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Epigastric Artery Flap Extremity Reconstruction

Through and through traumatic defects in the hands and feet of children produced by high-energy penetrating injuries are considered difficult surgical problems requiring complex reconstruction of affected bone, nerves, tendon and associated vascular structures. After a series of debridement procedures surgeons need to provide skeletal fixation and stable coverage. Form, function and safety of each reconstructive option should be considered carefully and weighted against each other when considering a reconstructive plan. Free flap transfer has become the preferred treatment option for reconstruction of the damaged extremities. The rectus abdominis muscle flap and free latissimus dorsi flap with their sizable areas and large vascular pedicles are the most commonly performed. Disadvantage of using this flap is the sacrifice of important muscles that may lead to functional deficit and potential donor site morbidity. The deep inferior epigastric artery perforator (DIEP) flap has been used extensively in breast reconstruction. It provides a huge amount of skin and soft tissue coverage with minimal donor morbidity. The DIEP flap has been utilized in extremity reconstruction, including foot and ankle, thumb reconstruction and repair of massive lower limb soft tissued defects. Advantages of the DIEP flap include: no need to sacrifice the abdominal musculature, provides a longer pedicle allowing tension free anastomoses and has a reliable and safe vascular supply. The free DIEP flap is suitable for any type of head, neck and extremity defect. The dissection of perforator's vessel is the key to achieve the successful free DIEP flap transfer in children. The US Doppler is still the most effective and economic method to locate the perforators. The overall survival rate of the flap is 96%. The venous outflow is easier to be compromised because of slower flow and thin vessels wall. Disadvantage includes fat hypertrophy and the scar left in the abdominal wall.

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Recurrent Achalasia after Esophagomyotomy

Achalasia is the most common motility disorder of the esophagus causing dysphagia due to loss of primary esophageal peristalsis and impaired relaxation of the lower esophageal sphincter (LES). Balloon dilatation and Botox injection are considered short-lived method of managing achalasia. Effective long-term management of achalasia results with laparoscopic Heller esophagomyotomy and Dor partial fundoplication. Factors known before the procedure such as patient characteristic, degree of esophageal dilatation or tortuosity, manometry findings and prior treatments have little effect on long-term outcome. Common causes of surgical failure are gastroesophageal reflux and recurrent dysphagia. When dysphagia occurs after myotomy is more often recurrent than persistent. Changes in the esophagus and/or LES that develops after the operation is more important in defining recurrence of symptoms. Almost 25% of all patients still experience dysphagia once per week after surgery. It is believed obstructive scar tissue or distortion of the myotomy develop in theses cases. The only predictor of the need for postop dilation is history of preop dilation. Specific causes of recurrent achalasia after surgery include: 1- Incomplete myotomy or scarring of the distal edge of the myotomy. Longer and more separated myotomy reduces this problem; 2- Not performing a fundoplication. This causes abnormal symptoms of reflux and heartburn. A 360-degree fundoplication aggravates dysphagia. Partial fundoplication prevents reflux and does not impair esophageal emptying; 3- GE reflux is considered a common cause of recurrent dysphagia due to esophagitis, scarring and development of Barrett's esophagus; 4- the effects of previous treatment due to scar tissue created from endoscopic manipulation or Botox injections; 5- Esophageal cancer since achalasia patients are at increased risk of developing squamous cell carcinoma. Diagnostic evaluation of recurrent symptoms must include: barium swallow, upper endoscopy and manometry. Management of recurrent achalasia include pneumatic balloon dilatation, revisional surgery, POEM or esophagectomy.

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Nontuberculous Mycobacteria Lymphadenitis

Atypical mycobacteria, also known as nontuberculous mycobacteria (NTM) are acid-fast bacteria other than Mycobacterium tuberculosis. Nontuberculous mycobacteria can cause difficult to diagnosed lymphadenitis in immunocompetent children. Exposure of the human oral cavity and respiratory tract to NTM comes from soil, specially after putting wet dirt or soil into their mouths. Incubation periods are variable but can reach five years in some cases. A diagnosis of NTM lymphadenitis should be suspected in children less than five years of age, female predominance, with subacute, unilateral, non-tender cervicofacial lymphadenitis resistant to standard antibiotic therapy. Submandibular and anterior cervical lymph are most commonly involved. Diagnosis is established by acid-fast staining, mycobacterial culture and histopathology. Sampling method for diagnosis includes FNA aspiration, curettage, drainage or complete excision. Polymerase-chain reaction testing of lymph node material has the highest diagnostic yield, followed by mycobacterial culture and microscopy for acid-fast bacilli. Positive culture will confirm the diagnosis but it can take six weeks. Growth characteristic of NTM lymphadenitis include slow-growing (M Fortuitum, Chelonei and Abscessus) and fast-growing mycobacteria (M. Marinum, Kansasii, Avium-intracellulare). In the US the majority of NTM lymphadenitis in children are caused by M Avium complex. A PPD skin test might be positive. The course of NTM lymphadenitis might be variable and involve eruption of the lymph node and tract formation with drainage; the lymph node might also remain indurated. Systemic symptoms are unusual in immunocompetent children. Reactivation of NTM can occur after trauma or injury near the affected area. Management of NTM lymphadenitis includes surgical (complete resection is gold standard if technically feasible), less likely prolonged antimycobacterial oral therapy (Clarithromycin is preferred).

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