Traumatic rupture of the right subclavian artery

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The number of patients admitted to hospital with major cardiovascular injuries following high-speed motor vehicle accidents is increasing. Injury to the left subclavian artery is well documented but there are only two case reports of injury to the right subclavian artery following blunt trauma without fractures of the clavicle or first rib. In one of the patients the artery was transected distally (Reid, 1967) and ultimately the vessel was ligated through a small cervical incision. The second patient (Matloff and Morton, 1968) presented with multiple injuries, one of which was a grossly ischaemic right arm. He died before surgical correction could be performed.

Our patient presented with multiple injuries and a pulseless right arm. The clinical diagnosis of a right subclavian artery rupture was suspected because a good right carotid pulse was palpable.

CASE REPORT

A man aged 33 years who had been involved in a motor vehicle accident was found on admission to be unconscious and to have the following injuries: a right-sided tension pneumothorax, a flail chest due to bilateral fractured ribs (2 to 6 on the right and 3 to 7 on the left; Fig. 1) and marked paradoxical respiration. An endotracheal tube was immediately inserted, and the right-sided tension pneumothorax was treated by an intercostal tube and underwater drainage. Following this the patient regained consciousness but in view of his flail chest intermittent positive pressure respiration was continued.

The right radial, brachial, and axillary pulses were absent. The right carotid pulse was good and no bruit could be heard over it. All other pulses were synchronous and no bruits were present. There was no evidence of a Horner's syndrome. There was a small ecchymosis over the mid portion of the sternum but no sternal tenderness. Good air entry was present throughout both lung fields. The right arm was colder than the left but the patient could move the fingers and arm well and the sensation was normal.

The anteroposterior chest radiograph revealed re-expansion of the right lung following pleural drainage and a wide mediastinum, and confirmed the rib fractures which had been diagnosed clinically (Fig. 2). Radiographs of the skull and pelvis were normal. During the first three hours his condition remained haemodynamically stable and serial chest films failed to show further widening of the mediastinum.

Three hours after admission, however, a soft ejection systolic murmur was heard at the root of the right carotid and a soft pericardial rub was also noted. Arrangements were made for an aortic arch angiogram but before this could be performed the patient suddenly became cyanosed, restless, and shocked. This necessitated curarization. A chest film at this time (Fig. 3) revealed further widening of the upper mediastinum. An attempt at pericardial aspiration was unsuccessful and in view of his very poor general condition arrangements were made for immediate exploration with a heart-lung machine standby.

The chest was opened through a mid-sternal incision and immediately torrential bleeding occurred through clots in the upper mediastinum. The blood pressure fell dramatically and because the bleeding point was deep and impossible to control through the infiltrated tissues and clots, a cannula was placed in the left external iliac artery and partial heart-lung bypass begun using the open-heart suckers for venous return. Following this the sternal retractor was inserted, the heart exposed by opening the pericardium, and a single venous cannula inserted in the right atrium. On full perfusion the dissection was started in the upper mediastinum, and the bleeding was found to be controlled after cross-clamping the innominate artery. The body temperature was lowered to 28°C and the blood pressure was maintained above 100 mmHg with the heart beating. The sternal incision was extended upwards and to the right in the neck in order to gain access to the carotid and subclavian arteries. The innominate artery was unclamped and another clamp was applied at the origin of the right
subclavian artery in order to perfuse the right carotid. This also had the desired effect of controlling the bleeding. After further dissection it was possible to reapply the clamp distal to the origin of the right vertebral artery, the cerebral circulation thus being completely re-established. The right subclavian artery was found to be completely disrupted immediately beyond the origin of the vertebral and this rupture involved all the distal branches which were ligated. The distal end of the subclavian artery was dissected free and anastomosed to its proximal end. The clamps were removed and the blood flow re-established while the body temperature was brought up to 36°C and the perfusion discontinued. Haemostasis was secured and the chest wall was closed around underwater drains. The patient maintained a good blood pressure and became conscious after warding.

The postoperative course was rather complicated. The patient developed jaundice on the first postoperative day. The serum bilirubin reached its highest level (39% mg) on the fourth day and returned to normal by the 42nd day. The jaundice may be explained by the following facts: Only old citrated blood was available for the emergency procedure; haemolysis occurred during the first part of the perfusion when open-heart suckers were used for venous return; and the considerable amount of blood infiltrating the tissues could not be removed and was subsequently reabsorbed during the days following surgery.

Regular chest physiotherapy was performed and to reduce pain omnopon and fentanyl were given, but after 10 days this was changed to methoxyflurane and it was considered that any further doses of omnopon might cause addiction. A paradoxical segment was still present when the patient was allowed to breathe spontaneously. Before this change tetracycline had been given because Klebsiella organisms had been isolated from the bronchial secretions on several occasions.

On the 15th postoperative day the patient developed evidence of high-output renal failure which was thought to be due to the combined effect of methoxyflurane and tetracycline. Methoxyflurane was discontinued and the patient was allowed large amounts of oral fluid to prevent dehydration. The urine output varied between $5 \frac{1}{2}$ and 6 litres a day.

Seventeen days postoperatively when the median sternotomy wound was healed and the risk of infection reduced a tracheostomy was performed and the

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**FIG. 1.** Chest radiograph on admission showing widened upper mediastinum, right pneumothorax, fractured ribs 2 to 6 on the right and 3 to 7 on the left. This film was taken after the insertion of auffed endotracheal tube.
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FIG. 2. Chest radiograph after insertion of an intercostal drain.

FIG. 3. Chest radiograph taken three hours after admission showing further widening of the superior mediastinum.
nasotracheal tube was replaced with a tracheostomy cannula. Laryngoscopy performed at this time revealed oedematous cords with some erosion, mainly in the posterior third. The flail chest segments became stabilized and between the 27th and 33rd postoperative days the intermittent positive pressure ventilation was gradually discontinued. The subsequent course was uneventful and the patient was discharged from hospital in good condition. The description of the renal problems encountered in this case are reported separately (Proctor and Barton, 1971).

DISCUSSION

Traumatic injuries to the subclavian vessels do not occur as commonly as those to the heart and aorta (Goggin, Thompson, and Jackson, 1970) during high-speed motor vehicle accidents. It appears that the intrathoracic rupture of the right subclavian artery is the rarest traumatic lesion of the great arteries. To our knowledge there are only two cases reported in the literature of intrathoracic rupture of the right subclavian artery without adjacent fractures or penetrating wounds. The injuries to the subclavian arteries reported in the literature are laceration (Fisher and Rienhoff, 1966; Steenburg and Ravitch, 1963; Pate and Wilson, 1964), contusion, thrombosis (Guilfoil and Christiansen, 1967) or aneurysm formation (Fahlsing, Magoon and Sproul, 1964). The injury appears to be due to trauma of adjacent bony structures, such as fracture of the first rib or the clavicle, or as a result of penetrating injuries to the upper chest or cervical region (Jones, Terrell, and Salyer, 1967). However, in our case neither of these causative factors was present. It is, therefore, considered that the subclavian artery was ruptured due to shearing forces produced by rapid deceleration as suggested by Binet, Langlois, Cormier, and de Saint Florent (1962). They reported the successful repair of traumatic avulsion of the innominate artery at its origin from the aortic arch using deep hypothermia and suggested that the mechanism of injury was a crushing force applied to the thorax in an anteroposterior direction which shortens the distance between the sternum and the vertebral column. This pushes the heart and ascending aorta posteriorly and to the left. The curvature of the aortic arch is accentuated and there is increased tension in its convex portion where the major vessels arise. The usual attitude assumed by the patient in these circumstances is one of defence against facial injury. There is hyperextension of the cervical spine and the head is rotated to one side or the other. This manoeuvre places the opposite carotid artery under tension in its long axis. The tearing force occurs at the point of maximum tension, which is at the junction of the carotid artery with the aortic arch. The presence or absence of rib fractures in these circumstances will depend on whether or not the crushing force applied to the chest exceeds the elasticity of the ribs.

The early recognition of traumatic injuries to the aorta and great vessels is essential. Parmley, Matlolly, Manion and Jahnke (1958) estimated that 80% of such cases die at the scene of the accident. Of the 20% who reach hospital alive, 66% are dead within two weeks, 82% within three weeks, and 90% are dead within 10 weeks. This high mortality was partly due to associated injuries such as flail chest, head injuries, long bone fractures, ruptured spleen, and pulmonary contusion and partly to missed diagnosis. Absent pulses in the arm associated with ischaemic changes should alert the clinician to the possibility of disruption of the subclavian artery and when other necessary life-saving procedures have been performed the appropriate area should be examined radiologically. The presence of a good right-sided carotid pulse suggests that the innominate artery is not involved. This may be elucidated by aortography. Serial chest films are most helpful in that they may show widening of the upper mediastinum which warrants immediate angiography. Our patient and Matlolly and Morton's (1968) deteriorated so rapidly that surgery had to be performed without waiting for precise angiographic confirmation of the site of disruption of the vessel. Without this precise information and in the presence of a widening mediastinum radiologically it is essential to have the facilities for heart-lung bypass available.

In view of the high mortality in these patients, even when they have reached hospital alive, we consider that the indications for angiography should be extended if a missed diagnosis is to be avoided. DeMeules, Cramer, and Perry (1971) regard the following as indications for angiography:

1. evidence of a widened superior mediastinum;
2. fractured sternum;
3. multiple rib fractures with crushed chest;
4. first rib fracture;
5. posteriorly displaced clavicular fracture;
6. peripheral pulse deficit involving branches of the aortic arch;
7. unexplained hypotension following inadequate blood replacement;
8. selected patients with massive haemothorax or continued bleeding at a rapid rate from chest tubes.

In the series of DeMeules et al. (1971), 50% of immediate post-injury chest films failed to show mediastinal widening. Angiography should be per-
formed before thoracotomy because 20% of patients have multiple aortic lacerations which may be undetectable on operative exploration alone (Blazek, 1965).

A median sternotomy provided adequate exposure in our patient. The lower transverse cervical incision combined with downward splitting of the manubrium sterni, as described by Shumacker (1948), Elkin (1945), and Steenburg and Ravitch (1963), would not have been practical in our case. If the injury involves the subclavian artery without mediastinal widening then access through a lower transverse cervical incision may be used for exploration and repair. At the time of thoracotomy the systemic blood pressure fell to 20 mmHg for a short period before heart-lung bypass was begun. It was not possible to find the site of bleeding during the control of much blood clot and contused tissue had been removed and all the great arteries had been dissected out beginning at the aortic root. Hypothermia was used for cerebral protection during the clamping of the right innominate artery and the subclavian artery end-to-end anastomosis.

Postoperatively nasotracheal intubation was maintained for a longer period than usual because it was feared that if a tracheostomy was performed the upper mediastinum might become infected. A tracheostomy was ultimately necessary to maintain positive pressure respiration while the fractured ribs and the flail chest were healing.

CONCLUSION

1. In all patients sustaining severe chest injuries the possibility of trauma to the heart and great vessels should be considered.

2. The indications for angiography must be extended if such injuries are not to be overlooked.

3. The patient may recover initially, suggesting that the injury is not severe. This is presumably due to a large haematoma occurring in a confined space, creating high pressure and thereby temporarily stopping the bleeding. But immediately the pressure is reduced by opening the chest and removing the clots bleeding may re-occur.

4. Median sternotomy is preferred and cardiopulmonary bypass facilities are essential.

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REFERENCES


