VASCULAR PRESSURE MONITORIZATION FOR NECESSITY OF VASCULAR AUGMENTATION IN A RAT EXTENDED ABDOMINAL PERFORATOR FLAP MODEL

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In case blood perfusion compromises, vascular enhancement with arterial supercharge or venous superdrainage can increase viability of the flap. In this study, vascular pressure monitorization was used in a rat extended abdominal perforator flap model to reveal intraoperative vascular compromise and the need for vascular augmentation. A rat abdominal perforator flap was designed, which was based on the right second cranial perforator of epigastric artery. Vascular pressures of the flap were monitored continuously for 60 min, by catheters placed in the right superficial inferior epigastric artery and vein. Forty rats were divided into four experimental groups, as follows: group 1 ($n = 10$, no vascular augmentation), group II ($n = 10$, arterial supercharge), group III ($n = 10$, venous superdrainage), and group IV ($n = 10$, arterial and venous augmentation). Arterial supercharge and/or venous superdrainage were performed by using the left superficial inferior epigastric artery and vein. After the rats were sacrificed on the 7th day, total flap area and necrotic regions were evaluated. Mean arterial blood pressure was found significantly lower ($P < 0.05$) and mean venous blood pressure was measured significantly higher ($P < 0.05$) in group I than the groups II, III, and IV. Flap survival area was also larger in the groups II, III, and IV than the group I ($P < 0.05$). The results of this experimental study demonstrate that arterial insufficiency and venous congestion are almost always present in the rat extended abdominal perforator flap model, similar to deep inferior epigastric perforator flap. When such an extended perforator flap is used, arterial and venous pressure monitorization may be considered as a tool to support intraoperative clinical findings to reveal the need of vascular augmentation and ascertain flap viability. © 2012 Wiley Periodicals, Inc. Microsurgery 32:303–308, 2012.

Flap necrosis often occurs in the distal parts of the flaps due to the tissue ischemia or venous congestion, leading to a clinical problem related to coverage of the defect area. Many researches have focused on improving flap survival and avoiding tissue necrosis by increasing the blood supply or venous drainage. Although many techniques have been advocated to improve the survival of skin flaps, flap necrosis is still an important issue in reconstructive surgery. Deep inferior epigastric perforator (DIEP) flap has been widely used clinically as a useful autologous tissue source for soft tissue reconstruction of the breast mound. However, in DIEP flap technique, compromised flap circulation particularly in the distal area such as zone IV is still a challenging problem waiting to be solved.[1–4] In studies using the rat abdomen perforator flap which mimicks DIEP flap, it has showed that vascular augmentation of the flaps with arterial supercharge or venous superdrainage provided a greater flap survival rate, when flap ischemia or venous congestion existed.[5–11] Intraoperative detection of the changes of flap perfusion seems to be important in deciding the necessity of vascular augmentation, resulting in successful flap surgery.

In this study, we aimed to investigate whether arterial insufficiency and/or venous congestion was present in an extended perforator-based abdominal flap model in rats and whether arterial supercharging and/or venous superdrainage was necessary to ascertain flap viability, using arterial and venous blood pressure monitorization.

MATERIALS AND METHODS

In this study, 40 male Wistar rats, weighting 300 and 350 g, were randomly divided into four groups ($n = 10$ each). The rats were housed in individual cages and fed standard rat chow and water ad libitum upon completion of the experiment. This experiment was approved by the Ethical Committee of University for Animal Researches.

After the rats were anesthetized with intramuscular injections of ketamine 10 mg kg^{-1} and subcutaneous injections of xylazine hydrochloride 3 mg kg^{-1}, the abdomens were depilated. A supplement dose was given in case it was required during the experiment. A rat abdominal perforator flap of 6×6 cm^2 was designed, which was based on the right second cranial perforator of epigastric artery penetrating the rectus abdominis muscle. This flap design was an extended version of the abdominal flap model described by Oksar et al. to fabricate a DIEP flap in the rats.[3] Abdominal perforator flap was raised from lateral to medial under the panniculus carnosus (Fig. 1). The second fascial perforator on the right rectus abdominis muscle was found and protected as a main pedicle of the flaps in all groups of the experiment and contralateral superficial inferior epigastric vascular

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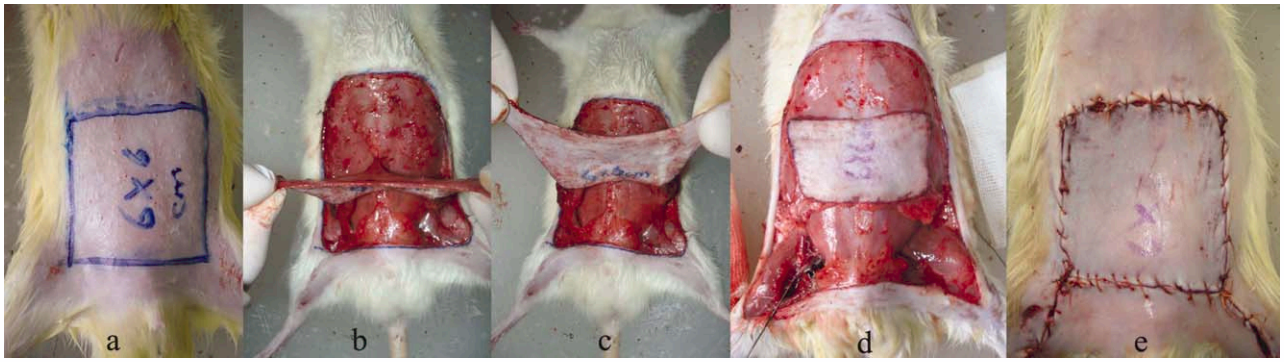

Figure 1. ***a****. Marking of the perforator based abdominal flap with which a rat abdominal perforator flap of 6 × 6 cm^2 was designed, which was based on the right second cranial perforator of epigastric artery.* ***b****. Appearance of the right second cranial perforator of the epigastric artery as a main pedicle after the flap dissection had been completed.* ***c****. Left and right superficial epigastric vessels. The left superficial inferior epigastric artery and vein were used for arterial and venous augmentation and the right ones were prepared for pressure measurement.* ***d****. Vascular pressure monitorization by means of placing the pressure catheters into the left femoral artery and vein.* ***e****. The flap was sutured back in place. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]*

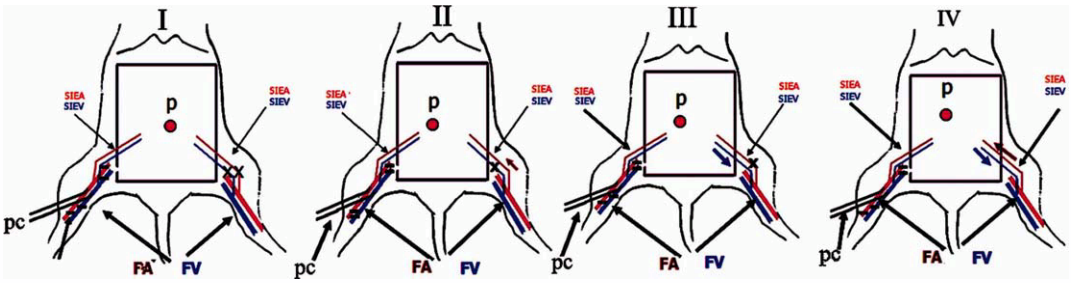

Figure 2. Schematic illustration of the experiment. In all flaps, main pedicle was the right second cranial perforator of epigastric artery (p). I represents group I in which the left superficial inferior epigastric artery and vein were ligated, so flap was perfused only by main pedicle during the pressure monitorization. II demonstrates group II in which the left superficial inferior epigastric vein was ligated so that the left SIEA augmented the flap blood supply in the experiment. III shows group III in which the left SIEA was ligated and venous superdrainage was provided by leaving the left SIEV intact in its place. IV represents group IV in which both the left SIEA and SIEV were in continuity with the flap, providing arterial and venous augmentation when arterial and venous pressures of the flap were measured through the pressure catheters (pc). [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

pedicle was dissected in groups II, III, and IV whereas all vessels of contralateral superficial inferior epigastric pedicle were cut and coagulated with a bipolar device in the control group (group I). The right (ipsilateral) superficial inferior epigastric pedicle and femoral artery and vein were then dissected in all experiment groups. The right femoral artery and vein were ligated proximal to the superficial inferior epigastric pedicle to monitor the pressures of the superficial inferior epigastric artery (SIEA) and vein (SIEV). In group II, the left superficial inferior epigastric artery was preserved for arterial supercharging, whereas its vein was cut and ligated. In group III, the left superficial inferior epigastric vein remained to be intact for venous superdrainage, but its artery was cut and ligated. In group IV, both the left superficial inferior epigastric artery and vein were left intact for arterial and venous augmentation (Fig. 2).

Catheters (P23IdD; Statham Gould, Oxnard, CA, 0.7 mm) were inserted into the right femoral artery (FA) and vein (FV) at the distal to the origins of the SIEA and SIEV. The pressure changes of the SIEA and SIEV were monitored via these catheters, which reflected the pressure of the flap vascular network. The flap was then sutured in place and followed for viability during 7 days.

After the pressure catheters were connected to a fluid pressure transducer (P23ID, Statham Gould, Oxnard, CA), zero calibrations were made at the level of the right atrium. Pressure changes of the right superficial inferior epigastric artery and vein were then recorded simultaneously with a computerized data acquisition system (PowerLab, ADInstruments, Australia) for 60 min after the flap inset (Fig. 3). Also data of 15th, 30th, 45th, and 60th min were recorded separately for comparison between the groups. After the rats were sacrificed on the

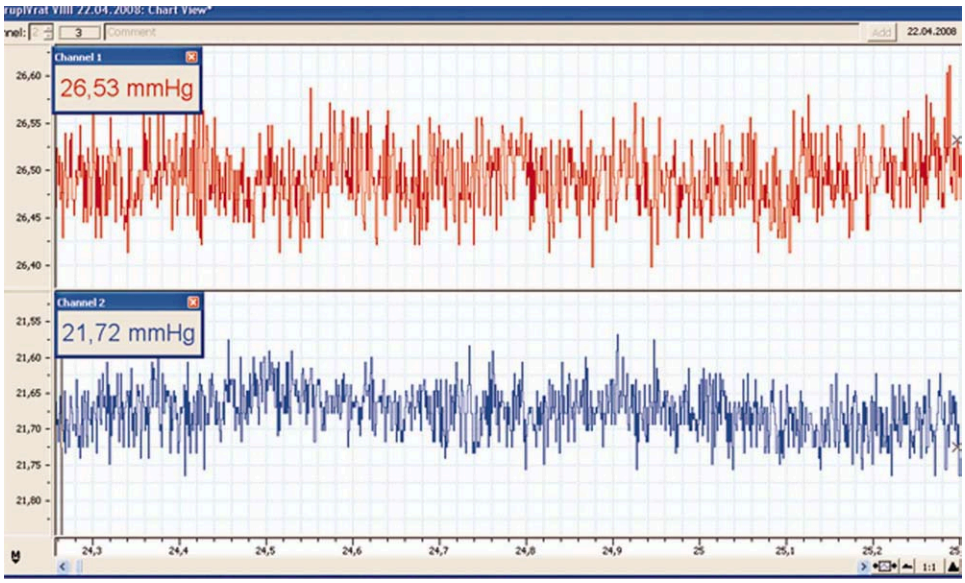

Figure 3. A representative tracing of the arterial (above) and venous (below) pressures during the measurement. Arterial and venous pressures were stable during the experiment, neither significant elevation nor considerable decline in the vascular pressures was recorded during the experiment. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

Table 1. Superficial Inferior Epigastric Artery Pressures Varying With Time (mmHg) and Statistical Values

Groups	15 min	30 min	45 min	60 min	*X* and *P*
I	4.97 ± 4.03	5.61 ± 7.09	5.39 ± 6.07	6.32 ± 5.08	$X = 6.15$; $P = 0.049$*
II	29.03 ± 8.97	25.07 ± 8.54	24.35 ± 5.60	23.19 ± 5.65	$X = 7.90$; $P = 0.048$*
III	20.46 ± 9.86	16.19 ± 6.13	18.06 ± 7.65	17.98 ± 6.84	$X = 5.88$; $P = 0.118$
IV	27.74 ± 5.14	25.72 ± 4.72	23.61 ± 5.14	23.61 ± 5.14	$X = 2.16$; $P = 0.540$
Results	$K_w = 23.43$; $P = 0.000$*	$K_w = 23.19$; $P = 0.000$*	$K_w = 21.22$; $P = 0.000$*	$K_w = 22.14$; $P = 0.000$*	

Data were represented as mean ± SD.
*$P < 0.05$; Kruskal Wallis test, Mann Whitney U test, Friedman test, and Wilcoxon test.

7th day, total flap area and necrotic regions were marked on clear acetate templates and these were electronically scanned, and then the area of flap survival for each rat (cm^2) was determined by using SPSS software.

For the statistical analysis, $P < 0.05$ was regarded as indicating statistical significance and data are expressed as the mean ± SD. Kruskal Wallis Test, Mann Whitney U Test, Friedman Test, and Wilcoxon Test were used to detect the differences between the groups.

RESULTS

Both arterial and venous vascular pressures were monitored during the experiment for 60 min in all flap groups. Mean arterial blood pressure in all of time intervals, was significantly lower in the group I (6.32 ± 5.08 mmHg) when compared with the groups II (23.19 ± 5.65 mmHg), III (17.98 ± 6.84 mmHg), and IV (23.31 ± 5.48 mmHg) ($P < 0.05$). Arterial pressures measured at 15th, 45th, and 60th min were observed to be significantly lower in the control group than those in the groups II, III, and IV. However, there was no significant difference in arterial pressures among the groups II, III, and IV. Similarly, arterial pressure at the 30th min was lower in the control group (5.61 ± 7.09 mmHg) than the other groups, whereas statistically significant differences in the arterial pressures were observed between groups II (25.07 ± 8.54 mmHg) and III (16.19 ± 6.13 mmHg), and between groups III and IV (25.72 ± 4.72 mmHg) due to the lower arterial pressure of the group III ($P = 0.000$), (Table 1). Mean venous blood pressure in all of the time intervals remained to be significantly higher in group I (21.53 ± 5.56 mmHg) when compared with the groups II (16.44 ± 1.73 mmHg), III (11.87 ± 3.0 mmHg), and IV (9.81 ± 4.42 mmHg), suggesting that vascular augmentation led to a decrease in venous pressures ($P = 0.001$). However, there were no significant differences in venous pressures among the groups II, III, and IV. Venous pressures measured at the 15th min were not significantly different among the groups, showing a gradual decrease in

Table 2. Superficial Inferior Epigastric Vein Pressures Varying With Time (mmHg) and Statistical Values

Groups	15 min	30 min	45 min	60 min	*X* and *P*
I	20.15 ± 7.08	20.57 ± 6.44	20.34 ± 4.67	21.53 ± 5.56	$X = 1.44$; $P = 0.696$
II	16.08 ± 2.04	15.98 ± 1.51	15.97 ± 1.56	16.44 ± 1.73	$X = 0.36$; $P = 0.948$
III	13.27 ± 3.84	11.71 ± 2.36	11.15 ± 2.15	11.87 ± 03.00	$X = 6.84$; $P = 0.77$
IV	14.63 ± 9.19	14.03 ± 9.73	12.75 ± 7.35	9.81 ± 4.42	$X = 7.20$; $P = 0.066$
Results	$K_w = 5.98$; $P = 0.113$	$K_w = 15.81$; $P = 0.001$*	$K_w = 18.98$; $P = 0.000$	$K_w = 26.87$; $P = 0.000$*	

Data were represented as mean ± SD.
*$P < 0.05$; Kruskal Wallis test, Mann Whitney U test, Friedman test, and Wilcoxon test.

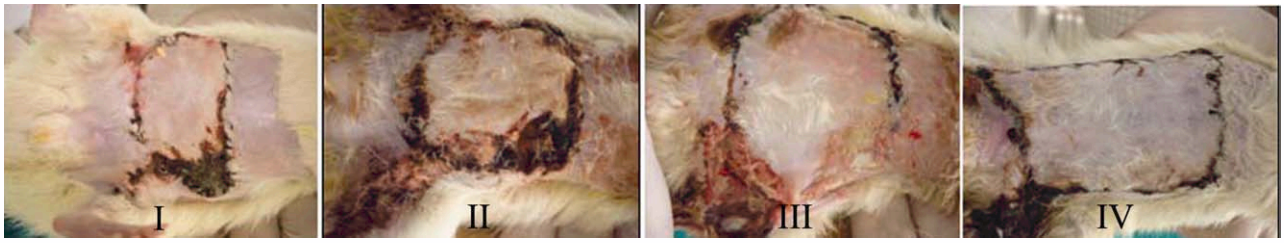

Figure 4. Appearance of the perforator flaps on postoperative 7th day. As flap survival considerably increased in all groups enhanced by the way of a vascular augmentation procedure, in the group I the least flap survival area was observed on that day. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

venous pressure after the first 15 min. There were significant differences among all of the groups in the venous pressures of 30th, 45th, and 60th min. Significant difference was also observed between the group I (20.57 ± 6.44 mmHg) and III (11.71 ± 2.36 mmHg) at the 30th min ($P = 0.000$), and between the group II (16.44 ± 1.73 mmHg) and IV (9.81 ± 4.42 mmHg) at the 60th min ($P = 0.012$) (Table 2).

Individual variations in the extent of the vascular pressure changes were not observed in the groups regarding different time intervals. Arterial and venous pressures were stable during the experiment, neither significant elevation nor considerable decline in the vascular pressures was recorded during the experiment. Venous pressure measurements in 15th, 30th, 45th, and 60th min showed no statistically significant difference among the groups. Arterial pressure values of groups III and IV had no statistically significant difference during the time intervals of the experiment, however; in the groups I (4.97 ± 4.03, 5.61 ± 7.09, 5.39 ± 6.07, 6.32 ± 5.08 mmHg) and II (29.03 ± 8.97, 25.07 ± 8.54, 24.35 ± 5.60, 23.19 ± 5.65 mmHg), *P* values were found to be 0.049 and 0.048 respectively, showing a statistical difference.

Flap survival area was significantly higher in the groups II (31.40 ± 4.83 cm^2), III (32.50 ± 3.13 cm^2), and IV (31.80 ± 3.52 cm^2) compared to the group I (control) (23.60 ± 7.32 cm^2) ($P = 0.022$). Flap survival considerably increased in all groups enhanced by the way of a vascular augmentation procedure except group I, however; there was no statistically significant difference among the groups II, III, and IV (Figs. 4 and 5).

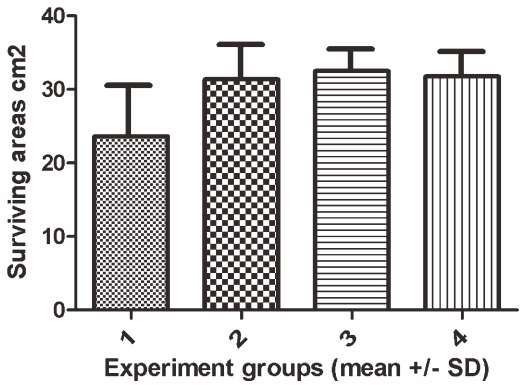

Figure 5. Comparison of mean survival areas (±SD) among the groups 7 days after flap elevation. Note that flap survival area was significantly higher in the groups II, III, and IV compared to the group I (control), so statistically significant difference was observed between the control group and the others (P = 0.022).

DISCUSSION

Perforator flaps have been used as autologous tissue sources for soft tissue reconstruction. Compromised flap circulation and flap necrosis, especially in the distal area of the extended flaps are still among the major drawbacks of the technique, which usually result from either arterial insufficiency or venous congestion, or both. To overcome these obstacles, arterial supercharge and venous superdrainage have been shown as both clinically and experimentally effective techniques, but timing and necessity of these are obscure during the perforator flap surgery, although there are many data about clinical findings sug-

gesting the need of arterial supercharge and venous superdrainage.[4–8] In this experiment, there was an attempt to reveal a pressure-based evidence for necessity of vascular augmentation. We considered that measurement of vascular pressures far from the vascular pedicles in this flap might be a reliable and safe way to determine pressure changes of flap vascular network reflecting the intraparenchymatous pressure and to decide the necessity of vascular augmentation, as an increase or decrease in vascular pressure might be considered to be a sensitive parameter indicating either venous congestion or arterial insufficiency, or both. The pressures of the right superficial inferior epigastric artery and vein represented pressures of flap vascular network and reflected intraparenchymatous arterial and venous blood pressures, as this pedicle was in continuity with only flap vascular network in this experiment model. After the dissection of the superficial inferior epigastric pedicle, right femoral artery and vein were ligated proximal to it, so that superficial inferior epigastric pedicle could be left with the flap vascular network alone.

As perforator flaps usually go into a shock phase in the acute term, which can be seen in clinical monitoring, and show a stable hemodynamic state after a couple of days,[12,13] pressure monitoring for a longer time period seems to be more useful to reveal whether arterial supercharge and/or venous superdrainage is really necessary for hemodynamic stability in this model, however; present study aimed to detect intraoperative pressure changes in the acute phase to detect the problem intraoperatively and to overcome the flap ischemia or venous congestion, by using vascular augmentation. On the other hand, it would be impossible to keep the animals stable under general anesthesia for longer than 3 or 4 h. In the present study, 1 h of monitorization was chosen to simulate the clinical settings intraoperatively by starting to measure vascular pressure just 1 h after flap inset.

Effectivity of arterial supercharge and venous superdrainage in flap survival has been emphasized in many clinical and experimental studies.[5–9] In a rat deep inferior epigastric perforator flap model, Hallock showed that venous supercharging provided greater flap survival by means of an alternative venous outflow tract. It is advisable for perforator flaps to overcome venous congestion.[5] In a study of Rooks et al. on the effect of venous hypertension on rabbit free flap survival, a correlation between the flap failure and venous hypertension was determined, suggesting that venous superdrainage resulted in greater free flap survival.[9] In our study, group III which had the venous superdrainage, showed significantly higher arterial blood pressure than the control group, suggesting that reduced venous pressure led to increase in arterial blood perfusion because of the decline in venous congestion and peripheric vascular resistance. This finding also proves that venous congestion is already present in this flap model, and venous superdrainage overcomes it successfully, resulting in increase in arterial blood perfusion and pressure. Greater flap survival in the group with venous superdrainage may arise from increased blood perfusion due to venous superdrainage.

It has been well known that in some specific situations where venous congestion or arterial insufficiency exist, vascular augmentation is the most effective way in increasing flap survival in experimental and clinical practice.[4] When arterial augmentation is compared with venous superdrainage in a flap, arterial supercharging results in a higher survival rate, suggesting that ischemia leads to flap necrosis more frequently than venous congestion in the rat abdominal flaps.[10] In this study, our results related to arterial supercharge and venous superdrainage was similar to the previous studies. We found that the survival area with arterial supercharge was larger than that of venous augmentation and both had statistically significant differences with the control group.

The realization of the intraoperative flap ischemia and venous congestion which are mainly due to arterial insufficiency or venous congestion, or both, is crucial for the success of perforator flap surgery. Precautions for the salvage of hemodynamically critical distal part of the flap should be taken immediately in the operation such as excision of the distal flap, using double pedicle, arterial supercharge and venous superdrainage. In deciding the need for one of these procedures, arterial and venous pressures of the flap may be considered as parameters reflecting the hemodynamical status of the flap, which also support the clinical findings such as the flap color and bleeding from the margin. Yamamoto et al. investigated the effect of vascular augmentation on the hemodynamics and survival area in a rat abdominal perforator flap model. They concluded that the venous pressure measurement was a reliable parameter for deciding the necessity of venous superdrainage.[10]

Our experiment was similar to Yamamoto's study considering its aim, but there were differences in his study design which can be criticized regarding the lack of standardization of the pressure measurement protocol and the timing of the measurement. Yamamoto's study seemed to be insufficient to reflect all aspects of vascular pressures of an extended perforator flap mimicking the DIEP flap. They used a rat abdominal perforator flap model described by Oksar.[3] Apart from our flap design which was an extended version of this model, they used the left superficial epigastric pedicle not only to monitor vascular pressure, but also to perform arterial supercharge and venous superdrainage. With this design, pressures of SIEV and SIEA may affect the pressures of femoral artery (FA) and vein (FV) due to the neighborhood of the vascular structures during vascular augmentation, resulting in unstable pressures and individual variations. The measured pressures were also only intravascular, and did not reflect intraparenchymatous vascular pressures. Changes in the SIEV and SIEA pressures were recorded

within a few minutes, while proximal sites of these vessels were opened and closed.[10] Therefore, it is difficult to give a decision on vascular pressures and the need for vascular augmentation depending on these measurements obtained within a few minutes. Appearance of considerable individual variations in the extent of pressure elevation was emphasized by Yamamoto,[10] which may be related to measurement of unstable vascular pressure. Moreover, as there were no differences in the vascular pressures among the three groups with neither of the manipulations, there was no comment on arterial pressures in the article. In our study, measurements of the pressures of artery and vein were made from the right superficial epigastric pedicle which was far from the flap vascular pedicles, possibly reflecting a more stable and actual intraparenchymatous pressure. In our present study, both arterial and venous pressures were recorded continuously for 60 min to obtain enough information on vascular pressure changes. There were no individual variations in the pressure monitoring. Mean arterial and venous blood pressures were found to be significantly different from the control group. The survival area of the flap also increased in the present study both in the groups of venous superdrainage and arterial supercharge whereas there was no change in flap survival area in the group of venous superdrainage in the study of Yamomoto et al.[10] Moreover, in our study, in the group IV in which both arterial supercharge and venous superdrainage were performed, survival area of the flap increased significantly, suggesting that all vascular augmentation procedures were effective on the survival of the flap in the present experimental model.

In a clinical study, Sakurai et al. monitored the intraparenchymatous venous pressure in various composite tissue grafts to ascertain flap viability at the time of surgery or for 3 days after the completion of surgery by placing a small-caliber catheter in the vein of transferred tissue.[11] They concluded that measurement of the venous pressure by means of a venous catheter was found to be accurate in assessing the patency of the venous channel and the viability of the transferred tissue.[11] In the present study, arterial and venous pressures were similarly monitored far from the pedicles of the flap and the same outcomes were observed.

In the control group of our study which had an extended abdominal flap without vascular augmentation, arterial insufficiency and venous congestion appeared in the flaps, resulting in a larger flap necrosis. Also the arterial pressures were measured to be decreased and venous pressures were increased in the superficial epigastric pedicle for 60 min. When a vascular augmentation procedure, such as arterial supercharge, venous superdrainage, or both were applied to this flap, venous pressure significantly decreased while measured arterial pressure was elevating. In this flap model, hemodynamic changes in flap circulation were monitored successfully, so in case of a decrease in arterial pressure or an elevation in venous pressure during the operation, arterial supercharge, venous superdrainage or both could be considered according to changes in the vascular pressures; otherwise there was a strong possibility of flap necrosis as occurred in the control group.

The results of this experiment demonstrate that arterial insufficiency and venous congestion is almost always present in the extended perforator-based rat abdominal flap model, similar to the extended DIEP flap. Our results also revealed that there was a clear correlation between the arterial supercharge and/or venous superdrainage and flap survival area and all vascular augmentation procedures increased the flap survival. It may be advised to perform arterial supercharge and/or venous superdrainage in an extended perforator flap after the arterial and venous pressure monitorization to ascertain flap viability, if clinical findings alone are not sufficient to decide on the need of vascular augmentation.

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