

Hypoxia as a Manifestation of Neurogenic Pulmonary Dysfunction

LANCE K. PARKS, M.D.,* AND NORMAN A. BERGMAN, M.D.†

Numerous clinical and experimental studies indicate that events occurring in the central nervous system can cause alterations in pulmonary function. Neurogenic pulmonary dysfunction is extensively discussed in a recent review of therapeutic and anesthetic considerations relative to intracranial hypertension.¹ In previous studies involving man, there were appreciable intervals between cerebral injury and the manifestations of pulmonary abnormalities. The present report describes a case in which severe systemic hypoxia occurred in an anesthetized patient immediately after a cerebral injury which was subsequently shown to have caused left partial hemiparesis.

REPORT OF A CASE

A 92-kg, 193-cm, 51-year-old Caucasian man was admitted to the University of Oregon Medical School Hospital for investigation of chronic proteinuria from an atherosclerotic left kidney secondary to chronic pyelonephritis. Significant findings in the history included transient episodes of dizziness and blurred vision during the preceding several months and a 25-pack-a-year smoking history. On physical examination there were bruits over both external carotid arteries and the right subclavian artery. Fine rales and wheezes were present over both lung bases but cleared with cough. Blood pressure was 130/90 mm Hg. Results of laboratory studies, including hemogram, blood chemistry, and arterial blood-gas studies (1, table 1) were within normal limits with the exception of 2-plus proteinuria found on urinalysis. Electrocardiogram and chest x-ray were unremarkable. Angiographic studies showed bilateral narrowing of the carotid arteries, more marked on the right, as well as severe right subclavian stenosis. Following the angiogram, weakness of the left arm developed, but resolved spontaneously within 24 hours. It was therefore decided to defer further

urologic studies and proceed with a right carotid endarterectomy.

Premedication for the endarterectomy was morphine sulfate, 10 mg, and atropine sulfate, 0.5 mg, im, one hour prior to anesthesia. Induction of anesthesia was with fentanyl, 0.25 mg, and thiopental, 200 mg, iv. An 8.5-mm cuffed endotracheal tube was inserted without difficulty with the aid of succinylcholine, 100 mg, preceded by pancuronium, 1.0 mg. Anesthesia was maintained with nitrous oxide, 70 per cent in a total flow of 5 l/min, supplemented with an additional 0.25 mg fentanyl in divided doses. Pancuronium, 4 mg, was given before incision of the skin. Respirations were controlled at a frequency of 12/min and a tidal volume of 800 ml. The patient's condition was monitored with an esophageal stethoscope, temperature probe, and electrocardiograph. Blood pressure was measured with a cuff and transcutaneous Doppler flow sensor. A percutaneous plastic cannula was placed in the left radial artery. Results of arterial blood-gas analysis of blood obtained 15 min after induction and 10 min prior to the beginning of operation (2, table 1) were interpreted as indicating suboptimal oxygenation with slightly more alveolar ventilation than was desired. The concentration of nitrous oxide was reduced to 60 per cent, with total flow remaining at 5 l/min. Respiratory frequency was reduced to 8/min. During the first 15 min of operation systolic blood pressure increased from 130 to 180 mm Hg. Enflurane, approximately 1 per cent, was subsequently added to the anesthetic mixture. Another arterial blood-gas determination after 30 min of operation (3, table 1) showed that $P_{a_{O_2}}$ had increased. $P_{a_{CO_2}}$ and pH were unchanged from the previous determination.

Insertion of a shunt across the abnormal carotid segment began about 45 minutes after the start of operation. Considerable technical difficulty was experienced in accomplishing this. Several attempts at clearing persistent air bubbles from the shunt tubing using back-flushing from the distal carotid segment were necessary. Flow was re-established after a period of carotid occlusion estimated to 2-3 min in duration. Within a minute of resumption of flow, blood in the wound became considerably darker. There had been no alteration in flow-meter settings, respiratory frequency, or tidal volume. Thoracic respiratory excursions and esophageal breath sounds were grossly unchanged. Pulse rate and arterial blood pressure also remained stable. An arterial blood specimen was quickly obtained and the anesthetic gas mixture was immediately changed to 99 per cent oxygen and 1 per cent enflurane. $P_{a_{O_2}}$ (4, table 1) was considerably lower than that of the sample obtained 15 minutes earlier although $F_{I_{O_2}}$ and respiratory

* Resident.

† Professor and Chairman.

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Address reprint requests to Norman A. Bergman, M.D., Department of Anesthesiology, University of Oregon Medical School, 3181 S. W. Sam Jackson Park Road, Portland, Oregon 97201.

TABLE I. Selected Respiratory and Circulatory Values in a Patient before, during and after Carotid Endarterectomy

	Time	$F_{I_{O_2}}$	$P_{a_{O_2}}$	$P_{a_{CO_2}}$	pH	Systolic Blood Pressure (mm Hg)	Comments
1	Preoperative	0.2	78	36	7.38	130	
2	10:35	0.3	81	35	7.36	180	15 min after anesthetic induction
3	11:15	0.4	115	35	7.36	150	10 min before carotid clamping
4	11:30	0.4	65	34	7.38	160	2 min after opening of shunt
5	11:55	0.99	73	35	7.38	140	25 min on $F_{I_{O_2}} = 0.99$
6	12:20	1.0	316	39	7.37	140	3 min before extubation
7		0.4	139	38	7.37		48 hours postoperatively during mechanical ventilation

pattern were identical. $P_{a_{CO_2}}$ and pH were essentially unchanged. No abnormality in the anesthesia circuit could be found, and correct placement of the endotracheal tube was again verified by auscultation of the chest. Twenty-five minutes later, $P_{a_{O_2}}$ was 73 mm Hg despite $F_{I_{O_2}}$ 0.99 during the interval (5, table 1). After another 25 minutes, at the completion of operation, $P_{a_{O_2}}$ had increased to an acceptable level (6, table 1). Spontaneous ventilation promptly returned following administration of prostigmine, 2.0 mg, atropine, 1.5 mg, and naloxone, 0.2 mg, iv. Prior to removal of the endotracheal tube, purposeful movements of both legs, right arm and head occurred, but the left arm and left side of the face failed to move. The endotracheal tube was removed and the patient was taken to the recovery room, where vital signs remained stable. The presence of partial left hemiparesis involving the arm and face was confirmed. The postoperative course was complicated. Angiography was done and the operative site was re-explored that afternoon because of the persistent hemiparesis. A few intimal fragments were removed from the carotid lumen. Following this procedure an episode of acute respiratory and circulatory failure necessitated intensive care with ventilatory support for 48 hours. Toward the end of this period of mechanical ventilation $P_{a_{O_2}}$ was 139 mm Hg with $F_{I_{O_2}}$ 0.4 (7, table 1). The patient subsequently made an uneventful recovery and was discharged from the hospital on the tenth postoperative day with mild residual weakness of the left arm and face. The electrocardiogram, chest x-ray, and blood enzyme levels did not change significantly at any time during the postoperative period.

DISCUSSION

Derangements of structure and function in previously normal lungs as a consequence of

events occurring in the central nervous system have been reported by many investigators. In animals, pulmonary edema, systemic hypoxia and changes in elastic properties of the lung have been produced by experimental head injury,² creation of anatomic lesions in the central nervous system,³ experimental elevation of intracranial pressure,⁴ perfusion of the brain with hypoxic blood,⁵ and sympathetic stimulation.⁶ In some of these studies, pulmonary dysfunction could be prevented by prior high section of the spinal cord, alpha-adrenergic receptor blockade, or denervation of the lung. Analogous changes in pulmonary function associated with abnormalities of the central nervous system have been reported to occur under clinical conditions in man. Pulmonary edema has been reported to occur within 2 hours of acute head injury and is particularly resistant to treatment unless intracranial pressure is reduced.⁷ The presence of respiratory abnormalities has been confirmed in a high proportion of patients following acute head injury.⁸ Such patients frequently have a large alveolar-arterial oxygen tension difference and venous admixture.⁹ Battle casualties dying within a short interval after sustaining severe head injury already had well-defined anatomic pulmonary changes characteristic of adult respiratory distress syndrome. Fatalities with head injuries who also sustained high spinal cord transection did not manifest the pulmonary changes of RDS.³

We postulate that the hypoxia of the patient described in the present communication was a manifestation of neurogenic pulmonary dysfunction. A significant and confirmed decrease in arterial oxygen tension occurred abruptly and immediately following a probable cerebral insult that caused objective neurologic changes. No associated primary respiratory or circulatory change that could explain the hypoxia could be demonstrated. Possible factors involved in producing localized cerebral injury include embolization of air or atheromatous material and a period of carotid occlusion. Specific neurally mediated alterations in pulmonary function responsible for the large pulmonary shunt that must have existed in this patient when his alveolar-arterial oxygen tension difference was greater than 600 mm Hg are not well established.¹ This case is presented because we have been unable to find any other report in which a documented change in pulmonary function was temporally so closely associated with an identifiable cerebral injury under clinical conditions in man. It is possible that if arterial blood gases were systematically monitored in comparable clinical situations, transient neurogenic pulmonary dysfunction might be encountered relatively frequently.

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The Psoas Compartment Block

D. CHAYEN, M.D.,* H. NATHAN, M.D.,† M. CHAYEN, M.D.‡

Analgesia of the inferior segments of the lower limb can easily be achieved by a femoro-sciatic block, but a similar possibility, other than spinal or epidural analgesia,

has not been developed for the thigh because of the multiplicity of its nerve supply.^{1,2} Winnie³ has described an anterior inguinal approach for paravascular anesthesia of the lumbar plexus, and has also suggested the possibility of a lumbar paravertebral method of injecting the lumbar plexus.

* Resident, Department of Anaesthesiology Municipal Governmental Medical Center Tel Aviv.

† Professor of Anatomy, Tel Aviv Medical School.

‡ Clinical Associate Professor of Anaesthesiology, Tel Aviv Medical School, and Head of Department of Anaesthesiology, Municipal Governmental Hospital, Tel Aviv.

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Address reprint requests to D. Chayen, M.D., Department of Anaesthesiology, Ichilov Hospital, Weizman Street, Tel Aviv, Israel.

We have developed an approach to the lumbar plexus based on the fact that most of the branches of the lumbar plexus and some of the sacral plexus supplying the thigh are found close to each other in the region of the fourth lumbar vertebra in what we call the "psoas compartment." Here the plexus can be reached by a single injection.