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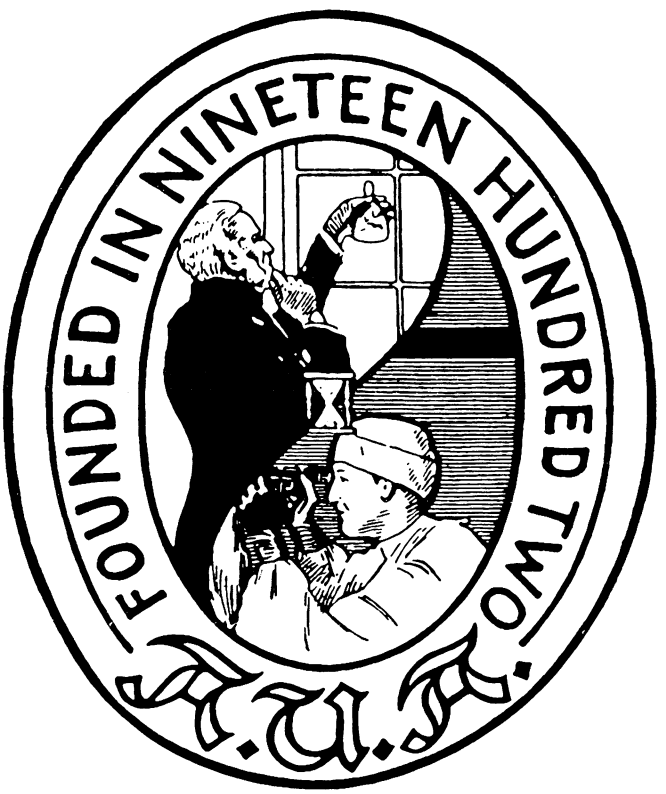


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# THE EFFECT OF VENOUS INCOMPETENCE AND ARTERIAL INSUFFICIENCY ON ERECTILE FUNCTION: AN ANIMAL MODEL

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ABSTRACT

We designed an animal model to elucidate the effect of venous leakage and arterial insufficiency on erectile function. In 10 dogs, electrodes were implanted around the cavernous nerves for electroerection and blood flow in the internal pudendal artery was recorded. Venous leakage was mimicked by inserting needles of varying gauges (30 to 16G) into the corpus cavernosum and the erectile response to neurostimulation was recorded before and after the creation of the leak. The relationship between the size and the amount of the venous leakage, the changes in the intracavernous pressure (peak and drop), and the changes in the peak and maintenance arterial blood flow were documented.

Arterial blood flow was then reduced by 25 and 50 per cent by means of a screw clamp on the terminal aorta. The erectile response to neurostimulation was again determined, with the same electrical parameters, first with reduced blood flow alone, then in combination with leakage of varying size.

Our results showed that minor cavernous vein leakage in the presence of normal arterial flow and a healthy sinusoidal system had a minimal effect on erectile function owing to a compensatory increase in penile blood flow. However, when reduction of arterial blood flow was superimposed on venous leakage, even of a minor degree, the erectile response to neurostimulation was markedly impaired. (*J. Urol., 144: 790–793, 1990*))

Many experiments have been devised to elucidate the relative contribution of the arterial and the venous systems to erectile capacity. In this century Lydston[1] and Lowsley and Bray[2] were the first to report the venous contribution to impotence, but later investigators attributed erection solely to increased arterial flow.[3,4] Accordingly, most attention was given to investigation of the arterial system by both noninvasive and invasive techniques.[5,6] The controversy regarding the role of the venous system had its basis in the conflicting results from the human penile perfusion studies of Newman and Reiss[7] and the $^{133}$-xenon wash-out studies of Shirai and associates[8] and Wagner and Uhrenholdt.[9]

Recent studies have clearly demonstrated that penile erection is the result of three synergistic hemodynamic events: increased arterial flow, sinusoidal relaxation, and venous outflow restriction.[10-12] With the recognition of the importance of both the arterial and venous systems and the frequency of both arterial insufficiency and venous incompetence as causes of organic impotence, we undertook this study to determine the contribution of each system in the hemodynamics of erection and the effect of impairment of each alone or in combination on erectile function in dogs.

## MATERIALS AND METHODS

Ten healthy adult mongrel dogs, weighing 20 to 30 kg., were used in this study. Dogs were premedicated with acepromazine (0.1 mg./kg. body weight [B.W.]) and anesthetized with intravenous sodium pentobarbital (30 mg./kg./B.W. with bolus injections of 25 to 50 mg./hr. as needed to maintain an adequate level of anesthesia and spontaneous respiration). Fluid intake consisted of intravenous infusion of normal saline solution (two ml./kg. B.W./hr.).

Through a midline incision from the xiphoid process to the pubic bone, the abdomen was opened and the bladder and prostate were exposed. By electrostimulation, the cavernous nerves were identified along the posterolateral aspect of the prostate, and bipolar cuff electrodes (Avery Laboratory) were placed around them to induce erection (fig. 1). The ipsilateral internal pudendal artery was exposed and an ultrasonic blood

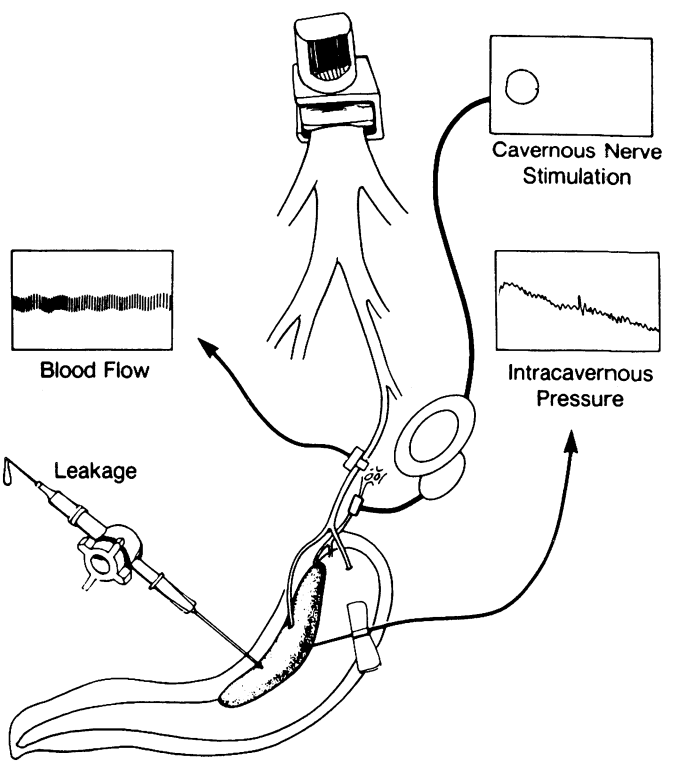


FIG. 1. Schematic drawing of experimental set-up.

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TABLE 1. *Effect on flow within the internal pudendal artery of leakage produced by needles of varying gauges*

| Needle Gauge | Arterial Flow | |
|---|---|---|
| | Peak* | Maintenance† |
| | (ml./min.) | |
| none | 46.3 ± 7.3 | 22.4 ± 2.5 |
| 30 | 46.3 ± 6.6 | 26.2 ± 3.3 |
| 27 | 46.6 ± 5.9 | 32.7 ± 7.3 |
| 25 | 45.7 ± 5.5 | 33.7 ± 4.4 |
| 22 | 47.3 ± 6.6 | 33.6 ± 5.4 |
| 19 | 48 ± 6.1 | 32.9 ± 1.8 |
| 16 | 48.1 ± 6 | 33 ± 3 |

All values represent the mean ± S.E. in 10 dogs.
* Change in peak flow with different gauges was not statistically significant.
† Increase in maintenance flow with different gauges was statistically significant at 95% by both Student's *t* test and Fisher PLSD test. p = 0.01; F = 2.962.

TABLE 2. *Effect on intracavernous pressure of leakage produced by needles of varying gauges*

| Needle Gauge | Intracavernous Pressure* | | Detumescence† (sec.) |
|---|---|---|---|
| | Peak (cm. $H_2O$) | Drop from Control Peak (%) | |
| None | 139.4 ± 6.6 | | 83.2 ± 19.1 |
| 30 | 123 ± 6.5 | 12.0 | 73.8 ± 15.2 |
| 27 | 112 ± 6.5 | 20 | 47 ± 11.1 |
| 25 | 110 ± 6 | 21.4 | 35 ± 11.8 |
| 22 | 87 ± 6 | 37.8 | 32 ± 8.4 |
| 19 | 63 ± 7.3 | 55 | 28 ± 9.1 |
| 16 | 57.1 ± 9.6 | 59.2 | 14.6 ± 2.9 |

All values represent the mean ± S.E. in 10 dogs.
* All control vs different gauges statistically significant at 95% by both *t* test and Fisher PLSD. p = 0.0001; F = 49.258.
† All control vs different gauges statistically significant by both tests. p = 0.0001; F = 9.173.

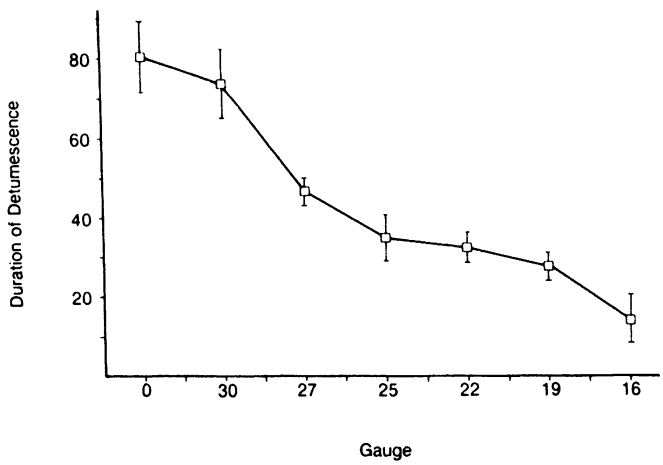


FIG. 2. Reduction in duration of detumescence (sec.)

TABLE 3. *Effect of reduced blood flow on intracavernous pressure*

| | Blood Flow (ml./min.) | Latent Period* (sec.) | Intracavernous Pressure† | | Detumescence‡ (sec.) |
|---|---|---|---|---|---|
| | | | Peak (cm. $H_2O$) | Drop from Control Peak (%) | |
| Normal | 9 | 29.6 ± 2.6 | 139.4 ± 7.7 | | 83.25 ± 19.1 |
| 25% reduction | 6.75 | 42.4 ± 3.4 | 96 ± 7.3 | 31 | 45.8 ± 5.8 |
| 50% reduction | 4.5 | 56.8 ± 5.3 | 60.6 ± 4.8 | 57 | 39.7 ± 7.8 |

All values represent the mean ± S.E. in 10 dogs.
* Control vs 25, 50% statistically significant by Student's *t* test and Fisher PLSD. p = 0.0001; F = 20.839.
† Control vs 25, 50% statistically significant by both tests. p = 0.0001; F = 54.926.
‡ Control vs 25 and 50% not statistically significant.

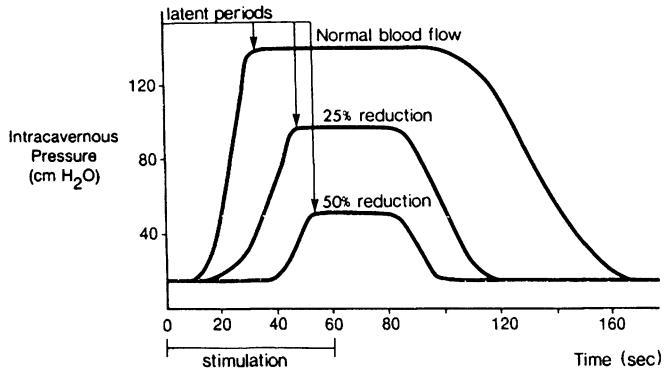


FIG. 3. Effect of reduction of arterial flow on duration of latent period and peak intracavernous pressure.

flow probe with a 1.5-mm. diameter (Transonic System, Inc.; Ithaca N.Y.) was placed to record arterial blood flow.

The entire penis was denuded, exposing both corpora cavernosa down to the ischial rami. A 21-gauge butterfly needle was placed in the corpus cavernosum and connected to a Statham pressure transducer for intracavernous pressure recording on a Grass polygraph. Systemic blood pressure was monitored through a 16-gauge angiocatheter placed in the femoral artery and connected to the same polygraph.

For reduction of penile blood flow, the terminal part of the aorta below the inferior mesenteric artery was dissected circumferentially and a screw clamp (Bel-Art) capable of controlling flow in flexible tubing up to 14 mm. (outer diameter) was placed around it. Once all monitoring and stimulating devices were in place, an intravenous bolus of 1000 U sodium heparin was delivered for anticoagulation with a maintenance dosage of 50 U to ensure that all pressure-recording cannulae remained patent.

Baseline measurements of the arterial blood flow and intracavernous pressure were recorded. Erection was induced by cavernous nerve stimulation and the following were measured: peak and maintenance penile blood flow; latency period (time from the beginning of stimulation until the maximal intracavernous pressure was attained); rise in intracavernous pressure; and the duration of detumescence (time from cessation of stimulation to return to baseline intracavernous pressure).

*Venous leakage study.* For creation of cavernous leakage, needles of different sizes (30 to 16G) were inserted into one corpus cavernosum. Previous heparinization helped to keep the needles patent. Electrostimulation of the cavernous nerve was repeated with each needle with the same stimulation parameters as in the control study. In addition, the volume of blood leaking from the corpus cavernosum during the period of stimulation was measured.

*Reduction of arterial blood flow.* Penile blood flow was reduced by 25 and 50 per cent by means of the screw clamp around the terminal aorta, and neurostimulation was repeated.

*Venous leakage and reduction in arterial flow.* The erectile response to neurostimulation was recorded again in the presence of both cavernous leakage and reduced penile blood flow with the same parameters as in the control study.

Data were analyzed with both Student's *t* test and analysis of variance (ANOVA) Fisher PLSD test, and values are expressed as the mean ± S.E.

## RESULTS

An immediate response to cavernous nerve stimulation was sinusoidal relaxation, evidenced by a slight drop in the basal intracavernous pressure and a marked increase in blood flow through the internal pudendal artery to a peak of almost six times the basal flow rate (seven ml./min.). The arterial flow then dropped to a maintenance level of an average of 22.5 ml./

min. and the intracavernous pressure gradually rose to an average peak of 140 cm. $H_2O$ with an average latency period of 32.8 sec. After stopping the stimulation, the intracavernous pressure gradually dropped to baseline after an average of 80.6 sec. (detumescence period).

*Venous leakage.* In the presence of small venous leakage (produced by 25 to 30 G needles), the peak arterial flow in the internal pudendal artery was not significantly different from the control value (table 1). However, the maintenance arterial flow gradually showed a compensatory increase to a peak of 33.7 ml./min. (control = 22.4 ml./min.) with the 25G leak and remained at that level with increasing needle gauge. This was statistically significant at 95% by both Student's *t* test and the Fisher PLSD test (p = 0.01, F = 2.962). This compensatory increase in the maintenance blood flow was reflected in the changes in intracavernous pressure: leakage created by needles up to 25G resulted in only a slight drop in pressure from 139.4 cm. $H_2O$ to an average peak of about 110 cm. $H_2O$ (21.4 per cent) (table 2). At this point, with the maintenance arterial blood flow at its peak, a further increase in the size of the leak resulted in a steeper drop in the peak intracavernous pressure to about 57 cm. $H_2O$ with the 16G needle (60 per cent reduction from control).

In addition to the reduction in the peak intracavernous pressure, we also noticed a reduction in the duration of detumescence from 83.2 sec. in the control study to 14.6 sec. in the presence of 16G leakage (fig. 2).

*Reduction of arterial flow.* When the blood flow in the aorta was reduced by 25 per cent (normal nine ml./min.), the first change noticed on cavernous nerve stimulation was a marked increase in the latent period to an average of 42.4 sec. (table 3) and a further prolongation to 56.8 sec. when the blood flow was reduced to 50 per cent (fig. 3). The effect on the peak intracavernous pressure was directly related to the percentage of reduction in the arterial blood flow: 96 cm. $H_2O$ at 25 per cent and 60.6 cm. $H_2O$ at 50 per cent. Interestingly, the duration of detumescence was also affected with reduction of the arterial blood flow alone, from an average of 83.2 sec. to 39.7 sec. with 50 per cent reduction.

TABLE 4. *Effect of combined cavernous leakage and reduced arterial flow on intracavernous pressure*

| Needle Gauge | Intracavernous Pressure | | | | Detumescence (sec.) | |
|---|---|---|---|---|---|---|
| | Peak (cm./$H_2O$) | | Drop from Control Peak (%) | | | |
| | 25%* | 50%† | 25% | 50% | 25%‡ | 50%§ |
| 30 | 69.7 ± 3.6 | 47.8 ± 6.8 | 49.7 | 66 | 40 ± 8.9 | 24.8 ± 5.4 |
| 27 | 58.8 ± 5.3 | 38.2 ± 5.6 | 57.6 | 77 | 37.6 ± 9.7 | 19 ± 3.8 |
| 25 | 49.7 ± 4.5 | 32.0 ± 6.4 | 64 | 78 | 34.1 ± 7 | 17.5 ± 3.8 |
| 22 | 43.7 ± 1.7 | 25 ± 7.6 | 68 | 83 | 24.5 ± 7.6 | 15.7 ± 3.5 |
| 19 | 33.5 ± 4.2 | 19.4 ± 7.2 | 76 | 90.2 | 18.0 ± 3.2 | 9 ± 3.5 |
| 16 | 26 ± 5.4 | 12 ± 4.4 | 80.7 | 93.3 | 10.5 ± 2.7 | 5.5 ± 2.3 |

All values represent the mean ± S.E. in 10 dogs.

* Control vs 25% reduction statistically significant at 95% by Student's *t* test and Fisher PLSD. p = 0.0001; F = 54.248.

† Control vs 50% reduction statistically significant at 95% by both tests. F = 61.08, p = 0.0001.

‡ Control vs 25% statistically significant. p = 0.0002; F = 6.245.

§ Control vs 50% statistically significant. p = 0.000; F = 9.099.

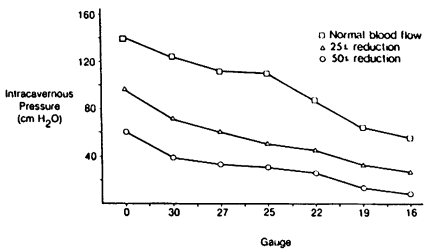


FIG. 4. Effect of cavernous leakage and reduction of arterial flow on peak intracavernous pressure.

TABLE 5. *Venous leakage**

| Needle Gauge | Arterial Blood Flow | | |
|---|---|---|---|
| | Normal Flow | 25% Reduction | 50% Reduction |
| 30 | 0.1 | 0.07 | 3 drops |
| 27 | 0.49 | 0.3 | 0.2 |
| 25 | 1.2 | 0.82 | 0.6 |
| 22 | 2.8 | 1.5 | 1.1 |
| 19 | 5.9 | 3.2 | 2.35 |
| 16 | 7 | 4.8 | 3.2 |

All values represent the mean ± S.E. in 10 dogs.

* In ml./min.

*Venous leakage and reduction of arterial flow.* When venous leakage was combined with reduction in arterial blood flow, the erectile response was markedly impaired (table 4). The effect on the latent period was similar to that induced by the reduction of arterial flow alone: it was prolonged by 43 and 63 per cent with a 25 and 50 per cent reduction, respectively. The changes in the peak intracavernous pressure were dramatic. Minor venous leakage alone (created by a 30G needle) had initially resulted in a very slight drop (from 139.4 to 123 cm. $H_2O$); however, when a 25 per cent reduction of arterial blood flow was superimposed, the peak intracavernous pressure dropped to almost 50 per cent of the control (to 69.7 cm. $H_2O$) (fig. 4). When the blood flow was further reduced to 50 per cent of normal, the same size leakage almost completely abolished the erectile response (an average peak of only 47.8 cm. $H_2O$). In addition, the shortening of the period of detumescence was also more pronounced. In the presence of a 30G leak alone, the duration had decreased from 83.2 sec. in the control to 73.8 sec., whereas with a 25 and 50 per cent arterial flow reduction, it was 40 and 24.8 sec., respectively. This decrease in the duration of detumescence was enhanced with increasing leakage size until it reached 10.5 and 5.5 sec. in the presence of 16G leakage and 25 and 50 per cent reduction in arterial flow.

*Volume of venous leakage with normal and reduced blood flow.* In table 5 are tabulated the average amounts of blood leaking from the corpus cavernosum during the period of stimulation in the presence of needles of different gauges and both normal and reduced blood flow.

## DISCUSSION

The results presented in this study further confirmed the role of both the arterial and venous systems in the hemodynamics of erection.

When cavernous leakage was created by inserting needles of different size into the corpus cavernosum, we noticed that the effect on erection depended not only on the size of the leak but also on the efficiency of penile arterial blood flow. All dogs had exhibited a good erectile response to control neurostimulation—evidence of healthy sinusoidal smooth muscle. In these animals minor venous leakage (30 to 25G) with unimpaired arterial flow had only a minor effect on the erectile response to neurostimulation owing to a compensatory increase in the maintenance arterial blood flow. However, when the size of the leakage increased beyond the compensatory capacity of the arterial flow (22 to 16G), the peak intracavernous pressure demonstrated a steeper drop to approximately 57 per cent of the control with the 16G needle. In contrast to these results (a noticeable response only to a large leak), a significant decrease in the duration of detumescence was seen with even a small leak. Thus, in the presence of mild to moderate venous leakage, the increase in the maintenance flow and the decrease in the duration of detumescence were the earliest parameters affected. This is in agreement with our previous report on patients with venogenic impotence.[13] In that study, we found these same parameters to be the most sensitive on cavernosometry in the

diagnosis and quantification of mild to moderate venous leakage.

This first part of our study might resemble the clinical situation of venogenic impotence in young patients with a normal arterial system. Usually this condition is asymptomatic unless the leak is very large or arterial insufficiency is superimposed, in which case inability to maintain a prolonged erection is the usual complaint. Similar findings have also been reported by others.[14,15]

When the arterial blood flow was reduced in the presence of normal venous and sinusoidal systems, the decrease in the erectile response to neurostimulation was proportional to the percentage of reduction: the average peak intracavernous pressure dropped by 31 and 63 per cent when the blood flow in the aorta was reduced by 25 and 50 per cent respectively. In addition, the latent period was markedly prolonged. These results are similar to our previous findings on the effect of acute penile artery occlusion[16] and are in agreement with several clinical studies reporting a delayed weak response to intracavernous injection of vasoactive agents in patients with arterial disease.[17-19] Interestingly, we found that with the reduction in arterial blood flow the duration of detumescence was significantly reduced—by about 34 and 53 per cent with 25 and 50 per cent reduction, respectively. In other studies in our laboratory in which detumescence was characterized as tri-phasic, the first phase was determined to be dependent mainly on arterial flow (manuscript in preparation). Reduction will abolish this phase, and may provide an explanation for our results.

When both venous leakage and arterial insufficiency were combined, the erectile response was markedly impaired. Minor venous leakage (created by a 30G needle) or a 25 per cent reduction in arterial flow alone resulted in a slight decrease in erectile response; when combined, they caused a 49.7 per cent decrease in peak intracavernous pressure, close to that induced by either 16G leakage or a 50 per cent reduction alone.

These findings draw attention to the importance of evaluating the competence of both the arterial and venous systems when a vascular origin is suspected.[20-23] Two main factors underlie this recommendation. First, evaluation of one system alone might reveal mild impairment that would otherwise require no surgical treatment (especially true in patients with mild venous leakage, which usually does not require surgical intervention). Second, the degree of impairment of each system will influence the choice of surgical treatment.[24] If the venous system alone is impaired, venous ligation would be the appropriate procedure; [25] if arterial insufficiency is the cause, a balloon dilation or arterial revascularization procedure may be indicated.[26-27] If both systems are impaired, a procedure that will increase both arterial flow and venous resistance should be considered: deep dorsal vein arterialization[22,28,29] or cavernous vein arterialization.[30]

From this study, we conclude: 1) both the arterial and venous systems play an important role in the hemodynamics of erection; 2) mild impairment of the venous system alone can be compensated for to some extent by other systems; and 3) diagnostic evaluation of patients with vasculogenic impotence should include assessment of both the arterial and venous systems for proper quantification of the degree of impairment of each system and better selection of surgical treatment. Investigation should begin with the arterial system because, if adequate, it has the potential to compensate for minor venous leakage.[19] Moreover, treatment of minor venous leakage may not prove beneficial to the patient if an existing arterial insufficiency has not been uncovered and treated.

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