Scand. J. clin. Lab. Invest. **43,** 381–388, 1983.

Transcapillary forces in subcutaneous tissue of patients following operation for lower limb atherosclerosis

EINAR STRANDEN

Department of Vascular Surgery, Aker Hospital, Oslo 5, Norway

Stranden, E. Transcapillary forces in patients following operation for lower limb atherosclerosis. *Scand. J. clin. Lab. Invest.* **43,** 381–388, 1983.

Colloid osmotic pressure in plasma (COP_{pl}) and in interstitial fluid (COP_{if}) was measured in 18 healthy controls and 38 patients with leg oedema following femoropopliteal arterial reconstruction. Interstitial fluid was collected from nylon wicks which had been implanted subcutaneously for 1 h. Interstitial fluid pressure was measured with the 'wick-in-needle' technique.

The patients were examined once in the period 1–16 days after surgery. Twenty-three had oedema at the time of examination. Nearly all recordings of patients with oedema were performed 4–16 days postoperatively. Mean increase in leg volume in patients with oedema was 20%. Mean COP_{if} of the operated extremity were 5.4, 6.8 and 7.5 mmHg in the periods 1–3, 4–7 and 8–16 days after surgery, respectively. These values were lower than in controls (9.3 mmHg, $P<0.05$).

Mean COP_{if} in the operated leg was 1.2 mmHg lower than in the contralateral leg of patients without oedema. In patients with moderate oedema (<15% leg volume increase) this difference was approximately three times higher. For more extensive oedema the difference declined, and above 20% leg volume increase, COP_{if} of nearly all legs operated on was higher than the contralateral. This probably reflects an increased transcapillary fluid filtration in patients with moderate oedema whereas lymphatic obstruction due to the surgical procedure is the main causative factor in patients with extensive oedema.

Compared to the contralateral leg, mean increases in P_{if} of the leg operated on were 0.6, 2.3 and 3.6 mmHg in the three investigation periods respectively. P_{if} in operated legs in the two last periods was also higher than in controls (−0.7 mmHg, $P<0.005$). Increased P_{if} may thus contribute towards limiting oedema formation postoperatively.

Key-words: arterial occlusive disease; colloid osmotic pressure; interstitial fluid pressure; oedema; postoperative complication

Einar Stranden, M.Sci., Department of Vascular Surgery, Aker Hospital, Oslo 5, Norway

Oedema of the foot and leg is a common complication after femoropopliteal arterial reconstruction [3, 8]. The oedema is mainly subcutaneous [16] and may last for several months.

Several causes have been proposed: deep venous thrombosis [7], lymphoedema due to iatrogenic disruption of lymph vessels during the

0036-5513/83/0900-0381 $02.00

operation [4, 19] and increased net capillary filtration when the microcirculation is suddenly exposed to normal blood pressure after prolonged adaptation to low pressure [8, 9, 10].

The transcapillary fluid balance is governed by the Starling forces across the capillary wall. Any increase in interstitial fluid volume ($\triangle IFV$) is caused by a net capillary filtration (F) exceeding lymphatic flow (L): $\triangle IFV = \int F\,dt - \int L\,dt$. Net capillary filtration is described by the equation:

$$F = CFC\ [(P_c - P_{if}) - \sigma\ (COP_{pl} - COP_{if})], \quad (1)$$

where P_c and P_{if} are hydrostatic pressures in the capillaries and interstitial fluid respectively. COP_{pl} and COP_{if} are colloid osmotic pressures of plasma and interstitial fluid, and σ is the capillary reflection coefficient. In subcutaneous tissue σ is probably between 0.9 and 1.0 [2]. The capillary filtration coefficient (CFC) represents the amount of net filtrate formed in 100 g of tissue per min for each mmHg rise in net capillary filtration pressure. This factor is probably increased in patients with post-reconstructive oedema [15]. The remaining factors in the Starling equation may affect the formation of local oedema as follows:

1. Resetting of pre- and post-capillary resistance to limit increase in P_c [2].

2. Increase in P_{if} may create a counter-pressure acting against the capillary filtration [2].

3. A reduction in COP_{if} is probably an important oedema-preventing mechanism [1].

The aim of this investigation was to examine the possible role of these factors in the formation of local oedema in patients following arterial reconstruction for lower limb atherosclerosis.

MATERIAL AND METHODS

The control group. This included 13 women and five men with a mean age of 30 years (13–55) and no signs of cardiovascular disease.

The study group. This consisted of 13 women and 25 men with a mean age of 64 years (45–86) examined once in a period 1–16 days (mean 6) following femoropopliteal reconstruction for atherosclerosis. For bypass graft material autogenous saphenous vein was used in 27 cases, Dardik biograft® in five and PTFE (Gore Tex®) in six patients. They had no clinical signs of venous insufficiency. In no patients could deep venous thrombosis be detected plethysmographically. Patients with heart failure, diabetes or gangrene were excluded.

All patients received intra-operative anticoagulation consisting of 3000 IE Heparin® but no colloids such as Dextran or albumin were given during this investigation. On the day of the operation the patients received an average of 4000 ml crystalloids usually consisting of 1000 ml isotonic saline solution (155 mmol/l) and 3000 ml Ringer's Lactate (130 mmol/l).

The patients were mobilized on the second postoperative day, and were given a normal peroral diet until one day prior to the operation and from the first postoperative day. All measurements were performed with the participants' informed consent.

Ankle systolic blood pressure. This was measured by Doppler ultrasound technique and expressed as percentage of brachial pressure (ankle pressure index, %) [21].

Interstitial fluid colloid osmotic pressure (COP_{if}). This was measured by the 'wick method' [6, 18]. Four double-stranded nylon wicks (0.8 mm, 210 filaments) were sewn subcutaneously at a length of 3–4 cm on the distal anterolateral aspect of the leg 10–15 cm above the ankle. A small dose (approximately 0.05 ml) of local anaesthetic (Xylocain®, Astra, 20 mg/ml without adrenaline) was given where the needles were inserted and pulled out. After an implantation period of 1 h the wicks were pulled out and the wick fluid isolated by centrifugation in mineral oil. The colloid osmotic pressure was measured by a small sample oncometer with a membrane active to proteins with a molecular weight above 30 000 daltons (Diaflo® PM 30 ultrafilter, Amicon, Lexington, USA).

Plasma colloid osmotic pressure (COP_{pl}). This was measured in blood drawn from a cubital vein using the above mentioned oncometer.

Interstitial fluid pressure (P_{if}). This was measured on the distal anterolateral aspect of the leg with the 'wick-in-needle' technique [5, 17]. Hypodermic needles (0.8 mm OD, 40 mm length) were provided with a 3–4-mm side-hole about 7 mm from the tip, filled with a loosely packed cotton tread and sterilized by gamma irradiation. The needles were connected via a polyethylene tube to a pressure transducer (Statham P 23 Db).

The force opposing filtration (reabsorption pressure, P_r, derived from eq. (1) is defined as:

$\sigma COP_{pl} - \sigma COP_{if} + P_{if}$. When no net filtration occurs (i.e. when filtration equals reabsorption) P_r is equal to capillary pressure.

In some of the patients only one of the two parameters COP_{if} and P_{if} was obtained because of problems (technical, bleeding) connected with the measurements.

The clinical pitting oedema was quantified from surface measurements regarding the leg as a truncated cone. The method compared well with the results obtained by water volumetry [14]. The technique was based on the assumption that the oedema was unilateral and that the initial leg volumes were equal. Oedema was then expressed as percentage increase in leg volume of the operated side.

Statistics. The results were analysed according to standard statistical methods including Student's *t*-test for paired and non-paired data. Regression lines were calculated with the least squares method.

RESULTS

Fifteen patients were studied in the period 1–3 days postoperatively. Of these, two had oedema of the leg operated on. Twenty-three were examined during the period 4–16 days after the operation; 21 had oedema. Mean leg volume increase in patients with oedema was 19.9± 6.2% (± SD, $n=23$).

P_r was 15.7± 3.1 mmHg (± SD, $n=18$) in controls (σ assumed to be 1.0). In patients the value obtained from the leg operated on was higher for all periods than in the contralateral. P_r in the leg operated on was not different from controls in the periods 1–3 and 4–7 days after surgery, but significantly increased in the period 8–16 days postoperatively (Table I). On the other hand, P_r in patients with oedema was not significantly different from the non-oedematous group.

The results of COP and P_{if} measurements are summarized in table I, showing a lower COP_{pl} in patients compared to controls ($P<0.01$). Even within the study group there were differences, as COP_{pl} in the first period (1–3 days) was lower than in period 8–16 days postoperatively ($P<0.05$).

COP_{if} of patients was significantly lower bilaterally than in healthy controls (Table I). The results corresponding to the different postoperative days are shown in Fig. 1. Except for the measurement in the latest included period (8–16 days postoperatively), mean COP_{if} of the operated leg was always lower than the contralateral values. COP_{if} of the operated leg was lowest on the first postoperative day and increased during the investigation period. In patients without oedema the difference in COP_{if} between the two legs was 1.2± 1.2 mmHg (± SD, $n=13$) ($\triangle$ COP_{if}, Fig. 2), the leg operated on

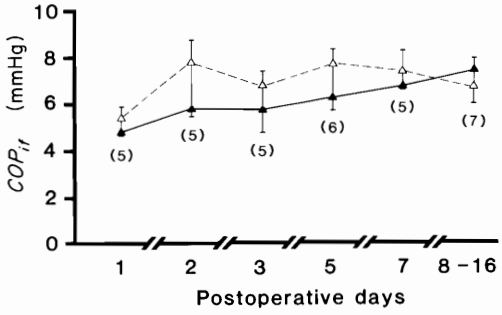

FIG. 1. Relationship between colloid osmotic pressure of interstitial fluid (COP_{if}± SEM) and the number of days after femoropopliteal arterial reconstruction for lower limb atherosclerosis. The measurements were performed once in the postoperative period. The filled triangles (▲) represent the leg operated on, the open triangles (△) the contralateral leg in those patients with successful bilateral measurements. Number of patients in parentheses.

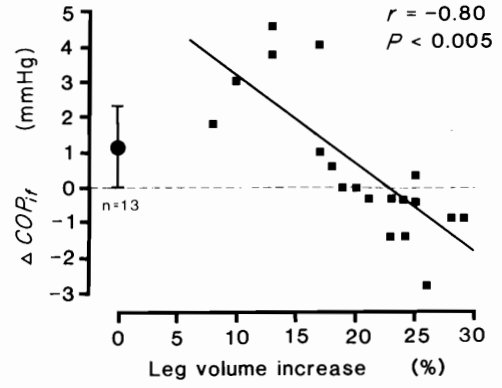

FIG. 2. Relationship between difference in COP_{if} between the two legs (ΔCOP_{if}=COP_{if} (contralateral leg) $-COP_{if}$ (operated leg)) and the calculated percentage leg volume increase in patients operated on for lower limb atherosclerosis. The filled circle represents mean ΔCOP_{if} (± SD) of the operated patients without oedema, and the filled squares represent ΔCOP_{if} of the patients with leg oedema. The regression line representing the patients with oedema is described by the equation: $\Delta COP_{if} = -0.261 \cdot x + 6.1$, where x represents percentage leg volume increase, $P<0.005$.

TABLE I. Colloid osmotic pressure of plasma (COP_{pl}), interstitial fluid (COP_{if}), and interstitial fluid pressure (P_{if}) of healthy controls and patients following arterial reconstruction for lower limb atherosclerosis. $COP_{if(pl\ 25)}$ is the calculated interstitial fluid colloid osmotic pressure where variations in COP_{pl} are compensated for. P_r is the calculated reabsorption pressure ($COP_{pl} - COP_{if} + P_{if}$). Mean values ± SD. Number of subjects in parentheses.

		1–3 days after surgery		4–7 days after surgery		8–16 days after surgery	
	Controls	Operated leg	Contralateral leg	Operated leg	Contralateral leg	Operated leg	Contralateral leg
Number of patients with oedema		2/15		11/13		10/10	
Leg volume increase (%)		2.2± 5.5 (15)		14.0± 10.2 (13)		22.5± 5.0 (10)	
COP_{pl} (mmHg)	25.7± 2.0 (18)		21.3± 2.5‡ (15)		23.0± 3.3‡ (13)		23.4± 2.4† (10)
COP_{if} (mmHg)	9.3± 2.0 (18)	5.4± 1.2‡ (14)	6.9± 2.3‡ (14)	6.8± 1.3‡ (13)	7.2± 1.5‡ (13)	7.5± 1.8* (10)	6.8± 1.8‡ (10)
$COP_{if(pl\ 25)}$ (mmHg)	9.0± 2.1 (18)	7.7± 0.9* (14)	9.3± 1.6 Ns (14)	8.1± 1.5 Ns (13)	8.5± 1.5 Ns (13)	8.5± 1.7 Ns (10)	7.8± 1.2 Ns (10)
P_{if} (mmHg)	−0.7± 1.3 (18)	−0.2± 1.1 Ns (10)	−0.8± 0.9 Ns (10)	1.3± 1.5‡ (12)	−1.0± 0.7 Ns (12)	1.9± 2.0‡ (9)	−1.7± 1.5* (9)
P_r (mmHg)	15.7± 3.1 (18)	15.8± 1.7 Ns (10)	13.4± 1.3* (10)	17.4± 2.0 Ns (12)	14.6± 2.0 Ns (12)	17.8± 2.6* (9)	14.7± 2.0 Ns (9)

*$P<0.05$, †$P<0.01$, ‡$P<0.005$ versus controls. Ns: not statistically different from controls.

having lowest COP_{if}. In subjects with moderate oedema there was a considerable reduction in COP_{if} of the operated extremity compared to the contralateral. In cases with more extensive oedema the difference declined, and finally, with more than 20% leg volume increase, COP_{if} of the operated leg (with one exception) was higher than the contralateral. There was also a significantly negative correlation when $\triangle COP_{if}$ was correlated to the number of days postoperatively $\triangle COP_{if} = -0.132x + 1.6$, where x is number of days after surgery, $1 < x < 16$, $r = -0.33$, $P < 0.05$).

There was a positive correlation ($P < 0.005$) between COP_{pl} and COP_{if} (Fig. 3). The regression line of the leg operated on in Fig. 3 was less steep than that of the contralateral, indicating that reduction in COP_{pl} to a lesser extent is compensated by reduction in COP_{if} in the afflicted leg. In order to reduce the plasma-dependent variation of COP_{if} the data were transformed to a standardized COP_{pl} of 25.0 mmHg ($COP_{if\ (pl\ 25)}$). Each point in Fig. 3 was moved along a parallel to the regression line of the non-operated leg to a vertical line at $COP_{pl} = 25.0$ mmHg to give the corrected COP_{if}. Except for the operated leg in the first postoperative period $COP_{if\ (pl\ 25)}$ was not different from controls (Table I).

When measured in the two last periods (4–7 days, 8–16 days postoperatively) P_{if} in the leg operated on was higher than in legs of controls ($P < 0.005$, Table I). In the first period (1–3 days postoperatively) the difference was not statistically significant.

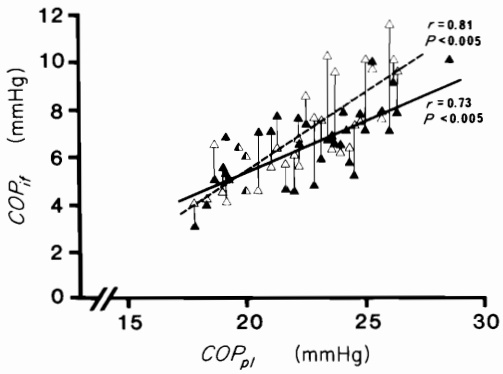

FIG. 3. Relationship between colloid osmotic pressure of interstitial fluid (COP_{if}) and plasma (COP_{pl}) in patients operated on for femoropopliteal atherosclerosis. The filled triangles (▲) represent the operated leg, the open triangles (△) the contralateral leg and the semiclosed triangles (◭) those subjects where COP_{if} in both legs was equal. Observations from the same patient are connected with vertical lines. The continuous line represents the linear regression of the legs operated on and the interrupted line represents the linear regression of the contralateral leg.

All atherosclerotic patients had a significantly lower ankle pressure index (both pre- and postoperatively) than controls (Table II). After reconstructive surgery there was a significant increase in both ankle systolic blood pressure and ankle pressure index of the leg operated on ($P < 0.005$). There was no significant correlation between preoperative ankle systolic pressure, ankle pressure index or increase in these parameters after reconstructive surgery and the

TABLE II. Ankle systolic blood pressure and ankle pressure index of healthy controls and patients with lower limb atherosclerosis, measured pre- and postoperatively. Mean values ± SD. Number of subjects in parentheses.

		Preoperatively		Postoperatively	
	Controls	Most diseased leg	Contralateral leg	Operated leg	Contralateral leg
Ankle systolic blood pressure (mmHg)	128.3± 10.4 (18)	[a] 68.4± 25.0† (32)	[b] 107.8± 33.8* (32)	[c] 104.8± 26.8† (32)	[d] 91.4± 30.5† (32)
Ankle pressure index (%)	106.8± 8.6 (18)	[e] 46.0± 15.2† (32)	[f] 71.7± 20.1† (32)	[g] 84.5± 18.4† (32)	[h] 71.8± 22.1† (32)

*$P < 0.01$, †$P < 0.005$ versus healthy controls. [a] versus [c]: $P < 0.005$, [b] versus [d]: $P < 0.05$, [e] versus [g]: $P < 0.005$, [f] versus [h]: no significant difference.

occurrence of local oedema in the operated leg, postoperatively.

DISCUSSION

The wick method used in the present study is well suited for clinical use. It is relatively painless and a sample of interstitial fluid can be obtained after an implantation period of 1 h. In addition, it corresponds well with other methods including empty wick catheter and implantable colloid osmometers [12].

Without net filtration the calculated P_r is equal to P_c. Actually $P_c > P_r$ but the difference is estimated to be in the order of 0.5 mmHg under normal conditions [2]. When net filtration increases, the underestimation of P_c is larger. Therefore, the increased P_r in the last investigation period (which implies even higher P_c) probably indicates that precapillary resistance vessels are unable to meet the increased arterial pressure (Table II) after reconstruction. This in itself would increase transudation [20].

The increased P_{if} observed in the last two periods is probably the major factor opposing further oedema formation. P_{if} was 2.3 and 3.6 mmHg higher than at the contralateral side, which compares well with findings in a previous study [17]. P_{if} may therefore counteract the increase in capillary pressure postoperatively.

The reduction in COP_{pl} postoperatively can be explained partly by haemodilution, caused by the fluid therapy given during and after the operation. This is supported by the finding of a low COP_{if} in both legs on the first postoperative day. In addition, the supine position following surgery may contribute to the reduction in COP_{pl}. Noddeland [11] found that COP_{pl} in healthy subjects was about 4 mmHg lower in the horizontal compared to the upright position. However, if the low COP_{pl} had been caused solely by a postural effect, a higher, not a reduced COP_{if} would have been expected, since transudation of fluid in the legs is less in the supine position [11].

The lower COP_{if} *compared to controls* could theoretically be explained by dilution of the interstitial proteins or reduction in interstitial protein content. The mean increase in leg volume in the patients with oedema was 20%. This was previously found to correspond with an increase in subcutaneous tissue volume of approximately 140% [16]. Provided that the oedema was produced by transudation of a fluid without protein, the calculated $COP_{if\ (pl\ 25)}$ would have been approximately 3.8 mmHg (9.0 mmHg $\cdot 100\%/(100\% + 140\%)$) using control subjects as reference. We have previously found a $COP_{if\ (pl\ 25)}$ of 5.9 mmHg preoperatively in the same type of patients as in the present study [18]. Based on that value, the calculated $COP_{if\ (pl\ 25)}$ would have been approximately 2.5 mmHg, which is substantially lower than the value found in this study. The reduction in COP_{if} compared to controls is thus less than would have been expected if only transudation of fluid with no protein increased the amount of interstitial fluid.

The 'normal' $COP_{if\ (pl\ 25)}$ in the last two periods indicate that the observed reduction in COP_{if} was parallel to the reduction in COP_{pl} (Table I, Fig. 3). Consequently, the reduction in COP_{pl} in the three postoperative periods was to a large extent neutralized by an equal reduction in COP_{if} (within 0.5 mmHg) in the operated leg. The 'colloid osmotic term' in equation 1 is therefore practically unchanged. On the other hand, the reduced $COP_{if\ (pl\ 25)}$ in the operated leg compared to controls observed in the first postoperative period may indicate dilution of the interstitial fluid or net 'washout' of protein. This finding indicates that the transcapillary fluid balance in the first period is in a non-steady state, in which oedema is *formed* by increased transcapillary filtration.

However, *compared to the same type of patients preoperatively* [18] $COP_{if\ (pl\ 25)}$ was increased in all postoperative periods ($P < 0.005$). This implies that there is an increasing fraction of plasma proteins in the interstitial space following arterial reconstruction. The increased COP_{if} may be due to increased interstitial protein concentration because of impaired lymphatic drainage (A), increased transcapillary protein transport (B), or both.

A. Reduced lymph flow capacity could be a causative factor for the negative ΔCOP_{if} found in patients with extensive oedema (>20% increase in leg volume, Fig. 2). Most of them were examined 8–16 days postoperatively. Possibly an increased P_{if} (induced by lymphatic obstruction) reduces net filtration. This in turn increases COP_{if} caused by a protein diffusion along a concentration gradient. A new steady-state with a low net filtration has presumably occurred. It is supported by the finding of an

increased COP_{if} of the afflicted leg in patients with chronic unilateral lymphoedema (approximately 5 mmHg higher than in the contralateral leg, Stranden: unpublished data). Lymphangiographic studies showing damage to the lymphatics after femoropopliteal bypass surgery [19], also point to the surgical trauma as an important factor.

B. Increased transcapillary protein filtration in rat hindquarters was found by Rippe and coworkers when venous pressure was experimentally increased [13]. They observed that serum albumin clearance increased abruptly at venous pressure above 35 mmHg ('stretched pore phenomenon'). Because capillary pressure in humans may be considerably increased in the upright position, such a phenomenon may be present postoperatively. There is no experimental evidence for such a hypothesis in patients in this study, however.

The change in interstitial fluid composition may be related to time after surgery and not to degree of leg volume increase *per se*. Because oedema developed gradually during the first 2 weeks (Table I), the measurements from the first postoperative days were probably performed before oedema formation had started. The changes in protein concentration seen in Fig. 2 may therefore to some extent be time-dependent. This is supported by the significantly negative correlation between $\triangle COP_{if}$ and number of days postoperatively.

In agreement with a previous study [4] no correlation between severity and duration of the oedema and increase in ankle systolic blood pressure was found. Other studies did not confirm this finding [8, 10].

In conclusion, reduced interstitial fluid protein concentration was observed in the first days following arterial reconstruction. This was probably caused by an increased transcapillary filtration. Later in the postoperative period both P_{if} and COP_{if} increased, which could be associated with lymph vessel destruction and reduced lymphatic drainage.

ACKNOWLEDGMENTS

This study was supported by grants from the Anders Jahres Fund for the Promotion of Science and the Professor Carl Sembs Fund. Einar Stranden, M.Sci. is a research fellow at the Department of Vascular Surgery, Aker Hospital, Oslo, Norway.

REFERENCES

1 Aukland, K. Autoregulation of interstitial fluid volume. Edema-preventing mechanisms. *Scand. J. clin. Lab. Invest.* **31,** 247, 1973.
2 Aukland, K. & Nicolaysen, G. Interstitial volume: local regulatory mechanisms. *Physiol. Rev.* **61 (3)** 556, 1981.
3 Connolly, J.E. & Stemmer, E.A. The nonreversed saphenous vein bypass for femoral-popliteal occlusive disease. *Surgery* **68,** 602, 1970.
4 Eickhoff, J.H. & Engell, H.C. Local regulation of blood flow and the occurrence of edema after arterial reconstruction of the lower limbs. *Ann. Surg.* **195,** 474, 1982.
5 Fadnes, H.O., Reed, R.K. & Aukland, K. Interstitial fluid pressure in rats measured with a modified wick technique. *Microvasc. Res.* **14,** 27, 1977.
6 Fadnes, H.O. & Aukland, K. Protein concentration and colloid osmotic pressure of interstitial fluid collected by the wick method. Analysis and evaluation of the method. *Microvasc. Res.* **14,** 11, 1977.
7 Hamer, J.D. Investigation of oedema of the lower limb following successful femoropopliteal by-pass surgery: the role of phlebography in demonstrating venous thrombosis. *Br. J. Surg.* **59,** 979, 1972.
8 Husni, E.A. The edema of arterial reconstruction. *Circulation* **35** (Suppl. 1), 169, 1967.
9 Kroese, A. Hyperaemia of the calf after arterial reconstruction for lower limb atherosclerosis. *Acta chir. scand.* **141,** 104, 1975.
10 Myhre, H.O. & Dedichen, H. Haemodynamic factors in the oedema of arterial reconstructions. *Scand. J. thorac. cardiovasc. Surg.* **6,** 323, 1972.
11 Noddeland, H. Influence of body posture on transcapillary pressures in human subcutaneous tissue. *Scand. J. clin. Lab. Invest.* **42,** 131, 1982.
12 Noddeland, H., Hargens, A.R., Reed, R.K. & Aukland, K. Interstitial colloid osmotic and hydrostatic pressures in subcutaneous tissue of human thorax. *Microvasc. Res.* **24,** 104, 1982.
13 Rippe, B., Kamiya, A. & Folkow, B. Transcapillary passage of albumin, effects of tissue cooling and of increases in filtration and plasma colloid osmotic pressure. *Acta physiol. scand.* **105,** 171, 1979.
14 Stranden, E. A comparison between surface measurements and water displacement volumetry for the quantification of leg edema. *J. Oslo Cy Hosps* **31,** 153, 1981.
15 Stranden, E. Transcapillary fluid filtration in patients with leg edema following arterial reconstruction for lower limb atherosclerosis. *VASA.* (in press, 1983).
16 Stranden, E. & Enge, I. Computed tomography in the investigation of leg edema following arterial reconstructions. *Eur. J. Radiol.* **2,** 113, 1982.
17 Stranden, E. & Myhre, H.O. Pressure-volume recordings of human subcutaneous tissue: a study

in patients with edema following arterial reconstruction for lower limb atherosclerosis. *Microvasc. Res.* **24,** 241, 1982.

18 Stranden, E. & Myhre, H.O. Transcapillary forces in patients with lower limb ischemia. *Scand. J. clin. Lab. Invest.* **43,** 233, 1983.

19 Støren, E.J., Myhre, H.O. & Stiris, G. Lymphangiographic findings in patients with leg oedema after arterial reconstructions. *Acta. chir. scand.* **140,** 385, 1974.

20 Wiederheim, C.A. Dynamics of capillary fluid exchange: A non linear computer simulation. *Microvasc. Res.* **18,** 48, 1979.

21 Winsor, T. The influence of arterial disease on the systolic blood pressure gradients of the extremity. *Am. J. med. Sci.* **220,** 117, 1959.

Received 3 October 1982
Accepted 28 March 1983