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PNEUMATIC TOURNIQUET AND LIMB BLOOD FLOW

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The response of limb circulation to tourniquet ischaemia in the hind limb of the rabbit was studied. Muscle blood flow was evaluated by means of a local isotope technique using Xe^{133} as the tracer and the changes occurring after tourniquet ischaemia of 60–180 minutes duration were investigated. The results obtained suggest that even when the tourniquet time is extended to 3 hours, no blood flow occurs in the limb distal to the tourniquet when the cuff is inflated to 300 mmHg. In all animals a hyperaemic reaction was noticed after releasing the tourniquet, and a peak reactive hyperaemia was registered 1 minute after the return of circulation. The magnitude of the reactive hyperaemia was independent of the length of tourniquet time. The hyperaemia was regularly of short duration and peak flow values were reached in 1 minute with the flow returning to normal or subnormal values after 5 minutes. Phlebography studies in 12 rabbits after 120–240 minute tourniquet blockade showed only one thrombosis in a deep popliteal vein after the 240-minute tourniquet time.

Key words: pneumatic tourniquet; limb; blood flow; ischaemia; thrombosis

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Pneumatic tourniquet is a commonly used technique for the exsanguination of a limb and obtaining a bloodless field for surgery of the extremities (Esmarch 1873, Cushing 1904). Some authors suggest that despite correct application of the tourniquet blood may flow into the distal part of the limb by the way of the medullary canal (Spira et al. 1965, Furlow 1971), the amount varying from 1 to 26 per cent of the flow to the unoperated limb. Klenerman & Crawley (1977) reported, however, in an experimental study, that the circulation was reduced to less than 1 per cent of that of the control limb.

Reddening of the skin of the limb after removal of the tourniquet as a sign of reactive hyperaemia was observed by Bier already in 1897. Even a short occlusion of the arterial blood flow of less than 5 minutes produces reactive hyperaemia (Myhre 1975). However, there is little accurate data on the blood flow to the limb after releasing the tourniquet. Several methods have been used to study reactive hyperaemia, venous occlusion plethysmography being one of the most common. However, this technique may impede the arterial blood flow and, furthermore, the measurement must be made at intervals (Dahn 1965). Estimation of venous outflow by drop recording technique and electromagnetic flowmetry have been successfully used to measure rapid changes in blood flow after brief arterial occlusion (Thulesius 1962, Myhre 1975). Recently, the local isotope clearance technique using Xe^{133} as the tracer has proved to be a convenient method to evaluate muscle blood flow (Lindbjerg 1966, Siggaard-Andersen & Bonde-Petersen 1967). In these experiments the circulatory occlusion was extended to 60 minutes. A reactive hyperaemia was always noted after the return of circulation.

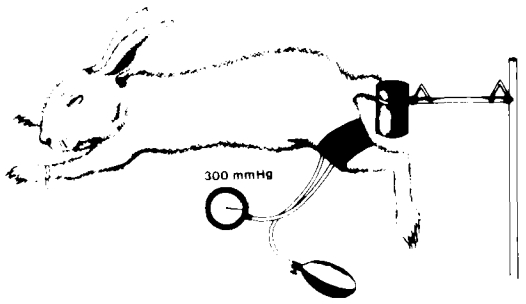

Figure 1. Schematic illustration of the model used in the experiments. The scintillation detector for the Xe^{133} wash out registration was placed 5 cm above the limb.

Postoperative venous thrombosis has been considered as a rare complication after pneumatic tourniquet (Geens et al. 1969, Potter et al. 1972). However, other authors have reported, in phlebographic studies, that the incidence of venous thrombosis after pneumatic tourniquet varies from 10 to 60 per cent (Cohen et al. 1973, Kroese & Stiris 1976). Evidence has been presented that the thrombosis appears already during the first postoperative hour (Flanc et al. 1968, Negus et al. 1968).

The purpose of this study was to clarify the circulatory situation in the limb during the use of the pneumatic tourniquet, to evaluate the blood flow after releasing the tourniquet and to find out if postoperative thrombosis appears as a complication.

MATERIAL AND METHODS

Thirty-one rabbits of both sexes, weight 2.7–3.4 kg, were used in this study. Anaesthesia was induced with intravenous sodium pentobarbitone and maintained by continuous ether inhalation.

The left hind limb was exsanguinated with an Esmarch rubber bandage and tourniquet blockade was applied by means of a 6 cm broad, Riva-Rocci-type cuff around the upper part of the leg, the cuff being inflated to 300 mmHg (Figure 1). The response of different periods of tourniquet blockade on the extremity blood flow was investigated. Six rabbits were subjected to 60 minutes of tourniquet ischaemia, another six to 120 minutes, the remaining six being subjected to 180 minutes of tourniquet ischaemia.

The hind limb blood flow was measured by the $Xenon^{133}$ wash out technique. Into the middle part of the anterior tibialis muscle 0.2 ml of a solution of Xe^{133} was injected in sterile isotonic saline (5 mCi/ml, Rotop®, Isocommerz GmbH, Berlin-Buch, DDR) with a thin needle (No. 16 OD 0.5 mm). The injection was performed for 10 seconds, the needle being withdrawn 20 seconds after finishing the injection. The disappearance rate of the isotope from the limb was followed by means of a scintillation detector with a thallium-activated NaI-crystal, which was placed 5 cm above the limb. The scintillation detector was coupled to a Wallac® ratemeter (Wallac Ltd, Turku, Finland). The output from the ratemeter was recorded on a logarithmic Servogor® chart recorder (Goertz Electro GmbH, Vienna, Austria). On the assumption of diffusion equilibrium of Xe^{133} between tissue and blood, calculation of the muscle blood flow was made according to the Fick principle (Lindbjerg 1966).

Phlebography of the hind leg after pneumatic tourniquet was performed on a further 12 rabbits. Four rabbits had a 120-minute tourniquet ischaemia and in four rabbits the duration of blockade was 180 and 240 minutes, respectively. Radiographic examination was performed within 30 minutes of the release of the tourniquet. To inject the contrast medium, the skin in the lower leg was incised and a catheter was inserted in the distal part of the great saphenous vein. An X-ray was taken also with phlebography tourniquet to visualize the deep veins of the limb.

In one rabbit an angiogram of the common iliac artery and its distal ramifications was performed. After laparatomy, a catheter was introduced through the left renal artery into the aorta. An angiogram was taken during the tourniquet occlusion and 30 minutes after the release of the tourniquet.

RESULTS

The average of 18 determinations of the normal blood flow baseline in the hind limb was 24 ± 8.1 ml/min/100 g muscle (mean and s.e.m.).

During the tourniquet blockade no decrease of Xe^{133} activity could be demonstrated indicating that no wash out occurred. This observation was made in all the animals under investigation and the tourniquet time did not affect the results.

When the tourniquet blockade was released

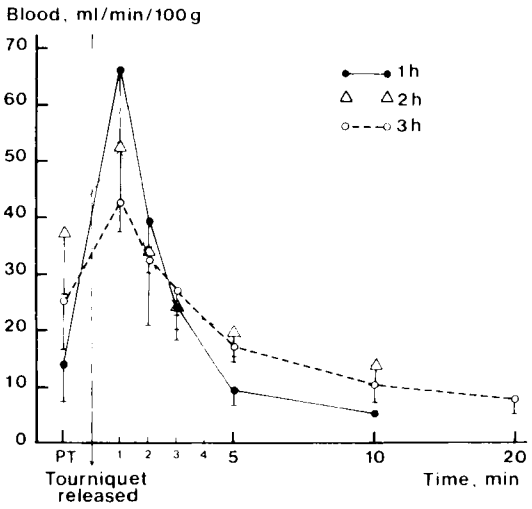

Figure 2. Reactive hyperaemia registered after releasing the tourniquet. Note the peak of the blood flow 1 minute after onset of the circulation. The magnitude of the hyperaemia was not significantly dependent on the tourniquet time. Within 5 to 10 minutes the flow had turned to normal or subnormal values. PT = pretourniquet value.

after 60 minutes, the Xe^{133} wash out increased 1 minute after return of the circulation to 68.8 ± 12.4 ml/min/100 g ($P < 0.05$). After this, the wash out decreased sharply and 5 minutes after releasing the blockade the wash out was lower than the normal baseline (9.2 ± 4.2 ml/min/100 g) (Figure 2).

In the 120-minute group the Xe^{133} wash out increased 1 minute after tourniquet release to 52.1 ± 8.5 ml/min/100 g. Five minutes after the release the value for wash out was 19.2 ± 2.1 ml/min/100 g.

In the 180-minute group 1 minute after release of the tourniquet blockade the wash out had increased to 42.1 ± 8.7 ml/min/100 g, but had declined 5 minutes after the release to 18.5 ± 2.0 ml/min/100g.

Phlebography revealed one venous thrombosis in a deep popliteal vein after 4 hours of tourniquet blockade (Figure 3b). In all the other animals phlebography revealed a patent venous flow. In all rabbits the phlebography was technically successful, but thrombosis could not be demonstrated (Figure 3a).

In the angiogram a clear occlusion of the arterial blood-flow was seen just above the inflated tourniquet cuff.

The control angiogram 30 minutes after the onset of circulation showed a normal angiogram in the tourniquet limb.

DISCUSSION

In 1949, Kety suggested that tissue blood flow could be measured by means of the disappearance of locally injected radioactive isotopes. Applying this principle, a radioactive isotope of the inert gas Xenon (Xe^{133}) has been used for experimental and clinical measurements (Lassen et al. 1964, Lindbjerg 1966). This isotope has proved to have theoretical advantages over other isotope clearance methods (Lassen et al. 1964).

The results obtained in the present investigation, using the Xe^{133} clearance method, suggest that even when the tourniquet time is extended to 3 hours, no blood flow occurs in the limb distal to the tourniquet cuff. This result correlates with those obtained by Klenerman & Crawley (1977). They reported a blood flow to the occluded limb of less than 1 per cent, when compared with the control limb. Their study was performed using the Na^{22} clearance method. It seems obvious that during these experimental conditions the circulation via the medullary canal is almost totally occluded and that the limb is virtually isolated from the circulation.

Hyperaemia following brief arterial occlusion has been recognized previously (Thulesius 1962, Lindbjerg 1966, Siggaard-Andersen & Bonde-Petersen 1967, Myhre 1975). However, tourniquet ischaemia is in surgical practice often extended to 2 hours or even longer. It was thought to be of interest to investigate the effect of releasing the tourniquet blockade after 1-, 2- and 3-hour periods of ischaemia. In the present work the peak of reactive hyperaemia was in all animals registered 1 minute after releasing the tourniquet blockade. Lindbjerg (1966)

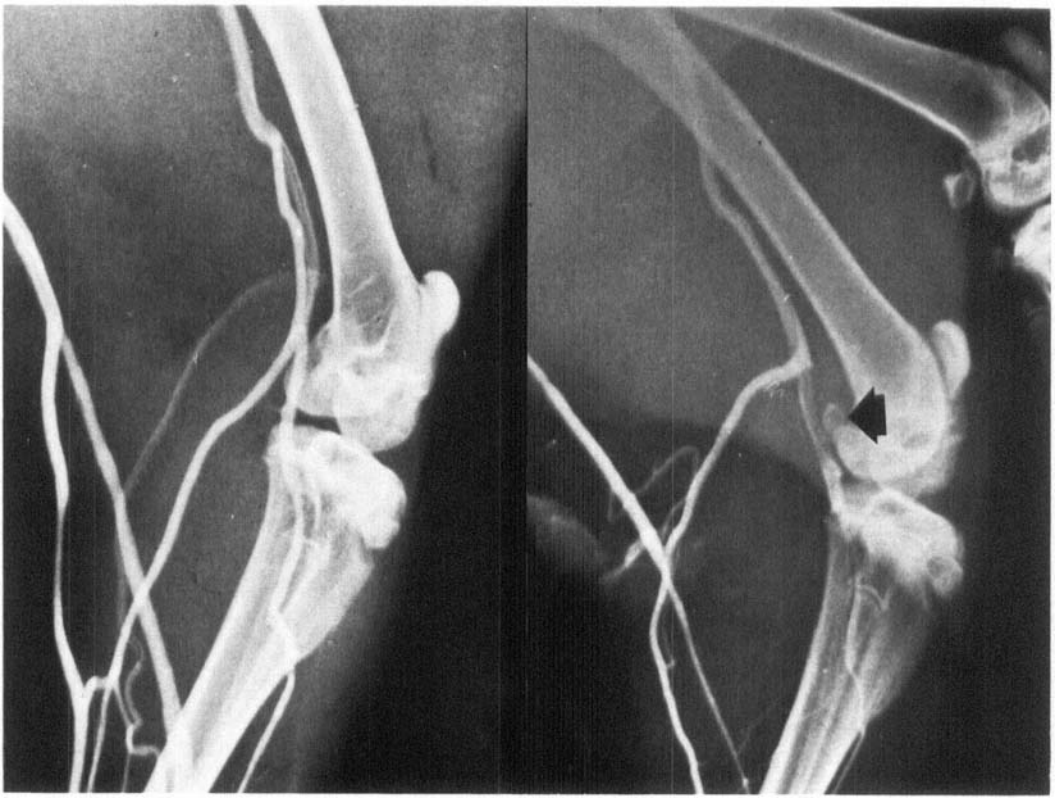

Figure 3. (a) Normal phlebography.
(b) Thrombosis in a deep popliteal vein following 4 hours of tourniquet occlusion.

reports in human experiments that up to a period of 5 minutes of occlusion the amount of hyperaemia increased in accordance with the duration of the ischaemia, but that further prolongation of the ischaemia did not cause a further increase in hyperaemia. In Lindbjerg's work the maximal blood flow in the human tibialis anterior muscle was measured after 5 minutes ischaemia combined with simultaneous exercise, and the peak flow of 70.8 ml/min/100 g was often maintained for more than 1 minute. In our study the maximal peak flow was registered after 60 minutes of tourniquet ischaemia and was comparable with the maximal muscle blood flow reported by Lindbjerg.

Shepherd (1950) and Hillestad (1963) have reported a peak blood flow of 40–50 ml/min/100 g in human calf muscles after brief strenuous exercise. These values resemble the flow in the rabbit tibialis anterior muscle during hyperaemia following release after 120 and 180 minutes of tourniquet blockade. Thulesius (1962) suggests that blockade of the extremity blood flow has an effect on the limb circulation comparable with strenuous exercise, since both result in a considerable reduction of collateral resistance.

Several mechanisms have been proposed for the reactive hyperaemia, such as accumulation of vasodilatator substances (Lewis & Grant 1925), and reduced vascular tone due to a fall in transmural pressure distal to the occlusion (Bayliss 1902, Folkow 1964). Local oxygen deficiency has also been held responsible for reactive hyperaemia (Crawford et al. 1959). Myhre (1975) suggests that the metabolites which are accumulated during circulatory arrest enter circulation and maintain increased blood flow, at least after

release of prolonged occlusions. The cause of the hyperaemia seems to be complex and can not be attributed to the influence of a single factor only.

The blood flow in the tibialis anterior muscle 5–10 minutes after release of the tourniquet is lower than the pretourniquet baseline value, but still within the range of values obtained under normal conditions (Shepherd 1950, Hillestad 1963, Lindbjerg 1966).

Experimental phlebographic studies to clarify the incidence of thrombosis after tourniquet ischaemia have not previously been performed. Our results with only one thrombosis in 12 rabbits do not justify conclusions of clinical consequences, but suggest, however, that further experimental work should be done in this field.

On the basis of the present investigation, the results may be summarized as follows:

A pneumatic tourniquet inflated to 300 mmHg caused a total blockade of the circulation in a hind limb of a rabbit;

The magnitude of reactive hyperaemia after releasing the tourniquet did not significantly depend on the tourniquet time;

Blood flow peak values were reached 1 minute after the return of circulation, with re-establishment of normal or subnormal values occurring within 5–10 minutes; and

Postoperative venous thrombosis was found in only one animal after 240 minutes of tourniquet time.

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