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Your patients deserve consistent care from experienced surgeons who are Board Certified in Pediatric Surgery. Regardless of which PSA surgeon happens to be on call, or which one is seeing your patient that day, you will find little variation in expertise among the four of us; we have the most total years of surgical experience, and most importantly, each of us is Board Certified in Pediatric Surgery.

JULY

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
1 David Bliss	2 José Iglesias	3 Glaze Vaughan	4 Tom Black	5 José Iglesias	6 Glaze Vaughan	7 José Iglesias
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15 Tom Black	16 José Iglesias	17 Glaze Vaughan	18 Tom Black	19 José Iglesias	20 Tom Black	21 José Iglesias
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29 David Bliss	30 Tom Black	31 Glaze Vaughan	We are available 24 hours every day. Page us directly to the number listed for direct referrals or for an immediate consultation.			

HYPERTROPHIC PYLORIC STENOSIS

Fewer neonatal conditions are more common but less well-understood than is hypertrophic pyloric stenosis (HPS).

The incidence of HPS is about three in 1000 births with males outnumbering females about 4 to 1 and first-born children more commonly affected. HPS among African-Americans and Asian infants is less common than among Caucasians. The overall incidence seems to be increasing. The cause of HPS is unknown, but women who had HPS

themselves are four times more likely to have an offspring with the condition, while men who had HPS are eight times more likely. Interestingly, infants with blood types B or O are more likely to be affected. Also, infants of mothers who took erythromycin during the latter stages of pregnancy or while nursing seem to have a higher risk. The cause seems most likely to be multifactorial.

HPS is generally diagnosed at between 3 and 6 weeks

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of life, although the diagnosis has been made even *in utero*. An infant suspected of having HPS may have had a history of 'spitting' since birth with recent worsening. When measured over time, the infant's weight will usually have decreased. Since formula intolerance is frequently suspected by the health care provider, most infants will have had their formula changed several times before the possibility of HPS is ultimately investigated. As HPS develops, the pylorus muscle which surrounds the gastroduodenal junction becomes hypertrophied, firm, and resistant to relaxation. The pyloric channel between stomach and duodenum narrows and gastric emptying decreases but only rarely ceases completely. The increasingly starving child eats avidly, but the only significant gastric emptying is by emesis. This eventually becomes forceful and remarkably 'projectile'. Although the emesis may be dark brown due to a small amount of blood caused by a mild gastritis, it is never bilious as the narrowed pylorus does not allow bile to reflux from the duodenum into the stomach. Peristaltic gastric waves are often seen coursing across the surface of the child's quite distended abdomen. Defecation will generally not have ceased completely, but urine output may well be diminished. Infants with HPS may be mildly jaundiced due to temporary inhibition of the liver enzyme gluconyl transferase. Nursing infants may have a slightly less severe clinical picture since breast milk produces smaller curds than does formula, allowing it to pass through the narrowed pylorus more easily.

An experienced and patient examiner will detect the hypertrophied pylorus, the so-called "olive", as often as 90% of the time, although this ability is becoming a lost art now that objective imaging studies have become so readily available and accurate. The key to a successful examination is a relaxed infant. He must be made comfortable with his knees drawn up toward his chest a bit to relax his abdominal wall musculature. One trick is to place an oro- or nasogastric tube, then allow the child to drink some glucose water, which is aspirated back through the tube as soon as the water enters the stomach. This will generally pacify the hungry child and cause him to stop crying and relax long enough for a thorough examination.

Assuming a diagnosis has not been made clinically, any child less than 3 months of age, regardless of gender or birth order, who has exhibited persistent non-bilious emesis, should be evaluated for HPS. The imaging study of choice at this time is ultrasonography, when available. An upper GI series, which was once the preferred study, although generally diagnostic, may increase the risk of aspiration during the study or during later induction of anesthesia.

Most infants with HPS will have had a significant amount of persistent emesis and will consequently display some degree of dehydration at diagnosis. The typical metabolic derangement seen with any gastric outlet obstruction and persistent vomiting is related to the loss of hydrochloric acid and fluid volume. Loss of H^+ ions induces a metabolic alkalosis while loss of Cl^- ions results in hypochloremia. The aldosterone that is released in response to volume depletion causes the kidney to retain Na^+ ions; in doing so, K^+ ions are exchanged and excreted, and the classic picture of

hypochloremic, hypokalemic metabolic alkalosis is complete. The volume deficit, a bicarbonate level above 30, and a chloride level less than 90, when present, must all be corrected prior to surgery. The ideal initial resuscitation regimen is debatable, but normal saline, 10 to 20 ml per Kg over 30 minutes for volume replacement (depending on estimated degree of dehydration) followed by one-half-normal saline with potassium until the child is well hydrated and any abnormal electrolytes have normalized, has proven to be quite satisfactory. This correction, when necessary, should not be completed too rapidly and may take hours or even days.

Attempts at non-surgical management of HPS have not gained wide acceptance. Endoscopic balloon dilation of the pylorus has been reported but is inconsistently successful. Intravenous atropine in slowly increasing dosages followed by maintenance dosing for 2 weeks has a reportedly high success rate, but the length of hospitalization and associated side effects make this option less desirable at the present time than a brief operation and short hospital stay.

The standard method for the surgical correction of HPS consists of pyloromyotomy or division of the pyloric muscle while leaving the mucosa of the stomach intact. Just how the procedure is carried out varies from surgeon to surgeon. The most frequent approach is through a laparotomy, either vertically through the midline or more commonly transversely through a right upper quadrant incision. Many surgeons prefer an umbilical incision because the resulting scar is essentially invisible. A neonate whose umbilical cord has not completely separated or whose navel has not completely healed following separation of the cord may not be an ideal candidate for this incision due to the risk of wound infection. The usual operating time for each of these procedures varies between 10 and 20 minutes. We have recently begun performing laparoscopic pyloromyotomy; this approach features equally excellent results with a minimally shorter operative time and perhaps even less visible scarring.

Recovery from pyloromyotomy is generally prompt and is associated with minimal discomfort. Most infants are begun on oral feedings 4 hours after surgery, but the exact feeding regimen varies significantly from surgeon to surgeon. Despite these differences, most infants will have resumed tolerating expected volumes of formula or a normal nursing regimen within 18 hours, and most infants will be discharged within 24 hours. Breast-fed infants generally return to full feedings sooner than do formula-fed infants, presumably because the smaller curds pass the pylorus more easily. Any associated jaundice should clear rapidly. Persistent "spitting" over the next few weeks is not uncommon, but significant emesis more than two weeks after surgery should prompt a further evaluation for gastroesophageal reflux. The only postoperative side effect that may be expected to occur with relative frequency is an increase in the infant's appetite; this may be partly due to recovery from a period of relative negative nitrogen balance and partly from more rapid gastric emptying due to a temporarily incompetent pyloric muscle. This generally resolves within 3 weeks as the hypertrophied muscle returns to its normal caliber and heals. No other long-term residua are associated with HPS.

Disclaimer: All material is intended for informational purposes only and is not intended, and should not be used, to replace medical advice offered by a qualified physician. We are always available and willing to discuss questionable conditions with you and we invite your request for our assistance.