values. As noted above, Gypsies consider the body below the waist impure and always cover the legs completely in their dress. They thus find usual hospital attire extremely degrading, a fact those caring for them should bear in mind.

The majority of the American Gypsy population have very poor health habits, with a high percentage of smoking, heavy salt and fat intake, obesity, and consanguineous marriages. Not surprisingly, this has led to an extremely high prevalence of hypertension, diabetes, and occlusive vascular disease, with strokes and myocardial infarctions observed in patients as young as their 20s. Many of these habits, such as smoking and obesity, are culturally ingrained and are very difficult for them to break.

GYPSY FUNERAL RITUALS

Should a Gypsy die in the hospital, it is imperative that hospital personnel allow the family to perform a brief grieving ritual. This involves lighting a candle under the bed with the patient positioned next to an open window. He is then rubbed down with holy oil as the entire extended family engages in a brief but intense grieving period, with hair torn out, faces scratched, and relatives throwing themselves to the floor. A 3-day wake then follows, and after burial, four large feasts are held to mark the 3-day, 9-day, 6-week, and 1-year anniversaries of the patient's death. These feasts are full of symbolic rituals, in which food, clothing, and incense are offered to the departed relative.

Understanding a patient's culture and outlook is critical to any doctor-patient interaction, but especially so when dealing with the Gypsy population. Understanding the reasons behind some Gypsy requests and behavior should facilitate effective medical care and allow for a more satisfying interaction with their care-givers.

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SUGGESTED READING

Thomas JD. Gypsies and American medical care. Ann Intern Med 1985; **102**:842–845.

Thomas JD, Doucette MM, Thomas DC, Stoeckle JD. Disease, lifestyle, and consanguinity in 58 American Gypsies. Lancet 1987; **2**:377–380.

Mandell F. Gypsies: culture and child care. Pediatrics 1974; **54**:603–607.

Sutherland A. Gypsies: the hidden Americans. London: Tavistock, 1975.

McDonald B. Gypsies: wanderers of the world. Washington, DC: National Geographic Society, 1970.

ENDOCRINE CAUSES OF IMPOTENCE

Impotence affects 10 to 20 million men in the United States. Yet, despite this prevalence, little is known about the disorder from a biologic perspective, especially in terms of endocrinology.

Overall, probably 5% to 10% of impotence cases have an endocrine component. Clinicians should consider endocrine disorders as a possible cause of impotence, because these disorders can be diagnosed readily with laboratory tests and, in the majority of patients, can be cured with appropriate therapy.

ANDROGEN DEFICIENCY

Hypoandrogenism may cause impotence. Low circulating androgen levels may be due to primary gonadal dysfunction or may result from hypothalamopituitary disease. In the majority of patients, testing for low androgen levels is straightforward, namely, the measurement of total serum testosterone. Gonadotropin measurements may be helpful in delineating the etiologic site of hypoandrogenism. Particularly in obese individuals, total testosterone levels may be misleadingly low as a result of low levels of sex hormone-binding globulin, but free (unbound, biologically active) testosterone levels may be normal. In such patients, gonadotropin levels are usually normal and treatment with androgen is generally of no value.

HYPERPROLACTINEMIA

Hyperprolactinemia is another endocrine disorder for which patients with impotence should be screened. About 80% of men who have prolactin levels above 50 ng/mL (normal = 2 to 12 ng/mL) complain of diminished libido and impotence. Prolactin excess results in impaired secretion of gonadotropin-releasing hormone, so that there is usually concomitant hypogonadotropic hypogonadism. Additionally, there is a direct inhibitory effect of prolactin excess on sex drive and function. Thus, treatment with androgen alone is not generally effective in such patients. Primary therapy should be directed

towards lowering prolactin levels and adding testosterone only if there is evidence of organic hypogonadotropism, ie, if low androgen levels fail to recover 1 to 3 months after attainment of normoprolactinemia.

DIABETES MELLITUS

Approximately 25% to 50% of patients who have had *type I diabetes mellitus* for 25 years or more have impotence. The cause may be vascular or neurologic or a combination of both.

Type II diabetes is also a major risk factor for the development of impotence. Routine glucose tolerance testing for all impotent men is not recommended, however, since mild abnormalities are common and are rarely the cause of impotence.

OTHER ENDOCRINE DISORDERS

Other endocrine disorders that may be associated with impaired sexual function include Cushing's syndrome (which is generally easy to diagnose), hyperthyroidism, and hypothyroidism.

DIAGNOSIS

The history may provide clues to the cause of the impotence. A comprehensive drug history is essential. A declining libido suggests an underlying endocrine problem. Diminished beard growth may be an additional hint. Gynecomastia should also be considered, since excessive estrogen may be a factor. Indeed, gynecomastia and impotence were prevalent among workers in early birth control pill factories. The presence of a sex drive, nocturnal penile tumescence, and erections with autostimulation point towards a psychogenic problem such as "performance anxiety."

The physical examination should include an assessment of hair growth, fat and muscle bulk, breasts, testes size, prostate size, fundi and visual fields, and the presence of other endocrinopathies.

Two critical laboratory tests that help to identify the cause of impotence are total testosterone and prolactin levels. Other tests that may be helpful include free testosterone levels (especially in obese patients), follicle-stimulating hormone and luteinizing hormone levels, and estradiol levels.

ENDOCRINOLOGIC TREATMENT

Treatment involves either lowering prolactin or replacing testosterone. Normalization of elevated prolactin levels usually can be achieved with the use of bromocriptine even in cases with pituitary tumors. Surgery or radiotherapy are additional options if a pituitary tumor is present, particularly if it is not a prolactinoma. Testosterone is administered intramuscularly as the cypionate or enanthate ester in oil at a dose of 200 mg every 2 to 4 weeks. Excessive androgen is not useful and may be dangerous. Hyperlibido is probably a myth but aggressive behavior (steroid rage) and acute cardiovascular deaths have been reported with androgen abuse.

Testosterone therapy may also be a consideration for men with low-normal levels. The threshold level of serum testosterone is 200 to 450 ng/dL (6 to 15 nmol/L). However, the potential efficacy of a therapeutic trial should not be overstated, and a placebo effect must be recognized.

Before administering androgen, a measurement of prostate-specific antigen (PSA) is advisable, particularly in men over age 50. It is recommended that blood for PSA should be drawn prior to the obligatory digital examination of the prostate since prostatic massage may falsely elevate PSA levels. There are two reasons for these recommendations. First, older men are at higher risk for prostate cancer. Second, androgen may enhance tumor growth, so that androgen deficiency may actually be desirable. In addition, liver function tests are recommended before starting therapy and at intervals thereafter. An occasional patient may develop reversible cholestasis.

Androgen therapy may be associated with a lag phase, so improvement might not be evident for up to 1 month after therapy is started.

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SUGGESTED READING

Carter JN, Tyson JE, Tolis G, Van Vliet S, Faiman C, Friesen HG. Prolactin-secreting tumors and hypogonadism in 22 men. N Engl J Med 1978; **299**:847–852.

Krane RJ, Goldstein I, Saenz de Tejada I. Impotence. N Engl J Med 1989; **321**:1648–1659.

NIH Consensus Development Panel on Impotence. NIH consensus conference. Impotence. JAMA 1993; **270**:83-90.