Intramuscular Pressure, Venous Function and Muscle Blood Flow in Patients with Lymphedema of the Leg

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Summary

Intramuscular pressure, muscle blood flow and venous emptying was studied in seven patients with unilateral lymphedema of the leg. Intramuscular pressure was measured with the wick technique. Muscle blood flow was assessed by means of the [133]Xenon clearance technique. Venous emptying was studied with strain gauge technique. Intramuscular pressure in the anterior tibial compartment of the edematous leg was 30 ± 14 mmHg at rest, rising to 49 ± 16 mmHg during exercise ($p < 0.05$). In the healthy leg the pressure rose from 16 ± 8 mmHg at rest to 28 ± 6 mmHg during exercise ($p < 0.05$). In the deep posterior compartment similar pressure values were obtained. Muscle blood flow during exercise was significantly higher ($p < 0.05$) in the healthy leg, 32.4 ± 6.8 ml x min^{-1}x $(100 \text{ g})^{-1}$ than in the edematous leg, 29.9 ± 4.8 ml x min^{-1} x $(100 \text{ g})^{-1}$. Venous emptying was significantly lower ($p < 0.05$) in the diseased leg, 44.7 ± 18.7 ml x min^{-1} x $(100 \text{ g})^{-1}$ than in the healthy leg, 61.4 ± 18.9 ml x min^{-1} x $(100 \text{ g})^{-1}$. Thus, lymphatic obstruction of the leg causes edema which leads to an increased intramuscular pressure and a decreased muscle blood flow and venous emptying.

Introduction

Compartment syndromes in the lower extremity are often associated with leg edema (1). It has previously been shown that leg edema of venous origin causes an increased intramuscular pressure and a decreased muscle blood flow (2). Fluid return via lymphatic vessels is probably important for removing excess extracellular fluid in muscle since lymphatic obstruction causes edema of the leg (3). The present study was undertaken in order to evaluate the effect of leg edema of lymphatic origin on intramuscular pressure, venous function and muscle blood flow.

Material

Seven patients, five females and two males, aged 46–72 years (mean 57) with unilateral lymphedema of the leg were studied. The left leg was affected in three cases, the right leg in four cases. The lymphedema was secondary to exstirpation of lymph nodes and radiation therapy in the treatment of malignant melanoma in six patients. In one patient the lymphedema was essential. The duration of lymphedema was 31 ± 22 months. The leg swelling was considerable with an increase in calf circumference of 5.2 ± 1.5 cm in the sick leg as compared with the contralateral leg. The patients had moderate pain in the leg on exhaustion. No other treatment than elastic stockings was used.

Methods

Intramuscular pressure measurement was performed with the wick technique described in detail previously (4). The wick catheters were placed into the anterior tibial and the deep posterior compartments under local anesthesia at the same level about 15 cm below the knee joint. In the wick catheter fibrils protrude from the bore of the catheter to prevent an obstruction of the catheter. The pressures were recorded with an electromagnetic transducer and recorder (Siemens Elema, Sweden).
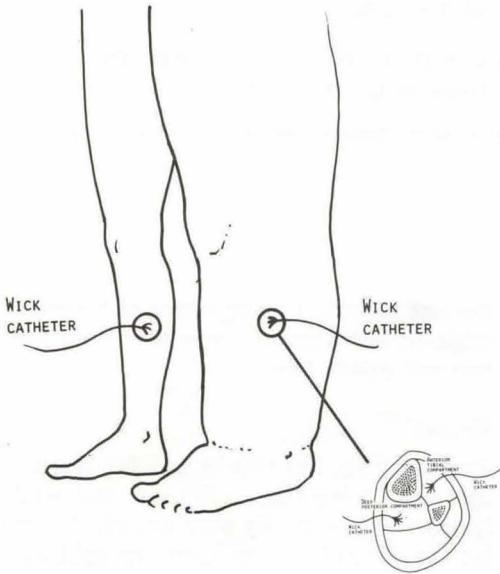
INTRAMUSCULAR PRESSURE
MM HG

INTRAMUSCULAR PRESSURE
MM HG

Fig. 1 Intramuscular pressure measurement with the wick technqiue in both legs in unilateral lymphedema. Inset: wick catheters placed in anterior tibial and deep posterior compartments

Fig. 2 Intramuscular pressures at rest and during exercise in the anterior tibial (upper diagram) and the deep posterior (lower diagram) compartments of both legs in 7 patients with unilateral lymphedema. SD is given. ▥ = lymphedematous leg. ☐ = control leg

The compartment pressures were measured in the lymphedematous and the contralateral leg at rest with the patient in a standing position. Compartment pressures were then measured during heel-raising every second for at least 5 min (Fig. 1).

Venous emptying was estimated according to *Hallböök* (5). A tourniquet applied around the thigh was pumped up to 50 mmHg. The calf volume was registered with strain gauge technique. When no further increase of calf volume was noted the tourniquet was released and the venous outflow measured. Values below 40 ml x min^{-1} x (100 g)$^{-1}$ were regarded as subnormal (5).

Systolic pressure of the big toe was measured with a strain gauge technique to evaluate any arterial disease.
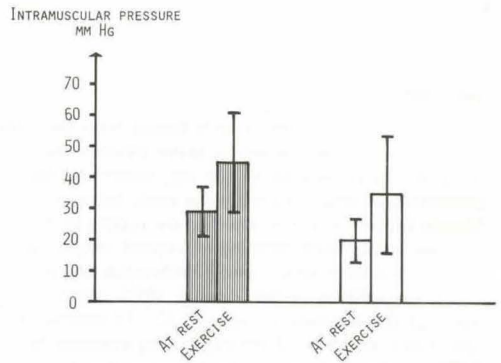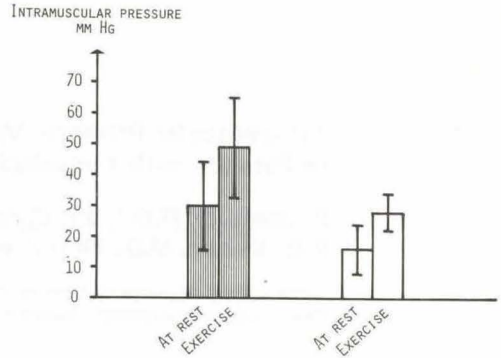
Muscle blood flow was measured in the anterior tibial muscles bilaterally using the ^{133}Xenon clearance technique. The legs were studied one after the other. Approximately 3.5 MBq ^{133}Xenon in 0.1 ml of isotonic saline was injected into the anterior tibial muscle about 15 cm below the knee joint at a depth of about 2 cm using a needle with 0.4 mm outer diameter. The isotope was slowly injected during 15 sec and the needle was withdrawn 15 sec thereafter. Records were taken from both legs with the patient at rest in a standing position, during heel-raising 60 times per min for 5 min and after exercise. Counting rate was recorded directly over the site of injection with a collimated cadmium telluride detector (TE 101, Pharmacia Electronics, Denmark) attached to the skin surface of the legs with adhesive tape. The detector was connected to a
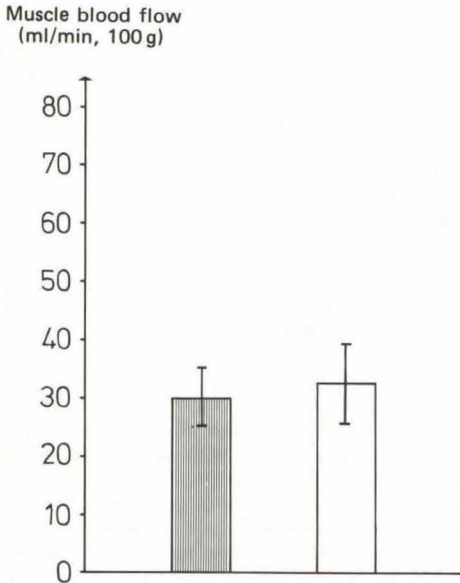
Muscle blood flow
(ml/min, 100 g)

Fig. 3 Muscle blood flow during exercise in both legs of 7 patients with unilateral lymphedema. SD is given. Symbols as in Fig. 2

portable memory, Memolog-500 (Pharmacia Electronics, Denmark) working as multiscaler giving digital information of the counting rate variation in 8 sec intervals which after each study was written directly into a computer (ABC 80). The blood flow (f) was calculated from the half-time (T1/2) of the disappearance of the isotope according to the following formula: $f = \frac{\ln 2 \cdot \lambda \cdot 60 \cdot 100}{T1/2}$, where λ represents the partition coefficient for [133]Xenon between tissue and blood. A standardized value of 0.7 μl/g was used for λ, since it has been used previously for muscle tissue (6). The calculated muscle blood flow is expressed as ml x min^{-1} x (100 g)$^{-1}$ tissue. Muscle blood flow was calculated from the decrease in counting rate during the last 2–3 min of the exercise.

Results

Intramuscular pressure in the anterior tibial compartment of the edematous leg was 30± 14 mmHg at rest, rising to 49±16 mmHg dur-

ing exercise (p< 0.05). In the healthy leg the pressure rose from 16±8 mmHg at rest 28±6 mmHg during exercise (p < 0.05). In the deep posterior compartment of the diseased leg the intramuscular pressure was 29±8 mmHg at rest, rising to 45 ± 16 mmHg during exercise (p < 0.05). The corresponding pressure values for the healthy leg was 20 ± 7 mmHg and 35 ± 19 mmHg (p< 0.05), respectively. The intramuscular pressures were significantly (p< 0.01) higher in the diseased than in the healthy leg both at rest and during exercise (Fig. 2).

Venous emptying was significantly lower (p< 0.05) in the diseased leg, 44.7±18.7 ml x min^{-1} x (100 g)$^{-1}$ than in the healthy leg, 61.4 ± 18.9 x min^{-1} x (100 g)$^{-1}$ ml. In two edematous legs venous emptying was markedly below 40 ml x min^{-1} x (100 g)$^{-1}$.

Systolic pressure of the big toe was normal in both the diseased and the contralateral leg. No significant difference in distal pressure was found between the legs.

Muscle blood flow as determined with the [133]Xenon clearance technique did not differ significantly between the leg with lymphedema, 2.9±0.9 ml x min^{-1} (100 g)$^{-1}$, and the contralateral leg, 2.5±1.0 ml x min^{-1} x (100 g)$^{-1}$, at rest. During exercise, however, muscle blood flow was significantly (p< 0.05) lower in the diseased leg, 29.9± 4.8 ml x min^{-1} x (100 g)$^{-1}$, than in the contralateral leg, 32.4±6.8 ml x min^{-1} x (100 g)$^{-1}$ (Fig. 3). After exercise blood flow decreased to the initial rest value within 5 min in both legs.

Discussion

Lymphatic obstruction leads to an increased tissue concentration of protein giving an increased colloid osmotic pressure in the tissue and resulting in an increased muscle water content (7). An increased muscle water content is known to cause an increased intramuscular pressure (1). In the present study the intramuscular pressure was found to be elevated in both the anterior tibial and the deep posterior compartments of the lymphedematous leg. The increased intramuscular pressure in the present lymphedematous patients may thus be due to an increased interstitial fluid

content (1). Venous obstruction in the leg has previously been shown to cause a marked increase in intramuscular pressure (2, 8, 9). The venous function in the present patients was tested by venous emptying. It was significantly lower in the lymphedematous leg than in the contralateral leg although it was subnormal in only 2 patients out of seven. The reason for the decreased venous emptying may be a partial compression of veins by the increased intramuscular pressure secondary to lymphedema.

Lymphatic obstruction may lead to an increased interstitial (intramuscular) pressure, which may cause a reduced capillary blood flow (7). Many investigators have demonstrated that increased intramuscular pressure reduces muscle blood flow (10, 11, 12). This was also seen in the present study where muscle blood flow during exercise as measured by the clearance of [133]Xenon was significantly decreased in the leg with increased intramuscular pressure due to lymphedema.

It is concluded from the present study that lymphatic obstruction of the leg causes edema which leads to an increased intramuscular pressure. The decreased venous emptying and the decreased muscle blood flow may be due to a compression of the veins and capillaries by the increased intramuscular pressure.

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