Edema in the lower limb following arterial reconstruction for atherosclerosis.

A study of pathogenetic mechanisms.

EINAR STRANDEN
(Department of Surgery, Aker Hospital)
<table>
<thead>
<tr>
<th>CONTENTS</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preface</td>
<td>5</td>
</tr>
<tr>
<td>Acknowledgements</td>
<td>6</td>
</tr>
<tr>
<td>Introduction</td>
<td>7</td>
</tr>
<tr>
<td>Discussion of methods</td>
<td>8</td>
</tr>
<tr>
<td>Discussion of results</td>
<td>12</td>
</tr>
<tr>
<td>Measures to limit edema formation</td>
<td>18</td>
</tr>
<tr>
<td>Summary and conclusions</td>
<td>18</td>
</tr>
<tr>
<td>References</td>
<td>18</td>
</tr>
</tbody>
</table>
PREFACE

The purpose of the present investigation was to study pathogenetic mechanisms causing edema of the lower limb following arterial reconstruction for atherosclerosis. This paper is based on the following articles which will be referred to by their Roman numerals:


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This study was carried out in the years 1979-83 when I was research fellow at the Department of Surgery, Section of Vascular Surgery, Aker Hospital, Oslo.

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INTRODUCTION
Edema of the foot and leg is a common complication after successful femoropopliteal reconstruction (Husni 1967) with a reported incidence ranging between 40% and 100% (Conolly and Stemmer 1970, Vaughan et al. 1970, Hamer 1972, Kooz and Stansel 1972). The edema usually appears within a few days postoperatively and may last for several months. It may worry the patient and represents a problem in the postoperative rehabilitation.

The exact mechanism for the development of post-reconstructive edema is unknown, but several causes have been proposed:

1. Deep venous thrombosis (Taylor 1962, Ernst et al. 1964). However, the incidence of deep venous thrombosis in patients with post-reconstructive edema is low (Myhre et al. 1974). This may be due to a hyperemia and concomitantly increased venous flow velocity which usually develops after vascular reconstruction (Sumner and Folse 1968, Myhre and Dedichen 1972, Kroese 1975). In addition anticoagulation therapy including heparin is used during the operations (Kakkar et al. 1971).

2. Destruction of lymph vessels caused by local dissection with resultant lymphedema (Linton and Darling 1962, Vaughan et al. 1970, Porter et al. 1972, Størren et al. 1974, Eickhoff and Engell 1982). The presence of lymph vessel disruption, visualized by lymphangiography, was not correlated with occurrence of postoperative edema (Porter et al. 1972). However, lymphangiography is probably an inadequate technique to study lymph vessel function in the development of edema.

3. Increased filtration from the capillaries which are suddenly exposed to a rise in blood pressure after prolonged adaptation to low pressure with concomitant degeneration of the arterial wall (Simeone and Husni 1959, Husni 1967, Husni and Manion 1967).

Fluid exchange between intra- and extravascular space takes place across the capillary wall. This structure is regarded as semipermeable: impermeable to plasma proteins and freely permeable to water and low molecular solutes. The interstitial fluid volume (IFV) is normally kept within narrow limits (Aukland and Nicolaysen 1981). A net increase in interstitial volume (Δ IFV) which is present in patients with postoperative edema, must be caused by a net capillary filtration (F) exceeding lymphatic flow (L):

\[ \Delta \text{ IFV} = \int F \, dt - \int L \, dt \]  \hspace{1cm} (1)
Studies on transcapillary fluid balance (Starling forces eq. 2) in patients with lower limb atherosclerosis have probably not been performed previously, mainly because of lack of suitable methods for clinical investigations. A «wick» method for collecting samples of interstitial fluid and «wick-in-needle» technique for measuring interstitial fluid pressure (Aukland and Fadnes 1973, Fadnes et al. 1977, Noddeland 1982a, 1982b) are valuable tools in the clinical investigation of transcapillary fluid balance.

In the present thesis Starling factors (eq. 2) were studied in patients with lower limb atherosclerosis before and after femoropopliteal reconstruction. The aim of the study was to gain information on the etiology of post-reconstructive edema.

The following questions were posed:

1. What is the magnitude of the Starling forces (i.e. what is the safety margin against edema formation) in patients with lower limb atherosclerosis?

2. In which compartment (subcutaneous, intramuscular) is the post-reconstructive edema located? This may have influence on the management of the edema, for example prophylactic fasciotomy.

3. To what extent does the increased volume influence interstitial fluid pressure, i.e. what is the tissue compliance?

4. Are the capillary filtration coefficient and Starling forces changed in legs with edema?

5. Does lower limb atherosclerosis affect arteriolar regulatory mechanisms?

**DISCUSSION OF METHODS**

**Femoropopliteal arterial reconstruction**

In all patients vascular surgery consisted of a femoropopliteal bypass. Usually a reversed autogenous saphenous vein was
used. If a suitable vein was not available, Dardik Biograft® (umbilical vein homograft) or Gore Tex® (expanded Teflon - PTFE) were chosen as a graft. Multiple skin incisions were made along the course of the great saphenous vein (Fig. 2A) (Cooley and Wukasch 1979). Following end-to-side anastomosis to the popliteal artery the graft was placed subfascially and anastomosed end-to-side to the common femoral artery (Fig. 2B).

Intraoperative anticoagulation consisted of 3000 IE Heparin®. To avoid interferences with the Starling forces no colloids were given postoperatively. On the day of operation the patients received an average of 1000 ml of isotonic saline solution and 3000 ml of Ringer’s lactate.

The patients received normal diet from the first postoperative day and were mobilized from the second. The measurements were performed with the patients in supine position in a room with an air-temperature of 23-25 degrees centigrade, after a resting period of at least 20 min.

**Interstitial fluid colloid osmotic pressure (COP)** measurements

Two main approaches to collect interstitial fluid for protein analysis have been used in previous investigations (Aukland and Nicolaysen 1981):

I. Direct sampling by micropipettes and catheters (Creese et al. 1962, Garetto and Hargens 1976, Haljamäe and Fredén 1970). However, the applied suction might increase net capillary filtration and thereby reduce interstitial fluid protein concentration (Aukland and Nicolaysen 1981).

II a. Technique based on fluid equilibrium by implantation of perforated capsules during a 4-6 weeks period (Guyton 1963). Use of the method in human investigation is for obvious reasons limited.

II b. In the present investigation a wick method was used (Scholander et al. 1968, Aukland and Fadnes 1973). In a recent study by Noddeland et al. (1982) similar values of COP\(_{iw}\) were found in fluid collected by implanted nylon wicks, empty wick catheter (no suction) and implantable colloid osmometer. Their investigation supported the assumption that COP of the fluid collected by the wick method reflects «true» COP\(_{iw}\).

Wicks made from 0.8 mm-thick nylon thread consisting of 210 filaments were soaked in saline. They were sewn subcutaneously. Wick insertion causes an inflammatory reaction during the first half hour after implantation, with marked increase in capillary protein leakage during that period. More than 50% of albumin in the wick is derived from plasma. In the next half hour the capillary protein leakage is reduced, and the wick equilibrates osmotically with the surrounding fluid (Fadnes and Aukland 1977). Following an implantation period of one hour the wick fluid was isolated as described by Johnsen (1974). The sample (2-10 μl) was centrifuged and transferred to an oncometer modified from those described by Tyhjaerg Hansen (1961) and Aukland and Johnsen (1974). For the oncometer an amplifier unit was constructed («5 μl Oncometer», Fig. 3), which permitted
easy read-out of the measured colloid osmotic pressure.

Sometimes wicks had to be discarded because of blood staining, but in most subjects at least one of the four wicks was without serious contamination. Usually one double strand was enough to obtain a sample of 3-5 µl which was needed for the oncometer. Pink wicks were included, since there is no significant difference between COP in clear and pink wicks obtained from the same person (Noddeland 1982b).

The wicks were introduced under local intracutaneous anesthesia (lidocaine chloride, 20 mg/ml, without adrenaline). The parts of the wicks passing anesthetized areas were cut away and did not contribute to the sample.

**Interstitial fluid hydrostatic pressure (Pif) measurements**

Pif was measured by a «wick-in-needle» (WN) technique (Scholander et al. 1968, Fadnes et al. 1977). The method is based on fluid equilibrium, with a water column between a pressure transducer and the interstitium. Hypodermic needles (0.8 mm OD, 40 mm length) were provided with a 4-mm side-hole approximately 7 mm from the tip. The needles were filled with cotton thread and sterilized by gamma irradiation. The thread provided a continuous water connection between tissue and needle lumen. When a needle without wick is used the tip is often obstructed.

The needle was connected to a pressure transducer (Statham P 23 Db) with a polyethylene tube (Portex manometer line, 60 cm, 200/490/060) (Fig. 4). The pressure transducer had a volume displacement of 0.04 µl/100 mm Hg, and compliance of the total measuring system was 0.7 µl/100 mm Hg.

After insertion of the needle under local anesthesia a negative pressure of 10 to 20 mm Hg was usually recorded in subcutaneous tissue. This was regarded as an insertion artifact. The pressure increased rapidly and stabilized after 1-10 min. In legs with edema the plateau was reached almost instantaneously. In normally hydrated tissue the process lasted longer. The patency
of the fluid phase was checked by slight compression of the polyethylene catheter by means of a screw clamp which was tightened to induce a pressure rise of approximately 5 mm Hg. Loosening the clamp gave the opposite response. The measurements were accepted only when the pressures were re-established (±0.5 mm Hg).

WN technique might result in erroneously high $P_j$ because of traumatic exudation by the needle insertion (Brace 1979). However, from investigations with WN and micropuncture techniques it was concluded that inflammatory reaction does not interfere with reliable measurements of $P_j$ by WN technique (Wiig et al. 1981).

**Measurement of calf blood flow and capillary filtration coefficient.**

Calf blood flow and capillary filtration coefficient (CFC) were measured by venous occlusion plethysmography (Hillestad 1963). A mercury strain-gauge plethysmograph with electrical calibration was used (Loosco Plethysmograph, G. L. Loos & Co., Amsterdam, Holland). The electrical calibration was compared with mechanical stretching of the strain-gauges by use of a micrometer. Mean difference between the two calibration procedures was approximately 3% which was regarded as satisfactory. Furthermore, an exact linear relationship was found between extension of the gauges and the recorded volume change.

The width of the venous occlusion cuff was approximately 1/3 of the circumference of the thigh (Graf 1964). The cuff was placed just proximal to the knee and inflated instantaneously to 50 mm Hg. The resulting change in leg volume was recorded by a Watanabe Mark V linear recorder (Watanabe Instruments Corp., Tokyo, Japan).
Calf blood flow (ml/min · 100 g of tissue) was calculated from the initial slope of the volume curve (Fig. 5) and estimated as the average of 3 consecutive measurements.

CFC was calculated from the slope of the volume curve during 3-6 min following venous occlusion (Fig. 5), assuming that filtration pressure increased 50 mm Hg. This is necessarily an approximation, since venous outflow pressure before inflation and postcapillary resistance were unknown. These factors may lead to an underestimation of CFC. However, the underestimation is probably similar in both limbs and the CFC-ratio between the limbs is therefore unchanged. This hypothetical technical error was investigated in two patients in whom cuff pressure was increased in step from 40 to 50 mm Hg. The increase in filtration pressure was assumed to be 10 mm Hg in both limbs, irrespective of the individual resting capillary pressures. In both patients leg volume increased more in the edematous leg than in the contralateral when filtration pressure increased by 10 mm Hg. CFC-ratio between operated and contralateral leg was almost equal at the three pressure levels (40, 50 and 40 → 50 = 10 mm Hg). CFC was slightly higher when calculated from the step increase of 10 mm Hg, which confirms that CFC is normally underestimated.

Edema may in itself affect the calculation of CFC. When tissue volume increases by X% due to edema, the amount of filtrate formed is underestimated X% by the present method.

**DISCUSSION OF RESULTS**

**Preoperatively**

Patients with peripheral atherosclerosis had a significantly lower COP_{if} than controls (Fig. 6) (I). Since COP_{if} already was low preoperatively, its potential as an edema preventing factor was equally reduced and could predispose to postoperative edema (Noddeland et al. 1982).

The reduction in COP_{if} was not due to a fall in plasma protein concentration;...
COP_{pl} was not different from controls. Neither did the patients show signs of ischemic edema (Thulesius and Sivertsson 1973). Therefore the lower COP_{if} was probably caused by a net reduction in interstitial protein mass i.e. «washout» of proteins. Since the capillary filtration coefficient was normal (VII), «washout» was probably caused by increased net filtration pressure. A reduced COP_{if} by «washout» of interstitial proteins may accompany increased venous pressure (Fadnes 1976, Wiederhielm 1979). In a recent study of patients with deep venous thrombosis and increased venous pressure of the lower limb we found a reduction in COP_{if} of 3.2 mm Hg compared to the contralateral leg (Seem and Strandén 1982). However, the condition was associated with edema, and the reduced COP_{if} could also be caused by dilution of interstitial proteins.

The mean age of patients was higher than in the control group. Age difference did not influence the measurement for COP_{if}. COP_{if} of the six oldest control subjects with a mean age compatible to the patients', was 9.5 mm Hg, not different from the other controls (p>0.05).

The force opposing filtration (reabsorption pressure, P_F) is defined as:

\[ P_F = \sigma \cdot COP_{pl} - \sigma \cdot COP_{if} + P_{if} \]

Assuming no net filtration across the capillary wall (i.e. filtration equals reabsorption), P_F is equal to capillary pressure (Pappenheimer and Soto-Rivera 1948, Hargens et al. 1981). Normally net transcapillary filtration occurs and mean P_C is higher than P_F. The difference between the pressures (i.e. net filtration pressure) is however small, probably about 0.5 mm Hg (Aukland and Nicolaysen 1981). Net filtration pressure may be defined as lymph flow divided by CFC (L/CFC) (Aukland and Nicolaysen 1981). The calculated net filtration pressure in legs where lymph vessels were cannulated (VI) was 0.5 ± 0.09 mm Hg. In this calculation it was assumed that the amount of soft tissue drained by the cannulated vessel was 100 g, and that the value of CFC did not change throughout the leg.

In patients with lower limb atherosclerosis P_C was higher than in controls (Fig. 7) (I). This finding was unexpected since we assumed that lower ankle systolic blood pressure would reduce capillary pressure.

Impaired arteriolar regulatory mechanisms seem to be among causative factors for the increased P_F in patients. This was concluded from the absence of vasoconstrictor response to increased venous distention (VIII). In atherosclerotic patients lower limb capillary pressure is thus more susceptible to increase in arterial pressure, for example in upright position.

An increased P_F, which means even higher P_C since P_C is normally higher than P_F, may explain the reduced COP_{if} in patients by causing increased filtration and
«washout» of proteins as mentioned above. Both factors (reduced COP_{if} and increased P_F) are unfavorable with respect to postoperative edema prevention.

Postoperatively
All patients had a successful operation. Venous thrombosis was not detected plethysmographically. Leg edema developed progressively during the first two weeks after surgery (V). During the first three postoperative days two out of 15 limbs became edematous. In the group studied 8-16 days postoperatively all 10 patients had edema. In the latter group the average leg volume increase was 22.5% calculated from surface measurements (II). Virtually all distally located edema (i.e. 98%) was subcutaneous (III). In the calf a relatively higher fraction of edema was intramuscular. Here the mean volume increase compared to the contralateral leg was 9.7% in the muscular compartments and 78.3% in subcutaneous tissue. This finding and registration of normal intramuscular pressure in edematous limbs confirmed that prophylactic fasciotomy was not indicated in this group of patients (III).

Capillary filtration coefficient
In 15 patients with leg edema CFC in the operated limb was twice the value of the contralateral (0.0032 versus 0.0017 ml/min \cdot 100 g \cdot mm Hg) (VIII). Thus, at otherwise equal net filtration pressure the amount of transudate formed in the operated limb was twice as high as in the contralateral.

CFC is the product of the hydrodynamic conductivity of the capillary wall and the capillary surface area available for exchange of fluid. Both factors may contribute to the increased CFC in limbs with edema. Because of the positive correlation between CFC and calf blood flow and postoperative hyperemia, it is tempting to assume that the increased CFC was due to increased capillary surface area by recruitment of capillaries (Kitchin 1963, Worm and Nielsen 1981). During venous occlusion plethysmography, however, primarily closed capillaries are probably filled and opened retrogradely when venous pressure is elevated to 50 mm Hg. The capillary surface area is therefore assumed to be maximal during the measurements, irrespective of precapillary tone.

Most legs with edema had postoperative hyperemia and increased skin temperature. Increased temperature reduces plasma viscosity and hence increases the rate of fluid transfer across the capillary wall (Curry 1979). Capillary permeability may also be enhanced by local increase of vasoactive substances such as histamine and bradykinin due to surgical trauma. These substances may increase capillary permeability (Boykin et al. 1980, Gawlowski 1982).

Calf blood flow was calculated from the slope of the volume curve immediately after inflation of the venous occlusion cuff - before venous pressure had increased considerably. Blood flow during measurement of CFC is probably lower than the value obtained at the beginning of venous occlusion. CFC was calculated at venous pressure of 50 mm Hg, presumably enough to trigger the veno-arteriolar reflex. Consequently in that period calf blood flow may have been reduced considerably (VIII). In addition, the increased venous pressure will in itself reduce local perfusion pressure. However, these considerations do not invalidate but increase the positive correlation between CFC and calf blood flow.

Transcapillary forces
Compared to the values obtained preoperatively (I), COP_{if} of the operated limb
was slightly lower in the first period (1-3 days) after surgery and increased in the later phase (Fig. 6) (V). However, the role of COP_{pl} in the transcapillary fluid balance is not determined by its absolute value, since the balance itself also depends on COP_{pl}. This was indicated by a positive correlation between COP_{pl} and COP_{pl} (V). Therefore a new parameter was introduced, COP_{pl} (pl 25). Each COP_{pl}-recording was given a new value where deviation in COP_{pl} from a standardized plasma value of 25 mm Hg was compensated for. Two examples are given (Fig. 8). The measured COP_{pl} (triangles) were moved along a parallel to the regression line of the non-operated leg to a vertical line at COP_{pl} = 25 mm Hg, to give the corrected COP_{pl} (squares). The two recordings obtained quite opposite values when variations in COP_{pl} were corrected. This strongly emphasizes that the value of COP_{pl} should be considered when COP_{pl} is discussed. (An alternative way is to calculate the colloid osmotic gradient, grad COP = COP_{pl} - COP_{pl}, and compare this parameter).

Because mean COP_{pl} was close to 25 mm Hg in both controls and patients preoperatively, COP_{pl} (pl 25) was almost identical to the measured COP_{pl}. Postoperatively, however, protein concentration of plasma was reduced, causing «uphill» transposition in figure 8. As a consequence COP_{pl} (pl 25) in all postoperative periods was higher than the measured COP_{pl}. The corrected value was higher than in the same type of patients preoperatively (p <0.005). This implies that the fraction of plasma proteins in the interstitial space is increased. Impaired lymphatic drainage is the most likely cause. It may increase P_{pl} (Fig. 9) which reduces net filtration by reduction in net filtration pressure. Protein transport by diffusion, depending on transcapillary concentration gradient, is however maintained.

Fig. 8. Relationship between COP_{pl}(A) and COP_{pl}(pl 25) (■). In the latter, variations in plasma colloid osmotic pressure (COP_{pl}) are compensated for. The measured COP_{pl} was moved along a parallel to the regression line of the non-operated leg (interrupted line) to a vertical line at COP_{pl} = 25 mm Hg, to give the corrected value.

A new steady state has presumably been established, in which both P_{pl} and COP_{pl} are increased. Increase in COP_{pl} caused by moderate lymphatic obstruction is disputable (Olszewski 1977). On the other hand the hypothesis is supported by measurements on patients with chronic unilateral lymph-

Fig. 9. Subcutaneous interstitial fluid pressure in controls, patients with lower limb atherosclerosis (Preop.) and after arterial reconstruction (Postop.). Filled symbols (●) represent control legs, the most diseased leg preoperatively and the operated leg: open symbols (○) represent contralateral leg. Number of subjects in parentheses. *p<0.05. **p<0.005 versus controls. Ns: not statistically different from controls.
edema of the lower limb. These patients had a mean increase in COP if of 5 mm Hg in the afflicted compared to the contralateral leg (Stranden, unpublished).

In all patients with lymph vessel cannulation (VI), maximal (systolic) intralymphatic end pressure waves ranged between 30 and 40 mm Hg, similar to those found in healthy controls (Olszewski and Engeset 1980). Thus preoperative ischemia did not reduce the efficiency of intrinsic mechanisms for lymph propulsion.

A high lymph flow measured after cannulating lymphatic vessels at the ankle (VI) does not imply high flow before cannulation. Because the lymphatics were only cannulated in distal direction, an obstructed system may have been opened during the cannulating procedure to give an artificial high value. Lymph flow was positively correlated to both CFC and \( P_i \), which indicated that the increased flow represented increased transcapillary filtration and not merely drainage of accumulated edema fluid.

**Compliance of the subcutaneous tissue**

Tissue compliance determines the change in \( P_{if} \) resulting from a change in interstitial fluid volume (IFV) and is defined as \( \Delta IFV/\Delta P_{if} \). A high compliance means that interstitial fluid can accumulate with a relatively small increase in \( P_{if} \) and vice versa.

Increased \( P_{if} \) may be an edema preventing factor by reducing net filtration pressure. The magnitude of this factor depends largely on tissue compliance. Pressure-volume measurements in patients with postoperative edema showed that subcutaneous tissue compliance varied considerably at different degrees of edema (Fig. 10) (IV).

When a logarithmic curve-fitting program was applied to the data the regression curve was represented by the equation:

\[
\ell(\zeta) = 0.71 \cdot \ln \zeta - 1.4, \tag{3}
\]

where \( \ell(\zeta) = P_{if} \) and \( \zeta (\zeta>0) \) is percentage increase in subcutaneous tissue volume, \( r = 0.62, p<0.001 \). This equation is represented by the interrupted line in figure 10. The first order derivative of eq. 3 is expressed by:

\[
\ell'(\zeta) = d\ell(z)/dz = \Delta P_{if}/\Delta IFV = 0.71/\zeta, \tag{4}
\]

which is the reciprocal of compliance. Assuming that eq. 3 is representing the pressure-volume relationship of the edematous leg in these patients, the subcutaneous tissue compliance is:

\[
\text{Compliance}_{(z)} = \zeta/0.71, \tag{5}
\]
where $z (z>0)$ is percentage increase in subcutaneous tissue volume. According to eq. 5 compliance increases linearly with IFV.

As an example the calculated compliance corresponding to a subcutaneous tissue volume increase of 50% was 70 ml/mm Hg · 100 g subcutaneous tissue. With extensive edema amounting to 600% the compliance was 845 ml/mm Hg · 100 g. There was a relatively large scatter in the recorded pressures, and the calculated examples must be regarded with caution.

The clinical consequence of the obtained pressure-volume curve is that initial accumulation of fluid in the interstitial space may be counteracted by a concomitant increase in $P_{1f}$ (the steepest part of the compliance curve). In the later phase of edema formation (>100% subcutaneous tissue volume increase) the compliance has increased considerably, permitting large quantities of fluid to transudate from the capillaries without any significant increase in $P_{1f}$. In this state of hydration $P_{1f}$ plays only a minor role in limiting further edema formation.

Interstitial tissue compliance in humans has not previously been established. The shape of the pressure-volume curve obtained in our study (IV) is similar to what has been found in animal experiments (Guyton 1965, Eliassen et al. 1974, Wiig and Reed 1981) although compliance (according to eq. 5) did not change abruptly. The calculated compliance was much higher than in previous studies. One explanation may be that the magnitude of volume changes and composition of tissues were different.

**Artiolar regulatory mechanisms**

To elucidate the arteriolar function in patients with lower limb atherosclerosis the veno-arteriolar (VA) reflex was studied (VIII). When vascular transmural pressure is increased more than 25 mm Hg (e.g. by lowering a limb) local blood flow is normally reduced (Gaskell and Burton 1953). The reduction is caused by arteriolar constriction due to a local sympathetic axon reflex triggered by venous distention (Henriksen 1977). In the present study (VIII) increase in transmural pressure was achieved by exposing a limb to subatmospheric pressure (limb negative pressure exposure: LNPE) of 75 mm Hg in a negative pressure chamber (Stranden et al. 1979).

The reflex was absent in patients with atherosclerosis studied before revascularisation. This could indicate dysfunction of the arteriolar smooth muscle cells or nerves participating in the reflex. In patients with pain at rest the peripheral resistance was probably reduced due to passive distention during LNPE. This finding indicated that autoregulation as well as vasoconstrictor response were hampered, possibly due to ischemia.

Atrophy of the vessel wall due to chronically reduced arterial pressure may be a causative factor for the long lasting postoperative hyperemia and edema (Husni and Manion 1967). However, other causes presumably play a part since the VA reflex, although absent in the immediate postoperative period, already returned one week after surgery. Accumulation of vasodilation metabolites in ischemic areas could make arteriolar smooth muscle cells less sensitive to constrictor stimuli (Henriksen and Wisborg 1975). On the other hand, although the VA reflex was normalized from the second postoperative week, postoperative hyperemia was still present in that period (VII). Therefore, a normal VA reflex does not exclude increased capillary pressure to be among the factors causing late postoperative edema.
MEASURES TO LIMIT EDEMA FORMATION

Prophylactic. Based on the assumption that lymphedema due to lymph vessel disruption is an etiological factor for post-reconstructive edema, measures may be taken to reduce surgical trauma in the inguinal and popliteal region (Eickhoff and Engell 1982). Lymph vessels may be visualized by injection of Patent Blue-Violet between first and second toe a few hours preoperatively, to save as many vessels as possible.

Symptomatic. To reduce the formation of edema postoperatively we have started compression treatment after arterial reconstruction for lower limb atherosclerosis. The compression increases Pff and thereby reduces net filtration pressure. Compression has been achieved by: Intermittent inflation of a cuff surrounding the limb (Jobst Extremity Pump® Jobst Ireland Ltd., Thurles, Ireland) for periods of 45 min three times a day, or: 2: By use of elastic stockings (T. E. D.® Anti-embolism stockings, Kendall, Boston, USA) commonly used by patients with deep venous insufficiency. The experience so far is that elastic stockings reduces edema formation most efficiently.

SUMMARY AND CONCLUSIONS

Reduced lymph flow and increased transcapillary filtration are major pathogenetic factors for lower limb edema following arterial reconstruction.

Decreased lymphatic drainage may explain the increased colloid osmotic pressure of interstitial fluid (COPff) and greater fraction of plasma proteins found in interstitial fluid postoperatively. Since the intrinsic pumping mechanism in the lymphatics was normal, reduced lymph flow was probably caused by lymph vessel damage during dissection in the groin and popliteal region.

In patients with postoperative edema, the capillary filtration coefficient (CFC) was significantly greater than preoperatively. This corresponded with a subcutaneous transudate formation twice as high as in the contralateral limb at equal net filtration pressure.

Based on measurements of Starling forces, the calculated capillary pressure in the operated leg was at least 2.5 mm Hg higher than in the contralateral. This rendered the operated leg even more susceptible to edema formation.

In addition, reduced COPff preoperatively may predispose to postoperative edema formation. Impaired arteriolar regulatory mechanisms in patients with atherosclerosis probably increase capillary pressure, resulting in a greater net filtration pressure. This in turn increases transcapillary filtration and «washout» of interstitial proteins.

Subcutaneous tissue compliance increased proportionally to increase in subcutaneous tissue volume. Interstitial fluid pressure (Pff) may initially serve as an important edema limiting factor. When edema has formed, its potential is reduced.

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