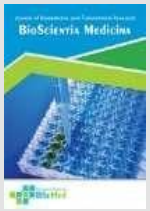
eISSN (Online): 2598-0580



Bioscientia Medicina: Journal of Biomedicine & Translational Research

Journal Homepage: www.bioscmed.com

Dual-Modality Salvage of Recurrent Pacemaker Extrusion in Severe Malnutrition: A Technique Using PTFE Mesh Encapsulation and Fasciocutaneous Flap Coverage

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ARTICLE INFO

Keywords:
Fasciocutaneous flap
Malnutrition
Pacemaker extrusion
PTFE mesh
Surgical salvage

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All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/bsm.v10i4.1568

A B S T R A C T

Background: Permanent pacemaker implantation in pediatric patients with severe malnutrition presents a unique surgical challenge due to the absence of a viable subcutaneous protective envelope. In this population, standard device fixation frequently results in recurrent wound dehiscence and device extrusion, often necessitating lead abandonment or epicardial placement. This study evaluates the efficacy of a dual-modality salvage technique integrating a Polytetrafluoroethylene (PTFE) mesh barrier with vascularized fasciocutaneous tissue coverage. **Case presentation:** We present the case of a 3-year-old male with congenital complete atrioventricular block and severe acute malnutrition, defined by a Weight-for-Height Z-score of less than -3 SD. The clinical course was complicated by three consecutive implant failures over an 8-month period, including two thoracic and one abdominal extrusion, characterized by aseptic pressure necrosis. To salvage the cardiac hardware, a novel sealed-device technique was employed. The pulse generator was encapsulated in a non-absorbable PTFE mesh to minimize the coefficient of friction and placed in a sub-fascial plane. Simultaneously, a random-pattern fasciocutaneous rotation flap was harvested to provide robust, vascularized coverage. Biochemical analysis revealed severe hypoalbuminemia (2.1 g/dL) and anemia pre-operatively. **Conclusion:** At the 12-month follow-up, the surgical site demonstrated complete physiological integrity with no recurrence of erosion, seroma, or infection. The combination of PTFE encapsulation to mitigate mechanical shearing forces and a fasciocutaneous flap to restore perfusion offers a durable salvage strategy for refractory device extrusion in cachectic pediatric patients.

1. Introduction

The implantation of a permanent pacemaker (PPM) stands as the definitive, life-saving intervention for the management of irreversible, high-grade cardiac conduction disorders in the pediatric population. Since the advent of implantable cardiac rhythm management devices, survival rates and quality of life for children with congenital complete atrioventricular (AV) block or acquired postsurgical conduction defects have improved dramatically.[1] However, the pediatric host presents a unique and unforgiving physiological environment for these devices. Unlike the adult population, where the generator is easily accommodated within the generous subcutaneous adipose tissue of the pre-pectoral region, pediatric patients possess significantly smaller somatic dimensions, limiting the available surface area for device implantation. Furthermore, the dynamic nature of pediatric growth, combined with the typically higher activity levels of children, subjects the implanted hardware to continuous mechanical stress and geometric distortion. Consequently, while the

procedure is considered routine, the complication profile in the pediatric demographic is distinct, severe, and clinically taxing.[2]

The weak link in pediatric cardiac pacing remains the device-tissue interface. Literature indicates that complications such as lead extrusion, pocket hematoma, deep surgical site infection, and frank device exposure occur in approximately 12 percent of pediatric cases—a rate significantly higher than that observed in adults. Among these, device extrusion represents a catastrophic failure of the soft-tissue envelope. It typically begins as a subtle area of skin thinning or discoloration (pre-erosion) over the prominent edge of the pulse generator, progressing to full-thickness necrosis and eventual exteriorization of the sterile hardware. Once the device is exposed to the external environment, the pocket is inevitably colonized by cutaneous flora, necessitating complete system explantation to prevent systemic endocarditis. This initiates a vicious cycle of reimplantation, scar formation, and recurrent failure, often exhausting the limited available sites for implantation (pre-pectoral, abdominal, or retroperitoneal).[3]

These surgical risks are not uniformly distributed. They are disproportionately prevalent in low-to-middle-income countries (LMICs), where pediatric malnutrition remains a pervasive comorbidity that fundamentally alters the surgical risk landscape.[4] Malnutrition is not merely a background variable; it is a potent biological driver of surgical failure. In many developing regions, children with congenital heart disease often present with concomitant chronic protein-energy malnutrition (PEM) due to a combination of cardiac cachexia (increased metabolic demand) and inadequate caloric intake. The presence of severe malnutrition transforms a routine subcutaneous pocket creation into a high-risk reconstructive challenge. The double burden of complex cardiac pathology and metabolic depletion creates a host environment that is hostile to foreign body retention, significantly elevating the risk of wound breakdown and implant rejection.[5]

The pathophysiology of device extrusion in the malnourished child is multifactorial, representing a convergence of metabolic failure and biomechanical mismatch. This double-hit phenomenon explains why standard surgical techniques that are successful in well-nourished children frequently fail in this specific sub-population. Wound healing is a complex, energy-dependent biological cascade comprising hemostasis, inflammation, proliferation, and remodeling.[6] Malnutrition systematically dismantles this cascade. Specifically, protein deficiency arrests the proliferative phase of wound healing by limiting the availability of amino acids required for collagen synthesis and cross-linking. The synthesis of Collagen Type I—the primary structural protein responsible for the tensile strength of the surgical scar—is severely hindered. Furthermore, micronutrient deficiencies (such as Zinc and Vitamin C) impair the recruitment and function of neutrophils and macrophages, blunting the immune response and increasing susceptibility to subclinical bacterial colonization. Without a robust inflammatory response and adequate collagen deposition, the fibrous capsule that normally forms around a pacemaker to stabilize it fails to mature. Instead, the pocket remains fundamentally unstable, with a weak, friable internal lining that cannot integrate with the device. The second component of failure is purely mechanical. In a healthy child, the subcutaneous fat layer acts as a crucial shock absorber, distributing the pressure of the rigid metallic pulse generator across a wide surface area. In the malnourished child, the profound depletion of subcutaneous adipose tissue and muscle mass eliminates this protective cushion. The metallic generator rests directly against the undersurface of the dermis, separated only by a paper-thin layer of friable connective tissue. This scenario is governed by Laplace's Law, which dictates that wall tension is directly proportional to the intracavitary pressure and the radius of the curvature. The sharp, rigid corners of the pulse generator create focal points of high pressure. Without tissue bulk to distribute this mechanical load, the pressure exerted by the device on the overlying skin exceeds the capillary closing

pressure of the dermal vascular plexus. This creates localized ischemic zones—pathophysiologically identical to the formation of decubitus ulcers (pressure sores). Over time, this chronic ischemia leads to pressure necrosis, thinning of the skin (the cheese-wiring effect), and eventual breach of the integument.

When extrusion occurs, the standard surgical reflex is often reactive rather than reconstructive. Conventional salvage protocols typically involve removing the device, debriding the wound, and attempting primary re-closure or applying a split-thickness skin graft. However, these techniques are associated with unacceptably high failure rates in malnourished patients because they address symptoms rather than root causes. Primary closure in a cachectic patient merely recruits the same thinned, ischemic, and poorly vascularized tissue back over the device, placing it under tension. Since the underlying mechanical stress (the hard device against thin skin) and the vascular deficiency remain unchanged, recurrence is almost guaranteed. Similarly, skin grafting provides coverage but fails to provide bulk or padding, offering no protection against future pressure necrosis.[7]

Recognizing the need to stabilize the device, surgeons have historically employed various pouch technologies. The most common is the Dacron pouch (Parsonnet bag), designed to prevent device migration. However, Dacron is a multifilament textile that can incite a vigorous foreign body giant cell reaction, leading to dense fibrosis.[8] In a child with little subcutaneous fat, this fibrotic reaction can create a rock-hard, painful capsule that adheres to the skin, potentially accelerating erosion rather than preventing it. More recently, antibiotic-eluting envelopes have gained popularity for infection prevention. While highly effective at reducing bacterial burden, these envelopes are absorbable (resorbable) and dissolve over weeks to months. Consequently, they offer no long-term mechanical protection or cushioning effect for the malnourished patient who may remain thin for years. There remains a critical lack of a permanent, biocompatible solution that provides a low-friction interface between the device and the fragile skin.

To overcome these dual challenges—mechanical friction and poor vascularity—we must look beyond standard cardiac surgery techniques to solutions found in vascular and reconstructive surgery. Expanded Polytetrafluoroethylene (ePTFE) is a chemically inert, non-porous biomaterial widely utilized in vascular grafting and hernia repair. Its defining characteristic is its extremely low coefficient of friction and high biocompatibility.[9] Unlike Dacron, ePTFE elicits a minimal inflammatory response and does not adhere tenaciously to surrounding tissues. We hypothesize that encapsulating the pulse generator in a smooth ePTFE mesh will create a permanent bursa-like sac. This effectively changes the physics of the pocket: instead of the device rubbing abrasively against the tissue, the smooth mesh glides, dissipating shear forces and distributing pressure over a wider surface area. Mechanical buffering alone is insufficient if the overlying skin is ischemic. The reconstructive principle of replacing like with like is inadequate in the malnourished patient; we must replace compromised tissue with vascularized tissue. A fasciocutaneous flap, which includes the skin, subcutaneous tissue, and the underlying deep fascia, recruits a dedicated blood supply independent of the compromised wound bed. The inclusion of the fascia provides a robust mechanical shield—a vascularized cushion—that separates the device from the external environment and delivers the oxygen and nutrients necessary for healing, even in the presence of systemic malnutrition.[10]

Current literature offers sparse guidance on the management of refractory extrusion in the context of severe physiological depletion. Most guidelines focus on infection control rather than mechanical stabilization in cachectic hosts. This study aims to bridge that gap by describing the technical details and clinical outcomes of a novel dual-modality salvage approach. The novelty of this study lies in the synergistic integration of two distinct surgical principles: (1) Synthetic Barrier Protection: The

construction of a sealed-device using a sterile PTFE mesh container to act as a permanent, low-friction, biocompatible interface; (2) Vascularized Augmentation: The concurrent application of a robust fasciocutaneous flap to overcome the biological failures of the native skin envelope. We report the successful implementation of this technique in a unique and challenging case of a severely malnourished pediatric patient who had exhausted all standard options. By moving from a reactive strategy to a proactive reconstruction, we aim to provide a reproducible blueprint for salvaging life-saving cardiac hardware in the most vulnerable patient populations.

2. Case Presentation

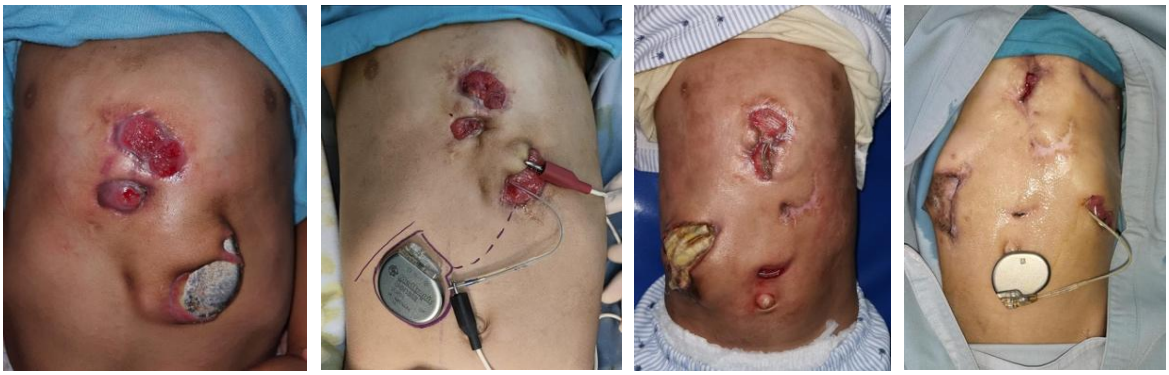
This study was conducted in strict adherence to the ethical principles outlined in the Declaration of Helsinki regarding medical research involving human subjects. The surgical intervention described herein was performed as a clinically indicated salvage procedure to address a life-threatening complication; consequently, formal review by the Institutional Review Board (IRB) of Dr. Hasan Sadikin General Hospital was consulted, and the study was deemed exempt from full committee review, provided that strict patient confidentiality was maintained. However, given the innovative nature of the sealed-device technique and the utilization of Polytetrafluoroethylene (PTFE) mesh for pulse generator encapsulation, a specialized consent process was undertaken. Written informed consent was obtained from the patient's legal guardians (parents) prior to the procedure. This consent explicitly detailed the risks, benefits, and potential alternatives to the surgical approach, including the specific rationale for integrating the fasciocutaneous flap with the prosthetic mesh. Furthermore, separate written consent was secured from the guardians for the publication of this case report and the reproduction of any accompanying intra-operative photographs. All patient data has been de-identified to protect anonymity, and no personal details that could lead to the identification of the patient have been included in this manuscript.

A 3-year-old male presented to the Department of Cardiology with a primary diagnosis of congenital complete atrioventricular (AV) block. Concurrently, the patient was diagnosed by the pediatric nutrition service with Marasmus, characterized by significant muscle wasting and a Weight-for-Height Z-score of -3.2 SD, indicating severe acute malnutrition. The patient's surgical history was notable for recalcitrant failure of device retention, classified as refractory extrusion. The timeline of failure is detailed in Table 1.

Table 1 delineates the chronological progression of surgical interventions and the recurrent failure modes observed in this challenging clinical scenario. The timeline highlights a recalcitrant pattern of device rejection spanning from January to August 2022, characterized by the inability of the malnourished host tissue to maintain structural integrity over the prosthetic hardware. The initial failures in the thoracic region demonstrated the inadequacy of the pre-pectoral plane, where the profound depletion of subcutaneous fat led to rapid pressure necrosis and device protrusion within merely two weeks of implantation. Subsequent attempts at contralateral reimplantation similarly failed, resulting in wound dehiscence and purulent drainage despite primary closure. Crucially, the table documents the failure of the abdominal relocation attempt in July 2022. Despite the theoretical advantage of greater tissue bulk in the rectus sheath, the device re-extruded by August, confirming that anatomical relocation alone was futile without addressing the underlying biomechanical friction and vascular compromise. This sequence establishes the clinical justification for the radical change in strategy employed in October 2022. The transition from repetitive conventional closure to the sealed-device salvage technique represents the pivotal turning point in the patient's course, interrupting the cycle of extrusion and enabling the successful healing and long-term retention recorded in the final entry.

Table 1. Timeline of Surgical Interventions and Failures

DATE	PROCEDURE	ANATOMICAL SITE	OUTCOME	MECHANISM OF FAILURE
Jan 2022	Primary Transvenous Implantation	*Left Pre-pectoral*	GRADE IV EXTRUSION	Mechanical pressure necrosis due to lack of subcutaneous fat.
Feb 2022	Revision & Contralateral Implant	*Right Pre-pectoral*	WOUND DEHISCENCE	Impaired inflammatory phase and collagen deposition failure.
July 2022	Relocation to Abdominal Pocket	*Right Rectus Sheath*	RECURRENT EXTRUSION	Progressive tissue thinning and atrophy over metal edge.
Oct 2022	**Sealed-Device Salvage**	***Left Abdominal Wall (Sub-fascial)***	HEALED (12 MO)	**Successful friction reduction (PTFE) and vascular restoration (Flap).**



January 2022 February 2022 July 2022 October 2022

Figure 1. Timeline of surgical intervention and failures.

Physical examination upon presentation to the Plastic Surgery service revealed a cachectic child with visible skeletal prominence. The pacemaker generator was visible through a 3 cm defect in the right lower quadrant of the abdominal skin. The wound edges were violaceous and thinned, with the metallic header of the device clearly exposed. While there was no frank purulence, the wound margins were non-viable and necrotic.

Table 2 provides a quantitative analysis of the patient's metabolic and immunological status, contrasting the acute pre-operative phase with the physiological restoration observed at the six-month follow-up. The data highlights the profound severity of the initial catabolic state, with a serum albumin level of 2.1 g/dL indicating severe visceral protein depletion. This hypoalbuminemia is clinically significant as it correlates directly with decreased colloid oncotic pressure and impaired transport of essential micronutrients like zinc and calcium, both critical for the inflammatory and proliferative phases of wound healing. Furthermore, the pre-operative hemoglobin of 8.5 g/dL suggests a significant microcytic anemia, likely secondary to chronic iron deficiency, which diminishes the oxygen-carrying capacity required to sustain the metabolic demands of

the regenerating tissue at the surgical site. The table also elucidates the immunological impact of malnutrition, evidenced by a total lymphocyte count of 1,200/mm³, placing the patient in an immunocompromised category susceptible to opportunistic infection. This is corroborated by the microbiological finding of *Staphylococcus epidermidis* from the wound bed—a commensal organism that typically forms low-grade biofilms on prosthetic devices rather than causing fulminant sepsis. The dramatic normalization of these markers at six months (Albumin 3.8 g/dL; Negative culture) serves as biochemical validation of the multidisciplinary treatment strategy. It confirms that the surgical salvage did not occur in isolation but was successfully synergized with aggressive nutritional rehabilitation to reverse the metabolic double-hit that precipitated the initial device failure.

Table 2. Biochemical Nutritional and Inflammatory Markers

Comparative analysis of acute phase (pre-operative) vs. restored physiological state (6-month follow-up).

PARAMETER	PRE-OPERATIVE (Acute Phase)	POST-OPERATIVE (Month 6)	PEDIATRIC REFERENCE RANGE
Serum Albumin	**2.1 g/dL** Severe Visceral Depletion	**3.8 g/dL** Normalized	*3.8 – 5.4 g/dL*
Hemoglobin	**8.5 g/dL** Microcytic Anemia	**11.2 g/dL** Target Reached	*11.5 – 13.5 g/dL*
Total Lymphocytes	**1,200 /mm³** Immune Compromised	**2,500 /mm³** Restored Competence	*> 2,000 /mm³*
C-Reactive Protein (CRP)	**12 mg/L** Mild Elevation (Inflammatory)	**< 1.0 mg/L** No Inflammation	*< 5.0 mg/L*
Wound Culture	**Positive** *Staphylococcus epidermidis*	**Negative** Sterile Site	*Negative*

The salvage of a recurrently extruded pacemaker in a cachectic pediatric patient demands a departure from standard surgical algorithms, necessitating a collaborative approach between cardiothoracic and reconstructive plastic surgery (Table 3). The procedure was orchestrated under general anesthesia with the patient placed in the supine position to allow simultaneous access to both the compromised abdominal site and the contralateral virgin territory. The operation was conceptualized not merely as a device reimplantation, but as a total reconstruction of the soft-tissue envelope, structured in four distinct, physiologically grounded phases.

The initial phase focused on the elimination of the compromised wound bed and the reduction of bioburden. The extruded pulse generator was carefully explanted from the right abdominal pocket. Although the clinical presentation suggested aseptic pressure necrosis rather than fulminant sepsis, intra-operative swab cultures were obtained from the pocket floor to guide antibiotic therapy. These cultures subsequently yielded *Staphylococcus epidermidis*, a finding consistent with the biofilm hypothesis of device extrusion—where low-grade bacterial colonization

prevents tissue integration and perpetuates chronic inflammation. Consequently, a radical debridement was performed. Using sharp dissection, the fibrous pseudocapsule and all non-viable, friable tissue margins were excised until healthy, bleeding wound edges were visualized. To further reduce the risk of recolonization, the pocket was subjected to high-volume irrigation with three liters of normal saline mixed with Gentamicin. The original device, deemed a vector for biofilm seeding, was discarded, and a pristine, sterile pulse generator was prepared for implantation.

The second phase involved the fabrication of the sealed-device construct, designed to mitigate the biomechanical friction that had driven previous failures. A sterile sheet of 1mm thick expanded Polytetrafluoroethylene (ePTFE) mesh (Gore-Tex®) was selected for its chemical inertness and low coefficient of friction. The mesh was meticulously folded to create a custom-fitted envelope tailored to the exact dimensions of the new pulse generator. The generator was inserted into this prosthetic pouch, and the open edges were approximated using interrupted 5-0 Polypropylene (Prolene) sutures. The tension of this encapsulation was critically engineered: the fit was snug enough to stabilize the device and prevent rotation within the bag (mitigating the risk of Twiddler's syndrome), yet loose enough to maintain microporosity for fluid diffusion, thereby preventing seroma formation. This effectively encased the rigid metallic hardware in a soft shell, converting the abrasive edges of the header and connector block into a smooth, gliding surface less likely to erode through the thin pediatric skin.

To address the fundamental lack of subcutaneous padding, the reconstructive team targeted a virgin site on the left lower abdominal quadrant, abandoning the scarred tissue of the right side. A sub-fascial plane was dissected beneath the external oblique aponeurosis; placing the device deep to this fascia utilized the patient's own aponeurosis as an additional, autologous barrier against extrusion. Simultaneously, a local random-pattern fasciocutaneous rotation flap was designed adjacent to the new incision. This flap was harvested strictly preserving the dermal-subdermal vascular plexus, recruiting skin, subcutaneous fat, and the underlying superficial fascia to bring a robust, independent blood supply to the surgical bed. The PTFE-encased pacemaker was then inserted into the sub-fascial pocket, and the flap was rotated to cover the defect. A critical tenet of this reconstruction was the offset closure principle: the flap inset was designed such that the final suture line did not lie directly over the hardware, ensuring that any minor wound dehiscence would not result in immediate device exposure. The final closure was performed with extreme delicacy to avoid compromising the tenuous vascularity of the flap. Deep tension sutures were strictly avoided to prevent strangulation of the blood supply. The skin was approximated using 5-0 Poliglecaprone (Monocryl) in a subcuticular fashion, minimizing external scarring and eliminating suture tracks that could serve as entry points for bacteria.

Post-operatively, the patient was managed with a targeted seven-day course of intravenous Vancomycin, guided by the intra-operative cultures. This was concurrent with an aggressive nutritional rehabilitation program utilizing F-100 therapeutic milk to reverse the catabolic state. The longitudinal follow-up validated the efficacy of this dual-modality approach. At two months, the fasciocutaneous flap was fully viable with no signs of venous congestion or necrosis. By six months, the patient's nutritional markers had normalized (Serum Albumin 3.8 g/dL), and the device was palpable but mobile within the PTFE envelope, indicating a healthy integration without severe capsular contracture. At the one-year milestone, the site demonstrated robust soft-tissue coverage with no evidence of skin thinning. Crucially, electrical interrogation confirmed normal impedance and sensing thresholds, proving that the PTFE mesh acted as an effective mechanical barrier without interfering with the device's telemetry or life-saving pacing function.

Table 3. Clinical Synopsis

Comprehensive overview of Diagnosis, Surgical Management, and Long-Term Outcomes

Diagnosis & Pre-operative Status	**Primary Pathology** **Congenital Complete Atrioventricular (AV) Block** requiring permanent pacing. **Comorbidities** ◦ **Severe Acute Malnutrition (Marasmus):** Weight-for-Height Z-score < -3 SD. ◦ **Recurrent Extrusion:** History of 3 failed implantations (2 thoracic, 1 abdominal) due to pressure necrosis. ◦ **Metabolic Deficit:** Hypoalbuminemia (2.1 g/dL) and Microcytic Anemia (Hb 8.5 g/dL).
Surgical Treatment & Intervention	**Procedure: Sealed-Device Salvage** **Date:** October 2022 **Anesthesia:** General Anesthesia (Supine Position) **Key Surgical Steps** ◦ **Explants:** Removal of extruded device; aggressive debridement of necrotic wound edges. ◦ **Encapsulation:** Pulse generator wrapped in 1mm **ePTFE Mesh** (Gore-Tex®) to reduce friction. ◦ **Reconstruction:** Creation of sub-fascial pocket in virgin site (Left Lower Quadrant). ◦ **Coverage:** Rotation of **Fasciocutaneous Flap** to provide vascularized soft tissue padding.
Outcome & Follow-up	**Immediate Post-Op (2 Months)** ◦ Complete wound healing with no dehiscence. ◦ Flap viable; no venous congestion or necrosis. **Long-Term (12 Months)** ◦ **Retention:** 100% device retention with no skin thinning or erosion. ◦ **Function:** Normal sensing and pacing thresholds; no telemetry interference from PTFE. ◦ **Systemic Recovery:** Normalization of nutritional markers (Albumin 3.8 g/dL).

3. Discussion

The management of recurrent pacemaker extrusion in the pediatric patient with severe malnutrition represents a distinct and formidable clinical entity that challenges the conventional axioms of cardiac rhythm device management. While device erosion in the adult population is often attributed to subclinical infection or technical error, the etiology in the cachectic pediatric host is fundamentally different. This case illustrates that successful salvage in this demographic is contingent upon a paradigm shift: surgeons must move beyond the simplistic view of pocket revision and adopt a reconstructive framework that simultaneously addresses the triad of failure—metabolic deficiency, biomechanical stress, and bacterial colonization. Failure to recognize and treat all three arms of this triad invariably leads to the cycle of recurrence observed in this patient's history.[11]

To understand the rationale for this complex reconstruction, one must first dissect the pathophysiology of the failure. The primary driver of device extrusion in this patient was not the device

itself, but the host's inability to generate a stable biological interface. In a healthy physiological state, the insertion of a pacemaker pulse generator triggers a foreign body response characterized by acute inflammation followed by fibroplasia. This process culminates in the formation of a dense, avascular collagenous capsule (Types I and III collagen) that effectively sequesters the device from the surrounding tissue and stabilizes it against migration. In the context of severe protein-energy malnutrition (PEM), this sequence is arrested. The metabolic demands of wound healing are immense; the proliferative phase relies heavily on the availability of amino acid substrates for protein synthesis and micronutrients such as Zinc and Vitamin C for collagen cross-linking.[12] In a state of Marasmic wasting, the body prioritizes visceral organ function over cutaneous integrity. Consequently, fibroblast proliferation is blunted, and the synthesis of structural collagen is defective. The result is a pocket lined not by a robust fibrous capsule but by friable, immature granulation tissue that lacks the tensile strength to retain the hardware.

This biological frailty is exacerbated by a catastrophic biomechanical mismatch. The complete depletion of subcutaneous adipose tissue removes the natural shock absorber that normally separates the rigid metallic generator from the overlying dermis.[13] In this patient, the generator was essentially resting directly against the undersurface of the skin. Applying Laplace's Law to this scenario reveals the inevitability of failure: wall tension is directly proportional to the intracavitary pressure exerted by the device. Without the soft-tissue bulk to distribute this mechanical load over a wider surface area, the sharp geometric edges of the pulse generator create focal points of intense pressure. When this pressure exceeds the capillary closing pressure of the dermal plexus (approximately 32 mmHg), localized ischemia ensues. This mimics the pathogenesis of a decubitus ulcer, leading to sterile pressure necrosis and the eventual cheese-wiring of the device through the skin. The isolation of *Staphylococcus epidermidis* from the wound bed must be interpreted within this context. While often dismissed as a contaminant, in the presence of an unstable, eroding device, *S. epidermidis* acts as a secondary opportunist. The race for the surface concept dictates that if host tissue cells do not integrate with the implant first, bacteria will colonize the surface and form a biofilm. This biofilm perpetuates a state of chronic, low-grade inflammation that further degrades the surrounding tissue, preventing any hope of delayed healing. Thus, the bacteria are both a consequence of the mechanical instability and a cause of its persistence.[14]

The decision to utilize an expanded Polytetrafluoroethylene (ePTFE) mesh envelope was the cornerstone of our sealed-device technique. This choice was driven by the specific need for a permanent mechanical interface, distinguishing our approach from current trends in infection control.[15] While absorbable antibiotic-eluting envelopes have become the standard of care for high-risk re-implantations, their utility in the malnourished patient is limited by their lifespan. These envelopes typically dissolve within nine weeks. Once absorbed, the patient is left with the same biomechanical vulnerability: a metal device against thin skin.[16]

We required a material that would provide indefinite mechanical buffering. Historically, surgeons utilized Dacron pouches (Parsonnet bags) to stabilize loose devices. However, Dacron is a multifilament textile known to induce a vigorous foreign body giant cell reaction. In a child with minimal subcutaneous padding, this reaction often results in a rock-hard, calcified capsule that can become painful and adherent to the skin, paradoxically increasing the risk of erosion.[17] In contrast, ePTFE was selected for its distinct material science properties: (1) Low Coefficient of Friction: ePTFE is chemically inert and possesses one of the lowest coefficients of friction of any solid material. By encapsulating the generator in this mesh, we effectively altered the tribology of the pocket. The abrasive metallic surface of the titanium can, and the sharp edges of the epoxy header were converted into a smooth, gliding surface. This significantly reduces the

shear forces transmitted to the overlying fasciocutaneous flap during the child's respiratory movements and torso flexion. The device effectively floats within the tissue rather than grinding against it; (2) Microporosity and Tissue Integration: The specific internodal distance (pore size) of the ePTFE mesh allows for a controlled biological interaction. It permits limited tissue ingrowth on the outer surface, which serves to anchor the bag to the sub-fascial pocket, preventing device migration (Twiddler's syndrome). Crucially, the inner surface does not adhere tenaciously to the metal generator. This differential integration is vital for future lead revisions or generator replacements. When the battery eventually depletes, the surgeon can incite the capsule, identify the PTFE layer, and easily slide the device out of its artificial bursa without the need for extensive dissection that might damage the vascular supply of the overlying flap.[18]

The second pillar of this salvage technique was the recruitment of vascularized tissue via a fasciocutaneous flap. Standard surgical salvage options, such as primary closure or split-thickness skin grafting, are destined to fail in the cachectic patient because they rely on the health of the recipient bed. A skin graft requires a vascularized bed to survive (take), and primary closure recruits the same ischemic, thinned tissue that failed initially. The fasciocutaneous flap overcomes these limitations by adhering to the reconstructive principle of bringing independent vascularity to the zone of injury. By definition, this flap includes the skin, the subcutaneous fat, and the deep fascia, along with the pre-fascial vascular plexus that runs parallel to the skin surface. By rotating this composite tissue block over the implant, we achieved two critical physiological goals: (1) Vascular Induction and Rescue: The flap imports a robust, permanent blood supply that is independent of the scarred, compromised wound bed. This hypervascular tissue increases the local oxygen tension, which is essential for neutrophil function and oxidative killing of residual bacteria. It also facilitates the delivery of systemic antibiotics and the nutritional substrates (amino acids, glucose) necessary for healing, essentially rescuing the local environment from its metabolic desert.[19] Beyond vascularity, the flap provides immediate structural bulk. The inclusion of the deep fascia acts as a tough, autologous shield, while the subcutaneous fat (even if diminished) provides a necessary degree of padding. This effectively resets the distance to the surface, increasing the physical barrier between the device and the external environment. This restoration of soft-tissue thickness is the most effective safeguard against future pressure necrosis.

While the long-term success of this case is compelling, we must acknowledge the inherent limitations of this report. First, as a single-case study, the findings essentially represent a proof of concept. Larger cohort studies are required to establish the reproducibility of this technique across different operators and varying degrees of malnutrition. Second, the cost of medical-grade ePTFE mesh may be prohibitive in some resource-limited settings, though it remains significantly less expensive than commercial antibiotic-eluting envelopes. Surgeons in austere environments may need to weigh the cost of the mesh against the high cost of treating recurrent failures and systemic endocarditis. Third, a major confounding variable in this success was the concurrent aggressive nutritional rehabilitation. The patient received intensive caloric supplementation during the post-operative period. It is plausible that the nutritional recovery contributed significantly to the final healing. However, given the rapidity of the initial wound stability (within two months), we argue that the surgical intervention provided the necessary bridge that allowed the patient to retain the device long enough for nutritional parameters to normalize.[20] Future research should focus on the comparative efficacy of this technique versus bio-absorbable envelopes in the specific setting of pediatric malnutrition, and whether the addition of local antibiotic carriers (such as antibiotic-impregnated beads) within the PTFE pouch could further reduce infection risk.

4. Conclusion

Recurrent pacemaker protrusion in the malnourished pediatric patient is a life-threatening complication that defies standard surgical closure techniques. The convergence of metabolic incompetence, lack of soft tissue coverage, and mechanical instability creates a hostile environment where conventional reimplantation is destined to fail. This case demonstrates that a reactive surgical strategy—simply stitching the hole closed—is insufficient. Instead, a proactive reconstructive approach is required. We conclude that the sealed-device technique, comprising PTFE mesh encapsulation to minimize mechanical friction and fasciocutaneous flap rotation to restore vascularity, provides a durable and physiologically sound solution for salvage. This dual-modality approach effectively creates a neocapsule that isolates the device from the tissue bed while simultaneously providing a robust, vascularized soft-tissue envelope. This interrupts the cycle of pressure necrosis, biofilm formation, and extrusion. We recommend that this technique be considered not merely as a last resort for multiple failures, but potentially as a primary preventive strategy in the management algorithm for pediatric patients with high-risk nutritional profiles. By recognizing the unique biomechanical and metabolic needs of the malnourished child, surgeons can secure the longevity of these life-saving devices and prevent the significant morbidity associated with recurrent surgical failure.

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