

# Radial Artery And Cephalic Vein Interposition Graft To Salvage A DIEP Flap And Prevent No-Flow Phenomenon: A Case Report And Literature Review.

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## Abstract

Autologous tissue from the lower abdomen represents the optimal method for delayed breast reconstruction in the previously irradiated chest, as these flaps are capable of reproducing a reconstructed breast that approximates the shape, volume, softness and ptosis of the native breast.<sup>1</sup> Unfortunately, delayed breast reconstruction in the setting of previous postmastectomy radiotherapy has been shown to have a higher complication rate<sup>2,3</sup> due the soft-tissue sequelae caused by radiation including tissue fibrosis, edema and vasculitis which can interfere with healing of the reconstructed breast and can injure the internal mammary vessels increasing the risk of intraoperative and postoperative microvascular anastomotic complications. We present the relevant literature review and a case where a deep inferior epigastric perforator (DIEP) flap was not only salvaged with a radial artery vascular pedicle conduit graft, but fat cell damage was minimized by prompt revascularisation of flap to the arm. 1 Baumann DP, Lin HY, Chevray PM. Perforator number predicts fat necrosis in a prospective analysis of breast reconstruction with free TRAM, DIEP and SIEA flaps. *Plast Reconstr Surg.* 2010;125:1335-1341. 2 Tran NV, Chang DW, Gupta A, Kroll SS, Robb GL. Comparison of immediate and delayed TRAM flap breast reconstruction in patients receiving postmastectomy radiation therapy. *Plast Reconstr Surg.* 2001;108:78-82. 3 Kronowitz SJ, Robb GL. Breast reconstruction with postmastectomy radiation therapy: Current issues. *Plast Reconstr Surg.* 2004; 114:950-960.

## INTRODUCTION

Autologous tissue from the lower abdomen represents the optimal method for delayed breast reconstruction in the previously irradiated chest, as these flaps are capable of reproducing a reconstructed breast that approximates the shape, volume, softness and ptosis of the native breast. <sup>1</sup> Unfortunately, delayed breast reconstruction in the setting of previous postmastectomy radiotherapy has been shown to have a higher complication rate <sup>2</sup> due the soft-tissue sequelae caused by radiation including tissue fibrosis, edema and vasculitis which can interfere with healing of the reconstructed breast and can injure the internal mammary vessels increasing the risk of intraoperative and postoperative microvascular anastomotic complications.

We present the relevant literature review and a case where a deep inferior epigastric perforator (DIEP) flap was not only salvaged with a radial artery vascular pedicle conduit graft, but fat cell damage was minimized by prompt revascularisation of flap to the arm.

## CASE REPORT

A 48 year old non-smoking woman underwent left breast mastectomy and irradiation for breast cancer. (Figure 1) She underwent DIEP flap breast reconstruction 7 years after her mastectomy by the senior author (PH). The flap was based on 4 perforators on the left deep inferior epigastric vascular axis. Simultaneous harvest of the left internal mammary vessels was performed through the 3<sup>rd</sup> intercostal space, revealing a macroscopically viable internal mammary artery and vein, which were deemed usable for microvascular anastomosis. The DIEP flap was divided and transferred to the chest wall for microvascular anastomosis. The irradiated artery was fragile but initial anastomosis of both artery and vein was uncomplicated with primary ischemic time of 55 minutes. After initially showing good blood flow with a well perfused flap the artery thrombosed. The decision was made to remove the 2<sup>nd</sup> costal cartilage and attempt a more proximal anastomosis. This was performed yet again with minimal technical concerns, but unfortunately the arterial anastomosis suffered the same thrombotic fate. At this stage the total ischemic time was 3,5 hrs with concerns of flap

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damage and impending no-reflow phenomenon an alternate recipient vessel and interposition vascular conduit were required. The DIEP pedicle after the two anastomotic revisions was considerably shortened and thus wouldn't reach the axillary vessels without a vascular graft. The flap needed to be immediately nourished along with the harvest of a long reliable vascular conduit to avoid no-flow phenomenon. No dissection in the scarred axilla had been performed at this stage and as ischaemic time was progressing the decision was made to revascularise the flap with the radial vessels. After performing of an Allen's test on the right hand the DIEP flap was anastomosed to the right radial artery and the cephalic vein at the wrist (Figure 2). The flap was allowed to perfuse allowing us plenty of time to dissect out the circumflex scapula vessels in the irradiated and scarred axilla. Once the recipient vessels were adequately prepared, approximately 10 cm of the radial artery and cephalic vein were dissected as conduits and the DIEP flap with its new extended pedicle was transferred to the chest wall and microvascular anastomosis was performed. Eight hours after the first operation venous congestion was noted and a reoperation performed immediately. A venous thrombosis was found in the cephalic vein graft. This was surprisingly localized to the center of the graft not in any of the 2 anastomoses. The left cephalic vein was dissected 14 cm down on the upper arm and mobilized to the mastectomy defect. An uneventful end to end anastomosis was performed between this vessel and the deep inferior epigastric vein of the flap. Healing was uneventful thereafter. The flap survived completely and at 1 year follow-up, the breast was soft and supple with no signs of fat necrosis (Figure 3). In a secondary procedure a right sided reduction and adjustment of the reconstructed NAC on the reconstructed breast was done with simultaneous liposuction and fat grafting. At final healing the patient was very happy with the outcome.

**Figure 1**

Figure 1: Pre-operative Photograph



**Figure 2**

Figure 2: Anastomosis of the DIEP flap to the radial vessels at the wrist



**Figure 3**

Figure 3: Final post-operative photographs



## DISCUSSION

The use of the radial artery as a vascular conduit is by no means a unique concept, however to our knowledge the use of the radial artery vascular pedicle as a conduit has not been reported for the salvage of a DIEP flap. The plastic surgical literature is abundant with reports of salvage techniques for DIEP/TRAM flaps. Barnett <sup>3</sup> reported a technique of venous supercharging free TRAMs by dividing the cephalic vein distally and rotating it about the infraclavicular fossa before anastomosing it to the superior inferior epigastric vein. This technique was also successfully used in the second phase of occlusion in this case. Blondeel <sup>4</sup> described salvaging a congested DIEP and free transverse rectus abdominis myocutaneous (TRAM) flaps by interposition grafting of the superficial inferior epigastric vein to the internal mammary vein. Wechselberger <sup>5</sup> solved insufficient venous drainage by anastomosis of the superficial inferior epigastric vein to a thoracodorsal, lateral thoracic, or intercostal vein. Obviously these techniques would not be successful in our case as no arterial inflow had been established. Niranjana <sup>6</sup> used several different anastomoses: superficial circumflex iliac vein to cephalic vein, superficial inferior epigastric vein to lateral pectoral vein, or an interposition vein graft between the cephalic vein and opposite pedicle to augment the venous system. The use of the contralateral internal mammary system was also entertained, however that would have required interposition vascular grafts to both the artery and vein to gain adequate pedicle length.

All these techniques require dissection of a separate recipient system and/or harvesting of an interposition graft, adding to the operative time.

Our concerns with the increasing ischemic time was the inevitable and irreversible sequelae of no-reflow phenomenon. Hence, the urgent anastomosis to the right radial artery vascular pedicle. No-reflow phenomenon is well documented in the plastic and reconstructive surgical literature. It consists of progressive vascular damage during reperfusion and an expanding zone of poor blood flow. <sup>7</sup> It was first described by May and colleagues <sup>8</sup> in 1978, they described the phenomenon in a free flap experiment and hypothesized that the no-reflow phenomenon is caused by cellular swelling, intravascular aggregation, and the leakage of intra-vascular fluid into the interstitial space, with reperfusion counterintuitively providing the main component of tissue injury after prolonged ischemia. Reperfusion injury, on the other hand, was first described by Cerra <sup>9</sup> in 1975.

They initially studied myocardial pedicles in dogs and, in an experiment designed to detect the extent of reperfusion injury, found that restoration of blood flow was associated with subendothelial hemorrhagic necrosis.

Several studies have subsequently examined the critical ischemic time (the maximum amount of time that a tissue can tolerate and yet survive) in skin <sup>10</sup> and myocutaneous flaps. <sup>11</sup> In general, skin and fascia are more resistant to ischemia than muscle. <sup>12</sup>

The tolerance against ischemia varies depending on the type of tissue, for example, the critical ischemic time for human muscles at normothermic temperature is around 2 hours, whereas jejunum develops histologic changes after approximately 30 minutes of ischemia. <sup>13</sup> Studies have shown the critical time of total venous occlusion for skin flaps to be between 7 and 8 hours. <sup>14</sup> Harashina <sup>15</sup> also observed 100 percent necrosis in rat island skin flaps subjected to 8 hours or more of venous ischemia.

Unfortunately, very little has been published on the ischemic tolerance of DIEP flaps. The best available experimental model for the tolerance of fat to ischemia still remains the pivotal paper by May et al. <sup>16</sup> In their study they found that the denervated epigastric flap could be raised and reliably transferred by microvascular anastomosis, with 100% flap survival seen in the 1 and 4 hour ischemic flap models (30 flaps). They also concluded that using their free flap model of rat epigastric flaps the no-flow phenomenon changed from a predictably reversible to a uniformly irreversible process over a relatively short period, 4 hours of ischemia. Also the decrease in flap survival, at 1 week post-operative, seemed to be related to increasing periods of ischemia prior to reperfusion. This may explain the long-term aesthetic success of our flap and the absence of fat necrosis.

Vijan and colleagues <sup>17</sup> attempted to clinically quantify how long a DIEP/TRAM flap can survive after pedicle thrombosis. They performed a retrospective review involving patients under going microvascular autologous breast reconstruction with TRAM or DIEP flaps all anastomosed to the thoracodorsal vascular bundle with no interposition grafts. Their study revealed 290 free flaps with an overall success rate of 97.6% and a failure rate of 2.4%. Pedicle thrombosis occurred in 6.2% (18 of 290) flaps. All the thromboses were detected within the first 48 hours after surgery. 10 flaps (55%) were saved and 8 flaps (45%) were lost. The greatest time interval between detection of vascular

compromise and re-exploration that resulted in flap survival was 188min. Of the flaps that survived, 4 healed well without complication. 6 flaps subsequently demonstrated 2-3cm superficial skin necrosis. Minor fat necrosis (<2cm) was identified in 2 patients and major fat necrosis (>2cm) in another 2 patients. For take-back operations, the mean time to incision after thrombosis detection was 127min (range 50-188min) in the saved group and 192 min (range 20-324) in the lost flap group. They concluded, although not statistically significant ( $p=0.0832$ ) due to small take-back sample, there was a trend towards a favorable outcome if re-exploration occurs within 188min of vascular compromise. How these events affected the final flap volume was not presented or discussed.

Immediate breast reconstruction is popular because of its psychological and aesthetic advantages<sup>18</sup> its use is usually deferred until postmastectomy radiation therapy has been delivered. Reasons for this include avoiding irradiating the flap, which has been shown to result in unpredictable fibrosis of the reconstructed breast, necessitating revisional surgery<sup>19</sup> and maintaining a planar chest wall to optimize the delivery of radiation therapy.<sup>20</sup>

Whereas in the past, irradiation was not often offered following mastectomy, recent recommendations suggest that patients with as little as one to three positive axillary lymph nodes may benefit.<sup>21</sup> With several large randomized controlled trials demonstrating improved outcomes in select patients receiving post-mastectomy radiation therapy.<sup>22</sup> These findings support the use of delayed breast reconstruction in women with breast cancer as postmastectomy radiotherapy is becoming a part of their comprehensive treatment program.<sup>23</sup>

The technical challenges of delayed breast reconstruction after postmastectomy radiation therapy, and the higher complication rates compared with immediate reconstruction, have been well established in plastic surgical literature.<sup>24</sup> The reasons for this are potentially multifactorial, with radiation injury contributing to chronic inflammation and vasculitis of the recipient vascular bed, but also the unsuitability of implant based reconstructions in irradiated tissue due to considerably increased risk for capsular contracture.

The choice of recipient vessels varies between surgeons, with studies showing that the thoracodorsal vessels are unusable in 7 to 15% of breast reconstructions and the

internal mammary vessels in 2%.<sup>25</sup>

However, these studies included only a few irradiated patients, 17% in Feng's study,<sup>26</sup> and not all the reconstructions were performed in a delayed setting. Temple and colleagues<sup>27</sup> in their study of a homogenous group of patients presenting for delayed breast reconstruction previously exposed to irradiation examined the internal mammary vessels in 56 patients and rejected them in 11 cases, for an unusability rate of 20 percent. The thoracodorsal vessels were explored in 74 patients and rejected in 19, for an unusability rate of 26 percent. Their conclusion was that there was no significant difference between the unusability rate for the internal mammary and thoracodorsal vessels in the setting of previous irradiation. ( $p=0.42$ )

Radiation therapy has long been used as adjuvant therapy for breast conservation therapy. The recognition that breast cancer is less likely to spread by means of the internal mammary nodes, in conjunction with higher rates of pulmonary and cardiac abnormality following irradiation, has led toward a declining inclusion of the internal mammary chain in the primary radiation field.<sup>28</sup> However, regardless of field design or intent to treat, the internal mammary vessels routinely receive at least a 40 percent partial dose, if not the full dose of radiation, thus placing the primary recipient vessel in free flap breast reconstruction for most surgeons at risk.<sup>29</sup>

The plastic surgical literature is relatively sparse in looking at the direct effect of radiotherapy on recipient vessels for microvascular anastomosis, however borrowing from the field of cardiology, we may see a corollary for vascular risk following radiation therapy. Women treated for left-sided breast conservation with adjuvant radiation therapy have a substantially increased risk of future coronary artery disease.<sup>30</sup> The effect of radiation on the internal mammary artery also decreases the usability of this vessel for coronary revascularization.<sup>31</sup>

Fosnot and colleagues<sup>32</sup> specifically addressed the effect of prior radiation therapy on the rate and types of microvascular challenges associated with therapy delivered before free flap breast reconstruction. In their retrospective series they concluded that previous irradiation was associated with a higher percentage of flaps with a vascular problem (9.6 percent versus 17.3 percent;  $p=0.001$ ). Radiation therapy was also the only variable identified as an

independent risk factor for vascular complications (odds ratio, 1.68;  $p=0.04$ ). There were also trends toward increased rates of intraoperative arterial and venous thromboses when comparing non-irradiated to irradiated patients. Intraoperative technical difficulties nearly reached statistical significance, with irradiation likely increasing the risk (4.8 percent versus 8.0 percent;  $p=0.06$ ). In addition, there appeared to be a trend toward higher anastomotic revision rates and a need for additional dissection on the recipient vessel to reach a usable target.

As a whole, they concluded that there was a significantly increased risk of having an intraoperative vascular complication with preoperative radiation therapy, (7.6 percent versus 14.2 percent;  $p=0.003$ ) as was the case in our report. Interestingly, there was no significant difference in rates of delayed arterial, venous, or any delayed complications (2.4 percent versus 4.0 percent;  $p=0.19$ ). Nor was there a significant difference in conversion rate to the thoracodorsal vessels between these groups (3.2 percent versus 5.0 percent;  $p=0.24$ ).

The effects of radiation on the soft tissues of the chest wall continue to develop beyond the period of radiation delivery and thus reconstruction is often postponed until these effects subside, however the exact optimal duration of this interval is not known. <sup>33</sup> Fosnot et al <sup>34</sup> suggested that the risk of vascular complication is greatest in the first few months after treatment and diminishes over time, but never reaches equivalence to non-irradiated patient.

Baumann and colleagues <sup>35</sup> attempted in their study to determine the optimal timing of delayed autologous breast reconstruction following post-mastectomy radiation therapy. In their series of one hundred and eighty-nine patients, the patients were classified as having undergone reconstruction less than 12 months after post-mastectomy radiation therapy (Group 1: 82 patients) or 12 months or more after post-mastectomy radiation therapy (Group 2: 107 patients). They found that those operated on in the first 12 months following irradiation had a statistically significant increase in flap loss ( $p=0.014$ ) and re-operation rate ( $p=0.022$ ). In addition, group 1 patients trended toward a higher incidence of microvascular thrombosis, infection and wound dehiscence.

## CONCLUSION

Autologous breast reconstruction utilizing the lower abdominal tissue represents the optimal method for delayed breast reconstruction in the previously irradiated chest. The

first attempt at reconstruction represents the best time to achieve the desired aesthetic results. Therefore, all measures should be taken to ensure a safe and reliable operation.

On the best available evidence one should wait 6-12 months post irradiation to perform the reconstruction, keep the ischemic time to a minimum with the knowledge that fat may tolerate up to 4 hours of ischemia and that both the thoracodorsal and internal mammary vascular system are equally usable post irradiation.

In threatening no-reflow due to prolonged ischemia time a possible way to salvage the flap is to connect it to the radial artery and cephalic vein at the wrist and use the vessels as a conduit to elongate the vascular pedicle.

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