OXYPHIL PARATHYROID ADENOMAS *

SHELDON C. SOMMERS, M.D., and THOMAS L. YOUNG, M.D.
(From the Laboratory of Pathology and the Cancer Research Institute, New England Deaconess Hospital, Boston 15, Mass.)

Oxyphil parathyroid adenomas have generally been regarded as incidental pathologic curiosities encountered at necropsy and without practical clinical or endocrinologic importance. However, numerous scattered reports in the literature and our observations combined would indicate that this is frequently not the case.

Histologically, the parathyroid gland consists of pale and dark principal or chief cells, dark or transitional oxyphil cells, water-clear or wasserhelle cells, and pale or mature oxyphil (Welsh) cells, mingled with fat and blood vessels. It is the pale oxyphil cell adenoma with which this paper is mainly concerned. So-called transitional oxyphil cells are smaller, contain vacuoles, and resemble dark chief cells. They are frequently present in those parathyroid adenomas which usually produce hyperparathyroidism and which it is not the present purpose to discuss.

Grossly, the oxyphil adenoma is composed of soft, solid, gray-brown or yellow-brown tissue, not readily distinguishable from other parathyroid adenomas. Microscopically, the oxyphil cells are large, with abundant eosinophilic granular cytoplasm and small, regular nuclei. The cells have sharp borders and occur in sheets, cords, and occasionally form acini containing colloid. They contain little lipid, and the cytoplasmic granules take fuchsin stains. As in other parathyroid adenomas, mitotic figures are rare. To our knowledge, no acceptable oxyphil cell carcinoma of the parathyroid gland has been reported.

Including the first case as reported by Erdheim[1] (1903), a total of 25 cases of oxyphil adenoma have been recorded (Table I).[1-18] For 6 of the earlier cases the microscopic descriptions were incomplete and these are susceptible to other interpretations, but from the illustrations and text are considered acceptable oxyphil growths.

Six new cases of oxyphil adenoma have been added, including 3 observed at necropsy.

Case 1

F. F. (no. 132190) was a Jewish woman who had had 14 hospital admissions and had died at the age of 63 years. At 21 years of age, having borne 2 children, she had puerperal fever, and at 23 years, bilateral ectopic tubal pregnancies. Kidney gravel

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TABLE I
Reported Parathyroid Oxyphil Adenomas

Author	Sex	Age	Size	Site	Osteitis fibrosa cystica	Remarks
Erdheim[1] (1903)	M	18	2.5 cm.	RL		Healed rickets
Zehbe[2] (1909)						
Möller[3] (1911)	F	80	0.3 x 0.2 cm.			
Schmorl[4] (1912)						Osteitis deformans
Schmorl[4] (1912)	F	47			+	"Acromegaly," pituitary adenoma
Molineus[5] (1913)	F	59			+	
Molineus[5] (1913)	F	48	1.8 cm. 2.3 cm.	RL LL	+	Pituitary adenoma
Danisch[6] (1925)	M	72		LL		Diabetes mellitus, arteriosclerosis
Danisch[6] (1925)	M	71		LL		Carcinoma of stomach, obliterative pericarditis
Cushing and Davidoff[7] (1927)	M	35	0.9 cm. 0.4 cm.	RU LU		Acromegaly, cardiac failure
Hadfield and Rogers[8] (1932)	F	58	5 x 3.5 x 3 cm. (52 gm.)	L		Serum calcium, 11.8 mg.%
Gutman, Swenson, and Parsons[9] (1934)	F	60	2.0 cm. 1.5 x 0.7 x 0.4 cm.	RU RL	+	Polycystic kidneys, uremia; calcium, 10.2 mg.%
Lawrence and Zimmerman[10] (1935)	M	44	0.3 cm.			Cushing's syndrome; dissecting aneurysm
Warren and Morgan[11] (1935)	F	54	1.7 x 0.9 x 0.9 cm.	RU	+	Carcinoma of breast
Pappenheimer and Wilens[12] (1935)				•		4 cases, 2 of nephritis

Chown[13] (1937)	F	46	0.22 x 0.1 cm.			Ruptured cerebral aneurysm, hypertension, renal calculi
McQuillan[14] (1938)	F	46		R		Serum calcium, 10.2 mg.%; phosphorus, 4.2 mg.%
Cope[15] (1944)	F	70	6 x 3 x 3 cm. (23 gm.)			Died of cardiac failure; phosphorus, 3.8 mg.%
Norris[16] (1946)	F	23	3.5 cm.	L		Serum calcium, 12.3 mg.%
Willis[17] (1948)	F	55	2.5 x 1.5 cm.			Uterine fibroids, meningioma, adrenal cortical adenomas
Black and Ackerman[18] (1950)	F	59	3 x 3 x 1.3 cm.	LL	+	Hypertension, rheumatoid arthritis, nephritis; serum calcium, 10.6 to 13.8 mg.%; phosphorus, 3.9 to 6.3 mg.%
Black and Ackerman[18] (1950)	F	66	4.3 x 2.2 x 1.9 cm.	R		Died of renal insufficiency, renal calculi

R = right; RL = right lower; RU = right upper.
L = left; LL = left lower; LU = left upper.

was found when she was 30 years old. Eight years later a colloid goiter with multiple nodules and secondary hyperplasia was removed surgically. One year later hysterectomy and salpingo-oophorectomy were performed for adenomatous endometrial hyperplasia, adenomyosis, and leiomyoma. The ovarian cortical stroma showed marked hyperplasia and thecomatosis. There was postoperative hemorrhage. Pneumonia occurred at 47 years of age. The next year for the first time she developed hypertension of 170 to 190/88 mm. of Hg, and weight had increased from 145 to 165 lbs. Diabetes mellitus was diagnosed, requiring 18 units of insulin daily, but this the patient used erratically. At 57 years of age she survived perforating appendicitis with retrocecal abscess. Pyelitis was treated 3 years later. At age 62, a sigmoid cancer was resected with a low rectal anastomosis, because the patient refused abdominoperineal resection. Adenocarcinoma with blood vessel invasion and metastases to two of nine lymph nodes was diagnosed pathologically. The tumor recurred locally in 14 months and an abdominoperineal operation was performed, but the patient died the next day of atelectasis.

At necropsy, hypertensive heart disease, arteriolar nephrosclerosis, multiple mucosal polyps and submucous lipoma of the remaining colon, mesenteric lipoma, focal chronic pancreatitis, old cystic softening of the right basal ganglia, and cranial thickening consistent with old inactive Paget's disease were diagnosed.

Four parathyroid glands were isolated; they were not enlarged grossly, measuring from 0.2 to 0.5 cm. in greatest diameter.

One gland contained two sharply demarcated adenomatous nodules of oxyphil cells (Fig. 1), a cyst with colloid, and less well delimited areas of oxyphil cells. The other three parathyroid glands were within normal limits.

Microscopically, the pituitary body contained an eosinophilic adenoma measuring 0.7 by 0.5 cm.

Case 2

E. L. D. (no. 51A34) was 76 years old at death. A woman of Italian descent, she had had six successful pregnancies, and was first seen at age 70 because of 15 years of post-menopausal bleeding. She weighed 160 lbs., and had hypertension of 220 mm. of Hg systolic, with cardiac enlargement. On three occasions over the next 3 years specimens of endometrium taken for biopsy showed hyperplasia. There was estrogen effect apparent by vaginal smear and skin examination, but the source could not be found. The patient continued to have vaginal bleeding until her death from heart failure.

At necropsy, facial hirsutism was present. There were thrombi in the pulmonary arteries with pulmonary infarcts. A carcinoma simplex of the left adrenal cortex had metastasized to lymph nodes, right kidney, diaphragm, liver, and lungs. The endometrium showed adenomatous hyperplasia with foci of secretory activity, indicating endogenous production of both estrogen and progesterone, and there was cortical stromal hyperplasia of the ovaries. Cholelithiasis and arteriolar nephrosclerosis were found also. The head was not examined.

Three parathyroid glands were isolated. All were hyperplastic. In the largest gland there was a sharply encapsulated mass composed mainly of oxyphil cells, with a few included chief cells (Fig. 2). Foci of oxyphils were present in the other glands.

Case 3

E. V. H. (no. 36A12) was 68 years of age at death. A Canadian-born mother of 2 children, when 44 years old she had had a gastroenterostomy performed elsewhere for a large gastric ulcer eroding into the pancreas. At 57 years of age, appendectomy and removal of infarcted omentum were necessary. Two years before death a sore inside the left cheek was biopsied, with the diagnosis of epidermoid carcinoma grade II. The Hinton test was negative and blood pressure was 120/70 mm. of Hg. Many x-ray treatments were administered without arresting the extension of cancer to the maxilla and hard palate, with involvement of the tongue and death from terminal bronchopneumonia.

At necropsy, a mucinous adenocarcinoma was found in a 1.4 cm. gastric polyp, and there was also a 5 mm. pyloric peptic ulcer. Ovarian stroma showed cortical hyperplasia and granuloma formation. In the atrophic thyroid gland a 0.2 cm. oxyphil adenoma was present, compressing uninvolved parathyroid tissue composed of chief cells (Fig. 3).

Case 4

E. M. (no. 62214) was a woman of Irish extraction who had borne 3 children. At 33 years of age menopause was induced by radium in the treatment of an epidermoid carcinoma of the cervix. When she was 37 years old, removal of bladder stones was performed, and 10 years later an anomalous vessel to the right kidney producing hydronephrosis was cut. Because of a positive Hinton test, antiluetic therapy was administered for 2 years at 38 years of age. When she was 49 years old abdominal pain and gastro-intestinal roentgenograms led to a diagnosis of duodenal ulcer, which was treated by milk, cream, and amphojel for 2 years. At 51 years of age, roentgenograms showed increased density of the skull with mottled and punched out areas, fuzziness of the vertebrae, and coarse trabeculation of the innominate bones. Diagnosis was difficult, but repeated chemical examinations of the blood revealed serum calcium, 12.5 to 13.0 mg. per cent; phosphate, 2.0 to 5.0 mg. per cent; alkaline phosphatase, 15.8 to 16.9 Bodansky units; and Sulkowitch test, 3 plus. After 6 months, further roentgenograms showed cystic changes in the long bones, considered indicative of hyperparathyroidism and possibly also of Paget's disease. The urine contained Bence Jones protein, but marrow puncture showed only hyperplasia.

Operation at 52 years of age resulted in the removal of a right lower parathyroid adenoma, 3.5 by 2 by 1.5 cm., and 25 gm. of colloid goiter with multiple nodules. Postoperative roentgenograms showed increased bone density, confirming pre-existing hyperparathyroidism. After operation the patient suffered from hypertension of 180/110 mm. of Hg and synovitis. Six years postoperatively, at 58 years of age, she died of progressive bulbar muscular atrophy.

Microscopically, the parathyroid tumor was composed chiefly of interdigitating cords of large oxyphil cells, which made up 80 per cent of the gland (Fig. 4). Necropsy elsewhere showed in addition to muscular atrophy, cardiac hypertrophy and dilatation of hypertensive type, pulmonary congestion and edema, fibrous pleuritis, moderate hydronephrosis, a 1.5 cm. cortical adenoma of the right adrenal gland, and radiation damage to the cervix and bladder with ulcerative cystitis.

Case 5

M. A. (no. 104582) was 31 years old and mother of one child. She had had an x-ray diagnosis of a cyst of the mandible 1 year previously, and 5 months previously three more cysts had developed in the jaw and one in the pubic bone. Calcium stones were found in the kidneys. An operation was performed at this time in another hospital without discovery of a parathyroid tumor. Chemical findings later included serum calcium, 14.5 per cent; phosphorus, 3.9 mg. per cent; alkaline phosphatase, 4.8 Bodansky units; Sulkowitch test, 2 plus. Roentgenograms showed bone changes typical of hyperparathyroidism and tiny renal calculi. At the second operation a tumor 2 by 2 by 0.9 cm. was removed from the lower right area of the neck.

Microscopically, the tumor had a thick fibrous capsule with delicate trabeculae and was composed in part of solid areas of oxyphil cells and elsewhere of mixed oxyphils and dark chief cells (Fig. 5).

Case 6

L. B. O. (no. 128085) was a woman who had had three pregnancies and two successful deliveries. When she was 59 years old she had been seen because of excessive vaginal bleeding for 2 years, previous to which she claimed normal menses. She weighed 202 lbs. and had hypertension of 210/100 mm. of Hg. By biopsy, a patho-

logic diagnosis of epidermoid carcinoma of the cervix was made. Successful radiation therapy was given. At 67 years of age a cyst of the right kidney was removed. At 69 years she developed diabetes mellitus. One year later she was seen with a basal metabolic rate of plus 24, a nodular thyroid gland, auricular fibrillation, and loss of weight to 149 lbs. At operation a colloid goiter with multiple nodules was removed. Posterior to the right thyroid lobe was a soft, yellow-brown, 0.9 cm. nodule; coincidentally with its manipulation the systolic blood pressure dropped from 170 to 70. The nodule was removed, and proved microscopically to be an oxyphil parathyroid adenoma with admixture of dark chief cells (Fig. 6). A normal parathyroid gland also was identified.

Of the 6 cases reported, all were in married women with children. Five were over 50 years of age. Two had evident hyperparathyroidism, 2 possibly Paget's osteitis deformans, 2 diabetes mellitus, and 5 had malignant neoplasms—carcinoma of cervix (2), sigmoid colon, adrenal cortex, mouth, and stomach—one with multiple primary cancers. Of the 4 cases without manifest parathyroid hyperfunction, one had kidney stones. Nodular goiters were present in 3, with secondary hyperplasia in 2. Endometrial hyperplasia was present in 2 patients. Hypertension was found in 4.

The frequent combinations of physiopathologic changes of a parathyroid gland, thyroid gland, pancreas, and uterus suggest a polyglandular syndrome and make one suspect pituitary dysfunction. Studies by others of acromegaly, Cushing's syndrome, and other hyperpituitary states already have shown a surprising incidence of parathyroid hyperplasia and adenoma formation (Table II). Additional cases[35-39] have been collected which, on anamnestic and morphologic grounds, have been thought to indicate that parathyroid hyperplasia or adenomas may form part of a polyhormonal pituitary disorder, particularly in women. Our material would support this thesis.

It has been asserted on good authority that oxyphil adenomas are non-functioning and do not produce hyperparathyroidism.[18,40] Yet 6 cases reported previously and 2 presented here have had osteitis fibrosa cystica, kidney stones, elevated serum calcium, or a combination of these abnormalities usual in parathyroid hyperfunction. It would appear sound to conclude that while not the most common source of hyperparathyroidism, oxyphil adenomas can exhibit hyperfunction. Assumption of the existence of a small, hidden, active chief cell or water-clear cell adenoma seems unjustified. However, in agreement with the belief that the oxyphil is an involuted cell form,[40] it may be that its adenomas are relatively quiescent or "burned out" when removed and examined.

For comparison, 28 adenomas of more common parathyroid types were re-examined microscopically. Of these, 25 were mainly of chief

cell type, and 3 of water-clear cells. No oxyphils were identified in 13, rare scattered oxyphils in 11, and from a few to moderate numbers in 4 adenomas.

One unusual tumor of the parathyroid region was found in a man, 57 years old, suffering from slight difficulty in swallowing and desire to clear his throat. At operation 110 gm. of colloid goiter with multiple nodules was removed. A mass 1.5 cm. in diameter was resected from the right lower thyroid pole, and considered to be a parathyroid adenoma. A normal parathyroid gland was removed also. On the day of operation serum calcium was 9.2 mg. per cent; phosphorus, 4.1 mg. per cent; and phosphatase, 3.8 Bodansky units. There was slight postoperative tetany, with calcium falling to 5.6 mg. per cent. Microscopically, the 1.5 cm. nodule proved to be a granular cell myoblastoma (Fig. 7).

Myoblastoma cells differ from parathyroid oxyphil cells in their irregular shapes and larger size. Prominent eosinophilic cytoplasmic granules of the myoblast cells are characteristic. Adjoining cells have the cross striations characteristic of voluntary muscle. Eosinophilic cells occur also in other cervical neoplasms, particularly in carotid body tumors and Hürthle cell adenomas of the thyroid gland. Microscopically the pattern of the carotid body tumor is vascular and glomangioid, accentuated by silver stains. The perivascular pink cells are quite irregular in size and shape, with clear nuclei, prominent nucleoli, and little resemblance to epithelium. Hürthle cell adenomas are mostly sharply encapsulated, relatively large intrathyroid tumors. Histologically the Hürthle adenoma pattern is relatively organoid or acinar, contrasting with the pavement-like polygonal parathyroid oxyphil cells. Hürthle cells are larger with more prominent vesicular nuclei. Colloid is more easily found in Hürthle cell tumors, and scanty in parathyroid adenomas.

Discussion

One objective of this presentation has been to indicate that oxyphil parathyroid adenomas may occur with hyperparathyroidism. Other such tumors had no evident function, although some gave hints of antecedent activity by slight hypercalcemia or kidney stones. They occurred predominantly in women (20 of the 25 cases in which the sex was stated). All but 5 patients were over 45 years old. Because of their relatively small size, oxyphil adenomas are not often discovered except by careful surgical or post-mortem exploration, but are probably not rare.

Based upon our experience, we believe that persons with oxyphil parathyroid adenomas frequently show indications of more generalized

TABLE II
Abnormal Parathyroid Glands in Hyperpituitarism

Author	Sex	Age	Size of parathyroid glands	Parathyroid glands, microscopic findings	Pituitary body	Other findings
Erdheim[1] (1903)	M	42	RU 1.2 x 0.5 x 0.3 cm. LU 0.8 x 0.8 x 0.3 cm. RL 1.1 x 0.6 x 0.4 cm. LL 1.7 x 0.6 x 0.3 cm.	3 hyperplastic	Eosinophil adenoma (acromegaly)	Aortic insufficiency
Claude and Baudouin[19] (1911)	F	51	(5 or 6 times normal)	Hyperplasia, oxyphil cells and colloid	Eosinophil adenoma (acromegaly)	Thyroid and adrenal hyperplasia
Schmorl[4] (1912)	F	47	(Enlarged)	Oxyphil and chief cells	Basophil adenoma (Cushing)	Osteitis fibrosa cystica, hirsutism, obesity
Carnot, Rathery, and Dumont[20] (1913)	F	58	0.8 x 0.3 cm. 0.8 x 0.3 cm. (3 times normal)	Hyperplasia, chief and oxyphil cells	Acidophil adenoma (acromegaly)	Splanchnomegaly
Molineus[5] (1913)	F	48	1.8 cm. 2.3 cm.	Hyperplasia and oxyphil adenomas	Basophil adenoma (Cushing)	Scar of peptic ulcer, thyroid gland enlarged, obese
Harbitz[21] (1915)	M	75	1 x 1.2 cm. 2 x 2.5 cm.	Multiple adenomas	Eosinophil hyperplasia	Splanchnomegaly
Josefson[22] (1915)*	M	26	6 x 4 x 2.5 cm.	Adenoma, oxyphil(?)	Chromophil adenoma (acromegaly)	Splanchnomegaly
Raab[23] and Kraus[24] (1924)†	M	31	Together, 9.16 gm.	Large nests of oxyphil cells	Basophil adenoma (Cushing)	Obese
Cushing and Davidoff[7] (1927)	M	52	1 x 0.8 x 0.5 cm.	Hyperplasia	Eosinophil adenoma (acromegaly)	Adenomas of thyroid gland and adrenal cortex
	M	35	0.9 cm. 0.4 cm.	Chief cell and oxyphil adenoma; oxyphil adenoma	Eosinophil hyperplasia (acromegaly)	Adenomas of thyroid and adrenal glands, and pancreas
Lloyd[25] (1929)	F	22	LU 0.3 cm. RL 1.5 x 0.4 x 0.3 cm. LL 1.7 x 1.0 x 0.8 cm.	Negative Hyperplasia of oxyphil and clear cells	Eosinophil adenoma (acromegaly)	Pancreatic islet adenomas

Hadfield and Rogers[8] (1932)	M	51	8 x 4 x 2.5 cm.	Chief cell adenoma	Enlarged (acromegaly)	Kidney gravel, thyroid adenomas
Hoff[26] (1934)	M	16	Generalized enlargement	Not given	Basophil adenoma	Osteitis fibrosa cystica, calcinosis
Hora[27] (1935)‡	F	38	Enlarged, RL 5 times normal	Not given	Basophil adenoma (Cushing)	Adrenal cortical hyperplasia and adenoma
Minciotti[28] (1935)‡	F	38	Not given	Hyperplasia	Non-granular adenoma (Cushing)	Osteoporosis; adrenal, thyroid hyperplasia
Lawrence and Zimmerman[10] (1935)	M	44	0.3 cm. 0.3 cm.	Adenoma, atrophic (type?)	Basophil adenoma (Cushing)	Adrenal adenomas, dissecting aneurysm
Kalbfleisch[29] (1937)	M	23	RU, LL, RL: "pea sized" LU larger	Hyperplasia, chief cell and oxyphil (?) adenomas	Invasive chromophobe adenoma	Obesity, 5 pancreatic islet adenomas
Franck and Hjerrild[30] (1937)	F	63	RL 4.5 x 2.5 x 1.5 cm. 3 x 1.5 x 1 cm. LL 2.0 x 0.8 x 0.6 cm.	Bilobed chief and clear cell adenoma Clear, chief, and oxyphil cell adenoma	Basophil hyperplasia	Osteitis fibrosa cystica, thyroid adenoma, fibrosed ovaries
Gerstel[31] (1938)	M	36	RL "egg size"	Chief cell adenoma	Eosinophil adenoma (acromegaly)	Pancreatic tumor, nodular adrenal and thyroid glands
Black and Ackerman[18] (1950)	M	60	Sup. mediastinum, 4 x 3 x 3 cm.	Chief cell adenoma	Eosinophil adenoma	Pancreatic islet adenomas; nodular adrenal and thyroid glands
Sprague *et al.*[32] (1950)	M	15	RL	Chief and transitional cell adenoma	Not examined (clinically, Cushing)	Adrenal cortical and thymic hyperplasias, thymic adenoma

* Cited by Cushing and Davidoff.[7]
† Cited by Cushing.[33]
‡ Cited by Kessel.[34]

metabolic disturbances. Pregnancy has been claimed as a condition in which pituitary activity favors initiation of parathyroid hyperfunction.[38] Peptic ulcers, now regarded by some as by-products of pituitary adrenocorticotropin overproduction following stress, also were seen in our cases and are occasional precursors of other types of parathyroid disease. In so-called primary parathyroid hyperplasia, clinical and metabolic studies have indicated possible participation of pituitary hormones.[41] Parathyroid hyperplasia and adenoma formation secondary to anterior pituitary stimulation have been tabulated in the attested cases of acromegaly and Cushing's disease. Other less clear-cut hyperpituitary states, including some cases of Morgagni-Stewart-Morel syndrome,[35] also have shown evidence of hyperparathyroid function.

Since acromegaly, Cushing's pituitary basophilism, and less specific anterior lobe hyperplasias are all implicated, the cellular source of the stimulus cannot be identified accurately.[39] However, the cases collected indicate human pituitary-parathyroid gland relationships more intimate and important than those suggested by animal experiments.[42]

The 6 new cases reported had no characteristic clinical endocrine stigmata, but rather a variety of diseases and hyperplastic states such as diabetes mellitus, endometrial hyperplasia, ovarian cortical stromal hyperplasia, adrenal cortical adenoma or carcinoma, hypertension, and nodular goiters with hyperthyroidism. Various workers have considered anterior pituitary stimuli important in the development of each of these conditions.

As has been repeatedly pointed out, no satisfactory criteria are available to distinguish with certainty between adenoma and localized hyperplasia of the parathyroid gland. Suggestive indications of a benign neoplasm include the localized homogeneous character of growth and giant cells of tumor type. Nodular hyperplasia has qualities merging with adenoma in the parathyroid gland as in adrenal and pituitary glands. This may indicate that in these tissues such morphologic distinctions are of only secondary importance.

If one accepts the tenet that all parathyroid cells are of a single type, with different appearances in varying functional states, oxyphil cells seem most comparable to involuted forms,[40] such as Hürthle cells of the thyroid gland and onkocytes of salivary glands. Oxyphil adenomas in general are removed in a condition of waning or spent function. They are considered to connote likelihood of past or present hyperparathyroidism, and to be morphologic evidence of anterior pituitary hyperactivity of varying type, degree, and duration. Besides the production of parathyroid adenomas, pituitary stimuli appear to

have initiated concurrent pathologic changes in various other susceptible tissues. Other etiologic factors of major importance doubtless exist in some parathyroid adenomas, and in the absence of a better understanding of the hormones involved, the suspected causes and effects remain unproved.

SUMMARY

Six cases of parathyroid oxyphil adenoma are reported, in addition to 25 collected from the literature. Eight patients had osteitis fibrosa cystica and evident hyperparathyroidism. Usually oxyphil adenomas appear to be a less active, involuted type of parathyroid growth. Evidence collected from the cases presented and the literature shows that anterior pituitary hyperfunction may contribute to parathyroid hyperplasia or adenoma formation. Some parathyroid adenomas occur as part of a polyglandular endocrine syndrome. Human pituitary-parathyroid gland relationships appear to warrant an increased emphasis.

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[*Illustrations follow*]

DESCRIPTION OF PLATES

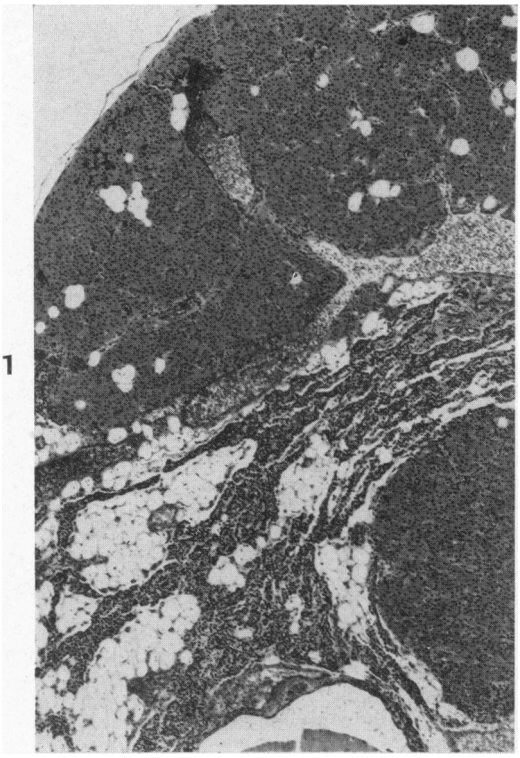
PLATE 95

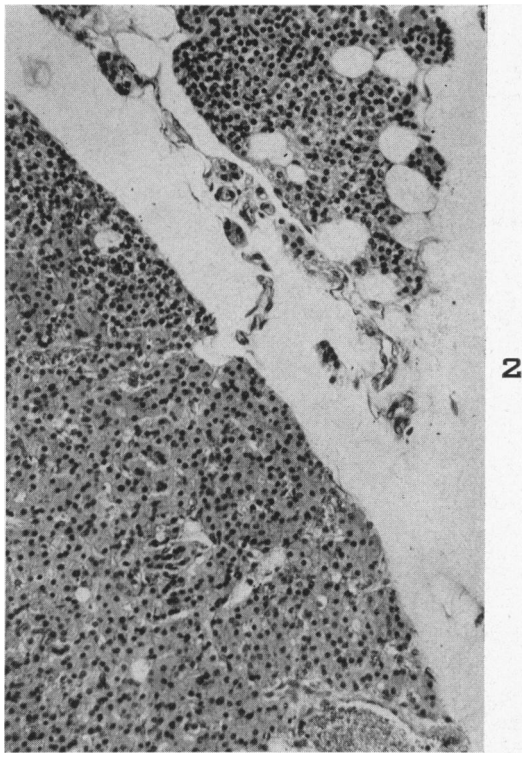
FIG. 1. Case 1. Two adenomatous oxyphil parathyroid nodules. Aside from one small cyst, the parathyroid glands were otherwise normal. Hematoxylin and eosin stain. × 50.

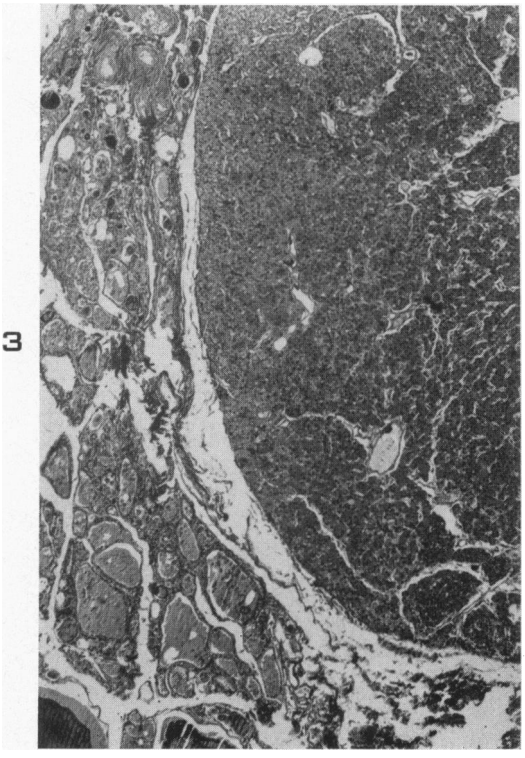
FIG. 2. Case 2. Portion of a solid encapsulated oxyphil adenoma of the parathyroid gland. Hematoxylin and eosin stain. × 150.

FIG. 3. Case 3. Intrathyroid parathyroid oxyphil adenoma. Hematoxylin and eosin stain. × 50.

FIG. 4. Case 4. Characteristic interwoven cords of large cells with granular eosinophilic cytoplasm, from an oxyphil parathyroid adenoma. Eosin and methylene blue stain. × 300.







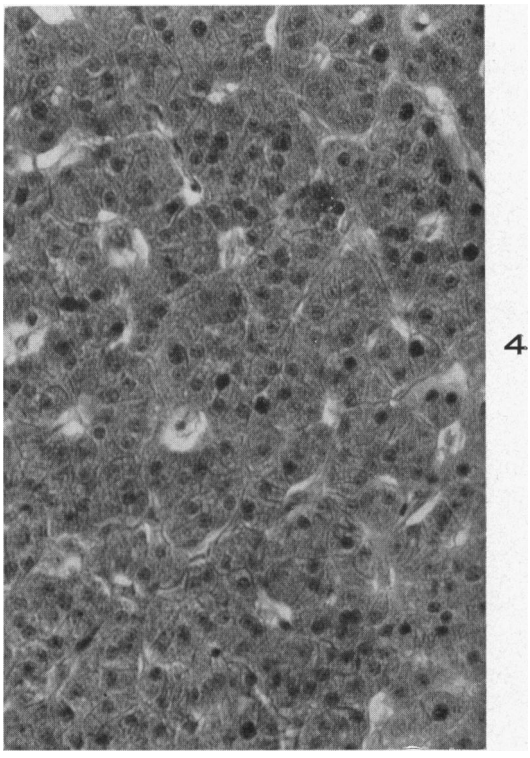


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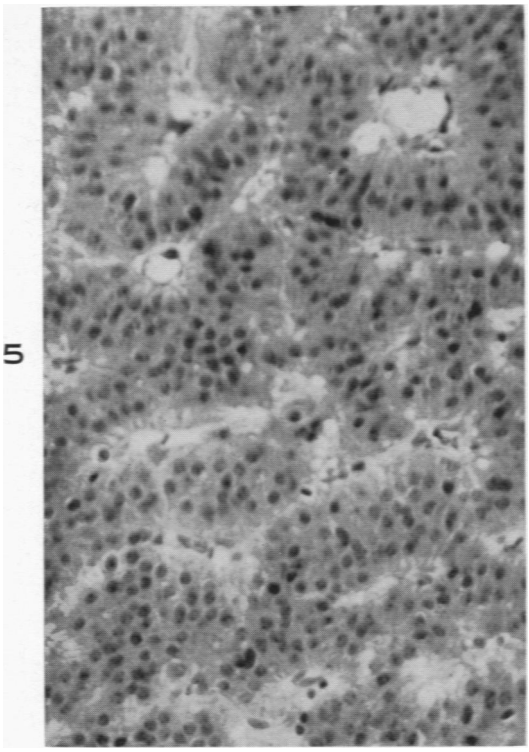
PLATE 96

FIG. 5. Case 5. Solid oxyphil adenoma of the parathyroid gland. Eosin and methylene blue stain. × 300.

FIG. 6. Case 6. A part of the parathyroid adenoma demonstrating intermingled oxyphil and chief cells. No clinical hyperparathyroidism was observed. Hematoxylin and eosin stain. × 300.

FIG. 7. Part of the granular cell myoblastoma removed from the parathyroid region. Cytoplasmic granularity and suggestive striations are present. Cells are much larger than parathyroid oxyphil cells. Hematoxylin and eosin stain. × 300.



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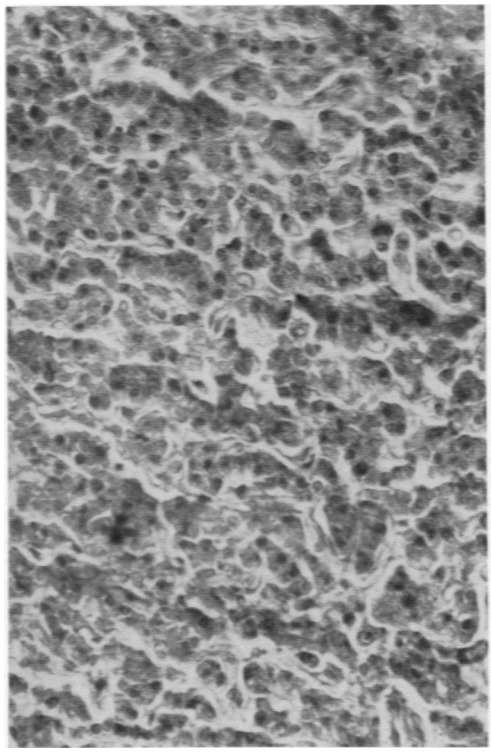

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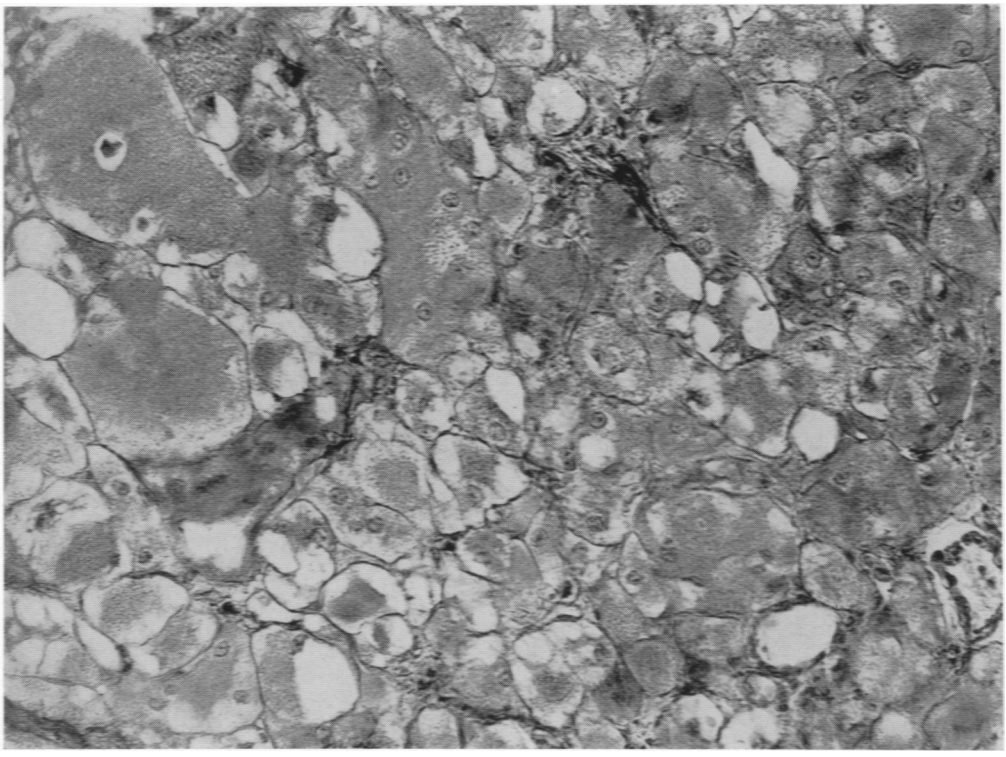

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