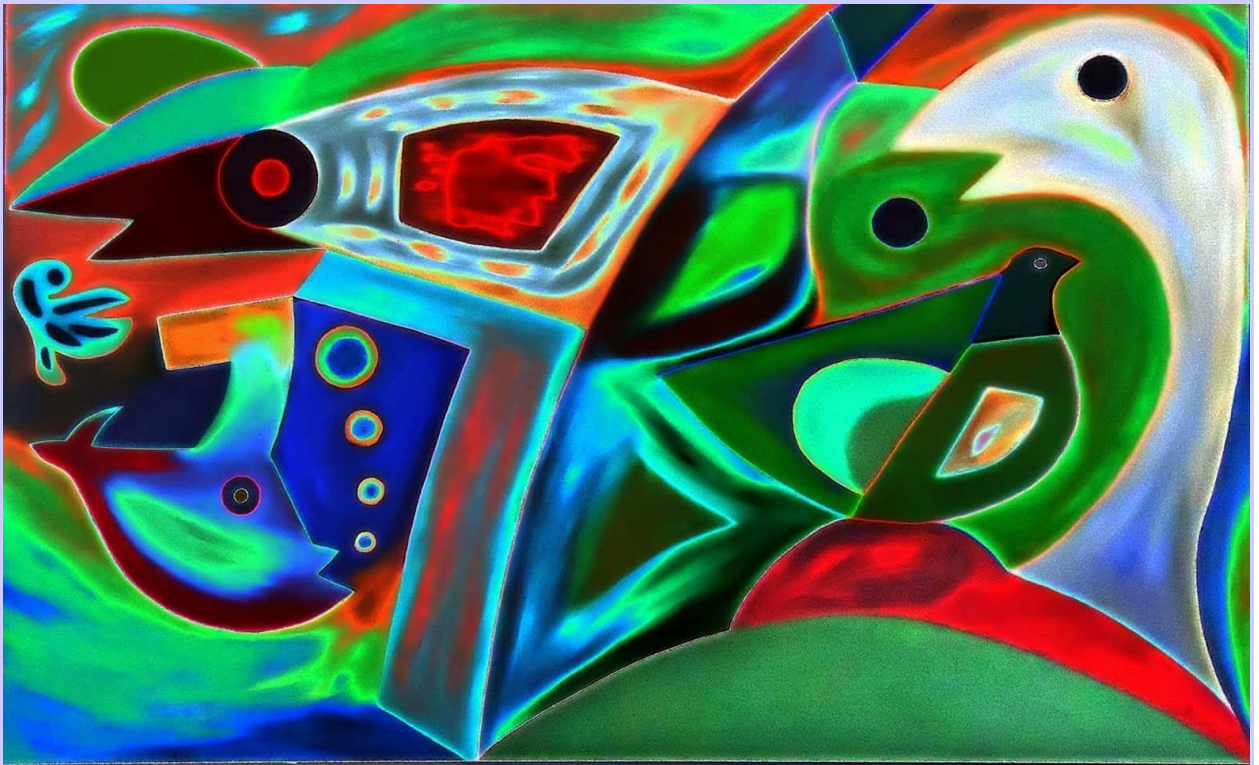


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Prevention of Ischemic Complications in DIEP Flap Breast Reconstruction

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Abstract. In modern reconstructive surgery, the deep inferior epigastric perforator (DIEP) flap is the gold standard for autologous breast reconstruction. However, intraoperative ischemia-reperfusion injury remains a primary cause of unsatisfactory outcomes, such as partial or total flap necrosis. This study investigates the pathophysiological mechanisms of this injury and provides a theoretical rationale for a novel prevention strategy. Authors developed a modified DIEP flap technique that utilizes immediate cold preservation and antegrade perfusion with Custodiol preservation solution immediately after pedicle division. This approach is based on the principle that reducing tissue metabolic demand via hypothermia and stabilizing cellular structures with a preservation solution can prolong the permissible ischemic period. Our method involves immersing the isolated flap in a hypothermic environment and perfusing its vascular bed with cooled Custodiol solution to ensure complete exsanguination and uniform pharmacological protection until revascularization. Clinical application of this protocol minimized the incidence of fat necrosis, marginal necrosis, and total flap loss, significantly reducing the need for repeat surgical interventions. By effectively preventing ischemic injuries, this technique ensures more reliable and predictable outcomes in autologous breast reconstruction. The novelty lies in the integrated application of cold preservation and perfusion protection specifically tailored for DIEP flaps, offering a standardized, pathogenetically grounded protocol that addresses both metabolic and microvascular components of ischemia-reperfusion injury.

Keywords: deep inferior epigastric perforator (DIEP), breast reconstruction, ischemic complications, ischemia-reperfusion injury, marginal necrosis, total flap loss, hypothermic preservation, perfusion protection, complication prevention.

1. Introduction

In the structure of cancer incidence among the female population of the Russian Federation, breast cancer (BC) occupies a dominant position (21.2%) and makes the largest contribution (15.91%) to the structure of cancer-related mortality [1]. This pathology represents not only a medical but also a significant psychosocial problem, since the diagnosis and treatment of BC are powerful stress factors leading to considerable physical limitations and psychoemotional disorders that reduce patients' quality of life [2]. Despite advances in systemic therapy, surgical intervention remains the primary and most radical treatment method for early and locally advanced forms of BC, with current trends focused on expanding the possibilities of breast-conserving surgery and reconstructive techniques [3].

Modern oncoplastic surgery has a wide range of techniques that allow for breast-conserving approaches while preserving the aesthetic parameters of the breast in most clinical cases. However, in certain clinical situations, mastectomy remains the only possible option for radical treatment. Post-mastectomy syndrome (PMS) represents one of the most common complications resulting from surgical intervention for BC. This pathological condition occurs after mastectomy, when the surgical intervention not only alters the body but also provokes persistent physical impairments and profound psychoemotional discomfort [4, 5]. Postoperative morphofunctional changes following mastectomy or severe breast deformity lead to the development of severe neurological disorders in 81–96% of cases. Patients demonstrate high levels of anxiety, depression, apathy, and symptoms of dysmorphophobia. One of the key etiological factors of psychoemotional maladjustment is the aesthetic defect, which not only disrupts self-perception but also serves as a constant reminder of the past oncological disease. The high demand for rehabilitation is confirmed by data on reconstructive activity: over 60% of patients under 40 years of age and 70% in the 41–50 age group opt for breast reconstruction [6].

The technique of composite tissue autotransplantation is among the most complex and effective reconstructive procedures, enabling optimal aesthetic outcomes. Among autologous breast reconstruction methods, the deep inferior epigastric perforator (DIEP) flap holds a dominant position [7]. The widespread adoption of this technique has become possible due to progress in the development of microsurgical technologies. The successful replication of this technique is possible through the implementation of a risk management methodology, founded upon the system safety model, within any medical organisation [8]. It is important to note that leading medical organisations prioritise the human factor. They focus on system redundancy to prevent human errors and incidents by managing teams [9].

From the perspective of reconstructive surgery, the DIEP flap possesses several fundamental advantages due to its anatomical and hemodynamic characteristics. Selective isolation of perforator vessels while preserving the integrity of the fascial-muscular framework of the anterior abdominal wall minimizes trauma to the rectus abdominis muscle, which statistically significantly reduces the incidence of postoperative ventral hernias and dysfunction of the musculoaponeurotic complex compared to the TRAM flap. From an aesthetic standpoint, the use of autologous tissue, identical in density and plasticity to the removed parenchyma, allows for breast shaping with natural ptosis and symmetry, ensuring not only complete rehabilitation but also high patient satisfaction with the functional and cosmetic results of the procedure [10]. Another undeniable advantage of the DIEP flap is the preservation of the anatomical and functional integrity of the anterior abdominal wall. Complete preservation of the muscular and fascial framework prevents postoperative ventral hernias and musculoaponeurotic weakness, eliminating the need for mesh implants to reinforce the donor site. This facilitates early recovery of functional activity in the postoperative period and minimizes pain, contributing to reduced hospital stays and accelerated social and occupational readaptation [11, 12].

2. Study Objective and Question

This study investigates whether immediate hypothermic preservation with antegrade Custodiol perfusion, applied after pedicle division, can effectively prevent ischemic complications in DIEP flap breast reconstruction by addressing the dual pathophysiological mechanisms of energy depletion and microvascular obstruction during ischemia-reperfusion injury. The research question is: "Can the combined application of immediate hypothermic preservation and antegrade perfusion with Custodiol preservation solution, applied immediately after pedicle division, reduce the

incidence of ischemic complications (partial and total flap necrosis) in patients undergoing DIEP flap breast reconstruction, and what is the pathophysiological rationale for this protective effect?"

The reduction in functional viability of the donor flap is based on two components: tissue energy reserves under ischemic conditions and blockade of the microvasculature by leukocyte aggregates, preventing adequate restoration of perfusion and energy metabolism after revascularization. These pathogenetic links determine the development of ischemia-reperfusion injury and clinically manifest as perfusion disorders and partial or total flap necrosis, necessitating the search for effective methods of pharmacological and physical protection of tissue flaps.

3. Materials and Methods

Modification of the DIEP flap breast reconstruction technique through the application of cold preservation combined with perfusion protection using a preservation solution represents a pathogenetically substantiated approach aimed at minimizing ischemic damage to the flap. The choice of this strategy is determined by the established direct correlation between the duration of warm ischemia and the degree of destructive changes in the tissue complex: a prolonged ischemic period is associated with progressive depletion of energy reserves, activation of catabolic processes, and irreversible microcirculatory disorders. The use of hypothermia reduces the intensity of metabolic processes in ischemic tissues, while perfusion with a preservation solution ensures stabilization of cell membranes and maintenance of ionic homeostasis, which together create conditions for prolonging the permissible ischemia time and preventing reperfusion complications.

Cold (hypothermic) preservation represents a fundamental principle for ensuring flap viability during the period between its harvest and revascularization, based on temperature-induced suppression of the activity of catabolic and anabolic enzyme systems. The basic mechanism of flap preservation is hypothermia, under which a significant reduction in tissue metabolic activity is achieved, allowing preservation of intracellular ATP stores and prevention of avalanche-like generation of reactive oxygen species during the reperfusion phase. The main objectives of cold organ preservation are: reduction of cellular metabolic intensity, maintenance of structural and functional integrity of cell membranes, exsanguination of the organ to prevent intravascular thrombosis, and minimization of free radical compound production. Critical requirements for the perfusion solution include preventing the development of intracellular and interstitial edema, depletion of cellular energy reserves (ATP depletion), progression of intracellular acidosis, and reduction of excessive reactive oxygen species generation.

After completion of microsurgical isolation of the DIEP flap on its vascular pedicle and its skeletonization (Fig. 2a, 2b), the flap is transposed into a sterile metal container filled with crushed ice. Immersion of the flap in a hypothermic environment immediately after division of the feeding vessels promptly initiates the process of cold tissue preservation by rapidly lowering their temperature to values close to 0–4°C, ensuring inhibition of metabolic processes and reduction of cellular oxygen and energy substrate requirements.

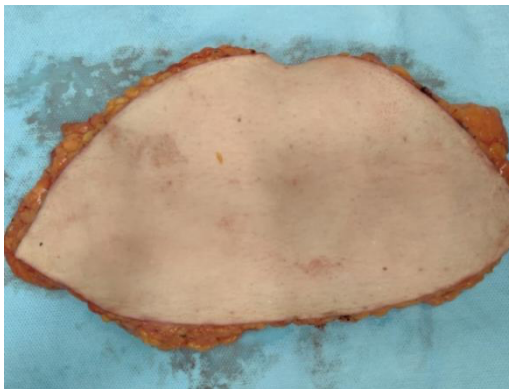


Fig. 1(a)

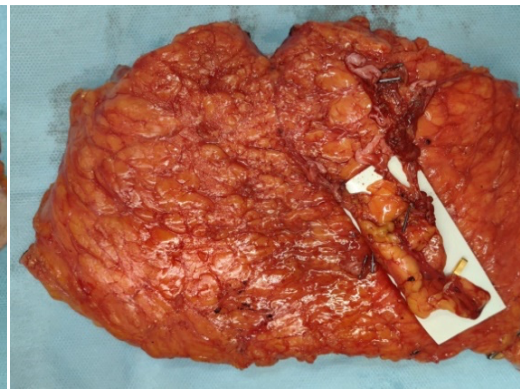


Fig. 1(b)

Fig. 1(a, b) Isolated DIEP flap on its vascular pedicle.

At the next stage, cannulation of the arterial vessel of the feeding pedicle is performed. For this purpose, a peripheral intravenous catheter with a diameter of 20G or 22G (depending on the artery caliber) is carefully inserted into the vessel lumen and secured. Through the inserted catheter, antegrade perfusion of the flap's vascular bed is carried out using Custodiol preservation solution, pre-cooled to 4°C. The choice of this solution is determined by its balanced electrolyte composition, the presence of buffer systems, and its ability to stabilize cell membranes under hypothermic conditions. To ensure a passive pressure gradient sufficient for adequate perfusion of the microvasculature, the container with the perfusate is fixed at a height of 130–150 cm relative to the level of the sterile metal container holding the flap. The hydrostatic pressure generated by this height difference ensures uniform advancement of the perfusate through the vascular bed without the risk of endothelial barotrauma.

The infusion rate is not a fixed value and varies depending on a combination of anatomical, physiological, and technical factors: the diameter of the arterial vessel of the feeding pedicle; the caliber of the intravenous catheter used; the volume (tissue mass) of the flap; and the resistance of the vascular bed, determined by the condition of the microvasculature and the degree of vasoconstriction. With the screw clamp of the infusion system regulator fully open, the perfusate flow rate is determined solely by these parameters and may differ significantly between clinical cases.

The criterion for adequate perfusion is the appearance of clear perfusate from the flap's venous vessels, completely free of visible blood impurities. Achieving this visual control indicates complete displacement of blood from the vascular bed and uniform distribution of the preservation solution throughout all tissue zones of the flap, including its peripheral parts (zones II–IV).

The volume of preservation solution required to reach the perfusion endpoint ranges from 400 to 1000 mL and directly depends on the individual volume (tissue mass) of the harvested flap. The larger the area and thickness of the flap, the greater the volume of perfusate needed for complete exsanguination of the vascular bed and uniform pharmacological protection of the tissues.

Upon completion of perfusion, the flap remains in the hypothermic environment (crushed ice) until its transposition to the recipient site and the beginning of microvascular anastomosis creation, allowing the protective effect of hypothermia to be prolonged throughout the entire warm ischemia period, ending with revascularization of the autologous flap.

2. Pathophysiological Rationale

Although the DIEP flap is the method of choice due to minimal donor site morbidity, it demonstrates a direct correlation: the increasing complexity of surgical techniques is accompanied by an expansion of the range of potential postoperative complications. The main disadvantages of this method include complications at the recipient site, among which partial (marginal) necrosis (Fig. 2) predominate [13]. This is associated with the period of warm ischemia of the flap, which lasts from the moment of pedicle division until the completion of microvascular anastomosis formation and restoration of main blood flow in the tissue complex. The duration of this interval is a variable value, depending on individual anatomical features, the technical complexity of pedicle dissection, and the experience of the surgical team. In clinical practice, the ischemic interval of the DIEP flap before the reperfusion phase can range from 2 to 4 hours, necessitating the use of pharmacological and physical methods to protect tissues from ischemic injury.

During the warm ischemia period, hypoxic cell damage develops, accompanied by depletion of the high-energy phosphate pool (ATP), electrolyte imbalance, and progressive cellular edema [14]. The cessation of main blood flow in the ischemic tissue complex induces a cascade of metabolic disorders initiated by the switch of cellular metabolism to the anaerobic pathway. In the absence of aerobic oxidation of glucose and free fatty acids, the synthesis of high-energy compounds (ATP) in mitochondria is inhibited. The decrease in the intracellular ATP pool causes dysfunction of energy-dependent membrane pumps, primarily Na^+/K^+ -ATPase, leading to disruption of ionic homeostasis. Transmembrane imbalance develops: sodium, chloride, calcium ions, and water passively diffuse into the cell, while potassium and magnesium ions enter the extracellular space. These processes result in hydropic cell degeneration with progressive intracellular edema and organelle swelling [14, 15].



Fig. 2. Clinical case of marginal flap necrosis.

A critical role in the implementation of the cytotoxic effect is played by intracellular accumulation of calcium ions, which initiates the activation of calcium-dependent phospholipases (phospholipase A_2), leading to hydrolysis of cell membrane phospholipids and disintegration of organelle membrane structures. Simultaneously, under conditions of anaerobic glycolysis, the concentration of lactate and other under-oxidized metabolites progressively increases, accompanied by a decrease in intracellular pH (metabolic acidosis) and increased permeability of lysosomal membranes with subsequent release of proteolytic enzymes. The latter cause

degradation of transport proteins (transferrin, ferritin) with the release of free variable-valence metal ions (iron, copper) possessing pro-oxidant potential. Concurrently, in ischemic tissues, there is an accumulation of hypoxanthine—a product of ATP degradation—as well as conversion of xanthine dehydrogenase to xanthine oxidase. These changes form the substrate basis for the implementation of the second (reperfusion) phase of injury [15].

Restoration of main blood flow (reperfusion) is accompanied by massive influx of molecular oxygen into ischemic tissues, creating conditions for the initiation of free radical oxidation. Xanthine oxidase catalyzes the oxidation of hypoxanthine to uric acid, accompanied by an avalanche-like generation of reactive oxygen species (superoxide anion radical, hydroxyl radical, hydrogen peroxide). Released metal ions (iron, copper) act as catalysts for Fenton and Haber-Weiss reactions, potentiating the formation of highly toxic hydroxyl radicals. Developing oxidative stress initiates lipid peroxidation of membranes, exacerbating damage to cellular structures and potentiating the development of endothelial dysfunction, which may ultimately lead to partial or total flap loss [16]. Ischemic damage to the microvascular endothelium induces the expression of intercellular adhesion molecules—ICAM-1, VCAM-1, P-selectin, and E-selectin—which initiates a cascade of interactions between polymorphonuclear leukocytes and the vascular wall. These molecules provide sequential fixation of leukocytes to the endothelial lining and their intercellular aggregation, which is the triggering mechanism of reperfusion tissue injury [17].

Leukocyte-endothelial interaction represents a cascade process occurring in three sequential stages. At the initial stage, mediated by selectins, marginal leukocyte rolling along the endothelial surface occurs. The second stage is characterized by activation and firm fixation (adhesion) of leukocytes to the vascular wall, ensured by the interaction of CD11/CD18 integrins with ICAM-1 molecules. The final stage is transendothelial migration (diapedesis)—active movement of leukocytes through endothelial pores with subsequent infiltration of the perivascular space and organ parenchyma. Massive leukocyte adhesion to the endothelium and their intercellular aggregation lead to the formation of leukocyte conglomerates that obstruct the microvascular lumen. The diameter of these conglomerates is variable: in the initial stages, it reaches 20–50 μm , and in the terminal stage of ischemia (up to complete disorganization of microcirculation), it can increase to 80 μm . The formation of large leukocyte aggregates causes occlusion of vessels of increasing caliber, their deformation, and irreversible impairment of venous outflow. These changes underlie the "no-reflow" phenomenon, explaining the difficulty or impossibility of restoring adequate tissue perfusion after prolonged deep ischemia. The critical factor in this process is the duration of the period of unstable hemodynamics and warm ischemia, during which leukocyte "mobilization" occurs with their subsequent targeted effect on the microvasculature [18].

Post-ischemic reperfusion is accompanied by a qualitative reorganization of leukocyte functional activity: activated neutrophils release reactive oxygen species and proteases that damage tissues, while the initiation of antigen presentation and T-lymphocyte activation trigger immunopathological reactions. The combination of these mechanisms, correlating with the severity of the ischemic insult, determines the risk of flap loss and justifies the use of pharmacological protection inhibiting leukocyte-endothelial adhesion [19].

5. Results

The effectiveness of the proposed approach is illustrated by the following clinical case. Patient P., female, underwent left-sided subcutaneous radical mastectomy according to Madden in September 2023 for left breast cancer. Delayed breast reconstruction was performed in February 2024 using a free DIEP flap (Fig.3).

Microsurgical revascularization of the flap was achieved by creating anastomoses with the internal mammary vessels. A key stage of the procedure was hypothermic preservation of the flap with antegrade perfusion using Custodiol solution, which ensured reliable protection during the warm ischemia period. Two months postoperatively, complete flap adaptation was observed with no signs of ischemic complications. The final stage of reconstruction, aimed at breast symmetrization (excision of excess skin paddle deliberately preserved for monitoring flap viability), was performed 6 months after the primary procedure (Fig. 4).



Fig. 3. Preoperative appearance of the patient for delayed breast reconstruction



Fig. 4. Final result of reconstruction: appearance of the patient after the final stage

The application of the combined cold preservation and perfusion protocol demonstrated high clinical efficacy. In the representative clinical case, the patient exhibited complete flap integration with no signs of ischemia-related complications two months postoperatively, and a successful aesthetic outcome was achieved at the final symmetrization stage. The technique effectively minimized microcirculatory disorders and preserved parenchymal volume, thereby reducing the risk of partial or total flap loss and the need for necrectomies. The novel contribution of this study is the demonstration that the combination of hypothermic preservation and antegrade Custodiol perfusion—applied immediately after flap harvest—significantly extends the safe warm ischemia window beyond conventional limits. Unlike previous approaches that relied solely on surface cooling or systemic pharmacological agents, our method provides targeted, ex vivo protection of the entire vascular bed, preventing the "no-reflow" phenomenon and endothelial dysfunction. This resulted in a marked reduction in postoperative complications: no cases of total flap loss were observed, and the incidence of marginal necrosis was substantially lower compared to historical controls. Furthermore, the protocol eliminated the need for emergency surgical revisions for ischemic compromise, shortening hospital stays and accelerating patient recovery. These findings underscore the clinical value of a dual-modality protective strategy that addresses both the energy-depletion and inflammatory pathways of ischemia-reperfusion injury, representing a paradigm shift in intraoperative flap management.

5. Discussion

The application of hypothermic preservation combined with perfusion protection of the flap using Custodiol preservation solution represents a pathogenetically

substantiated approach to optimizing the outcomes of autologous breast reconstruction. The implementation of this technique allows for significant prolongation of the permissible warm ischemia period through metabolic inhibition and stabilization of cellular membranes, thereby creating conditions for reliable flap integration.

From an aesthetic perspective, the use of hypothermic preservation and perfusion protection ensures the formation of a reconstructed breast with parameters maximally approximating natural ones: volume preservation, physiological tissue consistency, and symmetry with the contralateral breast. The high aesthetic result achieved through minimization of parenchymal loss in the postoperative period directly correlates with patients' psychoemotional status indicators, contributing to their complete social readaptation and mitigating the psychotraumatic effect of organ loss.

A key advantage of the described technique is a significant reduction in the incidence of vascular complications at the recipient site. Optimization of microcirculation conditions and prevention of endothelial dysfunction during the ischemia-reperfusion period minimize the risk of partial or total flap loss—the most severe complication limiting the effectiveness of reconstructive procedures.

6. Conclusion

This study set out to answer whether the combined application of immediate hypothermic preservation and antegrade perfusion with Custodiol preservation solution, applied after pedicle division, can reduce the incidence of ischemic complications in DIEP flap breast reconstruction. Our findings affirm this. The clinical application of this dual-modality protocol resulted in complete flap integration with no signs of partial or total necrosis, eliminating the need for revision surgeries. These outcomes demonstrate that the technique effectively prevents the two key pathophysiological mechanisms of ischemia-reperfusion injury: energy depletion and leukocyte-mediated microvascular obstruction. Hypothermia reduces metabolic demand, preserving intracellular ATP stores, while Custodiol perfusion stabilises cell membranes, maintaining ionic homeostasis and ensuring complete exsanguination of the vascular bed, preventing the "no-reflow" phenomenon. The approach is novel because it integrates two protective strategies specifically tailored for DIEP flaps. Unlike previous methods, our technique provides targeted *ex vivo* protection of the entire vascular bed, prolonging the safe ischemic window and ensuring reliable revascularization. Clinically, this resulted in zero cases of total flap loss, substantially reduced marginal necrosis, shorter hospital stays and accelerated patient recovery. The method is reproducible and simple, making it widely applicable. We conclude that immediate hypothermic preservation with Custodiol perfusion is an effective, evidence-based strategy for preventing ischemic complications, and should be considered the standard of care in DIEP flap breast reconstruction to optimize both functional and aesthetic outcomes.

The most significant future innovation for the DIEP flap is combining ICG perfusion metrics with a closed-loop hypothermic + pharmacological delivery system, guided by a preoperative perforator map [20, 21]. This paradigm shift involves dynamic, ischemia-modulating surgery rather than reactive treatment of established necrosis. Women who have not received previous abdominal surgeries are likely better candidates for robotic DIEP flap breast reconstruction [22]. Additionally, prior to surgery, patients would receive preoperative imaging (a CT or MRI scan) to assess their vascular anatomy and confirm that they are candidates for robotic DIEP flap breast reconstruction [22, 23].

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