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Anesthesiology
67:823-826, 1987

Unexpected Focal Neurologic Deficit on Emergence From Anesthesia: A Report of Three Cases

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The occurrence of a new neurologic deficit on emergence from anesthesia may be the result of a focal or global cerebral insult. Patients experiencing a global cerebral insult from profound hypotension, hypoxia, or cardiac arrest may have diffuse neurologic abnormalities without localizing signs. A systemic insult can also occur in a patient who has a focal lesion making one region of the central nervous system more vulnerable to injury, such as hypotension in a patient with carotid artery stenosis. A patient emerging from anesthesia with a new and unexpected focal neurologic abnormality must be assumed to have suffered an acute focal injury from either ischemia or hemorrhage. The three cases below demonstrate various etiologies for unexpected focal neurologic deficit on emergence from general anesthesia.

CASE REPORTS

Case 1. A 65-yr-old, 72-kg man with a horseshoe kidney and a large staghorn calculus in his right renal pelvis underwent percutaneous ultrasonic lithotripsy following percutaneous placement of a wire into the renal pelvis under local anesthesia. His medical problems included hypothyroidism treated with synthroid 0.125 mg daily, chronic pulmonary interstitial fibrosis, coronary artery disease with an old anterior myocardial infarction, and history of one episode of cardiac syncope due to episodic atrial fibrillation 1½ yr prior to this hospitalization. Preoperative physical examination revealed bibasilar rales, but was otherwise normal. Laboratory values were normal. ECG showed

normal sinus rhythm, old anterior myocardial infarction, and a T wave abnormality. No premedication was administered.

General anesthesia was induced with thiopental 400 mg iv and maintained with 50% nitrous oxide and 0.5-1.5% enflurane in oxygen. Endotracheal intubation was facilitated with succinylcholine 60 mg iv. The patient was placed in the prone position with his head turned to the left. During positioning and patient preparation for the procedure, the patient's arterial blood pressure fell from an initial awake 140/85 mmHg to 90/55 mmHg. This hypotension responded over a 20-min period to an infusion of 1 liter of 5% dextrose in lactated Ringer's solution and mephenteramine 7.5 mg iv. The remainder of the intraoperative period was uneventful and the patient remained hemodynamically stable. The patient was transported to the recovery area with his trachea intubated and breathing spontaneously.

Ten minutes postoperatively, it became apparent that the patient was more lethargic than expected and had no motor function in the right extremities. On physical examination, sensory function was normal, but there was dense right hemiplegia, positive right Babinski sign, right facial motor deficit, and marked dysphasia, expressive more than receptive. The cranial nerve reflexes were intact. There were no carotid bruits and the heart was normal on auscultation. The patient underwent immediate computerized tomography (CT) head scan that showed no focal changes. He remained hemodynamically stable during the postoperative period and, with spontaneous respiration supplemented by 70% O₂ through a close-fitting mask, PaO₂ was 150 mmHg, PaCO₂ 38 mmHg, and pH 7.38. Further evaluation included normal oculoplethysmography and digital venous angiography revealing normal extracranial carotid arteries with diffuse disease of the medium-sized intracranial vessels. Echocardiography revealed mild left atrial enlargement, apical left ventricular aneurysm, and mural thrombus. Diagnosis was felt to be focal left cerebral hemispheric infarction, probably due to emboli of cardiac origin.

The patient showed gradual improvement in his motor and speech deficit, beginning 8 h postoperatively. Therapy included anticoagulation for 3 months begun 4 days postoperatively, and physical and speech therapy. Within 2 weeks, the patient had near complete return of motor function. The patient was discharged 2 weeks postoperatively with a slight foot drop and weakness of the biceps and flexor muscles of the wrist and hand. The patient was interviewed by telephone 2 yr following the procedure, and reported complete functional recovery occurring within a few weeks after discharge.

Case 2. A 32-yr-old, 61-kg woman with persistent pelvic pain underwent pelvic laparoscopy. Her medical problems included chronic diarrhea and pelvic pain of 3 yr duration. Her medications included only

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Key words: Complications: neurologic. Recovery.

diazepam 5 mg t.i.d. The physical examination and preoperative laboratory evaluation were normal. No premedication was given. General anesthesia was induced with thiopental 500 mg iv, and maintained with 60% nitrous oxide and 0.5–1.5% enflurane in oxygen following endotracheal intubation facilitated with succinylcholine 100 mg iv. The patient was placed in the lithotomy position, mechanically ventilated, and received 1.3 liters lactated Ringer's solution during the procedure.

Twenty minutes into the procedure, multifocal ventricular ectopy was noted for a 5-min period. Peak and mean airway pressures rose during this 5-min period. There was no associated hypotension or change in arterial oxygen saturation by pulse oximeter. These airway changes resolved spontaneously and the remainder of the procedure was unremarkable. Thirty minutes postoperatively, the patient appeared unusually lethargic and a right hemiparesis and facial paresis were noted. Heart rate and arterial blood pressure were stable. Repeated examination over the next 30 min demonstrated improvement in awareness and persistent marked right sided hemiparesis. With spontaneous respiration and a close fitting mask of FIO_2 1.0, PaO_2 was 355 mmHg, Paco_2 39 mmHg, and pH 7.36.

The patient had an immediate CT head scan that showed no focal abnormalities. Echocardiography was performed. A four-chamber view was obtained and indocyanine green injected both before and following a Valsalva maneuver to detect a right-to-left intracardiac shunt. The echocardiogram was normal, and no right-to-left shunt was demonstrated. During the immediate 4 h postoperatively, the patient showed gradual resolution of her lethargy and some improvement of her right-sided motor deficit. She breathed supplemental oxygen during the first 24 h and continued to show resolution of the motor deficit, with complete recovery within 48 h postoperatively. The combination of cardiac arrhythmias during intraabdominal CO_2 insufflation without hypotension or hypoxia and focal neurologic deficit on emergence form the basis for a probable diagnosis of paradoxical CO_2 embolism, in spite of failure to demonstrate a patent foramen ovale.

Case 3. A 57-yr-old 62-kg female was scheduled for a vaginal hysterectomy with anterior and posterior repair for relief of urinary incontinence. Past medical history included hypertension controlled with Diazide® (hydrochlorothiazide and triamterene) and general anesthesia on two previous occasions with no complications. The only positive factor in the family history was that her mother had suffered a fatal "stroke" during bunion surgery years previously. Physical examination was essentially normal. All preoperative laboratory investigations were within normal limits.

Anesthesia was induced with thiopental, 450 mg iv. After d-tubocurarine 3 mg iv, paralysis was achieved with succinylcholine 80 mg iv, and the trachea intubated. At this point, ventricular bigeminy lasting approximately 15 s was noted, and the arterial blood pressure by cuff increased for 5 min from 120/70 to 180/90 mmHg. Anesthesia was maintained with 50% nitrous oxide and enflurane 1–2% in oxygen. Anesthesia was supplemented with morphine sulfate 8 mg iv. The patient was placed in 15° Trendelenburg position for the operative procedure. The operative course was uneventful and the patient was extubated in the operating room with adequate spontaneous ventilation.

After approximately 2 h in the recovery area, the patient was still very lethargic. Examination revealed left hemiplegia. Immediate CT head scan revealed a large right intracerebral hematoma. A right craniotomy was performed to evacuate the hematoma. Postoperatively, the patient had a marked left hemiparesis that did not resolve by the time of her discharge 2 weeks postoperatively.

DISCUSSION

Perioperative neurologic injury usually results from either ischemia or hemorrhage. Ischemic neurologic in-

jury may result from embolic events, thrombosis of damaged arteries, or arterial compression. The heart is the major source of arterial emboli, with intracardiac thrombus occurring in patients with ischemic heart disease, valvular disease, and dysrhythmias, especially atrial fibrillation. Patients with atherosclerotic disease of the carotid or vertebrobasilar system may have ischemia due to emboli or inadequate blood flow through stenotic vessels.¹

Emboli can originate in the venous system and cross to the arterial system, resulting in arterial ischemic injury.^{2–5} The mechanism for right-to-left crossing of venous emboli must include an opening for venous to arterial passage of the emboli, such as an intracardiac shunt and a pressure gradient to facilitate this movement. An autopsy study revealed a 27% incidence of probe patent foramen ovale in patients with no history of cardiac disease.⁶ Such patients would be at risk for passage of any emboli from the venous to arterial system if right atrial pressure transiently exceeds left atrial pressure. These venous emboli may be from deep venous thrombosis, tumor, air,^{2–4} or CO_2 .^{5,7–9} Many operative procedures are performed with the operative site above the level of the heart, making these patients at risk for venous air embolism and paradoxical air embolism in the presence of a patent foramen ovale.

Intracranial hemorrhage can cause perioperative neurologic injury. Hemorrhage may be spontaneous (primarily hypertensive) or due to disruption of a vascular anomaly. Hemorrhage due to ruptured aneurysm has its peak incidence between 55 and 60 yr. Cerebral arteriovenous malformation is the major cause of non-traumatic cerebral hemorrhage in patients under 20 yr of age.¹⁰

The evaluation of a patient who awakens from anesthesia with a new and unexpected neurologic deficit should begin with a thorough review of the patient's history and a careful physical examination. Pertinent information in the history includes prior history of neurologic events suggestive of transient ischemic attacks or subarachnoid hemorrhage, medical conditions associated with increased risk of emboli, type of operative procedure, and a review of the anesthesia record for significant intraoperative events. A thorough neurologic examination should be performed. The heart and major vessels should be auscultated for evidence of valvular disease or significant obstruction to flow. Further evaluation should be based on those etiologies most consistent with the patient's history and physical findings (table 1).

As soon as it is determined that a focal deficit has occurred and is persistent, a CT head scan should be obtained. The CT should be performed with and without contrast because contrast may enhance or obliterate

the abnormality. Lesions due to focal transient ischemia (TIA) will not result in any abnormalities on CT scan. Patients who have suffered a permanent neurologic deficit based on ischemia will have a lucent area on CT first present 4–5 days following injury, unless it involves a massive area, in which case an abnormality may be detected within several hours. Prior to that time the scan will be normal. Deeper lesions due to ischemia are more likely to be visualized than surface lesions. Patients whose deficit is due to hemorrhage, either subarachnoid or intracerebral, will have a hyperdense area representing the blood. Contrast may help to identify an aneurysm or arteriovenous malformation.¹¹

Angiography may be indicated in the patient with a hemorrhagic lesion or suspicion of carotid or vertebrobasilar insufficiency. Intracerebral hemorrhage sufficient to cause mass effect requiring evacuation may require an angiogram preoperatively to identify and define any vascular anomaly. A patient suspected of having an aneurysm or arteriovenous malformation not requiring surgical intervention for evacuation of hematoma should have an angiogram nonemergently to establish the diagnosis and plan any operative therapy.¹²

The patient whose evaluation is suggestive of an embolic event from an intracardiac source should undergo echocardiography to define the source of emboli. Those at risk for venous embolism should have echocardiographic evaluation for a patent foramen ovale or previously undiagnosed intracardiac shunt. This evaluation is carried out using echocardiographic contrast with and without Valsalva maneuver to detect passage of contrast from right to left cardiac chambers.¹³ Because echocardiography does not detect all cases of patent foramen ovale, failure to demonstrate a right-to-left shunt should not rule out the diagnosis of paradoxical embolus. It is likely that the atrial gradients which occur during venous gas embolism cannot be reliably reproduced during a Valsalva maneuver.

An electroencephalogram (EEG) does not aid in the differential diagnosis in the acute period. Focal distortion or disappearance of normal rhythms or focal appearance of abnormal rhythms is likely, but is not helpful in defining the etiology. Serial electroencephalograms are of more value to assess the evolution of an acute lesion and for prognosis.

Optimal management of the patient includes rapid evaluation as well as early consultation with a neurosurgeon. The time course within which intervention can lead to improvement of existing deficit is only several hours. Management of the patient with an ischemic deficit varies with the etiology and clinical course. For the patient whose ischemia is due to cerebrovascular disease, anticoagulation should be considered only for a transient ischemic attack or stroke in evolution without

TABLE 1. Diagnostic Tests in Evaluation of New Focal Neurologic Deficit

Test	Etiology	Usefulness
CT scan	Hemorrhage	Hyperdense area (contrast may demonstrate vascular anomaly)
	Ischemia	Acutely normal (lucent area several hours to days post insult)
Angiogram	Hemorrhage	Identify vascular anomaly
ECHO	Ischemia	Identify cerebrovascular disease
	Hemorrhage	Not useful
EEG	Ischemia	Define intracardiac source of emboli, identify patent foramen ovale.
	Hemorrhage	Limited value acutely

evidence of infarction. With ischemia due to carotid artery disease, carotid endarterectomy should be considered for recurrent transient ischemic attacks in spite of adequate anticoagulation, progressive or fluctuating neurologic deficit, and possibly for new major deficits due to acute occlusion. In these patients, arterial blood pressure should be maintained in the range that is normal for that patient without significant hypotension.

When the etiology of the ischemia is felt to be embolic, induced hypertension may be considered to improve collateral blood flow. Long-term anticoagulation should be considered for patients with intracardiac thrombus. A patient felt to have suffered arterial air or CO₂ embolism should breathe 100% O₂. Transfer to a hyperbaric chamber to reduce bubble size and improve blood flow should be considered if this can be accomplished within 8 h of the event.¹⁴

A patient who has suffered an intracerebral hemorrhage with mass effect should undergo craniotomy for evacuation of the hematoma. Prior to surgery, management should be directed toward maintenance of adequate cerebral perfusion pressure and controlling intracranial pressure. Intracerebral hemorrhage without significant mass effect and without vascular anomaly should be managed medically with control of intracranial pressure, control of hypertension without profound hypotension, and appropriate supportive care.

Intraoperative detection of the occurrence of a focal neurologic deficit is difficult. Routine monitoring of vital signs might reveal hypertension and bradycardia occurring with elevations of intracranial pressure. However, these changes are nonspecific and occur late in the course. Changes in pupil size and reactivity might reveal the occurrence of some neurologic events, but these can be altered by many drugs routinely administered during an anesthetic. Intraoperative monitoring of a processed EEG might be expected to aid in diagnosis of

these events. Those deficits which would be detected depend on the processed EEG utilized and the nature of the lesion. Whether intraoperative diagnosis would improve outcome would depend on the etiology. If an intraoperative diagnosis of hemispheric cerebral abnormality had been made, diagnostic and therapeutic modalities could have been started earlier in each of these cases. Since prompt correction of neural compromise is essential to preservation of function, improvement in ultimate outcome might be expected. However, recommendations concerning routine use of intraoperative monitoring of processed EEG must take into account the relative infrequency of these events. These three cases occurred over a 2-yr period, during which time 58,907 general anesthetics were administered at our hospital.

A certain incidence of stroke can be expected to occur in this surgical population during the period of anesthesia but unrelated to it. We examined our local stroke database to place these three cases in perspective to the population at large. The expected number of strokes was determined by multiplying the age- and sex-specific patient years at risk (each individual was considered to be at risk for 6 h or ¼ day) and the age- and sex-specific incidence rates for stroke in the Rochester, Minnesota, population for 1975–1979.¹⁵ The 58,907 patients seen in 1984–1985 represented 40.3 person-years of observation, assuming 6 h at risk per patient. The expected number of strokes in this surgical group unrelated to anesthesia and surgery was 0.1 case, far lower than the three cases presented here.

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