chorionic gonadotrophins in the urine in these patients and lowered plasma hydrocortisone. The eruption started at any time during the pregnancy and was controlled only by corticosteroid therapy except in the one controlled by progesterone mentioned above. They believe the disorder to be due to placental activity.

THE PINEAL GLAND

The pineal body, which Descartes once thought to be the seat of the soul, is beginning to yield some of its secrets and now deserves to be called the pineal gland. Many investigators have tried over the past thirty years to ascribe to it an endocrine function consistent with its acinar structure. Animal experiments involving pinealectomy or the administration of crude extracts of the gland whilst yielding a mass of conflicting data have nevertheless suggested the possibility of a gonadotrophic action. This once seemed to be the cause of the sexual precocity present in some males with pineal tumours, but the phenomenon is now thought to originate from damage to near-by structures in the hypothalamus owing to pressure or direct extension by the tumour.

Recent biochemical researches have shown that the pineal gland contains a group of active substances related to indole—the "indole hormones." These include serotonin, present in highest concentration in the pineal gland, melatonin, and adrenoglomerulotrophin.[1] Melatonin, or N-acetyl-5-methoxytryptamine, has been isolated from bovine pineal glands by A. B. Lerner and colleagues,[2] and is the most potent of all agents known to lighten the colour of frog skin. Melatonin activity has been found in extracts of human pineal glands and urine,[3] but at present there are no clues to its possible role in human physiology and disease.

Adrenoglomerulotrophin (A.G.T.) was first postulated by G. Farrell[4] as a trophic hormone regulating the secretion of aldosterone by the zona glomerulosa of the adrenal cortex. Farrell found that extracts of beef diencephalon stimulated the secretion of aldosterone in decerebrate dogs, and that a large part of this activity could be accounted for by that present in the pineal gland.[4 5] But pinealectomy only temporarily reduced the secretion of aldosterone; within a week it returned to normal, and the usual increase in secretion of aldosterone during sodium depletion was elicited in pinealectomized dogs.[6] Thus it seemed that A.G.T. was being secreted somewhere else in the hypothalamus, not necessarily or exclusively in the pineal gland. A.G.T. has been tentatively identified as 1-methyl-6-methoxy-1,2,3,4-tetrahydro-2-carboline.[7] It is interesting that in structure it closely resembles harmaline, the active agent in caapi, a potion of jungle vine which Peruvian Indians take to induce hallucinations and delusions, and that it derives from serotonin. Synthetic A.G.T. has been shown to stimulate secretion of aldosterone in hypophysectomized dogs.[7] Whatever the role of A.G.T. in controlling secretion of aldosterone—and this is a hotly disputed field—it is certainly not the only trophic factor concerned. J. O. Davis and colleagues[8] found that decapitated dogs responded normally to haemorrhage by increasing their secretion of aldosterone. This is thought to be mediated by angiotensin II secreted by the juxtaglomerular apparatus of the kidney, and now considered to be a major factor in the control of the secretion of aldosterone.

Recently A. Kennedy, D. Kilshaw, N. C. R. W. Reid, and W. H. Taylor[9] reported on a woman of 58 with recurrent thyrotoxicosis and several features of primary aldosteronism associated with an enlarged pineal gland. Biochemically she had hypernatraemia and hypokalaemic alkalosis with adequate urinary output of potassium. Her urinary aldosterone was slightly above the normal range. At necropsy the pineal gland was about three times normal size; the adrenals were normal and without hypertrophy of the zona glomerulosa. Though the evidence of primary aldosteronism is tenuous in this case, and thyrotoxicosis an unexplained complicating factor, it is worth bearing in mind the possibility that pineal over-secretion of A.G.T. might give rise to hyperaldosteronism. Unfortunately, in 15 cases of pineal hypertrophy cited by J. I. Kitay and M. D. Altschule[10] there were no consistent clinical findings and disturbances of electrolyte balance were not reported.

SCHIZOPHRENIA RESEARCH FUND

Mentally ill patients occupy nearly half of the hospital beds in Britain, and the numbers admitted to mental hospitals in England and Wales rose from 54,921 in 1949 to 94,083 in 1958. What is being done to encourage research into mental disease so that this suffering can be prevented and this expensive drain on the funds of the hospital service be reduced?

In 1949 the Mental Health Research Fund was set up by a group of far-seeing doctors and scientists. It has been energetically managed and has sponsored some invaluable research. Between March, 1954, and March, 1962, it distributed £208,310 in the form of grants. But in the year ended March 31, 1962, its income was only £43,685; it had spent £25,186 on fellowships and grants, and had a balance of income over expenditure of £12,054.[1] These are small figures in relation to the numbers of people who are mentally ill. It is true that research into mental disease is supported by other bodies, notably by the Medical Research Council, but is enough being done? Professor K. W. Cross, in a letter to *The Times*,[2] has complained about the anomalous position of medical research in Britain. Mr. Billy Butlin has recently given £30,000 to maintain research on kidney grafting at the Hammersmith Hospital, but, as

[1] McIsaac, W. M., *Cleveland Clin. Quart.*, 1962, 29, 76.
[2] Lerner, A. B., Case, J. D., and Takahashi, Y., *J. biol. Chem.*, 1960, 235, 1992.
[3] —— —— and Heinzelman, R. V., *J. Amer. chem. Soc.*, 1959, 81, 6084.
[4] Farrell, G., *Endocrinology*, 1959, 65, 29.
[5] —— ibid., 1959, 65, 239.
[6] —— *Circulation*, 1960, 21, 1009.
[7] —— and McIsaac, W. M., *Arch. Biochem.*, 1961, 94, 543.
[8] Davis, J. O., Carpenter, C. C., Ayers, C. R., and Bahn, R. L., *1960 Proc. 42nd Meet. Endocrinol. Soc.*, 1960.
[9] Kennedy, A., Kilshaw, D., Reid, N. C. R. W., and Taylor, W. H., *Brit. med. J.*, 1962, 2, 641.
[10] Kitay, J. I., and Altschule, M. D., *The Pineal Gland*, 1954. Harvard University Press, Cambridge, Mass.

[1] Mental Health Research Fund—Annual Report and Accounts for the Year Ended March 31, 1962.
[2] *The Times*, January 1, 1963.
[3] Ibid., January 4.