CIRCULATORY CHANGES DURING ANESTHESIA FOR NEUROSURGICAL OPERATIONS * †

WILLIAM S. HOWLAND, M.D., AND E. M. PAPPER, M.D.

New York, New York

Received for publication November 6, 1951

Undesirable cardiocirculatory manifestations during anesthesia for neurosurgical operations are not unusual. They are frequently ascribed to compression of the vital medullary centers, increased intracranial pressure or hemorrhage. There is no doubt that these are the most serious and most obvious causes of circulatory disturbances observed in this field of surgery. However, there are other causes of disorders of the cardiovascular system which are associated with anesthetic agents and methods or the result of surgical manipulation within the cranium. This group of complications may, in many instances, respond favorably to treatment. This report is concerned with these less frequently observed alterations in the circulation. It is based upon the anesthetic experiences gained at the Neurological Institute of the Columbia-Presbyterian Medical Center and the Psychiatric Institute of the State of New York.

Methods

A total of 1,180 patients was anesthetized in one year at the two institutions. Eighty of these were studied in connection with the psychosurgical project of Pool and his associates (1). For purposes of uniformity with respect to anesthesia and the avoidance of the hazard of explosion in the presence of the electrocautery, all patients were anesthetized with thiopental sodium in small doses and nitrous oxide-

* From the Department of Anesthesiology of the College of Physicians and Surgeons, Columbia University, and the Anesthesia Service of the Presbyterian Hospital, New York, New York.

† Presented at the Annual Meeting of the American Society of Anesthesiologists, Inc., Washington, D. C., November 6, 1951.

‡ Supported in part by The Wrightsman Fund for Research in Anesthesiology at the Presbyterian Hospital.
oxygen through an endotracheal tube. The concentration of oxygen in the inhaled atmosphere was never less than 25 per cent. Endotracheal intubation was performed during topical anesthesia with 2 per cent pontocaine hyaluronidase solution according to a technic described previously (2).

Intravascular pressures in the femoral, or carotid arteries, or in both, and in a peripheral vein were measured in the special group of 80 patients with Lilly capacitance manometers (3). In the remainder, arterial pressure was determined by auscultation. A crude index of respiratory function was obtained with a thoracic pneumograph. In some cases electrocardiograms were made.

RESULTS

There is no attempt to imply that the circulatory disturbances to be described occurred with regularity. These changes are examples of the events which have been observed together with suggested interpretations when the data permit such generalizations.

Fig. 1. The upper tracing is the femoral arterial pressure. The lower tracing was taken from a thoracic pneumograph. Thiopental (pentothal) reduced the peripheral resistance as measured by the height of the dicrotic notch with deep anesthesia.
Circulatory Changes During Anesthesia

The blood pressure was reduced by elevating the head and upper torso during thiopental, nitrous oxide-oxygen anesthesia. The hypotension was corrected with ephedrine.

The Influence of Thiopental Upon Arterial Pressure.—It is well known that the anesthetized patient does not respond to the stress of postural change, hemorrhage or trauma in the same manner as the unanesthetized one. It has been suggested that important factors in the diminution of vasomotor compensation during anesthesia may be the loss of skeletal muscular tone and depression of the carotid and aortic reflexes (4). There is a subsequent decrease of peripheral resistance resulting in vasodilatation (3). The height of the dicrotic notch in the pulse pressure contours observed during direct intra-arterial recordings has been accepted as an index of peripheral resistance in the arterial bed (5, 6). It is obvious, however, that the height of the dicrotic notch is, at best, only a semiquantitative measurement of total peripheral resistance if the volume of perfusing blood and other variables in the intact subject are unknown (5).

The effect of thiopental in doses large enough to produce apnea on peripheral vascular resistance in the femoral artery as measured in this manner is depicted in figure 1. The decrease in the height of the dicrotic notch over a period of six minutes is readily apparent. This effect was not observed during nitrous oxide-oxygen anesthesia reinforced with fractional doses of meperidine hydrochloride.

The Effect of Postural Change During Thiopental, Nitrous Oxide-
**Oxygen Anesthesia.**—It is frequently necessary to elevate the head and upper torso during craniotomy. This maneuver may result in hypotension without significant alteration in heart rate. The incidence of circulatory depression under these circumstances is greater in hypertensive than in normotensive patients. Such depression is observed in figure 2. The failure to maintain a stable arterial pressure in the presence of postural change appears to be associated with the inability to develop compensatory vasoconstriction during thiopental anesthesia.

![Graphs showing blood pressure readings](image)

*Fig. 3.* Transient hypertension and increased peripheral venous pressure were followed by hypotension and irregular pulse beats during "bucking" on an endotracheal tube.

Correction of this complication was accomplished with the intravenous administration of ephedrine. The dosage required was larger than that usually employed with success.

**The Role of Intratracheal Stimulation.**—Most intracranial procedures are facilitated in many ways by the use of an endotracheal airway. This practice has been employed in all the patients anesthetized for craniotomy. However, the stimulation of the tracheal mucosa during thiopental nitrous oxide-oxygen anesthesia with inadequate topical anesthesia has not been completely innocuous. The resultant reaction on the tube consists of bouts of forced expiration which may be severe.
enough to be accompanied by cyanosis. In figure 3 the effects of this phenomenon upon carotid, femoral and peripheral venous pressures are observed. There is some irregularity of the arterial pulse, a transient hypertension followed by hypotension and a narrowing of the pulse pressure. This is accompanied by episodes of transient increase in venous pressure. This situation produces an increase in intracranial pressure which may be significantly dangerous in the presence of an expanding intracranial lesion (7).

These deleterious effects are not confined to cerebral function, but may be seen in the coronary circulation as well. At least one death with acute myocardial infarction has been observed and attributed to the sequelae of "bucking" on an endotracheal tube. It is possible that damage of coronary arteries may occur following the strain of violent cough (8).

![Graph showing reflex bradycardia and hypotension with breath-holding during stimulation of the trachea at or near the carina.]

An unhappy by-product of "bucking" with the type of anesthesia employed in this group of patients results from the injudicious injection of large quantities of thiopental directed toward quieting the patient. Peripheral circulatory collapse may occur. Fortunately, this complication can be corrected with ephedrine intravenously. It is possible that curarization may be safer than the employment of unusually large doses of thiopental. However, it has become increasingly clear that adequate topical anesthesia is an efficient prophylactic measure. In the occasional failure to produce good surface anesthesia, small doses of morphine or meperidine intravenously are partially effective in overcoming the "bucking" phenomenon.

**Inadequate Respiration and the Circulation.**—One of the most dis-
tressing technical problems encountered in neurosurgery is that of severe venous oozing from the operative site which follows the elevation of venous pressure that is associated with inadequate respiratory exchange. The venous oozing may be so severe that hemostasis, even with the electrocautery, is not effective. Anoxia, cerebral venous hemorrhage and difficult operating conditions are the consequences of improper pulmonary ventilation (9, 10).

The two most common causes of inadequate respiratory exchange are obstruction of the airway and insufficient mechanical ventilation. The former is obvious. The latter may follow improper support of the patient in the prone position during operations on the spinal cord or posterior fossa. The error is the failure to provide sufficient space for expansion of the lower chest and upper portion of the abdomen.
to permit free breathing. The net result is enforced elevation of the weight of the upper part of the body with each breath since this is the only area of free movement during respiration. In most instances the clinical picture of inefficient respiration develops slowly over a period of several hours and is manifested by tachypnea, tachycardia and hypertension. In addition, the active thrusting motion of the upper half of the body with each respiratory effort is transmitted to the brain where movement makes operation difficult. Proper support and elevation of the sides of the chest avoid these difficulties.

Figure 6. A variety of pulse pressure contours, electrocardiographic and respiratory changes was associated with surgical manipulation of several cranial structures.

**Reflex Circulatory Changes.**—There was a group of circulatory disturbances characterized by alterations of heart rate or rhythm and a decrease in arterial pressure. The causative factors differed, but it is possible that there was a common underlying mechanism. It cannot be stated with certainty what that mechanism is, but the hemodynamic manifestations resemble those of vagal stimulation.

Figure 4 illustrates an effect that was commonly seen with suction in the trachea near or at the carina. There was a slowing of the heart rate together with some irregularity of beat, and significant transient hypotension. Although the pneumographic tracing indicated clearly the absence of adequate ventilation, the pulse pressure change was so
rapid (two seconds) that it must be ascribed to a reflex effect rather than anoxia.

The oculocardiac reflex is well known. Its afferent arm is generally presumed to be the trigeminal nerve and its outflow is over the vagus (11). Figure 5 represents two such reflex responses, the first with cardiac slowing and the second with respiratory arrest superimposed on the vascular response. This reflex is included for two reasons. In the first place, many neurosurgical patients are placed in the prone position on a head rest which may press upon the eyes and produce hypotension. Several such cases have been encountered. The blood pres-

![Graph](image_url)

**Fig. 7.** Bradycardia and apnea were produced by electrical stimulation of the uncen gyrus.

sure returned to previous levels when the ocular pressure was relieved. Secondly, the respiratory and cardiovascular responses exhibited are similar to those that occurred during surgical manipulations in the cranial area.

An interesting series of cardiovascular disorders was observed during manipulation or stimulation in various parts of the cranium and its contents. Some of these are demonstrated in figure 6. They consist of lengthening the duration of diastole with little change in the duration of systole, nodal rhythm, bigeminy and ventricular premature contractions. These reflexes are similar to the traction reflexes originating
during abdominal manipulation. The sites of stimulation evoking the reflex responses were the galea, pericranium, dura, cortical veins and the temporal and frontal lobes. Stimulation was usually mechanical, manipulation or retraction, although occasionally the electrocautery initiated the changes in circulatory activity.

On two occasions the manipulation of the dura resulted in cardiac arrest. The first patient was a 16 year old girl with dienecephalosis who suffered cardiac arrest during the suturing of the dura at the end of a bilateral topectomy. The second was a 43 year old woman whose heart stopped beating as the bone flap was being elevated and traction was exerted on an adherent dura. Both patients were resuscitated with complete success by immediate cardiac massage. The second patient was operated on uneventfully one week later with no unusual vascular changes during the same thiopental, nitrous oxide-oxygen anesthesia. The absence of cardiocirculatory effect was demonstrated by continuous electrocardiographic and intra-arterial pressure recording.

The last type of reflex change is a special consideration resulting from stimulation of the uncal gyrus of the temporal lobe with a current of known voltage, frequency and pulse width. This observation was made on one of the patients studied by Pool and his associates at the
Psychiatric Institute of the State of New York (1). Figure 7 shows the response obtained with suitable uncal stimulation. It consisted of bradycardia and respiratory arrest of fifty-five seconds’ duration after cessation of the stimulus. However, a special current is unnecessary as the same type of response has been noted in patients having temporal lobotomy with the ordinary electrocautery unit.

Of the changes caused by surgical intervention, only two are sufficiently different in their effect on the cardiovascular system to require separate comment. The first of these occurs with a sudden acute increase in intracranial pressure following ventriculography. The effect is not immediate and takes about an hour to develop. The delay is due to the gradual accumulation of edema of the brain and parenchymal shifts after the relatively incompressible cerebral spinal fluid is removed and air substituted for it. The sharp rise in pressure in the cerebral ventricles apparently causes a sudden increase in both systolic and diastolic blood pressures which may or may not be accompanied by tachycardia. This response and its immediate relief by ventricular drainage are shown in figure 8.

The second type of change in blood pressure is usually gradual in onset and again is related to increased intracranial pressure. Severe hypertension may occur after the cranium has been opened if there is obstruction to the flow of cerebrospinal fluid. Usually the obstruction is the result of the excessive use of cotton pledgets in an effort to control bleeding or during a difficult exposure of a tumor. The removal of the pledgets will allow the intracranial and arterial pressures to return to normal.

**Summary**

Certain cardiocirculatory disturbances were observed during thiopental, nitrous oxide-oxygen anesthesia for 1,180 neurosurgical operations with the aid of direct intravascular-pressure and electrocardiographic recording in 80 patients. These disturbances consist of:

- Decreased peripheral resistance in the arterial tree with deep thiopental anesthesia.
- Decrease in arterial pressure after change in posture in the anesthetized patient.
- Hypertension, tachycardia and tachypnea in the prone position if ventilation is inadequate.
- Diminution in pulse pressure, hypotension and the production of irregularities of heart rate and rhythm following stimulation of the carina and surgical manipulation of various pericranial and intracranial structures.
- Severe hypertension resulting from increases of intracranial pressure.
REFERENCES

THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.
THIRD ANNUAL REFRESHER COURSE PROGRAM

SPONSORED BY COMMITTEE ON MEDICAL SCHOOLS AND
POSTGRADUATE EDUCATION

The Bellevue-Stratford, Philadelphia, Pennsylvania

NOVEMBER 11-14, 1952

LIST OF LECTURES AND LECTURERS

<table>
<thead>
<tr>
<th>Course No.</th>
<th>Title</th>
<th>Instructor</th>
</tr>
</thead>
<tbody>
<tr>
<td>W11-W41</td>
<td>Intravenous Pentothal Anes.: Technique, Complications and Treatment</td>
<td>R. Charles Adams, M.D.</td>
</tr>
<tr>
<td>W37-W67</td>
<td>Vasopressor Agents</td>
<td>John Adriani, M.D.</td>
</tr>
<tr>
<td>W19-T39</td>
<td>Anesthesia for Burn Cases</td>
<td>Charles R. Allen, M.D.</td>
</tr>
<tr>
<td>F36</td>
<td>Ether—Techniques, Contraindications, Complications and Rx</td>
<td>Virginia Apgar, M.D.</td>
</tr>
<tr>
<td>F23</td>
<td>Pentothal-Curevue Mixtures</td>
<td>R. M. S. Barrett, M.D.</td>
</tr>
<tr>
<td>T13</td>
<td>The Chemical Relief of Pain</td>
<td>Henry K. Beecher, M.D.</td>
</tr>
<tr>
<td>F18</td>
<td>Trauma and Preparation for Anesthesia</td>
<td>Henry K. Beecher, M.D.</td>
</tr>
<tr>
<td>T29-F28</td>
<td>Airways, Topical Anes., Intubation, Tracheotomy</td>
<td>Harold F. Bishop, M.D.</td>
</tr>
<tr>
<td>W15-W45</td>
<td>Clinical Data on Dextran and Similar Substances</td>
<td>Walter Bloom, M.D.</td>
</tr>
<tr>
<td>F31</td>
<td>Brachial Plexus Block</td>
<td>John J. Bonica, M.D.</td>
</tr>
<tr>
<td>F63</td>
<td>Paravertebral Somatic Block</td>
<td>John J. Bonica, M.D.</td>
</tr>
<tr>
<td>T27</td>
<td>Oxygen Therapy</td>
<td>Bernard D. Briggs, M.D.</td>
</tr>
<tr>
<td>T25</td>
<td>Acetone in Thoracic Surgery</td>
<td>John P. Bunker, M.D.</td>
</tr>
<tr>
<td>T18-F46</td>
<td>Continuous Caudal and Peridural Anesthesia</td>
<td>John G. Cleland, M.D.</td>
</tr>
</tbody>
</table>

(Continued on page 360)