CORTISONE IN THE TREATMENT OF POSTOPERATIVE CEREBRAL EDEMA

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Modern surgical and anesthetic techniques have diminished markedly the desperate problems of postoperative cerebral edema that plagued the pioneer neurological surgeons, but cerebral edema of lesser degree remains an important factor in postoperative morbidity and mortality following many intracranial operations. The introduction of intravenous urea has been a major contribution in the prevention or temporary alleviation of the mechanical complications of brain swelling, but does not prevent or relieve the neurological deficits associated with cerebral edema that are not caused by brain-stem herniation and other mechanical complications of the swollen brain.

Although there has been evidence in the literature over the past 20 years that the anti-edema and anti-inflammatory effects of adrenal cortical steroids extend to cerebral tissue, this present attempt to evaluate the effectiveness of cortisone in the prevention and amelioration of the neurological symptoms of postoperative cerebral edema actually was undertaken because of an incidental observation made during an analysis of the endocrinological aspects of pituitary tumors and other tumors of the sellar region. This analysis showed that the protective effect of cortisone in operations in and about the sella was equally dramatic in those patients who had little or no preoperative hypopituitarism as in those with marked hypopituitarism. This raised the possibility that part, at least, of the well-known protective effect of cortisone in reducing the mortality and morbidity following these operations might be ascribable to a nonspecific effect of cortisone in reducing cerebral edema in a critical area of the brain rather than to the specific effect of supporting the pituitary-adrenal axis.

Since temporal lobectomy for focal cerebral seizures involves a reasonably standard amount of surgical manipulation of the brain, it seemed worth while to compare the effects of prophylactic administration of cortisone in a series of patients, undergoing temporal lobectomy, with a similar series of patients, operated upon by the same surgeon (T.R.), in whom cortisone had not been administered. It was hoped that a comparison between these two groups of the incidence and severity of the temporary postoperative symptoms which we attribute to postoperative cerebral edema might give an indication as to whether or not cortisone was of value in affording some protection against these postoperative problems.

REVIEW OF LITERATURE

The striking effect of cortisone in reducing the mortality and morbidity of operations about the sellar region became apparent soon after cortisone became generally available.9,10,18,24,25 In this connection it is of interest that Adson, in a discussion on surgery of sellar and suprasellar lesions at the St. Louis meeting of the Society of Neurological Surgeons on April 29 and 30, 1940, reported beneficial effects from the use of adrenal cortical extract for "pituitary asthenia" in the early postoperative period.

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The earliest pertinent study on cerebral edema, however, is that of Prados et al.,\textsuperscript{16,17} who studied the effect of subcutaneous injection of extract of anterior pituitary lobe (prepared in the Research Institute of Endocrinology of McGill University) on experimental cerebral edema produced by exposure of the cat brain to air. They concluded that adrenal cortical extract and extracts of the anterior lobe of the pituitary gland containing the corticotropic factor prevent or minimize the swelling of the brain, the changes in permeability of cerebral capillaries and the electroencephalographic alterations that follow exposure of the brain to air. Similar conclusions were reported by Grenell and McCawley\textsuperscript{8} in a study using a similar model. In a subsequent related study, Grenell and Mendelson\textsuperscript{7} showed that Cortisal was more effective in preventing cerebral edema than either ACTH or cortisone.

Foley et al.\textsuperscript{3} studied the effect of cortisone on early repair of brain wounds in guinea pigs. Whereas an appropriate inflammatory response appeared in control animals the first week, in the cortisone-treated animals there was a pronounced inhibition of this inflammatory response. There was no edema about the wound, no extravasation of serum into adjacent tissue, and no fibrin formation.

Dr. Aronson,\textsuperscript{4} in discussion of this paper, remarked that after a certain amount of time, whether or not cortisone was continued, the reactive elements managed to catch up to those of the control animals.

In recent years cortisone has been tried out in a number of neurological conditions in which suppression of edema and/or inflammatory response was felt to be therapeutically important. Of particular importance to this study are the reports of Russek et al.\textsuperscript{20,21} and Roberts\textsuperscript{19} who used cortisone in the immediate therapy of apoplectic strokes. Within 24 hours of commencement of therapy, the majority of patients were reported to show significant lessening of paresis, increased sense of well-being and improved motivation.

Similar but temporary improvement in neurological status was reported by Kofman et al.\textsuperscript{15} in their report of 15 cases of cerebral metastases from carcinoma of the breast treated with Prednisolone. They reported remarkable temporary improvement in 14 of these 15 patients. This was attributed to the anti-inflammatory effect on the cerebral edema surrounding the metastatic lesions, since these patients all had had previous adrenalectomy and the metastatic tumors themselves, therefore, probably were not hormone-dependent at this time, and also because a similar improvement occurred in 3 cases of cerebral metastases from bronchial carcinoma. Galicich et al.\textsuperscript{4,5} reported similar dramatic clinical improvement in cases of recurrent gliomata treated with large doses of Dexamethasone. In some of their patients, in addition to improvement in consciousness and reduction of other neurological deficits such as hemiparesis and papilledema, there was objective evidence of reduction of intracranial pressure with bulging decompressions becoming flat and soft, and in 2 patients, angiographic evidence of reduction in size of the expanding lesion. Säker and Rust\textsuperscript{22} also reported on the use of Prednisolone as an anti-inflammatory and anti-edema agent in patients with brain tumors, and recommended the combination of Prednisolone with Marsalid.

Gurdjian and Webster\textsuperscript{8} commented on the use of cortisone and Prednisolone in the treatment of severe head injuries. It was their impression that cortisone produced marked improvement in some of their deeply comatose patients, with the result that patients unconscious for several days as a result of head injuries but without intracranial mass lesions seemed to become ambulatory more quickly when treated with cortisone than similar patients who did not receive cortisone.

Smith and Ross\textsuperscript{23} studied the effect of steroids on meningeal inflammation caused by injection of small amounts of Pantopaque into the brains of rabbits and guinea pigs. They found that cortisone and Prednisolone given by mouth or parenterally were effective in preventing chronic adhesive arachnoiditis by inhibiting the production of
fibrous changes in the leptomeninges but that intrathecal administration of hydrocortisone produced no such beneficial effect.

Lippert et al. studied the effects of cortisone on cerebral edema produced by implantation of psyllium seeds into the dog brain, and reported suggestive evidence of improvement in morbidity, mortality, and severity of edema in the cortisone-treated animals as compared with the controls.

Jefferson commented on the possible relationship between cerebral edema and suprarenal insufficiency on the basis of necropsy studies of 2 patients in whom cerebral edema and suprarenal failure were associated, and 1 patient with chromophobe adenoma of the pituitary with papiledema and, at the time of operation, a tight, swollen brain which could not be attributed either to the size of the tumor or ventricular obstruction.

METHODS

In 1958 all patients operated upon for temporal-lobe epilepsy by the senior author (T.R.) were put on a regime of prophylactic cortisone as follows (Cortisone 1 series):*

1. During the operation and while the patient was receiving intravenous fluids postoperatively, hydrocortisone, 10 mg. per hr., was administered in the intravenous fluid.

2. During the first 4 postoperative days the patient received 25 mg. cortisone-acetate orally every 6 hrs. (100 mg. total daily dose).

3. Oral cortisone-acetate administration was reduced progressively over the following 4 days:
   a. 5th postoperative day, 25 mg. q. 8 hrs. (75 mg. total daily dose).
   b. 6th postoperative day, 12.5 mg. q. 6 hrs. (50 mg. total daily dose).
   c. 7th postoperative day, 12.5 mg. q. 8 hrs. (37.5 mg. total daily dose).
   d. 8th postoperative day, 12.5 mg. b.i.d. (25 mg. total daily dose).

4. Preoperatively, all patients had the following electrolyte determinations: serum sodium, potassium, chlorides, nonprotein nitrogen, and blood sugar ½ to 2 hrs. after breakfast. These were repeated on the 12–14th postoperative day.

5. The fluid intake and output for the entire 8-day period was charted on each patient.

* The assistance of Dr. John Beck, Department of Endocrinology of the Royal Victoria Hospital, in planning the cortisone regimes in this study is gratefully acknowledged.

At the end of the year the 31 consecutive patients undergoing temporal lobectomy on the above cortisone regime were compared with 35 consecutive temporal-lobe patients operated upon by the senior author (T.R.) prior to the beginning of the cortisone series.†

Following this analysis the next consecutive 35 patients undergoing temporal lobectomy by the senior author (T.R.) were placed on the following regime of increased cortisone dosage (Cortisone 2 series):

1. 100 mg. of cortisone-acetate orally the day before operation.

2. 100 mg. of cortisone-acetate orally on the morning of operation.

3. During operation and while intravenous fluids were being administered postoperatively, hydrocortisone, 10 mg. per hr., was given for a total dose of 100 mg. intravenously during the day of operation and the night following operation.

4. On the 1st and 2nd postoperative days patients were given 50 mg. of cortisone-acetate orally every 8 hrs. (total daily dose of 150 mg.).

5. On the next 4 postoperative days patients were given 25 mg. of cortisone-acetate orally every 6 hrs. (total daily dose of 100 mg.).

6. Oral cortisone-acetate administration was reduced progressively over the next 4 days:
   a. 7th postoperative day 25 mg. q. 8 hrs. (total daily dose 75 mg.).
   b. 8th postoperative day 12.5 mg. q. 6 hrs. (total daily dose 50 mg.).
   c. 9th postoperative day 12.5 mg. q. 8 hrs. (total daily dose 37.5 mg.).
   d. 10th postoperative day 12.5 mg. b.i.d. (total daily dose 25 mg.).

7. The electrolyte studies and charting of intake and output were carried out as in the Cortisone 1 series.

The records of these three series of patients (No Cortisone, Cortisone 1 and Cortisone 2) were then analyzed with reference to the incidence, severity and duration of postoperative hemiparesis, seizures during the first 2 weeks postoperatively, and presence and duration of the syndrome of aseptic meningitis. In the case of patients undergoing temporal lobectomy on the hemisphere

† The comparison of these two series of patients was reported at a Symposium on Cerebral Edema at the meeting of the Neurosurgical Society of America, Hot Springs, Virginia, on April 3, 1939.
dominant for speech, the incidence, duration and severity of dysphasia, and comparison of the intelligence quotient and memory quotient recorded 2½ to 3 weeks after operation with the preoperative test rating were analyzed.*

RESULTS

HEMIPARESIS

Patients were graded in regard to hemiparesis on the following scale:

Grade 0 = no hemiparesis.

Grade 1 = slight or moderate weakness, principally in the hand and of 4 days’ duration or less.

Grade 2 = weakness of the proximal muscles of the arm as well as of the hand or weakness of the hand alone persisting for 5–10 days.

Grade 3 = paresis of the arm of 11 or more days’ duration, or weakness of leg as well as weakness of arm.

Fig. 1 shows the results of this comparison. In the No Cortisone group of 35 patients, 43 per cent had no hemiparesis as compared with 68 per cent in the Cortisone 1 series and 89 per cent in the Cortisone 2 series. The difference between the control group and the two cortisone groups combined is significant beyond the .001 level of confidence ($X^2 = 16.4$) and the difference between the Cortisone 2 series and the Cortisone 1 series is also significant ($p < .05$).

In the No Cortisone series 17 per cent had Grade 1 hemiparesis as compared with 23 per cent in the Cortisone 1 series and 9 per cent in the Cortisone 2 series. Of the No Cortisone series 36 per cent had hemiparesis of Grade 2 severity as compared with 10 per cent in the Cortisone 1 series and 3 per cent in the Cortisone 2 series. Of the No Cortisone series 14 per cent had a Grade 3 hemiparesis. There were no patients in either of the cortisone series with this degree of temporary postoperative hemiparesis.

Those patients who had some temporary hemiparesis were classified according to the duration of the hemiparesis and divided into two groups, those in whom the weakness lasted 4 days or less, and those in whom it persisted for 5 to 30 days. In these patients who had some hemiparesis, the duration was 4 days or less in 35 per cent in the No Cortisone group as compared with 60 per cent in the Cortisone 1 series and 75 per cent in the Cortisone 2 series. The difference between the control group and the two cortisone groups combined is significant beyond the .001 level of confidence ($X^2 = 17.9$).

Summary. This comparison suggests that the prophylactic cortisone therapy exerted a definite effect in reducing the incidence, severity and duration of temporary hemiparesis in the early postoperative period following temporal lobectomy for focal cerebral seizures, and that this effect was enhanced by increasing and prolonging the administration of cortisone.

APHASIA

The operation was carried out on the hemisphere dominant for speech in 14 of the patients in the No Cortisone series, 22 of the patients in the Cortisone 1 series, and 23 of the patients in the Cortisone 2 series. The patients were graded in regard to dysphasia on the following scale:

Grade 0 = no dysphasia.

Grade 1 = slight dysphasia of 1–4 days’ duration.
Grade 2 = moderate dysphasia, or slight dysphasia of 5–10 days’ duration.

Grade 3 = severe dysphasia, or moderate dysphasia of 11 or more days’ duration.

The results of the comparison of the incidence, severity and duration of dysphasia are shown in Fig. 2. Fifty per cent of the No Cortisone patients had a Grade 3 temporary postoperative dysphasia as compared with 32 per cent in the Cortisone 1 series and 30 per cent in the Cortisone 2 series. Because the numbers are small, the comparison of the three groups is more meaningful when Grades 0 and 1 are combined and compared with Grade 2 and Grade 3. Thus only 14 per cent of the No Cortisone patients had either no dysphasia or minimal and brief dysphasia as compared with 55 per cent of the Cortisone 1 series and 62 per cent of the Cortisone 2 series. The two cortisone series combined show a significantly lower incidence of Grade 2 and Grade 3 dysphasia than the control series ($x^2 = 8.1$, $p < .01$).

Those patients who had some temporary dysphasia were classified according to the duration and divided into two groups, those in whom the dysphasia lasted 6 days or less and those in whom it persisted for 7 to 30 days. When dysphasia developed it persisted for 7 days or longer in 86 per cent of the control group who had dysphasia as compared with 47 per cent of those who had dysphasia in the Cortisone 1 group and 41 per cent in the Cortisone 2 group.

**Summary.** The prophylactic administration of cortisone apparently resulted in a decrease in the severity and duration of dysphasia in the early postoperative period following temporal lobectomy for temporal-lobe seizures. The protective effect was somewhat less striking than that on hemiparesis, but was more clearly evident in the Cortisone 2 series.

**PSYCHOLOGICAL DEFICITS**

No significant alterations in the postoperative intelligence or memory quotients were recorded by Dr. Milner and her associates in patients following temporal lobectomy on the nondominant cerebral hemisphere. A temporary fall in these test ratings did occur in many patients when the temporal lobe of the hemisphere dominant for speech was removed. The analysis of these test results was therefore limited to those patients undergoing operation on the cerebral hemisphere dominant for speech. The comparison of these three series of patients in this regard is shown in Fig. 3. The postoperative full-scale I.Q. fell 6 points or more in 9 of the 10 patients (90 per cent) in the No Cortisone series as compared with 55 per cent in the Cortisone 1 series and 48 per cent in the Cortisone 2 series. The memory quotient (M.Q.) fell 14 points or more in 90
per cent of the patients in the No Cortisone series as compared with 50 per cent in the Cortisone 1 series and 43 per cent in the Cortisone 2 series. The difference between the combined cortisone groups and the controls is significant for both these measurements (I.Q.: $x^2 = 5.30, p < .05$; M.Q.: $x^2 = 6.19, p < .02$).

**Summary.** The prophylactic administration of cortisone seemed to produce a moderate degree of protection against reduction in the early postoperative period of certain aspects of mental function as tested by the full-scale I.Q. and by the M.Q. rating.

**SEIZURES**

The patients were classified in regard to seizures during the first 2 postoperative weeks as follows:

- Grade 0 = no attacks.
- Grade 1 = auras and/or minor attacks only on 1 to 4 days.
- Grade 2 = minor attacks on 5 days or more, or minor and major attacks on 1 to 4 days.
- Grade 3 = minor and major attacks on 5 days or more, or episodes of status epilepticus, major or minor.

The results of this comparison are shown in Fig. 4. Twenty-nine per cent of the patients in the No Cortisone and in the Cortisone 1 group had no seizures as compared with 54 per cent in the Cortisone 2 series, a statistically significant difference ($x^2 = 6.34, p < .02$). At the other end of the scale 20 per cent of the No Cortisone series had seizures of Grade 3 severity as compared with 10 per cent in the Cortisone 1 series and 6 per cent in the Cortisone 2 series. This difference is not significant. There was likewise no significant difference in the duration of the seizures in the control group as compared with the two cortisone series.

**ASEPTIC MENINGITIS**

The syndrome of aseptic meningitis was considered to be present whenever there was an unexplained elevation of temperature of more than 1°F. after the 2nd postoperative day if there was stiffness of the neck or other signs of meningeal irritation were present, or, when lumbar puncture was carried out because of headache or elevation of temperature, the cerebrospinal fluid showed more than the number of white cells that might be expected in view of the number of red cells in the spinal fluid. This syndrome was present in 34 per cent of the 35 patients in the No Cortisone series, in 19 per cent of the 31 patients in the Cortisone 1 series, and in 3 or 9 per cent of the 35 patients in the Cortisone 2 series (Fig. 5). The incidence of aseptic meningitis was significantly lower in the combined cortisone groups than in the control group ($x^2 = 5.92, p < .01$).

**Summary.** The analysis gives evidence that the prophylactic cortisone therapy gave some protection against the syndrome of aseptic meningitis and suggests that the protective effect was greater with the larger dosage of cortisone. In those who had this syndrome there was no difference in the duration of the symptoms in the No Cortisone and the Cortisone 1 and 2 groups.
Although the postoperative symptoms analyzed in this report have not been a major problem in most patients following temporal lobectomy, the increase in smoothness of the postoperative course and the patient’s comfort, as well as the reduction in incidence, severity and duration of the symptoms studied in this report, were clearly evident from the clinical standpoint well before the actual analysis of the first series was carried out. In 1959, following the analysis of the Cortisone 1 series, we started using the prophylactic cortisone regime on all patients with seizures as well as on the patients undergoing temporal lobectomy who are reported here as the Cortisone 2 series. With this increased experience and again with no complications during this second year, starting in 1960 we added the prophylactic cortisone regime to all patients undergoing craniotomy for brain tumor. The increased smoothness of the postoperative course has been equally apparent in this group of patients, both those undergoing suboccipital craniotomy for tumors of the posterior fossa as well as those undergoing operation for hemispheric lesions. Starting in 1961, we began to use this prophylactic cortisone regime also in patients with severe head injuries who do not regain consciousness promptly.

When the first cortisone series was started we were concerned about four possible complications of the cortisone administration: (a) effect on wound healing, (b) possible reduced resistance to infection, (c) clinically significant upset in electrolytes, and (d) possibility of hemorrhage from the gastrointestinal tract. In none of the patients in these two cortisone series nor in patients undergoing other types of craniotomies for seizures or brain tumors has there been any evidence of interference with wound healing, either from the standpoint of rate of wound healing or firmness of the cutaneous cicatrix.

Because of the possibility of reduced resistance to infection, the patients in the Cortisone 1 series were put on prophylactic penicillin and erythromycin for the duration of the administration of cortisone. No infections occurred at all, so that during the Cortisone 2 series, the penicillin was discontinued and patients were given only erythromycin during the period of administration of cortisone. Again no infections at all occurred in this second series, so that during 1961 the patients received no prophylactic antibiotics at all unless the patient had had a previous craniotomy at the same site or unless mastoid air cells or the frontal sinus had been opened during the operation. We have now to date a consecutive series of over 200 craniotomies for focal epilepsy done on a prophylactic cortisone regime without a single infection.

None of the patients on prophylactic cortisone had any clinical evidence of electrolyte disturbance and the postoperative electrolyte determinations showed no significant alterations as compared with the preoperative determinations. Minor changes in these determinations have not yet been analyzed in detail, however.

There was no hemorrhage from the gastrointestinal tract in any of the patients in these two cortisone series or in patients undergoing other types of craniotomy for focal seizures. This complication has appeared, however, in 2 patients with brain tumor undergoing craniotomy while on a prophylactic cortisone regime.

The first patient, undergoing a total hypophysectomy for acromegaly, had a pre-
vious history of gastric ulcer. He had, on his 6th postoperative day, a massive gastrointestinal hemorrhage which responded promptly to blood transfusions and appropriate gastrointestinal therapy. His further postoperative course was uneventful.

The second patient, operated upon in 1961, had von Hippel-Lindau’s disease and was on a regime with double the daily dosage of cortisone used in the Cortisone series of this report. A radical but still partial removal was carried out of a hemangioendothelioma, filling the 4th ventricle and arising from and infiltrating the upper end of the medulla and lower portion of the floor of the 4th ventricle. On her 9th postoperative day she went into shock from hidden gastrointestinal hemorrhage. This responded promptly to emergency and rapid blood transfusions, and administration of antacid medication and ulcer diet. Approximately 2 weeks later, after the patient had been off the cortisone, a second but less severe episode of gastrointestinal bleeding occurred, which ultimately required laparotomy. The hemorrhage in this instance may represent a Cushing’s ulcer as a complication of her lower brain-stem lesion, but the possibility of the cortisone causing or contributing to it must be recognized. History or symptoms of a peptic ulcer probably constitute a contra-indication for elective administration of cortisone.

Of the various symptoms and signs evaluated in this present study, it would seem that the transient postoperative hemiparesis gives the best measure of postoperative cerebral edema, since the brain removed in a temporal lobectomy does not come close to the motor system. The area of cortical excision approaches the temporal speech zone much more closely, and the variability in size and location of this speech zone from patient to patient adds a complicating factor despite the fact that removal of the temporal lobe in the dominant hemisphere in this series has been a relatively standard one from the anatomical standpoint. As a rule the cavity after removal extended 5 cm. back from the end of the middle fossa measured along the fissure of Sylvius, and 6–7 cm. measured along the base of the skull. Dysphasia, therefore, would seem to be a less useful parameter in evaluating the effects of cerebral edema than hemiparesis. Similarly, the postoperative seizures in the early period, which in some instances are caused by increased irritability of the adjacent brain and are secondary to cerebral edema, may also be the result of incomplete removal of the patient’s epileptogenic area, so that this, like aphasia, is probably a less accurate measure of the effect of postoperative cerebral edema than hemiparesis. It is of interest, however, that both in the case of aphasia and early postoperative seizures there seems to be a definite protective effect from the prophylactic cortisone regimes, and that this protective effect seems to be greater in the second series with the increased dosage as compared with the first cortisone regime.

The syndrome of aseptic meningitis occurs more frequently following large removals for focal epilepsy than following craniotomy for brain tumors, and therefore has been of interest to the group at the Montreal Neurological Institute for a number of years. This symptom is the least definite and easily quantitated of the symptoms analyzed in this report, but with strict application of the criteria described, again there seems to be a fairly definite protective effect of the prophylactic cortisone regime. More striking than the results reported, however, is the subjective impression that the patients on the cortisone regime who had aseptic meningitis were considerably less disturbed by headache, stiff neck and elevation of temperature than was the case with the patients in the No Cortisone series. In view of Jackson’s evidence that this syndrome is an inflammatory reaction of leptomeninges to the breakdown products of blood, it seems likely that the anti-inflammatory rather than the anti-edema effect of the cortisone is primarily responsible for its apparent protective effect against this syndrome of aseptic meningitis.

It seems quite clear from the results of the present study that with increasing dosage of cortisone plus a preoperative loading dose
and the continuation of the cortisone for a little longer period, the beneficial effect was enhanced. In an effort to determine whether still larger doses of cortisone would provide still greater protective effect from these postoperative symptoms, we have put our patients undergoing temporal lobectomy during 1961 on double the daily cortisone dosage utilized in the Cortisone 2 series of the present report. The results in this third cortisone series will then be compared with those of this report.

The amount of manipulation and exposure of the brain has gradually increased during the period of time covered by these three series of temporal-lobeectomy patients as a result of the introduction of depth recording with first one and then two depth electrodes and a progressive increase in the number of electrical stimulations of these depth-electrode contacts. It seems unlikely, therefore, that these results can be explained on a mechanical basis related to refinement of surgical technique.

The basic nature of the protective effect of the cortisone against cerebral edema is not clear, and further work, both experimental and clinical, will be necessary to determine the optimum dosage of cortisone and to decide whether or not some of the synthetic corticosteroids might be still more effective.

Although prophylactic cortisone is not a panacea, does not completely prevent cerebral edema, and is not a substitute for gentle manipulation of the brain, its effectiveness in promoting a smooth postoperative course and in reducing postoperative morbidity has been sufficiently striking in our experience to warrant putting our present experience on record.

SUMMARY AND CONCLUSIONS

1. A consecutive series of 31 patients undergoing a relatively standard surgical manipulation of the brain (temporal lobectomy for focal cerebral seizures) were placed on a prophylactic cortisone regime during the operation and for the first 8 postoperative days. These patients were compared with respect to the incidence, duration and severity of transient symptoms attributed to cerebral edema (hemiparesis, early postoperative seizures, aseptic meningitis, and aphasia and decrease in full-scale intelligence quotient and memory quotient in the case of dominant temporal lobectomy) with a previous consecutive series of 35 patients undergoing the same procedure at the hands of the same surgeon (T.R.).

2. A subsequent series of 35 patients were placed on a prophylactic cortisone regime of increased dosage, extending from the day before operation through the 10th postoperative day, and compared with the previous two series.

3. There was a definite lessening of the incidence, severity and duration of the above symptoms, which was most marked in the case of hemiparesis, but was evident to a lesser degree in the other symptoms as well.

4. There was an increased smoothness of the postoperative course in the patients on the cortisone regimes that was clearly evident, quite apart from the specific symptoms analyzed and quantitated roughly.

5. There were no complications from the prophylactic cortisone regimes in these two series of patients.

6. The use of a prophylactic cortisone regime seems well worth exploring further as an aid in increasing the safety of craniotomies for lesions in which postoperative cerebral edema may be a significant factor, and also as an aid in the treatment of patients with severe head injury and other neurological and neurosurgical conditions in which cerebral edema is apt to play an important role.

REFERENCES


