BLOOD VESSEL INJURY: FEATURES OF THE PATHOLOGY

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THE pathology of trauma to vessels may be discussed under the heading of injuries with breach of the vessel wall and the less common occlusions of apparently intact vessels. Concomitant injury, such as that of the nerves, will not be considered.

In every series of vascular injuries, various degrees of division of vessels are commoner than occlusions (Morris, Creech and DeBailey, 1957; Neely, Hardy and Artz, 1961). The former give the following frequency of lesions in their series:

Laceration—36, 3 with subintimal dissection

Transection—71.

Contusion—6, 4 with thrombosis

Spasm—3.

Lacerations and Transections

The most common cause of penetrating vessel injury in America is wounding by knife or gunshot, less commonly by a fracture fragment, or by the surgeon's rongeur or curette (as when operating on a herniated intervertebral disc.) Blunt trauma may also rupture arteries or veins, especially in the abdomen or chest (McBurney and Gegan, 1962; Shuck and Trump, 1961). Special interest focuses on the rupture of the thoracic aorta in car accidents. The rupture occurs at the junction of the arch and the descending part. It has been attributed to contusion of the aorta, downward acceleration of the heart and arch, or to forward acceleration of the descending aorta (Zehnder, 1956). With large tears, death is immediate; smaller tears lead to aneurysm formation. Surprisingly, these aneurysms may remain unruptured for years, as in five cases recently reported by Steinberg (1957). Primary operative injury to vessels needs no comment, but secondary erosion of a major artery by infection is reported by Brunschwig and Brockunier (1960) as the cause of iliac artery rupture in a group of patients

after gynaecological procedures. Extensive dissection for malignancy and previous irradiation were additional factors in these patients.

Hæmostasis after a vessel is opened depends upon three mechanisms (Quick, 1942). Contraction of the vessel is most significant in the case of arteries, especially after complete transection. An incomplete laceration may continue to bleed because contraction of the arterial muscle tends to open the lips of the wound. The other two mechanisms are those of cellular aggregation, especially platelets, and of fibrinous clot formation. All three processes become more effective after hæmorrhage has caused some hypotension. Hæmorrhage thus usually ceases spontaneously in wounds of even the largest veins, until operative manipulation releases the pressure of the perivascular hæmatoma (Starzl, Kaupp, Beheler and Freeark, 1962). It should be noted that embolism into the jugular or subclavian veins is favoured if the patient is placed in the sitting position and the local venous pressure is thus lowered.

Aside from immediate hæmorrhage, a significant result of arterial laceration is the formation of an aneurysm or fistula. Trauma rarely produces a true aneurysm, but it may cause rupture or thrombosis of an existing one. This sequence is well known in popliteal arteriosclerotic aneurysms, following sharp knee flexion as in squatting. False aneurysms or pulsating hæmatomas are very common after incomplete arterial division. A systolic murmur is characteristic. Severe hæmorrhage is a constant threat and the lesion, when recognized, should be dealt with at once.

A side-by-side injury of an artery and vein produces an arteriovenous fistula. Usually the decompression offered by the low pressure of the vein obviates the danger of hæmorrhage, although rarely an intervening dissecting hæmatoma may exist. Small arteriovenous fistulæ have closed spontaneously, generally by thrombosis, in months or years (Takaro and Scott, 1960), but as Schumacher (in Elkin and DeBailey, 1956) observes, this happens so infrequently as to make this a factor of little significance in planning treatment.

The tendency for arteriovenous fistulæ to induce cardiac enlargement and failure depends upon the size of the communication and the proximity of the lesion to the heart. Thus large fistulæ between the abdominal aorta and inferior vena cava produce early heart failure (Hufnagel and Conrad, 1962). Janes and Jennings (1962) have reported on a group of young patients in each

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whom a fistula, one half-inch in length, was produced in the femoral artery and vein, in order to stimulate growth of a short limb. These fistulae did produce some cardiac enlargement, but no instances of heart failure were observed over the several years between the making and the surgical closure of the fistulae. Occasionally, infection may be established on the interior of the fistula with the production of a subacute bacterial endocarditis. As with the similar process in cases of patent ductus arteriosus, cure is usually effected after excision of the fistula, unless the heart valves are also involved (Parmley, Orbison, Hughes and Mattingley, 1954).

The degree of ischaemia resulting from blood vessel injury depends on many factors and will be discussed later.

Non-Penetrating Occlusions

Blunt injury, such as fracture and dislocation at the elbow or knee, is the commonest cause of traumatic occlusion of apparently intact vessels (Hoover, 1961; Makins, 1919). Vascular spasm, internal parietal dissection and thrombosis are three of the important mechanisms of this occlusion (Gryska, 1962). More insidious injury, as by infection, irradiation (Wolbach, 1909), or chemical irritation, causes both inflammation and intimal proliferation. The latter is also a stage in healing after mechanical trauma (Valls-Serra, 1951) as well, so that partial or total occlusion may develop slowly in a vessel not occluded early after injury. There is little opportunity for recanalization of a post-traumatic thrombosis, since injury also leads to fibrosis and contraction of the vessel.

That local spasm of an injured vessel is myogenic in origin and thus unrelieved by nerve block, but relieved by the local application of papaverine, is well documented by Kinmonth, Hadfield, Connolly, Lee and Amaroso (1956). There may be added to this a spasm of the distal arterial tree, often highly significant in the upper extremity, which is under nervous control and amenable to nerve block. However, spasm alone is rare in an artery, and experience dictates that a vessel in apparent spasm which cannot be adequately relieved should be explored, since thrombosis is usually present (Gryska, 1962; Hoover, 1961; Morris and others, 1957). More rarely, an intimal rupture, with subintimal haematoma, may cause obstruction in the contused vessel, as it may in vessels which have also been lacerated externally.

Certain special situations deserve mention. Ballistic wounds produce blast effects over wide areas, and arteries thus contused may show long thrombi (Makins, 1919). Fibrosis may later develop far beyond an apparently localized wound. Jahnke (1958), in a long-term study of repaired war injuries of arteries, found a high incidence of late fibrosis and thrombosis, and was of the opinion that the initial seemingly adequate debridement should have been more extensive.

FIG. 1.—Post-amputation arteriogram after high tension electrical burn of the forearm in a boy of 14. The soft tissues of the forearm were extensively destroyed, with thrombosis of both the radial and ulnar arteries. Thrombosis did not extend beyond the wrist. The limit of this process is indicated for the ulnar artery and its branches ('Thromb'). Some of the arteriographic medium is extravasated ('Extrav').

The intravascular injection of chemical irritants may produce thrombosis. This occurs frequently after intravenous therapy and is usually harmless. Intra-arterial injection is more serious. Gangrene of a limb has occurred after intra-arterial transfusion and is frequent after inadvertent injection of pentothal (Stone and Donnelly, 1961).

Burns of thermal or electrical origin cause extensive local thrombi. In the case of high tension contact, the current is said to pass for great distances along the main vessels, with resultant extensive thrombosis and gangrene (Muir, 1958). Fig. 1, however, shows that the

FIG. 2.—Effect of femoral venous occlusion on flow in the companion artery in a dog, under nembutal anaesthesia. The attenuation ('atten.') of venous pressure recording was modified during the experiment.

As venous pressure rose, arterial flow fell transiently, but returned to its original level at 25 minutes. Flow increased after venous release, but this may have been due to an increasing general arterial pressure.

thrombosis need not extend to the very end of the vascular tree. Frostbite readily produces major venous or arterial thrombosis, especially in the presence of pre-existing arterial disease.

Variations in Ischaemic Effect

The degree of ischaemia produced by occlusion or division of an artery depends first of all upon the degree to which the artery is normally endowed with collaterals. Thus, injuries to the popliteal artery show a greater incidence of gangrene than do injuries to the femoral artery (Edwards, 1958). Concomitant injury to collaterals obviously adds to the ischaemia.

It is well appreciated that effective collaterals are lacking for the supply of most viscera. The same is true for some portions of skin, the nerves and the muscles of the limbs (Edwards, 1954). Some individual muscles are notoriously limited in their supply and may become ischaemic or necrotic, quite out of proportion to the state of the remainder of the limb (Edwards, 1953).

The rectus femoris, for example, may become entirely necrotic on interruption of its single supply from the lateral femoral circumflex artery. The gastrocnemius is vulnerable because of the single artery to each of its two heads. The biceps humeri likewise may have but one artery supplying it, although in most subjects it possesses up to eight arteries. Occlusion of the anterior tibial artery may cause necrosis of the entire contents of the anterior tibial compartment.

Theoretically, collaterals may exist in sufficient number so as to re-establish the original flow, but as the accompanying Table 1 (taken from a work on plumbing engineering) illustrates, these small vessels must be exceptionally numerous indeed to attain such a happy result.

Flow is, of course, also directly related to pressure, and inversely related to viscosity. Adequate restoration of blood volume and pressure is thus of great importance in restoring blood flow, while the judicious use of saline or low-molecular weight dextran may assist flow considerably.

TABLE 1.—The number of ½-inch pipes that will discharge as much as a single pipe of other sizes for the same pressure loss. (From Babbitt, H. E., Plumbing. McGraw-Hill Book Company, New York, 1960).

Size of pipe, in.	½	1	2	4	8	10
Number of ½-in. pipes with same capacity	1	6.2	37.8	189	1,200	2,090

Subfascial œdema, alone or associated with hæmorrhage, can produce ischæmic changes in a limb. This is not infrequently the dominant mechanism of ischæmia following injury such as supracondylar fracture of the humerus. The occlusion is relieved in these cases by division of the deep fascia.

Muscle is very sensitive to ischæmia and at least partial necrosis follows arterial occlusion lasting four hours or more (Edwards, 1953). Indeed, contracture of muscle may be noted within an hour of ischæmia. This early contracture wears off completely if blood flow is quickly restored. If high grade ischæmia continues, autolysis then produces a flail limb within 24 hours. Lesser degrees of unrelieved ischæmia go on to the fibrotic contracture of Volkmann (Griffiths, 1940).

Since World War I, there has been a good deal of conflicting testimony regarding the effect of venous ligation for concomitant arterial occlusion. Since flow is directly related to differential pressure, any increase in resistance to the outflow of an *in vitro* system lowers flow, but even the more recent physiologic experiments, with the better instrumentation now available, do not always show this result in the living animal. The factor operating to help maintain the flow is the increased transmural pressure which produces passive vasodilatation of the vascular tree and a consequent reduction in resistance to flow (McDonald, 1960; Read, Kuida and Johnson, 1958).

The results in the dog, as observed in our laboratory, have varied somewhat. An immediate diminution in arterial flow was constant, but this could be quite transient and succeeded by a substantial rise, while the venous pressure, although diminishing, was still far above its initial level (Figs. 2 and 3). When the inflow arterial pressure was very low, no such secondary rise in arterial flow was observed throughout the hour or two of duration of our experiments, and in one case thrombosis of a narrowed arterial lumen resulted (Fig. 4). One clinical counterpart of these experiments is arteriosclerosis, where venous ligation or thrombosis may diminish flow sufficiently to precipitate gangrene.

FIG. 3.—Effect of femoral venous occlusion on arterial flow in another dog. While the flow vacillated somewhat, there was a tendency to a considerable increase during the venous occlusion.

FIG. 4.—Effect of femoral venous occlusion on arterial flow when stenosis is present in the artery. Flow falls with the venous occlusion, and thrombosis of the stenotic lumen finally occurs.

To return to arterial injury, it would seem from our own observations and the work of others that there is no adequate justification for adding venous insult to arterial injury, especially as reconstructive surgery is so well established that the new point of discussion is whether to reconstruct the vein as well as the artery, when both are injured.

Summary and Conclusions

The most common cause of penetrating vessel injury is wounding by knife or gun-shot, although blunt trauma may also rupture major vessels. Spontaneous hæmostasis more readily follows complete, than incomplete, transection. Incomplete laceration may be followed by dissecting hæmatoma, aneurysm or arteriovenous fistula.

Blunt injury, as in fractures, is the commonest cause of traumatic vascular occlusion, which may also follow repeated lesser trauma, irritation of a vessel by infection, irradiation, or the accidental injection of chemical irritants. Electrical burns cause thrombosis which does not necessarily extend to the acral arterial tree.

Variation in ischæmic effect depends at the outset upon the excellence of collateral endowment. Not only the viscera, but also other tissues, notably the muscles, may anatomically lack adequate collaterals, and may thus be subject to total necrosis after relatively localized vascular interruption.

Experiment also demonstrates that local venous obstruction produces a fall in arterial flow which is transient unless a general or local arterial hypotension exists. In other instances the fall may be followed by a moderate increase. This has been ascribed to passive vasodilatation of the vascular tree and a consequent reduction in peripheral resistance.

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