

Operative pediatric surgery. Achalasia in children

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Annotation. Achalasia is a rare disease that makes it hard to swallow foods and liquids. In achalasia, there is a problem with the tube that carries food from the mouth to the stomach (esophagus). The muscles that make the esophagus contract and push food down to the stomach don't work well. The muscle contractions get weak. The LES (lower esophageal sphincter) also doesn't work well. The LES is the muscle at the bottom of the esophagus, where it joins the stomach. With achalasia, the LES gets narrow and tight. Food does not pass into the stomach as it should. Over time, food and liquid can collect in the esophagus. Achalasia happens more often in adults. It is rare in children. It affects males and females equally. In some cases, there may be a family history of achalasia.

Key words: achalasia, sphincter, cardiomyotomy, antireflux, nasogastric, gastrohepatic, phrenoesophageal

Open achalasia : Lewis Spitz

History. Achalasia was first described by Willis in 1672. He treated the patient by fashioning a rod out of a whale bone with a sponge on the end with which the patient was able to force food into his stomach. In 1877, Zenker and von Ziemssen, and in 1884 Mackenzie, suggested that achalasia was due to diminished contractile power of the esophageal musculature. In 1888, Meltzer and Mikulicz independently postulated that spasmodic contraction of the cardiac sphincter was the etiologic factor. In the same year, Einhorn proposed that the condition was due to failure of relaxation of the cardia on swallowing

Principles and justification. Achalasia is a motility disorder of the esophagus characterized by an absence of peristalsis and a failure of relaxation of the lower esophageal sphincter. The cardinal symptoms in childhood are vomiting, dysphagia, chest pains and recurrent respiratory infections, and weight loss. The child learns to eat very slowly and to drink large quantities of fluid to encourage food to enter the stomach. At first, there is only regurgitation of food, but later vomiting of undigested food eaten days earlier occurs. The child with achalasia is often first referred to a psychiatrist for treatment of food aversion or anorexia

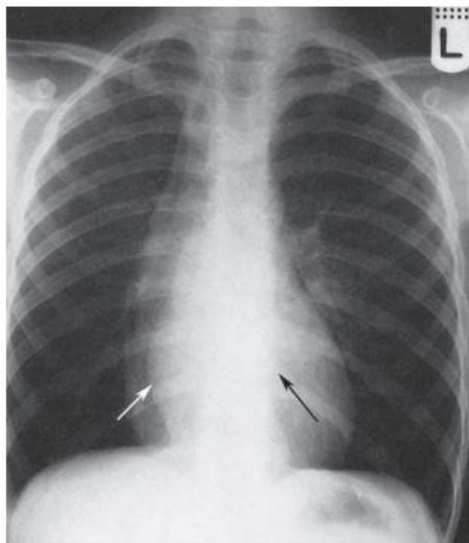
Histopathology. Strips of muscle from the distal esophagus reveal varying pathologies from complete absence of ganglion cells to chronic inflammatory changes through to normal ganglia.

Histochemistry reveals a significant reduction in all neuropeptides, particularly vasoactive intestinal polypeptide, galanin, and neuropeptide Y. Treatment Medical treatment Transient relief of symptoms can be achieved with nifedipine, a calcium antagonist that reduces the pressure at the lower esophageal sphincter. Forceful dilatation The aim of this treatment is to physically disrupt the muscle fibers of the lower esophageal sphincter by means of pneumatic or balloon dilatation. A fluid-filled (Plummer) or air-filled (Browne–McHardy, Rider–Moller, angioplasty catheter) bag of fixed diameter, or the balloon dilator, is radiologically positioned in the distal esophagus and gently inflated. Relief of symptoms in children is at best temporary, but may occasionally last for prolonged periods. Recently, it has been shown that botulinum toxin injected into the lower esophageal sphincter musculature results in symptomatic relief, but the effect is short lived.

Surgical treatment. The basis of all surgical procedures is the cardiomyotomy described in 1914 by Heller. Controversies concern the length of the myotomy, the extent to which the myotomy extends onto the stomach, and the necessity for an antireflux procedure. The principle of the procedure is to perform a myotomy over the distal 4–6 cm of esophagus, extending the incision for 1 cm onto the anterior wall of the stomach. The myotomy is covered by a short, floppy Nissen fundoplication to protect against subsequent gastroesophageal reflux.

Preoperative Diagnosis. Radiologic features .

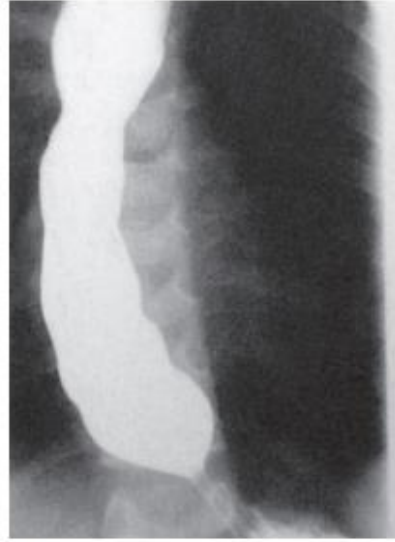
1 A plain chest x-ray may show a dilated, food-filled esophagus with an air–fluid level. There may be radiologic signs of recurrent aspiration pneumonitis.



1

2 The diagnostic features of achalasia on barium swallow are a dilated esophagus, absence of stripping waves, incoordinated contraction, and obstruction at the gastroesophageal junction with

prolonged retention of barium in the esophagus. Failure of relaxation of the lower esophageal sphincter gives rise to the classical ‘rattail’ deformity of funneling and narrowing of the distal esophagus.

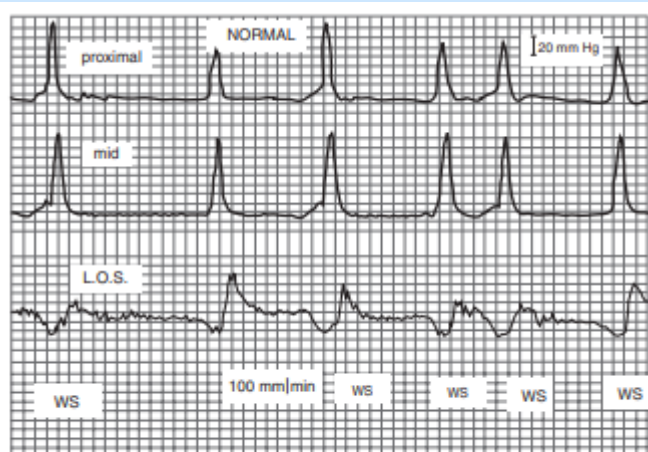


2

Endoscopy. The main value of esophagoscopy is to exclude an organic cause for the obstruction.

Esophageal manometry

3a,b The criteria for diagnosis include: (1) a high-pressure (>30 mmHg) lower esophageal sphincter zone; (2) failure of the lower esophagus to relax in response to swallowing; (3) absence of propulsive peristalsis; and (4) incoordinated tertiary contractions in the body of the esophagus.



3a

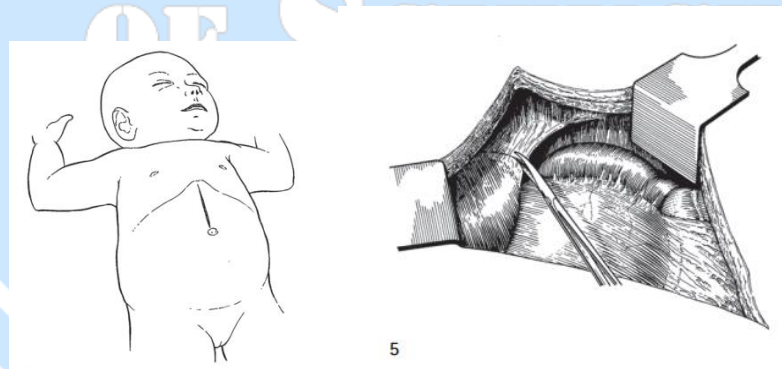


3b

Anesthesia . General endotracheal anesthesia is administered, with the patient supine on the operating table. Measures must be taken to avoid aspiration of esophageal contents during the induction of anesthesia. Preoperative esophagoscopy is recommended to ensure complete evacuation of retained food and secretions from the esophagus. A medium-caliber nasogastric tube is passed into the stomach.

Operation

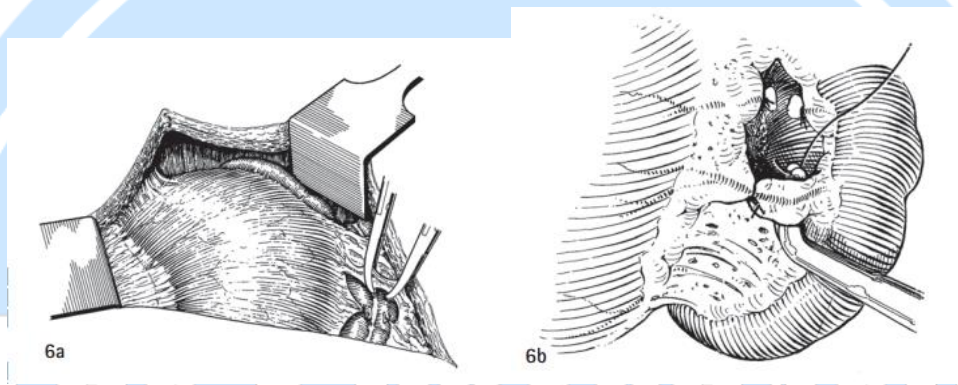
Incision . The approach is via an upper abdominal midline incision extending from the xiphisternum to the umbilicus.(picture 5)



Exposure. In most cases, adequate exposure of the abdominal esophagus can be obtained by retracting the left lobe of the liver anterosuperiorly with a wide retractor. If necessary, additional exposure may be attained by dividing the left triangular ligament in the avascular plane and retracting the left lobe of the liver towards the midline.

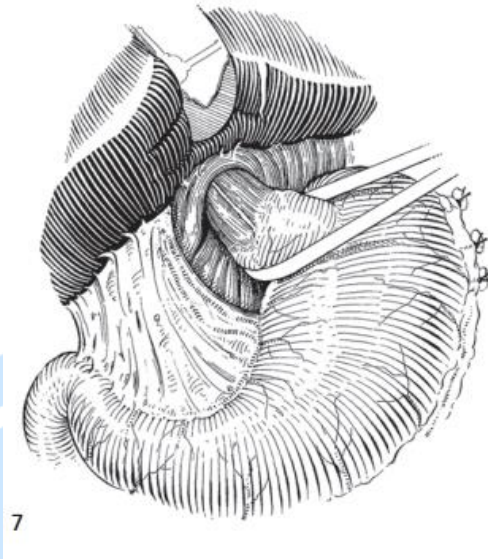
Mobilization of fundus of stomach . 6a,b As a Nissen fundoplication will be performed in addition to the extended gastroesophageal myotomy, the operative procedure for fundoplication should be

followed at an early stage. The proximal one-third of the greater curvature of the stomach is liberated from its attachment to the spleen by ligating or coagulating with bipolar diathermy and dividing the short gastric vessels in the gastrosplenic ligament. This is accomplished most safely using a right-angled forceps passed around each vessel in turn. When the vessels in the upper part of the gastrosplenic ligament have been divided, the spleen should be allowed to fall back into the posterior peritoneum, thereby avoiding inadvertent trauma. Splenectomy should never be necessary in this procedure. The fundus is now sufficiently free to allow for a loose (floppy) fundoplication. The esophageal hiatus is completely exposed by dividing the upper part of the gastrohepatic omentum above the left gastric vessels.

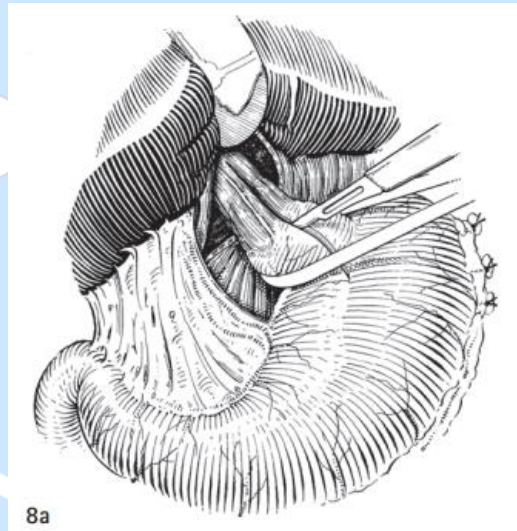


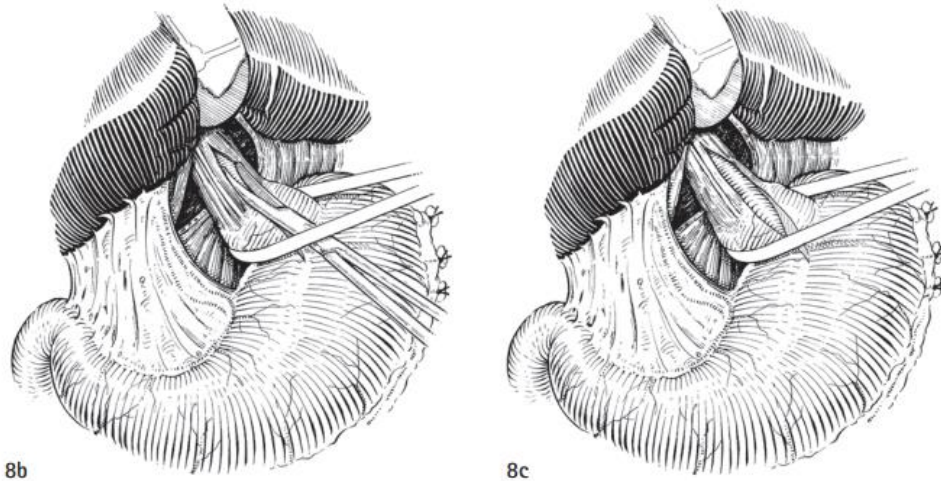
Exposure of esophageal hiatus. The phrenoesophageal membrane is placed on stretch by downward traction on the stomach while the diaphragmatic muscles are retracted superiorly. The avascular membrane is incised with scissors and the musculature of the esophagus displayed. The anterior vagal nerve will be seen coursing on the surface of the esophagus; it should be carefully protected and preserved.

Mobilization of the distal esophagus. Using a combination of sharp and blunt dissection, the lower end of the esophagus is encircled, taking care not to injure the posterior vagal nerve. A rubber sling is placed around the esophagus. The lower 5–8 cm of esophagus is now exposed through the esophageal hiatus into the posterior mediastinum using blunt dissection with either a moist pledget or right-angled forceps. (picture 7)

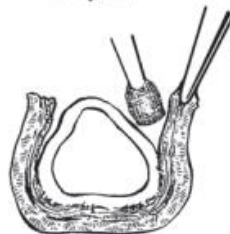
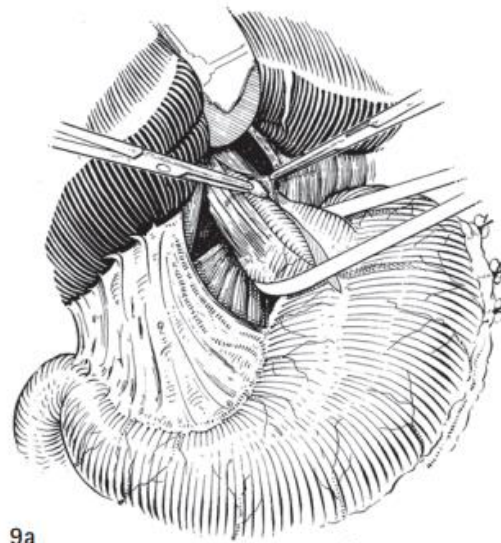


Gastroesophageal myotomy. 8a–c The myotomy is performed on the anterior wall of the esophagus, extending for 1 cm onto the fundus of the stomach. A superficial incision (1–2 mm in depth) is made in the musculature of the distal 4–6 cm of the esophagus. The divided muscle is gently parted with a blunt hemostat until the underlying mucosa of the esophagus is encountered. The thickness of the muscle of the lower esophagus varies from a few millimeters to 0.5 cm or more. Great care must be taken to avoid opening into the lumen of the esophagus

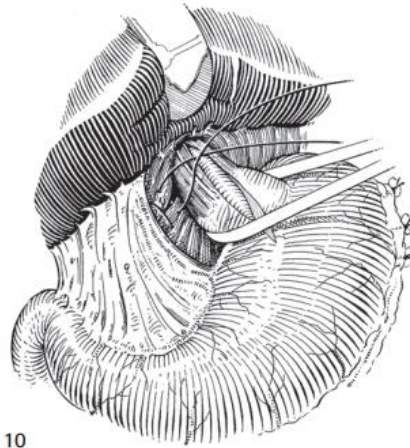




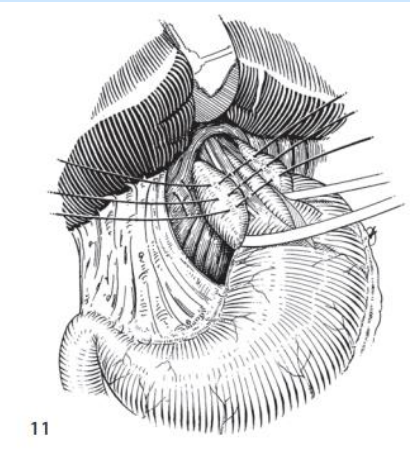
9a,b The divided muscle is now separated from the underlying mucosa by blunt pledget dissection in the submucosal plane. The dissection is continued until at least 50 percent of the circumference of the esophagus is free of the overlying muscle. The myotomy is extended through the gastroesophageal junction for 1 cm onto the fundus of the stomach and the musculature is similarly elevated from the underlying mucosa.



Testing for esophageal perforation. The stomach and esophagus are distended with air introduced through the nasogastric tube, and the exposed mucosa is carefully inspected for perforation. A mucosal defect should be carefully closed with fine polyglycolic acid sutures.



Narrowing of hiatus 10 . The esophageal hiatus is narrowed posteriorly to the esophagus by placing deep sutures through the crura of the diaphragm. The sutures are tied loosely to prevent them from cutting through, leaving sufficient space alongside the esophagus to allow passage of the tip of a finger. Two or three sutures may be required for this purpose.



Fundoplication 11. A loose (floppy) Nissen fundoplication is now constructed over the distal 1–1.5 cm of the esophagus. The esophageal sutures are only placed through one side of the divided esophageal muscle in order to prevent reapproximation of the edges of the myotomy

Wound closure . The wound is closed either in layers or with interrupted en masse sutures of 3/0 polyglycolic acid. A subcuticular suture approximates the skin edges.

Postoperative care . Nasogastric decompression and intravenous fluids are continued until the postoperative ileus has resolved (mean of 3–4 days).

Complications. These can include mediastinitis due to failure to detect a mucosal perforation, and recurrence of symptoms if the muscle is not separated from the underlying mucosa for at least half

the circumference of the esophagus. Gastroesophageal reflux is due to an inadequate fundoplication, and dysphagia for solids is due to too tight a fundoplication.

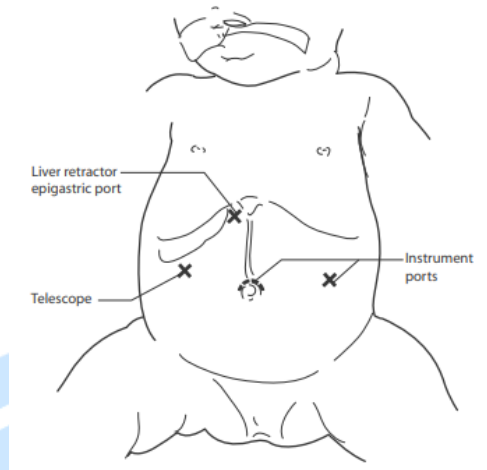
Outcome. After myotomy alone without an antireflux procedure, the long-term incidence of gastroesophageal reflux is around 15 percent. Relief of the dysphagia and respiratory problems is usually complete, but residual or recurrent pain may occur in 25 percent of patients and is due to diffuse esophageal spasm. The esophageal pain generally responds to pneumatic dilatation

Laparoscopic achalasia

Preoperative preparation of the patient and anesthesia . Neither bowel preparation nor placement of a Foley catheter into the bladder is required. General anesthesia with full relaxation and endotracheal intubation is mandatory. It is recommended to perform esophageal endoscopy and clear the esophagus from retained food after induction of anesthesia. Thereafter, a medium size stiff nasogastric tube is inserted and left until the end of operation to fixate the esophagus and to ensure that the stomach is empty for the duration of the operation. Single shot intravenous perioperative antibiotic prophylaxis is given with induction of anesthesia.

Patient and team positioning . The patient is placed in a supine position at the lower end of the operation table. The operation is performed by the surgeon who stands between the legs of the patient and the camera assistant placed to his left. The scrub nurse is to the right hand of the surgeon. The laparoscopic tower including the video monitor is placed at the head or the left head of the table. The operation table is set in a reversed Trendelenburg position.

Port placements . A four-port technique is used. A 5- or 10-mm port for the telescope is inserted through or below the umbilicus with an open technique. Carbon dioxide is insufflated at a pressure of 8–10 mmHg. Two 3.5- or 5-mm instrument ports are inserted in the right and left mid-upper abdomen (one in each upper quadrant). A fourth trocar for the liver retractor may be introduced below the subcostal margin to the left of the falciform ligament. The grasper for liver retraction may also be introduced directly without using a trocar. In case of difficulties with exposure, an additional 5-mm port may be introduced above the umbilicus right to the falciform ligament for the telescope. After changing the position of the telescope, an additional instrument may be used via the umbilical port for grasping the stomach and pulling it downward in these cases.



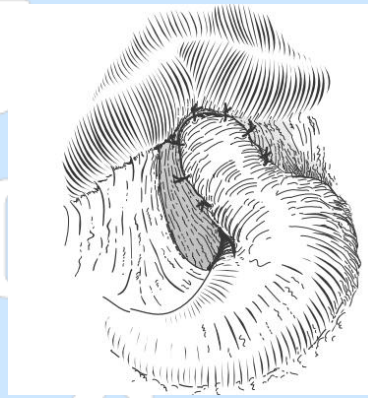
Liver retraction . The left lobe of the liver can be easily retracted upward with a single-toothed ratcheted grasper which is introduced through the subxyphoid incision with or without use of a port. The grasper is fixed to the muscular diaphragm by grasping it just above the hiatus. It can then be left in situ during the duration of the operation.

Exposure of cardia and esophagus . The ‘white line’, which is the edge of the diaphragmatic crura, is easily identifiable by gentle downward traction of the stomach. The phrenoesophageal junction is divided using the monopolar hook and the anterior wall of the esophagus is freed. The dissection includes only the anterior and lateral esophagus and the posterior esophagus is not mobilized to prevent gastroesophageal reflux. The esophagus should be exposed from the crus down to the esophagogastric junction. The anterior vagal nerve should be identified, preserved, and pushed away from the myotomy incision. Thereafter, the anterior crus is lifted away from the esophagus to gain entry into the mediastinal esophagus. An easy plane can be developed between the overarching crus and the esophagus, allowing the esophagus to be exposed in the mediastinum for up to 5 cm. Once an adequate length of intrathoracic esophagus has been exposed, attention should be redirected to the abdominal esophagus.

Esophageal myotomy. An esophageal myotomy is best started with a superficial incision on the anterior wall of the esophagus about 1 cm proximal to the esophagogastric junction. While it is possible to use monopolar diathermy or an ultrasonic scalpel to make this initial myotomy incision, both these instruments can produce deeper thermal damage, which may cause unrecognized damage of the underlying mucosa, resulting in delayed perforation. Blunt curved scissor dissection is used and the longitudinal muscle layers are separated. The mucosa can be clearly seen as it herniates through the myotomy. The muscle layers should be spread further to the mediastinum for up to 5 cm using blunt dissection. Dissection is continued down to the gastric junction extending onto the fundus of the stomach, which can be identified when one sees the edge of

circular gastric muscle fibers. The underlying mucosa should be allowed to pout outwards as much as possible. Bleeding from the esophageal muscle layers usually stops spontaneously and cauterization is unnecessary. The mucosa is inspected for evidence of perforation and if there is concern, air may be instilled into the esophagus and stomach to check for mucosal leak. A mucosal leak can be repaired laparoscopically with an absorbable mucosal suture. Intraoperative endoscopy may facilitate identification of the esophagogastric junction and dynamic manometry may identify the adequate length of myotomy, but the author considers these maneuvers unnecessary.

Fundoplication . A 180° anterior fundoplication is performed to prevent reflux and to protect the mucosa. The anterior gastric fundus is fixated to the anterior esophageal muscle layers with between four and six non-absorbable sutures. The upper sutures are attached to the anterior hiatus to prevent slipping of the wrap. There is no need to divide the short gastric vessels.



Postoperative management . The nasogastric tube is removed immediately after anesthesia. Patients can usually commence feeding with liquids immediately following the operation, with immediate relief of symptoms. A routine contrast study is not required before commencement of feeding. Oral feeding should be completed by day 3. Long-term follow up is essential and clinical assessment is performed at 1, 6, and 12 months postoperatively. Contrast study, pH study, or manometry should only be used in case of persisting or new symptoms.

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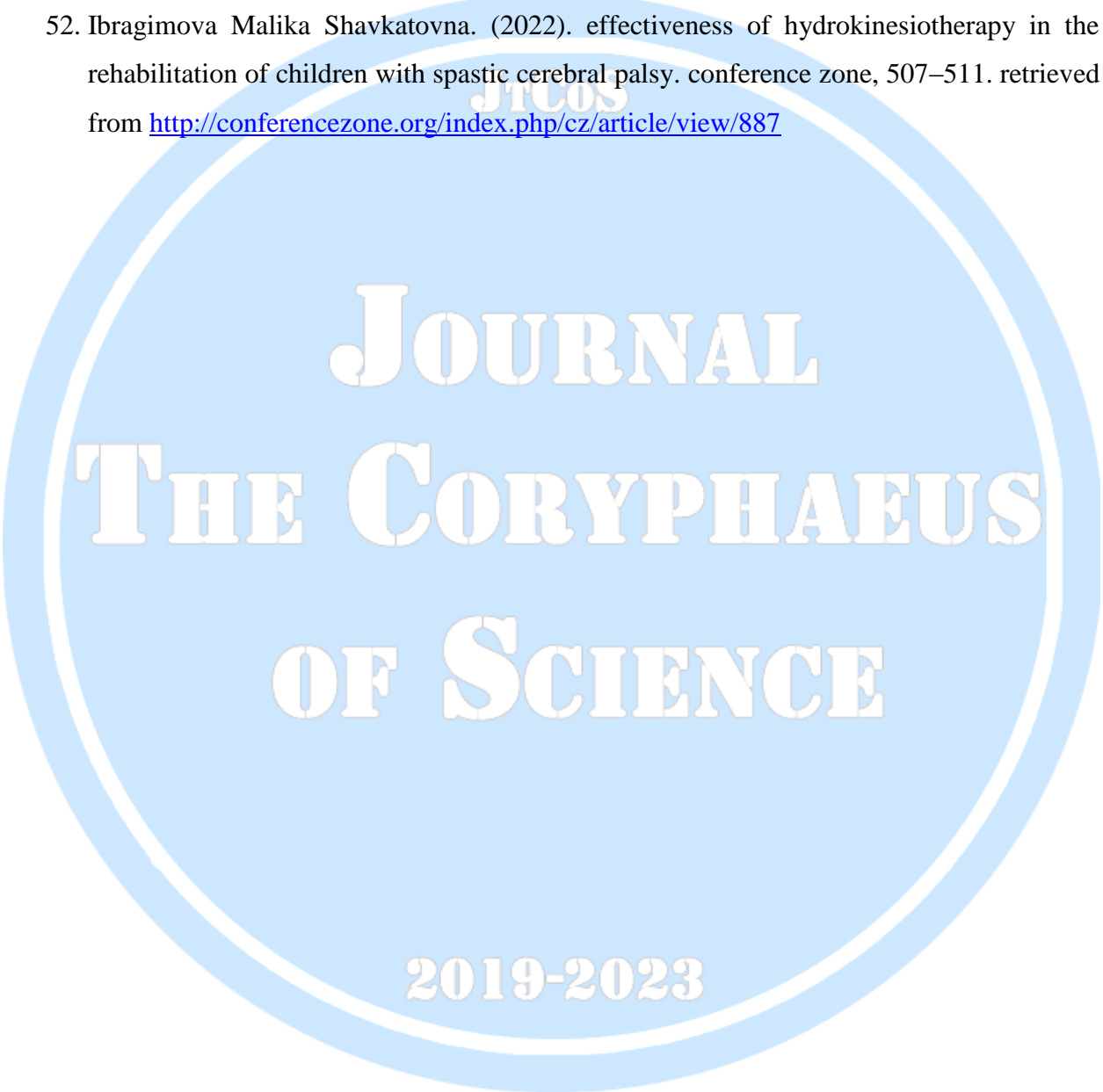
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