The Blood Flow through the Calf of the Leg during Acute Occlusion of the Femoral Artery and Vein

By John T. Shepherd, M.D., M.Ch.

The calf blood flow has been studied in the human subject before and during acute occlusion of the common femoral artery and vein. The results suggest that the concomitant venous occlusion has a detrimental effect on the development of the collateral circulation.

Opinion is divided on the effects of concomitant venous ligation on the nutrition of the limb following injury to the main artery. In the present investigation the calf blood flow has been studied in the human subject following acute occlusion of the common femoral artery and vein, and evidence is presented which suggests that the simultaneous venous occlusion has a detrimental effect on the blood flow and nutrition of the limb.

Methods

A mechanical compressor was used to occlude the artery and vein. The maximum combined width of these vessels, measured post mortem, in 17 male subjects was 2.7 cm. The compressor consisted of a hinged horizontal arm fastened at one end to a table; a wooden block 5 cm. by 3.5 cm. surrounded by a 1.5 cm. thick rubber pad was attached to the under surface of the free end of this arm, and this pad was centered as nearly as possible over the artery and vein. A 9.5 Kg. weight, placed directly over the wooden block, was used to occlude the vessels. Blood flows were recorded through the calf of the leg by means of venous occlusion plethysmography before, during and after application of compression. The subjects were all healthy males, age 19 to 30 years.

Results

Resting calf blood flows were recorded and when these became stable the compression was applied to the femoral artery and vein and the calf flow again determined. An immediate decrease in flow to about one-sixth of the resting value occurred on application of compression and this was followed by a rapid recovery, the blood flow returning to approximately its previous resting level in one half to six minutes (average two minutes). The flow remained more or less stable for the next two and one-half to seven and one-half minutes and then decreased throughout the remainder of the period of compression (fig. 1A). Compression of the vessels was not usually maintained for longer than 10 to 11 minutes, as after this time it became painful. This paper deals only with the final decrease in flow which was observed in 13 out of 15 subjects tested.

At this stage it was observed that there was an increase in calf volume when both artery and vein were occluded (fig. 1C) and as the superficial veins were distending during the period of compression, there seemed little doubt that the increase in calf volume was due to a rise in venous pressure. Estimations of venous pressure were made on six subjects. During compression of the femoral vessels the pressure in the long saphenous vein gradually rose to about 50 mm. Hg (fig. 1B). As the collecting pressure used in the recording of the blood flow was 75 mm. Hg, the observed decrease in blood flow was not the result of using too low a collecting pressure.

The rise in venous pressure was due solely to occlusion of the femoral vein and not to obstruction of the collateral venous return by pressure against the couch. This was shown by the following experiment carried out on six of the subjects. Using a compressor with a central fulcrum, it was possible to occlude the artery and vein with the subject in the prone position in order to compare the increase in calf volume

From The Department of Physiology, The Queen’s University of Belfast, Northern Ireland.
with that obtained in the same subject in the supine position. The marked similarity of the volume changes in the two positions made it clear that the increase in calf volume and the rise in venous pressure resulted from the inability of the collateral venous circulation to cope with the collateral arterial inflow when the femoral vein was occluded.

It was now necessary to decide whether the observed decrease in blood flow represented a true decrease, or an apparent one due to a fault in the method of recording. That is, the plethysmographic method, which depends on accommodation of arterial inflow in the vessels of the limb, may have failed to register accurate flows when the veins and other vessels were already partially distended.

If this were a true decrease in blood flow, then a reactive hyperemia should follow the removal of compression and the more marked the decrease the greater should be the resultant hyperemia. The blood flow through the calf after compression of the femoral vessels was therefore measured and compared over a similar period of time with the blood flow through the calf immediately preceding the period of compression. In figure 2 the abscissa represents the calf blood flow after compression expressed as a percentage of the calf blood flow before compression, and the ordinate represents the peak blood flow obtained on removal of compression. It is clear that the lower the percentage the calf flow after compression of the femoral vessels is of the flow before compression, the greater is the resultant hyperemia.

A further analysis made it clear that little if any of this debt was incurred during the initial decrease in calf blood flow immediately after the application of compression, as most
of this had been repaid before the terminal decrease commenced. It seems, therefore, that
the rise in venous pressure which follows the venous occlusion causes a true decrease in the
collateral blood flow to the calf.

Some workers agree that a rise in venous pressure following concomitant venous occlusion
may decrease the blood flow, but they argue that this decrease may be more than
compensated by a more homogeneous distribution of the blood in the limb.2

To see if any compensation for the decrease in flow occurred in the present series a further
test was performed. The weights were applied to the femoral artery and vein and the subject
immediately began to plantar and dorsiflex the foot at the rate of one flexion and extension per
second. For approximately half a minute from the start of the exercise no pain was felt in the
limb. Then a deep seated pain was felt in front of the ankle and occasionally extending upward
for a short distance into the extensor compartment of the leg. As the exercise continued the pain
steadily increased in severity for about one minute. It then became unbearable so that the exercise had to be stopped.
On discontinuing the exercise the pain diminished and completely or almost completely disappeared. Now followed a period of half a
minute to three minutes of complete or almost complete freedom from pain. Finally the pain reappeared at the previous site even though exercise was not recommenced, steadily increased in severity, once again becoming intolerable, at which point compression was removed. Just before pain reappeared venous distension was obvious and cyanosis commenced and gradually increased in intensity (fig. 1D).

These results confirm the previous findings that a true decrease in blood flow occurred after the collateral circulation had been established for a short period. In addition the return of the pain showed that this decrease was not compensated by keeping the blood longer in the tissues or by a more homogeneous distribution of blood in the limb. In order to show that these results were due to compression of the concomitant vein, it was decided to repeat this test, using digital compression, in an endeavor
to occlude the artery only, leaving the vein completely or almost completely patent. This
type of compression was adopted in preference to moving the mechanical compressor more
laterally in an attempt to occlude the artery only, as it was felt that the sensitive digital compression offered a more reliable and con-
stant method of producing a pure arterial occlusion, especially as the subjects chosen for this test had easily palpable femoral arteries.

![Graph A](http://circ.ahajournals.org/)

**Fig. 3.** A. Occlusion of femoral artery and vein. B. Occlusion of femoral artery with only partial occlusion of the femoral vein.

It was found that the onset of the pain, its situation and maximum intensity coincided with the previous tests but that following the cessation of exercise, the pain decreased as before, died away completely and did not return. There was no venous distension or cyanosis (fig. 1E). In addition, in some cases this test was repeated with digital compression of both artery and vein, and results similar to those of mechanical compression were obtained.
Finally it might be argued that the decrease in collateral blood flow was not the result of the rise in venous pressure but was due to reflex vasoconstriction as a result of the pressure of the weights on the femoral vessels. The following observations suggest that reflex vasoconstriction was not responsible.

1. In some of the 13 subjects, who had a small amount of subcutaneous fat in the inguinal region, moving the compression pad further laterally in an attempt to leave part of the femoral vein unoccluded while still occluding the femoral artery resulted in no decrease in blood flow and little or no increase in calf volume (fig. 3).

2. There was no evidence of contralateral reflex vasoconstriction, the flow through the normal circulation in the opposite calf being unchanged during the period of compression.

**DISCUSSION**

All who are in favor of ligating the concomitant vein following injury to main arteries are agreed that the resulting benefit is dependent upon the production of a partial venous obstruction in the limb concerned. The essential question to be answered is whether following arterial occlusion any benefit can be conferred upon the nutrition of the limb by producing a partial venous obstruction.

Holman and Edwards have suggested that the rise in venous pressure following venous ligation would increase the blood flow through the limb. This has been repudiated by Brooks, Wilson and others. In the present investigation it was found that the extent of the venous obstruction following the occlusion of the femoral vein near its termination led to a marked decrease in collateral arterial inflow in the 5 to 10 minute period following the occlusion.

It could be argued that the rise in venous pressure, while decreasing the blood flow, may be more than compensated by permitting increased interchange of nutritional substances between blood and tissues, first as a result of blood remaining longer in contact with the tissues and second on account of the action of the increased intravascular pressure keeping more capillaries open, and thus facilitating nutritious exchange. The decrease in flow in the present experiment did not appear to be compensated for in this way, as evinced by the return of pain in the exercise test, when the artery and vein were both compressed.

**SUMMARY**

1. The calf blood flow has been studied in the human subject for 10 minutes during acute occlusion of the common femoral artery and vein.

2. After an initial decrease the calf blood flow rapidly returns to approximately the level present before occlusion, and then decreases again. This latter decrease is due to the rise in venous pressure which results from the occlusion of the femoral vein.

3. The decrease in calf flow due to the rise in venous pressure is not compensated by other changes accompanying the increased venous pressure.

**ACKNOWLEDGMENTS**

I wish to express my thanks to Professor Henry Barcroft for his advice and helpfulness throughout this work; and to those members of the Belfast Medical Students Association who willingly cooperated in the experiments. I would also thank the Medical Research Council for a grant for laboratory assistance.

**REFERENCES**


Ligation of the external iliac artery and vein above and below a communicating wound of these two vessels. Ann. Surg. 73: 265, 1921.


The Blood Flow through the Calf of the Leg during Acute Occlusion of the Femoral Artery and Vein

JOHN T. SHEPHERD

Circulation. 1952;6:281-285
doi: 10.1161/01.CIR.6.2.281

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1952 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/6/2/281

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/