Treatment.—Septic shock is best treated by *prevention*, i.e. prompt recognition of the presence of sepsis and institution of its proper treatment before the state of shock supervenes. This prevention necessitates:

1. Identification of the source of infection.
2. Administration of antibiotics as specific as possible.
3. Institution of surgical drainage, e.g. drainage of abscess, surgical debridement or removal of septic focus.

Once shock has set in, an early manifestation of which is increased fluid requirement to maintain the urine output, very rapid action has to be undertaken to save the patient. This consists of:

A. Treatment for the sepsis.

B. Treatment for the shock.

C. Other supportive measures.

A. TREATMENT FOR THE SEPSIS:

1. Identification of the source.—In the majority of cases the source of infection is evident. If no source is apparent, the cause probably lies in the abdomen—USG, CT or MRI may be helpful to detect localised collections.
2. Antibiotics:
   a. If previous culture-sensitivity tests are available, the specific antibiotic can be started immediately.
   b. In other cases, especially when gastro-intestinal tract organisms are suspected, combination of cefazolin, gentamicin/amikacin and metronidazole usually works well.
3. Surgery.—Drainage of abscess or localised collections, surgical debridement, removal of products of abortion, etc.

B. TREATMENT FOR THE SHOCK.—Essentially the shock is hypovolaemic and the aim is restoration of the blood volume. Prompt correction of the pre-existing fluid deficit is necessary and large quantities of fluid are often needed. However, care must be taken that there is no overloading, because often the lungs are already damaged by the septic process. The types of fluid are:

1. Usually crystalloid solutions, e.g. lactated or acetated Ringer's solution, or isotonic saline buffered with sodium bicarbonate, can effect resuscitation. As much as 10 to 15 litres may be necessary in the first 24 hours.
2. In the absence of specific needs, colloid solutions (5% albumin in isotonic saline or synthetic plasma-expanders like dextran, gelatin or hydroxyethyl starch) are better avoided because of their escape into interstitium of the lungs, resulting from enhanced capillary permeability in septic shock, may be dangerous.
3. Any deficit in RBC count should be corrected by blood transfusion because the damaged lungs must be properly oxygenated.

the extensive fibrosis on the wall of the ulcer. A true pyloric stenosis results from a pyloric ulcer or a pyloric cancer.

There is a typical history of duodenal ulcer, usually for a long period. When stenosis sets in, characteristic clinical features are noted as follows:

1. Periodicity is lost.
2. Pain changes its character. It becomes less intense and loses relationship to food. More distressing is a sensation of fullness in the epigastrium, which is continuous but is exaggerated after taking food.
3. Vomiting becomes a constant feature:
   a. It is projectile.
   b. It is copious.
   c. It is often self-induced.
   d. It usually contains *old food.*
4. Majority of the patients complain of a lump, moving in the upper abdomen, particularly after taking food.
5. Constipation becomes more severe.
6. There is rapid loss of weight due to inanition.

On Examination

1. A cricket ball-size lump is seen moving across the epigastrium, from left to right, at the rate of 3 to 4 per minute. It is particularly noticed after the patient is asked to drink something.
2. A *suction splash,* due to presence of large quantities of fluid together with gas in the stomach, is heard by the naked ear, on vigorously shaking the abdomen.
3. On *ausculto-percussion,* as the greater curvature is traced, it is found that the stomach is grossly dilated.

Special Investigations

1. *FTM Analysis:*
   a. Huge quantity of fasting content, often containing old food.
   b. A low hydrochloric acid level. This is due to lack of function of the oxyntic cells, resultant upon the severe chronic gastritis set up by fermentation of food from stasis.
   c. A high total acid level because of organic acids derived from fermentation.
   d. Absence of bile in all the samples.
   e. Copious amount of mucus because of chronic gastritis.
2. *Barium Meal Examination:*
   a. A grossly dilated stomach.
   b. Evidence of gross chronic gastritis.
   c. Failure of the stomach to evacuate the meal even after 6 hours (stasis of the meal may also be found in cases of pylorospasm; in these cases, however, injection of an antispasmodic evacuates the meal; this does not occur in cases of actual stenosis).
   d. Simultaneous finding of the meal in the stomach and the transverse colon.

Treatment.—The treatment is operative and consists of either a vagotomy with gastrojejunostomy or a subtotal gastrectomy, as is done for chronic duodenal ulcer. Though the acid secretion in these patients is low and the only problem is the obstruction, a simple gastrojejunostomy is not advisable. As soon as the obstruction

Radical Surgery.—This is called *radical gastrectomy* which means en bloc resection of the growth-bearing area of the stomach with at least 1½ inch of healthy stomach wall at either end, together with all the nodes draining the stomach. According to the situation of the growth, radical gastrectomy may be:

1. *Lower Radical Gastrectomy.*—For growths in the lower part of the stomach.
2. *Upper Radical Gastrectomy.*—For growths in the upper part of the stomach.
3. *Total Radical Gastrectomy.*—The whole of the stomach is resected in cases of growths infiltrating widely along the wall of the organ.

Gastrectomy has been described in details in Chapter on instruments under gastrointestinal clamps.

Palliative Surgery

A. For inoperable pyloric growths:
   1. *Gastrojejunostomy.*—The difficulties after a simple gastrojejunostomy are:
      a. The bleeding from the growth still continues.
      b. Loss of appetite and vomiting continues.
      c. The stoma is quickly infiltrated by the growth.

      Thus, a better procedure is:
   2. *Devine's exclusion operation.*—In this operation, the stomach is transected well above the growth and the distal cut end is closed. A gastro-jejunostomy is then performed between the proximal cut end of the stomach and the jejunum. The disadvantages of a simple gastrojejunostomy are overcome as the growth is excluded from the pathway of food. This operation is contraindicated if the pylorous is completely occluded because the secretions of the isolated chamber cannot come out.

B. For inoperable growths in the upper part of the stomach and for leather-bottle stomach:
   1. Oesophagojejunostomy.
   2. Permanent jejunostomy for feeding.

C. For inoperable growths at the cardiac end:

   An oesophageal tube, as used for oesophageal cancers, is passed down to the stomach through the growth, and the patient is fed through it.

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

Etiology and Pathology.—This is a congenital condition of pyloric obstruction and distinctly differs from the common post-ulcer pyloric stenosis of the adults in that there is no fibrosis in the pylorus or duodenum; instead, there is a gross hypertrophy of the pyloric musculature, so much so that its lumen is obliterated.

The factors, leading to this pyloric obstruction, are as follows:

1. Gross hypertrophy of the musculature of the pyloric antrum, particularly the circular muscle fibres, and the hypertrophic musculature encroaching into the lumen of the pylorus. Distally, the hypertrophic fibres end abruptly at the pylorus, so that the duodenum is absolutely normal. Proximally, the hypertrophy gradually fades off into normal gastric musculature.
2. A spasm of the hypertrophic musculature. Whether the spasm is primary and the hypertrophy is secondary to it, or whether the hypertrophy is congenital and the spasm is secondary, is a matter of debate. According to

many pathologists, again, the condition is due to an achalasia of the pylorus (i.e. primary failure to relax) and, in order to overcome this, the antral musculature hypertrophies.

3. Oedema of the pyloric mucous membrane. This is believed to be due to irritation by curd, resulting from stasis of milk in the stomach.

Onset.—Though the condition is called congenital, the symptoms of obstruction seldom start at birth. In the majority of cases, symptoms begin between the second and third weeks of life. This may be explained by two factors:

a. The oedema of the mucous membrane, which makes the obstruction complete, takes 2 to 3 weeks' time to develop.
b. The spasm of the pyloric musculature starts at that time.

Incidence.—There are certain interesting features as regards the incidence of the disease:

1. Males suffer much more frequently than females (9:1).
2. It is usually the first-born male child that suffers and the incidence steadily declines according to the position of the child in the stem.
3. There is often a hereditary factor—the disease appears in some families with greater frequency.

Symptoms.—As has already been stated, symptoms usually start between the second and third weeks of life. A child, who was so long healthy and gaining weight, suddenly starts deteriorating as follows:

1. Vomiting.—Forcible and projectile, and not containing bile. It occurs almost after every feed. In spite of vomiting, the appetite is good and the child wants food after each vomit.
2. Constipation.—Small, dry stools.
3. Urine output diminishes gradually, so that there is less wetting of the napkins.
4. Loss of weight.

Physical Signs

1. There are signs of dehydration, including depressed frontanalles.
2. Visible Peristalsis.—The dilated stomach, proximal to the obstruction, is seen as a ball-shaped lump, moving from left to right across the epigastrium, particularly after a meal.
3. If, by gentle palpation, the firm hypertrophic pylorus can be felt as a lump just below the right costal margin, the diagnosis is confirmed.

Special Investigations.—Diagnosis is usually made on clinical examination. In case of difficulty, a *barium-meal examination* may be done and this shows:

a. Dilated stomach.
b. Retention of the meal in the stomach even after six hours.
c. The pyloric canal shows an elongation and persistent gross narrowing—the *'string sign'*.

If a barium examination is done, the remnant meal must be sucked out since, otherwise, the child may vomit and aspirate the meal.

A better procedure, to avoid the hazards of a barium examination, is to give the child a measured quantity of milk and to aspirate the stomach after 4 hours. Recovery of more than 75 per cent of the meal confirms the diagnosis.

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