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Murine skin flap survival may not be affected by underlying fat viability☆

David L. Brown [a,b,c], **Anthony J. Penington** [a,b,*]

[a] *The Bernard O'Brien Institute of Microsurgery, Melbourne, Australia*
[b] *The Department of Surgery, St. Vincent's Hospital, Melbourne, Australia*
[c] *The University of Michigan, Ann Arbor, MI, USA*

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Summary One problem in the treatment of degloving injuries is the accurate prediction of the survivability of the avulsed tissue. Initial evaluation frequently underestimates the degree of eventual flap loss, and in many cases, there is a progressive necrosis that continues over the ensuing days. The pathophysiology of this phenomenon is unclear. We undertook this study to test the theory that underlying devascularised fat contributes to overlying skin necrosis.

A dorsal random skin flap model was used in the rat. Sixty-six rats were divided into three groups: flaps with viable fat and silicone sheeting underneath, flaps with devascularised fat and silicone sheeting underneath and control flaps with only silicone sheeting underneath. Flap necrosis (% area $\pm$ SEM) was evaluated at one week, and found to be $27.1 \pm 4\%$ in the live fat group, $33.2 \pm 4\%$ in the dead fat group and $33.6 \pm 5\%$ in the control group. One-way analysis of variance showed no statistically significant difference between the three groups at a power of 80%.

In this study, we have shown that neither live nor dead fat has a significant influence on the survival of an overlying random skin flap in the rat.

Degloving injuries are common problems with potentially devastating clinical consequences.[1–12] The treatment of choice is early and repeated debridement,[13,14] with tensionless repositioning of the degloved skin. Sometimes the flap is excised and replaced as a full or partial thickness skin graft.[1,15] Following defatting, skin graft survival has been shown to be augmented by the use of vacuum-assisted closure.[2,16]

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* Corresponding author. The Bernard O'Brien Institute, 42 Fitzroy Street, Fitzroy, Melbourne 3065, Australia. Tel.: +61 3 9288 2545; fax: +61 3 9288 2605.
E-mail address: a.penington@unimelb.edu.au (A.J. Penington).

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In traumatically elevated flaps initial evaluation frequently underestimates the degree of eventual flap loss. Skin that has capillary return and bleeds upon pinprick at the first assessment can become progressively necrotic days after the injury. It is not clear whether this represents progressive necrosis of the skin flap or merely progressive recognition of the extent of non-viability.[17]

The pathophysiology of skin flap necrosis and its progression in degloving injuries has not been fully elucidated. At least two possible aetiologies for skin necrosis following avulsion injury can be postulated: interruption of blood supply to the skin and a toxic effect of underlying devascularised fat on the overlying skin. In the first theory, blood vessel damage in avulsion injury is not localised solely to the immediate plane of tissue separation. Avulsion is known to cause wide-spread vascular injury, and this commonly witnessed clinical phenomenon has been well-documented experimentally.[8–12,18] Intimal damage as a result of traction injury can cause progressive thrombosis of small arterial and venous perforators, resulting in arterial insufficiency, venous congestion or a combination of the two, over a period of several days. This phenomenon would explain why avulsed flaps frequently undergo progressive necrosis, while similar flaps, elevated surgically, might survive. Theories of ischaemia and/or congestion are also bolstered by the finding that aggressive defatting of an avulsed flap and replacement of the skin as a full thickness graft frequently result in near complete survival.

In the second theory, release of toxic metabolites from underlying devascularised fat may affect necrosis of overlying skin. Several authors have implicated free radicals in skin flap necrosis due to ischaemia reperfusion.[2,16] Evidence of free radical formation is present in marginally viable skin flaps.[2,16] Studies of warm stored skin flaps have shown that free radical formation increases in adipose tissue but not in skin prior to reperfusion,[2,16] implying that in a marginally viable, traumatically raised skin flap, the adipose tissue might play an active role in causing skin necrosis. There would then be a potent case for debriding traumatically raised flaps of adipose tissue.

This study was designed to address the second of these two hypotheses: that devascularised fat under a marginally viable skin flap contributes to skin flap necrosis.

Materials and methods

Model

Male Sprague–Dawley rats weighing between 225 and 275 g were used throughout the study. A caudally-based dorsal skin flap, as a modification of the original McFarlane flap[19] and other recent modifications,[20,21] was used.

Stage I: elevation of the epigastric fat pad

Under sterile conditions, with the animal in a supine position, a 2 cm oblique incision was made in the right groin. The epigastric fat pad in the rat is fed by two pedicles, the dominant superficial inferior epigastric vessels inferiorly, and the iliac branch of the iliolumbar artery supero-laterally. With dissection of the epigastric fat pad from its surrounding tissue attachments, mobilisation of the fat pad on the two pedicles is possible. Lateral dissection deep to the panniculus carnosus was performed towards the lateral dorsum of the animal, to facilitate transfer of the fat pad to lie under the dorsal flap. The fat pad was then returned to its original position and the wound was closed with interrupted 4/0 silk sutures.

Stage II: elevation of a caudally-based dorsal skin flap

Following dissection of the epigastric fat pad, the animal was repositioned in a prone position and reprepped. The outline of the proposed flap was marked on the dorsum, to the right of midline to allow rotation of the ventral epigastric fat pad underneath it. Dimensions of the flap were 6×2 cm. Positioning of the flap was standardised by placing its medial edge 5 mm to the right of the spinal midline, and its caudal base 5 mm caudal to the posterior superior iliac spine (Fig. 1A). Outlines of the flap were incised through the panniculus carnosus and elevation performed with cauterisation of feeding vessels by bipolar electrocautery. Lateral dissection, deep to the panniculus carnosus, afforded access to the previously dissected epigastric fat pad. According to the randomly chosen experimental group (see below), the fat pad was either left in-situ in its native ventral position (control), transferred to the back with its vascular pedicles intact (viable fat), or transferred to the back after cauterisation and division of its vascular pedicles (devascularised fat).

Pilot experiments showed that these skin flaps, as well as the devascularised fat pads, when replaced onto the original bed of the flap, had improved survival based on their ability to take as grafts. This finding is also documented in the literature.[20] Therefore, silicone sheeting ($6 \times 2 \times 0.2$ cm) was used to separate the flap (plus or minus the underlying fat pad) from the wound bed. Further pilot studies confirmed validity of the model, with consistent necrosis of the fat grafts.