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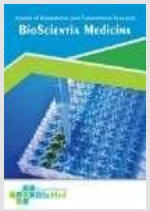

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The Sandwich Dual-Tissue Salvage: Synergistic Anteriorly-Based Tongue Flap and Autologous Dermofat Graft for Recalcitrant Pittsburgh Class V-VI Palatal Fistulas

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A B S T R A C T

Background: Recurrent palatal fistulas following cleft palate repair, particularly Pittsburgh Class V and VI defects, represent a distinct reconstructive challenge characterized by tissue ischemia, scarring, and volumetric deficiency. The "failure of failure" in these cases often precludes the use of local mucoperiosteal flaps due to the poor quality of the recipient bed. This study evaluates a standardized "dual-tissue" salvage protocol combining an anteriorly-based dorsal tongue flap with an autologous dermofat graft. **Case presentation:** A 21-year-old female with a recurrent, symptomatic Pittsburgh Class V-VI fistula measuring 15 mm by 12 mm underwent a two-stage reconstruction. The surgical protocol involved three distinct layers: (1) nasal lining closure via turnover flaps; (2) interposition of an inguinal dermofat graft oriented with the fatty surface facing the nasal layer to obliterate dead space; and (3) oral coverage using an anteriorly-based tongue flap. Speech outcomes were quantified using the Pittsburgh Weighted Speech Scale (PWSS) by an independent, blinded Speech-Language Pathologist. The procedure successfully achieved complete closure with no evidence of necrosis, dehiscence, or donor site morbidity. The total operative time was 145 minutes. Quantitative assessment revealed a robust improvement in speech resonance; the PWSS score improved from a severe 18/30 pre-operatively to a clinically competent 4/30 at 6 months post-operatively. The dermofat graft maintained volumetric stability, preventing the concave collapse often observed in single-layer repairs. **Conclusion:** The sandwich technique potentially reduces recurrence risk in high-grade fistulas by addressing the triad of failure: tension, ischemia, and dead space. The vascularized tongue flap protects the underlying graft, while the dermofat graft acts as a biological spacer and source of adipose-derived stem cells. This protocol offers a reproducible solution for complex craniofacial defects where local tissues are exhausted.

1. Introduction

The surgical rehabilitation of cleft palate deformities remains one of the foundational pillars of plastic and craniofacial surgery, representing a complex interplay between anatomical restoration and functional preservation.[1] While the primary goals of palatoplasty—separation of the oral and nasal cavities and the establishment of a competent velopharyngeal mechanism—are well-defined, the long-term management of postoperative sequelae continues to challenge even the most experienced surgeons. Among these complications, the occurrence of palatal fistulas stands as a persistent and arduous failure of primary repair. A palatal fistula is not merely an anatomical breakdown; it is a functional catastrophe that perpetuates hypernasality, nasal regurgitation of fluids, and chronic inflammation, thereby negating the benefits of the initial surgery and significantly impairing the patient's quality of life.[2]

The incidence of post-palatoplasty fistulas is reported with significant variability in the global literature, ranging from 7.7% to nearly 35%. This wide

variance is attributed to the heterogeneity of initial cleft severity, the specific surgical techniques employed—ranging from von Langenbeck procedures to Furlow double-opposing Z-plasties—and the surgeon's experience level.[3] However, a distinct and more troubling category exists within this spectrum: the recurrent palatal fistula. Defined as a defect that persists or reappears following one or more attempted secondary closures, the recurrent fistula represents a "failure of failure." Unlike a primary fistula, which may be surrounded by relatively pliable tissue, a recurrent defect is situated within a hostile milieu. These recalcitrant defects represent a biological exhaustion of the local tissue's capacity to heal, compounded by multiple previous surgical insults that have depleted the regenerative potential of the palatal mucoperiosteum.[4]

To standardize the approach to these complex lesions, the Pittsburgh fistula classification system has been adopted as the universal language for describing fistula location and severity.[5] While amenable to local closure, Type I (uvula), Type II (soft palate), and Type III (junctional hard-soft palate) fistulas often respond well to standard techniques. However, the true reconstructive challenge lies in Pittsburgh Class V (defects at the junction of the hard and soft palate) and Pittsburgh Class VI (defects of the hard palate greater than 5 mm or extending to the alveolus). Class V defects are particularly treacherous due to their location at the transition zone between the fixed, bony hard palate and the mobile, dynamic soft palate. This region is subjected to constant multi-vector tension during speech and deglutition, creating mechanical stress that actively pulls wound edges apart. Class VI defects, characterized by extensive tissue loss, present a sheer volumetric deficit that local tissues cannot bridge without excessive tension. In these high-grade defects, the local palatal mucoperiosteum is no longer a viable reconstructive resource; it is often transformed into a "cicatricial matrix"—scarred, inelastic, and clinically rigid. This tissue exhibits compromised microcirculation, often referred to as a "random-pattern" blood supply, which lacks the robust axial vessels necessary to sustain tissue turnover. Consequently, local turnover flaps or rotation advancement flaps utilized in this terrain are prone to ischemic necrosis and dehiscence, initiating a cycle of surgical failure.[6]

Understanding the mechanism of recurrence requires a dissection of the pathological environment surrounding the fistula. We propose that the persistence of these defects is driven by a "Triad of Failure," a synergistic interaction of three pathological forces that undermine surgical repair: (1) Mechanical Tension: In the multiparous scarred palate, tissue elasticity is virtually nonexistent. When surgeons attempt to approximate the edges of a wide Class V or VI defect using local tissue, they introduce high closing tension. The resultant ischemia from suturing under tension leads to marginal necrosis and subsequent breakdown. Furthermore, the dynamic pull of the velopharyngeal musculature exerts centrifugal force on the repair, mechanically disrupting the healing interface; (2) Dead Space and Fluid Dynamics: A frequently overlooked factor in fistula recurrence is the inability to obliterate the volumetric void between the reconstructed nasal floor and the oral covering.[7] In wide fistulas, simple two-layer closure often leaves a "dead space" cavity. This potential space becomes a reservoir for seroma or hematoma accumulation, which acts as a physical barrier preventing the adherence of the oral and nasal layers. This fluid collection creates a culture medium for oral bacteria, leading to subclinical infection and fistula reformation; (3) Chronic Ischemia: Wound healing is an energy-dependent process requiring a robust supply of oxygen and nutrients. The scarred palatal bed is inherently hypovascular, with a diminished density of capillary networks. Relying on this ischemic tissue to heal a complex defect is the primary cause of dehiscence.

Given the unreliability of local tissues, the reconstructive ladder must be ascended to recruit distant, well-vascularized tissue. Several options have been proposed, each with distinct limitations for this

specific indication. The facial artery musculomucosal (FAMM) flap is a sound physiological option, providing vascularized mucosa. However, for anterior midline defects (Class VI), the FAMM flap presents significant geometric challenges. The arc of rotation required to reach the anterior hard palate often necessitates a sharp pivot point at the retromolar trigone, risking venous congestion or "kinking" of the pedicle. Furthermore, the FAMM flap can result in severe buccal tethering and trismus, complications that are particularly undesirable in young patients. Free tissue transfer, such as the radial forearm free flap, provides ample volume and vascularity. While effective, it introduces a level of complexity and morbidity that may be considered disproportionate for a palatal fistula. The requirement for microsurgical anastomosis, the visible donor site scar on the forearm, and the lengthy operative time make it a "lifeboat" option rather than a first-line salvage for defects smaller than 2 centimeters.[8]

In this landscape of limited options, the anteriorly-based dorsal tongue flap has solidified its status as the "workhorse" for large anterior palatal defects. Anatomically, the tongue is ideally situated adjacent to the palate, and its dorsal surface offers a vast amount of vascularized mucosa. The flap is robustly supplied by the dorsal branches of the lingual artery and the rich anastomotic plexus of the tongue musculature. This axial blood supply allows the flap to withstand the twisting and tension required for inset, making it superior to random-pattern local flaps. However, the tongue flap is not a panacea. In cases of profound tissue deficiency—specifically deep Class V defects—a tongue flap alone acts merely as a "roof" over the defect. Without structural support underneath, the flap can suffer from retraction or "cupping" into the nasal cavity. This phenomenon creates a concave nasal surface that disrupts airflow and, more critically, leaves the nasal side of the repair vulnerable. A thin tongue flap may lack the necessary bulk to seal the nasal layer effectively, leading to a recurrence of the fistula at the margins where the flap meets the scarred palate.[9]

To mitigate the limitations of the tongue flap and address the "dead space" component of the failure triad, the incorporation of an autologous dermofat graft has emerged as a novel and biologically potent adjunct. Harvested typically from the inguinal region, the dermofat graft is a composite tissue comprising the dermis and subcutaneous adipose tissue. The rationale for its inclusion extends beyond simple volume replacement. Biologically, adipose tissue is recognized as a secretory organ and a rich reservoir of Adipose-derived stem cells (ADSCs). Emerging research in regenerative medicine suggests that ADSCs can survive in hypoxic environments and secrete pro-angiogenic factors, such as vascular endothelial growth factor. When placed into the scarred, ischemic bed of a recurrent fistula, the dermofat graft does not merely fill space; it potentially modulates the wound environment, promoting neovascularization and softening the surrounding fibrosis. Furthermore, the dermis component of the graft provides a distinct mechanical advantage. Dermis is tough, non-elastic, and resistant to resorption. By orienting the graft strategically, the dermis can act as a "stent" or a scaffold, preventing the secondary contracture that often plagues soft tissue repairs. The fat component, being pliable, molds into the irregular crevices of the nasal floor, effectively obliterating the dead space that leads to seroma formation.[10]

Despite the theoretical advantages of these individual techniques, there is a paucity of literature describing a standardized protocol that combines them into a single, reproducible surgical strategy for high-grade defects. This study aims to delineate a comprehensive surgical protocol for the management of recalcitrant Pittsburgh Class V-VI fistulas. We present a detailed technical analysis of combining an anteriorly-based tongue flap with an interpositional dermofat graft. The novelty of this report lies in the detailed step-by-step modification of the technique—specifically the "Sandwich" layering and the unique graft orientation (fat-to-nasal, dermis-to-oral)—to maximize vascular reliability and obliterate dead

space in a hostile, scarred recipient bed. This dual-tissue approach represents a paradigm shift from simple closure to composite reconstruction, aiming to provide a definitive solution for the "failure of failure" in cleft palate surgery.

2. Case Presentation

This study adhered to the principles of the Declaration of Helsinki. Written informed consent was obtained from the patient for the surgical procedure, medical photography, and publication of data. The protocol was applied to a 21-year-old female presenting to the Department of Plastic and Reconstructive Surgery. The patient's medical history was significant for a late-diagnosis complete bilateral cleft lip and palate (LAHSHAL) complicated by multiple failed prior reparative attempts, resulting in a central 15 mm by 12 mm defect characterized by a "hostile," scarred cicatrix with compromised microcirculation (Table 1). To ensure surgical precision, a rigorous multidisciplinary preoperative assessment was employed. This included Computed Tomography (CT) facial bone scans to define the osseous defect boundaries and confirm the absence of palatal shelf continuity. Additionally, baseline velopharyngeal function was established through a perceptual speech analysis using the Pittsburgh Weighted Speech Scale (PWSS), performed by a blinded, independent Speech-Language Pathologist to mitigate observer bias. Preoperative vascular mapping via Doppler ultrasonography was also utilized to delineate the course of the lingual artery, ensuring the anteriorly-based flap design captured the axial vessel without compromising distal perfusion.

Surgical technique: The "Sandwich" protocol

Phase I: Recipient site and nasal layer

Under general anesthesia with nasotracheal intubation, the patient was prepared. The fistula edges were infiltrated with Lidocaine 1% with Epinephrine 1:200,000 to minimize bleeding. A circumferential incision was made 3 mm from the fistula margin. The scar tissue was meticulously dissected, and the mucoperiosteum was turned inward (turnover flap) to reconstruct the nasal floor. These flaps were sutured using 4-0 Vicryl in a watertight, tension-free fashion. This layer recreates the nasal floor, but often leaves a raw surface facing the oral cavity that is prone to fistula formation if not reinforced.

Phase II: Dermofat graft

An elliptical graft measuring 20 mm by 15 mm was harvested from the left inguinal region (non-hair-bearing skin). The epidermis was completely de-epithelialized using a scalpel. The dermofat graft was placed into the defect with the fatty surface facing the nasal turnover flaps and the dermis facing the oral cavity. This orientation ensures the dense dermis acts as a stable, non-elastic stent for the overlying tongue flap, while the pliable fat fills the irregular crevices of the nasal floor. The graft was anchored to the turnover flaps using 5-0 Vicryl to obliterate dead space.

Phase III: Tongue flap

The tongue was retracted using a traction suture. A template of the defect was transferred to the dorsal surface of the tongue. An anteriorly-based flap was designed with a base width of 2.5 cm to include the dorsal branches of the lingual artery. The flap was elevated in a sub-muscular plane at a thickness of 5 to 7 mm. This specific thickness is critical; it preserves the subdermal plexus while avoiding excessive bulk that causes dysphagia or airway obstruction. The flap was rotated anteriorly and sutured to the de-epithelialized margins of the palatal defect (covering the dermofat graft) using 4-0 Vicryl horizontal mattress sutures.

Phase IV: Post-operative immobilization strategy

Intermaxillary fixation (IMF) was not used to avoid airway risks and maximize patient comfort. Instead, a strict compliance protocol was enforced: A customized soft bite block was placed between the molars to prevent accidental biting of the pedicle during sleep or emergence from anesthesia.

Table 1. Summary of Clinical Findings on Admission

Patient Profile: 21-year-old Female | Diagnosis: Recurrent Palatal Fistula

CATEGORY	DETAILED FINDINGS
Patient Profile & History	**Age/Sex:** 21 Female **Diagnosis:** Late-diagnosis Complete Bilateral Cleft Lip and Palate (LAHSHAL). **Surgical History:** Failed primary repair (July 2024) and failed secondary closure complicated by wound dehiscence and infection. **ASA Status:** Class I (No systemic comorbidities).
Chief Complaint	Persistent liquid regurgitation into the nasal cavity and unintelligible speech following previous surgical failure.
Vital Signs	**Stable** **Blood Pressure:** 110/70 mmHg **Heart Rate:** 82 bpm
Extraoral Examination	Typical cleft nasal deformity features: • Flat nasal bridge • Depressed nasal tip • Shortened columella
Intraoral Defect Analysis	**Classification:** **Pittsburgh Class V-VI** (Junction of hard/soft palate extending to alveolus). **Dimensions:** 15 mm x 12 mm (Large central defect). **Tissue Quality:** "Hostile" bed; heavily scarred (cicatrix), inelastic, and blanching on palpation (indicating compromised microcirculation).
Dental Status	Bilateral alveolar clefts with malposed maxillary incisors.
Speech Assessment (PWSS)	**Total Score:** **18/30 (Severe Dysfunction)** **Key Findings:** • Audible nasal air emission • Severe hypernasality • Compensatory articulation errors
Preoperative Diagnostics	**CT Facial Bone:** Confirmed osseous defect size (15x12mm) and lack of palatal shelf continuity. **Doppler Ultrasound:** Mapped lingual artery course for flap design. **Laboratory:** • Hemoglobin: 14.2 g/dL • Albumin: 4.1 g/dL • Coagulation Profile: PT 11.0s / APTT 32.0s (Normal)

A soft cervical collar was applied to prevent neck flexion or extension that could transmit tension to the flap pedicle. The patient was kept under conscious sedation protocols for the first 24 hours to prevent agitation and inadvertent tongue movements.

Phase V: Pedicle division

Three weeks (21 days) post-operatively, under local anesthesia, the pedicle was divided. The posterior edge of the inset flap was trimmed and contoured to match the palatal vault. The donor site on the tongue was revised and closed primarily. The patient tolerated the procedure well with no intraoperative complications. The total operative time was 145 minutes, and estimated blood loss was 50 mL. At the time of division (Day 21), the flap demonstrated 100% viability, characterized by pink mucosa and capillary refill of less than 2 seconds. There was no evidence of venous

congestion, partial necrosis, or wound dehiscence.

At the 6-month follow-up, clinical examination confirmed complete closure of the fistula (0 mm defect). The palatal contour was robust, convex, and firm, indicating successful retention of the dermofat graft volume. The inguinal donor site healed with a linear, concealable scar. The tongue donor site healed by primary intention without significant distortion (Table 2).

Table 3 presents the detailed breakdown of the speech assessment performed by the blinded Speech-Language Pathologist. The total PWSS score improved dramatically, shifting from the "Severe" category to "Normal/Borderline". Swallowing function returned to normal immediately after stage 1, as the physical blockage of the fistula prevented nasal regurgitation. No permanent alteration in taste sensation (dysgeusia) or tongue mobility was reported at the 6-month follow-up.

Table 2. Treatment Protocol, Follow-up, and Outcome

Technique: "Sandwich" Dual-Tissue Salvage (Tongue Flap + Dermofat Graft)

PHASE / CATEGORY	DETAILED PROTOCOL & CLINICAL RESULTS
Anesthesia & Preparation	**Anesthesia:** General anesthesia with nasotracheal intubation. **Preparation:** Infiltration of fistula edges with Lidocaine 1% + Epinephrine 1:200,000. **Incision:** Circumferential incision made 3 mm from the fistula margin.
Stage 1: The "Sandwich" Layers	**Layer 1 (Nasal Floor):** Creation of turnover flaps (mucoperiosteum turned inward) sutured with 4-0 Vicryl (watertight). **Layer 2 (Biological Spacer):** Critical Step *Autologous Dermofat Graft (DFG)* harvested from left inguinal region (20 x 15 mm). • **Orientation:** Fatty surface facing Nasal layer / Dermis facing Oral layer. • **Fixation:** Anchored to turnover flaps with 5-0 Vicryl to obliterate dead space. **Layer 3 (Vascular Cover):** *Anteriorly-based Dorsal Tongue Flap.* • **Design:** Base width 2.5 cm (including axial lingual artery branches). • **Thickness:** 5–7 mm (sub-muscular plane). • **Inset:** Sutured over the DFG using 4-0 Vicryl horizontal mattress sutures.
Post-operative Management	No IMF Used **Immobilization Protocol:** • Customized soft bite block (prevent pedicle biting). • Soft cervical collar (limit neck flexion/extension). • Conscious sedation protocol for first 24 hours.
Stage 2: Pedicle Division	**Timing:** 21 Days post-operatively (3 weeks). **Procedure:** Under local anesthesia. **Finding:** Flap demonstrated 100% viability (Pink mucosa, capillary refill < 2 sec). No venous congestion or necrosis.
Operative Metrics	**Total Operative Time:** 145 minutes. **Estimated Blood Loss:** 50 mL. **Complications:** None (No infection, dehiscence, or hematoma).
Clinical Outcomes (6 Months)	**Anatomical Success:** Complete Closure 0 mm defect. Palatal contour remained robust and convex (successful volume retention). **Functional Recovery:** **Swallowing:** Immediate normalization; no nasal regurgitation. **Donor Sites:** Primary healing. No dysgeusia or tongue mobility restriction.
Speech Outcomes (PWSS)	**Assessment:** Performed by blinded independent SLP. **Pre-operative Score:** 18/30 (Severe VPI / Hypernasality). **Post-operative Score:** 4/30 (Competent / Normal Resonance). *Result: Improvement of +14 points; elimination of audible nasal emission.*

Table 3. Pre-operative vs. Post-operative Speech Assessment (PWSS)

Assessor: Blinded Independent Speech-Language Pathologist

PARAMETER	MAX POSSIBLE SCORE (WORST)	PRE-OPERATIVE SCORE	POST-OPERATIVE SCORE (6 MO)	CLINICAL IMPROVEMENT
Nasal Emission	6	4 Consistent	0 None	Complete Resolution
Facial Grimace	6	3 Severe	1 Mild	Significant
Hypernasality	6	5 Severe	1 Mild	Significant
Phonation	6	3	1	Improved
Articulation	6	3	1	Improved
TOTAL SCORE	30	18 Severe VPI	4 Competent	+14 Points Overall Improvement

*PWSS Scoring System: 0 = Normal/No Deficit, 30 = Severe Dysfunction. Scores < 5 are considered clinically competent.

3. Discussion

The management of recurrent palatal fistulas, particularly those classified as Pittsburgh Class V and VI, represents one of the most formidable challenges in craniofacial reconstruction.[11] To understand the necessity of the "Sandwich" technique, one must first rigorously deconstruct the pathophysiology of recurrence in this specific patient population. A recurrent fistula is not merely an anatomical void; it is the physical manifestation of a "hostile" recipient bed characterized by biological exhaustion. In patients who have undergone primary palatoplasty and subsequent failed secondary closures, the local palatal mucoperiosteum undergoes profound micro-architectural changes. The native tissue is replaced by a dense cicatricial matrix, characterized by disorganized collagen deposition and, critically, a significant reduction in capillary density.[12]

This transformation creates a precarious hemodynamic environment. When this scarred tissue is elevated as a turnover flap or a rotation-advancement flap, it relies entirely on a "random-pattern" blood supply—perfusion dependent on the dermal-subdermal plexus rather than a defined axial vessel. In a scarred bed, this plexus is often attenuated or obliterated. Consequently, when the surgeon places tension on these flaps to bridge a wide Class V defect (junctional defects), the intraluminal pressure of the capillaries is exceeded by the mechanical tension of the closure. This results in a zone of relative ischemia at the suture line, leading to marginal necrosis, dehiscence, and inevitably, the recurrence of the fistula. Furthermore, the biomechanics of the Class V region exacerbate this ischemic insult. The junction of the hard and soft palate is a dynamic zone, subjected to the constant, multi-vector pull of the tensor veli palatini and levator veli palatini muscles. These muscular forces exert a centrifugal pull on the repair, actively distracting the wound edges during every act of swallowing and

phonation. Therefore, the "Triad of Failure"—ischemia, tension, and dynamic distraction—renders local tissue closure fundamentally inadequate for these high-grade defects.[13]

Our proposed "Sandwich" protocol—incorporating an anteriorly-based dorsal tongue flap and an autologous dermofat graft—was designed to systematically dismantle this Triad of Failure through architectural innovation (Figure 1). The central tenet of this approach is the concept of "breaking the line of repair." In conventional single-layer or simple double-layer closures, the nasal and oral suture lines are often superimposed or lie in close proximity. If the nasal layer creates a pinhole dehiscence due to the aforementioned ischemia, saliva and oral flora can immediately track through to the nasal cavity, establishing a patent tract.[14]

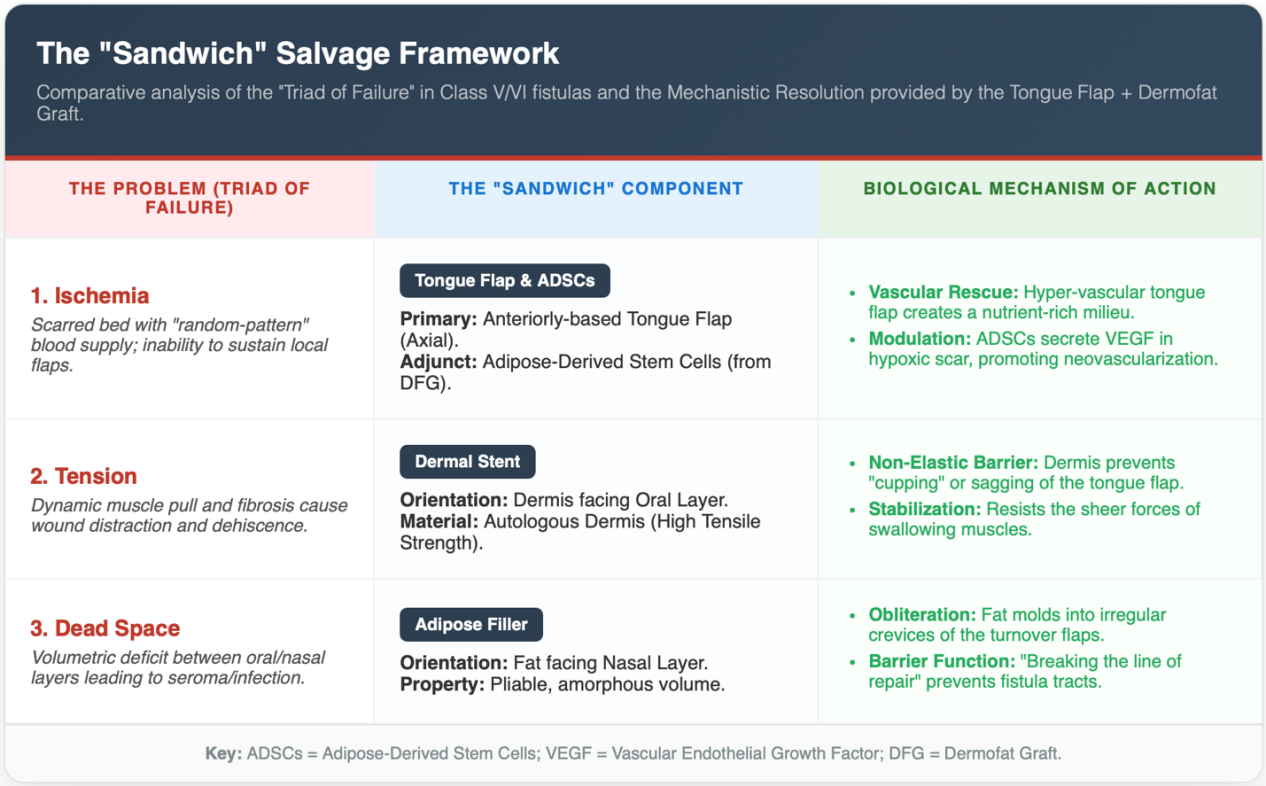

Figure 1. The sandwich salvage framework.

By interposing a vascularized, autologous dermofat graft between the nasal turnover flap (the "floor") and the tongue flap (the "roof"), we physically uncouple the two disparate biological environments. The graft serves as a biological buffer, increasing the physical distance between the oral and nasal cavities. Even in the event of a minor dehiscence of the nasal turnover flap, the overlying dermofat graft acts as a hydrophobic barrier, preventing the formation of a through-and-through communication. Crucially, the specific orientation of the graft—fatty surface facing the nasal layer and dermis facing the oral tongue flap—is not arbitrary but functionally distinct. The dermal component of the graft serves a structural role. Dermis is composed of dense, irregular connective tissue rich in elastin and Type I collagen.[15] Unlike fat or mucosa, it possesses significant tensile strength and resistance to mechanical shear. By placing the dermis against the undersurface of the tongue flap, it acts as a non-elastic "stent," stabilizing the repair and

preventing the tongue flap from sagging or "cupping" into the defect. Conversely, the adipose component, which is pliable and amorphous, serves as a volumetric filler. It molds itself into the irregular, scarred crevices of the nasal turnover flaps, effectively obliterating dead space. This management of dead space is critical; the elimination of potential cavities prevents the accumulation of seroma or hematoma, which are potent culture media for subclinical infection and subsequent graft loss.[16]

A critical and valid critique of fat grafting in any reconstructive domain is the unpredictability of long-term volume retention. The "Peer's Cell Theory" posits that surviving adipocytes maintain volume, while non-viable cells are replaced by fibrous tissue or oil cysts. Current literature suggests that non-vascularized fat grafts can lose between 30% and 60% of their volume within the first year. This resorption is primarily driven by the "zone of necrosis"—the central core of the graft that succumbs to ischemia before neovascularization can penetrate from the periphery.

However, our specific "Sandwich" technique mitigates this high resorption rate through two distinct biological mechanisms. First, we hypothesize a "Vascular Rescue Effect." Unlike free fat grafting into a generic subcutaneous pocket, the dermofat graft in this protocol is immediately covered by the anteriorly-based tongue flap. The tongue is an organ of exceptional vascularity, supplied by the robust dorsal lingual arteries and a rich anastomotic plexus. By suturing this hyper-vascular flap directly over the graft, we bathe the graft in a nutrient-rich milieu, accelerating the process of inosculation (the connection of donor and recipient vessels).[17] This reduces the duration of the ischemic phase, thereby maximizing the survival of the fragile adipocytes.

Secondly, and perhaps more profoundly, is the potential role of Adipose-derived stem cells (ADSCs). The dermofat graft is not merely an inert filler; it is a biologically active tissue. Adipose tissue is a rich reservoir of multipotent mesenchymal stem cells. In the context of a "hostile," scarred palatal bed—which is essentially a chronic ischemic wound—evidence suggests that ADSCs are activated by local hypoxic cues. Once activated, these cells function via paracrine signaling, releasing pro-angiogenic factors such as vascular endothelial growth factor (VEGF) and hepatocyte growth factor (HGF). These factors promote neovascularization of the surrounding scar tissue, effectively modulating the recipient bed. By converting a hypovascular, fibrotic environment into a more receptive, vascularized matrix, the dermofat graft may improve not only its own survival but also the healing of the overlying tongue flap.[18]

The selection of the tongue flap over the facial artery musculomucosal (FAMM) flap for this specific indication was driven by geometric and functional superiority for midline defects. While the FAMM flap is a valuable tool in the cleft surgeon's armamentarium, it possesses inherent limitations when addressing anterior Class VI defects. The FAMM flap relies on a rotational arc centered at the retromolar trigone. To reach the anterior hard palate or the incisive foramen region, the flap must be harvested with significant length, increasing the risk of distal tip necrosis. Furthermore, the acute angle required to pivot the flap into the midline often results in "kinking" of the pedicle, leading to venous congestion—a complication that can compromise the entire reconstruction.

In contradistinction, the anteriorly-based dorsal tongue flap is centrally located, offering a direct, linear approach to the palate without the need for acute angulation. This "mirror image" geometry ensures that the vascular pedicle remains straight and patent, minimizing the risk of venous compromise. Additionally, the functional quality of the tissue must be considered. The dorsal surface of the tongue is covered by specialized masticatory mucosa (papillae), which is texturally more similar to the rugae of the hard palate than the smooth, slippery buccal mucosa of the FAMM flap. This textural similarity may offer subtle advantages in the articulation of lingual-alveolar sounds as the tongue tip interacts with a surface that provides appropriate friction and proprioceptive feedback.[19]

Despite the promising clinical and functional outcomes presented in this study, it is imperative to acknowledge the limitations inherent in this report to maintain scientific integrity. First and foremost, this study represents Level V evidence—a single case presentation. While the biological rationale is sound and the surgical technique is reproducible, a sample size of one precludes any statistical generalization regarding recurrence rates. We cannot definitively state that the dermofat graft was the sole determinant of success; it is plausible that a meticulously executed tongue flap alone might have sufficed, although the risk of concave collapse would have remained.

Secondly, the follow-up period of 6 months, while sufficient to confirm initial fistula closure and the return of speech competency, represents the minimum threshold for assessing graft stability. Adipose tissue resorption is a dynamic process that can continue for 12 to 18 months post-operatively. While the clinical contour remains robust in our patient, late-stage volumetric loss cannot be ruled out. The lack of objective volumetric quantification—such as pre- and post-operative Magnetic Resonance Imaging (MRI) or 3D intraoral scanning—renders our assessment of "volume retention" subjective. Future comparative studies utilizing such imaging modalities are necessary to precisely quantify the percentage of fat retention in the sub-lingual environment.

Finally, the morbidity of a two-stage procedure cannot be overlooked. The three-week period of pedicle attachment imposes a significant burden on the patient, including temporary dysphagia, dysarthria, and the psychological stress of oral tethering. While our patient tolerated this well under a strict compliance protocol, this requirement may contraindicate the procedure in non-compliant patients or those with pre-existing airway compromise. Nevertheless, for the specific cohort of patients with recalcitrant, multi-recurrent Class V-VI fistulas who have exhausted local tissue options, the "Sandwich" technique offers a robust, biologically augmented salvage strategy that prioritizes the physiological principles of vascularity and dead-space obliteration over the convenience of single-stage repair.[20]

4. Conclusion

The management of recurrent Pittsburgh Class V-VI palatal fistulas requires a departure from standard local flap techniques. The "Sandwich" technique—combining an anteriorly-based tongue flap with an autologous dermofat graft—represents a biologically sound salvage strategy. By providing reliable axial vascularity and obliterating dead space with autologous tissue, this protocol addresses the root causes of recurrence. While this case demonstrates proof-of-concept with excellent functional speech outcomes (PWSS 4/30), comparative studies with larger cohorts are required to statistically validate the superiority of this composite graft over the standard tongue flap alone. Nevertheless, for the recalcitrant "failure of failure" case, this technique offers a robust and reproducible mechanism to minimize the theoretical risk of recurrence, providing a pathway to speech competency for complex cleft patients.

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