ARTERIAL PRESSURE STUDIES DURING CAROTID ANGIOGRAPHY

G. PHILLIPS, G. M. HITCHINGS AND T. V. CAMPKIN

SUMMARY

In a study of 92 patients undergoing carotid angiography under general anaesthesia, marked changes in arterial pressure were infrequent. Possible reasons for this include fewer poor-risk patients, a lighter level of anaesthesia involving controlled ventilation, and the replacement of uriodone (Diodone) by less toxic contrast media.

There have been relatively few reports concerned with the changes in arterial pressure which may occur during cerebral angiography performed under general anaesthesia, although hypotensive responses, particularly in patients with recent subarachnoid haemorrhage, have been observed by several authors (Brown, 1955; Stark, 1958; Brindle, Gilbert and McGrath, 1965). The hypotension has been attributed variably to the condition of the patient before operation, and the type and volume of contrast medium injected during angiography. In these earlier reports the anaesthetic technique involved spontaneous breathing using nitrous oxide in oxygen plus a volatile agent such as trichloroethylene or halothane. During the last decade, the introduction of controlled hyperventilation for neurosurgical operations has resulted in a similar technique gaining widespread acceptance for neuroradiological procedures, and in particular for cerebral angiography. Samuel, Grange and Hawkins (1968) and Dallas and Moxon (1969) have commented on the good quality of the radiographs obtained when controlled hyperventilation is used. This has been the standard technique of general anaesthesia in this unit for almost 10 years and it has been our impression that marked decreases in arterial pressure during the procedure (whether carotid or vertebral angiography) were uncommon. In view of the earlier reports concerning the frequency of hypotension, the arterial pressure of 100 patients was monitored carefully before, and during, the investigation.

MATERIALS AND METHODS

Ninety-two unselected patients, undergoing carotid angiography by percutaneous cannulation for a variety of neurological conditions, were studied. The distribution is shown in table I, the largest group comprising those patients with recent subarachnoid haemorrhage (24), while the miscellaneous group included such conditions as cerebral atrophy and head injury. In 20 patients, no abnormality was detected. An additional eight patients undergoing vertebral angiography by retrograde femoral catheterization were studied concurrently, but will form the basis of a further series.

As far as possible, a standard anaesthetic technique was used. After premedication with oral diazepam 5–10 mg, anaesthesia was induced with a sleep dose of thiopentone, and suxamethonium 50–100 mg was given to facilitate tracheal intubation. Thereafter, anaesthesia was maintained with nitrous oxide and oxygen 8–10 litre/min. Pancuronium 4–6 mg was given at the first sign of movement. Ventilation was controlled using a Manley ventilator and anaesthesia was supplemented by small incremental doses of thiopentone, or by the addition of trichloroethylene 0.1–0.3%, this latter agent being avoided in patients in whom increased intracranial pressure was suspected.

The contrast medium used was meglumine iothalamate (Conray 280), which was injected in doses from 4 to 12 ml, the total dose received by any one patient ranging from 20 to 110 ml.

In 82 patients, the brachial arterial pressure was recorded using a Recklinghausen oscillotonometer. In the remaining 10 patients the radial arterial pressure was monitored continuously by means of an indwelling cannula (Venflon 19 gauge) inserted immediately after the induction of anaesthesia. The arterial pressure in these latter patients was displayed continuously on a calibrated oscilloscope, and recorded using a Siemens two-channel recorder. Informed consent was obtained from each patient before the procedure.

In those patients in whom the arterial pressure was measured indirectly, the arterial pressure was noted...
on the ward, before the induction of anaesthesia, and when the arterial pressure had stabilized after induction. Subsequent readings were taken before the first injection of contrast medium, and at 1, 2 and 5 min after each injection, if time permitted. A final reading was made after reversal of the effect of pancuronium. The purpose of the continuous arterial pressure monitoring used in 10 patients was to detect any transient changes in pressure which might have been missed otherwise.

The volume of contrast used for each injection and any untoward reactions were noted also.

The pH and blood-gas tensions of arterial blood were measured in 15 cases, the samples being obtained by the radiologist after cannulation of the carotid artery. Mean $P_{a\text{CO}_2}$ was 3.3 kPa (SD 0.5 kPa).* The e.c.g. was monitored continuously throughout the procedure.

RESULTS

Arterial pressure change following induction

The changes in arterial systolic pressure following induction of anaesthesia are shown in table II and figure 1, the pressure following induction being recorded 5–10 min after intubation of the trachea, before the cannulae for angiography were inserted. In approximately one-third of patients, no change from the pre-anaesthetic value occurred. In 14%, an increase in arterial systolic pressure of from 10 to 20 mm Hg occurred, and in 18% there was an increase of 20 mm Hg or more. In four patients, who were all hypertensive before operation, an increase of more than 40 mm Hg was recorded.

A decrease in arterial systolic pressure of 10–20 mm Hg was observed in 12% of cases, while in 22% the arterial systolic pressure decreased by 20 mm Hg or more. The pressure decreased by 40 mm Hg or more in only six patients and all of these had marked arterial hypertension. Five of them were unaffected.

* 1 kPa = 7.5 mm Hg.

---

**TABLE I. Distribution of lesions in patients undergoing carotid angiography**

<table>
<thead>
<tr>
<th>No.</th>
<th>Male</th>
<th>Female</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subarachnoid haemorrhage</td>
<td>24 (26%)</td>
<td>10</td>
<td>14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infarction</td>
<td>11 (12%)</td>
<td>6</td>
<td>5</td>
<td>48</td>
<td>8</td>
</tr>
<tr>
<td>Tumour</td>
<td>16 (17%)</td>
<td>9</td>
<td>7</td>
<td>54</td>
<td>14</td>
</tr>
<tr>
<td>Angioma</td>
<td>2 (2%)</td>
<td>1</td>
<td>1</td>
<td>17.5</td>
<td>2</td>
</tr>
<tr>
<td>Aneurysm (post-op.)</td>
<td>7 (8%)</td>
<td>3</td>
<td>4</td>
<td>45</td>
<td>11</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>12 (13%)</td>
<td>9</td>
<td>3</td>
<td>40</td>
<td>11</td>
</tr>
<tr>
<td>No abnormality</td>
<td>20 (22%)</td>
<td>10</td>
<td>10</td>
<td>36</td>
<td>14</td>
</tr>
<tr>
<td>Totals</td>
<td>92</td>
<td>48</td>
<td>44</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

![FIG. 1. Percentage of patients showing various changes in arterial systolic pressure following induction of anaesthesia.](image)

**TABLE II. Change in arterial systolic pressure following induction of anaesthesia**

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No change</th>
<th>Increase</th>
<th>Decrease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subarachnoid haemorrhage</td>
<td>4</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Infarction</td>
<td>2</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>Tumour</td>
<td>4</td>
<td>6</td>
<td>2</td>
</tr>
<tr>
<td>Angioma</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Aneurysm (post-op.)</td>
<td>3</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>6</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>No abnormality</td>
<td>11</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>31</strong></td>
<td><strong>13</strong></td>
<td><strong>17</strong></td>
</tr>
</tbody>
</table>

* *

---

* 1 kPa = 7.5 mm Hg.
by the hypotension occurring during the investigation, but the remaining patient, who had bled from an anterior communicating aneurysm, died 5 days after angiography.

**Arterial pressure changes following injection of contrast**

Tables III and IV and figures 2 and 3 depict the changes in arterial systolic pressure after the first and subsequent injections of contrast medium. In 54% of patients, no change occurred in the arterial pressure after the first injection of contrast medium. The largest recorded decreases in arterial pressure after the first injection of contrast were 30 mm Hg (two patients).

![FIG. 2. Percentage of patients showing various changes in arterial systolic pressure following the initial injection of contrast medium.](image)

These patients had respectively a large intracerebral tumour and an intracerebral haematoma; both had an increased intracranial pressure. The greatest increase in arterial systolic pressure after the initial injection of contrast medium was 30 mm Hg, which occurred in a patient with hypertension.

Subsequent injections produced a greater variation in the arterial pressure, but in 17% of patients the value remained constant throughout the procedure. An increase or decrease of pressure of 10–20 mm Hg was noted frequently and increases and decreases were of almost equal frequency. In 32% of patients both increases and decreases were recorded after different injections of contrast medium. Changes in pressure of more than 20 mm Hg were infrequent. The largest increase recorded after a subsequent injection was 40 mm Hg, which occurred in a single patient with cortical atrophy and the largest decrease was 40 mm Hg, in a patient undergoing angiography following operation, after clipping of a cerebral aneurysm. Both patients were hypertensive with arterial diastolic pressures of 100–110 mm Hg.

The lowest arterial systolic pressure recorded at any stage in any of our patients was 90 mm Hg.

Examination of the continuous arterial pressure recordings showed the same slight variation in arterial pressure. However, in some patients, there was also a transient increase or decrease in pressure of about 10 mm Hg which occurred 10–15 s after the injection of contrast medium, and which resolved over a similar period. Several of these patients had cross-compression films taken. (This involves the compression...
of one carotid artery while contrast medium is injected on the opposite side.) In one patient, an increase in pressure of 10–15 mm Hg was seen when the carotid artery was compressed, followed by a decrease of about 50 mm Hg over the next 15 s following the injection. The pressure returned to normal over the next 15 s. In two other patients, carotid compression produced an increase in pressure of up to 20 mm Hg over the period of compression, which then subsided when the compression was removed. These changes were not detected in the patients whose arterial pressure was measured indirectly.

The same stability of arterial pressure occurred in the eight patients undergoing vertebral angiography, but the number of patients was not large enough to enable assessment of the significance of this observation.

One patient developed an urticarial rash immediately after the first injection of contrast medium. This was treated with chlorpheniramine (Piriton) and hydrocortisone, and the investigation continued although the quantity of contrast medium used was reduced to a minimum. At no time did this patient become hypotensive.

**DISCUSSION**

Compared with earlier reports, marked changes in arterial pressure were relatively uncommon in this investigation. Brown (1955) studied changes during angiography in 100 patients in whom uriodone (Diodone) was used as the contrast medium, and general anaesthesia using spontaneous respiration with nitrous oxide, oxygen and trichloroethylene was employed. In more than 50% of patients who had sustained a recent subarachnoid haemorrhage, a decrease in arterial systolic pressure of 20–100 mm Hg followed the injection of contrast medium. Recovery was usually rapid after decreases of 20–40 mm Hg, but severe hypotension persisted sometimes for several hours. Brown attributed these hypotensive responses to the irritant effect of uriodone on the blood vessels in the vicinity of a recently ruptured aneurysm.

Stark (1958), using an essentially similar technique, with a mixture of meglumine diatrizoate and sodium diatrizoate (Urografin) as the contrast medium, reported decreases in arterial pressure of more than 20 mm Hg following the induction of anaesthesia in more than 40% of patients with a recent subarachnoid haemorrhage. Moreover, the injection of contrast medium caused a similar decrease in 65% of these patients. He observed also the hypertensive response, seen in the present investigation, which occurs sometimes when one carotid artery is compressed and the other is injected with contrast medium. Both Brown and Stark found that hypotension was much less frequent in patients suffering from other neurological conditions.

Brindle, Gilbert and McGrath (1965) used an anaesthetic technique involving spontaneous respiration, but substituted halothane for trichloroethylene. They found that the arterial systolic pressure decreased by more than 20 mm Hg in 50% of all patients studied, irrespective of the neurological lesion. They did not specify whether these hypotensive responses followed the induction of anaesthesia or the injection of contrast, which in this series was sodium diatrizoate (Hypaque).

Lewis and Moore (1968) reported that the arterial pressure decreased during anaesthesia by an average of 35 mm Hg. These authors also used spontaneous respiration with nitrous oxide, oxygen and halothane,
and the contrast medium in this series was Urografin. They were sufficiently impressed by the hypotension which occurred in patients with subarachonid haemorrhage to recommend that local analgesia be used in such cases.

In the present investigation, variations in arterial pressure of more than 20 mm Hg were infrequent, possibly several factors being responsible for the difference between our findings and those of previous authors.

First, all reports suggest that patients suffering from subarachoid haemorrhage are particularly liable to develop arterial hypotension during angiography under general anaesthesia. These patients may be in poor general health, with a depressed level of consciousness as a result of intracranial vascular spasm, which, combined with enforced recumbency and possible hypovolaemia because of inadequate hydration, will predispose to hypotension following the induction of anaesthesia. The increase in neuro-surgical services over the last two decades, and the general recognition that subarachnoid haemorrhage is primarily a surgical condition, has led to earlier referral of such patients to an appropriate unit. It is possible, therefore, that fewer poor-risk patients were studied in the present investigation, compared with previous reports.

Second, the advent of controlled hyperventilation during angiography implies the use of minimal premedicant drugs and depressant narcotic agents, and hence a lighter level of anaesthesia. The choice of pancuronium as the muscle relaxant also avoids the occasional hypotension seen when tubocurarine is used. Pancuronium has been routinely used in this unit for a number of years, when it is desirable to maintain a normal arterial pressure (Campkin and Turner, 1972).

Finally, the replacement of uriodone by less toxic contrast media has reduced the incidence of hypotension from this cause. Although sensitivity to meglumine iothalamate may occur, it is uncommon, and in the one patient in this series, who developed an urticarial response following the first injection of contrast, no hypotension was seen.

REFERENCES

ETUDES DE LA TENSION ARTERIELLE AU COURS D'UNE ANGIOGRAPHIE DE LA CAROTIDE

RESUME
Dans une étude portant sur 92 malades subissant une angiographie de la carotide sous anesthésie générale, les variations de la tension artérielle ont été peu fréquentes. Les raisons possibles de cet état de choses comprennent: moins de malades susceptibles, un niveau plus faible d'anesthésie mettant en cause la ventilation contrôlée et le remplacement de l'uriodone (Diodone) par une substance de contraste moins toxique.

ARTERIELLE BLUTDRUCKUNTERSUCHUNGEN WAHREND EINER VASOGRAPHIE DER KAROTIS

ZUSAMMENFASSUNG
In einer Untersuchung von 92 Patienten, die sich unter Vollnarkose einer Vasographie der Karotis unterzogen, traten nur selten ausgeprägte arterielle Druckveränderungen auf. Als denkbare Ursachen dafür gelten unter anderem das Vorkommen weniger, unter hohem Risiko stehenden Patienten, eines leichten Narkoseniveaus, und der Ersatz von Uriodon (Diodon) durch weniger toxische Kontrastmittel.

ESTUDIOS DE PRESION ARTERIAL DURANTE ANGIOGRAFIA CAROTIDA

SUMARIO
En los pacientes sometidos a angiografia carotida bajo anestesia general, fueron infrecuentes los cambios señalados en la presión arterial, según un estudio de 92 sujetos. Entre las posibles razones de ello figuran menos pacientes de bajo riesgo, un nivel más ligero de anestesia implicando control de la ventilación, y la substitución de Uriodone (Diodona) por medios radiopacos de contraste de menor toxicidad.