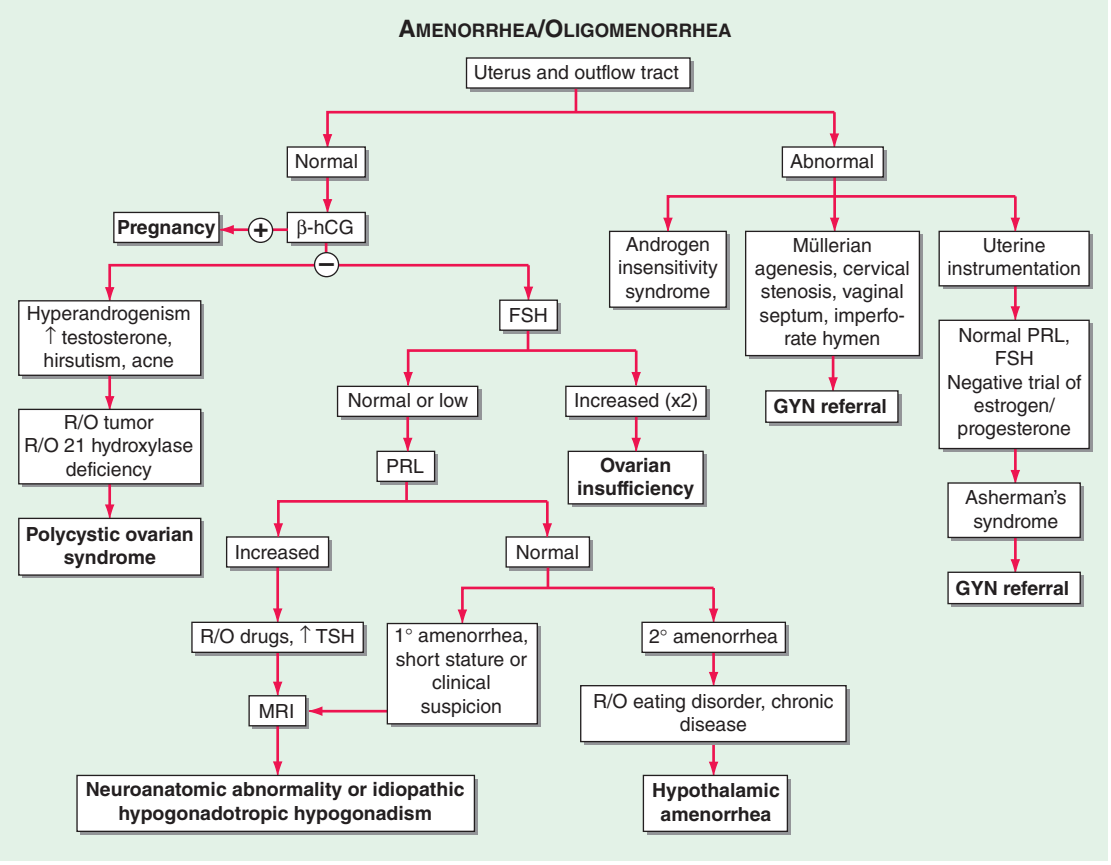

FIGURE 69-2 Algorithm for evaluation of amenorrhea. β-hCG, human chorionic gonadotropin; FSH, follicle-stimulating hormone; GYN, gynecologist; MRI, magnetic resonance imaging; PRL, prolactin; R/O, rule out; TSH, thyroid-stimulating hormone.

Hypergonadotropic hypogonadism occurs rarely in other disorders, such as mutations in the FSH or LH receptors. Aromatase deficiency and 17α-hydroxylase deficiency are associated with decreased estrogen and elevated gonadotropins and with hyperandrogenism and hypertension, respectively. Gonadotropin-secreting tumors in women of reproductive age generally present with high, rather than low, estrogen levels and cause ovarian hyperstimulation or dysfunctional bleeding.

TREATMENT HYPO- AND HYPERGONADOTROPIC CAUSES OF AMENORRHEA

Amenorrhea almost always is associated with chronically low levels of estrogen, whether it is caused by hypogonadotropic hypogonadism or ovarian insufficiency. Development of secondary sexual characteristics requires gradual titration of estradiol replacement with eventual addition of progestin. Hormone replacement with either low-dose estrogen/progesterone regimens or oral contraceptive pills is recommended until the usual age of menopause for bone and cardiovascular protection. Patients with hypogonadotropic hypogonadism who are interested in fertility require treatment with exogenous FSH combined with LH or pulsatile GnRH. Patients with ovarian failure can consider oocyte donation, which has a high rate of success in this population, although its use in women with Turner's syndrome is limited by significant maternal cardiovascular risk.

POLYCYSTIC OVARIAN SYNDROME (PCOS) PCOS is diagnosed based on a combination of clinical or biochemical evidence of hyperandrogenism, amenorrhea or oligomenorrhea, and the ultrasound appearance of polycystic ovaries. Approximately half of patients with PCOS are obese, and abnormalities in insulin dynamics are common, as is metabolic syndrome. Symptoms generally begin shortly after menarche and are slowly progressive. Lean oligo-ovulatory patients with PCOS generally have high LH levels in the presence of normal to low levels of FSH and estradiol. The LH/FSH ratio is less pronounced in obese patients in whom insulin resistance is a more prominent feature.

TREATMENT POLYCYSTIC OVARIAN SYNDROME

A major abnormality in patients with PCOS is the failure of regular, predictable ovulation. Thus, these patients are at risk for the development of dysfunctional bleeding and endometrial hyperplasia associated with unopposed estrogen exposure. Endometrial protection can be achieved with the use of oral contraceptives or progestins (medroxyprogesterone acetate, 5–10 mg, or prometrium, 200 mg daily for 10–14 days of each month). Oral contraceptives are also useful for management of hyperandrogenic symptoms, as are spironolactone and cyproterone acetate (not available in the United States), which function as weak androgen receptor blockers. Management of the associated metabolic syndrome may be appropriate for some patients (Chap. 422). For patients interested in fertility, weight control is a critical first step. Clomiphene citrate is highly effective as a first-line treatment, and there is increasing evidence that the aromatase inhibitor letrozole may also be effective. Exogenous gonadotropins can be used by experienced practitioners; a diagnosis of polycystic ovaries in the presence or absence of cycle abnormalities increases the risk of hyperstimulation.

PELVIC PAIN

The mechanisms that cause pelvic pain are similar to those that cause abdominal pain (Chap. 20) and include inflammation of the parietal peritoneum, obstruction of hollow viscera, vascular disturbances, and pain originating in the abdominal wall. Pelvic pain may reflect pelvic disease per se but also may reflect extrapelvic disorders that refer pain to the pelvis. In up to 60% of cases, pelvic pain can be attributed to