2. Carnett JB: Intercostal neuralgia as a cause of abdominal pain and tenderness. Surg Gynecol Obstet 42:625, 1926
3. Ashby EC: Abdominal pain of spinal origin. Ann R Coll Surg Engl 59:242, 1977
4. Hall PN, Lee APB: Rectus nerve entrapment causing abdominal pain. Br J Surg 75:917, 1988
5. Doouss TW, Boas RA: The abdominal cutaneous nerve entrapment syndrome. N Z Med J 81:473–475, 1975
6. Applegate WV: Abdominal cutaneous nerve entrapment syndrome. Surgery 71:118–124, 1972
7. Tung AS, Tenicela R, Gioranetti J: Rectus abdominis nerve entrapment syndrome. JAMA 240:738–739, 1978
8. Gallergos NC, Hobsley M: Recognition and treatment of abdominal wall pain. J R Soc Med 82:344, 1989
9. Mehta M, Ranger I: Persistent abdominal pain. Anaesthesia 26: 330, 1971
10. Ghia JN, Duncan GS, Scott DS, Gregg JM: Therapeutic nerve block for chronic pain. Am Fam Physician 20:74, 1979

Anesthesiology
73:780–782, 1990

Acute Upper Airway Obstruction Due to Arterial Puncture during Percutaneous Central Venous Cannulation of the Subclavian Vein

ANTHONY M. O'LEARY, M.B.B.S., M.R.C.G.P., D.C.H., D.R.C.O.G.*

This case report documents the clinical course following attempted, percutaneous, infraclavicular central venous catheterization of the subclavian vein leading to rapid, extensive arterial hemorrhage and total upper airway obstruction necessitating emergency tracheal intubation.

CASE REPORT

An 85-yr-old white woman (height, 166 cm; weight, 75 kg) was admitted to the coronary care unit with a history of episodes of loss of consciousness and collapse. Examination revealed bradycardia of 30–40 beats per min and signs of left ventricular failure. An ECG confirmed complete heart block, and a chest x-ray showed pulmonary edema.

A diagnosis of Stokes-Adams attack was confirmed, and a decision was made to institute temporary transvenous pacing. Hemoglobin at this time was 9.9 g/dl, and prothrombin time was 16 s (control, 14 s), which was considered evidence that there was no significant clotting abnormality.

A 14-G needle (ID, 1.6 mm; OD, 1.9 mm) was inserted one finger breadth below the midpoint of the clavicle. The needle tip was advanced along the inferior border of the clavicle in the direction of the sternal notch. At all times during advancement of the needle, gentle negative pressure was maintained by slightly withdrawing the plunger to identify entry into a vessel. The procedure was difficult, and after four separate attempts using different infraclavicular entry sites and different depth of needle insertion and direction of needle, the needle entered a vessel; the syringe was removed from the needle hub, and a jet of pulsatile arterial blood was witnessed. The needle was removed, and local pressure was applied for 2 or 3 min. At this point, the suprasternal notch suddenly disappeared, and the soft tissues of the neck rapidly became swollen. This occurred 20 min into the procedure and 5 min after documented arterial puncture.

Intense cyanosis was noted, and there was a marked degree of neck swelling that was very tense and hard. Assisted manual ventilation was attempted with no success, and the patient lost consciousness. At this point 10 min after arterial puncture, total upper airway obstruction supervened, and a decision was made to intubate the trachea. Initially, laryngoscopy revealed a grade 4 view (neither cords nor epiglottis visible), and the soft tissues were of such a hard consistency that they resisted attempts to improve the view with the laryngoscope blade. A grade 3 view (epiglottis visible, cords not visible) was soon achieved, and the trachea was intubated with a standard, cuffed, oral endotracheal tube that passed easily through the vocal cords. Attempts to advance the tube further, however, were associated with considerable resistance, giving the impression that the trachea was either collapsed or angled acutely below the level of the cords. A satisfactory position was eventually obtained, and the lungs were ventilated with ease, after which cyanosis resolved and consciousness returned.

Subsequently, the patient was admitted to the intensive care unit (ICU), and her lungs were mechanically ventilated overnight. Cardiac pacing was achieved after an extremely difficult insertion of a left internal jugular central venous catheter that was due to the marked swelling of the neck. Ten hours later, the patient's hemoglobin concentration had decreased from 9.9–7.7 g/dl, and the results of clotting studies were normal.

Tracheal extubation was achieved uneventfully 4 days after admission. There was no electrocardiographic or enzyme evidence of acute myocardial infarction.

During the patient's first day in the ICU, a number of procedures were performed to accurately document the nature of this complication. A lateral radiograph of the neck (fig. 1) illustrated marked widening of the retropharyngeal space consistent with retropharyngeal hematoma. A chest x-ray demonstrated the absence of significant hemothorax or pneumothorax and also showed tracheal deviation to the left. Computerized axial tomographic (CAT) scans through sections at the level of the sixth and seventh cervical vertebrae, respectively, illustrated a right-sided mass without well-defined boundaries. The first CAT scan (fig. 2) showed the trachea to be relatively central, but a further scan (fig. 3) showed the trachea to be deviated toward the left.

DISCUSSION

Percutaneous central venous catheterization is associated with many potential complications that have been well-documented in the literature. There is little doubt that in this case an artery was lacerated and acute hem-

* Registrar in Anaesthesia.

Received from the Department of Anaesthesia, Derbyshire Royal Infirmary, London Road, Derby, England. Accepted for publication May 6, 1990.

Address reprint requests to Dr. O'Leary: Registrar in Anaesthesia, 1 Parkers Fields, Quorn, Loughborough, Leicestershire, LE12 8EJ, England.

Key words: Anesthetic techniques: central venous catheterization. Complications: airway obstruction.

FIG. 1. Lateral radiograph of neck showing marked widening of the retropharyngeal space 10 h after the acute hemorrhage.

orrhage into the neck produced acute respiratory complications.

The infraclavicular approach to the subclavian vein is associated with arterial puncture (usually the subclavian artery),[1-3] but laceration of the carotid artery has also been described.[4] There are also many documented cases of respiratory complications, such as pneumothorax[4,5] and hemothorax either alone[5] or associated with a laceration of the subclavian vein,[6] occurring using this approach. Laceration of the innominate vein leading to mediastinal hemorrhage also has occurred.[4] However, there is only one documented case of arterial rupture leading to respiratory complications.[7] This case involved rupture of the ascending cervical artery during attempted catheterization of the internal jugular vein in an anticoagulated patient that led to a dissecting neck hematoma, hemothorax, and death.

An additional report describes a patient with a coagulopathy who developed a large hematoma due to attempted cannulation of the internal jugular vein that accumulated over 3 h and eventually caused respiratory obstruction requiring tracheal intubation.[8] In our case, despite normal coagulation, sufficient bleeding into soft tissue occurred to decrease hemoglobin concentration by 2 g/dl.

This case is interesting because acute respiratory obstruction due to sudden upper airway obstruction secondary to arterial puncture has not been described previously in the literature. Further, the subclavian approach is associated with lower respiratory tract complications, such as hemothorax and pneumothorax, and is rarely associated with bleeding around the trachea, which occurs

FIG. 2. CT scan at the level of the sixth cervical vertebra, showing a soft-tissue mass in the right side of the neck 12 h after acute hemorrhage. a = tracheal tube in trachea; b = nasogastric tube; c = central venous catheter (line) and cardiac pacing wire; d = central venous catheter (hub, at skin surface); e = hematoma; f = thyroid cartilage.

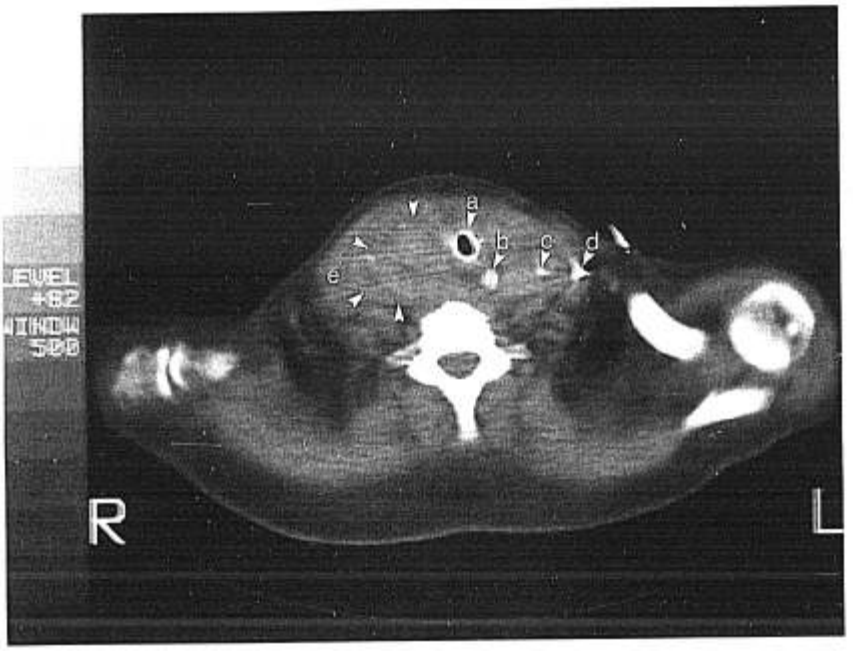

FIG. 3. CT scan at the level of the seventh cervical vertebra, showing a soft-tissue mass in the right side with displacement of the trachea to the left of the midline 12 h after acute hemorrhage. Note the presence of the tracheal tube, nasogastric tube, central venous catheter and cardiac pacing wire. a = tracheal tube in trachea; b = nasogastric tube; c = central venous catheter (line) and cardiac pacing wire; d = central venous catheter (hub, at skin surface); e = hematoma; f = thyroid cartilage.

more often with carotid artery puncture using the internal jugular approach. It is important to point out that when this does occur, a difficult intubation should be anticipated.

It is not possible to determine which artery was responsible for the rapid bleeding. Both the subclavian artery and the ascending cervical artery are vulnerable. However, the size of the hematoma and the rapidity with which it developed would be more consistent with damage to a major vessel, such as the subclavian artery.

It is likely that the hemorrhage escaped from the neurovascular sheath *via* the needle puncture site and extended in a large compartment bounded posteriorly by prevertebral fascia and anteriorly by pretracheal fascia. The absence of any other limiting fascia would allow unobstructed access of blood under pressure to the region of both the trachea and the retropharynx.

Further, it is difficult to be precise as to the level at which obstruction occurred. Supraglottic obstruction due to retropharyngeal swelling is consistent with the difficult laryngoscopy, but tracheal obstruction is consistent with the difficult tracheal intubation and the deviation of the trachea. It may be that the trachea is not occluded on the CAT scans either because of a splinting effect of the tracheal tube or because these scans were performed 12 h after the acute event, during which time some resolution had occurred. In summary, the exact nature and location of the arterial damage and upper airway obstruction are a matter of conjecture.

This case illustrates the potential for rapid onset of severe tracheal obstruction in patients undergoing subclavian central venous cannulation who may even have normal clotting studies.

The author would like to thank the following for their help in the preparation of this case report: Dr. D. A. B. Turner, Consultant Anaesthetist; Dr. D. Chapman, Consultant Anaesthetist; and Mr. R. Nash, Consultant Surgeon. The author is indebted to Miss R. Finney for her secretarial assistance.

REFERENCES

1. Blackett RL, Bakran A, Bradley JA, Halsall A, Hill GL, McMahon MJ: A prospective study of subclavian vein catheters used exclusively for the purpose of intravenous feeding. Br J Surg 65: 393–395, 1978
2. Morgan WW, Harkins GA: Percutaneous introduction of long-term indwelling venous catheters in infants. J Pediatr Surg 7: 538–541, 1972
3. Defalque RJ: Subclavian venepuncture: A review. Anesth Analg 47:677–682, 1968
4. Ryan JA, Abel RM, Abbott WM, Hopkins CC, Chesney T: Catheter complications in total parenteral nutrition. N Engl J Med 290:757–761, 1974
5. Groff DB, Ahmed N: Subclavian vein catheterization in the infant. J Pediatr Surg 9:171–174, 1974
6. Smith BE, Modell JH, Gaub ML, Moya F: Complications of subclavian vein catheterization. Arch Surg 90:228–229, 1965
7. Wisheart JD, Hassan MA, Jackson JW: A complication of percutaneous cannulation of the internal jugular vein. Thorax 27: 496–499, 1972
8. Knoblanche GE: Respiratory obstruction due to haematoma following internal jugular vein cannulation (letter). Anaesth Intensive Care 7:286, 1979