no doubt borne by the profits reaped in consequence of its success. The *British Medical Journal* (1954) has referred to the role of family doctors in dissuading mothers from the routine administration of unnecessary medicaments to their offspring. Both they and all health workers, especially those whose efforts are chiefly directed towards infants and children, must play their part in discouraging the regular use of laxatives and the attitude of mind which accompanies it.

Summary

The regular taking of laxatives by a group of school entrants and leavers is investigated. Over 17% of these children took them at least weekly, proportionately more girls than boys doing so at each age. The practice was commoner among entrants than among leavers.

The frequency of administration and type of laxative used are analysed.

The undesirable nature of such medication is discussed.

I am indebted to the health visitors of the North Bucks Division and to my wife for help in conducting this survey, to Professor A. Mair for advice, and to Dr. G. W. H. Townsend, county medical officer, for permission to publish this article.

REFERENCES

- Boyd, J. T., and Doll, R. (1954). *Brit. J. Cancer*, **8**, 231.
British Medical Journal, 1954, **1**, 265.
 Ibid., 1955, **1**, 780.
 Cheyne, G. (1724). *An Essay of Health and Long Life*, p. 37. Strahan, London.
 Creery, R. D. G. (1955). *British Medical Journal*, **2**, 178.
 Gordon, I. (1951). *Ibid.*, **1**, 611.
Practitioner, 1955, **175**, 121.
 Reid, J. J. A., and Reid, M. (1954). *Med. Offr.*, **91**, 273.
 Saunders, L. (1954). *Cultural Difference and Medical Care*, pp. 147, 271. Russell Sage Foundation, New York.
 Stein, L., and Sklaroff, S. A. (1952). *Brit. J. soc. Med.*, **6**, 118.

UNUSUAL NEUROLOGICAL COMPLICATIONS AFTER SUDDEN WITHDRAWAL OF CORTISONE

BY

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It is well known that reactions may occur during treatment with cortisone and corticotrophin and when these drugs are withdrawn. In the case here described a severe and unusual illness occurred when the patient inadvertently stopped taking cortisone, with which she had been treated for a long period.

Case Report

A married woman of 38 was admitted to Addenbrooke's Hospital on March 26, 1953, in a comatose state. She had suffered from severe rheumatoid arthritis for 14 years, and for the previous 18 months had been having cortisone, with considerable benefit. During this period she had had some evidence of hypercortisonism: moonface, gain in weight, acneiform lesions, and difficulty in healing trivial wounds.

After 100 mg. intramuscularly daily for the first six days the usual maintenance dose was 50 mg. daily, at first intramuscularly and later orally. For the two months before admission the dose had been higher—75 mg. orally—on account of an increase in severity of the disease.

Ten days before admission she accidentally doubled the dosage of cortisone to 150 mg. daily, having misread instructions supplied. Seven days later she realized her error and discontinued the drug. The next day she com-

plained of headache and difficulty in sleeping, which she attributed to worry about her children, who had tonsillitis. Her joints were a little more painful. On the third morning after stopping cortisone she was found in bed in a comatose state.

On admission a comatose, well-nourished woman with joints showing rheumatoid changes was seen; she was rousable by painful stimulation. Her temperature was 105° F. (40.6° C.), pulse 140. Her face was moon-shaped, with acneiform lesions. She was not dehydrated or oedematous, and not cyanosed. Medium crepitations were heard at both lung bases. Her B.P. was 90/60.

Examination of the abdomen revealed no abnormality. Central nervous system: fundi normal, pupils reactive; tone generally flaccid; tendon and abdominal reflexes absent; plantar responses flexor.

Investigations.—Blood urea, 24 mg./100 ml.; serum potassium, 18.3 mg./100 ml.; serum sodium, 311 mg./100 ml.; serum calcium, 8.8 mg./100 ml.; Hb, 14 g./100 ml.; W.B.C., 15,000; neutrophil leucocytosis. Urinary 17-ketosteroid excretion, 6 mg./day. C.S.U.: protein+, otherwise normal. E.C.G. normal. Lumbar puncture: normal pressure and fluid.

Treatment and Progress.—She was treated with tepid sponging, fluid by intragastric Ryle tube, and penicillin. The intravenous injection of 50 ml. of 50% glucose had no effect. After 24 hours the right pupil was larger than the left. The next day the coma was lighter but she was unable to speak; she was at times conscious; there was pronounced weakness of the arms and legs, with some return of tone and tendon reflexes. Her arthritic joints were obviously very painful on slight passive movement and were much more swollen than when she was on cortisone.

On the fifth day she attempted to speak, but had severe dysarthria and was seen to be very labile emotionally, alternately weeping and laughing. A Parkinsonian tremor appeared in the hands; 24 hours later the feet were involved; by then the pupils were again equal. She was started on 25 mg. of cortisone daily, later increased to 50 mg. Six days after admission the temperature became normal; there was some dysphagia and almost complete bilateral palatal palsy which disappeared in a few days. Fourteen days after the onset of the illness her tremor was most pronounced, involving both upper and lower limbs. Benzhexol hydrochloride was given with good effect.

Her speech improved slowly, but after 30 months it remains slow, halting, and slurred, being confined to short sentences. She is still emotionally labile, but to a much less degree. She can sit up, but is unable to feed herself or to stand unsupported, having generalized loss of power in the limbs. There is now wasting compatible with disuse atrophy; the tendon reflexes are diminished, the plantar responses are flexor, and there is no sensory loss. Slight intermittent tremor of the hands remains.

Discussion

In this case, in which cortisone was withdrawn suddenly after a long period of therapy, the two features we would stress are the acute illness, presumably due in part to acute adrenal insufficiency and in part to cerebral lesions, and the permanent neurological sequelae.

Acute adrenal insufficiency, which may occur and prove fatal, can appear as sudden circulatory collapse, decrease in blood pressure, increase in pulse rate, or perhaps an elevation of temperature, and may cause the patient to lose consciousness quickly (Salassa *et al.*, 1953a, 1953b). In these circumstances the serum electrolytes may be normal (Thorn, 1949), whilst most of the features of chronic adrenal insufficiency will be absent.

Fatal adrenal insufficiency has been reported on several occasions. Fraser *et al.* (1952) described the case of a 34-year-old man with rheumatoid arthritis who died in severe shock two days after operation. He had been on cortisone for only two months, with an average daily dose of 50 mg.,

which was stopped two days before the operation, a cup arthroplasty on the hip. At necropsy the adrenals were atrophied; there were multiple haemorrhages in the lungs and in the interventricular septum; a few petechiae were present in the brain.

Proctor and Rawson (1951) described a similar case: adrenal cortical failure occurred on completion of a 24-day course of cortisone followed by corticotrophin for 6 days. There was cytolytic destruction of the adrenal cortex, most pronounced in the fascicular zone.

A state of shock and hyperpyrexia followed by death has been reported after a minor operation on a patient receiving cortisone for rheumatoid arthritis (Lewis *et al.*, 1953) and also after a course of cortisone given to a patient with ulcerative colitis (Rachet *et al.*, 1951).

Sprague *et al.* (1950) report the death of two patients, on treatment with cortisone for rheumatoid arthritis, who after operation became shocked, comatose, and anuric. In both cases the adrenals showed atrophic changes.

Norymberski *et al.* (1953) have shown that 50 mg. of cortisone daily will completely suppress the endogenous output of hydrocortisone-like steroids, while Sprague *et al.* (1950) suggest that a dose of cortisone as low as 20 mg. a day for five days may be suppressive. These authors suggest that patients who develop extensive hypercortisonism may be more liable to persistence of impaired adrenal and pituitary function.

Our patient, who had been on cortisone for 18 months and had mild hypercortisonism, abruptly stopped taking cortisone and developed coma, hyperpyrexia, tachycardia, and hypotension, the serum electrolytes being normal apart from a slightly low serum sodium. She thus had many of the features mentioned in the cases previously described. We think it likely that she had acute adrenal insufficiency, although as she recovered we do not know the nature of the pathological lesion.

With regard to the neurological lesions our patient had a series of cerebral disturbances during the acute phase and was left with serious incapacitating sequelae similar to Parkinsonism. It is possible that she had a coincidental encephalitis of bacterial or virus origin, but the normality of the C.S.F. is against this. The widespread nature of the cerebral damage suggests multiple small vascular lesions.

Cosgriff (1951) described a series of thrombo-embolic episodes which followed the cessation of cortisone. These he ascribed to the hypercoagulability of blood which is present during and following cortisone therapy. However, only two of these were cerebral vascular accidents and both occurred in elderly patients aged 69 and 70. Neither case resembled ours.

The withdrawal of cortisone from patients suffering from rheumatoid arthritis may be followed by panmesenchymal and panangiitic reactions which, when severe, may resemble acute disseminated lupus erythematosus or polyarteritis nodosa (Slocumb, 1953). In our case there was no evidence of vascular lesions outside the brain.

In the case described by Fraser *et al.* (1952) a number of petechial haemorrhages were found in the brain. It is possible that our patient had similar lesions. However, the exact pathology of the cerebral lesions must remain in doubt.

Summary

A patient with rheumatoid arthritis was treated with cortisone for 18 months. She inadvertently took a double dose for one week and then discontinued the drug. Coma, hyperpyrexia, hypotension, and, later, dysarthria, Parkinsonian tremors, and muscular weakness ensued. The patient has only partially recovered from these disabilities and is still bedridden after 30 months. We consider that this syndrome may have been due to acute adrenal insufficiency complicated by multiple cerebral vascular lesions.

REFERENCES

- Cosgriff, S. W. (1951). *J. Amer. med. Ass.*, **147**, 924.
 Fraser, C. G., Preuss, F. S., and Bigford, W. D. (1952). *Ibid.*, **149**, 1542.
 Lewis, L., Robinson, R. F., Yee, J., Hacker, L. A., and Eisen, G. (1953). *Ann. intern. Med.*, **39**, 116.
 Norymberski, J. K., Stubbs, R. D., and West, H. F. (1953). *Lancet*, **1**, 1276.
 Proctor, E. L., and Rawson, A. J. (1951). *Amer. J. clin. Path.*, **21**, 158.
 Rachet, J., Bussan, A., Roge, J., and Robineau, R. (1951). *Arch. Mal. Appar. dig.*, **40**, 1129.
 Salassa, R. M., Bennett, W. A., Keating, F. R., and Sprague, R. G. (1953a). *J. Amer. med. Ass.*, **152**, 1509.
 ——— Keating, F. R., and Sprague, R. G. (1953b). *Proc. Mayo Clin.*, **28**, 662.
 Slocumb, C. H. (1953). *Ibid.*, **28**, 655.
 Sprague, R. G., *et al.* (1950). *Arch. intern. Med.*, **85**, 199.
 Thorn, G. W. (1949). *The Diagnosis and Treatment of Adrenal Insufficiency*, p. 19. Blackwell, Oxford.

ROVSING'S SIGN

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In 1907 Professor Thorkild Rovsing, of Copenhagen, first described the sign which now bears his name. The following is a translation of his description. "The left hand is applied over the healthy colon in the left iliac fossa, the right hand applies pressure over it in an anti-peristaltic direction; because the ileo-caecal valve is competent, pain is produced in the right iliac fossa with inflammation of the appendix and caecum. Where there is muscular rigidity in the right iliac fossa and therefore accurate palpation is impossible, it will give a clue to the diagnosis—that is, it will differentiate between a lesion of the caecum and the appendix in the right iliac fossa from another lesion giving inflammation in the right iliac fossa."

This sign, with its suggested mechanism, has since found a place in the minds of countless medical students, and it receives tacit approval in surgical textbooks—for example, Aird (1949) and Bailey and Love (1956). The following note and the accompanying well-known diagram are those of Hamilton Bailey (1954): "Even pressure is exerted over the descending colon. This forces gas into the caecum. If, when the left iliac fossa is pressed, pain is appreciated in the right iliac fossa, the case is probably one of acute appendicitis."

I have been intrigued by this sign, and for this reason, with the following questions in mind, a series of observations were carried out: (1) Does Rovsing's sign occur? (2) Is the suggested mechanism correct? (3) If not, what is the explanation?

Does It Occur?

During a series of 303 consecutive cases of acute appendicitis confirmed at operation, Rovsing's sign was found to be positive on five occasions. The findings in these patients were confirmed by at least two other surgeons.

Case 1.—A woman aged 34. The right lower quadrant of the abdomen was blocked by 1/2000 amethocaine hydrochloride. After this, Rovsing's sign was no longer present, although the rebound phenomenon still remained. A grid-iron incision with gentle retraction of the wound edges revealed an acutely inflamed retro-ileal appendix; the proximal part of the appendix between the caecum and the ileum was visible, and was found to be in direct contact with the overlying parietal peritoneum, which showed signs of inflammation.

Case 2.—A man aged 37. This patient presented with a classical history of appendicitis. The right lower abdomen was blocked by local analgesia as in Case 1. Rovsing's sign