Anesthesia for Basilar Arterial Aneurysm with Elective Circulatory Arrest and Moderate Hypothermia

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Aneurysms of the basilar artery comprise 3 per cent of all intracranial aneurysms.[1] Surgical management of patients with basilar arterial aneurysms has always been hazardous because of the difficulty in approach and the proximity of the aneurysm to vital structures. Skultety and Nishioka,[2] in 1966, reported that a majority of neurosurgeons regarded aneurysms of the basilar and posterior cerebral arteries as unapproachable; of 11 patients who were treated surgically, eight died and two were left with neurologic deficits. The literature is replete with discouraging results, with mortality and morbidity rates of more than 80 per cent.[3-7] There has been some improvement in surgical technique in the past five years,[8-10] including development of techniques to reduce temporarily the blood flow to the brain. In the initial report from our institution, after using elective circulatory arrest in five patients,[11] it was concluded that the method was worthy of further trial. We have now treated 13 patients with basilar arterial aneurysms over a period of six years. The purpose of this paper is to describe our technique and the modifications made since our initial communication,[11] and to compare our approach with those of others.

METHOD

Forty-five minutes before being brought to the induction room, each patient was medicated with secobarbital, 100 mg, and scopolamine, 0.6 mg, given intramuscularly. The patient was placed supine on a thermal blanket. Leads for the electrocardiographic monitor and a blood pressure cuff were applied. General anesthesia was then induced with a sleep dose of sodium thiopental (250–375 mg, intravenously), followed by *d*-tubocurarine, 0.3 mg/kg body weight. The patient was hyperventilated with a mask using 100 per cent oxygen with halothane, 0.5 to 1.0 per cent. Fifteen to 20 minutes after administration of *d*-tubocurarine, the trachea was incubated with a wide-bore latex-armored tube following lidocaine spray (4 per cent, 120 mg). This waiting period provided adequate anesthesia and relaxation and prevented a rise in blood pressure associated with tracheal intubation. Following inflation of the endotracheal tube cuff to obtain an airtight fit, patients were ventilated mechanically to maintain Pa_{CO_2} at 25–30 torr using 40 per cent oxygen in nitrous oxide with halothane (0.5 to 1.0 per cent). An esophageal stethoscope and an esophageal thermistor were introduced to monitor heart sounds and core temperature, respectively. A second intravenous route was established and a radial artery was cannulated (having previously determined the adequacy of circulation) for measurement of intra-arterial blood pressure and blood gases using the Astrup microequipment, type AME 1b, Radiometer, Copenhagen. During blood-gas analysis, corrections for body temperature were made using the nomogram of Kelman and Nunn.[12]

Usually, by this time, the body temperature approached 36 C. Active cooling was commenced by wrapping the patient in two thermal blankets. Temperature of the cooling bath was maintained at 2 C. Ice bags were placed over areas where major vessels are in proximity to the surface. Chlorpromazine was then given intravenously, 2.5 mg every 15 minutes, until the temperature dropped to 32 C. The average total dose of chlorpromazine used was 10 mg. On the average, it took one and a half hours from induction of anesthesia for the core temperature to reach

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32 C. At this point the patient was taken to the operating room. The ice bags were removed, and the upper of the two blankets was lowered to a level below the umbilicus. When the esophageal temperature reached 31 C, active cooling was discontinued, following which the temperature drifted down to 28–29 C. It is important that active cooling be discontinued at 31 C. If it is allowed to continue to 28 C, the core temperature will stabilize at 24–25 C, predisposing to spontaneous ventricular arrhythmia or fibrillation.

The head, neck and chest were prepared in continuity and draped. Through a temporal craniotomy on the nondominant side, a small anterior temporal lobectomy was done to avoid retraction under the temporal lobe. The incisura was then exposed and the operating dissecting microscope introduced. Osmotic diuretics were not used, to avoid traction on the aneurysm from a shrinking brain. At this stage, the thoracic surgeon joined the team. A median sternotomy and pericardiotomy were done if the patient had a right temporal craniotomy or an anterior thoracotomy and pericardiotomy if a left temporal craniotomy was performed. The neurosurgeon then proceeded to explore the aneurysm.

When obliteration of the aneurysm was imminent, nitrous oxide and halothane was discontinued and an additional dose of *d*-tubocurarine (0.2 mg/kg body weight) was administered. Body temperature was 28–29 C at this stage. Ventricular fibrillation was instituted by applying a 6–8-volt RMS, 60-Hz alternating current directly to the heart. Mechanical ventilation of the lungs was stopped; the lungs were kept inflated with oxygen at a pressure of 10 torr. If obliteration of the aneurysm was not completed within 4 minutes of circulatory arrest, the intracranial procedure was stopped and the heart was massaged manually for 1–2 minutes. Arterial pressure of 60 torr was usually obtained during the manual massage. Following this, short periods of circulatory arrest (2–3 minutes) alternating with manual massage (1–2 minutes) were repeated if necessary.

Once the aneurysm was obliterated, an initial dose of 40 mEq sodium bicarbonate was given intravenously. Manual hyperinflation of the lungs with oxygen cleared atelectatic patches, while the surgeon massaged the heart until myocardial tone returned. Conversion to normal sinus rhythm was then effected with a single 30-watt seconds direct-current shock applied directly to the heart. There was no difficulty in instituting adequate circulation. Ventricular extrasystoles occurred rarely, and brief atrial fibrillation occasionally, before sinus rhythm became established. Blood-gas analysis was repeated and further appropriate correction for metabolic acidosis was made if necessary.

Active warming was then started using a second unit with a bath temperature maintained at 41 C. The use of two units for cooling and warming saved valuable time. After closure of the head and chest wounds, the core temperature was usually between 32 and 33 C. At this stage, nitrous oxide and halothane were discontinued. Atropine, 1.0 mg, and prostigmine, 2.5 mg, were given intravenously. Mechanical ventilation was discontinued and the endotracheal tube was removed when spontaneous ventilation became established. Early in the series, tracheostomies were done, but we found that with careful surgical dissection in the area of the brain stem, postoperative respiratory depression was avoided and patients breathed adequately at a temperature of 32–33 C. We now consider tracheostomy unnecessary. Following extubation, the patient was taken to the recovery room, where oxygen was administered through nasal catheters. Gradual warming was maintained, with the patient kept covered with warm blankets. In no instance was shivering encountered at this stage. Mediastinal drains were removed after 24 hours and ambulation begun within two to four days.

Results

The results of operation in all patients operated upon since 1967 are summarized in table 1. There were two deaths early in the series. One was related to dissection of the dominant temporal lobe and the other to inadvertent tearing of the vein of Labbe. The reasons for these surgical complications were recognized. With appropriate changes in sur-

TABLE 1. Blood Pressure and Heart Rate during Various Stages of Surgery, Total Duration of Cardiac Arrest and Manual Massage, and Metabolic Status following Defibrillation of the Heart

Patient	Prior to Induction of Anesthesia		Before Circulatory Arrest		After Circulatory Arrest		Time in Minutes Arrest/Massage	Number of Interruptions for Massage	Base Excess after Arrest following 40 mEq Sodium Bicarbonate
	Blood Pressure (torr)	Heart Rate (/min)	Blood Pressure (torr)	Heart Rate (/min)	Blood Pressure (torr)	Heart Rate (/min)			
1	110/70	70	95/60	60	95/60	60	12/7	3	− 1.0
2	125/80	80	110/75	70	110/80	70	2.5/0	0	+ 1.0
3	120/80	70	70/50	50	75/55	55	2.2/0	0	+ 2.5
4	140/90	80	85/60	65	85/60	60	3.7/0	0	− 0.7
5	130/80	80	100/65	75	90/60	70	28/12	8	− 6.0
6	120/75	80	110/80	70	110/80	70	9/4	2	− 3.0
7	100/70	80	80/60	65	75/50	70	6/2	2	− 1.0
8	110/70	80	85/60	55	80/55	60	12/5	3	− 4.4
9	140/80	75	100/60	50	110/60	55	23/8	7	− 5.0
10	105/70	70	80/60	60	80/60	65	6/2	1	+ 2.0
11	115/70	90	90/70	70	90/70	80	1/0	0	+ 4.0
12	160/90	82	110/70	55	105/70	55	1/0	0	+ 1.5
13	110/80	74	85/55	55	85/55	55	14/7	4	− 3.5
14	140/70	76	105/70	60	105/65	64	9/4	2	− 2.5

gical technique there was no further mortality. Three patients had minimal paresis related to surgery. These three and all other patients made satisfactory recoveries and were able to return to normal life. In one of these patients, a clip on the aneurysm fractured and slipped 32 days after the first operation. The patient had to have a second operation; hence, a total of 14 operations. In this series there was no morbidity or mortality related to the anesthetic procedure. During the interval of this study, four additional patients with basilar arterial aneurysms were admitted in poor condition. They had repeated subarachnoid hemorrhage and died while under observation.

DISCUSSION

Elective circulatory arrest and hypothermia are not new concepts. The question is how best they can be achieved, as several techniques are available.

The open-chest methods used previously include profound hypothermia (15 C) and circulatory arrest,[13] moderate hypothermia (30 C) and clamping of the superior and inferior vena cava and the aorta,[13] and moderate hypothermia (30 C) with occlusion of the superior and inferior vena cava and the innominate, left common carotid, and left subclavian arteries.[14]

The closed-chest methods that have been used include moderate hypothermia (31 C) and an external pacemaker in the right ventricle,[15] and moderate hypothermia (31 C) with a balloon catheter in the ascending aorta and a pacemaker in the right ventricle.[16] In these patients blood pressure was lowered (30–40 torr systolic) by rapid pacing of the heart (150–200 beats/min). The inflated balloon in the aorta stopped the outflow and increased coronary perfusion. All these methods have the drawbacks of damage to major vessels, thromboembolism, and arrhythmias. If blood pressure reached 25 torr systolic, ventricular fibrillation occurred, necessitating external massage and direct-current shock.

Rovit and co-workers[17,18] also used pacemaker-induced hypotension. They later discontinued the use of hypothermia because they felt it increased myocardial irritability. Blood pressure was initially lowered to 70–80 torr systolic using a ganglionic-blocking agent. Pacing was done through a catheter in the right ventricle at the rate of 150 beats/min. Blood pressure fell further to 30–40 torr systolic. When pacing was discontinued there was an increase in blood pressure to above previous levels, and some patients had ventricular arrhythmias during the procedure.

The reason offered for hypertension seen following cardiac pacing is that during hypotension induced by rapid pacing (ventricular tachycardia) the body's homeostatic mechanism comes into play with a release of catecholamines.[15] When the rapid pacing is discontinued the released catecholamines cause overshoot of the blood pressure. Attempts to avoid this by lowering the blood pressure initially with a ganglionic blocking agent did not fully resolve the problem.[18] Other workers suggested the use of propranolol. Beta-adrenergic blockade, however, did not resolve the problem completely.

We feel that our technique offers advantages over previously available methods. Moderate hypothermia has the advantage of reducing cerebral oxygen consumption, yet does not increase myocardial irritability. No active attempt was made to lower blood pressure beyond the levels associated with hypothermia, chlorpromazine, *d*-tubocurarine, and halothane. We did not observe a single episode of post-circulatory-arrest hypertension in our patients. This, we believe, was due to the sympatholytic effects of chlorpromazine and *d*-tubocurarine, and to the direct effect of halothane on the myocardium and blood vessels.

No major blood vessels were occluded, and as a result we had no associated problems of thrombosis, embolization, or arrhythmia. The use of dissecting microscope precludes methods requiring external cardiac massage. Gross movements of the patient would shift and disrupt the operative field. We have had no complications related to thoracotomy and pericardiotomy in our series.

With the use of pacemaker-induced hypotension, the blood pressure is 30–40 torr systolic. This may still be much too high. If a basilar aneurysm ruptures, the mortality is approximately 80 per cent.[2] It is therefore desirable to have a method where the possibility of rupture is very much reduced. With the use of our method, the operating table is adjusted to 15-degree head-up tilt, and when the circulation is arrested, a "cadaverous state" is obtained, during which the surgeon can dissect the aneurysm free and obliterate it with complete ease. There is no problem with time. If he is unable to do it in 4 minutes initially, he can continue following a short period of manual compression of the heart for 1–2 minutes. This freedom is not available with other methods.

Bendixen,[19] in his editorial on the report by Rovit and co-workers,[18] asked the question, "What effect does it have on surgical technique to be able to interrupt brain blood flow at will should rupture of the aneurysm occur?," as they had 11 ruptured aneurysms in 16 patients. He goes on to say, "We cannot know whether they would have had fewer ruptures had they not had the pacemaker available, but the question must be asked."

In the 14 operations performed, we had only one instance of ruptured aneurysm during the operation and the patient survived to make a complete recovery. This low incidence, we feel, was due to better operative conditions provided by our technique.

It is conceivable that in the future, with continuing improvement in surgical technique, the surgeon may be able to approch this problem without the help of circulatory arrest. Until such time, we feel that our method offers an excellent chance to reduce the mortality and morbidity associated with basilar arterial aneurysms.

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Arytenoid Cartilage Dislocation Following Prolonged Endotracheal Intubation

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Well-known laryngeal complications of endotracheal intubation include mucous membrane abrasion, glottic and subglottic edema of varying severity, and formation of laryngeal granuloma. We report here an unusual complication, arytenoid cartilage dislocation, in a patient with a complicated history of heart disease and prolonged intubation following open-heart surgery.

REPORT OF A CASE

A 61-year-old Caucasian man was transferred from another hospital for cardiac catheterization. He had had renal failure at the age of 12 years and rheumatic heart disease in childhood, with an asymptomatic residual heart murmur of mitral regurgitation. About four years prior to admission hypertension had been noted and treatment with digoxin and ethacrynic acid begun because of heart failure.

Six weeks prior to admission, the patient sustained a myocardial infarction, which was treated at another hospital, followed by pneumonitis and peripheral thrombophlebitis, which were treated with Dicumarol; however, treatment was discontinued when gastric bleeding occurred. The patient was readmitted to the same hospital five weeks later with a second myocardial infarction, accompanied by ventricular tachycardia which progressed to ventricular fibrillation. Resuscitation which included external cardiac massage and nasotracheal intubation was successful after 1½–2 hours. Before his transfer to this hospital an attempt to extubate the trachea was unsuccessful because of development of pulmonary edema.

On admission the patient was found to have a flail chest, probably the result of chest compression, infiltration of the right lower lobe, and collapse of the right upper lobe. Mechanical ventilation of the lungs with positive end-expiratory pressure was necessary for a few days to overcome hypoxia. Nasotracheal intubation was maintained for a total of 11 days.

Cardiac catheterization revealed almost complete obstruction of all main coronary vessels, as well as aortic regurgitation and aortic stenosis. After still another episode of pulmonary edema, the patient underwent coronary-artery bypass grafting and aortic valvuloplasty three weeks after admission. Tracheal intubation was easily done using a plastic tube of 8.5-mm internal diameter. The patient fared well during the operation, which lasted about 7½ hours. Upon extubation of the trachea the following morning, hoarseness of voice, thought to be the result of laryngeal

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