Myocutaneous Flap Ischemia: Flow Dynamics Following Venous and Arterial Obstruction

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To further clarify the pathogenesis of the poorer prognosis in skin flaps exposed to venous stasis compared with arterial insufficiency, a microsphere study was conducted in bilateral rectus abdominis island flaps in seven pigs. The relationship between capillary blood flow and arteriovenous (A-V) shunting was studied during progressive 1-hour intervals of arterial insufficiency and venous stasis and during 3 hours of reperfusion.

Under controlled conditions, total blood flow was reduced from 100 percent to both 50 and 25 percent by application of an adjustable clamp on the artery supplying one flap and on the vein draining the contralateral flap. The relative distribution between A-V shunt flow and capillary blood flow was different in arterial insufficiency when compared with venous stasis at both the 50 percent and the 25 percent blood flow levels. In the arterial insufficiency flaps, the A-V shunt flow and capillary blood flow shared the total blood flow in the following percentages: 64/36 (at 100 percent total blood flow), 44/56 (at 50 percent total blood flow level), and 22/78 (at 25 percent total blood flow level). In the venous stasis flaps, the A-V shunt flow and the capillary blood flow shared the total blood flow in percentages of 70/30, 66/34, and 55/45, respectively. Hence, in arterial insufficiency flaps, capillary blood flow was spared by a relatively greater decline in A-V shunting compared with venous stasis flaps. Redistribution of capillary blood flow from subcutaneous tissue to muscle was observed, whereas blood flow was equally distributed throughout the length of the flaps at all flow levels. After 1 hour of reperfusion, intravenous blood pressure and A-V shunting were similar to preischemic values, whereas capillary blood flow and total venous outflow were lower in venous stasis flaps than in arterial insufficiency flaps.

Venous stasis thus causes greater microcirculatory derangements than arterial insufficiency, and the slow reflow after venous stasis may indicate that even short periods of venous stasis are potentially damaging to the microcirculation.

Vascular complications secondary to free-flap procedures comprise a complex sequence of ischemia-reperfusion events leading to no reflow and tissue injury. Total venous occlusion appears to create more damage to skin flaps than arterial occlusion, and blood flow is lower during the immediate reperfusion phase. The clinical scenario is, however, more likely mimicked by a progressive reduction in flow that, in a worst-case scenario, would end in total flow cessation. Incipient ischemia caused by either reduced arterial inflow or increased venous outflow resistance influences the flap hemodynamics in a yet unclarified way. Arteriovenous (A-V) shunting is known to constitute a substantial proportion of the blood flow in skin flaps. In myocutaneous flaps, A-V shunts carry more than 50 percent of the blood flow in the uneventful postoperative phase. Attempts to improve capillary blood flow in normal myocutaneous flaps by closing A-V shunts have so far failed. Ischemia may cause earlier closure of the thin-walled capillaries than of A-V shunts. The venous congestion may further prolong or even prohibit the reperfusion when the obstruction is eliminated. Furthermore, the relative flow distribution among the various tissue components in a composite flap may be influenced differently due to a tissue priority in the flap.

The purpose of the present study was to compare the hemodynamic reaction of bilateral myocutaneous flaps during conditions of reduced arterial inflow and impaired venous outflow. The
hypotheses were (1) that venous stasis would lead to a greater degree of A-V shunting than would arterial insufficiency, (2) that the reperfusion after venous stasis would be less than that after arterial insufficiency, (3) that capillary blood flow would be spared in expense of A-V shunting, and (4) that muscle would be protected at the expense of skin and subcutaneous tissue with respect to capillary blood flow.

**Materials and Methods**

Seven Danish Landrace pigs weighing 42.4 ± 1.3 kg (mean ± SEM) were used. The care and use of these laboratory animals complied with the principles stated by the Danish law on animal experimentation.

**Experimental Preparation**

The pigs were premedicated by intramuscular injection of midazolam (30 mg, Roche A/S Hvidovre, Denmark) and ketamine (500 mg, Parke-Davis, Copenhagen, Denmark). After intubation, the pigs were ventilated mechanically on a Servo 900 ventilator with 50% oxygen (4 liters/min, 12 cycles/min). Anesthesia was maintained by continuous IV infusion of midazolam (15 mg/h), ketamine (500 mg/h), pancuronium (6 mg/h, Organon AS, Skovlunde, Denmark), and pethidine (25 mg/h, DAK, Copenhagen, Denmark). Isotonic saline (125 ml/h) was given continuously.

The carotid arteries were exposed, and a pigtail catheter (7 French, William Cook Europe A/S) was inserted into the left ventricle through the left carotid artery. A straight multiperforated catheter (7 French, William Cook Europe A/S) was directed into the aorta through the right carotid artery and advanced to the level of the fourth lumbar vertebra. A catheter was placed in one external jugular vein. The animals received ampicillin (1 gm/4 h) IV and continuous infusion of heparin (100 IU/kg/h) starting after catheterization. A warming blanket was used for maintaining a constant core temperature.

Cardiopulmonary homeostasis was ensured by repeated measurements of blood gases (ABL-3, Radiometer, Denmark) and continuous registration of heart rate and arterial pressures (Gould Statham transducers and Sirecost, Siemens monitor, Bilthoven, The Netherlands).

**Flap Model**

Bilateral rectus abdominis myocutaneous island flaps (7 × 14 cm) perfused by the superior epigastric arteries were studied. The superficial epigastric veins were located at the distal border of the marked flap areas, and after surgical exposure, intravascular catheters were placed in the veins for continuous registration of intravenous pressure. After elevation and reinserting of the flaps, the superior epigastric veins were cut in the costosternal triangle and cannulated by plastic tubings (OD 4 mm, ID 3 mm, IVAC, Codan, Lensahn, Germany). The venous blood from each flap was led through a drop counter in order to measure venous outflow (VO) continuously. This method has been described previously. Priming volume of the flowmeter was approximately 30 ml. A constant outlet pressure was chosen to be zero at heart level. The normal fluctuations in venous blood pressure close to the heart cannot be obtained in this system. The blood was autotransfused by means of the catheter placed in the jugular vein. The major part of the sympathetic nerves to this flap arises from the spinal nerves and only a minor part runs with the vessels. In order to obtain a comparable situation in all animals, adventitia was stripped from the supplying arteries.

**Hemodynamic Investigation**

NEN-TRAC 15-μm microspheres (New England Nuclear, Boston, Mass.) were used for assessment of the regional capillary blood flow (RBF). Four blood flow measurements were performed in each experiment using microspheres labeled with 141Ce, 103Ru, 95Nb, and 46Sc. The order of microspheres in each experiment was randomized. Each vial contained approximately 9.25 MBq (250 μCi) activity and 6 × 10⁵ particles (specified diameter of 15.5 ± 0.1 μm) in 6 ml physiologic saline with 10% dextran and 0.01% Tween 80. Each microsphere vial was agitated thoroughly on a Whirlmixer for 30 minutes prior to injection in the left ventricle (over 60 s). Reference blood was withdrawn from the aortic catheter (rate 5 ml/min for approximately 5 minutes) beginning 30 seconds before microsphere injection by means of siliconated glass syringes driven by a reversed infusion pump (Braun-Melsungen).

**Study Design (Fig. 1)**

Three hours postoperatively, the first microsphere blood flow measurement was performed (preischemia). On the basis of the actual venous outflow on each side, venous outflow was regulated down to 50 percent for 1 hour, to 25 percent for 1 hour, and to zero blood flow for 1 hour. Thereafter, a reperfusion period of 3 hours was
allowed. The blood flow reduction on the right side mimicked arterial insufficiency by application of a specially manufactured stepless clamp on the superior epigastric artery. On the left side, a similar clamping was performed on the tube cannulating the superior epigastric vein. The venous outflow was continuously adjusted to the desired flow level by manipulating the clamp screw. Second, third, and fourth microsphere blood flow measurements were performed at the 50 percent blood flow level, at the 25 percent blood flow level, and after 1 hour of reperfusion, respectively.

Simultaneously with each microsphere blood flow measurement, venous outflow from each flap was collected for assessment of the amount of nonentrapped microspheres in the flap. Venous blood was collected for 5 minutes at the microsphere flow level of 100 percent, at the 50 percent flow level, and at reperfusion. Outflowing blood was collected for 10 minutes at the 25 percent flow level because of low flow rate. Blood loss resulting from collection of reference blood from the abdominal aorta and flap veins and from collection of blood samples was substituted using donor blood from other pigs. After 3 hours of reperfusion, the animals were sacrificed with intracardiac potassium chloride. The entire flaps and biopsy specimens from the lower abdominal wall—roughly 10 cm distal to the flaps—were collected, frozen, and dissected into the four tissue layers: skin, subcutaneous tissue, panniculus carnosus, and muscle. Each tissue layer was cut into pieces of approximately 2 gm. The biopsy specimens, blood from the aortic reference sample, and blood from the venous outflow were placed into preweighed tubes and weighed.

Calculations

Cardiac output (CO) (liters/min) was calculated from the total injected microsphere count, the reference blood sample rate, and the microsphere count in the reference sampling using the equation

\[
CO = \frac{\text{activity in injectate}}{\text{activity in reference sample}} \times \text{reference sampling rate}
\]

Regional capillary blood flow (RBF) (ml/min/100 gm) in control tissue was calculated by use of the reference sampling, whereas all calculations of blood flow in the flaps were based on the fractional uptake of activity to avoid the potential error associated with the reference-sampling method. Absolute blood flow values were calculated as the fractional activity uptake multiplied by the venous outflow:

\[
RBF = \frac{\text{activity in tissue}}{\text{activity in entire flap} + \text{activity in venous outflow}} \times \text{venous outflow (ml/min)}
\]

A-V shunting (A-V) (%) was calculated on the basis of the microsphere activity in the collected venous outflow and the microsphere activity in the flaps as

\[
A-V = \frac{\text{activity in venous outflow}}{\text{activity in venous outflow} + \text{activity in flap}} \times 100\%
\]
Table I

Hemodynamics at the Four Blood Flow Measurements

<table>
<thead>
<tr>
<th></th>
<th>Preischemia</th>
<th>50 Percent</th>
<th>25 Percent</th>
<th>Reperfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output (liters/min)</td>
<td>3.5 (0.5)</td>
<td>2.8 (0.4)*</td>
<td>2.7 (0.3)*</td>
<td>2.8 (0.5)</td>
</tr>
<tr>
<td>Mean arterial pressure (mmHg)</td>
<td>87 (4)</td>
<td>99 (7)</td>
<td>101 (10)</td>
<td>91 (5)</td>
</tr>
<tr>
<td>Pao₂ (kPa)</td>
<td>19.7 (0.6)</td>
<td>19.2 (0.6)</td>
<td>19.1 (0.5)</td>
<td>19.4 (0.5)</td>
</tr>
<tr>
<td>Control capillary blood flow (ml/min/100 gm)</td>
<td>1.1 (0.2)</td>
<td>0.9 (0.2)</td>
<td>0.9 (0.2)</td>
<td>1.2 (0.3)</td>
</tr>
<tr>
<td>Skin</td>
<td>1.4 (0.5)</td>
<td>1.0 (0.2)</td>
<td>1.1 (0.3)</td>
<td>2.2 (0.6)</td>
</tr>
<tr>
<td>Subcutis</td>
<td>1.3 (0.3)</td>
<td>1.2 (0.3)</td>
<td>1.1 (0.2)</td>
<td>1.3 (0.5)</td>
</tr>
<tr>
<td>Pancreas</td>
<td>2.6 (0.3)</td>
<td>2.2 (0.2)</td>
<td>2.1 (0.1)</td>
<td>2.1 (0.1)</td>
</tr>
</tbody>
</table>

Note: Cardiac output, mean arterial blood pressure, arterial oxygen tension (Pao₂), and capillary blood flow in control tissue biopsy specimens measured four times throughout the experiment. Values are mean (SEM), n = 7.

* Significant difference from preischemic value (p ≤ 0.05).

Statistical Analysis

The mean values and the standard error of the mean (SEM) were calculated for all measurements. A probability plot showed that flow parameters were with good approximation normally distributed. Data were analyzed with analysis of variance (ANOVA) and with student's paired t test (two-tailed); p values ≤ 0.05 were considered significant. No corrections were made for multiple testing.

RESULTS

Clinical assessment prior to release of the clamps showed that the flaps subjected to venous stasis were cyanotic and congested during ischemia, whereas flaps with arterial ischemia were pale and without edema formation. Secondary to clamp release, the arterial insufficiency flaps showed reactive hyperemia, whereas congestion and cyanosis in the venous stasis flaps subsided.

Mean arterial blood pressure, arterial oxygen tension, and regional blood flow in control tissue were similar at the four blood flow measure-
ments. Cardiac output was higher in the preischmic phase than at the second and third blood flow measuring time periods (p < 0.05). Cardiac output at the reperfusion time was not statistically different from that at the preischemic level (Table I).

Venous outflow values at hourly intervals are shown in Table II. The 50 percent and the 25 percent blood flow levels were obtained within a small percentage. During the first minutes of the reperfusion period, a pronounced hyperemic response was observed in all arterial insufficiency flaps and in none of the venous stasis flaps. Thereafter, venous outflow increased steadily in both flap types. The venous outflow was higher in arterial insufficiency flaps compared with the venous stasis flaps during reperfusion.

Venous blood pressure increased secondary to flap surgery (p < 0.001) and reached a preischmic level that unexpectedly was higher in flaps destined to undergo venous stasis (p < 0.05) (Table III). During ischemia, venous blood pressure decreased from 7.4 ± 2.2 mmHg to 3.9 ± 1.7 mmHg.

Table II

Venous Outflow

<table>
<thead>
<tr>
<th></th>
<th>Arterial Insufficiency</th>
<th>Venous Stasis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ml/min</td>
<td>Percent</td>
</tr>
<tr>
<td>Preischemia</td>
<td>10.3 (1.9)</td>
<td>100</td>
</tr>
<tr>
<td>Partial ischemia</td>
<td>5.1 (0.8)</td>
<td>51.9 (5.1)</td>
</tr>
<tr>
<td>50 percent</td>
<td>2.6 (0.5)</td>
<td>25.1 (2.4)</td>
</tr>
<tr>
<td>25 percent</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total ischemia</td>
<td>10.8 (1.3)</td>
<td>121.0 (19)</td>
</tr>
<tr>
<td>Reperfusion: 1 hour</td>
<td>12.6 (1.5)</td>
<td>144.9 (22)</td>
</tr>
<tr>
<td>2 hours</td>
<td>13.1 (1.7)</td>
<td>150.2 (26)</td>
</tr>
<tr>
<td>3 hours</td>
<td>13.1 (1.7)</td>
<td>150.2 (26)</td>
</tr>
</tbody>
</table>

Note: Venous outflow (ml/min and percent of preischemic level) in control tissue to receive an abdominal island flap subjected to partial and total ischemia (arterial insufficiency) and simultaneously clamping the vein on the other flap (venous stasis). Values are mean (SEM), n = 7.

* Significant difference between arterial insufficiency and venous stasis (p ≤ 0.05).
TABLE III

<table>
<thead>
<tr>
<th>Arterial Insufficiency</th>
<th>Venous Stasis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prior to flap elevation</td>
<td>5.0 (0.9)</td>
</tr>
<tr>
<td>Preischemia flap elevated and venous outflow stabilized</td>
<td>7.4 (2.2)</td>
</tr>
<tr>
<td>Partial ischemia:</td>
<td></td>
</tr>
<tr>
<td>50 percent</td>
<td>5.7 (1.5)</td>
</tr>
<tr>
<td>25 percent</td>
<td>5.0 (1.8)*</td>
</tr>
<tr>
<td>Total ischemia</td>
<td>5.9 (1.7)†</td>
</tr>
<tr>
<td>Reperfusion:</td>
<td></td>
</tr>
<tr>
<td>1 hour</td>
<td>8.3 (2.2)</td>
</tr>
<tr>
<td>2 hours</td>
<td>7.1 (2.3)</td>
</tr>
<tr>
<td>3 hours</td>
<td>7.0 (2.2)</td>
</tr>
</tbody>
</table>

Note: Venous pressure (mmHg) in the veins draining myocutaneous rectus abdominis island flaps. Values were obtained before flap elevation, after elevation of the flap and cannulation of the vein for continuous registration of venous outflow (VO), after partial ischemia (blood flow reduced to 50 percent of preischemic level and 25 percent of preischemic level), after total ischemia, and during 3 hours of reperfusion. Values are mean (SEM), n = 7.

Different venous pressure when compared with preischemic values (*p < 0.05; †p < 0.001).

mmHg in flaps exposed to arterial insufficiency (p < 0.01) and increased from 13.4 ± 1.8 mmHg to 58.0 ± 6.0 mmHg in flaps exposed to venous stasis (p < 0.001). In both arterial insufficiency and venous stasis flaps, the venous pressure was normalized after 1 hour of reperfusion.

A-V Shunting (Fig. 2)

In the preischemic state, 3 hours after flap elevation, two-thirds of the microspheres entering the flap circulation reappeared in the collected venous outflow. The shunt percentage decreased in arterial insufficiency flaps to 44 percent at the 50 percent blood flow level (p < 0.01) and to 21.6 percent at the 25 percent blood flow level (p < 0.001). This left 56 and 78 percent of the total flap flow to run through the capillaries, respectively. In venous stasis flaps, the A-V shunt percentage was maintained during flow reduction to 50 percent but decreased to 55 percent at the 25 percent blood flow level (p < 0.01). Venous stasis flaps shunted more microspheres than the arterial insufficiency flaps at both levels of partial ischemia (p < 0.001). After 1 hour of reperfusion, the shunting percentage had returned to the preischemic level in both flap types.

Regional Capillary Blood Flow

The reduction in regional capillary blood flow was significantly more pronounced in venous stasis flaps than in arterial insufficiency flaps after partial ischemia (p < 0.05) (Fig. 3). During reperfusion, regional capillary blood flow was significantly higher in arterial insufficiency flaps than in venous stasis flaps (p < 0.01).

The blood flow was equally distributed throughout the lengths of the flaps at all flow levels. The regional distribution of blood flow inside the flaps showed no tissue priority in the arterial insufficiency flaps, except that a temporary reduction in the muscular regional capillary blood flow was observed at the 50 percent blood flow level when compared with the preischemic level (p < 0.05) (Fig. 4). In venous stasis flaps, partial ischemia resulted in a net shift of blood flow from subcutaneous tissue to muscle at both

![Fig. 2. Arteriovenous shunting. Percentage of the flap blood flow running through arteriovenous shunts. Measurements were performed at a preischemic level 3 hours after elevation of the flaps, after reduction of the flap blood flow to 50 percent for 1 hour, after further reduction to 25 percent for 1 hour, and after 1 hour of total occlusion followed by 1 hour of reperfusion. Ischemia was induced as isolated arterial ischemia in one of the two flaps (open bars) and at the same time an isolated venous ischemia in the other flap (closed bars). Values are mean ± SEM; n = 7. Significant difference from preischemic values (*p < 0.01; †p < 0.001). Significant difference between arterial and venous ischemia (*p < 0.01).](image)

![Fig. 3. Capillary blood flow (ml/min). See Figure 2. Arterial ischemia in one of the two flaps (open bars). Venous ischemia in the contralateral flap (closed bars). Values are mean ± SEM; n = 7. Significant difference between arterial and venous ischemia (*p < 0.05; †p < 0.01).](image)
Fig. 4. Capillary blood flow distribution (%) to skin, subcutaneous tissue, panniculus carnosus, and muscle in flaps subjected to arterial ischemia (left) and venous stasis (right). Measurements are performed before flow reduction (preischemia), at reduction in total flap flow to 50 and 25 percent, and after 1 hour of reperfusion. Significant difference between arterial and venous ischemia (*p < 0.05).
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partial ischemic blood flow levels (p < 0.05) (see Fig. 4).

DISCUSSION

This study has revealed a different hemodynamic response of myocutaneous island flaps to ischemia when an arterial etiology is compared with a venous etiology. Partial arterial ischemia resulted in a major blood flow reduction in A-V shunts. At the low flow level, A-V shunts carried, on average, 22 percent of the total blood flow, leaving 78 percent to the capillaries. In the venous stasis flaps, an almost parallel reduction in A-V shunt flow and capillary blood flow was observed. Compared with arterial ischemia, the incipient venous stasis was characterized by a relatively higher degree of A-V shunting, with A-V shunts carrying greater than 55 percent of the total blood flow at all flow levels and capillary blood flow being lower at all flow levels. Although this study only considered a relatively short ischemic insult, the data suggest that the poorer prognosis after venous problems than after an arterial insult may in part be due to these hemodynamic alterations.

Acute venous obstruction is known to be more damaging than acute complete pedicle obstruction in experimental skin flaps, as well as in myocutaneous flaps. There are several possible explanations for this observation. During venous obstruction, the microcirculation is subjected to a considerable increase in the intravascular pressure load. The resulting congestion and edema formation and the eventual breakdown of the endothelium are supposed to lead to an extrinsic compression of the microcirculation, with subsequent vascular collapse, an increased diffusion barrier for oxygen transport, and an increased risk of thrombosis in the microcirculation. Our flap model requires heparinization, and we are not able to assess the importance of thrombosis in the microcirculation. The edema formation in the venous stasis model results in a weight increase that is about 6 percent higher than after arterial insufficiency. The capillaries may be more susceptible to the external compression than the more thick-walled A-V shunts, and this is reflected in the different flow distribution pattern in the venous stasis flaps when compared with the arterial insufficiency flaps. The A-V shunting in flaps exposed to isolated venous stasis should be taken into consideration when venous blood samples are used in research for assessment of flap status and especially in comparing incipient arterial and venous ischemia. The normalization of the A-V shunt percentage during reperfusion suggests that precautions are less important in this stage. A-V shunt flow, which is abundantly present in skin flaps and in myocutaneous flaps, is controlled or modified by efferent nerve impulses, hormonal substances, oxygen tension, pH, intravascular pressure, and temperature. The current flap model is deprived of sympathetic nerves, and the changes in temperature, oxygen tension, and pH that normally are necessary to induce changes in A-V shunting have not been observed in this flap model. However, the persistent arterial inflow in cases of venous ischemia is able to induce maximal vasodilation.

Capillary blood flow in the arterial insufficiency flaps seems to support different tissue components equally, whereas the muscular capillary blood flow was favored at the expense of subcutaneous tissue in flaps subjected to a venous insult. The blood flow shift could theoretically be due to the greater vessel recruitment capacity in muscle or less edema formation in muscle compared with subcutaneous tissue. Although statistically significant, the clinical relevance of the internal redistribution may be minor.

The A-V shunt flow was normalized in both flaps after 1 hour of reperfusion, although the capillary blood flow was significantly lower in the venous stasis flaps than in the arterial insufficiency flaps. Venous outflow was still reduced in the venous stasis flaps compared with the arterial insufficiency flaps after 3 hours of reperfusion. In a previous study we found that venous outflow in myocutaneous flaps not subjected to ischemia shows a continuous increase throughout the first 9 postoperative hours. Hence the reflow in the venous ischemic flaps is slow compared both with flaps with arterial ischemia and with normal uncomplicated flaps. Reperfusion after 1 hour of arterial ischemia in skin island flaps is also more pronounced than that after venous stasis, and after prolonged venous stasis, the reflow is less when compared with arterial ischemia. This slow-reflow phenomenon may be induced by alterations in the vascular resistance, which can be explained by an inappropriate relationship between tissue pressure (edema), the intravascular pressure, and the rheologic changes secondary to the venous stasis (hemococoncentration). The pressure changes may lead to vessel collapse and impede blood flow. Viscosity is strongly dependent on changes in hematocrit, and the increased
hemoconcentration in venous stasis flaps can in part explain the slow reflow. Thrombosis occluding the microcirculation is potentially more pronounced in the slow-reflow situation. However, in the present study, thrombosis in the microcirculation may not play an important role because of the heparinization.

Two theories regarding the reactive hyperemia seen after arterial ischemia have been proposed. One is the metabolic hypothesis, which holds that slowly diffusible vasoactive substances accumulate in the extravascular space during ischemia. The vasodilation is continued into the reperfusion phase because the removal of these substances is very slow. The second theory, the myogenic hypothesis, states that secondary to the low transmural pressure after a proximal occlusion, vasodilation takes place and is maintained within the first period after reperfusion. The sustained intravascular pressure increase in venous stasis flaps may hinder the vasodilation and perhaps even initiate a protecting vasoconstriction. This vasoconstriction is independent of intact sympathetic nerve supply to the flap, and the phenomenon has been described in free skin flaps in humans.

The conclusions of the present study are as follows: (1) There is more A-V shunting in flaps exposed to partial venous stasis than in flaps exposed to arterial insufficiency. (2) The reperfusion after venous stasis is less than that observed after arterial insufficiency with respect to both total blood flow and capillary blood flow. After 1 hour of reperfusion, flaps subjected to arterial ischemia had normalized or showed hyperemic capillary perfusion, whereas flaps subjected to venous ischemia still had reduced perfusion. (3) We hypothesized that the capillary blood flow would be spared at the expense of A-V shunting. This was true for the arterial insufficiency flaps, where A-V shunt flow was relatively more decreased than capillary blood flow. However, venous stasis caused a relatively more pronounced decrease in capillary blood flow compared with shunt flow. In flaps exposed to isolated venous obstruction, a greater reduction in blood flow was necessary to close the A-V shunts. (4) The fourth hypothesis on protection of muscle flow at the expense of subcutaneous blood flow was not proven true for the arterial insufficiency flaps. In venous stasis flaps, a net shift in capillary blood flow from subcutaneous tissue to muscle during partial ischemia was observed. However, the changes were not striking, and it is disputable whether this phenomenon has any clinical significance.

The sluggish nutritional blood flow during incipient venous ischemia and the slow reflow after a short period of total venous occlusion give physiologic support for the worse prognosis when compared with arterial insults of the same duration and degree of flow reduction. However, the pathophysiologic mechanisms behind the different hemodynamic changes need further investigations.

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