Actually each instance presented a difficult diagnostic problem. In not one case could an absolute diagnosis of hernia as the cause of the obstruction be made, although hernia was suspected in all but the second case.

In any occasion of acute, intestinal obstruction where there is a history of previous hernia or any masses in the inguinal or femoral region, the possibility of strangulation with incarceration of the hernia should be excluded.

The incision should be in a region where the suspected hernia is easily accessible. Decision as to where the incision should be made, however, is often difficult when the etiologic factor is not localized.

Clinical diagnosis of high or low obstruction may help since the jejunum and proximal portion of the ileum usually lay high in the abdomen and to the left, while the distal portion of the ileum is apt to be in the lower part of the abdomen or pelvis. In the first case there was some difficulty in freeing the incarcerated bowel because the incision was distant from the site of herniation.

A small incision is preferable. It may be enlarged if indicated. Through a small wound a collapsed loop of intestine can be picked up and traced back to the point of obstruction. A large incision presents the problem of coping with an open abdomen full of distended loops of bowel. With a small incision shock from exposure and handling is minimized, and closure of the wound is greatly facilitated.

ABSENCE OF PITUITARY FAILURE IN FAT BOYS WITH TESTICULAR DEFICIENCY

E. PERRY McCULLAGH, M.D., AND IRENE T. KLINE, M.A.

The term Fröhlich's syndrome is commonly used to designate obesity and retarded sexual maturation without regard for the fact that the original description of the condition by Babinski[1] and by Fröhlich[2,3] included also the presence of a suprasellar tumor. While obesity and retarded maturation are a combination frequently seen, suprasellar tumor is a rare concomitant. Adiposogenital dystrophy, a term proposed by Bartels,[4] might be used appropriately for these more common cases without tumor were it not that it, too, was originally designed to include the presence of a suprasellar tumor.

Opinions on the testicular biopsies are based on reports of Dr. Earl T. Engle, who studied the sections.

Early workers[4,5,6] pointed out that obesity could not result from pituitary failure. This fact has been repeatedly confirmed. Bailey and Bremer[7] were the first to prove experimentally that a condition simulating adiposogenital dystrophy could be caused by a lesion in the hypothalamus alone, without injury to the pituitary gland. Substantiation of this concept has been abundant.[8] The implication has remained, however, that the lack of pituitary gonadotrophins is the explanation of the gonadal deficiency in patients who are usually designated as having adiposogenital dystrophy. In cases of tumor in the region of the pituitary gland this supposition is undoubtedly true. Suprasellar tumors in boys are uncommon. When such a tumor occurs, it is likely to be associated with normal or less than normal height. The genitalia may be relatively infantile, but such patients are seldom obese.

Fröhlich's patient was approximately 58 inches (145 cm.) in height according to Bruch's estimate[3] (no definite height was originally given) and was 118.8 pounds (54 kg.) in weight at the age of 14 years, which was approximately 30 per cent over ideal. The tumor in the pituitary region, the presence of which was proved at operation, was apparently a craniopharyngioma. On the other hand, the common phenomenon of testicular failure in adolescent boys is generally not associated with a tumor in the region of the pituitary gland. Those patients without pituitary tumor may be above average height; they do have various degrees of hypoplasia of the genitalia, and their obesity is frequently greater than that in patients with a tumor. In short, Fröhlich's syndrome in its true form is very rare, and the term as it is usually used is a misnomer.

Gonadal failure with obesity is diagnosed more frequently in boys than in girls largely because the external genitalia can be seen to be small, or may seem to be small, because of excess of surrounding fat; and because the normal physical activity and aggressive attitude of the adolescent boy is replaced by a somewhat feminine demeanor and often by a distinct tendency to placidity or laziness. The condition must be carefully distinguished from simple obesity. In fat girls the condition may be suspected when there is delay in breast development and delay in the appearance of the menses. In adolescent girls who are not fat delay in breast development and retarded appearance of such changes as fullness of the hips make ovarian deficiency more evident. Minor degrees of gonadal failure, however, are not easily determined in either sex during early adolescence, since wide variations are normal.

Hypothyroidism is commonly misdiagnosed upon the finding of low metabolic rates in these obese children with delayed adolescence. Such patients frequently show no other signs of hypothyroidism. Their skin

DIAGNOSTIC CHARACTERISTICS			CASE I		CASE 2		CASE 3	
			Before treatment. 14 yr. 3-6-44.	After treatment. 15¾ yr. 12-3-45.	Before treatment 10½ yr. 6-23-44	After treatment 11⅝ yr. 10-6-45	Before treatment 18 yr. 1-5-45	After treatment 18½ yr. 5-4-45
HYPOGENITALISM	Assays	Urinary Gonadotrophins	105-212 MU/24 hr. 3-8-44. 105-212 MU/24 hr. 9-11-44.	53-105 MU/24 hr. 1-11-45. (no injections previous 2½ wk.)	212-318 MU/24 hr.	6-13 MU/24 hr. 7-27-45 (no injections previous 2 wk.)	105-212 MU/24 hr.	not done
		Urinary 17-ketosteroids	2.2 mg./24 hr.	5.8 mg./24 hr. 1-22-45.	1.5 mg./24 hr.	highest assay after 6 mo. therapy: 1.3 mg./24 hr.	7.8 mg./24 hr.	3-31-45 6.4 mg./24 hr.
	Testes	Location and size	L: about 3x1.5 cm. in scrotum R: severely atrophied	L: normal R: still small, but in upper scrotum	about 1.5x0.75 cm. approximately at external ring; both drop into scrotum	normal	about 1.5x1.25 cm. in scrotum	unchanged
		Biopsy	8-12-44, L: "Progressive tubular fibrosis with spermatogenic arrest"		9-12-44 "Very early developmental arrest, bilateral, with complete lack of differentiation"		1-26-45 "Incomplete development bilateral with beginning tubular fibrosis and cellular atrophy"	
	Penis	Size	2.25 cm. long	normal; 6.75 cm. in length	small	normal	7 cm. long; with long prepuce; normal for age 12	9 cm. long
OBESITY	Height[1] (inches)		68 (avg. 62.2)	71¾ (avg. 66.2)	58¾ (avg. 54.8)	60¾ (avg. 57.5)	65½ (avg. 68.2)	66 (avg. 68.2)
	Weight[1] (pounds)		209 (134 ideal for h.)	225 (155 ideal for h.)	131 (77 ideal for h.)	106½ (85 ideal for h.)	155½ (136 ideal for h.)	150 (136 ideal for h.)
	Overweight[1] (pounds)		75	70 (did not follow diet)	54	21	19½	14
	Distribution of fat		feminine fat distribution	less suggestion of feminine configuration	abdomen, hips, mammary glands	normal except for slight "pot belly"	especially in face, abdomen, hips and breasts — perhaps gynecomastia	little change
HYPOMETABOLISM	Basal metabolic rate		—18%	—16%	—13% 6-27-44	—23% 8-1-45	—12%	
HYPOTRICHOSIS	Pubic hair		none	normal for 17 yr.	none	beginning	½ normal female	slight increase
	Axillary hair		none	½ normal	none	none	very scant	no change
	Facial hair		none	slight mustache and beard	none	none	none	no change
FAULTY SKELETAL DEVELOPMENT	Epiphyseal age		not estimated		10 yrs.	not repeated	not estimated	
	Span exceeds height by		2 inches		0 inches			
SUBJECTIVE SYMPTOMS	Facies		immature for age	much more mature	puerile	more mature	puerile	same
	Demeanor		distinctly unagressive	improved	alertness below normal	more alert	less agressive than normal	improved
	Activity		much below normal	improved	good average endurance	more active	moderate	slightly more active
	Voice		puerile	distinctly lower	puerile	puerile	puerile (high)	same
THERAPY	Chorionic Gonadotrophin		500 IU q 2nd day to 750 IU daily (except 2½ weeks) 3-6-44 to 5-1-45		50 IU q 2nd day 6-27-44 to 2-9-44 500 IU/day 2-5-45 to 3-10-45 500 IU 3xwk. 3-10-45 to 7-14-45		3 months 2-10-45 to 5-4-45; 750 IU q 2nd day	
	Thyroid		Gr. 1 daily 3-6-44 to 6-8-44; Gr. 2 daily 6-8-44		Gr. 1 daily throughout		Gr. 1 daily throughout	
	Diet		1200 cal. per day (followed poorly)		1400 cal. per day until 6-14-45 later, 1800 cal. per day		1200 cal. with 60 Gm. protein per day	

[1] From Baldwin, B. T. and Wood, T. D.: Weight-Height-Age Tables in English Units of American Born Boys (clothed) of School Age. (Iowa: The Iowa Child Welfare Station, State University of Iowa, 1931).

is fine, and their complexions are pink. Their teeth may be excellent. Their nails grow well. Their height is normal; their bone age is little, if at all, retarded, and their blood cholesterol is typically within normal range.

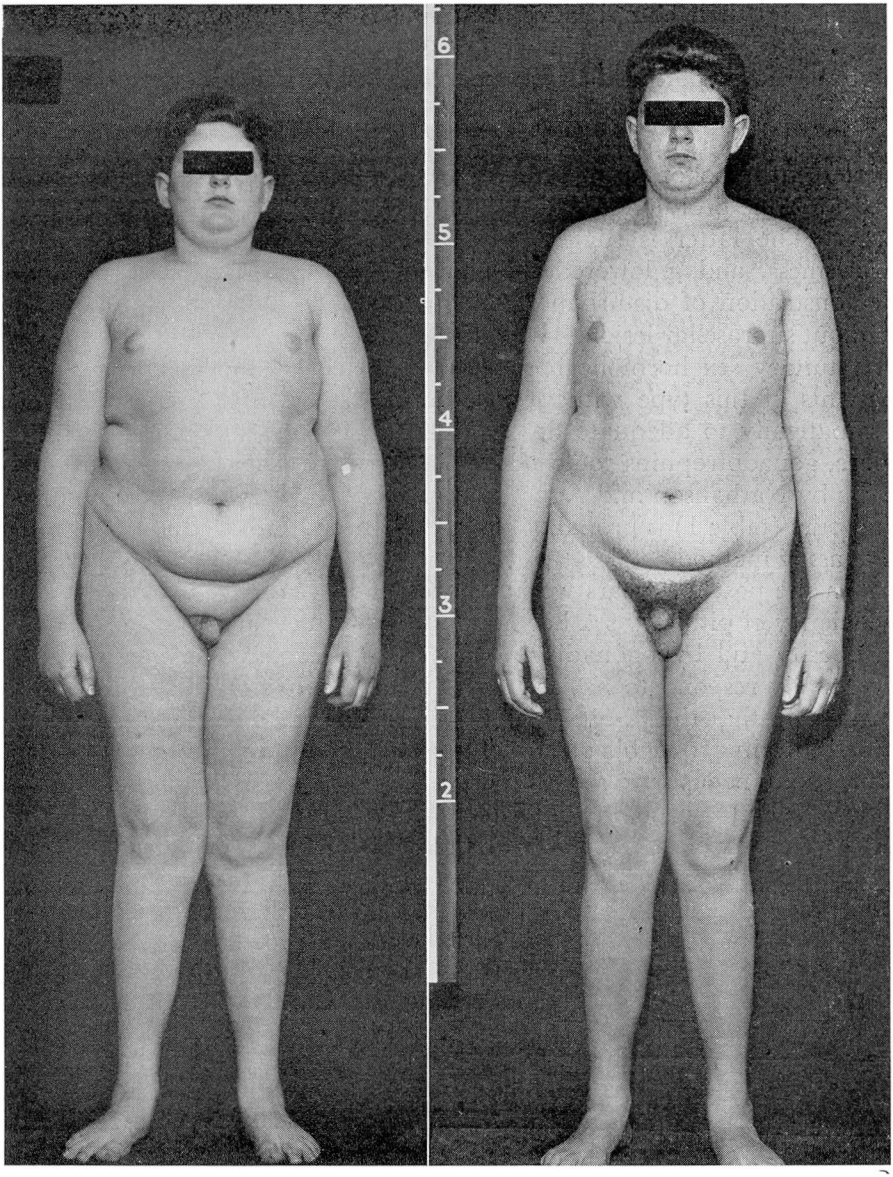
The usual conception of the physiologic mechanism involved in adolescent hypogonadism with obesity is that there is a lack of pituitary sex hormone production accompanied by a possible hypothalamic disorder. The latter is suggested more forcefully when hunger, thirst, drowsiness, and a lowered metabolic rate also are present. A clear demonstration of disturbance of hypothalamic function is impossible without suprasellar lesion. The belief that there is a deficient production of pituitary sex hormone has been supported by the fact that many patients of this type without pituitary or suprasellar lesions respond dramatically to adequate doses of chorionic gonadotrophin. Furthermore, gonadotrophins could not be found in the urine of any untreated cases by Nathanson and Aub.[9] The discrepancy between their findings and ours (table 1) is probably because we have employed a more sensitive assay method.

In the past few years we have become increasingly convinced that deficiency of pituitary sex hormone is not a usual part of this syndrome of obesity and hypogonadism. Many such persons, if untreated, may eventually respond to natural stimuli and attain normal adulthood,[10] suggesting that the pituitary gland in those who undergo spontaneous cure is, in time, capable of stimulating the gonads to a normal response. This spontaneous cure seems to be particularly possible for the patients whose weight is brought to normal.

In a group of our untreated adolescent boys with evidence of hypogonadism and varying degrees of obesity, titres of urinary gonadotrophins equal to or above those of normal adult males are present (tables 1 and 2). The studies of Greulich *et al.*[11] have shown that the urinary

TABLE 2
Urinary Gonadotrophin Titres
Normal Adult Males

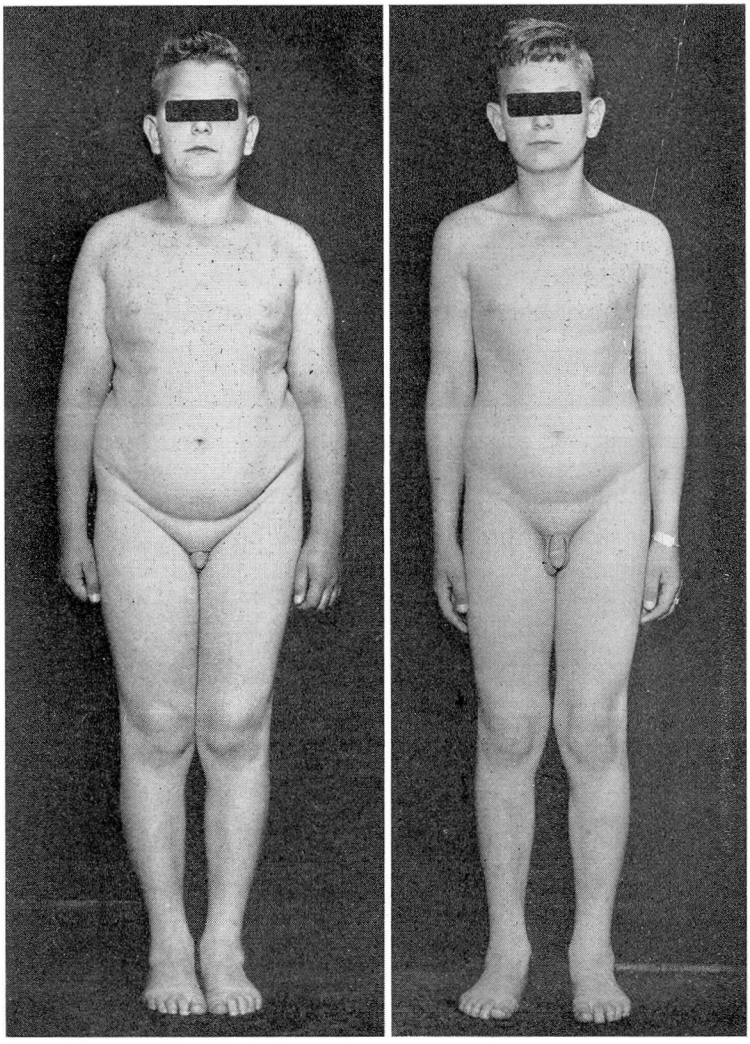
MU/24 hr.		No. of Cases
More Than	Less Than	
26	53	12
53	105	9
105	212	2
		Total 23



a *b*

Fig. 1, Case 1 (a) Before treatment. Age 14 years.
(b) After treatment. Age 15 years, 9 months.

excretion of gonadotrophins in normal adolescent boys reaches an adult level at 14 years of age, if skeletal development and secondary sexual



a *b*

FIG. 2, Case 2 (a) Before treatment. Age 10 years, 6 months.
(b) After treatment. Age 11 years, 6 months.

characteristics have attained a corresponding level of maturity.

The method we used for urinary gonadotrophin assays is the mouse uterine weight method described by Klinefelter *et al.*[12] Each test was done on a 16 hour aliquot of a 24 hour specimen. Normal adult male titres determined by this method in our laboratory are given in Table 2.

Urinary 17-ketosteroid determinations[13,14] were done on 24 hour or 72 hour urine specimens and expressed as mg. per 24 hours. Table 3 shows normal adult male tires for comparison.

The first explanation which comes to mind with regard to the excess titres of gonadotrophin in boys with obesity and genital dystrophy is that they have some degree of primary hypogonadism, or in milder cases a low testicular responsiveness, which may eventually attain relative normality. In some instances, however, these patients do respond to injections of chorionic gonadotrophin. Adequate or excessive amounts of urinary gonadotrophin, measured as follicle-stimulating hormone, do not necessarily imply the presence of an adequate amount or an excess of luteinizing hormone or interstitial cell-stimulating hormone. It does not seem likely, however, that there is an increase over the normal quantity of interstitial cell-stimulating hormone or even a lack of responsiveness to it, because of a prompt and often pronounced response to injections of chorionic gonadotrophin, which contains chiefly luteinizing hormone or interstitial cell-stimulating hormone.

Three adolescent boys (figures 1, 2, and 3) who have obesity and testicular deficiency with absence of pituitary or suprasellar tumor, and who might ordinarily be designated as Fröhlich's syndrome or adiposogenital dystrophy, are selected to demonstrate these points (table 1). It will be noted that two of these patients who had high titres of urinary gonadotrophin appeared to respond to injections of chorionic gonadotrophin. Their own gonadotrophins evidently were not producing the interstitial cell stimulation which was brought about by the pregnancy urine extract used in treatment.

Testicular biopsies performed in these and similar cases reveal that there are at least in some cases varying degrees of developmental arrest of the tubular tissues. There are also fibrosis of the intertubular tissues, varying from mild to severe, and a less than normal number of interstitial cells. Such studies will be presented subsequently in greater detail.

Wherever large amounts of follicle-stimulating hormone are present in the urine, pituitary dysfunction may be said to exist. Theoretically it is possible that there may also be a deficient production of luteinizing hormone, but until a satisfactory method for its measurement is available, that possibility cannot be ascertained. As an explanation of the hormonal imbalance the following might be considered: a primary pituitary disorder creates an excessive production of follicle-stimulating hormone, thereby causing testicular damage. It is more likely, however, that the main defect lies in early developmental arrest of the testis itself and that pituitary hyperfunction or dysfunction results as a secondary result of this change.

TABLE 3
Urinary 17-Ketosteroid Titres
Normal Adult Males

Mg./24 hr.	No. of Cases
4.0-4.9	1
5.0-5.9	1
6.0-6.9	4
7.0-7.9	7
8.0-8.9	6
9.0-9.9	6
10.0-10.9	4
11.0-11.9	5
12.0-12.9	1
13.0-13.9	2
14.0-14.9	2
15.0-15.9	0
16.0-16.9	1
17.0-17.9	1
18.0-18.9	1
Mean: 9.0-9.9	Range: 4.8-18.4 mg./24 hr. Average: 9.5 mg./24 hr.

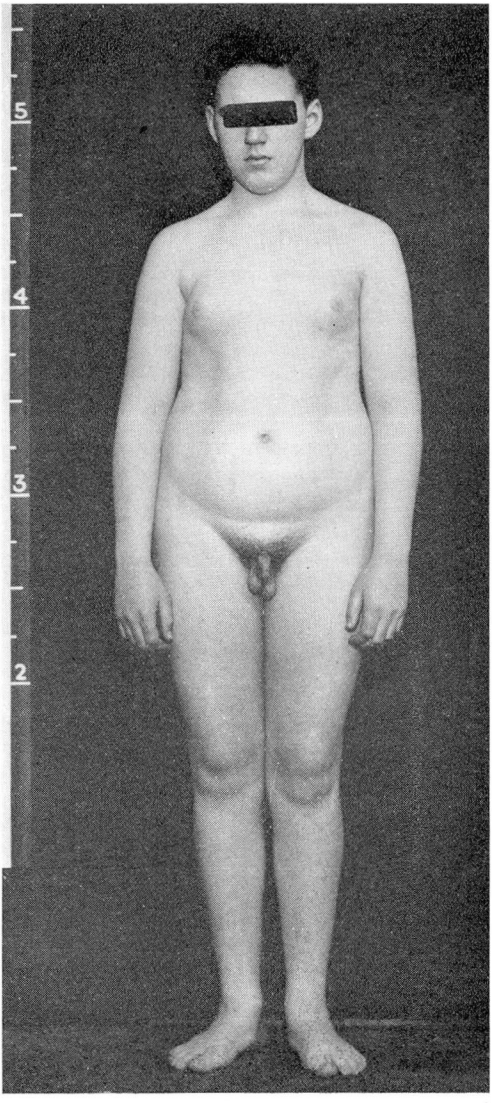


FIG. 3, Case 3—Before treatment. Age 18 years.

SUMMARY

An outline of the essential findings in three patients is presented. These patients represent a group now being studied whose primary features are testicular failure of the adolescent type, obesity, absence of tumor in the region of the pituitary gland, and the presence of a normal or an increased quantity of follicle-stimulating hormone in the urine.

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CERVICAL PERIARTHRITIS

Diagnosis and Treatment

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The term, periarthritis, by definition means inflammation in the tissues around a joint. Every case of arthritis has an associated periarthritis. The term, cervical periarthritis, we have reserved for those cases in which no arthritis or other abnormal anatomical bone change can be demonstrated. It may be the cause of pain and stiffness in the neck and is usually recognized by the presence of soreness in the supporting ligaments and muscles of the neck.

Many patients with cervical periarthritis complain of numbness and aching in the arms and hands and may have soreness in the shoulder muscles. In the absence of x-ray evidence of disease in the bone, the

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