and for three weeks. During this period each dose is adjusted in terms of response to previous doses—a kind of biological assay. The patient thereby gains a considerable insight into the principles underlying treatment. After the initial three weeks the patient attends the hypertensive clinic—weekly at first and later at longer intervals. Occasional all-day tests are made, but most of the control at this stage is in terms of hypotensive symptoms. Casual blood pressures are taken, but they are of very little use in the control of dosage. Quite high blood pressures may be encountered in out-patients whose blood pressure, with methonium, is satisfactory under home conditions.

Summary

On three and a half year's experience it appears already that the prognosis of malignant hypertension is much improved by effective control over the blood-pressure level by the administration of methonium salts.

The result of treating all of 28 malignant hypertensive patients, including seven with substantial renal insufficiency or advanced heart failure, is that 21 are living and seven dead. The average survival time of those who died is 9.7 months and of those who are still alive 22.8 months. A considerable majority of the survivors are leading productive lives, the men working at their previous occupations and the women doing housework.

In hypertensive patients with retinal grades II and III, taken together, the mortality figures are better in 122 treated cases (16.4%) than in 53 controls (28.2%), but no claim of statistical significance is made.

Objective improvements include: (a) the disappearance in most cases of such changes as papilloedema, retinal oedema, soft exudates, haemorrhages, and, after long treatment, of star-shaped macular figures; (b) the return to work of most patients under 60 presenting with malignant hypertension, cardiac asthma, or congestive heart failure, including in most instances the elimination of the use of digitalis, mersalyl, or substantial salt restriction; and (c) the electrocardiographic improvement and tendency to decrease of heart size. The consistency with which these results have been reproduced during three and a half years leads me to state that less satisfactory degrees of improvement are usually due to inadequacy of the regime used.

In a minority of patients the doses of methonium salts which relieve hypertensive manifestations lead to much discomfort from side-effects. Methods of alleviation which have helped some cases are suggested.

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REFERENCES

- Doyle, A. E. (1953). *Amer. Heart J.*, **45**, 363.
 Keith, M. A., Woolf, B., and Gilchrist, A. R. (1949). *Brit. Heart J.*, **11**, 287.
 Keith, N. M., Wagener, H. P., and Barker, N. W. (1939). *Amer. J. med. Sci.*, **197**, 332.
 Kilpatrick, J. A., and Smirk, F. H. (1952). *Lancet*, **1**, 8.
 McMichael, J. (1952). *British Medical Journal*, **1**, 933.
 Maxwell, R. D. H., and Campbell, A. J. M. (1953). *Lancet*, **1**, 455.
 Morrison, B. (1953). *British Medical Journal*, **1**, 1291.
 Restall, P. A., and Smirk, F. H. (1950). *N.Z. med. J.*, **49**, 206.
 Rosenheim, M. L. (1952). *Proc. roy. Soc. Med.*, **45**, 269.
 Smirk, F. H. (1950). *N.Z. med. J.*, **49**, 637.
 — (1951). *Amer. Heart J.*, **42**, 530.
 — (1952a). *Proc. Univ. Otago med. Sch.*, **30**, 13.
 — (1952b). *Lancet*, **2**, 695.
 — (1952c). *Ibid.*, **2**, 1002.
 — (1953a). *Ibid.*, **1**, 457.
 — (1953b). *N.Z. med. J.*, **52**, 325.
 — and Aitstad, K. S. (1951). *British Medical Journal*, **1**, 1217.
 Wien, R., and Mason, D. F. J. (1953). *Lancet*, **1**, 454.

ORAL CORTISONE TREATMENT OF HYPOPITUITARISM

BY

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In early trials of the therapeutic value of cortisone in hypopituitarism, the hormone was given in large dosage (75 to 100 mg. daily) for periods of about one to three weeks. The results were encouraging, but in some ways not satisfactory (Summers and Sheehan, 1951). Other workers using this dosage observed no beneficial effects (Robertson and Kirkpatrick, 1951; Brown and Greenblatt, 1952). Since then, a much lower dosage of cortisone has been tried for the continued treatment of these patients. Originally the cortisone was given either by injection or by implantation of pellets, but more recently oral therapy has been found just as efficient and far more convenient. Reports of single cases treated for long periods with doses of 12.5 to 25 mg. daily have been published by Thorn *et al.* (1950, 1951), Clark *et al.* (1951), Levy (1952), Perkins and Rynearson (1952), Roberts *et al.* (1952), McCullagh and Schaffenburg (1953), Tardy (1953), Dontigny (1953), Mathisen (1953), and Fourman and Horler (1954). More recently, a series of three cases treated in this way have been recorded by Hart (1953), and two cases by McCullagh *et al.* (1954). All these workers have reported good results from prolonged therapy at low dosage.

A few investigations have been made of the therapeutic value of cortisone in the treatment or prophylaxis of hypopituitary coma (Allott and Simmons, 1951; Sheehan and Summers, 1952; Paniagua *et al.*, 1952; Caughey *et al.*, 1952; Hijmans, 1952; Heyde, 1953). In addition there have been several reported studies of the effects of cortisone administered for very short periods for diagnostic tests or physiological studies, but these are not of direct relevance to the subject under consideration here.

Treatment of Present Cases

The following account concerns four patients with severe hypopituitarism who have been given small doses of cortisone orally over periods of about two years. Three of these were typical examples of severe hypopituitarism due to post-partum necrosis; the fourth was a man with hypopituitarism of unknown aetiology, probably due to some form of atrophy and scarring of the pituitary.

The patients were studied for a preliminary observation period of two to three weeks without treatment, and for a further control period of five weeks, during which 3 gr. (0.2 g.) of thyroid sicum was given daily. No significant effect was noted from the administration of thyroid. (This point merits a short interpolation. The warnings given in almost all textbooks about the dangers of thyroid therapy in patients with hypopituitarism appear to be overstated. Most of these patients tolerate ordinary dosage of thyroid without any recognizable ill effect. From a study of the occasional cases reported in the literature where the administration of thyroid was followed rapidly by coma, it seems probable that the patients were already on the point of developing coma in any event. Nevertheless, when

thyroid treatment is given, reasonable care should be taken. If the dosage is raised too much because of the lack of response, gastro-intestinal upsets and loss of weight can be produced; these two effects are serious in any patient suffering from hypopituitarism.)

After these periods of preliminary study, cortisone was given orally as "cortone" without any other treatment. The initial dosage was 12.5 mg. daily for one to two weeks, followed by 25 mg. daily for two to four weeks. In one patient it was increased to 37.5 mg. for two weeks and to 50 mg. for one week. Subsequently the amount was gradually reduced in all cases to 12.5 mg. daily as a maintenance dose. When the dosage was raised to 25 mg. one of the patients began to complain of insomnia. Another patient slept satisfactorily with this amount but had insomnia when the dose was increased to 37.5 mg. daily; she recovered when it was reduced to 25 mg. again. In all subsequent treatment the dosage was maintained at a level low enough to allow normal sleep.

Clinical Effects of Treatment

In all four cases there was a marked improvement in the general condition within three or four days. The patients became mentally alert and took a normal interest in their surroundings. They had an obvious sensation of well-being, became physically energetic, and were no longer sensitive to cold. Thus they were able to resume all their ordinary activities in the home and outside. The improvement was maintained satisfactorily on a daily dose of 12.5 mg.

The initial period of treatment with 25 mg. produced in all cases a normal capillary flush of the cheeks, a return of axillary secretion, and the occurrence of sweating on exercise. This improvement, once established, was maintained by 12.5 mg. daily. No changes developed in the face or elsewhere suggestive of a Cushing syndrome, even after two years' treatment. An experimental increase of the dosage to 50 mg. daily in one patient caused excessive perspiration.

Two of the female patients had no change in body hair, but the third female patient developed a small amount of pubic hair and some thickening of the head hair after six months' treatment. The male patient, in the course of six months' treatment, developed a slight amount of pubic hair and a slight return of hair on the legs; his beard became stronger, so that he needed to shave daily instead of once or twice a week.

The amenorrhoea was not affected in any way. No exact details are available about the libido of the female patients, but there was a return of a normal degree of pudor. Striking changes were, however, observed in the male patient, who for the previous five years had been completely without libido and had had no erections or other evidence of sexual activity. During the first three weeks of treatment with 12.5 mg. and 25 mg. of cortisone daily there was no obvious change. When he was given 37.5 mg. daily he began to have erections two or three times a week. After discharge from hospital on a maintenance dose of 25 mg. daily he continued to have frequent erections and had coitus two or three times a month, although no emissions occurred.

In two patients the blood pressure remained unchanged, the levels before treatment having been 130/80 and 140/90. In the third patient there was a slight rise, from 125/75 before to 140/75 during treatment. The fourth patient developed an irregularly increasing hypertension. The blood pressure was 130/80 previously and rose slowly during the first year of treatment to levels fluctuating between 150/90 and 180/130; subsequently it gradually returned to its original level.

Two of the patients had a definite increase in weight during several months' treatment; one rose from 104 to 135 lb. (47.2 to 61.2 kg.), and the other from 107 to 130 lb. (48.5 to 59 kg.). The third patient had a slight weight gain, rising from 92 to 96 lb. (41.7 to 43.5 kg.). The fourth patient lost weight during the thyroid treatment, falling from

162 to 151 lb. (73.5 to 68.5 kg.); on cortisone treatment she gradually regained this lost weight, and remained at 160 to 165 lb. (72.6 to 74.8 kg.).

Biochemical Changes

With these small and slowly increasing doses of cortisone there was none of the gross polyuria which follows immediately after parenteral treatment with high dosage. Nevertheless the urine volume slowly increased. Three of the patients had the following mean urine outputs before treatment and while on maintenance dosage respectively: (a) 1,120 and 1,680 ml., (b) 1,150 and 1,730 ml., and (c) 1,100 and 1,900 ml.

Kepler water diuresis tests were made on two of the patients. One whose maximum diuresis on the morning of the test had been 30 ml. an hour before treatment had a maximum of 140 ml. after treatment. The other had a previous maximum hourly excretion of 100 ml.; this increased during treatment to 165 ml. an hour.

It is well known that there is a great increase of nitrogen excretion in the urine when large doses of cortisone are given. This did not occur in the present series with low dosage. In one case the daily output of urea was unaffected by the treatment; in another there was a slight fall in the output of urea and creatinine, possibly in relation to nitrogen storage in the muscles.

There was always an increased output of 17-ketosteroids in the urine. Before treatment the output in the four patients had ranged between 0.2 and 1 mg. a day. On the maintenance doses of 12.5 to 25 mg. daily the urinary excretion of 17-ketosteroids ranged between 2.1 and 2.8 mg. a day. The increased output of 17-ketosteroids expressed in mg. daily corresponds to about 6% of the daily dosage of cortisone.

No significant changes were observed in the serum chemistry. The serum sodium remained at about the low range usual in these patients (280 to 300 mg. per 100 ml.) and the serum potassium fluctuated between 17 and 20 mg. per 100 ml. The serum cholesterol showed the usual wide fluctuations between 105 and 355 mg. per 100 ml., without any recognizable effect of treatment. The blood urea was rather high (commonly about 35 to 65 mg. per 100 ml.), but again there was no constant change related to the administration of cortisone.

There was no very significant alteration in the sensitivity to insulin, but the patients commonly showed some improvement in their response to hypoglycaemia. Several insulin-tolerance tests (using 0.03 unit per kg.) were performed in three of the patients; the mean blood sugars, expressed in mg. per 100 ml., were as follows:

Patient	Minutes:	0	20	40	60	120
A	Before treatment	72	52	37	40	42
	During ..	77	49	49	54	66
B	Before ..	95	67	50	63	65
	During ..	96	61	63	73	74
C	Before ..	102	57	66	73	88
	During ..	86	63	64	68	81

The basal metabolic rate was studied in detail in only one patient. It had been -20% before treatment and continued in the range of -20% to -30% during cortisone treatment.

There was a definite though variable improvement in the anaemia. One patient with R.B.C. 4,100,000 and Hb 80% improved gradually to R.B.C. 4,700,000 and Hb 93%. The second patient, with R.B.C. 3,400,000 and Hb 70%, rose to an average of R.B.C. 4,200,000 and Hb 82%. The third patient with R.B.C. 4,000,000 and Hb 72%, rose to R.B.C. 5,000,000 and Hb 100%.

Effect of Cessation of Treatment

In three of the patients the cortisone therapy was stopped for a period of three months after about nine months' treatment. This produced a gradual but progressive deterioration in the

clinical state, so that at the end of a month they were almost back to the same condition as before treatment. They complained of marked asthenia, lack of perspiration, sensitivity to cold, and low urine output. The female patient who had had some return of pubic hair and thickening of head hair during treatment showed considerable regression of this hair growth after cessation of the cortisone. The male patient noticed that he required to shave very much less often, and his libido and erections had disappeared entirely within about two months. Two of the patients began to complain of rheumatic pains in the knees and ankles about two months after stopping treatment, though there were no recognizable physical changes in the joints.

After the three months without cortisone these three patients were put back on the maintenance dosage of 12.5 mg. daily, as it was clearly necessary for their continued health. Their condition improved rapidly, and within about a week they were as well as they had been when under cortisone therapy previously. All the patients have continued in remarkably good health for the past year on this maintenance dosage, and there is nothing to suggest any loss of effect of the cortisone after two years of treatment. It is important to note that none of the patients have, up to the present, developed hypopituitary coma or any of the symptoms which accompany its incipient stages. This may possibly be related to the improved responsiveness to hypoglycaemia.

Effects of Large Dosage

As was recorded in our previous paper, though large dosage of cortisone produced a striking improvement in the physical condition, psychological disturbances changed the patient's attitude so much that prolonged treatment with large dosage was impossible. Similar observations have been made in three further cases given large doses during the past two years. Two of these patients were typical severe cases of post-partum hypopituitarism; the third patient, also female, suffered from hypopituitarism of uncertain aetiology. These patients were given cortisone orally in a dosage of 50 to 100 mg. a day for one to three weeks. There was usually an improvement in the physical condition for two or three days, but then various psychological disturbances occurred. One of these patients had previously been mentally normal, though dulled by the pituitary deficiency. When she was given large doses of cortisone she developed insomnia and had mild delusions. The second patient had always had a psychotic personality. During the control period she suffered from a variety of delusions, though she had lucid intervals lasting a few days. While on treatment she became definitely worse mentally and was very negativistic. The third case is of particular interest.

A woman aged 58 had had typical severe post-partum hypopituitarism since the age of 33. She had been receiving 100 mg. of testosterone weekly for two years before the present admission. During the last few months she had gradually developed, in addition to her hypopituitarism, what appeared clinically to be a true myxoedema (not what is sometimes called pituitary myxoedema), with coarse scaly skin, gross oedema of the orbits and lips, a croaking voice, and a rapidly progressive loss of head hair. Psychologically she varied between torpor and vulgar abusiveness; her behaviour became what is commonly described as "nasty drunk." She was given 100 mg. of cortisone daily for two weeks. This had no recognizable effect on the physical signs of myxoedema, and her mental condition deteriorated further. She was then given 3 gr. (0.2 mg.) of thyroid siccum daily. Within a week the appearances of myxoedema were much diminished and her mental state was improving. At the end of three weeks she had returned to her condition of six months before; she appeared clinically to be an ordinary case of hypopituitarism and was a peaceful and placid woman again. Continued treatment with this dosage of thyroid and testosterone kept her in reasonable health for several months. After this, cortisone (12.5 mg. daily) was then added to the other treatment. This rapidly produced the characteristic improvement in the general condition.

After about three or four days she developed a transient diuresis, and within the next two weeks she became able to do all her housework and go out shopping.

Conclusions

Several aspects of the syndrome of hypopituitarism could reasonably be considered as manifestations of the secondary thyroid hypofunction. These include the mental slowness and lack of interest, the physical sloth, the extreme sensitivity to cold, and the absence of sweating; symptoms which are characteristic of myxoedema. It is therefore very interesting that these particular aspects are influenced by cortisone, which is an adrenal hormone, but not by thyroid.

Until ten years ago a variety of treatments were used in hypopituitarism, but, apart from the effect of pregnancy, the results were not impressive. When testosterone was first introduced its value was obvious, and the patients and their relatives usually insisted that the treatment should be continued. Cortisone in small dosage produces even greater enthusiasm, and all the patients treated so far have invariably returned for a further supply of tablets well before their last supply is exhausted.

Testosterone produces in patients with hypopituitarism a sensation of well-being and improves the physical strength and mental activity. It causes a regrowth of pubic and axillary hair and some return of libido. Cortisone has similar but greater effects on the physical and mental activity, though it does not usually restore body hair. We have therefore tried the effect of giving both substances together, but have not observed any definite additive effect apart from that on the body hair. If after a course of this combined therapy the testosterone is stopped and the cortisone is continued alone, the patient's general condition remains as good as before. On the other hand, a patient who has had cortisone and testosterone together is not nearly so well if an attempt is made to continue treatment with testosterone but without cortisone.

It is a common practice to prescribe a combination of many kinds of treatment for hypopituitarism: thyroid, oestrogens, deoxycortone, sodium chloride, and various vitamins. There are theoretical reasons for administering all of these substances, but detailed studies are required to ascertain what effect is actually produced when each of them is administered singly for a period to patients who are being kept on maintenance treatment with cortisone. The only definite observation we have made is that in certain cases the puffiness of the eyes is reduced by a combination of thyroid and cortisone, but not by either given separately.

From our limited experience up to date it seems that cortisone therapy is the most useful method of long-term treatment of patients with hypopituitarism. A dosage of 12.5 mg. orally each day appears to be adequate for maintenance. A dosage of 25 mg. is equally satisfactory, but it does not produce a proportionate improvement in the signs or symptoms. Larger dosage, such as 50 to 100 mg. a day, is very apt to produce transient mental disturbances; this is in marked contrast to the tolerance of large dosage which is shown by patients with rheumatoid arthritis.

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REFERENCES

- Allott, E. N., and Simmons, J. H. (1951). *British Medical Journal*, 2, 568.
- Brown, N. H., and Greenblatt, R. B. (1952). *J. clin. Endocr.*, 12, 1040.
- Caughey, J. E., James, A., and MacLeod, E. K. (1952). *British Medical Journal*, 1, 1216.
- Clark, E. C., Franklin, M., and Sahs, A. L. (1951). *Arch. Neurol. Psychiat.*, Chicago, 65, 724.
- Dontigny, P. (1953). *Un. méd. Can.*, 82, 159.
- Fourman, P., and Horler, A. R. (1954). *Lancet*, 1, 334.
- Hart, F. D. (1953). In *Cortisone and A.C.T.H. in Clinical Practice*, edited by W. S. C. Copeman. Butterworth, London.
- Heyde, E. C. (1953). *Arch. intern. Med.*, 92, 442.
- Hijmans, W. (1952). M.D. Thesis, Leyden.
- Levy, M. S. (1952). *J. Amer. med. Ass.*, 149, 411.
- McCullagh, E. P., and Schaffenburg, C. A. (1953). *J. clin. Endocr.*, 13, 1283.
- Skilern, P. G., and Schaffenburg, C. A. (1954). *Cleveland Clin. Quart.*, 21, 31.

- Mathisen, H. S. (1953). *Acta med. scand.*, **145**, 326.
 Paniagua, M. E., Dominguez, A., and Marcial, R. (1952). *Bol. Asoc. méd. P. Rico*, **44**, 450.
 Perkins, R. F., and Rynearson, E. H. (1952). *J. clin. Endocr.*, **12**, 574.
 Roberts, L. N., Goldberg, W. M., and Pickard, H. (1952). *Canad. med. Ass. J.*, **66**, 458.
 Robertson, J. D., and Kirkpatrick, H. F. W. (1951). *Lancet*, **2**, 54.
 Sheehan, H. L., and Summers, V. K. (1952). *British Medical Journal*, **1**, 1214.
 Summers, V. K., and Sheehan, H. L. (1951). *Ibid.*, **2**, 564.
 Tardy, J. (1953). M.D. Thesis, Lyons.
 Thorn, G. W., Forsham, P. H., Frawley, T. F., and Hill, S. R. (1950). *J. clin. Endocr.*, **10**, 833.
 ———, Wilson, D. L., Renold, A. E., Frederickson, D. W., and Jenkins, D. (1951). *Amer. J. Med.*, **10**, 595.

ANALYSIS OF 1,400 CASES OF ACUTE INJURY TO THE HEAD*

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Between the years 1941 and 1951 2,000 cases of acute injury to the head were admitted to the General Hospital, Newcastle-upon-Tyne, and were treated in the department of neurological surgery. Of these, 1,544 were aged 13 and over (1,281 male, 263 female), and 456 were children up to the age of 12. The death rate in this consecutive series of cases was 10.5%.

In this paper a detailed analysis is given of 1,000 of the adults and 400 of the children; these cases were those of the above series who were admitted to hospital between the years 1945 and 1951. The adults were nursed in the department of neurological surgery; most of the children were nursed in the department of paediatrics.

The primary objects of this study were: (1) to determine the sources and causes of acute head injuries that occur in a provincial city and area engaged in the heavy industries of shipbuilding and mining; (2) to set on record a statistical statement of the severity and the nature of the injuries sustained; (3) to analyse the cause of death and to appraise methods of surgical treatment; (4) to inquire into the long-term results with the object of discovering whether and how morbidity can be minimized; and (5) to bring to the notice of the surgical world once again the magnitude and importance of this branch of medicine.

An analysis of 1,000 adult cases is given (Table I). It will be seen that 444 of them, or nearly half the total, were due to road accidents, and just over 19% of these accidents were fatal. Table II shows the great preponderance of these injuries in the productive period of life, underlining once more the serious social and economic aspect of injuries which for the most part should not happen.

Table III shows, what would be expected, a graver prognosis for patients admitted in a state of semicomatose and coma.

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TABLE I.—Analysis of 1,000 Consecutive Adult Cases

Cause of Injury	No.	Deaths
Road:		
Car	59	12
Motor-cycle	125	25
Pedal cycle	109	17
Pedestrian	151	33
Industry	248	38
Mines	59	7
Home	77	17
Simple fall outside	59	7
Struck by another	28	2
Sport	29	—
Gunshot wound	4	3
Air crash	2	—
Miscellaneous	10	4
Unknown	40	10
Total	1,000	175

TABLE II

Age	No.		Deaths	
	Male	Female	Male	Female
13 to 20	141	43	16	—
21 " 30	203	23	25	3
31 " 40	159	15	21	2
41 " 50	122	29	25	2
51 " 60	82	33	23	6
61 " 70	67	23	27	7
71 " 80	27	22	13	2
81 " 90	4	7	—	3
Total	805	195	150	25

TABLE III

Level of Consciousness on Admission	No.	Deaths
Consciousness to confusion	724	27
Semicoma	101	27
Coma	175	121
Total	1,000	175

In 940 of the cases the injury was closed, with possible internal compounding; among these there were 151 deaths. Sixty patients had an open injury of the vault, and 24 of them died. There were 221 associated injuries in the 1,000 cases, as follows: abdominal 3 (2 deaths); thoracic 19 (6 deaths); limbs 114 (16 deaths); spine 11 (no deaths); facio-maxillary 74 (9 deaths). Major operations had to be performed in 209 cases (74 deaths) and minor operations in 166 (16 deaths). The duration of stay in hospital averaged 12.9 days. The disposal of those who survived is shown in Table IV.

TABLE IV.—Disposal of 825 Patients

Home	756
Rehabilitation centre	18
Convalescent	12
Military or naval depot	12
Transfer to other centres (thoracic, facio-maxillary, psychiatric)	6
Miscellaneous	21

To complete the analysis of the cases we give (Tables V and VI) some figures relating to the 400 cases of injury to the head in children (up to the age of 12). Of these, 147 were due to road accidents; there were 20 deaths (13.36%). As many as 149 of the 400 children sustained injury as a result of falling from a height; 5 (3.3%) of these died.

Table VII gives details of the level of consciousness on admission. In the great majority of the cases (368) the injury was closed, with possible internal compounding; 26 (7.1%) of these children died. In 32 cases there was an open injury of the vault, with 8 (25%) deaths. Among the 400 cases there were 38 associated injuries, as follows: thoracic, 3 (no deaths); limbs, 20 (2 deaths); facio-maxillary, 14 (no deaths); pelvis, 1 (1 death). A major operation was necessary in 70 cases (6 deaths)