Importance of Venous Occlusion in Arterial Repair Failure: An Experimental Study

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ARTERIAL injuries of the femoral and popliteal arteries are repaired successfully in the majority of instances. Prolonged ischemia, tissue destruction, technical error, instability and sepsis are all well-recognized causes of either repair failure or loss of an extremity despite successful restoration of arterial patency. Another potential cause for loss of an injured extremity is inadequate deep venous return. Numerous clinical impressions from the conflicts in Korea [6, 7, 15] and Vietnam [2, 5, 12, 14] indicate that venostasis alone may jeopardize an extremity even when distal pulses and arterial patency have been restored and the aforementioned harmful conditions are absent.

Although vein repair has been advised whenever possible,[2, 5, 6, 12,15] ligation is more commonly done.[8, 15] This problem was addressed again as late as 1967, when Chandler and Knapp [1] in a report of 126 arterial repairs in Vietnam added, "Accurate assessment of the relative hazards of ligating or repairing concomitant venous injury is beyond the competence of this study but it is urgently needed." The answer to this question is of particular importance in the lower extremity since accompanying veins are injured in approximately 60% of arterial injuries.[1, 7, 12]

While failure of limb salvage was attributed to venostasis alone, it seemed possible that venous blockage might also decrease arterial flow sufficient to cause arterial repair thrombosis or dangerously poor perfusion despite a patent repair.

Review of the English language medical literature revealed one recent experimental study [16] designed to simulate phlegmasia cerulea dolens in which total extremity venous occlusion resulted in cessation of femoral arterial flow in dogs. By contrast, our experimental study was designed to test femoral arterial flow with only concomitant vein occlusion and with collateral flow intact. The results of the present study are strikingly in favor of repairing concomitant venous injury whenever possible since uncomplicated occlusion of the accompanying vein results in a prompt and continuing arterial low-flow state.

Methods and Results

Ten mongrel dogs weighing 14 to 17 Kg. were anesthetized with a halothane-oxygen mixture administered endotracheally. Aortic blood pressure was monitored continuously

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In conducting the research described in this report, the investigations adhered to the "Guide for Laboratory Animal Facilities and Care," as promulgated by the committee on the Guide for Laboratory Animal Facilities and Care of the Institute of Laboratory Animal Resources, National Academy of Sciences–National Research Council.

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TABLE 1. *Effect of Venous Occlusion on Arterial Flow*

Incident	Arterial Blood Flow* (ml./min.)	(Per Cent of Baseline)
Baseline	53.4	(100)
Venous occlusion		
Immediate effect	17.6	(33)
After 2 hours	28.0	(52)
Release after 2 hour-occlusion		
Immediate effect	41.8	(78)
After 10 minutes	45.6	(85)

* Mean values from 10 dogs; Blood pressures remained constant.

by means of a catheter placed in the contralateral femoral artery.

The selected common femoral vessels were exposed in the groin and baseline measurements of arterial flow were recorded with a square wave electromagnetic flow meter.[18]* Arterial flow rates (ml./min.) were then measured during and after periods of concomitant vein occlusion produced with a non-traumatic tube tourniquet. Observations were made several times to establish mean values for each animal. Table 1 shows mean values for all test animals.

Arterial flow decreased immediately and stabilized at 33% of baseline flow during a 2-minute period of venous occlusion. Release of obstruction was followed by prompt return of arterial flow to baseline levels.

When venous occlusion was applied for 2 hours, the initial response was the same but during the occlusive period, arterial flow gradually rose from 33% to 52% of baseline values. Improvement in flow suggested that collateral venous pathways were opening. Upon release of venous occlusion after 2 hours, arterial flow rose immediately to 78%, and 10 minutes later was 85% of baseline flow. These results were consistently reproducible in all test animals. A representative tracing is shown in Figure 1. Distal arterial pressure was not measured but peripheral pulses remained unchanged to palpation throughout. No changes suggestive of ischemia or venostasis were observed and all animals had immediate full use of their extremities indicating that the circulatory alterations were reversible under the conditions of this experiment.

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Discussion

The consistency and direction of even this relatively small number of observations is considered conclusive that significant reduction of arterial flow is produced by concomitant venous occlusion alone. While the 2-hour period of occlusion produced no clinical effect in the normal canine limb, it seems entirely possible that an arterial flow rate of one-half normal would eventually become clinically significant in a traumatized extremity with arterial suture line. Additional studies are now in progress to determine the duration and degree of reduced arterial flow necessary to cause arterial repair failure. Support for these conclusions is provided by the work of Stallworth *et al.*,[16] in which the canine hind limb was totally occluded by tourniquet with the femoral vessels excluded. When the femoral vein was occluded also, arterial flow dropped immediately to zero, while distal pulses and arterial pressure remained normal. After 6 hours of occlusion, changes similar to those of phlegmasia cerulea dolens occurred. After 12 hours distal pulses and pressure usually disappeared presumably due to arterial thrombosis progressing from smaller to larger arteries. After 24 hours one third of animals died, 60% had major arterial thrombosis and in no case could arterial flow be re-established. While their study was related primarily to clinical deep venous thrombosis, the results appear relevant to combined arterial and venous trauma.

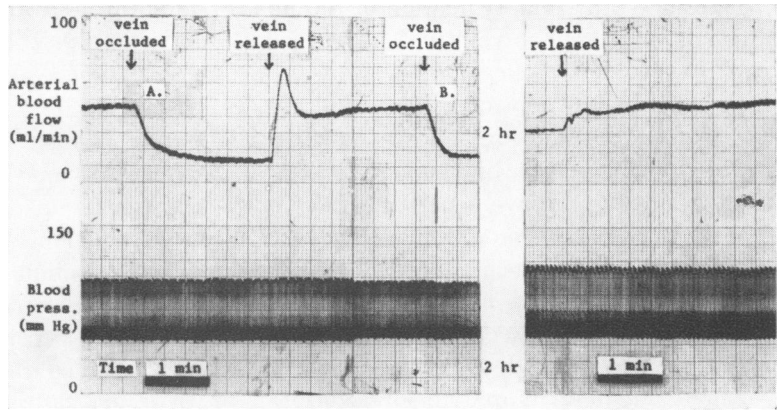

FIG. 1. Representative tracing from one animal demonstrating the effect of femoral vein occlusion on concomitant arterial flow. Venous occlusion (A) for 2 minutes reduced arterial flow from 46 ml./min. to 13 m./min. Upon release of occlusion, arterial flow returned to the baseline level within 30 seconds after a brief "overshoot." A second occlusion (B) produced essentially the same initial effect. During a 2-hour period of venous occlusion arterial flow gradually rose from 16 ml./min. to 32 ml./min. and upon release, flow returned to the baseline level within minutes.

We have recently confirmed the findings of Stallworth *et al.*[16] in our laboratory. Total venous occlusion of the canine hind limb resulted in essentially negligible femoral arterial flow. During four hours of observation, distal pulses were unchanged and tibial artery blood pressure remained similar to aortic blood pressure. The leg markedly increased in size and became ecchymotic with superficial bullae formation.

From these two studies, a mechanism by which venous blockage adversely affects an injured extremity can be inferred. Venous obstruction causes venous hypertension and venostasis which in turn cause increased tissue resistance to inflow. The resultant arterial low-flow state increases the tendency both for arterial repair thrombosis and for tissue death from poor perfusion even in the presence of a patent repair. Even modest venous occlusion, as typified by our study, decreases arterial flow significantly and consistently. Total venous occlusion [16] causes cessation of arterial flow and, as might be expected, progressively leads to arterial thrombosis and death of the limb from gangrene. Most clinical cases of combined vascular trauma will be found between these two extremes ranging from a clean low-velocity wound in which the artery is repaired and the vein ligated to a high-velocity war wound in which severe impairment of collateral venous flow is added. Perhaps the most important consideration is that the presence of peripheral pulses, which the clinician relies heavily upon to signify a successful repair, is not necessarily indicative of the true status of arterial flow and perfusion. Our study suggests also that obvious signs of venous obstruction are not required for the described mechanism to produce an arterial low-flow state.

A brief review of past thinking regarding management of the concomitant vein in arterial injury brings out the types of clinical problems for which the results of these experiments might prove meaningful.

Prior to World War I, when ligation was the accepted method for treating major extremity arterial injuries. Matas [10] stated that the danger of peripheral gangrene was doubly increased by simultaneous injury to the accompanying vein. However, during that conflict Makins [9] developed the opposite view and recommended ligation of the concomitant vein, even when uninjured, along with the artery, because of his belief that collateral arterial circulation would be better utilized and the incidence of gangrene would be lessened. His views were largely refuted in 1932 by Montgomery [11] in an experiment similar to the present one.

During World War II ligation of the uninvolved concomitant vein was not widely employed and DeBakey and Simeone [6] in their extensive review concluded that this practice afforded no protection whatsoever against development of gangrene. However, Makins' views were perhaps, in part, responsible for a continuing belief that ligation of injured concomitant veins, while not helpful, would do no harm.

During the Korean conflict, when for the first time a concerted effort was made to repair all acutely injured major arteries,[17] Hughes [6] and Spencer [15] independently noted that in certain cases gangrene was attributable to venous insufficiency and venostasis. Amputation was required even with "good arterial inflow" [6] and "the arterial tree was demonstrated to be patent in the amputated extremity." [15] Both authors found no complications following venous repair and recommended repair whenever possible.

In the Vietnam conflict even greater recognition has been given to the importance of concomitant venous repair whenever feasible, especially in the popliteal and distal femoral regions.[2, 12, 14] Approximately one third of lower extremity vein injuries are being repaired, including the use of vein grafts, which represents a significant increase in the use of repair as compared to previous experience. If possible, vein grafts for either artery or vein repair are obtained from the opposite saphenous or cephalic vein to preserve maximum venous flow in the injured limb. The short-term advantages of venous repair are avoidance of acute venostasis and improvement of venous return even if restoration is only temporary because of subsequent thrombosis.[2] The long-range goals are to provide a pathway for later recanalization if thrombosis occurs [3, 14] and to prevent chronic venostasis.[14]

While all these reports emphasize venostasis as contributory to development of gangrene, none has suggested that an associated arterial low-flow state is perhaps an important part of the process leading to repair thrombosis or distal ischemia.

Several clinical situations can be identified in which the previously described mechanism may play an important role. Massive tissue destruction is accompanied by destruction of venous collaterals and if the concomitant vein is also ligated, early loss of the extremity by this mechanism may occur. Sepsis or repeated debridement may produce the same effect. In certain areas such as the popliteal region, venous circulation may be more tenuous and relatively modest venous obstruction may cause critical deficits of arterial flow. In instances where delayed repair is attended by ischemic tissue swelling distally, a similar situation may be produced. The damaged tissue may present an inflow barrier, as with venostasis, resulting in decreased perfusion which becomes an added factor in tissue death.

This same mechanism may be a subtle cause of arterial repair thrombosis which occurs for no obvious reason and without the commonly recognized causes being present. It is possible that moderate venostasis not recognizable clinically may produce decreased arterial flow which over several days may lead to arterial repair thrombosis. It is suggested also that when the low-flow state produced by venous obstruction is added to any of the usual factors associated with failure of an arterial repair, then a successful outcome is less likely.

Summary

In combined arterial and venous trauma of the lower extremity, the choice of repair or ligation of the concomitant vein is encountered frequently. This experimental study shows that in the otherwise normal canine hind limb, uncomplicated occlusion of the concomitant vein produces a prompt and continuing femoral artery low-flow

state ranging from one third to one half of baseline flow measurements. It is suggested that this finding may have clinical importance in a wide range of situations involving combined vascular trauma. When concomitant veins are ligated or damaged beyond repair, the resultant arterial low-flow state may be a significant and previously unrecognized factor in either thrombosis of the arterial repair or in development of gangrene despite an apparently successful repair. It is also suggested that tissue swelling from prior ischemia may exert the same deleterious effect on arterial flow.

This arterial low-flow state is more pronounced when extensive tissue damage blocks collateral venous pathways as demonstrated by the experiments of Stallworth *et al.*,[16] in which total venous occlusion in the canine limb produced immediate cessation of all arterial flow. Of particular clinical interest, peripheral pulses, which the clinician relies heavily upon to signify a successful repair, may remain normally present for extended periods during the low-flow state and are, thus, not indicative of the true status of arterial flow and perfusion.

An increasing amount of clinical evidence from combat experience suggests that venous repair is critical to limb salvage particularly in the popliteal region and should be done whenever feasible even if it prolongs the operation and requires a vein graft. Restoration of venous return becomes even more critical when other causes for arterial repair failure, such as prolonged ischemia, instability due to fractures, technical problems with the arterial repair and sepsis, are present.

The results of this study strongly support clinical observations on the benefits of concomitant vein repair and suggest that decreased arterial flow may be an important clinical result of ligation of the concomitant vein.

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