keep the veins distended, the tourniquet is kept in place during the following procedure.

With the thumb or fingers, firm pressure is exerted over the skin where a vein is expected to be (fig. 2).

After the pressure has been maintained for 10 to 15 seconds, a pit will appear when the thumb is removed. A visible and prominent vein will appear in the depression if the right place has been chosen. If a vein does not appear, or if a better vein is thought to be present in another place, a new pit is formed. If necessary, the pit is easily enlarged. A vein that has been made visible and prominent by this method is easy to puncture (fig. 3).

CASE REPORTS

Acute Pulmonary Edema during Anesthesia and Operation in a Healthy Young Patient

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A case of pulmonary edema during anesthesia and surgery in a young patient without known cardiopulmonary disease is reported. The steep Trendelenburg position of the patient and the possible obstruction to lymphatic drainage from the lungs are discussed as possible contributing causes.

REPORT OF A CASE

The patient, a 30-year-old woman, had a diagnosis of undifferentiated carcinoma of the pericardium. Except for the present illness, she had been in excellent health, with no history of cardiopulmonary disease. Preoperative laboratory values were: hematocrit, 36 per cent; total protein, 6.6 g/100 ml (albumin, 4.2 g/100 ml); BUN, 9 mg/100 ml; Na, 138 mEq/l; K, 3.9 mEq/l; Cl, 100 mEq/l; CO₂, 26 mEq/l; blood pressure, 95/60 torr; heart rate, 66 beats/min; weight, 54 kg and height, 171 cm. Metastatic evaluation revealed no evidence of tumor dissemination.

The patient was taken to the operating room for exploratory laparotomy and possible total pelvic exenteration with bilateral groin dissections on October 7, 1969. Premedication was atropine, 0.4 mg intramuscularly, at 7:30 AM. Anesthesia was induced at 8:15 AM with nitrous oxide-oxygen (3:2) plus methoxyflurane via a Pentac vaporizer. The trachea was intubated with the aid of succinylcholine and preparation for operation, including wrapping both legs to mid-thigh with ace bandages, was begun. Arterial and central venous pressures (left radial artery and external jugular vein) and electrocardiogram were monitored and recorded on a Gilson polygraph. Nasopharyngeal temperature and expired tidal volume were monitored. Blood loss was determined by measurement of suction loss in calibrated bottles and that in sponges by a Crouse-Hinds blood-loss machine.

The patient was placed 35 degrees head-down at 9:15 AM. The initial incision, at 9:30 AM resulted in movement. Nitrous oxide-oxygen (3:2) and methoxyflurane, at the 0.1 setting on the Pentac vaporizer, were continued. Blood pressure increased from 90/65 to 110/85 torr. At the time of incision the arterial methoxyflurane concentration was equivalent to an alveolar concentration of 0.06 per cent, Pao₂, 160 torr, Paco₂, 30 torr (all corrected to patient temperature); BE, −3; tidal volume 950 ml (controlled respiration via a Bird respirator at 6 breaths/min); nasopharyngeal temperature 35 C; arterial microhematocrit 29 per cent. No evidence of tumor dissemination outside the confines of the pelvis was found. The large size and position of the primary tumor made total pelvic exenteration mandatory for cure. Be-
cause of the low hematocrit and the decision to proceed with radical operation, the first unit of A.C.D. blood was begun, via a blood warmer. At this time blood pressure was 105/75 torr; heart rate, 90 beats/min; central venous pressure, 15 torr. *d*-tubocurarine, 21 mg, was administered at 10:00 AM. At this time the arterial methoxyflurane concentration was equivalent to a 0.057 per cent alveolar concentration. After 2 units of A.C.D. blood (11:00 AM) the measured blood loss was 300 ml and arterial microhematocrit, 36 per cent. Blood pressure was 110/75 torr; heart rate, 90 beats/min, and central venous pressure 18 torr. Over the next 75 minutes (11:00-12:15) the blood loss increased, requiring 4 units of A.C.D. blood and 500 ml of plasmenate. At one point, during vigorous blood loss, the blood pressure decreased to 95/70 torr and central venous pressure to 10 torr. At 12:15 PM the blood pressure was 105/65 torr; heart rate, 75 beats/min; central venous pressure, 17 torr; hematocrit, 30 per cent. Measured blood loss was 3,000 ml and replacement consisted of 7 units of A.C.D. blood, 500 ml of plasmenate, and 1,200 ml of lactated Ringer's solution. Spontaneous respiration had returned, so an additional 21 mg of *d*-tubocurarine was administered at 12:45 PM. At this time secretions were heard via the esophageal stethoscope. Earlier breath sounds had been faint, but no rhonchi were noted. Tracheal suction was productive of 5-10 ml of thick yellow material. Yellow secretions continued and became profuse and watery, requiring suctioning every 3 to 5 minutes for the duration of the operation. At 1:00 PM *P*<sub>ao</sub> was 70 torr; *P*<sub>aco</sub>, 42 torr; *pH* 7.33; BE, -2; nasopharyngeal temperature, 34.5 C; hematocrit, 46 per cent. Measured blood loss was now 3,500 ml, with no further replacement since 12:15 PM. Blood pressure was 105/75 torr; heart rate, 84 beats/min; central venous pressure, 16 torr. The inspired gas was changed to 100 per cent oxygen and the tidal volume was increased to 1,000 ml. The tidal volume had fallen to 600 ml at 6 breaths/min. The patient was placed flat, with a decrease in the central venous pressure from 15 to 8 torr and no change in blood pressure from 105/75 torr. Both ureters had been placed in an ileal pouch so the output could not be measured, but the surgeons believed that urinary flow was minimal. After 30 minutes of 100 per cent oxygen *P*<sub>ao</sub> was 385 torr; *P*<sub>aco</sub>, 42 torr; *pH* 7.37; BE, -1. *d*-Tubocurarine was reversed with 5 mg prostigmine and 2 mg atropine, with no change in the vital signs. The total pelvic exenteration and bilateral groin dissections were completed at 2:00 PM, at which time the blood pressure was 110/75 torr; heart rate, 84 beats /min; central venous pressure, 7 torr. Coincident with the return of spontaneous respiration central venous pressure decreased to about 2 torr and the tracheal aspirate decreased to a very small volume. At this time arterial methoxyflurane was equivalent to 0.08 per cent. The patient awakened promptly and was taken to the intensive care unit, where respiration was assisted via an orotracheal tube. No secretions could be aspirated from the orotracheal tube, but auscultation revealed bilateral inspiratory and expiratory rhonchi. Blood pressure was 110/70 torr; heart rate, 110 beats/min; central venous pressure, 4 cm H<sub>2</sub>O; hematocrit, 42 per cent. Portable anteroposterior roentgenograms of the chest revealed bilateral interstitial infiltrates predominant at the bases and the left upper lobe, with no change in heart size. These findings were interpreted as pulmonary edema. Electrocardiogram was normal. At 7:00 PM rhonchi persisted, but the trachea was dry and the patient alert. Urinary output was 80 ml/hr. The patient was extubated and did well.

On postoperative day 1 the chest was clear to auscultation. Total proteins were 5.2 g/100 ml (albumin, 3.3 g/100 ml), and Na, 132 mEq/l. Roentgenogram of the chest revealed bilateral basilar infiltrates slightly improved over those seen the day before. The infiltrates receded gradually and the roentgenogram of the chest was read as clear on the seventh postoperative day. Analysis of tracheal fluid obtained during operation revealed a protein content of 4.8 g/100 ml; Na, 140 mEq/l; *pH* 7-8 by nitrazine paper.

The patient did progressively well for 24 days postoperatively, but then developed a series of unexplained complications, such as anemia, ileus and sepsis. Examination of the bone marrow re-

**Fig. 1.** Normal preoperative posterior-anterior roentgenogram of the chest.
revealed malignant cell invasion. At the same time, on the basis of the operative specimen, the diagnosis was changed from undifferentiated carcinoma of the perineum to alveolar rhabdomyosarcoma. The patient had a progressive downhill course and died on postoperative day 54. Autopsy showed a normal heart and lungs and revealed no evidence of local or metastatic tumor other than replacement of bone marrow by malignant cells.

**DISCUSSION**

Six causes of pulmonary edema during anesthesia and operation have been described by Adriani:

1) Incomplete cardiac emptying due to drugs that depress the myocardium, drugs that cause tachycardia, and drugs that produce severe arrhythmias: None of these mechanisms seems applicable to our patient. Alveolar methoxyflurane concentrations ranged from 0.057 to 0.08 per cent with 60 per cent inspired nitrous oxide. MAC for methoxyflurane is 0.16 per cent, and unpublished data suggest MAC with 60 per cent inspired nitrous oxide is about 0.07 per cent. Though all anesthetics produce some cardiovascular depression, the anesthetic dose in our patient was never excessive. Heart rate and electrocardiogram remained normal during the entire operation.

2) Redistribution of blood from the periphery. No vasoconstrictor drugs were used. Cold may produce peripheral vasconstriction, but 34.5 C is not an unusual degree of hypothermia in prolonged operations.

3) Negative airway pressure: Respiration was controlled throughout and no negative phase was used. Airway obstruction was not experienced until the onset of pulmonary edema.

4) Circulatory overload. Fluid replacement was carefully monitored to replace only lost blood and to provide for third-space loss plus normal daily maintenance. The measured blood loss was 3,500 ml, which did not take into account about 1,000 ml of blood (surgeon's estimate) on the drapes. Calculating fluid replacement in abdominal surgical operations as 7–10 ml/kg/hr, we should have given approximately 2,500 ml of balanced salt solution. Maintenance fluids were calculated as 1.5 ml/kg/hr. Total replacement was 2,000 ml of Ringer's lactate solution, 500 ml of plasmancate, and 7 units of A.C.D. blood.

5) Idiopathic.

6) Injury to alveolar membranes by noxious inhalant or aspirate. No suggestion of either possibility existed in our patient.

Our case had several interesting features. The central venous pressure ranged from 15 to 18 torr during most of the operation. This elevated pressure was attributed to the patient's steep head-down position plus positive-pressure ventilation and anesthesia. The central venous pressure fell to 8 torr when the patient was placed flat and to 2 torr with the return of spontaneous respiration. We speculate that the dependent portions of the lung may have been exposed to excessive hydrostatic pressure, with subsequent transudation of fluid. Paine et al. have shown an obstruction to pulmonary lymphatic drainage with elevated superior vena cavaal pressure. The combination of fluid transudation secondary to the steep head-down position with obstruction to lymphatic flow by the elevated central venous pressure may have left only an alveolar
escape route for the fluid. One would predict such a mechanism would take time to develop; it is only speculative that three to four hours would be sufficient. It should be noted that our patient was placed flat simultaneously with the onset of secretions without improvement, but it is likely that severe pulmonary edema was already established, as evidenced by the high hematocrit (46 per cent).

A single cause of pulmonary edema is rare, and it is probable that a combination of events was responsible in our patient. Indeed, many operations have been performed in this hospital with the patients in similar Trendelenburg position, without incident. However, the more common causes of pulmonary edema (i.e., pre-existing heart disease, anesthetic overdose, arrhythmia, increased blood pressure, tachycardia, hypoxia, negative airway pressure and fluid overload) can be eliminated or minimized in our case. Essentially we are left to consider the presence of elevated central venous pressure, an outpouring of frothy yellow tracheal fluid, and roentgenographic evidence of pulmonary edema, and to conclude that the steep head-down position combined with the elevated central venous pressure may have provoked the development of pulmonary edema.

REFERENCES

Transient Phrenic-nerve Paralysis Following Subclavian Venipuncture

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Percutaneous puncture of the subclavian vein, used in emergencies for monitoring of central venous pressure, recently has been used for the passage of permanent transvenous pacemaker catheters.1 The purpose of this communication is to document a hitherto unreported complication of subclavian venipuncture.

REPORT OF A CASE

A 65-year-old white woman sustained a series of episodes of Stokes-Adams syncope on the day of her admission to the Johannesburg Hospital. The electrocardiogram showed that the rhythm varied between 2:1 and complete A-V block. S-T and T wave changes were indicative of a recent anterior subendocardial myocardial infarction. Serial serum enzyme determination confirmed this diagnosis. The patient was treated by demand pacing with a temporary transvenous electrode passed via an antecubital vein but, in spite of steroids, heart block remained, and after three weeks permanent pacemaking was decided upon.

The skin and deeper tissues were infiltrated with 5 ml of 2 per cent lidocaine 1 inch below the midpoint of the clavicle to a depth of about 1½ inches. The skin was punctured with a Seldinger needle which was directed medially and cephalad towards the superior aspect of the sternoclavicular joint to enter the left subclavian vein. The guide wire passed with ease into the superior vena cava. Further manipulation was abandoned because within a few minutes the patient became dyspneic and cyanotic. Roentgenogram of the chest revealed that the heart and mediastinum

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