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Perioperative Management for Transplant of Autologous Adrenal Medulla to the Brain for Parkinsonism

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Parkinson's disease was originally described as "paralysis agitans" or the shaking palsy by James Parkinson in 1817. Parkinsonism (lower case "p"), the generic term for the disease, is characterized by tremor, bradykinesia, rigidity, and postural changes. The pathophysiology is dopamine deficiency in the basal ganglia associated with decreased numbers of melanocytes in the substantia nigra.¹

The only treatment before 1967 was placing lesions in the basal ganglia, although it was not very effective.² Since 1967, dihydroxyphenylalanine (DOPA) and other dopaminergic drugs^{3,4} have become the mainstay of therapy, owing to their ability to raise the central nervous system (CNS) concentration of dopamine.

Recently, surgical therapy has again been suggested. Transplantation of autologous adrenal medulla to the caudate nucleus of the brain has resulted in reduced symptoms and decreased drug requirements in two reports.^{5,6} Several of these operations have been performed

at our institution. This report describes perioperative management of these patients.

METHODS AND MATERIALS

Twelve patients have undergone this procedure, which was approved by the local ethics committee. Informed consent was obtained from each patient. Ages were 39 to 49 yr and parkinson disability was Stage II to Stage IV.⁷ All patients had been with L-DOPA, carbidopa, bromocriptine, amantadine, trihexyphenidyl, in various combinations and were experiencing worsening of symptoms.

Patients had nothing by mouth for at least 6 h before the operation. A Brown-Roberts-Wells stereotactic base and frame was placed on the head after sedation with midazolam 1-2 mg iv and infiltrating the scalp with a 50/50 mixture of 1% lidocaine with 1:100,000 epinephrine and 0.5% bupivacaine (total volume 10-15 ml). Computerized tomography was performed and target point coordinates determined.^{8,9}

After transport to the operating room, arterial blood pressure cuff, electrocardiograph, precordial stethoscope, and pulse oximeter were placed. Further monitoring after induction of anesthesia included nasopharyngeal temperature, esophageal stethoscope, radial arterial catheter, urinary catheter, and mass spectrometry.

Additional midazolam was given in 1-mg increments as the upper airway was anesthetized with nebulized 4% lidocaine (4-5 ml) and viscous 2% lidocaine (15 ml) in the nose.¹⁰ After spraying with 0.5% phenylephrine (1 ml total), a warmed tracheal tube was inserted through the

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TABLE 1. Percent Change in Mean Arterial Blood Pressure (MAP); (Event MAP)-(Baseline MAP*) \times 100 (Baseline MAP)

Patient	Event Number†						
	1	2	3	4	5	6	7
1	.88	18.4	9.65	49.1	16.67	3.07	28.1
2	-18.9	16.1	-14.3	7.14	16.1	-3.57	-10.7
3	-16.0	14.5	7.42	21.1	36.7	19.1	7.42
4	9.02	36.8	9.02	35.3	29.3	12.4	16.5
5	-19.3	8.47	2.46	21.4	22.8	-1.75	5.26
6	-32.1	9.28	-6.42	16.1	4.64	4.64	1.79
7	-39.0	5.82	-31.5	13.0	30.1	-17.8	-14.4
8	-25.3	6.67	-4.00	1.67	18.3	7.67	-2.00
9	23.6	31.1	-1.97	8.66	8.66	13.0	7.87
10	-20.3	-14.9	-22.3	-18.9	-9.46	-9.46	-5.40
11	-26.4	-2.14	-7.14	-18.6	-7.14	-18.6	-3.57
12	-25.8	-8.87	-6.45	-4.44	-8.06	-9.27	-12.9

* Baseline MAP is defined as the average arterial blood pressure 1 day preoperatively.

† Events are: 1) after induction, 2) during adrenal dissection, 3)

after adrenal dissection while waiting for caudate preparation, 4) during adrenal manipulation, 5) during adrenal vein ligation, 6) while placing the graft, and 7) during closing.

nose and a fiberoptic bronchoscope passed through the tube. The trachea was identified and cannulated, and the tube advanced. The connection with the anesthesia circuit was established below the plane of the stereotactic base.

Anesthesia was induced with thiopental 3-5 mg/kg, sufentanil 1 μ g/kg, and vecuronium 100 μ g/kg iv and maintained by sufentanil infusion, and N₂O/O₂ (2 l/1 l), with isoflurane (0.25-0.5% end-tidal) as needed to control heart rate and arterial blood pressure. Vecuronium infusion was titrated to maintain the first twitch in a train-of-four¹¹ at 10% of control as assessed by a linear force transducer.

Position was left lateral decubitus with the head rotated to the right. The adrenalectomy and the craniotomy were performed simultaneously. As the adrenalectomy began, an Ommaya reservoir was placed and a right frontal craniotomy begun for exposure of the caudate nucleus. After the adrenal was removed, the medulla was separated, fragmented, and placed in a "pouch" that was created in the caudate.

During wound closure, nitrous oxide, sufentanil, and vecuronium were discontinued and isoflurane increased as needed to prevent hypertension. After closure, isoflurane was discontinued and naloxone administered for respiratory rate < 12/minute. Neuromuscular blockade was reversed with neostigmine (50 μ g/kg) and glycopyrrolate (10 μ g/kg). The trachea was extubated when patients could follow commands and sustain head lift for 15 s.

Perioperative sympathetic response was studied in seven patients by measuring arterial blood pressure, heart rate, and serum catecholamines at eight events: 0) baseline, the day before operation, 1) after induction of anesthesia, 2) during adrenal dissection, 3) after adrenal dissection while waiting for caudate preparation, 4) during adrenal manipulation, 5) during adrenal vein ligation, 6)

while placing the graft, and 7) while closing. Baseline arterial blood pressure was defined as the average arterial blood pressure the day before operation. Arterial blood was placed in EGTA/glutathione tubes and the serum frozen at -70° C for later radioenzymatic assay of epinephrine, norepinephrine, and dopamine.¹² Linear regression of percent change in catecholamines *versus* percent change in mean arterial pressure was performed. Changes in catecholamine concentrations were analyzed by analysis of variance and Tukey's Honestly Significant Difference Test. All statistics were performed with Statgraphics™ (STSC, Inc, Rockville, MD) on an IBM-PC™.

RESULTS

All values are reported as mean \pm standard error of the mean.

A total of 3.54 ± 0.37 mg midazolam was administered. Some patients were sensitive to the sedative effects of midazolam but there were no adverse effects before induction. Operative time was 202 ± 14.8 min. Sufentanil dosage was 0.41 ± 0.02 μ g \cdot kg⁻¹ \cdot h⁻¹, excluding the initial bolus. Seven (58.3%) patients required naloxone 5.32 ± 0.8 μ g/kg. Vecuronium dosage was 0.67 ± 0.1 μ g \cdot kg⁻¹ \cdot min⁻¹. Twitch height and train-of-four ratio recovered to 100% in all patients after reversal.

Arterial blood pressure was well controlled throughout the operation. One patient had a transient increase in mean arterial pressure of 49.1% above baseline (table 1), but in general arterial blood pressure and heart rate remained within 30% of baseline arterial pressure by titrating the sufentanil infusion or adjusting the isoflurane concentration.

Dopamine and norepinephrine were significantly decreased after induction of anesthesia and epinephrine in-

creased significantly during adrenal manipulation (table 2). There was no correlation between any of the catecholamines and change in MAP ($r = 0.026-0.091$, $P > 0.05$).

Patients were subjectively drowsier than non-parkinsonian craniotomy patients, but they followed commands and their tracheas were extubated at the end of the case. Two patients had a right pneumothorax, and one developed retroperitoneal bleeding requiring exploration. No patients required postoperative tracheal intubation. One patient developed a right frontal lobe abscess 4 months postoperatively. There were no immediate deaths, but one patient died of a myocardial infarction several months postoperatively.

DISCUSSION

A number of challenging anesthetic problems occur secondary to placement of the stereotactic frame prior to induction of anesthesia, the simultaneous performance of the adrenalectomy and craniotomy, and the parkinsonism.

Stereotaxis simplifies identification of the caudate nucleus and the normal-sized cerebral ventricles, but the position of the frame obstructs easy airway access. The frame may be applied after induction of anesthesia or with local anesthesia and sedation before anesthetic induction. Application of the frame prior to induction of anesthesia eliminates transporting an anesthetized patient, an advantage that is important if the scanner is remote from the operating room. Fiberoptic endoscopy simplifies endotracheal intubation and has been used in over 50 stereotactic craniotomies without untoward effects (unpublished data).

Catecholamine release during adrenal manipulation makes the anesthetic choice similar to that for excision of pheochromocytoma; the anesthetic should not cause sympathetic stimulation, should suppress sympathetic stimulation, and should minimize the consequences of catecholamine release.¹³ Thiopental and narcotics appear to be free of such interactions. Nitrous oxide is an alpha-sympathomimetic,¹⁴ but this effect is offset by its myocardial depressant effects.¹⁵ Catecholamine-induced arrhythmias occur at lower concentrations with halothane than with isoflurane or enflurane.¹⁶ Droperidol should be avoided, since it prevents re-uptake of catecholamines in the adrenal medulla.¹⁷ Droperidol may also precipitate a parkinsonian crisis, since it is a central dopamine antagonist.^{18,19}

Muscle relaxants may affect sympathetic tone. Pancuronium is a sympathomimetic²⁰ and can augment sympathetic response during adrenal manipulation.²¹ Histamine has been associated with hypertension in pheochromocytoma,²² although d-tubocurarine,²³⁻²⁵ metocurine,²⁵ and atracurium²⁶ have been used without problems. Ve-

TABLE 2. Plasma Catecholamines (pg/ml) during Adrenal Caudate Transplant (n = 7 Patients, Mean \pm SEM)

Event*	Epinephrine	Norepinephrine	Dopamine
0	81 \pm 5	766 \pm 34†	998 \pm 170†
1	37 \pm 12	383 \pm 22	309 \pm 43
2	202 \pm 17	533 \pm 23	256 \pm 34
3	691 \pm 94‡	547 \pm 33	249 \pm 29
4	600 \pm 59	550 \pm 22	227 \pm 29
5	521 \pm 57	511 \pm 24	186 \pm 18
6	130 \pm 16	501 \pm 14	182 \pm 19
7	87 \pm 7	445 \pm 18	163 \pm 17

* Events are: 0) baseline, 1) after induction, 2) during adrenal dissection, 3) after adrenal dissection while waiting for caudate preparation, 4) during adrenal manipulation, 5) during adrenal vein ligation, 6) while placing the graft, and 7) during closing.

† Significantly higher than the other norepinephrine or dopamine values. ($P < 0.05$).

‡ Significantly higher ($P < 0.05$) than baseline.

curonium, on the other hand, does not alter hemodynamic stability^{27,28} or release histamine,^{**} and is probably the best choice of the available relaxants.

Postoperative drowsiness may be worsened by perioperative medications. Sufentanil may cause residual depression, even after naloxone. Midazolam used before induction of anesthesia should be nearly eliminated,²⁹ but since benzodiazepine sensitivity may be greater in basal ganglia disease,³⁰⁻³² low concentrations may be significant. Patients also received preoperative phenytoin. Early in the series, a loading dose was given intraoperatively. Later, the loading dose was given 2 days preoperatively, which resulted in less sedation. Whenever possible, drugs that may cause residual sedation are avoided or at least kept at minimum effective doses.

Bradykinesia affects evaluation of postoperative sedation because the slow movements give the appearance of sedation. Preoperative anti-parkinson medication was withheld early in the series, and patients required carbidopa/l-DOPA in the recovery room. Medical therapy is now continued to the time of operation and additional doses given if weakness occurs.

None of the patients became nauseated and/or vomited postoperatively. Many antiemetics, including phenothiazines and butyrophenones, are central dopamine antagonists, and may worsen dyskinesias. Diphenhydramine, a central anticholinergic, is a weak anti-emetic and lacks extrapyramidal side effects. It may, however, cause sedation. Nasogastric suction may reduce the need for medication. All patients received nasogastric suction and no further treatment was necessary.

Epinephrine was elevated manipulating the adrenal,

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but norepinephrine and dopamine concentrations fell and stayed below baseline after induction of anesthesia. This may partly explain modest intraoperative blood pressure changes; in pheochromocytoma, hypertension tends to be less marked in tumors secreting a relative excess of epinephrine.¹³ Mild arterial blood pressure changes may also be due to the tendency for hypotension in patients receiving l-DOPA.³³ There was no adrenal pathology, so this pattern of catecholamine response probably represents the response of normal adrenal glands to surgical manipulation under balanced anesthesia. Sufentanil is known to decrease norepinephrine,³⁴ but should not prevent release during adrenal manipulation.

The sensitivity of parkinsonians to d-tubocurarine is similar,³⁵ but no information is currently available concerning sensitivity to vecuronium. Cannon *et al.* reported infusion rates of $0.92 \pm 0.37 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ to maintain twitch height at 10% of control in non-parkinson patients employing balanced anesthesia with fentanyl³⁶ versus the $0.67 \pm 0.11 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ in our patients. Their variability in values would suggest that there is no difference in dosage requirements for vecuronium in patients with parkinsonism.

In summary, nitrous oxide, sufentanil infusion, and vecuronium for muscle relaxation, supplemented with low concentrations of isoflurane after an awake, sedated fiberoptic tracheal intubation provides adequate anesthesia for adrenal caudate transplant. Other anesthetic techniques could be used as long as emergence from anesthesia is rapid, there is no stimulation of the sympathetic-adrenal system, and drugs that exacerbate parkinsonism are avoided.

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Fatal Air Embolism after Gastrointestinal Endoscopy

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Although intraoperative venous air embolism (VAE) has been described in many settings, the conditions surrounding the case of VAE during gastrointestinal endoscopy have not previously been described.

REPORT OF A CASE

The patient, an infant girl, originally presented for medical care at age 1 month with persistent hyperbilirubinemia and was found to have biliary atresia. At age 5 weeks, the patient underwent a Kasai procedure (hepatoportoenterostomy), and her condition improved. Liver biopsy at that time revealed a mild degree of hepatic cirrhosis. Over the subsequent 3 months, the patient experienced multiple bouts of ascending cholangitis. On the current hospital admission, the 4-month-old patient's bile drainage had diminished significantly, and the surgeon wished to visualize the hepatoportal enterostomy site with a gastroscope.

In the operating room, monitors (including pulse oximeter) were applied, and the induction of anesthesia began by inhalation of nitrous oxide (70%) and oxygen (30%) via a mask with increasing concentrations of halothane. After atropine (0.04 mg) and succinylcholine (10 mg) were administered iv, nitrous oxide (N₂O) was discontinued. After 100% oxygen had been administered for 1 min, the trachea was intubated, and bilateral auscultation of the chest demonstrated clear and equal breath sounds. Vital signs were stable at this time with an arterial blood pressure of 90/40 mmHg, a normal sinus rhythm at 120/bpm, and 100% oxygen saturation of hemoglobin. Anesthesia was maintained with 2% halothane and 60% N₂O in oxygen. About 4 min after the gastroscope was inserted into the stoma, the peripheral oxyhemoglobin saturation dropped to 60% and the heart rate decreased from 140 to 70 bpm within 15-30 s. Almost immediately thereafter, peripheral pulses were not palpable, and the ECG showed ventricular fibrillation.

Cardiopulmonary resuscitation was begun, and the femoral artery was exposed by surgical dissection in order to obtain arterial blood for gas analysis. Air bubbles were noted in the arterial cannula as blood was withdrawn. The patient never regained a viable heart rhythm or arterial blood pressure.

At autopsy, massive air embolus was determined to be the cause of death. Air was found in the right atrium, right ventricle, and in a large hepatic vein in the area of the porta hepatis. A patent foramen ovale was noted, and air was present in the coronary arteries.

On closer examination of the hepatoportoenterostomy, the pathologist found a large hepatic vein just under the enterostomal surface of the liver. Examination of serial sections of tissue revealed that the hepatic vein approached and was almost contiguous with the denuded surface of the liver. The medical examiner's report also included an evaluation of the gastroscope. This gastroscope was capable of infusing air or water by operating one switch. By occluding the end of a button with light pressure, the operator would infuse air at 8-9 cc/s. If the button were completely depressed, water would be infused at a rate of 0.3 cc/s.

DISCUSSION

The effects of VAE may be minimal or clinically significant depending on the amount of air infused, its rate of infusion, and the location of the embolus. When an open vein is higher than the level of the heart with a pressure gradient of approximately 5 cm H₂O, the patient is at risk for VAE.¹ VAE is commonly reported during neurosurgical procedures in the sitting positions.² It has also been described during Cesarean section,³ liver transplantation,⁴ and total hip replacement.⁵ However, a review of the literature suggests that a case similar to ours has not been reported.

Studies in dogs revealed different physiologic responses to VAE depending on the rate and quantity of air infused.⁶ Slow infusion of air caused predictable cardiopulmonary changes, including, in order of increasing air infusion, a characteristic "gasp," ECG changes (peaking of P waves, later ST segment depression), increased central venous pressure, increase in heart rate, decrease in peripheral resistance, and eventually the presence of a "mill wheel"

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