the individual, changes in coagulation of the blood, and disturbances of the blood-brain barrier. This constellation of factors has its effect upon the symptomatology. Coma or convulsions may develop, or hypothermia or hyperthermia require our therapeutic intervention.

Within the group of cases probably, or possibly, due to cerebral thrombosis or thrombophlebitis, otitic hydrocephalus has a tendency towards spontaneous cure except in cachectic infants or children. Indeed, one may say that it is the only variety of cerebral thrombosis or thrombophlebitis in childhood in which the prognosis is not very gloomy. Palliative measures (repeated lumbar punctures, limitation of fluid intake, and anticonvulsive medication) are in other thromboses of little avail. The advisability of anticoagulant therapy is still under discussion. It certainly carries with it a serious danger if the cerebrospinal fluid is blood-stained. Our own view, however, is that, having regard to the severity of the illness before us, especially if the cerebral signs are progressive and the seizures show a tendency to become generalized, it is better to make use of the chance of success which the anticoagulant remedies offer. We would say that these remedies are particularly indicated where (though this is rare in childhood) the cerebral event is accompanied by thrombosis of the vessels of the limbs.

In the fulminant encephalopathies without hemiplegic signs and where there is no suggestion of thrombosis, the essential aim is the restoration of the ionic equilibrium of the blood. One must attempt to block the autonomic system by chemical insulation and should not, therefore, hesitate in the presence of convulsions to give barbiturates by transfusion.

Let us suppose a diagnosis of encephalitis, and probably a virus encephalitis, suggests itself; most of the viral encephalitides are, with rare exceptions, insensitive to both the antibiotics and the sulphonamides. We shall therefore reserve these two groups of substances for the treatment of the accompanying secondary infections. On the other hand, we will, of course, employ salicylates and iodine in high dosage as we did during the European encephalitis epidemics in 1920-5. Recently a place has been given to cortisone and corticotrophin in the treatment of these acute forms as well as the late forms of post-infectious encephalitis. In the acute forms we have not had the impression that these substances are of undoubted value. In regard to the late forms it is difficult to evaluate a therapy in a condition where the natural outcome is in any case so favourable.

In the presence of coma, and especially in the presence of those prolonged states of disturbed consciousness which are seen in late post-measles encephalitis, the treatment will be that usually given in the state of common shock.

I have mentioned the serous encephalitides; their treatment will also be that of states of severe shock.

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THE ELECTROENCEPHALOGRAM IN MYXOEDEMA COMA

CLINICAL AND ELECTROENCEPHALOGRAPHIC STUDY OF THREE CASES*

BY

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Myxoedema as a cause of death was first described by Hadden in 1879, when a patient under the care of Ord (1878) died in coma. That myxoedema might be other than an easily remediable condition, however, was largely ignored until recent reports made it clear that myxoedema coma was not in fact a rarity, and that it was almost invariably fatal. During the past four years at least 20 cases have been reported in this country alone—all these patients died except one in the series reported by Malden (1955). Summers (1953) reported five cases in which the patients died in hypothermic coma, the initial temperatures varying from 23.9 to 35° C. Treatment by immersion in a warm bath, thyroxine, and cortisone had no effect. Dyson and Wood's (1956) patient died after 100 µg. of triiodothyronine had been given intravenously. Malden (1955) reported three cases of hypothermic coma; two patients died but one recovered after treatment with electric blankets and intravenous corticotrophin and thyroxine. He noted that hypothermia was present in all three cases, the blood sugar was normal, and there was absence of shivering (due, he supposed, to failure of the thermo-regulatory centre).

Reports of recovery have been remarkably few. Apart from the case of Malden (1955) only two other survivals have been described; one a very early case which recovered spontaneously in 24 hours (Brotmacher and Coghill, 1953), the other a condition of deep coma in a woman of 83 who recovered with thyroid extract, but in whom the temperature was not noted (Martin and Whittle, 1953). That it is an extremely dangerous condition, however, is evident, and Summers (1953) thought that the changes produced by extremely low body temperatures are apparently irreversible or that the body reaches a state in which it can no longer respond to thyroxine.

Coma in myxoedema patients presents a clinical picture similar in many respects to coma in hypopituitarism (Sheehan and Summers, 1952). Caughey and Garrod (1954), in discussing factors which could precipitate coma in hypopituitarism, mentioned infection, hypoglycaemia, hypersensitivity to drugs and anaesthesia, sodium depletion, water retention, cerebral anoxia, and hypothermia as possible factors. It is possible that some of these factors may also operate in the production of myxoedema coma and that adrenocortical failure or even some degree of pituitary failure (Hubble, 1955) may be concerned.

It is interesting to note the effect of artificially induced hypothermia in man and animals. Dundee, Scott, and Mesham (1953) found that cardiac arrest occurred in three dogs at temperatures of 23° C., 25° C., and 26.5° C. Swan *et al.* (1953), however, described temperatures ranging from 21.5 to 26° C. in 15 patients

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undergoing cardiac operations with only one fatality. It therefore seems unlikely that hypothermia is the sole factor in preventing recovery from myxoedema coma, though it is doubtless an important one. One would expect that cerebral activity would be markedly affected in these cases (as of course the state of consciousness indicates). Scott (1955) has described the E.E.G. changes in hypothermia in man and found that cerebral electrical activity was much reduced, though it did not entirely disappear.

I have been able to find only one report of the E.E.G. changes in myxoedema coma. Biemond (1955) described the case of a woman of 66 who was stuporous when an E.E.G. was taken. She later died from bronchopneumonia and was found to have cirrhosis of the liver and cerebral changes of Wilson's disease as well as thyroid aplasia. There was no mention of hypothermia. The E.E.G. revealed "a dominant theta rhythm of 6-7 cycles per second at 30-40 microvolts; there was also some generalized 2-3 per sec. delta activity." Serial E.E.G.s have been taken in a case of hypopituitary coma by Salmon (1956). The coma was associated with hypothermia (87° F.; 30.6° C.), hypoglycaemia (blood sugar 35 mg./100 ml.), and sodium depletion (serum Na 276 mg./100 ml.). Treatment restored the patient (and her E.E.G.) to normal. He remarks that the form of the initial tracing (high-amplitude delta activity) was probably due to the hypoglycaemia.

I have been able to obtain E.E.G.s of two patients in myxoedema coma and a third patient during recovery from the same condition. They illustrate three phases of the same process and the accompanying changes in cerebral electrical activity.

Case 1

A married woman aged 62 was admitted in coma to the Royal Southern Hospital, Liverpool, under the care of Dr. R. W. Brookfield, on January 14, 1958. Although a known case of myxoedema, she had taken no thyroid for two years. On the night of January 13 (a very cold night) she went to bed, and next morning was found on the floor in her nightdress and could not be roused.

On admission her rectal temperature was 24.5° C. (76° F.). She was deeply comatose and unresponsive to any stimuli. An obese woman, she presented a grossly swollen, typically myxoedematous facies with coarse, dry, ice-cold skin. Her axillary hair was sparse, but her pubic hair was normal. Her respirations were slow and grunting, her pulse was impalpable, and her heart rate was 20 a minute. Her serum cholesterol was 440 mg., sodium 285 mg., potassium 16 mg., and sugar 80 mg./100 ml. Her E.E.G. on admission (Fig. 1) showed in addition to artifact (from head movement and snoring) an almost flat trace with a few random 4-5 c./sec. waves (10μ V). She was catheterized on admission, the bladder containing 120 ml. of urine. As it was known she had passed urine about 12 hours previously it was possible to make an estimate of 17-ketosteroid excretion during the preceding 24 hours (1 mg.). Treatment was begun as follows: (1) She was covered with blankets and a heat cradle placed outside; (2) 100μ g. of L-triiodothyronine in 500 ml. of 5% glucose-saline was given by intragastric drip; and (3) 100 mg. of hemisuccinate hydrocortisone was given intravenously.

During the next 24 hours her general condition did not change, but her rectal temperature rose from 24.5 to 27° C. However, she died 24 hours after admission. Further blood was taken about 10 minutes before death, and the estimations were as follows: serum sodium 320 mg., potassium 15 mg., and sugar 135 mg./100 ml. She was catheterized after death—there had been no secretion of

urine in the previous 24 hours. A post-mortem examination was not permitted.

Comment.—This is the type of case which led Le Marquand, Hausmann, and Hemsted (1955) to remark: "We were impressed . . . with our helplessness to deal with the final phase." It is difficult to see how with our present therapeutic armamentarium this patient could have been saved, especially as in recent cases intravenous triiodothyronine has brought no improvement (Dyson and Wood, 1956) and may indeed be dangerous in producing a too rapid acceleration of metabolism. The E.E.G. tracing in this case indicates that cerebral electrical activity was reduced to a minimum although it does not follow that any permanent brain damage would have ensued had recovery taken place.

FIG. 1.—Case 1. E.E.G. on admission.

FIG. 2.—Case 2. E.E.G. on June 14, 1955.

Case 2

This case illustrates the condition of myxoedema coma at an earlier stage, when recovery was possible with the use of thyroid extract.

A married woman had a subtotal thyroidectomy for non-toxic goitre in 1953 at the age of 45. Shortly after this she was judged to be hypothyroid and given thyroid, $1\frac{1}{2}$ gr. (0.1 g.) daily, with benefit. In June, 1955, her skin was somewhat dry and she was occasionally forgetful, but was otherwise quite well. Her E.E.G. at this time (Fig. 2) showed a 9 c./sec. alpha rhythm with an amplitude of 10μ V.

On September 3, 1957, she started to vomit, and two days later was admitted to the Royal Southern Hospital. She was found to be drowsy and uncooperative; she answered questions with difficulty and was dehydrated. The dehydration was corrected, the vomiting ceased and her serum electrolytes and N.P.N. were normal. However, she

became gradually weaker, more apathetic and quiet, drowsy, and difficult to rouse. By September 12 she was stuporous. She slept most of the time and would respond, by moving, to shouting and to painful stimuli, but would utter no more than a few drowsy words. She felt very cold: her temperature was recorded as 96° F. (35.6° C.), but was probably lower as a clinical thermometer was used. Her N.P.N. was 20 mg./100 ml., serum cholesterol 140 mg./100 ml., and 17-ketosteroids 2.8 mg. a day. At this stage it was discovered that she had not received any thyroid for six months. A diagnosis was made of myxoedema coma, and thyroid extract was given (via a Ryle tube). An E.E.G. (Fig. 3) obtained just before thyroid was started showed a

FIG. 3.—Case 2. E.E.G. on September 12, 1957.

FIG. 4.—Case 2. E.E.G. on October 2, 1957.

mixture of generalized 3 c./sec. delta activity and 5–6 c./sec. theta activity with an amplitude up to 50 μ V. A very small amount of 8–9 c./sec. alpha activity was detected in the parieto-occipital leads and a little rapid activity anteriorly.

She remained drowsy and apathetic (with lapses into coma when she could not be roused) for several days, although no further vomiting occurred. However, on September 20 she appeared more alert, though still sleepy and lethargic. Progress continued slowly, and on September 25, when a further E.E.G. was obtained, she was sleeping less and could recognize objects and talk lucidly at times, though her attention wandered. The E.E.G. showed some improvement—a fairly regular 7–8 c./sec. occipito-parietal alpha rhythm with much 4–6 c./sec. theta and a little low-amplitude delta activity in the temporal regions.

On October 2 she looked much less hypothyroid, was more alert, and could hold a cup. Her speech was slow and her memory poor. The E.E.G. (Fig. 4) showed a somewhat irregular 8 c./sec. alpha rhythm with scattered 6–7 c./sec. waves in all leads. On October 10 she was starting to walk and could feed herself. She knew the date and could tell the time, but could not attempt the 100–7 test. Her skin was still dry without axillary sweating,

otherwise her appearance was normal. Her E.E.G. showed a mixture of 8 and 9 c./sec. alpha activity posteriorly with a regular 7–8 c./sec. rhythm anteriorly. She was discharged on October 12, taking thyroid, 3 gr. (0.2 g.) daily. On November 26 she was seen again and appeared euthyroid, her only deficit being a rather poor memory for recent events, and a poor performance at mental arithmetic (failing the 100–7 test at 79). The E.E.G. showed an 8–9 c./sec. alpha rhythm, with no slow activity.

Comment.—In this case the episode of coma (less severe than in Case 1) appears definitely to have been associated with her hypothyroid state, although it was probably precipitated by vomiting and dehydration. At the time when she was comatose there was no clinical or biochemical evidence of dehydration, nor, as she was receiving dextrose by the intragastric route, could hypoglycaemia have played a part. Focal neurological signs were absent. Moreover, the gradual response to thyroid extract alone with concomitant gradual E.E.G. improvement suggests that this was indeed an episode of myxoedema coma. Treatment, of course, was begun at a much earlier stage than in Case 1 and when a response was possible to an agent as slow in action as thyroid extract. It seems reasonable to suggest that had the condition been allowed to progress further a more rapid agent such as thyroxine or triiodothyronine would have been required, and if treatment had been further delayed no response to treatment might be forthcoming. One may postulate a dynamic concept of a condition which in its earliest stage will respond to small doses of thyroid extract and in its latest stage may be irreversible because of damage to vital centres, including the brain.

An intermediate stage in which more rapid and energetic treatment may yet produce a response is exemplified by Case 3. (Also reported by Surtees and Ginsburg, 1958.)

Case 3

A married woman aged 70 had complained for one year of increasing coldness, gain in weight, deafness, and a puffy face. For one week she had been sleeping more than usual. On the morning of January 22, 1957, she had been found in bed and could not be roused. On admission to Walton Hospital under the care of Dr. V. K. Summers, she was stuporous and very restless. She looked myxoedematous with a yellowish puffy face and dry scaly skin. Her rectal temperature was 87° F. (30.6° C.). The axillae were dry and hairless, and the pubic hair was scanty. Neurological signs were absent. The pulse was regular at 52 a minute, the heart sounds were faint, and the blood pressure was 180/100. Her blood sugar was 122 mg., serum potassium 19 mg., serum cholesterol 645 mg., serum sodium 290 mg./100 ml., and haemoglobin 82%. The E.C.G. was negative T123.

She was treated initially by means of a heat cradle, and 100 μ g. of L-triiodothyronine was given by Ryle's tube. There was little change in the first 24 hours, but by 48 hours she was less drowsy, was speaking a few words, and her rectal temperature was 99° F. (37.2° C.). Treatment was continued with triiodothyronine, 40 μ g. a day. By January 25 she was speaking and was much more active. On February 2 the B.M.R. was –25%. On February 6 she was speaking rationally, and seemed normal mentally; the pulse and temperature were normal. An E.E.G. (Fig. 5) showed a dominant 8 c./sec. alpha rhythm with a good deal of scattered 7 c./sec. activity. The highest amplitude was in the temporal leads (up to 14 μ V) with very low frontal voltage. Triiodothyronine was stopped on February 13 as she felt weak and giddy, started to vomit, and developed tachycardia. Cortisone, 25 mg. a day, was given. On February 18 her condition had improved again: a further E.E.G. (Fig. 6) showed an 8 c./sec. alpha rhythm

in the parieto-occipital regions (15 μ V) with an increase in the amount of 7 c./sec. activity and a dominant 7 c./sec. frontal rhythm (suggesting that some deterioration had taken place).

Unfortunately the patient died suddenly on February 18. Necropsy revealed that death was due to a thrombosis of

FIG. 5.—Case 3. E.E.G. on February 6, 1957.

FIG. 6.—Case 3. E.E.G. on February 18, 1957.

the right coronary artery. The thyroid was small, consisting almost entirely of fibrous tissue. The pituitary and adrenals were normal.

Discussion

The electrical activity of the brain is correlated closely with its metabolism. The E.E.G. records differences in potential through the scalp and thereby reflects the behaviour of the brain in its internal environment. Changes in metabolic factors such as oxygen tension or availability of glucose are quickly reflected in the E.E.G. (Davis, Davis, and Thompson, 1938). Shifts in electrolyte balance, pH, and particularly CO₂ tension significantly influence the brain waves (Gibbs, Williams, and Gibbs, 1940). This prompt response to alterations in the internal environment of the brain provides a convenient means whereby the physiological effects of disease elsewhere may be detected and correlated with clinical observation.

Neuropsychiatric disturbances occur commonly in endocrine disorders, and abnormalities in the E.E.G. have been described in such conditions as hypopituitarism, Addison's disease, Cushing's syndrome, hyperinsulinism, myxoedema, and thyrotoxicosis. The biochemical disorder differs in these conditions, although ultimately the cerebral disturbances which may occur relate to altered respiratory exchanges in the brain cells.

The E.E.G. itself is an expression of the metabolic activity of the brain cells, and its normal features depend on numerous humoral and tissue factors whose stability is regulated, *inter alia*, by the endocrine system. It is logical to suppose, *a priori*, that cerebral bio-electrical activity undergoes important modifications in endocrine disturbances, and this is in fact what occurs.

Three cases of myxoedema coma have been presented. In the first and most severe case, which was associated with an extreme degree of hypothermia (24.5° C.), very little cerebral activity was demonstrable in the E.E.G. record. In Case 2 when coma was light the record showed generalized theta and delta activity of normal or somewhat reduced amplitude. In Case 3 the E.E.G. taken during recovery from coma resembled the records seen in untreated myxoedema of moderate severity in the waking state (Nieman, 1958). Numerous studies have implied that E.E.G. changes parallel changes in cerebral metabolism, and that in delirium and coma from widely differing causes the E.E.G. can be correlated with the clinical evaluation of the level of awareness (Engel and Romano, 1944). In general the E.E.G. in mild delirium shows a shift to frequencies below the normal alpha range; with more marked delirium there is greater slowing of frequencies accompanied by the appearance of intermittent low-voltage fast activity. In a severe derangement of cerebral metabolism the E.E.G. consists largely of irregular moderate to high-voltage 1–8 c./sec. slow waves. Engel and Romano (1944) think it is possible to correlate the E.E.G. picture and the level of awareness with considerable precision. It appears, however, from these cases that, whereas in other forms of delirium and coma the E.E.G. waves increase in voltage whilst decreasing in frequency, in myxoedema both frequency and amplitude decrease together.

There is evidence that the lowering of voltage is due, in part at least, to the hypothermia which may accompany the reduction of cerebral metabolism in myxoedema coma. The extent to which the E.E.G. of a patient in severe hypothermic coma with virtually no cerebral electrical activity demonstrable can recover is, however, uncertain. Whether the E.E.G. will return to normal and whether there will be any residual cerebral symptoms are problems which must remain until more satisfactory treatment can be employed to resuscitate such patients. Some residual mental impairment is present in Case 2—namely, poor memory for recent events and inability to calculate—and this may be the result of cerebral damage during myxoedema coma.

It is notable that hypoglycaemia, which may play a part in the production of hypopituitary coma (Caughey and Garrod, 1954), was not present in any of the above cases, nor does the E.E.G. resemble the tracing seen in hypoglycaemia (Davis *et al.*, 1938); and sodium depletion does not seem to have been responsible. Adrenocortical function, however, as implied by the 17-ketosteroid estimation, was reduced in all three cases, and in the most severe case was only 1 mg. in 24 hours. This does not necessarily imply that hypofunction of the adrenal cortex is responsible for the production of coma in myxoedema. It has been known for some time that adrenal cortex function might be sometimes, though not invariably, depressed in myxoedema. Friedgood (1942) demonstrated low excretion of 17-ketosteroids in 9 out of 10 patients with spontaneous and post-thyroidectomy myxoedema. Therapy did not raise the values.

Hill, Reiss, Forsham, and Thorn (1950) found that 9 out of 11 patients with primary hypothyroidism given a single 25-mg. injection of corticotrophin had an inadequate adrenal response, and Hubble (1955) found a defective water-load response in myxoedema. In a personal series of 18 patients in whom the 17-ketosteroid excretion was estimated the highest figure was 5.7 mg. a day and the lowest 0.95 mg. a day. In all but two patients the figure was below 5 mg. a day. Boulet and Mirouze (1955) believe that if adrenal function is investigated in myxoedema then hypocorticalism will be found almost as constantly as hypothyroidism: they emphasize, however, that thyroid hormone is the only treatment indicated. It is difficult, therefore, to assess the role of the adrenal cortex in the mechanism of myxoedema coma. It is unlikely to be a major factor, as clinical evidence of adrenal insufficiency is not found in myxoedema coma, and Summers (1956) found that cortisone was of no value in the treatment of myxoedema though it might produce some mental alertness. Moreover, the E.E.G. in myxoedema and in myxoedema coma does not resemble the records seen in Addison's disease (Hoffman, Lewis, and Thorn, 1942).

It is probable that hypothermia, which is a constant feature of myxoedema coma, and deficiency of thyroid hormone itself, are the most important factors, though precipitating factors may include environmental cold (note the seasonal variation), trauma (Macdonald, 1958), and dehydration (Case 2 of present series). Macdonald (1958) suggests that during periods of stress the requirement of thyroid may increase like that of the adrenal corticosteroids. In advanced myxoedema coma the tissues may become unresponsive to thyroid hormone and the condition be irreversible. Extreme reduction or absence of cerebral electrical activity may indicate this state of affairs and a poor prognosis.

Summary

The clinical features and E.E.G. traces in three cases of myxoedema coma are described. It is suggested that thyroid hormone deficiency and hypothermia are the most important factors in the production of coma in myxoedema. The E.E.G. may be correlated with the changes in cerebral metabolism.

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VALUE OF THE VOICE IN DIAGNOSIS OF MYXOEDEMA IN THE ELDERLY*

BY

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Adult myxoedema was first described by Gull in 1874, and it was Ord (1878), who first called it myxoedema. In Allbutt's *System of Medicine*, first published in 1897, Ord gives a masterly description of a female case, and there have been numerous studies of this disease since that time (Report, 1888; Bramwell, 1892; Howard, 1907; White, 1913; McCarrison, 1917; Janney and Henderson, 1920; Lerman, 1937; Means, 1937; Rundle, 1951; Spence, 1953; Werner, 1955; Asper and Wiswell, 1956). There seems to be little disagreement that the classical case of myxoedema is easy to recognize, but the onset is insidious, and by the time the diagnosis is made the disease has often been present for years (Means, 1937). Myxoedema is apt to be overlooked as a cause of symptoms in those over the age of 60, if only for the fact that so many in this age group ascribe the symptoms to their advancing years and regard them as inevitable.

The symptomatology of myxoedema tends to be diffuse and equivocal. Most people over the age of 60 attending as hospital out-patients will, on questioning, admit to some symptoms commonly associated with myxoedema. These are: increasing weight, constipation, falling hair, sensitivity to cold, dry skin, difficulty in walking, vague limb pains, failing of memory and concentration, slow thought, and dizzy spells. Cases of myxoedema respond very well to adequate treatment with thyroid. This, coupled with the fact that untreated cases of myxoedema are unusually prone to death from infection and coma (Hadden, 1879; Report, 1888; Summers, 1953; Le Marquand *et al.*, 1953, 1955; Malden, 1955; Marshall and McCaughey, 1956; Surtees and Ginsberg, 1958; Emslie-Smith, 1958), makes early diagnosis important.

Almost all authors (Bramwell, 1892; White, 1913; McCarrison, 1917; Spence, 1953) mention the alteration of the voice that may occur with the development of the disease, but few stress its value, although Means (1937) states that "the voice change and speech peculiarities, taken together, form a combination almost pathognomonic of the disease"; and Asher (1949) has described the voice as being like a bad gramophone record of a drowsy, slightly intoxicated person with a bad cold and a plum in the mouth.

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