Circulatory Embarrassment in Patients with Large Diaphragmatic Hernias

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It is generally known that diaphragmatic hemias in the newborn can present serious problems during anesthesia. Loehning, Takaori, and Safar¹ reported cardiac arrest in three patients with traumatic diaphragmatic hemias. The arrests followed institution of positive-pressure ventilation during anesthesia. Circulatory collapse during anesthesia in adults with nontraumatic diaphragmatic hemias has not been reported.

CASE REPORT

A 28-year-old man was scheduled for a left thoracotomy and repair of a large hernia of the foramen of Morgagni. His symptoms, which had started a year prior to admission, consisted of sharp sticking pain in the precordium associated with dyspnea. The pains occurred at night and were relieved by sitting up. The patient's weight was 200 pounds; blood pressure, 120/60 mm. Hg; pulse, 60 beats/min.; respiratory rate, 18/min. A grade ii/vi medium-pitched midsystolic murmur was heard at the second right interspace. X-ray revealed a large hernia of the foramen of Morgagni, containing colon in the left side of the Electrocardiogram showed minor intraatrial block and low-voltage QRS complexes in the precordial leads.

In the operating room, blood pressure was 100/ 50 mm. Hg; pulse, 70 beats/min.; central venous pressure 7 cm. H₂O. Induction of anesthesia was achieved with 250 mg. thiopentone sodium; intubation was facilitated with 80 mg. succinylcholine. Respiration was controlled manually until spontaneous efforts recurred. Periodic manual assistance of respiration was done with pressure not exceeding 10 cm. H₂O, using a semiclosed-circle Halothane, one per cent, two liters of N-O and two liters of oxygen were used for maintenance. Following induction, blood pressure remained at 100/50 mm. Hg; pulse rate ranged between 70 and 80 beats/min.; and central venous pressure rose to 10 cm. H₂O and remained at that level. After 30 minutes the anesthesia circle was connected to an Air Shields Ventimeter to control respiration. A tidal volume of 700 ml. was

delivered, resulting in a peak upper airway pressure of 15 cm. H-O at 16 times per minute. This was followed immediately by a sudden marked rise in central venous pressure to 35 cm. H₂O. Multiple premature ventricular contractions were observed on the oscilloscope; these occurred in groups of as many as four in succession, with a small number of sinus beats between them. Blood pressure fell to 70/50 mm. Hg. Injection of 75 mg. lidocaine intravenously was followed by prompt return to regular sinus rhythm with no premature beats and a rise in blood pressure to 100/60 mm. Hg. However, central venous pressure remained elevated at 34 cm. H-O. The respirator was disconnected and when spontaneous respiration was resumed, a reduction of the central venous pressure to 28 cm. H-O occurred. Heart rate and blood pressure were relatively stable until the end of the procedure. Opening the chest was associated with a slight drop of central venous pressure to 24 cm. H₂O and controlled respiration was resumed. A large hernial sac containing colon was opened and its contents reduced. Central venous pressure remained between 24 and 26 cm. H₂O until the hernia was completely reduced and the defect in the diaphragm closed, after which it dropped promptly to 12 cm. H-O. Blood loss during the procedure was minimal.

COMMENT

The commonest diaphragmatic hernias are those related to the esophageal hiatus.2 They pass into the posterior mediastinum and usually do not cause displacement of the heart.3 Bochdalek or posterolateral hernias in newborn infants usually displace the mediastinum, pushing the heart to the opposite side. Hernias of the foramen of Morgagni (retrosternal hernias) protrude through a defect in the anterior part of the diaphragmatic attachments to the sternum and costal cartilages, occur more commonly on the right, and contain omentum and colon.4 Such hernias may become large, as in the case described, and can compress the heart and lower lobe of the adjacent lung.

During positive-pressure ventilation the area

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of lung adjacent to the hernia expands and displaces the sac. The hernial sac, in turn, pushes against adjacent soft structures, namely the heart and great veins, resulting in reduction of cardiac output. This may precipitate arrhythmias. Although positive pressure ventilation usually is associated with some rise in the venous pressure, such rise is usually in the range of half the rise of mean airway pressure.5 The increase in central venous pressure observed in this case was far in excess of the rise of mean airway pressure. After cessation of positive-pressure ventilation, the venous pressure dropped to a level which was still more than twice the original value. Persistence of the rise in venous pressure after the chest was opened, and its return to a more nearly normal level after reduction of the hernia, is similar to the observation of Loehning et al.,1 who found a reduction in cardiac output and a rise in caval pressure following the inflation of a balloon inside the chest cavity in dogs. These changes persisted even after bilateral thoracotomies and subsided only after deflation of the balloon. They also observed that positive-pressure ventilation with low pressure in the presence of an inflated balloon was not associated with severe circulatory embarrassment. The mechanism of the persistent rise in central venous pressure is not very clear. It is possible that that a change in the size and position of the hernial sac occurred after positive-pressure ventilation and persisted after the return of spontaneous breathing.

Hunter showed that circulatory embarrassment can occur during nitrous oxide anesthesia in patients with artificial pneumothorax. Eger found that nitrous oxide can result in an increase in size of an enclosed gas-filled space in the body. Such an increase is significant when more than 70 per cent nitrous oxide is used. The volume of the air space in the chest can double in ten minutes using 75 per cent nitrous oxide. The volume changes associated with 50 per cent alveolar concentra-

tions of nitrous oxide are significantly less than those associated with 75 per cent alveolar concentration. It is doubtful that nitrous oxide caused a major change in the size of the hernia, because only 50 per cent nitrous oxide was used and there was no evidence of hernial obstruction or strangulation.

We suggest that anesthesiologists should be aware of the hazards of positive-pressure ventilation in similar conditions. A study of the X-ray finding is valuable. Monitoring of the electrocardiogram and central venous pressure may provide early warning of circulatory embarrassment in these cases. When the hernia involves the stomach and esophagus, the presence of a nasogastric tube helps keep the stomach empty. Assisted ventilation using low pressures can be performed provided that it is not accompanied by a severe rise in venous pressure and the occurrence of arrhythmias. In the presence of a closed air-containing space in the chest nitrous oxide should be avoided or should be limited to 50 per cent.

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