2, 3, 4] This report describes the advantageous use of the Doppler technique to monitor blood pressure in eight infants less than 6 months of age who underwent surgical operations.

During evaluation of the use of the technique for small infants we have used the Parks Model 801 Doppler Ultrasound Flow

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