HAEMODYNAMIC RESPONSES TO INDUCED ARTERIAL HYPOTENSION IN CHILDREN

M. R. SALEM, T. TOYAMA, A. Y. WONG, H. K. JACOBS AND E. J. BENNETT

SUMMARY

Cardiovascular measurements were made in 12 children in whom arterial hypotension was induced with pentolinium and halothane. Propranolol was given to five patients who exhibited tachycardia. Measurements were made at the following stages: before induction of hypotension, 5 min after the administration of pentolinium, when a "dry" operating field was obtained, at the time of maximum hypotension and when arterial pressure had returned to 90% of the control values. The decrease in arterial pressure 5 min after the administration of pentolinium was accompanied by tachycardia, but there was no significant change in cardiac index and the operative field was congested. A substantial decrease in cardiac output occurred when a "dry" field was obtained.

There have been several haemodynamic studies of the state of patients during anaesthesia with induced hypotension (Theye and Tuohy, 1965; Jordan et al., 1971; Scott et al., 1972; Styles, Coleman and Leary, 1973; Wildsmith et al., 1973; Prys-Roberts et al., 1974; Fahmy and Laver, 1976). Some investigators have concluded that a reduction in arterial pressure rather than blood flow is the main factor responsible for a reduction in bleeding (Styles, Coleman and Leary, 1973), while others noted that a dry operative field was dependent on a significant reduction in cardiac output (Didier, Clagett and Theye, 1965).

The present study was designed to measure the cardiovascular state of children during the various stages of hypotension induced with pentolinium and halothane.

It has been proposed that cardiac output may be measured from variations in transthoracic electrical impedance (Kubicik et al., 1966; Baker et al., 1971; Denniston et al., 1976). Measurements obtained by this technique have been shown to correlate well with values obtained with electromagnetic flowmeters (Baker et al., 1971). Recently, the technique has been evaluated in children (Labadidi et al., 1971). The impedance cardiograph enabled us to measure cardiac output in the present study by a non-invasive technique.

METHODS

The investigation was approved by our hospital's Human Experimentation Committee. Twelve patients (aged 1–13 yr) were studied. Children with a reduction in oxygen delivery to the tissues, including anaemia, hypovolaemia, a low cardiac output, right to left shunting and pulmonary disease, were excluded. The proposed surgical procedures included plastic surgery of the head (10 patients) and removal of a haemangioma from the thigh (two patients).

Premedication consisted of morphine 0.18 mg kg⁻¹ and hyoscine 0.15–0.4 mg i.m. 1 h before anaesthesia which was induced with either thiopentone or halothane and maintained with halothane in oxygen. Intubation of the trachea was facilitated by the use of suxamethonium i.v. Ventilation was controlled at a constant tidal volume and rate (between 12 and 22 b.p.m.) throughout the period of hypotension using an Air-Shields Ventimeter/Ventilator (with a paediatric attachment for the smaller children). Four aluminium electrodes were placed circumferentially around the subject's neck and abdomen for measurement of cardiac output with the impedance cardiograph.

When a clinically steady state was achieved and the end-expired halothane concentration was maintained between 0.8% and 1.2%, using a Narcotest-M halothane analyser, control haemodynamic measurements were obtained. Surgery was commenced before hypotension was induced and the operative field was observed continuously by an independent observer. Subsequently, hypotension was induced gradually, with the patient supine, by the i.v. injection of pentolinium tartrate 0.2 mg kg⁻¹. Propranolol in doses of up to 0.05 mg kg⁻¹ was
given to five patients with marked tachycardia. Inspired halothane concentrations were adjusted to produce stable hypotension and a satisfactory operative field. No patient was tilted to a head-up position.

Arterial pressure was measured directly in eight patients, or with a Doppler ultrasonic device (four patients). The duration of hypotension varied between 75 and 153 min (mean 110 min). The standard lead II e.c.g. was displayed continuously. Rectal temperature was maintained between 36.5 and 37.5 °C, using a heated blanket when necessary.

Towards the completion of surgery, arterial pressure was restored gradually to the pre-hypotension value by decreasing the inspired halothane concentration and by administering fluids i.v. Since there was little blood loss (less than 10% of estimated blood volume), no patient received a blood transfusion. Dextrose 5% in lactated Ringer's solution was infused at a rate of 3-5 ml kg⁻¹ h⁻¹. The rate of administration was adjusted to assist in the induction and maintenance of hypotension. Arterial blood-gas analysis was performed repeatedly throughout the study to ensure that \( P_{a} CO_2 \) was maintained between 4 and 5 kPa and \( P_{a} O_2 \) greater than 40 kPa.

Measurements were obtained before induction of hypotension (control), 5 min after the i.v. administration of pentolinium, when the independent observer regarded the operative field as satisfactory, and when the lowest arterial pressure was recorded. Measurements were made in eight patients when the systolic arterial pressure was restored to within 10% of the pre-hypotension value. All measurements were made in duplicate at each stage and the average was calculated. The following were measured or derived: heart rate (HR), systolic arterial pressure (SAP), mean arterial pressure (MAP), stroke volume index (SVI) and cardiac index (CI). The product of systolic arterial pressure and heart rate (SAP × HR) was calculated as an index of the likely changes in myocardial oxygen consumption (\( V_{Mo_2} \)) (Nelson et al., 1974). The data were analysed using the paired Student's \( t \) test.

RESULTS

The mean arterial pressure before the induction of hypotension was 71.08 ± 2.45 mm Hg (mean ± SEM) and the heart rate was 116.17 ± 6.15 beat min⁻¹. The administration of pentolinium resulted in a significant decrease in MAP to 64.52 ± 3.56 mm Hg (\( P<0.01 \)) and a significant increase in HR to 127.83 ± 4.77 beat min⁻¹ (\( P<0.05 \)). This was accompanied by a reduction in SVI from 36.01 ± 4.55 to 29.21 ± 3.58 ml beat⁻¹ m⁻² (\( P<0.05 \)), but no significant change in CI (table I). The operative field was regarded as unsatisfactory (congested) in all patients both at the time of control measurements and at 5 min after the administration of pentolinium.

A satisfactory operative field was noticed in association with either increasing the halothane concentration or the administration of propranolol i.v., or both. At this stage MAP had decreased to 43.71 ± 1.99 mm Hg. This decrease is statistically significant when compared with MAP before or 5 min after pentolinium (\( P<0.001 \)). This was associated with HR 114.92 ± 3.45 beat min⁻¹, which was not significantly different from the control value. When a dry operative

<table>
<thead>
<tr>
<th>Table I. Haemodynamic measurements before and at various stages of induced hypotension. (Values are expressed as means ± SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Control</strong> (before pentolinium)†</td>
</tr>
<tr>
<td>-------------------------------</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
</tr>
<tr>
<td>Heart rate (beat min⁻¹)</td>
</tr>
<tr>
<td>Stroke volume index (ml beat⁻¹ m⁻²)</td>
</tr>
<tr>
<td>Cardiac index (litre m⁻² min⁻¹)</td>
</tr>
<tr>
<td>Product of systolic arterial pressure and heart rate (mm Hg beat min⁻¹)</td>
</tr>
</tbody>
</table>

Student's paired \( t \) test: *** \( P<0.001 \); ** \( P<0.01 \); * \( P<0.05 \).
† The operative field was considered congested in all 12 patients.
‡ Data obtained in eight patients only.
field was obtained both SVI and CI were reduced significantly in comparison with the control values (table I).

There were no marked changes in the haemodynamic variables from the point at which a dry field was noted to the point of maximum hypotension and the operative field remained satisfactory. CI at its lowest value was significantly less than when arterial pressure was at the minimum (P<0.05). This was a result of a change in stroke volume (P<0.05) rather than of heart rate.

Haemodynamic measurements were obtained in eight patients when the arterial systolic pressure had returned to within 90% of control. In the remaining four patients, such measurements could not be obtained with accuracy because of muscle movement. The restoration of arterial pressure was associated with an increase in SVI to control values, but CI remained slightly, though significantly, less than the pre-hypotension value (P<0.05). Figure 1 shows the relationship between MAP and CI expressed as a percentage of control values during hypotension and after the return to normal pressure.

The product of systolic arterial pressure and heart rate was unchanged 5 min after the administration of pentolinium. However, a significant decrease occurred when a dry field was obtained, and at the various stages of hypotension (P<0.001). The restoration of arterial pressure to normal was accompanied by an increase in the SAP–HR product to the control values (fig. 2).

No arrhythmia or e.c.g. changes were observed during hypotension. All patients recovered from anaesthesia and there were no complications.
anastomosis easier, thereby avoiding stretching and tearing of a diseased vessel wall (Salem, Ei-Etr and Rattenborg, 1968; Dalal et al., 1974).

Most previous studies of hypotensive anaesthesia have assumed that the hypotension improved operative conditions. However, it is known that a dry field is not always accomplished with hypotension and bleeding may be excessive in spite of an adequate reduction in arterial pressure (Larson, 1964).

The present study indicates that in children the production of a relatively dry operative field by induced arterial hypotension is accomplished only by a substantial decrease in cardiac output. This is in agreement with earlier work of Didier and his colleagues (1965) who demonstrated that satisfactory operative conditions during trimetaphan hypotension in adults were related more closely to the reduction in cardiac output than to the decrease in arterial pressure. They found in several instances that the technique was successful in producing a “bloodless field” in association with a significant reduction in cardiac output but minimal changes in arterial pressure. When an unsatisfactory surgical field was present, the reduction in cardiac output was less than 20% despite adequate hypotension.

The safety of induced hypotension depends upon maintenance of adequate distribution of blood flow to vital organs, especially the heart and the brain. Utilizing the product of SAP and HR, an index of change in myocardial oxygen consumption, Fahmy and Laver (1976) have shown that the oxygen availability decreased less during hypotensive anaesthesia than did the index of myocardial oxygen consumption. In our study, we found that the decrease in CI was associated also with a decrease in SAP x HR. Although the oxygen content was not measured in our patients, it is unlikely that it had changed significantly during hypotension since changes in haemoglobin concentrations were minimal and $P_{aO_2}$ exceeded 40 kPa in all patients. Therefore, alterations in cardiac index were indicative of changes in oxygen availability. These observations suggest that in spite of substantial reduction in cardiac output and possibly coronary blood flow during hypotension in children, myocardial oxygen consumption was reduced in proportion.

During hypotension, cerebral vasodilatation occurs and cerebral oxygen extraction is increased (Eckenhoff et al., 1963). However, with a reduced cardiac output, it is important to consider other safety features: the avoidance of hypocapnia, early detection of warning signs indicative of cerebral hypoxia, the induction of hypotension gradually and a level of hypotension consistent with the patient’s condition, the use of adequate monitoring and the use of $F_{iO_2}$ in excess of 0.9 (Salem, Kim and Shaker, 1970; Salem, Ivankovic and Shaker, 1971). With meticulous attention to these points, our technique of deliberate hypotension has been used extensively in infants and children without any related sequelae (Salem et al., 1974).

Two basic techniques of producing hypotension are available at present. In one, hypotension is accompanied by a reduction in cardiac output and blood flow at the operative site, or both, which results in a drier field. This is accomplished best by the combination of pentolinium and halothane, and beta-adrenergic blockade when necessary. In the other technique, hypotension is produced without a decrease in cardiac output. This is useful for facilitating vascular surgery, since it is the reduction in vessel tension and not the decrease in blood flow that is desirable. In this instance, a hypotensive drug which does not decrease cardiac output would be indicated (for example sodium nitroprusside).

We feel that pentolinium provides certain advantages for inducing hypotension in children. In contrast with other agents, pentolinium produces a gradual decrease in arterial pressure which may allow time for the vascular bed of vital organs to dilate. Furthermore, stable hypotension is produced and a second dose is seldom necessary. Although pentolinium has a long duration of action, arterial pressure may be increased by rapid i.v. infusions and by discontinuing the administration of halothane.

Children are known to be resistant to many hypotensive drugs (Anderson, 1955), but rarely to pentolinium (Salem et al., 1974). In a recent report, Bennett and others (1974) found that blood loss in patients undergoing insertion of a Harrington rod was greater in those receiving nitroprusside than those receiving pentolinium. In our practice, the use of nitroprusside and trimetaphan has been limited to situations in which minute-to-minute control of pressure is required to facilitate surgery on large vessels.

ACKNOWLEDGEMENTS
We wish to express our thanks to Mr Frank Bernard Hurley, for his technical assistance with the impedance cardiograph, and to Ms Janice LaShea and Ms Cindy Shipyor, for secretarial assistance.

REFERENCES
INDUCED HYPOTENSION IN CHILDREN


RESPUESTAS HEMODINAMICAS ANTE LA HIPOTENSION ARTERIAL INDUCIDA EN NIÑOS

SUMARIO
Se efectuaron mediciones cardiovasculares en 12 niños en los cuales se había inducido hipotensión arterial mediante pentolinio y halotano. Se administró propranolol a cinco pacientes que acusaron taquicardia. Se tomaron mediciones en las siguientes etapas: antes de la inducción de hipotensión, 5 min después de la administración de pentolinio, cuando se obtuvo un campo de operación “seco”, en el momento de hipotensión máxima y cuando la presión arterial había vuelto a un 90% de los valores de control. La disminución en la presión arterial a los 5 min de haberse administrado el pentolinio fue acompañada por taquicardia, pero no se produjo cambio significativo alguno en el índice cardíaco y el campo operativo estaba congestionado. Se produjo una considerable disminución en la capacidad cardíaca al obtenerse un campo “seco”. 