

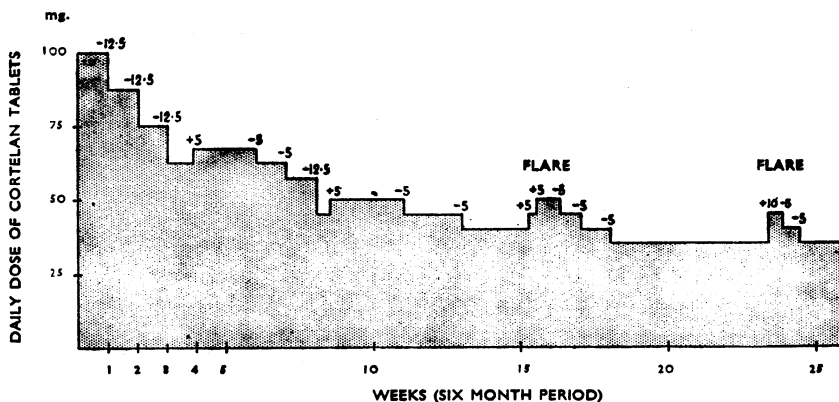
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Treatment must be individualized, but the way a typical course may run is shown by the graph.



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of little value it must be pointed out that most patients with Meniere's disease are able to continue living a reasonably normal life when treated on the above lines. If the attacks of vertigo do continue to interfere with the patient's life and if the condition is unilateral the attacks can be stopped by a destructive procedure as mentioned above. Impulses can be cut off from the affected labyrinth either by destruction of the labyrinth itself or by division of the vestibular portion of the auditory nerve. Destruction of the labyrinth also involves complete loss of hearing on that side and for that reason division of the vestibular portion of the nerve might seem more desirable. However, this latter operation is a much more formidable procedure with a greater morbidity and a definite mortality and in most cases there is no value to be gained in preserving the distorted remnant of hearing which remains. As a result division of the vestibular portion of the nerve is restricted to those cases which have troublesome attacks of vertigo but who still have good hearing.

Benign Paroxysmal Positional Vertigo

For this condition there is no specific cure but fortunately most cases settle down spontaneously. In the period when the condition is active the

patients are helped most by appreciation of the circumstances under which an attack develops and by realization that once an attack has occurred there is usually a period of several hours during which the offending position can be adopted with impunity. Some patients find it possible to avoid any posture which provokes the vertigo but in the others it is often best deliberately to produce an attack at a convenient moment and then enjoy a period of freedom. In this way patients with this complaint can often carry on with an occupation which involves climbing ladders, etc.

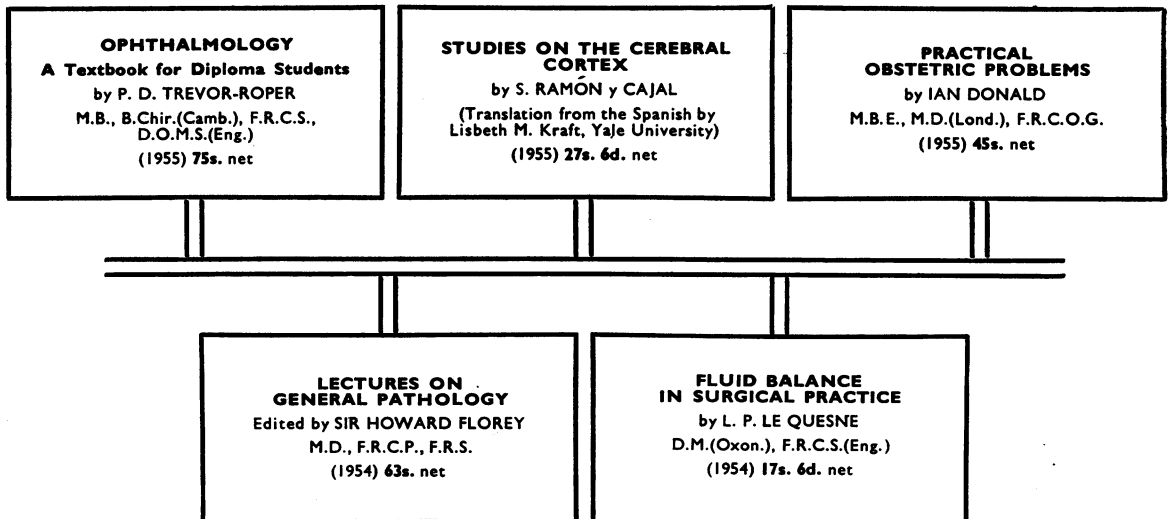
In addition, many of these patients are convinced that they are helped by those antihistaminics which seem most active in motion sickness.

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coagulants can cause marked bleeding into the lung and pleura.

Treatment of Pulmonary Embolism

Most of the points in treatment of pulmonary embolism have been dealt with already. When embolism occurs it is generally right to start heparin treatment immediately, whilst the injection of morphine, gr. $\frac{1}{4}$ and atropine gr. $\frac{1}{75}$ is also of value. Intravenous injection of papaverine sulphate (with the object of producing pulmonary vasodilatation) was formerly advocated but is almost certainly of no more value in dilating the pulmonary than the systemic arteries when given in this way (Kinmonth, 1952).

The need for cautious use of the anticoagulants in cases of extensive pulmonary infarction has already been emphasised. In such cases vein ligation may be the safest method of preventing further embolism. In this connection it should be noted that certain Australian authorities (Cummine and Lyons, 1948; Dew, 1953), have contended that pulmonary artery thrombosis is more common than embolism, and they have advanced various reports from the older literature in support of this view. However, it was rare for the leg veins to be completely dissected at the autopsies from which these reports were derived and, in the absence of such complete examination, it would be rash to deny the occurrence of embolism. That massive thrombosis of the pulmonary arteries does occur, is well-established. It is significant that in at least one of the more recent reports on pulmonary artery thrombosis (Keating *et al.*, 1953), the leg veins are described as rarely free from thrombosis.

Conclusions

With intelligent use of the various measures now available it is certainly possible to do much more for the patient with thrombosis than was possible a few years ago. Even in the more difficult cases

of thrombosis the clinician who has a good laboratory to help him should be able to treat his cases with relative safety. Prevention and treatment of thrombosis is still difficult in many cases, especially in those patients who may bleed from an operation site during anticoagulant therapy. A great deal of research is needed to clarify many obscure aspects of aetiology and pathogenesis without which truly rational therapy will never be possible. The author is conscious that many questions are left unanswered in this review, and that scientific information is sadly lacking on many aspects of thrombosis.

Acknowledgment

I am indebted to the editor of *Annals of the Royal College of Surgeons of England* for permission to reproduce Fig. 1.

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hospital in relapse at the time of this report. Three patients are in remission, two of whom no longer receive maintenance treatment.

Side effects produced by corticotrophin and cortisone have been prominent in this group. Familiar complications such as hypertension with fits, hypokalaemia, steroid diabetes, and acute mania have been encountered. Two patients have developed miliary tuberculosis during the course of treatment, and in neither case was a tuberculous focus suspected before the commencement of treatment; this potential hazard has been recorded previously (Harris-Jones and Pein, 1952; Walker, 1952).

Discussion

This group of patients has been studied and presented because the results of treatment differ from some previously reported (Cohen and Cadman, 1953; Rishards, 1954). In their study of a group comparable to the present one, Soffer and Bader (1952) quote a mortality of 33 per cent. In a recent survey of 62 cases treated with ACTH and cortisone, Harvey *et al.* (1954) record a mortality of 29 per cent.; in 26 per cent. the disease appeared inactive following a single course of steroid therapy; 18 per cent. required continuous maintenance therapy.

The impression gained from the present study, is that although these hormones may induce a remission, particularly in the acute phase, they have little influence on the subsequent course of the disease. Their effect on the pulmonary lesions is unpredictable, and in this respect their use may be hazardous. The thrombocytopaenic state appears uninfluenced by treatment. When the haematological and biochemical results of treatment are set against the clinical response, it seems difficult to conceive that the fundamental pathological process is being arrested. It appears more likely that ACTH and cortisone merely modify the hyper-immune state, and minimize the antigen-antibody reactions presumably responsible for the disease process. Despite their obvious

limitations however, these steroids remain the most valuable weapons yet available for the treatment of systemic lupus erythematosus.

Summary

The results of the treatment of 12 cases of systemic lupus erythematosus with ACTH and cortisone are recorded. An evaluation of these results has been attempted, and from this it has been concluded that although these steroids modify the acute phase of the disease, there is less evidence that they influence its subsequent course.

It is a pleasure to record my gratitude for the great help and encouragement I have received from the physicians and dermatologists of the Royal Hospital, and Royal Infirmary, Sheffield, under whose care the patients were admitted. I would also like to express my thanks to Professor C. H. Stuart-Harris for his helpful criticisms of this paper.

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Comment

Both central and peripheral factors here contribute to the cyanosis. The arterial oxygen saturation is remarkably low at 82 per cent., and the figures of this order have only previously been recorded in the presence of advanced heart failure and gross cardiac enlargement. It has been suggested that the cause of the central cyanosis is the presence of small pulmonary arteriovenous shunts such as have been demonstrated by Farber in Eisenmenger's complex. Ordway noted in two patients that the arterial oxygen saturation was not raised by breathing pure oxygen, but there are no other reports of respiratory function studies in cases of primary pulmonary hypertension. The arteriovenous oxygen difference of 5.7 ml. per cent. is raised and results from poor peripheral circulation and polycythaemia.

Haemoptysis, not a constant feature of this condition, was the presenting symptom here, and could well result from the rupture of small pulmonary arteriovenous shunts.

I wish to thank Col. R. C. Spicer and Dr. Evan Bedford for permission to publish this case.

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