PEDIATRIC SURGERY *Update**
Volume 66 No. 02 FEBRUARY 2026

Pyloric Atresia and Epidermolysis Bullosa

Pyloric atresia associated with epidermolysis bullosa represents one of the most severe congenital syndromes encountered in neonatal medicine, combining a mechanical obstruction of the gastric outlet with a profound disorder of skin and mucosal integrity. Although pyloric atresia alone accounts for only a small fraction of intestinal atresia, its association with epidermolysis bullosa markedly alters the clinical course, prognosis, and management priorities. This combined condition is rare, typically presenting in the neonatal period, and is characterized by early gastrointestinal obstruction, extensive skin fragility, and a high risk of multisystem complications that frequently culminate in early mortality .

Clinically, affected neonates usually present within the first days of life with non-bilious vomiting, feeding intolerance, and progressive abdominal distension caused by complete obstruction at the level of the pylorus. Radiographic imaging classically demonstrates a markedly distended stomach with absence of distal bowel gas, often referred to as a “single bubble” sign. These findings are often preceded by antenatal clues, particularly polyhydramnios and fetal gastric dilation detected on prenatal ultrasonography, reflecting impaired gastric emptying in utero. At the same time, cutaneous manifestations may be evident at birth or emerge shortly thereafter, including tense bullae, erosions, or areas of congenital skin absence. Even minimal mechanical trauma, such as handling or adhesive application, can provoke new blister formation, underscoring the extreme fragility of the integument in this disorder .

Epidermolysis bullosa with pyloric atresia is now recognized as a genetically determined condition most commonly inherited in an autosomal recessive pattern. The underlying defect involves proteins essential for dermo epidermal adhesion, particularly those associated with hemidesmosomes and the basement membrane zone. Pathogenic variants in genes encoding integrin α6, integrin β4, and plectin disrupt epithelial stability not only in the skin but also in the gastrointestinal tract, urinary system, and respiratory mucosa. This explains why the disease extends beyond the skin to involve pyloric development, renal structures, and internal epithelial linings. The phenotype varies in severity depending on the nature of the mutation, but many affected infants experience extensive disease with rapid clinical deterioration .

From a pathological standpoint, pyloric atresia in this syndrome may take several anatomical forms, ranging from a thin membranous web to a solid fibrous cord or a complete gap between the stomach and duodenum. These anatomical variations have important implications for surgical management. Less extensive lesions may permit pyloroplasty or excision of a pyloric membrane, whereas more complex forms require

bypass procedures such as gastroduodenostomy or gastrojejunal anastomosis. In practice, the choice of operation is often influenced not only by anatomy but also by the infant's overall condition, body size, tissue fragility, and the feasibility of safely mobilizing surrounding structures .

Surgical correction of the pyloric obstruction is essential for survival, yet it does not alter the underlying disease process. Even when surgery is technically successful and early postoperative feeding is achieved, the long-term outcome remains guarded. The postoperative period is frequently complicated by wound breakdown, infection, electrolyte disturbances, and feeding difficulties. Skin trauma during anesthesia, intubation, vascular access, and surgical positioning can lead to widespread blistering and erosions. As a result, meticulous perioperative planning is required, including avoidance of adhesive tapes, careful fixation of tubes, padding of pressure points, and gentle tissue handling. Central venous access is often necessary for nutritional and fluid management, but catheter placement itself carries significant risks in the context of fragile skin and impaired wound healing .

Beyond the gastrointestinal tract and skin, multisystem involvement is common and contributes substantially to morbidity and mortality. Renal and urinary tract anomalies, such as hydronephrosis, dysplastic kidneys, and obstructive uropathy, have been reported with notable frequency. Protein-losing enteropathy may develop due to mucosal fragility within the intestine, leading to chronic diarrhea, hypoalbuminemia, and failure to thrive. Respiratory complications are also prominent, including mucosal blistering of the airway, recurrent aspiration, and severe infections. These complications often interact, producing a cascade of clinical deterioration that is difficult to reverse despite intensive supportive care .

Infectious complications remain a leading cause of death in affected infants. Open skin lesions provide a portal of entry for bacteria, while immune compromise related to malnutrition and chronic inflammation further increases susceptibility. Sepsis may develop rapidly and prove refractory to broad-spectrum antimicrobial therapy. Recurrent pneumonia, whether infectious or aspiration-related, is another frequent terminal event. Even in cases where initial surgical and dermatologic management appears successful, late-onset infections can abruptly worsen the clinical course and lead to fatal outcomes weeks or months after birth .

Diagnostic confirmation relies on a combination of clinical features, imaging, and laboratory evaluation. While the diagnosis of pyloric atresia is usually established radiographically, confirmation of epidermolysis bullosa may involve skin biopsy with ultrastructural or immunofluorescence analysis, as well as molecular genetic testing. In practice, definitive genetic results are often obtained after clinical decisions have already been made, particularly in rapidly progressive cases. Nevertheless, establishing the genetic basis is important for prognostication, family counseling, and future reproductive planning. Prenatal diagnosis may be possible in families with known mutations, allowing informed decision-making and anticipatory perinatal care .

The overall prognosis of epidermolysis bullosa with pyloric atresia remains poor despite advances in neonatal intensive care and surgical techniques. Mortality is highest in the neonatal period, especially among infants with extensive skin involvement, severe mutations, and associated systemic anomalies. A minority of patients survive beyond infancy, and those who do often face chronic medical challenges, including persistent skin disease, nutritional deficiencies, and recurrent infections. Importantly, survival does not necessarily correlate with the success of pyloric surgery alone, emphasizing that the gastrointestinal obstruction is only one component of a broader systemic disorder .

Management therefore requires a coordinated, multidisciplinary approach that balances aggressive supportive care with realistic assessment of prognosis. Surgical correction of pyloric atresia should be accompanied by meticulous dermatologic care, nutritional support, infection surveillance, and careful handling at every stage of treatment. In some cases, early involvement of palliative care services may be appropriate to support families and guide decision-making, particularly when the burden of disease is overwhelming and the likelihood of long-term survival is low. Transparent communication with caregivers about the nature of the condition, expected complications, and potential outcomes is essential throughout the clinical course .

In summary, pyloric atresia associated with epidermolysis bullosa is a devastating congenital syndrome rooted in fundamental defects of epithelial integrity. Its presentation is marked by early gastric outlet obstruction and severe skin fragility, with frequent involvement of multiple organ systems. Although surgical intervention is necessary to relieve pyloric obstruction, it does not address the underlying genetic disease, and survival remains limited by infectious, nutritional, and respiratory complications. Continued recognition of this condition, careful multidisciplinary management, and advances in genetic diagnosis are essential to improving care and supporting affected families, even as the prognosis remains guarded in most cases .

References:

1- Lucky AW, Gorell E. Epidermolysis bullosa with pyloric atresia. In: GeneReviews® [Internet]. Seattle (WA): University of Washington, Seattle; 1993–2025. First published February 22, 2008; updated January 26, 2023.

2- Márquez K, Rodríguez DA, Pérez LA, Duarte M, Zárate LA. Epidermolysis bullosa with pyloric atresia: Report of two cases in consecutive siblings. *Biomédica*. 41(2):201–207, 2021

3- Pan P. Congenital pyloric atresia and epidermolysis bullosa: Report of a rare association. *Journal of Indian Association of Pediatric Surgeons*. 26(4):256–258, 2021

4-Luo C, Yang L, Huang Z, Su Y, Lu Y, Yu D, Zhang M, Wu K. Case report: Epidermolysis bullosa complicated with pyloric atresia and a literature review. *Frontiers in Pediatrics*. 11:1098273, 2023

5- Saleem A, Khan AM, Ahmed M. Pyloric atresia associated with epidermolysis bullosa: A case report. *Journal of Ayub Medical College Abbottabad*. 36(4):838–840, 2024

6- Sakamoto N, Masumoto K, Aoyama T, Shirane K, Homma Y. Pyloric atresia in a neonate with epidermolysis bullosa: A case report. *Clinical Case Reports*. 12(12):e9685, 2024

Tailgut Cysts

Tailgut cysts are rare congenital lesions that arise from remnants of the embryonic hindgut that fail to regress during early development. During normal embryogenesis, the tailgut

appears transiently as the most distal portion of the primitive gut and typically involutes by the sixth week of gestation. When this involution is incomplete, epithelial remnants persist and may later give rise to cystic lesions in the presacral or retrorectal space. These cysts are also referred to as retrorectal cystic hamartomas and represent a small but clinically significant subset of presacral tumors.

The retrorectal space is anatomically complex and relatively inaccessible, bordered anteriorly by the rectum, posteriorly by the sacrum and coccyx, superiorly by the peritoneal reflection, inferiorly by the pelvic floor musculature, and laterally by major vessels, ureters, and neural structures. Lesions arising in this space may remain clinically silent for years due to its capacity to accommodate slow-growing masses. As a result, tailgut cysts are frequently discovered incidentally during imaging performed for unrelated gynecologic, gastrointestinal, or spinal complaints.

Epidemiologically, tailgut cysts show a marked predominance in females and are most often diagnosed in adults between the third and sixth decades of life, although cases have been reported across all age groups, including children. The reasons for the female predominance remain unclear but may relate to increased detection during pelvic imaging or gynecologic evaluation. Despite their congenital origin, presentation in childhood is uncommon, and pediatric cases are particularly prone to misdiagnosis.

Clinical presentation varies widely. Approximately half of affected individuals are asymptomatic at the time of diagnosis. When symptoms occur, they are typically related to mass effect on adjacent structures. Patients may report constipation, tenesmus, pelvic or rectal pain, dysuria, urinary retention, or a sensation of incomplete evacuation. In women, symptoms may fluctuate with hormonal changes or be confused with gynecologic conditions such as endometriosis. In some cases, pain worsens with prolonged sitting or physical activity, reflecting pressure on sacral nerve roots.

Complications can arise when cysts become infected, rupture, or bleed. Infected tailgut cysts may present as recurrent perianal abscesses, fistulas, or chronic inflammatory masses, often leading to delayed diagnosis and repeated ineffective interventions. One of the most clinically significant concerns associated with tailgut cysts is their potential for malignant transformation. Although historically considered rare, malignant degeneration has been increasingly reported, with transformation into adenocarcinoma, neuroendocrine tumors, or squamous cell carcinoma. This oncologic risk underpins the consensus that complete surgical excision is indicated even in asymptomatic patients.

Radiologic imaging plays a central role in diagnosis and preoperative planning. Magnetic resonance imaging is generally considered the modality of choice due to its superior soft tissue contrast and ability to delineate the relationship between the cyst and surrounding pelvic structures. Tailgut cysts typically appear as well-defined, multiloculated cystic lesions with variable signal intensity depending on their content. High signal intensity on T1-weighted images may reflect mucinous or protein-rich material, while T2-weighted images often demonstrate a hyperintense, multicystic pattern. MRI is particularly valuable in

assessing extension above or below the levator ani muscle, involvement of the sacrum or coccyx, and features suggestive of malignancy, such as irregular walls, solid components, or enhancement after contrast administration.

Computed tomography can also be useful, especially when MRI is unavailable, but it is less specific in characterizing cyst contents and soft tissue planes. Ultrasonography may detect cystic masses but is limited in deep pelvic evaluation. Preoperative biopsy is generally discouraged due to the risk of infection, tumor seeding, and limited diagnostic yield, as definitive diagnosis relies on histopathological examination of the resected specimen.

Histologically, tailgut cysts are characterized by a heterogeneous epithelial lining that may include stratified squamous, columnar, transitional, or ciliated epithelium, sometimes within the same lesion. The cyst wall may contain fibrous tissue and smooth muscle but lacks the organized muscular layers and neural plexuses seen in true duplication cysts. This histologic diversity reflects the embryologic origin of the lesion and helps distinguish tailgut cysts from other presacral entities such as dermoid cysts, epidermoid cysts, teratomas, anterior meningoceles, and rectal duplications.

The definitive treatment of tailgut cysts is complete surgical excision with clear margins. The choice of surgical approach depends primarily on the size and location of the lesion, its relationship to the pelvic floor, and suspected involvement of adjacent structures. Lesions located above the level of the levator ani or sacral vertebrae are commonly approached from an anterior, transabdominal route, while those located lower in the presacral or retroanal space may be more accessible via posterior approaches such as the transsacral or parasacrococcygeal route. In selected cases, a combined anterior and posterior approach is required, particularly for large lesions, extensive adhesions, or suspected bony involvement.

Advances in minimally invasive surgery have significantly influenced the management of tailgut cysts. Laparoscopic and robotic techniques allow enhanced visualization, precise dissection in confined pelvic spaces, and improved preservation of nerves and vascular structures. Robotic-assisted surgery, in particular, offers technical advantages such as three-dimensional visualization, articulated instruments, tremor filtration, and improved ergonomics, which are especially valuable in the narrow presacral space. These techniques have been associated with reduced blood loss, shorter hospital stays, and faster recovery compared to traditional open surgery, albeit with longer operative times in some cases.

Despite these advantages, surgical resection of tailgut cysts remains technically demanding. Dense adhesions to the rectum, pelvic floor muscles, or sacrum may be encountered, especially in cases with prior infection or inflammation. Intraoperative cyst rupture can occur and should be managed with immediate evacuation and irrigation to minimize contamination. Injury to the rectal wall, although uncommon, is a recognized risk and requires prompt repair. In selected cases, partial or complete coccygectomy may be necessary to achieve complete excision and reduce recurrence risk.

Postoperative outcomes are generally favorable when complete resection is achieved. Recurrence is rare but may occur following incomplete excision or cyst rupture. Long-term follow-up with clinical evaluation and periodic imaging is advisable, particularly in cases with atypical histologic features or difficult dissections. When malignant transformation is identified, management must be individualized and may involve additional surgery, chemotherapy, or radiotherapy depending on tumor type and stage.

One of the ongoing challenges in the management of tailgut cysts is diagnostic delay. Nonspecific symptoms, rarity of the condition, and overlap with more common pelvic pathologies contribute to misdiagnosis and prolonged patient morbidity. Increased awareness among surgeons, radiologists, and clinicians is essential to ensure timely identification and appropriate referral. A high index of suspicion should be maintained when evaluating cystic lesions in the presacral space, particularly in middle-aged women with unexplained pelvic or rectal symptoms.

In summary, tailgut cysts are uncommon congenital lesions with variable clinical presentation and significant potential for complications, including malignant transformation. Accurate diagnosis relies on high-quality imaging, while definitive management requires complete surgical excision tailored to the lesion's anatomy. Advances in minimally invasive and robotic surgery have expanded the therapeutic options available and improved perioperative outcomes. Given the complexity of the presacral space and the rarity of these lesions, optimal management depends on careful preoperative planning, detailed knowledge of pelvic anatomy, and meticulous surgical technique. Continued recognition of tailgut cysts as a distinct clinical entity is essential to prevent delayed treatment and to ensure favorable long-term outcomes.

References:

1- Rompen IF, Scheiwiller A, Winiger A, Metzger J, Gass JM: Robotic-Assisted Laparoscopic Resection of Tailgut Cysts. *JSLS*. 25(3):e2021.00035, 2021

2- Solís-Peña A, Ngu LWS, Kraft Carré M, Gomez Jurado MJ, Vallribera Valls F, Pellino G, Espin-Basany E: Robotic abdominal resection of tailgut cysts – A technical note with step-by-step description. *Colorectal Disease*. 24(6):793–796, 2022

3- Haval S, Dwivedi D, Nichkaode P: Presacral tailgut cyst. *Annals of African Medicine*. 23(2):237–241, 2024

4- Shukla R, Patel JD, Chandna SB, Parikh U: Tailgut cyst in a child: A case report and review of literature. *African Journal of Paediatric Surgery*. ;21(3):184–187, 2024

5- Wojciechowski J, Skołozdrzy T, Wojtasik P, Romanowski M: Two cases of symptomatic tailgut cysts. *Journal of Clinical Medicine*. 13(17):5136, 2024

6- Abatli S, AlHabil Y, Hamad MS, Abulibdeh Y: Mature cystic teratoma mimicking a tailgut cyst in an adolescent female: A case report. *Journal of Surgical Case Reports*. (11):rjae719, 2024

Blunt Cerebrovascular Injuries

Blunt cerebrovascular injury represents one of the most elusive and potentially devastating consequences of pediatric trauma. Although relatively infrequent when compared with other traumatic injuries, its clinical importance lies in the disproportionate risk of ischemic stroke, long-term neurologic impairment, and mortality. The challenge in pediatric populations is amplified by anatomical, physiological, and developmental factors that obscure early recognition and complicate diagnostic decision-making. As a result, blunt cerebrovascular

injury remains both underdiagnosed and inconsistently managed, despite growing awareness of its clinical relevance.

Blunt cerebrovascular injury refers to nonpenetrating damage to the carotid or vertebral arteries caused by mechanical forces such as hyperextension, hyperflexion, rotation, or direct blunt impact. These forces may produce intimal tears, intramural hematomas, pseudoaneurysm formation, arterial dissection, or complete vessel occlusion. While these injuries may initially remain clinically silent, they carry a significant risk of delayed ischemic stroke, sometimes occurring hours or days after the inciting trauma. This delayed presentation contributes to diagnostic uncertainty and underscores the importance of early identification in at-risk patients.

In children, the incidence of blunt cerebrovascular injury has historically been reported as low, often below one percent of all blunt trauma admissions. However, increasing evidence suggests that this figure may reflect underdiagnosis rather than true rarity. Pediatric patients are less likely to undergo vascular imaging, in part due to concerns about radiation exposure and the absence of validated pediatric screening criteria. As imaging practices evolve and awareness increases, reported incidence rates have risen, with some contemporary cohorts identifying rates approaching or exceeding one percent when systematic screening is applied.

Several anatomical and biomechanical characteristics unique to children influence both injury patterns and detection. A proportionally larger head, weaker cervical musculature, greater ligamentous laxity, and increased elasticity of vascular structures contribute to distinctive injury mechanisms. These features may paradoxically offer some protection against vessel rupture while simultaneously predisposing to stretching and intimal damage. The result is a spectrum of vascular injury that may not produce immediate neurologic signs yet carries a substantial risk for delayed ischemic events.

Motor vehicle collisions remain the most common mechanism associated with pediatric blunt cerebrovascular injury. Within this context, restraint use plays a nuanced role. Proper restraint has been shown to reduce overall injury severity and may lower the risk of vascular injury in younger children. Conversely, improper restraint or high-energy mechanisms can transmit rotational and shearing forces to the cervical vasculature, increasing injury risk. Notably, while cervical seatbelt signs have historically been viewed as red flags, their predictive value for vascular injury in children appears inconsistent, and their absence does not exclude significant pathology.

Beyond mechanism of injury, several anatomical and clinical features have emerged as important predictors. Cervical spine fractures, particularly those involving the upper cervical segments, are among the strongest associated factors. Basilar skull fractures, facial fractures—especially Le Fort–type patterns—and intracranial hemorrhage also demonstrate strong associations. Depressed Glasgow Coma Scale scores and higher overall injury severity scores further increase suspicion. Conversely, isolated soft tissue injuries of the neck, once considered highly suggestive, have shown limited predictive value in pediatric

populations.

Despite these associations, no single clinical feature reliably predicts blunt cerebrovascular injury. This has led to the development of screening algorithms intended to identify high-risk patients. Many of these tools were initially developed in adult populations and later extrapolated to children. Unfortunately, when applied to pediatric cohorts, these adult-derived criteria demonstrate limited sensitivity. In some analyses, commonly used screening frameworks identify only a small fraction of affected children, missing a substantial number of cases that ultimately develop cerebrovascular complications.

More recent pediatric-focused screening models have attempted to improve sensitivity by incorporating age-specific injury patterns and mechanisms. When applied consistently, these approaches have increased detection rates, but at the cost of increased imaging utilization. This trade-off highlights the ongoing tension between minimizing radiation exposure and preventing devastating neurologic outcomes. Importantly, studies implementing structured screening protocols have demonstrated higher detection rates than historical controls, suggesting that underdiagnosis remains a central concern.

Imaging modality selection remains another critical consideration. Computed tomographic angiography has become the primary diagnostic tool due to its availability and rapid acquisition. However, its sensitivity in detecting subtle intimal injuries is imperfect, particularly in children. While specificity is generally high, false-negative results still occur. Digital subtraction angiography remains the gold standard but is invasive and rarely used as a first-line modality in pediatric trauma. Magnetic resonance angiography offers a radiation-free alternative, although its availability and feasibility in acute settings are limited. Consequently, clinical judgment continues to play a decisive role in determining when imaging is warranted.

Once identified, management strategies for blunt cerebrovascular injury in children largely mirror those used in adults, despite the lack of pediatric-specific outcome data. Antithrombotic therapy—either antiplatelet agents or anticoagulation—constitutes the cornerstone of treatment for most low- to moderate-grade injuries. Surgical or endovascular interventions are reserved for select cases involving high-grade lesions, progressive neurologic deficits, or failure of medical therapy. Observation alone may be appropriate in select low-risk cases, particularly when bleeding risk or concomitant injuries limit pharmacologic intervention.

Outcomes in pediatric patients appear comparable to those observed in adults when injuries are identified and treated promptly. Stroke remains the most feared complication and may occur even after diagnosis and initiation of therapy, although its incidence decreases significantly with early recognition. Reported stroke rates vary across studies, reflecting differences in screening intensity, diagnostic thresholds, and follow-up practices. Importantly, pediatric patients often demonstrate favorable neurological recovery compared with adults, potentially reflecting greater neuroplasticity.

Despite these advances, management remains inconsistent across institutions. Treatment strategies vary widely with respect to medication choice, duration of therapy, and follow-up imaging. Some children discontinue antithrombotic therapy prematurely, while others remain on prolonged treatment without clear evidence-based guidance. These inconsistencies underscore the need for standardized pediatric-specific protocols informed by prospective, multicenter data.

Comparative analyses between pediatric and adult populations reveal both similarities and distinctions. Injury mechanisms and vascular territories involved are broadly comparable, yet children tend to present with higher injury severity scores and more frequent carotid involvement, whereas vertebral artery injuries appear more common in adults. Despite these differences, overall outcomes—including stroke rates and mortality—are largely similar when comparable management strategies are applied. This suggests that adult-derived treatment frameworks may be pragmatically applied to children, though they are not ideal substitutes for pediatric-specific guidelines.

In summary, blunt cerebrovascular injury in children represents a complex and often underrecognized consequence of blunt trauma. Its detection is hindered by subtle clinical presentation, variable risk factors, and limitations of existing screening tools. Recognition of high-risk mechanisms and injury patterns, combined with judicious use of imaging and timely therapeutic intervention, can significantly mitigate the risk of catastrophic neurologic outcomes. Continued research and collaborative efforts are essential to refine screening strategies, optimize management, and ultimately improve outcomes for this vulnerable population.

References:

1- Farzaneh CA, Schomberg J, Sullivan BG, Guner YS, Nance ML, Gibbs D, Yu PT: Development and validation of machine learning models for the prediction of blunt cerebrovascular injury in children. *Journal of Pediatric Surgery*. 57(4):732–738, 2022

2- El Tawil C, Nemeth J, Al Sawafi M: Pediatric blunt cerebrovascular injuries: Approach and management. *Pediatric Emergency Care*. 40(4):319–322, 2024

3- Nickoles TA, Lewit RA, Notrica DM, Ryan M, Johnson J, Maxson RT, Naiditch JA, Lawson KA, Temkit M, Padilla B, Eubanks JW III: Lower incidence of blunt cerebrovascular injury among young, properly restrained children: An ATOMAC multicenter study. *Journal of Trauma and Acute Care Surgery*. 95(3):334–340, 2023

4- Schulz M, Weihing V, Shah MN, Cox CS Jr, Ugalde I: Risk factors for blunt cerebrovascular injury in the pediatric patient: A systematic review.
American Journal of Emergency Medicine. 71:37–46, 2023

5- Lewit RA, Nickoles TA, Williams R, Notrica DM, Stottlemyre RL, Ryan M, Johnson JJ, Naiditch JA, Lawson KA, Maxson RT, Grimes S, Eubanks JW III: Blunt cerebrovascular injury in children: A prospective multicenter ATOMAC+ study. *Journal of Trauma and Acute Care Surgery*. 99(2):245–252, 2025

6- Asaadi S, Rosenthal MG, Radulescu A, Mukherjee K, Luo-Owen X, Dubose JJ, Tabrizi MB; AAST PROOVIT Study Group: Pediatric versus adult blunt cerebrovascular injuries: Patient characteristics, management, and outcomes. *Annals of Vascular Surgery*. 116:1–8, 2025

***Edited by: Humberto Lugo-Vicente, MD, FACS, FAAP**
Professor of Pediatric Surgery, UPR - School of Medicine, UCC School of Medicine & Ponce School of Medicine.

Pediatric Surgery, San Jorge Hospital.
Postal Address: P.O. Box 10426, San Juan, Puerto Rico USA 00922-0426.
Tel (787) 340-1868 E-mail: *pediatricsurgerypr@gmail.net*
Internet: pedsurgeryupdate.com

ISSN 1089-7739