TABLE IV

Results of treatment of cases with achlorhydria

Type of anæmia	Number of cases	On admission result of histamine injections	At time of discharge		
			Free acid		No free acid
			Sine histamine	*Cum* histamine	
(*a*) Ancylostome	11	No free acid .. 7 Free acid .. 4	1 2	3 2	3 0
(*b*) Macrocytic	4	No free acid .. 2 Free acid .. 2	1 1	0 1	1 0
(*c*) Hypochromic other than (*a*) ..	5	No free acid .. 4 Free acid .. 1	0 1	0 0	4 0
TOTAL ..	20	No free acid .. 13 Free acid .. 7	6	6	8

curve was normal from the beginning, despite profound anæmia.

The examples below show that cases which commenced and remained achlorhydric and histamine-fast responded to treatment with iron just as rapidly as those with free hydrochloric acid in abundance.

Date	ACHLORHYDRIA		NORMOCHLORHYDRIA	
	Thambiah		Kannan	
	Red blood cells in millions	Hæmoglobin, per cent	Red blood cells in millions	Hæmoglobin, per cent
3-5-39	0.87	12	..	..
11-5-39	1.5	20	1.24	19
14-5-39	2.13	32	1.38	20
17-5-39	3.0	55	2.16	42
21-5-39	3.0	55	2.6	42
24-5-39	3.0	55	2.7	49
27-5-39	3.0	57	2.76	51
31-5-39	3.2	61	2.81	62
3-6-39	3.58	75	2.86	68
7-6-39	4.16	81	3.35	73
10-6-39	4.32	87	3.4	75
14-6-39	4.35	95	3.6	82
17-6-39	5.0	97	3.7	85
21-6-39	5.0	97	4.16	90
24-6-39	5.0	105	4.2	90
28-6-39	5.5	105	4.32	100
1-7-39	5.22	105	4.72	100
5-7-39	5.5	109	5.0	100

The series includes 4 cases of typical Witts' anæmia with severe koilonychia, no ancylostomiasis and histamine-fast achlorhydria. All responded well to iron.

The macrocytic anæmias all responded to marmite and 6 out of 7 were achlorhydric on admission in contradistinction to macrocytic anæmias which have been described in other parts of India with normal hydrochloric acid secretion.

A further series will be reported in more detail as opportunity presents but it is obvious that:—

(*a*) histamine-fast achlorhydria is a condition from which recovery may be made,

(*b*) the condition is not uncommonly found with microcytic ancylostome anæmia,

(*c*) high-dosage therapy with iron can be carried out efficiently in the absence of hydrochloric acid.

Our thanks are due to Professor Mannady Nayar and the staff of the Biochemistry Department of the Madras College for fractional-test-meal reports.

ABNORMALITIES OF THE SUPRARENAL GLAND AND ADDISON'S DISEASE

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THE study of the adrenal glands presents features which are of more than ordinary interest. The peculiar composition of the organ, consisting of two parts, which are entirely different embryologically, and moreover, the uncertain and incomplete knowledge of its functional activities claim continued study. Whatever little knowledge we possess on this subject is derived from pathological observations rather than from physiological experiments (Boyd, 1938). In view of the above facts we are recording a few abnormalities of the

adrenals that we have observed in the study of about two thousand autopsies from the year 1919 to March 1940. The following table shows the results of our investigation :—

TABLE

Total number of autopsies studied	Total number of suprarenal abnormalities	Nature of abnormalities in the gland	Total number of cases showing extensive tuberculous lesions in the body
2,007	16	(*a*) Tuberculosis .. 2 (*b*) Tumour— primary .. 1 secondary .. 1 (*c*) Hæmorrhage .. 2 (*d*) Congestion .. 8 (*e*) Actinomycosis— secondary .. 1 (*f*) Degeneration and hæmorrhage .. 1	352

From the above number the following seven cases are described in detail in consideration of the rare and interesting nature of the lesions.

Case 1.—M. B., female, aged 30, was admitted into the hospital on 9th August, 1938, in the following condition—

The patient complained of intense epigastric pain which was of about four months' duration. On examination pulse rate was found to be 170 per minute with very feeble volume. There was rigidity and tenderness of the abdomen especially over the pelvic region. A hard and tender mass was felt in the right iliac fossa. She died within a few hours of admission and an autopsy examination, which was done the next day, showed the following:—

A pint of blood-stained purulent fluid in the peritoneal cavity. Omentum was extensively adherent to the pelvic organs; tubo-ovarian masses were seen on both sides with extensive adhesions and fibrinous deposits. These masses, when cut, showed frank pus which yielded *B. coli* on culture. A tumour mass was seen above the left kidney. The other regions showed no marked change. Opinion as to the cause of death was peritonitis following ruptured suppurative tubo-ovarian mass. The tumour in the kidney region was found to arise from the left suprarenal gland, and it was globular in shape and pinkish in colour, somewhat cystic in feel and it measured 12½ by 6 inches. The tumour completely replaced the adrenal tissue of which a strip of cortical portion could be seen at the periphery of the tumour. On cutting through the mass the tumour was found to consist of partly solid and partly soft tissues which were of different colour at different places—red, hæmorrhagic, pale, brown and dirty greenish black (plate VI, figure 1). There was no fluid within the tumour. The entire growth was surrounded by a fibrous capsule which separated the new growth from the scanty remnant of the cortical portion of the adrenal gland.

Microscopical anatomy of the tumour—(hæmatoxylin-eosin stain and Mallory's phosphotungstic acid stain)—the growth consisted mainly of compact masses of large ganglionic cells with very little intercellular substance (plate VII, figure 2). Nerve fibres and intercellular neuro-fibrils were scanty. On higher magnification (plate VII, figure 3) the ganglionic cells were well seen; the nuclei were usually seen to be placed at the periphery and binucleated cells were also present. The nucleoli were distinct. Most of the cells showed definite vacuolations in their cytoplasm giving a foamy appearance; evidently this is a degenerative process which has set in within the tumour. No other abnormal finding in any other region. Right suprarenal gland was normal.

Commentary.—The structure of the tumour was of a ganglion neuroma. Tumours arising from the sympathetic nervous system are not common (Geschickter, 1935) and in the reported cases the tumours had their origin in the central nervous system, cranial nerves and their roots and ganglions, neck, thorax, alimentary tract, suprarenals, retroperitoneal region, pelvis and the peripheral nerves. Ganglion neuromas from the suprarenals are still rarer. McFarland (1931) collected about one hundred cases of this type of new growth of which only thirteen had their source in the adrenal gland. These tumours usually run a benign course although rarely they may take a malignant turn. Extensive destruction of the adrenal substance by neoplasm may give rise to Addison's disease syndrome. In Guttman's (1930) series of 566 cases of Addison's disease only 1.2 per cent was due to a neoplastic condition of the suprarenal. In this case there was practically no suprarenal substance left excepting a small strip of cortex, which was for practical purposes devoid of any function. Though the destruction was extensive there were no symptoms of suprarenal failure, probably because the right suprarenal was healthy but it showed no compensatory hypertrophy.

Case 2.—J. H., male, aged 51, admitted into the hospital on 2nd August, 1939, with the following complaints:—

(1) Generalized weakness and lassitude, progressive anæmia and loss of weight.

(2) Vomiting after meals—duration four months.

(3) Pain during swallowing behind the sternum and in the epigastrium, left lumbar and left iliac regions.

History.—About a year prior to his admission, the patient was suffering from 'acidity' and he felt he was getting weaker. About eight months back he felt difficulty in swallowing but took no notice of it. Since four months he started bringing up all food immediately after eating and about that time he began to feel occasional painful sensations in the left iliac region and chest.

Condition on admission.—Poorly nourished, moderately built, looked markedly anæmic. There was no jaundice and the temperature was normal; P/R = 80/20; blood pressure—systolic 115, diastolic 70.

EXPLANATION OF PLATE VI

Fig. 1. *Case* 1.—Exact size of tumour (cut surface). Note the different colours at different areas and a small rim of suprarenal structure at the periphery.

Fig. 6. *Case* 3.—Both suprarenals showing the hæmorrhagic appearances of the organs. The pale areas are composed of degenerated glandular elements.

Fig. 11. *Case* 6.—Suprarenal glands showing the gross appearance; both the glands have been partially cut open to show yellowish-grey nature of the necrotic change.

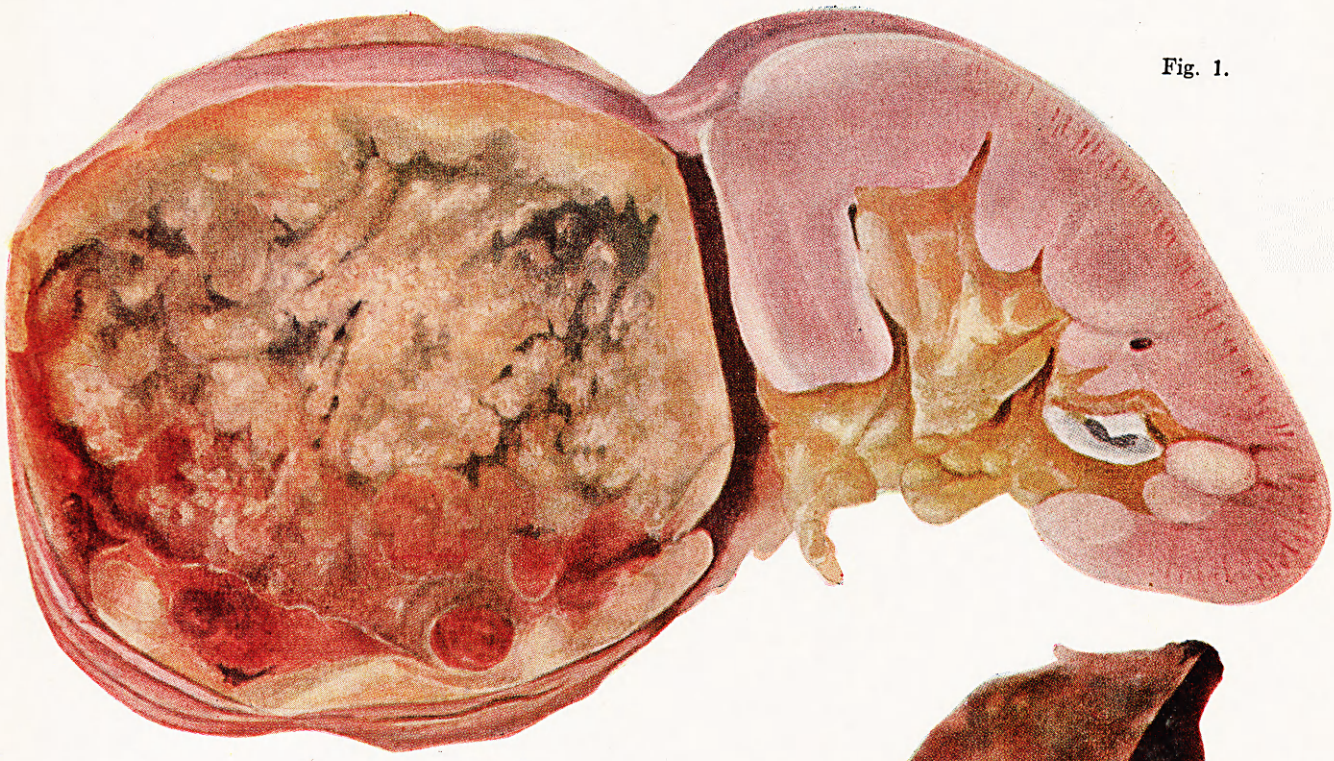

Fig. 1.

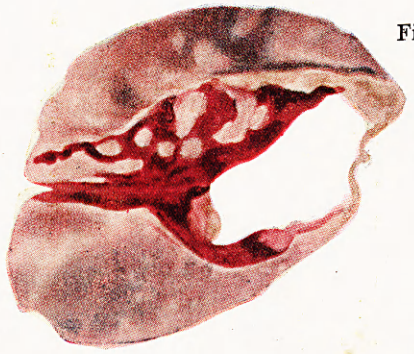

Fig. 6*a*.

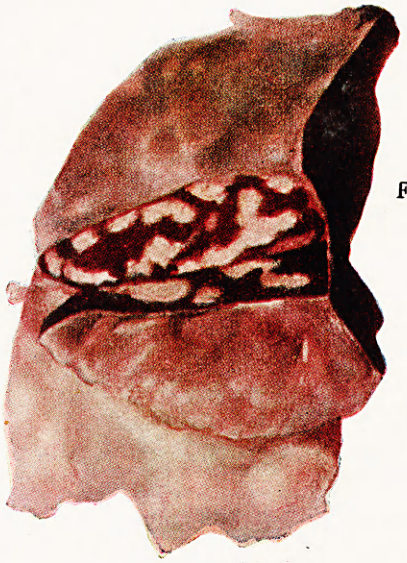

Fig. 6*b*.

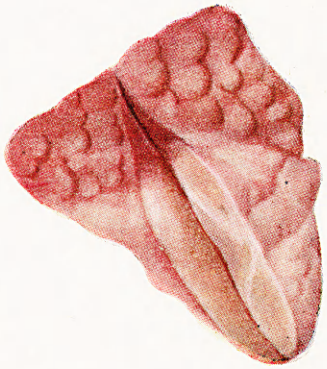

Fig. 11*a*.

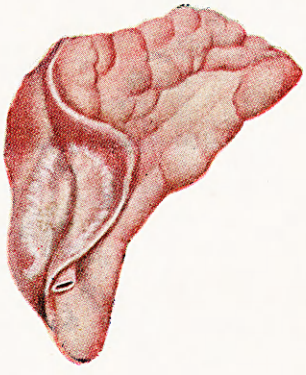

Fig. 11*b*.

After admission.—X-ray examination showed a new growth of the cardiac end of the stomach. A laparotomy was performed and a huge hard growth with uneven surface was found occupying the fundus and body of the stomach.

The patient died on 7th September, 1939, and a partial post-mortem was done to explore the abdominal cavity. Besides the gastric growth a number of huge masses were seen within the peritoneal cavity. These were removed and on examination showed extensive neoplastic involvement of the stomach. A mass 3 by 3½ inches was seen at the hilum of the right kidney but separate from it; another mass the size of a ping pong ball was seen just below the greater curvature of the stomach near the pylorus, but quite separate from it. A third mass, measuring 3 by 2 inches was noticed just above the left kidney (plate VIII, figure 4). On histological examination the gastric growth showed the structure of an adenocarcinoma; masses below the stomach and near the right kidney showed the structure of lymphatic glands infiltrated with adenocarcinomatous process. The mass just above the left kidney was found to be the left suprarenal, which was extensively infiltrated with the same tumour. On examining a number of blocks from the left suprarenal it was seen that very little of the adrenal structure was left. The entire growth consisted of adenocarcinoma with very scanty adrenal tissue here and there (plate VIII, figure 5).

Commentary.—Gastric carcinoma has a wide metastatic spread but infiltration into the suprarenals is rare (Ewing, 1931). The lymphatic spread of this tumour is obvious from the microscopic picture of the abdominal lymphatic glands but there must have been hæmatogenous spread also because otherwise the suprarenal metastasis cannot be explained. The extensive suprarenal involvement, which has almost entirely replaced the gland structure, must have been of long duration, putting the gland out of function for some time. The right suprarenal was not available.

Case 3.—J. M., male, aged 40, admitted into the hospital on 28th February, 1939, with the following history:—

Asthma for the last 12 years; extreme difficulty in breathing for about 24 hours. On examination signs of cavitation at the apex of the right lung were detected and a diagnosis of pulmonary tuberculosis was made. He died on 6th March, 1939, and the autopsy examination showed bilateral plastic pleurisy and emphysematous condition of both lungs. Right lung showed two cavities at the lower part of the base and miliary tubercles in the whole of the lower lobe. Left lung showed consolidation at the base. Liver was congested and enlarged. Spleen was congested. Both the suprarenals were palpably enlarged, the right one being the larger. No other gross abnormality was detected. The enlarged suprarenals were then studied. They felt hard; on cutting through, the glands were found to be hæmorrhagic looking with pale yellowish areas within (plate VI, figures 6*a* and 6*b*). The consistency was homogeneous.

Microscopical examination.—The normal pattern of the gland was completely disorganized and the cortical and medullary areas could not be differentiated. The gland structure showed marked degeneration; the shadow cells taking only eosin stain (plate IX, figure 7). The degenerated areas were filled with red blood cells. At some areas these hæmorrhages showed signs of organization and at other places isolated islets of atrophied cortical structure could be detected interspersed with masses of red blood cells and young fibroblasts (plate IX, figure 8). No signs of any inflammation nor caseation could be seen anywhere. These changes were more or less in all parts of both the organs.

Commentary.—The case presented a condition of widespread degenerative changes of the glandular structures and its infiltration with blood. These bilateral changes in the suprarenal were found in a case of active pulmonary tuberculosis. The initial change seemed to be essentially a degenerative one with subsequent hæmorrhage into the substance resulting in the gross increase of the size of the organ. Guttman (1930), while discussing the rôle of degenerative changes of the adrenals in the causation of Addison's disease, pointed out the rare possibility of tuberculosis as the ætiologic factor of the degeneration. In the present case, though associated with active pulmonary tuberculosis, the suprarenal itself showed no caseation or any picture to suggest a tuberculous process. Our figure 7 is very similar to some of the pictures produced by Guttman, *viz,* degeneration of the gland substance which is infiltrated with red blood cells but the case reported by Guttman showed the organs to be small and thin.

Case 4.—C. C. P., male, aged 60, was admitted into the hospital on 27th February, 1940, in the following condition:—

Unconsciousness with flaccid paralysis of the right upper and lower limbs, contracted pupils, blood pressure 165/115; pulse 100, respiration 28. A diagnosis of cerebral hæmorrhage was made. A week later the patient died; on post-mortem examination the following findings were made—extensive hæmorrhage in the left corpus striatum, hypostatic pneumonia, hypertrophied left ventricle and marked atheroma of the aorta. The left suprarenal was enlarged about twice the normal size. The right suprarenal was normal. On examination, left suprarenal was found to be purple-red in appearance, the normal cream colour being absent. On cutting through the organ it was found to be frankly hæmorrhagic. Histological examination showed that the structural units were present but there was enormous congestion and at places free red blood cells were found scattered within the gland substance. This hæmorrhagic condition was so marked that in most of the blocks studied the glandular element was seen as islets in the midst of masses of red blood cells (plate X, figure 9). There were no degenerative changes in the adrenal tissue but the usual normal arrangement was lost.

Commentary.—This is a case of senile cerebral hæmorrhage with associated suprarenal hæmorrhage. It seems probable that the factors which were responsible for the cerebral lesion caused the adrenal damage too, *viz,* vascular sclerosis.

Case 5.—A. R., aged 35, male, admitted on 30th March, 1928, with the following complaints:—

(1) Frequent motions, about ten times in 24 hours—duration a month and a half.

(2) Pain in the hypogastric region before and after the motions—the same period.

(3) General wasting.

(4) Fever, daily rise of temperature up to 102°F. for a month.

History.—About a year ago he had an attack of a similar nature, *viz,* loose motions seven or eight times daily for one month, with fever and cough. After a month he got over the attack which recurred after a month and a half and for which he went to hospital. A diagnosis of pulmonary tuberculosis was made. In the hospital he continued to have the loose motions and fever. Blood examination showed secondary type

of anæmia, formaldehyde test was positive. Stool examination showed no significant findings.

He died on 3rd July, 1928, and an autopsy showed advanced pulmonary tuberculosis a short summary of which was published (De and Chatterjee, 1934) but no detailed description of the suprarenal was noted. The left suprarenal gland was found to be grossly involved in a suppurative process. The organ was not much enlarged but appeared nodular. On cutting through the substance it was found to be composed mainly of suppurative material which almost completely filled the gland. On histological examination the characteristic appearances of actinomycotic infection (plate X, figure 10) were seen. Very little gland structure was present. Actinomycotic foci were present in a sinus on the left upper arm and the left kidney showed extensive actinomycotic involvement.

Commentary.—The actinomycotic invasion of the suprarenal gland was a part of the generalized actinomycotic infection accompanying pulmonary tuberculosis. The left suprarenal was evidently completely out of function; the gland did not show any compensatory reaction.

Case 6.—A. S., female, aged 30, was admitted into the emergency ward of the hospital on 1st June, 1938, with the following symptoms:—

Pain in the chest, cough with expectoration and fever; duration four months. She gave a history of previous hæmoptysis.

On examination—the patient was found to be extremely emaciated; temperature 101°F. and pulse rate 110 per minute, respiration 32 per minute. Both the lungs were full of râles and crepitations. Heart sounds were feeble but regular. A clinical diagnosis of pulmonary tuberculosis was made. She took her discharge on 3rd June. On the morning of 5th June she was found in a condition of exhaustion and was picked up by an ambulance and admitted into the hospital. The pulse was almost imperceptible and the patient was in a moribund condition. She died twenty-four hours after her re-admission. An autopsy was performed on 8th June and the following conditions were observed:—

The general appearance showed much emaciation. Both the pleural cavities were obliterated by dense adhesions. Right lung showed no gross abnormality. Left lung showed numerous cavities of varying size, which were distributed throughout the upper lobe. Extensive caseous pneumonic consolidations were found throughout the lower lobe. Left hilar lymph glands were enlarged and caseous. The small intestine at its lower part showed a number of ulcerated areas, tuberculous in nature. Adrenal glands—both the glands were enlarged; nearly half of the right adrenal was replaced by yellowish-white caseous looking areas; the left gland was also extensively involved (plate VI, figures 11*a* and 11*b*) and on section many small caseous foci were seen scattered throughout the organ. Scrapings from the lung and intestinal ulcers showed acid-fast bacilli; scrapings from the necrotic areas of the suprarenals did not show any acid-fast bacilli.

Microscopical anatomy.—The section of the lung showed some areas filled with large mononuclear phagocytic cells, lymphocytes, scanty fibrous tissue network, many neutrophilic polymorphonuclear and red blood cells, and other areas showed at homogeneous caseous areas. Giant cell formation was not seen; intestine—sections showed typical tuberculous type of granulation tissue.

Suprarenal—there were large areas of coagulative necrosis destroying the gland tissue completely. The cortex and the medulla of both the organs were extensively involved with very small isolated islets of cortical substance left (plate XI, figure 12). No giant cells were seen.

Commentary.—The case showed bilateral tuberculous involvement of the suprarenal glands in association with active pulmonary and intestinal tuberculous lesions. Though the destruction was extensive the case showed no obvious picture of adrenal failure.

Case 7.—J. R., male, aged 55, motor driver by occupation, was admitted into the hospital on 22nd February, 1940, with the following complaints:—

(1) Difficulty in breathing—duration four years.

(2) Palpitation—one year.

(3) Inability to walk because of general weakness—one month.

(4) Loss of weight.

History.—About five years back he had an attack of palpitation from which he suffered for about a year. It would come at about ten or twelve days' intervals and would last for about five minutes. After some treatment this trouble was relieved. After a few months of respite he began to experience difficulty in breathing off and on, which gradually increased in severity. Since the last four months prior to his admission the difficulty became very marked and the palpitation also reappeared. Because of his respiratory difficulty, palpitation and extreme weakness in walking and gradual loss of weight, he sought hospital aid. The patient had a past history of syphilis and gonorrhœa for which he was treated. He had several attacks of malaria. There was nothing particular in the family history. He was addicted to opium which he took daily. He had nocturnal sweats which sometimes were rather excessive.

On examination.—A man with an anxious look and gasping. There was slight cyanosis and the extremities were cold. He was orthopnæic and the difficulty was both inspiratory and expiratory in nature. There was epigastric pain and he often felt giddy. There was always a sort of sinking feeling.

Progress in the hospital.—The patient continued to go downhill with three main features, *viz,* extreme tired feeling, marked insomnia and dyspnœa.

Blood report—hæmoglobin 70 per cent, red cells 3,600,000 per c.mm., leucocytes 11,236 per c.mm., polymorphonuclears 74 per cent, lymphocytes 32 per cent, monocytes 4 per cent, eosinophils 2 per cent, sputum no acid-fast bacilli. Blood urea 40 mgm. per cent, N.P.N. 47 mgm. per cent, cholesterol 354 mgm. per cent, chloride 402.7 mgm. per cent, urine urea 0.3 mgm. per cent. The patient died on 22nd February, 1940.

Post-mortem findings: Left pleural cavity.—Recent adhesions at the apex and antero-laterally. Right pleural cavity—normal. Left lung was emphysematous and there were interlobar adhesions present; base œdematous and bronchioles were much dilated. Right lung—voluminous and œdematous. Heart—weight 250 gm. and flabby. Spleen—enlarged with capsular thickening. Liver—congested and blackish looking. Gastro-intestinal tract—no gross abnormality. Brain and spinal cord—no abnormality. Thyroid gland—no abnormality. Right kidney—on section a small necrotic area about ½ by ¼ inch in size was seen in the medulla near the lower pole (plate XI, figure 13). Histological sections from the area showed the typical tuberculous nature of the lesion, *viz,* caseation and giant cell formation (plate XII, figure 14).

Suprarenals.—Both the organs were much enlarged, to the size of 2½ by 2 by ¼ inches with marked nodular surfaces. On cutting through the glands these were found to be entirely composed of cheesy material which at places was seen broken down and a thick, creamy material escaped out of the mass (plate XI, figure 13). Both the glands showed the above picture and the normal glandular appearances were entirely absent. On histological sections—caseation was found everywhere. No giant cells were seen. The normal cortical and medullary structures had completely disappeared and in only one of the blocks a slight cortical element was seen (plate XII, figure 15).

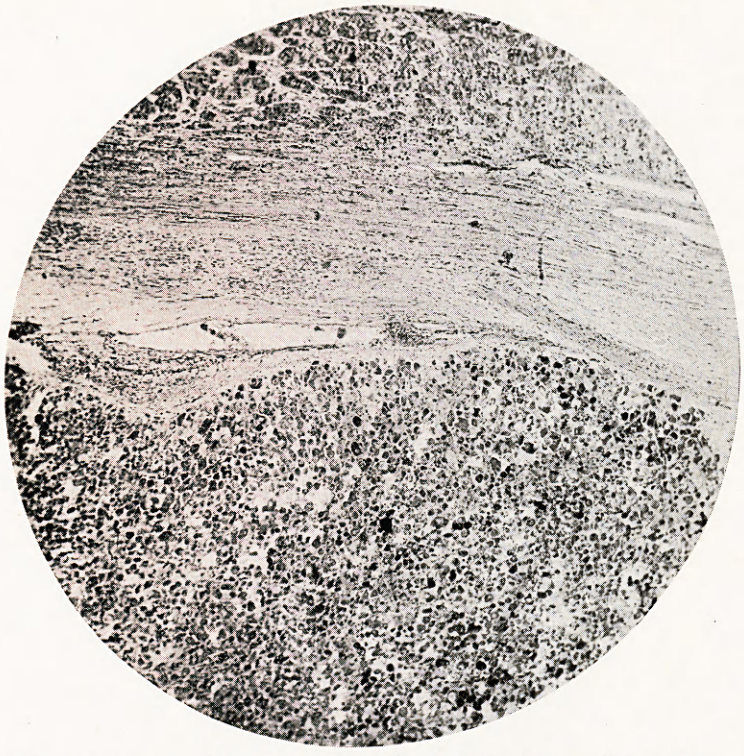

Fig. 2.—*Case* 1. Low power photomicrograph of a section of the tumour. It is composed of ganglion cells and it is encapsulated by fibrous tissue. At one corner the remnants of the normal gland structure is seen. × 175.

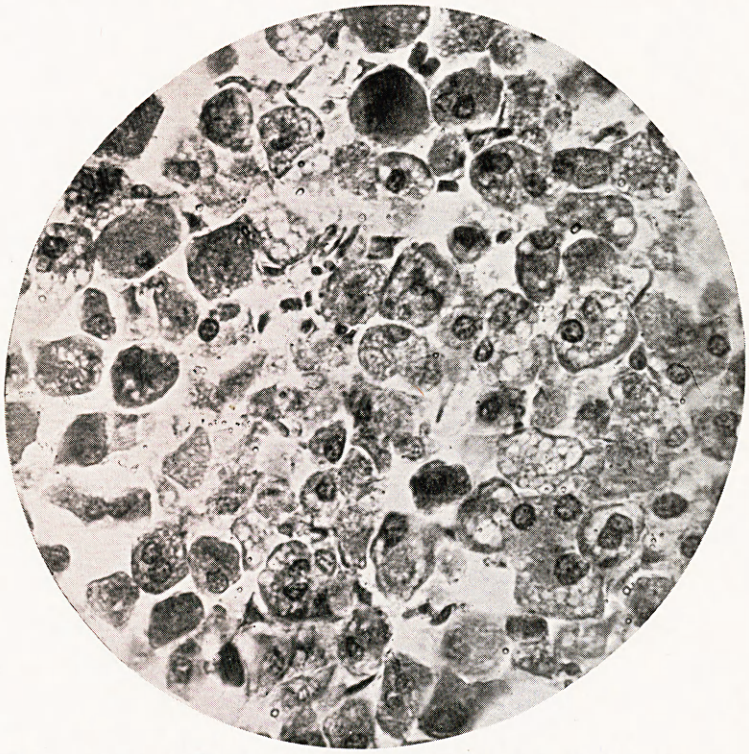

Fig. 3.—*Case* 1. High power photomicrographic view of the tumour cells. Note the double nuclei in some of the cells. The vacuolated appearance of the cells is apparent. × 500.

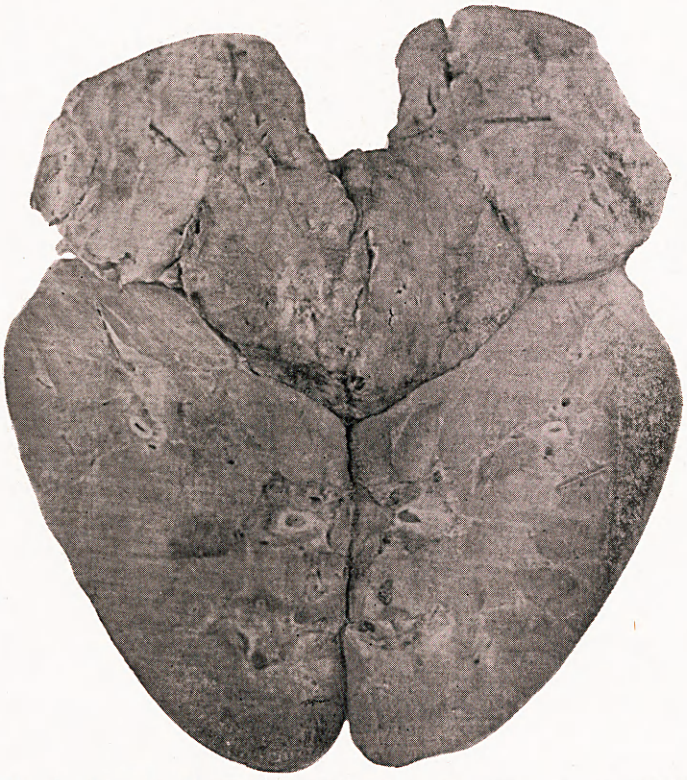

Fig. 4.—*Case* 2. Photograph of the suprarenal tumour with the left kidney. The suprarenal is completely replaced by the tumour mass.

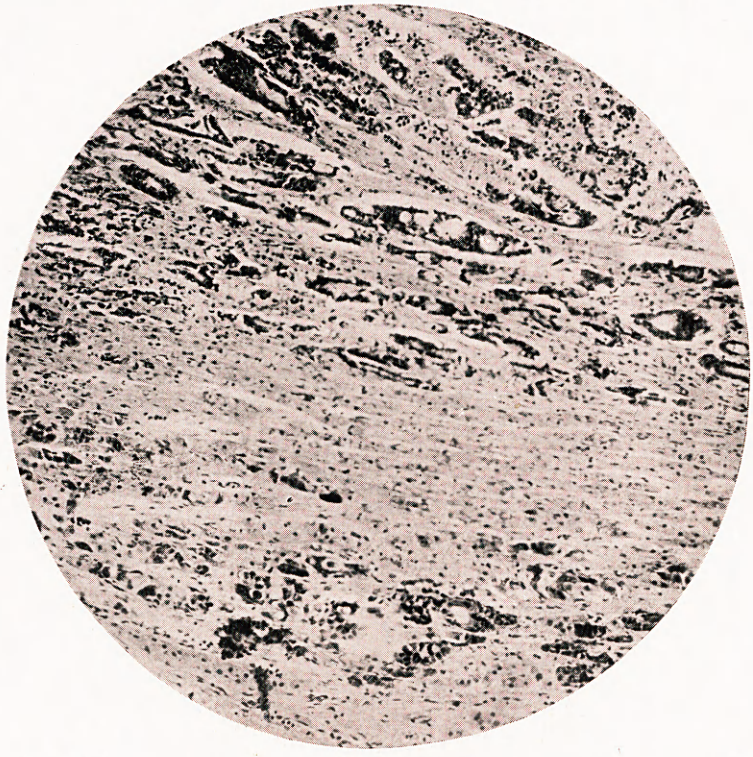

Fig. 5.—*Case* 2. Photomicrograph of the section of the suprarenal tumour. The adenocarcinomatous process is seen extensively infiltrating the gland substance. × 265.

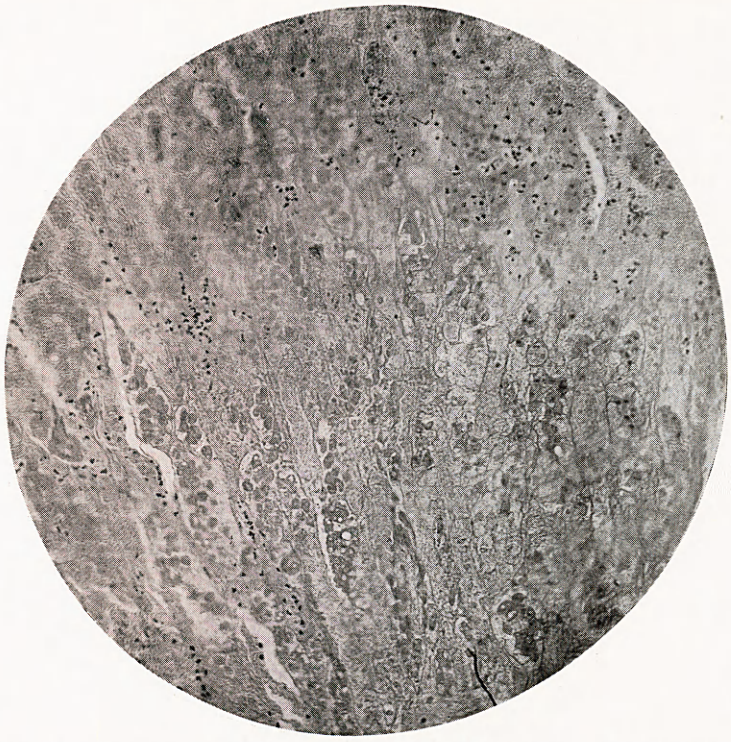

Fig. 7.—*Case* 3. Photomicrograph of a section of the gland well showing the advanced degenerated condition; a few shadow cells are still seen. × 340.

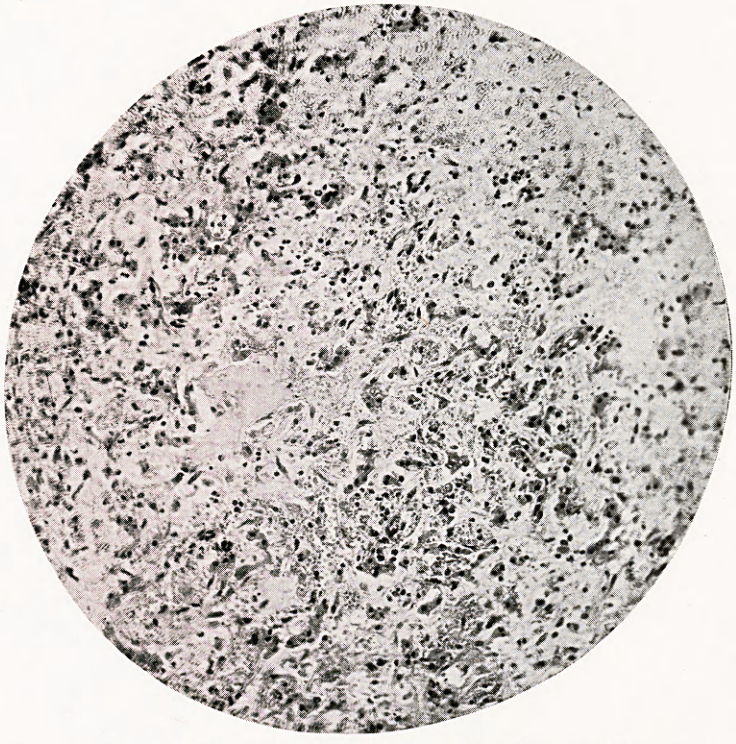

Fig. 8.—*Case* 3. Photomicrograph of a section from another area of the gland showing the islets of remnants of gland substance; masses of red blood cells are present all over and fibroblasts are also seen. × 265.

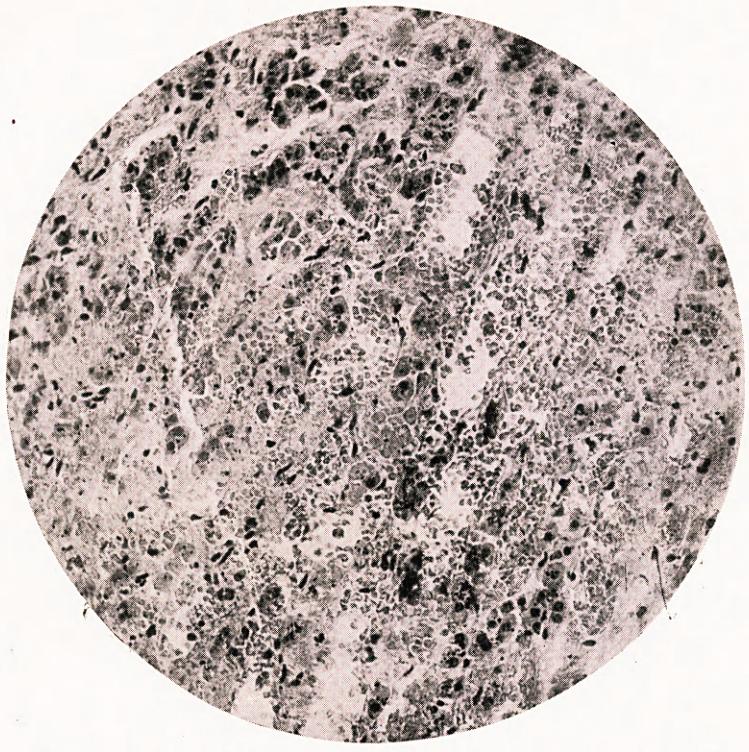

Fig. 9.—*Case* 4. Photomicrographic view of a section of the suprarenal gland showing the congestion and hæmorrhage into the gland substance. × 500.

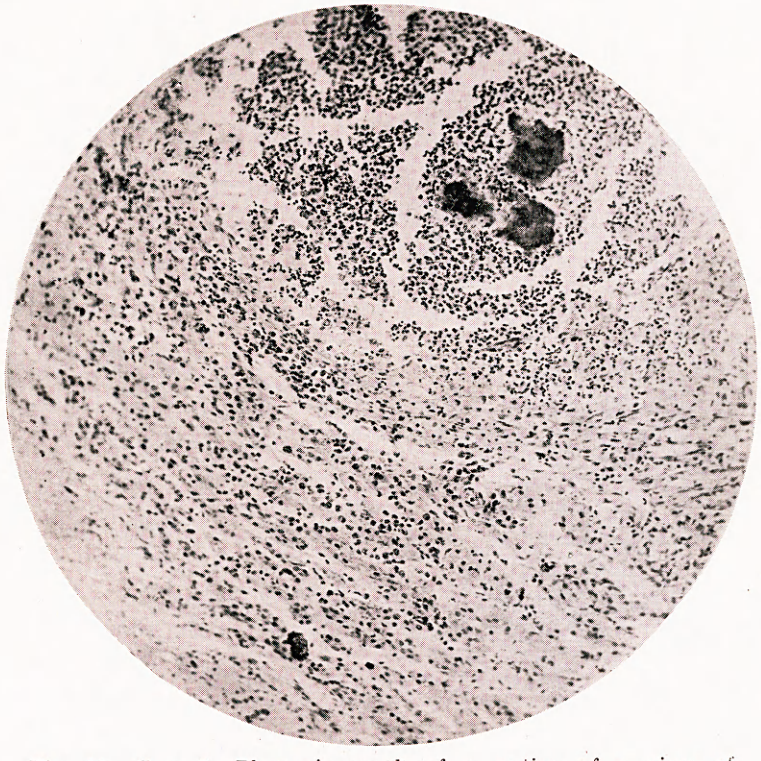

Fig. 10.—*Case* 5. Photomicrograph of a section of a piece of suprarenal gland showing the actinomytic foci within the gland; the leukocytic layer around the bacterial colony is well seen. × 265.

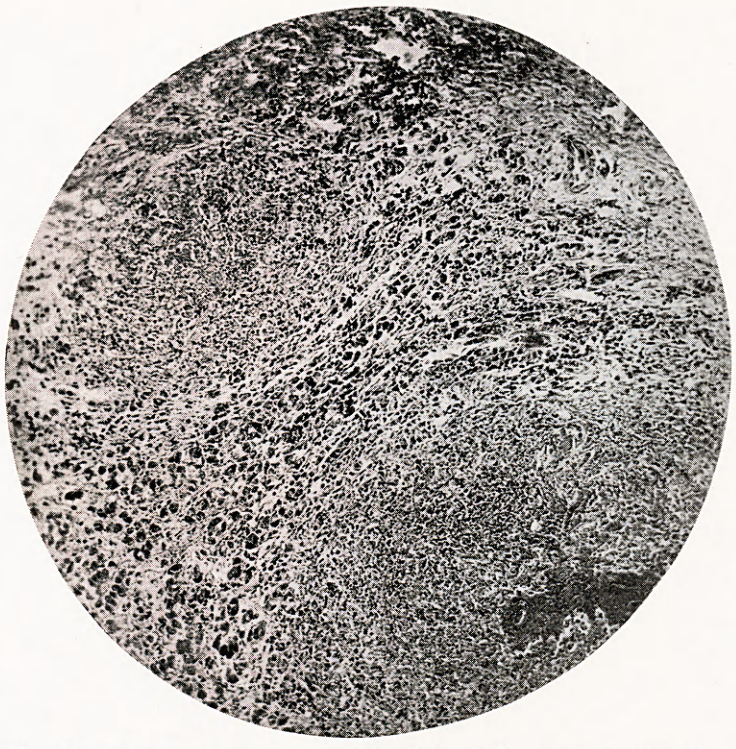

Fig. 12.—*Case* 6. Photomicrographic view of the suprarenal gland showing the extensive necrosis. A few altered gland cells are also seen in the field. × 265.

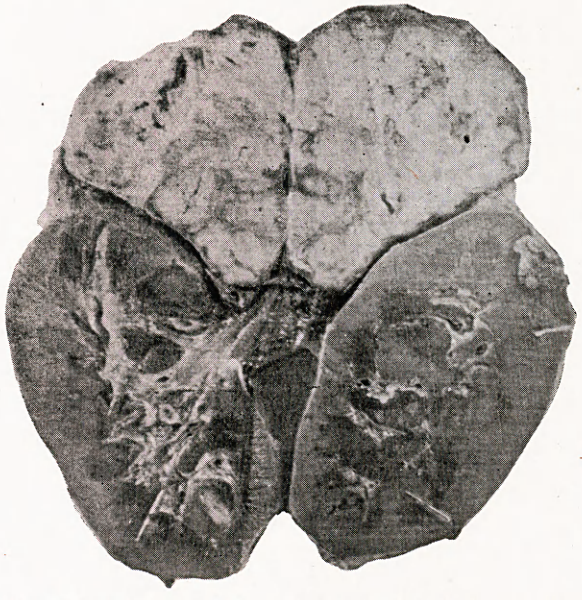

Fig. 13.—*Case* 7. Photograph of the left kidney with the suprarenal gland. In the medullary substance of the lower pole of the left kidney a small necrotic area is seen. The extensive involvement of the suprarenal is apparent. The other suprarenal showed similar change.

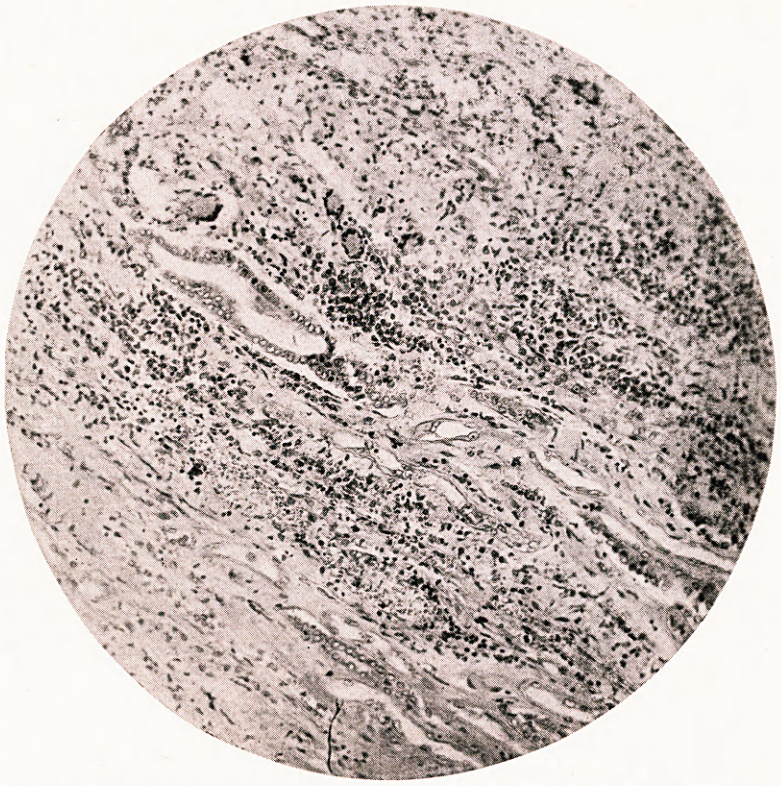

Fig. 14.—*Case* 7. Photomicrograph of the section of the renal tissue from the necrotic area at the lower pole of the left kidney. The typical tuberculous nature of the lesion with giant cells is well seen; some of the renal tubules which have undergone considerable structural change owing to fibrosis may also be seen. × 340.

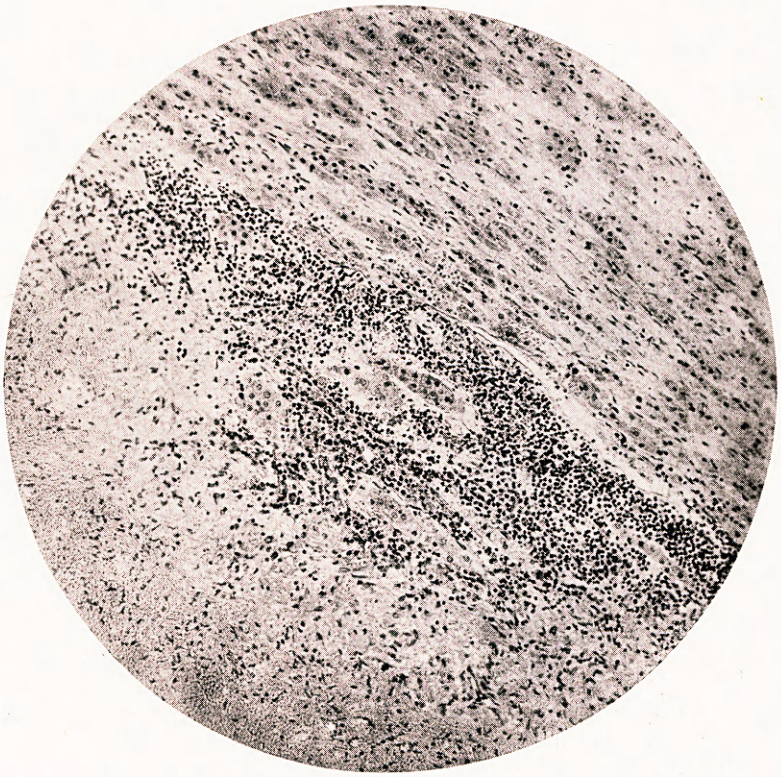

Fig. 15.—*Case* 7. Photomicrograph of a section from the suprarenal gland. Note the caseated area with round cell infiltration. An area of partially degenerated glandular substance is also seen. × 265.

Commentary.—Extensive bilateral tuberculous process was evident. There was no sign of tuberculous lesions anywhere else excepting the focus in the left kidney. Tuberculosis of the suprarenals is always secondary but the primary lesions may or may not be noticeable. The spread is by the hæmatogenous route. Secondary spread from the kidney is extremely rare (Boyd, 1935). In the present case the active lesion in the left kidney is in all probability the primary lesion.

Discussion.—The suprarenal glands are not a common site for disease. Out of two thousand unselected autopsies the organs were found to be diseased only in 16 cases. It is surprising that except in one case (case 7) none of these showed the usual signs of suprarenal failure—the Addison's disease syndrome—the characteristic pigmentation, extreme weakness, gastro-intestinal disturbances and low blood pressure. Unfortunately in the cases under review the blood pressure records were not complete and so no observation can be made on that point. Addison's disease is a rare condition. Diseased suprarenals are not necessarily associated with the Addison's disease syndrome because of the fact that a slight amount of healthy structure may be sufficient for life or an unnoticed aberrant adrenal structure might take up the function. In our series all the seven cases showed complete disorganization of one of the glands and both the glands in three cases but only in case 7 was there suggestion of adrenalin failure—gastro-intestinal disturbance and extreme weakness. Both in cases 6 and 7 bilateral destruction of the gland had existed for a long time yet neither showed the most constant characteristic, *viz,* pigmentation, although cases of Addison's disease are possible without pigmentation (Hadfield and Garrod, 1938, and Guttman, 1930).

Atrophy, tumour, syphilis, degenerative and vascular changes and tuberculosis are the usual causes of Addison's disease of which tuberculosis accounts for 70 per cent. Our table shows 352 tuberculosis cases out of two thousand autopsies (roughly 18 per cent). Out of these 352 cases the tuberculous involvement of the suprarenal occurred only in two cases (about 0.56 per cent). From the figures of different workers Guttman collected the percentages of tuberculosis of the suprarenals in association with pulmonary tuberculosis which ranged from 2 per cent to 5 per cent. In our series the figure is still smaller. Considering the prevalence of tuberculosis in Bengal the incidence of Addison's disease due to tuberculosis would be expected to be higher. Another interesting point is that in Addison's disease due to tuberculosis of the suprarenals, usually there is no manifestation of active tuberculosis anywhere else in the body. In the two cases under review one showed extensive active exudative type of tuberculosis of the lung and intestine and in the other one there was involvement of the kidneys. It is noted that because of the rarity of Addison's disease, unless all the classical signs and symptoms are present it is not surprising that the condition might be overlooked. In both the cases 6 and 7 on autopsy, the bilateral extensive destruction was clear but neither gave any definite indication of the possibility of frank Addison's disease.

With regard to the rôle of the cortex and the medulla in the production of Addison's disease, there were long controversies in the past, but the balance of evidence at present seems to place the cortical portion as mainly responsible for the signs and symptoms, although the part played by the medulla is not altogether insignificant. In our cases no differential study from this point of view is possible because in all instances both the cortex and the medulla have been markedly involved. In case 1 the origin of the tumour was of course from the medulla, but by simple increase of size the cortical part has been extensively involved by pressure effects. It is significant that in unilateral lesions there was no compensatory hypertrophy of the other healthy gland.

Summary

(1) In two thousand unselected autopsies, suprarenal lesions were found in sixteen cases.

(2) Out of three hundred and fifty-two cases of tuberculosis, pulmonary and intestinal, only two cases showed involvement of the suprarenal glands.

(3) A unilateral suprarenal lesion fails to produce Addison's syndrome and extensive bilateral destruction of the glands does not necessarily result in a frank picture of Addison's disease.

(4) Two cases of tuberculosis, a case of secondary actinomycosis, hæmorrhage, degeneration, secondary adenocarcinoma and ganglion neuroma of the suprarenal gland are described in detail.

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