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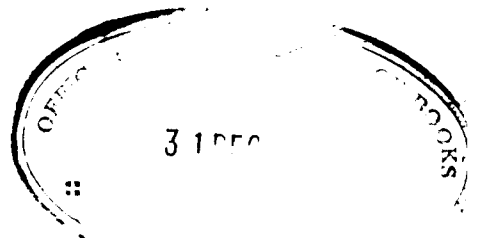
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[No. 3

NEUROLOGY

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NEUROLOGY

Vol. II

No. 3

CONTENTS OCTOBER 1954

Hypothermia in relation to Neurological & Neuro-surgical Problems	-	-	-	Baldev Singh and Jacob Chandy	43
Anaesthesia in Neuro-Surgical Procedures	-	-	-	V. Rajagopalan and B. Ramamurthi	54
Gargoylism	-	-	-	B. Ramamurthi	58
Association Notes	-	-	-		63

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1. Wells P. Eagleton, M.D., Brain Abscess (its surgical pathology and operative technic) Newark, N. J.: The Macmillan Company. 1922. p. 157.
2. Frederick P. Moersch, M.D., & George P. Sayre, M.D., Neurologic Manifestations associated with dissecting aortic aneurysm. The Journal of the American Medical Assn., Vol. 144, No. 14: December, 1950. 1145-1146.

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No. 3

HYPOTHERMIA IN RELATION TO NEUROLOGICAL AND NEUROSURGICAL PROBLEMS

BALDEV SINGH and JACOB CHANDY, Vellore.

INTRODUCTION

If legend can be a basis for any scientific thought, then hypothermia has a rich background in Indian mythology and mysticism. Some of the travellers in Tibet, and a book by name Tibetan Book of the Dead, have given descriptions as to how some ascetics would go into a state of inanimation for shorter or longer periods by burying themselves in the Himalayan snows and how accurate the ritual to revive them had to be, to bring them out safe from such suspended animation. Any time there was a flaw in administering nutrition or artificial warmth to the body, the recovery was jeopardised. These examples, however, were few and far between, and hence it is not easy to find full details and facts about the procedure.

EVOLUTION OF THERMO-REGULATION

The recent interest in the physiological and pathological effects of exposure to low temperature was greatly stimulated by the pioneer studies of Fay² and his co-workers, who reported in 1940 that humans could survive unexpectedly prolonged reduction in body temperature with no apparent ill-effects. Temperature is a measure of molecular agitation and there is very little known so far if there is any state of matter without such agitation. To think of a proton, the static energy, without electron, the dynamic energy, is well nigh impossible according to the present knowledge of physics. Temperature regulates the growth and metabolism in the widest sense. It limits the survival and distribution of animals on the earth. The temperature limits for active cell life are from about 0 to about + 45°C and many higher animals can live only within a still narrower range.

The cold-blooded or poikilothermic animals conform passively to the environmental temperature. They enter into a state of dormancy and lowered metabolism. These are the animals which make a large percentage of life in the sea or even on the crust of the earth.

The heterothermic animals according to thermic circumstances can behave like poikilothermics and yet have a rudimentary development to be able to keep a constant temperature within a certain range. The homoiothermics are comparatively few. They have a well developed mechanism to keep their body at a certain state of warmth and to maintain it they can control the production as well as the loss of heat from the body.

The temperature relations of animals to their environments are closely related to the temperature properties of water¹². Animals are 70 to 90% water. The low heat conductivity of water protects the animal against too sudden a rise or fall of temperature. The bound water inside the cell protects the cell against freezing and changing into a frozen mass. The unbound water by its circulation with the fluids of the body distributes the heat to the various tissues. The high heat of vaporization of water is a factor potent against high rises of temperature. The free water, however, is liable to freeze and lead to damage to the animal. Dehydrated animals, therefore, stand the extreme cold much better.

It is the quantity of bound water as compared to the free water which in evolution determined the change from Poikilothermy through Hetero, to Homoiothermy. The reptilia are the intermediate stage and they have variable proportions of these two types of water. Those who have more

bound water gave rise to the homoiothermic species. The bound water gives to the cell the property of vitrification — that is to form a sort of gel which does not freeze nor boil easily but remains fluid at adequate temperature. The temperature of vitrification determines the range of optimum temperature in a given species of animals. Vitrification is the quasi-chemical protein to water relationship and has a genetic basis. Proteins in the protoplasm disintegrate at low temperature without proper quota of bound water. The temperature for this disintegration is the lowest basic necessity of the cell of each species. Any temperature below this produces chill coma. Amongst the various cell communities in the organism, the neuron has the maximum of water bound to its proteins. It survives high and low ranges of temperature with greater tenacity. Vitrification also gives protection to cell life by its property of keeping the proteins intact against sudden freezing and prolonged cooling. The division of interstitial tissue into small compartments containing various quantities of bound water further adds to this protection for it is easier to affect the temperature of a mass of water when it is all together than when it is parcellated.

The binding of water to the proteins and the nature of the proteins in the animal depend on species characteristics and upon the quality and quantity of enzymes. Whilst in poikilothermics and in infants of all species, the phosphorilase is more predominant, in hetero and homoiothermics the oxidases play a more important role. The enzyme activity as well as the binding of water is further helped by the developing endocrines¹³ particularly, the thyroid, the supra-renal and the pituitary glands respectively. These glands in addition affect the basal metabolic rate. Thus when the poikilothermy passes to heterothermy, these changes in endocrines and B.M.R. react slowly and hence are suitable for the animal to live in the thermal medium of water. Terrestrial life needs meeting quickly with the emergency of variation of temperature. The chief function of nervous

control is to meet these emergencies and thus keep the body temperature stable.

The nervous control acts as a thermostat which is set for a certain range of temperature. It has influence on heat production and heat loss to hold the temperature at a steady range. In the infant animal when myelination¹⁴ is not yet complete the temperature regulation is not quite so stable as in the adult. This nervous thermostat is in the hypothalamus¹⁵ in its anterior part near the supra-optic nuclei and also its posterolateral part. It is both facilitatory and inhibitory and is acted upon by the frontal lobes and by the emotional centres. The effective stimulus for this heat regulating mechanism is the temperature of the blood. This region of the hypothalamus has a very rich capillary³ blood supply. The capillaries here are almost like sinusoids. The blood, however, is able to affect the neurons to act not only by the heat it has but also by chemical means due to chemo-reception which these neurons exhibit to the circulating hormones and other substances which can raise or diminish the temperature. The hypothalamic centres acting by the descending pathways through the autonomic fibres either raise the body temperature by muscle contraction as in shivering or diminish it both by radiation of heat due to vaso-dilatation of skin vessels and by evaporation due to sweat secretion.

The central mechanism of heat regulation is not only effected by blood heat and chemo-reception, but is also stimulated by the peripheral nerve end-organs of heat and cold, but whilst the latter has a graduated and mild effect the former produces more sudden and drastic changes. It must be stated, that the response of the animal organism to low abnormal ranges of temperature is species-specific and depends on developmental capacities for response, and the intrinsic mechanisms for adaptation to thermal stress. The animal therefore reacts to abnormally low environmental temperature in two phases; the first is the phase of effective mobilisation of defences when body temperature is maintained within broadly physiological limits, and the

second, is the phase, when the assault from without is so intense or prolonged that the intrinsic defences are overwhelmed and the body temperature falls below recognised physiologic limits. The techniques for survival are, in homoiothermics especially, qualitatively different in these two phases. In the first phase, the mechanism of alarm reaction and general adaptation comes into action. The region in the hypothalamus which controls A.C.T.H. liberation from the pituitary is located very close to the zone which controls heat regulation. Any sudden change in the temperature acts as an alarm stimulus and sets to action the protective mechanism through the pituitary suprarenal axis which brings about biochemical changes to meet the situation. The role of the hypothalamus and its correlation to the A.C.T.H. secretion mechanism is elaborated and discussed at length in the later part of this paper. In the second phase, this mechanism is overpowered and exhausted. For its survival in this phase each organ has to use its own protective stability to survive the emergency. It depends on the structural and biochemical organisation of the tissue involved.

There are three methods to produce hypothermia artificially:—

(1) By surface cooling, i.e. the immersion of the unshaven animal in cold water as has been elaborated by Talbot¹⁸ and modified by later workers. More recently the same results have been achieved by the use of cold air or cold blanket. The cold blanket contains coils through which water or air is kept circulating at the low temperature required.

(2) By an artificial arterio-venous anastomoses as practised by Delorme¹.

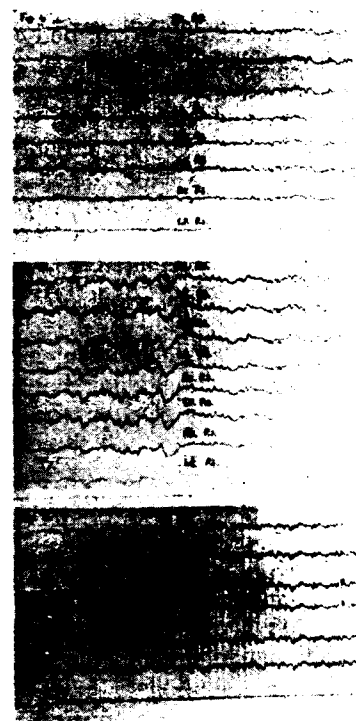
(3) By "artificial hibernation" which depresses the temperature regulating mechanism as developed by Laborit¹.

Surface cooling in dogs is done by exposing the unshaven animal to temperatures from 10—20°C. The premedication and preparation is as follows:—

The dog is given promethazine 5 mgms/kgm. body weight, subcutaneously. Anaes-

thesia is induced with intravenous Thiopentone sodium 0.15 gm/kgm. body weight, followed immediately by Succinecholine 5 mgms/kgm. body weight. The trachea is intubated, and anaesthesia is maintained with oxygen and ether in closed circuit apparatus with CO₂ absorption. Rectal temperature, pulse rate, blood pressure, E.C.G. and oxygen consumption are taken before and after the experiment. Further drop in water temperature is brought about by adding crushed ice. The temperature of the dog is stabilised at 22—34°C for 30 minutes. Re-warming is done in the bath gradually raising the temperature to 40°C. When shivering appears and the dog becomes conscious, it is removed from the bath. The temperature drops a couple of degrees more after removing the water from the iced bath. The temperature is measured with the thermo-couple. This, however, is not necessary, for rectal temperature is quite accurate within + 1°C. Blood pressure is taken with a mercury manometer. E.C.G. lead II runs all the time. Oxygen consumption is taken with a Spirometer. Defibrillation is done with intra-cardiac injection of adrenalin 0.5 mgm (1 in 1000) and shock voltage 150 to 200 volts with the duration of 0.2 milliseconds. We have not used this method and hence can say nothing more about it.





In human beings it is useful for baby patients. It is a slow method and produces skin ulceration. Talbot emphasizes that sometimes the subcutaneous fat solidifies, and leads to large gangrenous areas.

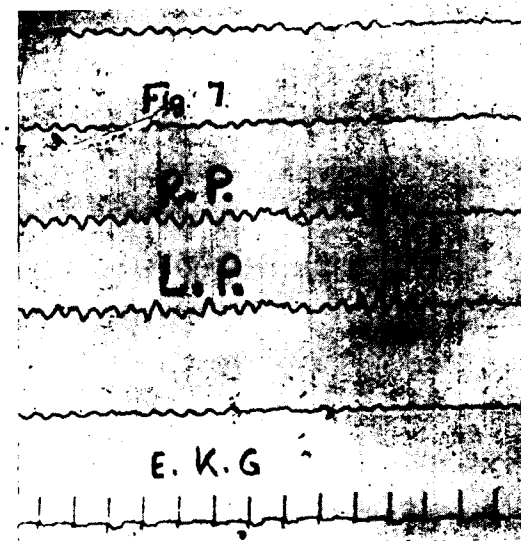
We have used modified Delorme technique of arterio-venous shunt. Protocols of an experiment done on a dog gives all the details of our technique.

9 p.m. Weight of dog: 6 lbs. normal and healthy. E.E.G. record taken without sedation whilst the dog was quiet, without any pre-medication by bipolar parasagittal electrodes (Figs. 1 & 3), both while the dog was awake as well as when it was drowsy. The brain waves showed a frequency of about 10-12 (Fig. 4) per second during awake period and 6-7 per second during drowsiness (Fig. 5). The experiment was done in the E.E.G. lab. on an animal experimental table. 9-30 p.m. the dog was anaesthetised with ether, both the groins shaved and sterilised for sterile operation. The site of the operation was draped and the femoral vessels were exposed in the right groin by a transverse incision 1" in length along the poutart ligament in the upper part of the femoral triangle. The femoral artery and vein were clearly defined. 150 international units of Heparin were given in the femoral vein, exposed

on the right side. A sterile polyethylene tube was bevelled at both ends and filled with citrate solution before the ends were introduced into the femoral artery and the vein. The tube was 10 feet in length and had a bore of 1 mm. It was kept in ice all through the experiment (Fig. 2). Rectal temperature of the dog was recorded before starting the experiment and it was 37°C. Subsequently, readings were taken every half hour, and the temperature gradually dropped to 32°C in four hours. During this time the E.E.G. record was taken every time the temperature dropped by half a degree. The record showed gradual slowing like as in an animal that passes into deeper and deeper anaesthesia (Fig. 6). Shivering became more and more intense as the temperature dropped to 33°C. This had to be controlled with an occasional whiff of ether. With each shivering the temperature drop became more slow. The experiment was terminated at 2 a.m. with the heart of the dog in a fairly good condition. The polyethylene tube was removed, the femoral artery and vein were ligated, and the wound was closed with silk sutures, and dressed with Tinc. Benzoin. The animal vomited twice as consciousness began to return in about 45 minutes time. Next day, the temperature of the dog was 39°C, but he still was partially drowsy although taking his feed spontaneously. Brain waves were still comparatively slow. Twelve hours later there was complete regaining of consciousness and the E.E.G. record showed normal awake activity.

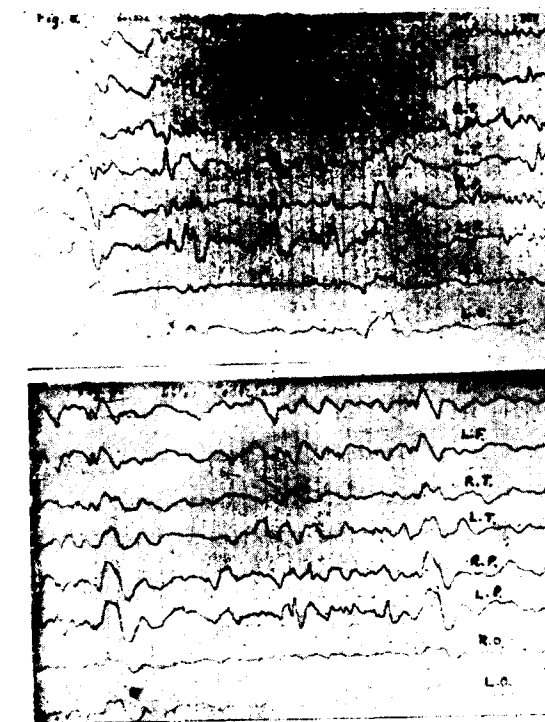
Remarks: In the choice of the dog, it has been found that an animal of about 8-10 pounds is the best to deal with this experiment. This eliminates difficulty experienced in intubating the vessels. Shivering has to be prevented by giving sympatholytic drugs. Heparin has to be repeated every three hours, otherwise there is likely to be clotting in the polyethylene tube. In the choice of polyethylene tube it has to be seen that it does not accommodate too much of blood so as to effect grossly the total circulating volume, nor should it offer too much resistance, so that arterial pressure is unable to push the blood into the vein. In the latter case, a three-way stop cock and syringe has to be used to keep the blood going from artery to vein through the tube. During the recovery phase the warming should not be done quickly otherwise there is such a peripheral dilatation of vessels that the animal bleeds in his own vessels and dies of shock.

Subsequently, we have used the same technique in three monkeys of 6-7 lbs.



weight. The E.C.G. was included in the E.E.G. recording (Fig. 7). The monkeys do not stand the arterio-venous shunt so well as the dog. For them the length of the polyethylene tube has to be reduced (1 meter in length, and 1 mm. in bore). The advantages of this method are the quick effect, the absence of skin complications and better control. Carotid to jugular anastomoses¹⁶ is more effective as it brings down the temperature much quicker due to the earlier and direct effect on the hypothalamus. It may, however, be remarked that a younger animal stands the hypothermia better than the adult, the lower species better than the higher ones, and the poikilothermics better than homoiothermics.

In some continental clinics, various agents are given before, during and after operation to lower metabolism by producing a state of artificial hibernation, and a large volume of work has been done in France in this connection. Hypothermia is



often used as an ancillary method. The drugs prescribed, facilitate cooling by depressing the temperature regulating mechanism. Much of the success depends on the use of Largactil. This drug depresses various parts of the autonomic and central nervous system as also the endocrines. We have attempted this method by using the Lytic cocktail, containing Largactil, Phenergan and Pethedine in the following proportions, run in the form of a drip for 72 hours to 4 days or by injecting the solution I.V. every 30 minutes or so. The formula used is as follows:—

Phenergan 6.25 mgms.
Pethedine 12.5 mgr.
Largactil 6.25 mgms. each in ¼ cc. distilled water.

The dose used is 1/20-1/10 cc. of the mixture kilogram body weight for 24 hours. It usually slows the brain waves and effects sleep, but the temperature drop is almost negligible (Figs. 8 & 9). We have yet to

see how it effects the normal people; for all the three cases in whom we used this drug, were very sick children suffering from acute virus encephalitis, for which this treatment is recommended. Werner²⁰ et al, however, emphasize that the drug shows its effect on hypothalamus best in decorticated animals. Evidently, cortex has an inhibitory effect and hence in species with highly developed cortex the drug will have a minimal effect.

There has been extensive data collected by various workers on hypothermia as it affects the nervous system, i.e. (1) Animal studies, (2) Neurological and Psychiatric effects on man, (3) Miscellaneous facts correlated to the neurological changes.

(1) Animal studies:— Neural effects of cold exposure are considered conveniently from 3 standpoints: (a) the local effect of cold on peripheral and central neural tissues, (b) the neural adaptive mechanisms manifest in the intact animal, and (c) the effect of hypothermia on the biochemistry of the cells of the supraoptic region, the anterior horn cells of spinal cord and the liver.

(a) Local effects of cold⁵:—

(i) There is a selective damage of nerves produced by exposure to cold. This selectivity depends upon the myelin sheath — the thicker it is, the greater the damage.

(ii) This type of selective sensitivity also is manifested by certain skeletal muscles which most probably depends upon the size of their motor nerves.

(iii) The sensitivity of the peripheral nerve to cold also depends on certain other factors; one of which is the species characteristics, for it has been found that size for size the golden hamster nerves are damaged at a comparatively lower temperature than the nerves of the rat.

(iv) It has been presumed that local changes of temperature in the diencephalon are effective to activate thermoregulatory mechanism. This, however, has not been confirmed finally by experimental studies so far. It has been found that no changes in oxygen consumption with changes in the

temperature are produced, either in the hypothalamus or in the cortical tissues in the rat. Further, the aerobic and anaerobic metabolism of brain slices is not in any way altered by chemical agents which change the "neural thermostatic set". However, whilst in febrile rabbits, no differences have been observed in Cholinesterase activity of brain tissue, in adult rats cooling the body temperature to 15°C causes liberation of free and total acetylcholine. It has to be further investigated whether the site of liberation of this acetylcholine is in the diencephalon or in the general cerebral tissue. Without such crucial experiments the original presumption as to the location of thermo-regulatory mechanism in the hypothalamus shall remain vague.

(v) Vasoconstrictor effect of cooling is also a local phenomenon as indicated by the fact that there is almost total vasoconstriction in the cooled separated paw of the dog which only is connected to the rest of the body by its anaesthetised blood supply.

(b) Neural adaptive mechanisms in the intact animal:—

(i) There is impressive evidence that the fluid shifts observed in the second phase hypothermic animals is abolished by lesion in the anterior hypothalamus. Embryological evidence shows that the density of myelination increases in the lateral hypothalamic area in the rat at an age when the thermo-regulating mechanisms are rapidly developing. It has been borne by several investigations that the nuclear masses in the ventro-paramedian hypothalamus between the tuberal nuclei and the mamillary bodies are vitally linked by fibre tracts to the neural mechanism which liberates A.C.T.H. from the anterior pituitary in response to cold as well as other stresses. All this evidence lends support to the fact that the hypothalamus plays an important role in the animal adaptation to the alarm stimulus of cold, even though the location of acetylcholine liberation is considerably uncertain. On the motor side this adaptation is channelled through the corticospinal system which is

necessary for shivering to set in when the temperature drops down below a certain range. It has yet to be established as to how the hypothalamic heat regulatory mechanism influences, and, is connected to the corticospinal system.

(ii) Further evidence that neural mechanism is definitely necessary for adaptation comes from the fact that in the second phase when damage to the animal becomes irreversible there is a progressive descending paralysis of the central nervous system. In the dog consciousness is first lost at a rectal temperature of 24°C. Shivering and vocalisation disappear at approximately 17°C and, spontaneous head jaw and limb movements disappear at slightly lower temperature at which stage the recovery of the animal is not possible. This irrecoverable damage to the nervous system coincides with the failure of survival of animal as a whole. This, however, is not damage to anoxia, for, electroencephalographically, it has been proved by Noell that the E.E.G. patterns induced by cold are not like those of anoxia or hypoglycemia. He points out that the brain wave activity at a body temperature ranging from 28°C to 32°C is very much like eserine or strychnine poisoning. Most probably it is an effect of excessive liberation of acetylcholine as already stated under the local effects of cold.

(c) Study of the effect of Hypothermia on the biochemistry of the cells of the supra optic region, the anterior horn cells of the spinal cord and the liver⁶:—

A semi-quantitative estimation of the mean concentrations of nucleic acids and proteins in the cytoplasm, nucleus, and nucleolus of liver cells, anterior horn cells and supra optic neurons in rats has been made by the ultra-violet micro-absorption technique of Casperson. The freezing-drying technique of fixation was used exclusively. Variations in the concentrations of these substances have been observed under a variety of experimental conditions. The observations made led to the following conclusions and inferences:

(i) The mean cytoplasmic concentration of nucleic acid (pentose nucleic acid) is approximately equal in the rat liver cell, the supraoptic neuron and the ventral horn cell.

(ii) Calculations from observed data justify the inference that the total concentration of nucleic-acid (of pentose-type) is within the same range, for the nucleolus of the rat supraoptic neuron, the aggregate of nucleoli in the rat liver cell, and the nucleolus of the cat ventral horn cell.

(iii) The nucleic acid identified chemically in these cellular structures is, as far as available criteria are concerned, apparently identical with what has been termed 'Nissl substance' in standard neuropathological parlance.

(iv) The magnitude of depletion of this cytoplasmic material under controlled environmental stress suggests that the concentration of a stable cytoplasmic nucleic acid component is approximately equal for liver cells and supraoptic neuron. Additional evidence suggests that this stable component is represented by the mitochondrial fraction of cytoplasm, at least in liver cells.

The effect of fasting:—

(i) After an 18 to 24 hour fast there is a marked depletion of cytoplasmic pentose-nucleic acid in the supraoptic neurons. This change is recognizable as chromatolysis using appropriately sensitive standard histological techniques. Similar but less reliable changes are observed in the nuclei and nucleoli of the same cells.

(ii) The chemical response of the rat liver cell during the same period of fast is similar to that of the supraoptic neurons.

The effect of exposure to cold:—

(i) A brief period of cold exposure in non-fasted rats, produces a profound depletion of cytoplasmic nucleic acid in the liver cell. The nucleus and nucleolus of these cells likewise appear to participate in these changes. In the same cold-exposed animals, an initial marked rise followed by a

precipitous fall below 'resting level' in cytoplasmic nucleic acids was observed in supraoptic neurons.

(ii) After an 18 to 24 hour fast, brief cold exposure produces a minimal and questionable depletion of cytoplasmic nucleic acid in the liver cell. No substantial changes are noted in the chemical composition of liver nuclei and nucleoli under these conditions. No measurable alteration in the protein concentration of any component of the liver cell was produced during brief cold exposure after fasting. Under the same circumstances brief cold exposure produce a marked initial rise in cytoplasmic nucleic acid in supraoptic neuron in spite of the fact that the resting fasted level is extremely low. With one hour of cold exposure, there follows again a secondary precipitous drop in cytoplasmic nucleic acid concentration as in the non-fasted animal. Similar trends are noted in the nucleus and nucleolus of the neurons.

These data suggest that the basic pattern of these chemical events is similar in the non-fasted liver cell and supraoptic neuron. The possibility of identical changes in the biochemistry of liver cells and supraoptic nucleus may offer one useful key to the understanding of certain presumed metabolic disturbances of the central nervous system in which liver changes are also conspicuous.

Brief fast does not produce any measurable depletion of adrenal ascorbic acid in the rat. There is a marked and uniform depletion of adrenal ascorbic acid in both fasted and non-fasted rats with brief cold exposure. This change manifests itself within the first twenty minutes of exposure. The correlation of this chemical change in the adrenals to the nucleic acid changes in liver cells and neurons is an interesting phenomenon. Prolonged exposure to very low temperature is likely to damage the liver cell, the neuron and the adrenals, irreversibly. The utility of hypothermia is beneficial with advantage to the animal only during the stage when due to its katabolic effect in the first phase, it has

not produced a permanent damage to the cell by a prolonged second phase of perpetuated metabolic depletion.

(2) Neurological and Psychiatric aspects of cold exposure in man⁷.

In man, clouding of consciousness first appears at a body temperature of about 34°C though with rapid fall in body temperature the level may appear to be as low as 31°C. During this phase the deep tendon reflexes are increased as an accompaniment to shivering. Discomfort in this first phase may be partially referable to increased muscular activity though surface pain is occasionally reported when the body is cooled in air or with immersion in cold water. Pain, however, is a transient phenomenon and is rapidly replaced by numbness with hyperesthesia. Hyperreflexia persists until the body temperature reaches 30°C or slightly below. At approximately 27°C response to the spoken word disappears and with a further drop of about 1°C the deep tendon reflexes, abdominal reflexes and gag reflex disappear. Pupils remain equal, pupillary light reflex diminishes progressively with falling body temperature and disappears completely at about 26°C. Sensitivity to pain and temperature change is not lost during or after artificial hypothermia, though painful subjective symptoms are relieved in almost all patients. In general there is a retrograde amnesia for that period during which body temperature is below 33°C. Such amnesia is likewise reported as a result of accidental immersion hypothermia, and exposure to cold air. Rapid cooling of the body presumably is accompanied by more violent reactions. It is reported that sometimes there is accompanied fixed upward gaze present in hypothermic states. The increase in C.S.F. pressure is variable. On autopsy, the brain is found oedematous, there is cerebrovascular congestion and extravasation of blood in the ventricles as well as the brain substance.

The influence of prolonged hypothermia on psychiatric disorders is not yet clearly established. An optimistic or even euphoric state described by Talbot among some

patients with advanced malignancies treated by artificial hypothermia is interesting in view of the probable adrenal cortical stimulation accompanying this form of therapy.

(3) The Miscellaneous facts include :

- (i) The effect of age and species⁸,
- (ii) Nutritional factors⁹,
- (iii) Oxygen consumption and related phenomenon¹⁰,
- (iv) Cardiovascular responses^{11, 19}.

All these factors influence the total behaviour of the animal towards hypothermia and the effects on the central nervous system are a concomitant happening connected with the over-all response.

(i) The effect of age and species :—

It has been demonstrated repeatedly that infant rats can survive near-freezing body temperature until well into the 2nd or 3rd week of life though mature rats consistently succumb when cooled below 14°C. Intrinsic thermo-regulatory mechanisms do not begin to appear in the rat until after the 12th to 18th day of life, and since these seem to involve an increasing dependence on aerobic oxidative mechanism by the activity of oxidases, it has been recognised with justification that the great tolerance of infant rats for extremely low temperature may in part be a reflection of their greater resistance to anoxia due to phosphorylase and phosphorylation. This applies particularly to the biochemical development of the nervous system for it has been well established that the embryonic brain is predominantly dependant on anaerobic metabolism due to phosphorylation in contrast to the adult brain which primarily makes use of the oxidases and the oxygen.

(ii) Nutritional factors :—

The observations made by Fuhrman and Crismon that rats fasted for 24 hours could be cooled to a significantly lower temperature than fed rats, before cessation of respiration, is explainable on the basis that dehydration produced thereby reduces the quantity of unbound water and thereby diminishes the risk to life. In man, high

carbohydrate and high fat diet favours high tolerance for cold. Presumably small feedings spaced at short intervals offer the best dietary regime. In cases where hypothermia is utilised as a therapeutic or operative procedure, due attention has to be paid to these observations regarding diet.

(iii) Oxygen consumption and related phenomenon :—

In both rats and dogs oxygen consumption and blood glucose level are elevated in the first phase of hypothermia. In the second phase with rapid fall in body temperature there is always almost linear decrease in the oxygen consumption values. Hyperglycemia persists but there is complete depletion of glycogen in the liver. Irreversible damage in the second phase is attributed to anoxia of the central nervous system. Observations in man confirm the phasic sequences observed in animals. The increase in oxygen consumption and blood glucose level in the first phase are attributed to increased muscular activity accompanied by the production of adrenaline as well as adrenal corticoids and A.C.T.H. due to the alarm stimulus. The second phase in human beings begins at a temperature of 35°C. Muscular rigidity and shivering persist in man even at a very low body temperature. The total energy may remain above basal level even at 27°C. The lethal body temperature for man is 25°C. At this stage there is an irreversible damage done to the temperature regulating mechanism.

(iv) Cardiovascular responses :—

In the rat and the dog the pulse rate and blood pressure increases in the first phase and then fall linearly in the second phase of hypothermia. Below 20° body temperature bradycardia is pronounced in the dogs. Atropine and Vagotomy have no effect in this phase. Death is attributed to neurogenic respiratory failure, heart block or both. In man the increase in pulse rate and blood pressure in the first phase vary linearly with the oxygen consumption. The rectal temperature of 35°C signals the onset of second phase and the pulse rate

and blood pressure progressively fall with the falling temperature. At a temperature below 30°C total cardiac irregularities intrude. This is due to the effect on the pace-maker and the conductivity, which are influenced by the metabolic debt, tachysystolic foci, defect in depolarisation and repolarisation, failure of oxygen uptake at low temperature and cardiac echymosis. These changes have been explained by Szent Gyorgi¹⁷ on the basis of histeretic condition of acto-myosin brought about by the low temperature due to the lack of plasticizing effect in the frozen serum, which loses what he calls the active steroids. These active steroids are very important in maintaining normal permeability of the cardiac muscle fibre and the utility of A.T.P. with the associated potassium and sodium exchange. The net result is cardiac failure, high venous pressure and rapid drop in blood pressure. This phase comes when there is also a corresponding irreversible damage in the central nervous system. The best utility of hypothermia for all operative procedures on the brain and the heart is during the earlier part of second phase when the blood pressure is low, there is bradycardia and the cell katabolism in general has not jeopardized the cell life. During this period the bleeding is minimum and the shock negligible.

The study of hypothermia has led to its use in homoiothermic animals especially in the following conditions:—

(i) Operative procedures:—

In this paper we are dealing with only its use in neurosurgery. As already emphasized, the early part of the second phase is the stage of choice for operative intervention. In this stage hypothermia produces narcoses, lowers the heart rate and blood pressure to an extent that bleeding is minimum and diminishes the metabolic rate of the parenchymatous tissue in this case the neurons and neuroglial tissue so that they can stand anoxia and lack of nutrition without any irreversible damage. There is a remarkable reduction in the total period of operative procedures and

this helps considerably to reduce shock and the complications produced by the shock. In the choice of the patient, the younger he is, the better. Partial dehydration and adequate nutrition with carbohydrates and fats before the operation under hypothermia further reduces the chances of any ill effects due to this procedure. In case, however, during the operation cardiac irregularities threaten to increase the danger of death, immediate venesection followed by I.V. plasma containing effective steroids, and, defibrillation should be resorted to. It has been emphasized already that raising the body temperature subsequent to hypothermia should be very gradual.

(ii) Hypothermia has been successfully utilised in treating mental derangements such as depressions, schizophrenia, and anxiety states. This therapy is based on the assumption that A.C.T.H. and supra-renal corticoids which produce euphoria are liberated when the body temperature is lowered beyond a certain range. This state of euphoria is further helpful in treating cases of intractable pain due to advanced malignancy. In this respect, hypothermia has the same status as pre-frontal lobotomy.

(iii) The drop in blood pressure and the contraction of the superficial blood vessels produced in hypothermia are utilised to prevent toxemia and shock in extensive burns of the skin surface.

(iv) The common effect of hypothermia on the nucleic acid and protein metabolism of liver cells, ventral horn cells of the spinal cord and neurons of the supra-optic nuclei has helped to explain why in certain diseases the liver and certain regions of the brain especially the basal ganglia are affected alike by the same disease process. This is also the basis to use hypothermia in the treatment of encephalitis due to viruses which thrive only if the neuron has normal protein metabolism. The results however in our hands are equivocal.

(v) Lowering of body temperature by methods of arterio-venous shunts are not so well tolerated by higher apes and by

man. In them it may be advantageous, therefore, to produce hypothermia by surface cooling supplemented with Largactil.

SUMMARY

The subject of hypothermia in relation to neurology and neurosurgery has been discussed in this paper, under the following headings:

(i) The evolution of homoiothermic control of temperature as it has developed from poikilothermic and heterothermic states has been described. The role of bound water, proteins, enzymes, endocrines, autonomic nervous system including hypothalamus and visceral regions of the cerebral cortex is elaborated.

(ii) The three procedures, i.e. hypothermia produced by surface cooling, arterio-venous shunt, and by Largactil are evaluated and details of technique as followed in our work, are given in the form of protocols. For human beings the method of arterio-venous shunt has more limitations than surface cooling supplemented with Largactil.

(iii) The effects of hypothermia on muscles, nerves, spinal cord and various regions of the brain have been discussed. The correlation of common features of metabolism in liver cells and some of the neurons is pointed out.

(iv) The utility of hypothermia in neurosurgery, neuropsychiatry, pain, malignancy, and shock due to burns has been commented upon.

REFERENCES

1. Delorme, E. J.: Experimental cooling of Blood stream, *Lancet*, II: 914-915, 1952.

2. Fay, T.: Observations on prolonged human refrigeration. *New York J. Med.* 40: 1351-1354, 1940.
3. Finley, K. H.: Angio-architecture of the Hypothalamus. *A. Research Nerve. & Ment. Dis. Proc.* XX: 286-309, 1940.
4. Laborit, H. and Huguenard, P.: "L' Hibernation artificielle par moyens pharmacodynamiques et physiques". *Pr. Med.* 59: 1329, 1951.
5. Nurnberger, J. I.: Clinical and Metabolic affects of exposure to low environmental temperatures. *A. Research Nerv. & Ment. Dis. Proc.* XXXII: 139-149, 1953.
6. *Ibid*, XXXII: 162-163, 1953.
7. *Ibid*, XXXII: 141, 1953.
8. *Ibid*, XXXII: 134, 1953.
9. *Ibid*, XXXII: 135, 1953.
10. *Ibid*, XXXII: 135-136, 1953.
11. *Ibid*, XXXII: 136-137, 1953.
12. Prosser, C. L.: *Comparative animal Physiology*, Lond. W. B. Saunders. 1950, P. 341.
13. *Ibid*, 1950, P. 369.
14. *Ibid*, 1950, P. 363.
15. Ranson, S. W.: Regulation of body temperature. *A. Research Nerv. & Ment. Dis. Proc.* XX: 342-399, 1940.
16. Ross, D. N.: Venous Cooling. *Lancet* I: 1108-1109.
17. Szent-Gyorgi, *Chemical Physiology of Contraction in body and heart muscle*. New York, 1954, Academic Press, P. 49, 1953.
18. Talbot, J. H.: The physiologic and therapeutic effects of Hypothermia. *New Engl. J. Med.* 224, 281-288, 1941.
19. Victor, E. Hall and Grant, R.: *Annual Review Physiology. Hypothermia*, 13: 91-92, 1951.
20. Werner, G. et al.: The Action of Chlorpromazine on the C.N.S. *Bull. Cal. School of Trop. Med.* 1: 14-15, 1954.

ANAESTHESIA IN NEURO-SURGICAL PROCEDURES

V. RAJAGOPALAN and B. RAMAMURTHI, Madras.

Modern anaesthesia has progressed so rapidly that it has benefitted all branches of surgery not the least neuro-surgery and has enabled the surgeon to attack deeper and more vital parts of the human anatomy. Surgery of the nervous system demands a highly skilled level of anaesthesia as the parts operated upon often deal with vital functions, as the postures adopted are most often difficult and as intracranial pressure relationships must be disturbed as little as possible.

Because of these factors neuro-surgical procedures were conducted for a long time under local anaesthesia. This was found quite satisfactory and even now in the European continent many neuro-surgical centres adopt local anaesthesia. But the chief disadvantage of this method lies in the fact that the one who suffers most is the hero of the show, viz., the patient. It is a great strain for the patient to adopt uncomfortable postures for a long time however co-operative he may be; and even though a strong sedative or hypnotic may be used. Hence in our centre general anaesthesia is adopted for most of the neuro-surgical procedures except those where a conscious patient is necessary for the surgical procedure, e.g., cortical excision for epilepsy. Occasionally local anaesthesia has been used for trigeminal root section. This enables the surgeon to ascertain the area of anaesthesia he has produced. The disadvantages of discomfort to the patient far outweighs this advantage.

Even when general anaesthesia is given, local infiltration of the operation site with novocaine is always done as it lessens the amount of anaesthetic required and as it shows the planes of separation of the tissues better.

To administer general anaesthesia for a neuro-surgical operation requires great care and skill. The requirements of the surgeon during the operation are exacting.

The patient should not strain during any part of the procedure or this may result in increased intracranial tension and consequent herniation of the brain with occasionally fatal results. This is more so during operations in the posterior fossa specially with high intracranial tension. An added responsibility in sitting postures, is to detect air embolism, the moment it occurs, so that proper measures may be taken. The requirements of neuro-surgical anaesthesia are that it must be induced without strain, it must be even and light — not too light as to make the patient strain, but light enough to ensure a proper and quick recovery of consciousness the moment the surgical procedure is over. It may have to be prolonged for a number of hours if need be and hence the drug should be non-toxic. They should not form explosive mixtures with air as surgical diathermy is invariably used.

Intracranial tension :

Many cases that come for operation have already a high intracranial pressure. The anaesthetic drug used should be such as not to increase the pressure further. Just as the principle in thoracic anaesthesia is to abolish paradoxical breathing and mediastinal movement, the fundamental principle in neuro-surgery is to control and prevent the rise of intracranial tension. Obstructed breathing, increased CO₂ tension, high venous pressure, coughing, bucking on the endotracheal tube, breath holding are highly dangerous and must be prevented not only during induction but also throughout the operation.

In all the cases coming for surgery with raised intracranial tension, a preliminary ventricular tapping must be done, if need be under local anaesthesia, so that, the intracranial pressure relationship may not be disturbed. This greatly facilitates anaesthesia and also prevents operative venous oozing and cyanosis.

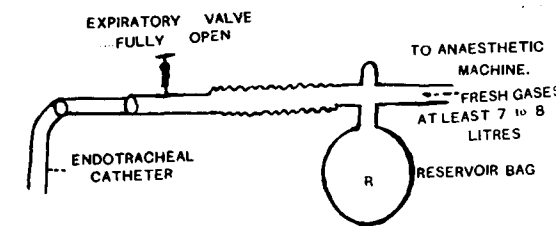


Fig. 1.

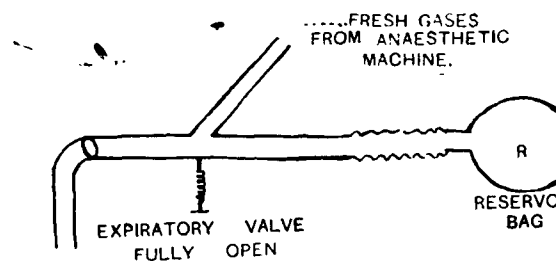


Fig. 2.

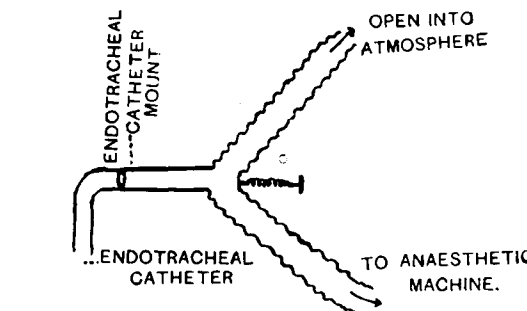


Fig. 3.

Modified Ayer's Technique.

The throat should be sprayed with 4% Xylocaine or 2% Nupercaine before intubation to avoid coughing.

Endotracheal catheters should preferably be of non-kinkable type. Armoured tubes which do not obstruct the lumen even when the neck is flexed, are now available and are most suitable for neuro-surgical patients. Cuffed tubes are better in anterior cranial fossa operations.

The circuit chosen should be one in which there is no CO₂ accumulation. Any one of the circuits (See Figures) may be employed in adults but in children Ayres T piece technique is the only choice. CO₂ absorption is not essential and is contra-indicated if Trilene is used.

Technique :

Anaesthesia is induced by intravenous injection of 0.5 gm. of Thiopentane Sodium and 80 mgms. of Flaxedil or 100 mgms. of Scoline. Then the patient is inflated with oxygen for about 1 minute and under laryngoscopic vision the larynx is sprayed with 4% xylocaine or 2% nupercaine. A good dose of relaxant and topical analgesia of the larynx are essential to facilitate smooth and easy intubation without coughing. After oral intubation with the biggest possible size of endotracheal catheter, the patient is connected to the anaesthetic machine and the anaesthesia is maintained by N₂O and O₂. The patient is then gently shifted to the operation table and positioned as required by the surgeon. By this time (about 5 minutes to 10 minutes) spontaneous respiration would have returned in normal cases. It is better to have the patient breathing on his own before shifting to the operation table. To prevent the patient biting the catheter, it is better to introduce a Water's metal airway or Bournels guard. The endotracheal catheter as well as the mount should be well fixed with adhesive plaster to prevent slipping out during the operation. Adequate oxygenation must be ensured throughout the whole procedure. Further maintenance is with gas and oxygen with a trace of Trilene

Premedication :

Morphia and omnopon are better avoided since they not only increase intracranial tension but also depress excessively the medullary centres in those cases who have already increased intracranial tension. Atropine 1,100 gr. in adults, alone or combined with a short acting oral barbiturate is the choice.

Anaesthetic agents :

Ether and Cyclopropane are unsuitable because of the explosive risk, apart from ether raising the intracranial tension. N₂O and O₂ with Trilene and intermittent doses of Pethidine offers the best combination.

and intermittent small doses of Pethidine about 10 mgms. at a time.

Blood loss :

Resuscitation and maintenance of proper general condition of the patient is an important responsibility of the anaesthetist during the operation. He must be helped by the surgeon by gentle technique with minimum blood loss. The erect posture minimises bleeding to a good extent in intracranial surgery and also reduces the intracranial tension to a certain extent. But this throws an added responsibility for the anaesthetist with difficulty in maintaining anaesthesia and watching for air embolism.

The anaesthetist controls the resuscitation measures. A glucose saline drip is started intravenously at the beginning of the operation and is changed over to blood if need arises. A correct estimate of the blood loss is kept and is replaced usually towards the close of the operation if it is minimal.

Hypotension :

Posture alone does not completely control blood loss, specially when dealing with vascular lesions of the brain. Harvey Cushing himself first laid down the principle of modern arteriotomy. Arteriotomy is however now surpassed by other methods like chemical hypotension with ganglion blocking agents which are comparatively safer. Controlled hypotension with Arfonad is used in all our cases where excessive bleeding is expected. With modern methods of diagnosis of intracranial lesions this information is available to the anaesthetist in many cases beforehand and he is able to plan for hypotension anaesthesia.

Chemical hypotension is preferable to other methods of hypotension and controlled hypotension is superior to the other methods of chemical hypotension in which there is no active control over variations of blood pressure. Methonium compounds were used to produce hypotension, but their use was much limited because of unpredictability of their action and of difficulty in exercising control over blood pres-

sure. With Arfonad, this difficulty has been got over and a minute to minute control of blood pressure is possible. Hypotension with Arfonad has been used in 8 of our cases during the last year and has been found to be useful.

It reduced operation time and reduced the amount of blood needed, thus diminishing the strain on the blood bank. Arfonad is administered as an intravenous drip of 1 in 1,000 solution (1 mgm. per 100 c.c.) in normal glucose solution, the rate of drip being adjusted to the requirement of the individual case. Stoppage of the drug restores the blood pressure.

Recovery :

A short time before the end of the operation the depth of anaesthesia must be lightened to have the patient conscious at the end of the operation. A conscious patient at the end of the operation is an important requisite and is a good guide for the patient's condition. Sometimes there is a delayed recovery specially in those who have suffered from some blood loss. In these, there may be a spasm of cerebral arterioles. In addition to restoration of blood volume, Papaverine 30 mgms. intravenous has been found useful for recovery of consciousness provided a watch is kept for signs for post operative bleeding.

Great care must be exercised in changing the position of the patient, specially after prolonged operation. The patient must be shifted to the cot gently after changing his position to the horizontal in slow stages if he had been operated in the erect posture. The endotracheal catheter can be removed if the patient is conscious. But in operations on the posterior fossa, specially if lower cranial paralysis is feared, the catheter can be left in situ or must be readily available in the post-operative ward for reintroduction.

Emergencies :

Occasionally an unconscious patient is brought in for surgery. If the unconsciousness is due to high intracranial pressure and if the surgeon proposes to do only a

small operation, (e.g., draining an intracranial abscess) it may be done under local anaesthesia. But if the surgical procedure is likely to be major and prolonged, then it is advisable to intubate the patient after spraying the larynx. This is preferable as very often the unconscious patient with higher intracranial tension may very quickly go in for respiratory paralysis and die. An endotracheal tube gives control over respiration and respiration can be maintained if need be while the surgeon is operating and removing the cause of increased intracranial tension.

Respiratory failure may occur in patients who are awaiting surgery. Immediate intubation and maintenance of respiration and oxygenation have helped to keep the patient alive so that the necessary surgical procedures may be carried out immediately.

To sum up the important principles :

- (1) Drugs and circuits chosen should not increase intracranial tension.

- (2) They must be non-explosive.
- (3) They should be fit for prolonged use and must not depress any vital processes in the body.
- (4) Induction must be smooth without coughing or breath holding or vomiting.
- (5) Intubation must be quick, smooth and easy without producing coughing or straining on the tube.
- (6) There must be no respiratory obstruction in the circuit any where.
- (7) Anaesthesia must be light and on an even plane throughout.
- (8) Adequate oxygenation throughout must be ensured.
- (9) There should not be any CO₂ accumulation; CO₂ accumulation is the direct cause of rise of venous pressure.
- (10) Quick recovery from anaesthesia at the end of operation must be ensured.

GARGOYLISM

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This syndrome characterised by a number of bizarre symptoms and signs, is easily diagnosed, when fully developed. It was described by Hurler¹² in 1919 and since then our knowledge of this condition has increased though no therapeutic progress has been made. This paper reviews our present knowledge of this condition with illustrative case records.

It was Hunter¹¹ in 1917 who reported the first two cases though the disease is named after Hurler (1919) to whom the priority of description is often given. Hunter described two cases with the characteristic facies, kyphosis and distension of the abdomen. The patients were brothers and they did not have corneal opacity. In 1919, Gertrude Hurler¹² published two cases in boys. She added mental deficiency and clouding of the cornea to the symptoms described by Hunter. Putnam and Peka¹⁷ (1925) gave the first report of a case in English literature. Helmholtz and Harrington¹⁰ (1931) reviewed a number of cases. It was only in 1934 that Tuthill²⁰ investigating one of the cases described by Hurler in 1919, gave a detailed account of the pathological changes occurring in the nervous system. In the same year, Sheldon and others presented cases before the Royal Society of Medicine. In 1936 Ellis, Sheldon and Capon⁸ described seven cases and reviewed eight previous cases and from the resemblance of the patients to a gargoyle gave the name Gargoylism to the disease. They suggested that the disease may be due to metabolic error. In 1938, Kressler¹⁴ and Aegerter published radiological illustrations with a report on 19 cases.

The disease has many synonyms (Hurler disease, Dyostosis multiplex, lipochondro dystrophy). It is characterised by a heavy ugly facies, kyphosis, distension of the abdomen with enlarged liver and spleen and mental deficiency. Clouding of the cornea is also often present, though it was absent in almost a fifth of the cases reported.

CASE REPORTS

Three cases suspected of gargoylism were seen by the neurosurgical unit, Madras.

V. aged 14 years (NS 3515) male was sent with a complaint of difficulty in walking, kyphosis and protuberant abdomen. He was born normally. The parents noted a shortening of the height and a waddling gait. On examination, he was an intelligent boy studying in the high school in the normal class for his age. There was no corneal opacity. Liver and Spleen were not palpable. X-rays of the spine showed typical changes of chondro osteo dystrophy of the (Fig 1 & 2) Marquio Brailsford type. Gargoylism was excluded.

R. aged 6 years (NS 110/6) female was referred for mental deficiency. Her facial features were definitely heavy and ugly with some spots of opacity in the cornea. The spleen was palpable. Mental power was far below her age. The head was enlarged and there was an early kyphosis. Her parents were not related. 2 other siblings were normal. X-rays of her spine showed the typical features of gargoylism (Fig. 3). Blood cholesterol was normal as also the glucose tolerance test.

P. aged 15 years (NS 3913) female was examined for mental deficiency. Her facial features were characteristic with typical features. She had also



Fig. 1.



Fig. 2.



Fig. 3.

had hypertelorism (Fig. 4 & 5). There was kyphosis with enlarged liver and spleen. There was no corneal opacity. Her mental quotient was far below normal. X-rays of the spine showed features of gargoylism. Her parents were related to each other before marriage. 6 other siblings were normal. Blood cholesterol was normal. Pneumo encephalogram had to be done under general anaesthesia as the girl would not co-operate. This showed normal ventricular system but atrophy of the frontal cortex.

There is doubtful evidence to suggest a hereditary factor in the cause of the disease. It has occurred in more than one member of the family. Cases have been described in three consecutive children of the same parents. In some instances the parents have been related before marriage. In the same family, there may also occur allied diseases like craniostenosis and chondro osteo dystrophy (Brailsford Marquio disease)³. Because of these familial factors, a genetic cause has been suggested. The high incidence of the disease in siblings and the frequency of occurrence in cousin marriages point to a genetic origin. It is believed to be inherited as a recessive character (Potter¹⁶). The disease definitely origi-

nates in utero and in a third of the reported cases more than one child in the family has been affected. Caffey⁵ reports a case in the first month of life suspected because of the facial features. X-rays of the long bones of the upper limb and further progress of the case confirmed the diagnosis. There have been only three reported cases that were recognised in the first month of foetal life. In one of these (in a family observed by Buchanan) it was the father of the child who made the first diagnosis as his previous two children developed Gargoylism.

Though in such rare instances the disease may be recognised in childhood, the first symptoms may not be noted till the child is 3 or 4 years old. Kyphosis may first attract attention or the large distended abdomen. In some cases the dwarfism is apparent. Mental disturbance may be obvious only a little later, but is an integral part of the clinical picture. There may be also other evidence of damage to the central nervous system. In a well marked case, the facies are typically heavy and ugly, with prominence of the supra orbital ridges and the temporal regions. The tongue may be thick



Fig. 4.

and protruberant. The eyes may be set wide apart and the bridge of the nose depressed (Fig. 4). The skull may be large or may show other changes of oxycephaly. The association of such changes with gargoylism is discussed below.

The abdomen is protuberant and there is hepato splenomegaly. When the patient stands, the thighs are partially flexed and adducted; there is genu valgum and the feet are everted.

Cloudiness of the cornea is a striking feature in many cases and is caused by multiple opacities in the deeper layers of the cornea.

Kyphosis is an early sign. Changes in the skin (like thickening, loss of elasticity) may also occur. Anderson and Tandberg¹ (1950) have reported the case of a boy of 6½ years old with features of gargoylism who showed skin nodules. Biopsy showed changes similar to those seen in the internal organs in gargoylism. Jervis¹³ (1950) publishing a study of 10 cases emphasizing



Fig. 5.

the 'Formes Frustes' of the disease in which congenital clouding of the cornea is associated with only slight changes in the skeleton. He points out that gargoylism may present itself with varied clinical manifestations and atypical variants may occur. Diagnosis of gargoylism is possible even in the absence of some of the clinical features. About 23 of the recorded cases showed only incomplete features.

The radiological features are interesting and characteristic. The features are such that gargoylism is considered as an atypical form of chondro osteo dystrophy (Marquio Brailsford). Though there is a general resemblance to chondro osteo dystrophy there are distinct differences (Cf. Fig. 1 and 3).

Caffey⁵ (1952) has described bone changes in very young children. In them, the bones of the upper limbs and the ribs show the most severe changes whereas the leg bones are not affected. There is expansion of the medullary cavity in the long

bones and thinning of the cortex. According to Caffey⁴, an uneven increase in the growth of the shafts of the long bones is the most important single diagnostic feature in the skeleton of a patient with gargoylism. At the epiphyseal areas, radiological changes are not diagnostic. This differentiates gargoylism from chondro osteo dystrophy, viz., the conspicuous and consistent diaphyseal expansion with minimal epiphyseal changes. This appearance is also in contrast to the findings in achondroplasia in which the ends of long bones form mushroom like expansions and overhang the shafts.

In the vertebrae, differences can be made out. The figures show the difference between chondro osteo dystrophy and gargoylism. In gargoylism the upper and lower surfaces of the vertebral bodies are convex so that they present a circular rather than a quadrilateral outline (Fairbank⁹) whereas in chondro osteo dystrophy the vertebrae are flattened out or biconcave with a tongue-like process projecting from the front. In addition in cases with well marked kyphosis in Gargoylism, one vertebral body may be smaller than the rest and may show beaking.

PATHOLOGY

Gargoylism has long been considered to be a disturbance of lipid metabolism though now there is the view that it is more likely to be a disturbance of carbohydrate metabolism. The idea of lipoidoses arose from histological and chemical examination of some autopsies. Tuthill²⁰ (1934) examined the brain of the patient, described by Hurler in 1919 and recorded the finding of a lipid in the brain with changes corresponding to those found in infantile amaurotic family idiocy.

Ashby¹, Stewart and Watkin (1937) performed autopsies on two cases lacking hepato-splenomegaly. In both enlarged persisting thymus was found. In every part of the nervous system, the cytoplasm of the ganglion cells was found distended by the deposit of an abnormal substance. The Nissl granules were found reduced in num-

ber and crowded to one side. The cell processes were not affected. Chemical analysis in one case showed a reduction in the cerebroside of the cerebral cortex but not of the white matter. Around many of the blood vessels of the brain, lipid deposits (with the staining reaction of a cerebroside) were found. So these authors considered Gargoylism as a form of lipoidoses even though no foam cells were found in the liver and spleen. Cornelia de Lange⁶ (1942) also found in a case of typical gargoylism that the brain contained nerve cells laden with lipid closely resembling juvenile amaurotic idiocy.

So far Gargoylism was accepted as a form of lipoidoses though the nature of lipid could not be ascertained. Laboratory investigations and analysis of blood lipoids did not show any changes. In the two cases recorded in this paper, the blood cholesterol was normal and there was no evidence of disturbance of fat or carbohydrate metabolism. But in 1944 de Lange⁷ and her colleagues published autopsy studies of 2 cases of gargoylism (though the writers were not quite sure that the cases were typical examples of gargoylism). In these they found accumulation of glycogen in the liver cells with cirrhosis. The nerve cells of the brain did not show any evidence of change as reported in other cases.

It was Strauss¹⁹ (1947) who suggested that the material found in the tissue cells may be a non-lipoid material of high molecular weight like glycogen. Lindsay et al¹⁵ (1948) publishing extensive clinical and autopsy findings in 8 cases, concluded that the lesion was due to the intra and extra cellular deposition of a substance giving the histo-chemical reactions of glycogen. It was also suggested that glycogen may be present combined with proteins. This theory will serve to explain the skeletal changes and also to explain the absence of blood lipid changes in gargoylism.

Changes in the skull are difficult to explain. Case no. 3 presented typical features of hypertelorism. In hypertelorism the head is broad and flat and the forehead

is prominent. The nose is upturned and there is an abnormal increase in the distance between the two eyes. This is due to an abnormality in the development of the sphenoid bone whose lesser wing is much hypertrophied whereas the greater wing is not well developed (Potter¹⁶). Just as in the limbs and the spine, the abnormal growth of the cartilaginous portion of skull may explain these skull changes. Association of Gargoylism with cranial dysostosis is recorded. In our experience, none of the 20 cases of oxycephaly recorded by us showed features of gargoylism. Helmholz¹⁰ and Harrington included gargoylism, along with hypertelorism and five other conditions under cranial dysostosis. But there is no doubt the conditions are different though occasional associations are reported. There is no definite evidence of any causal relationship between Gargoylism and cranial dysostosis. Occasionally dilatation of the ventricles has been reported in Gargoylism. In our case pneumo-encephalogram did not show any hydrocephalus though there was a cortical atrophy.

The changes in the cornea are due to replacement of Bowman's membrane by large polygonal cells having abundant granular cytoplasm. Corneal cells also showed similar changes. The changes though thought to be due to lipoidoses, have not been definitely shown to be so by any staining reactions (Rochat¹⁸ 1942).

Thus the exact aetiology of gargoylism is unknown. Here is a field for further study.

Our thanks are due to the Dean, General Hospital, Madras, for the kind permission to publish this paper.

REFERENCES

1. Anderson, B. and Tandberg, O.: Acta. Paediatrica. 1950.
2. Ashby, W. R., Stewart, R. M. and Watkins, J. H.: Brain. 60: 149, 1937.
3. Brailsford, J. F.: Amer. Jour. Surg. 7: 404, 1929.
4. Caffey, J.: Paediatric X-ray Diagnosis. Chicago Year Book Publishers. Pp. 633, 1945.
5. Caffey, J.: Am. J. Roentgenol. 67: 715-731, May, 1952.
6. de Lange, C. C.: Psychiat. Neurol. Bl. Amst. 46: 2, 1942.
7. de Lange, C. C. et al.: Acta. Paediat. Stock. 31: 398, 1944.
8. Ellis, R. W. B., Sheldon, W. and Capon, N. B.: Quarterly J. of Med. 29: N.S. 5: 119, 1936.
9. Fairbank, T.: Atlas of General affections of the skeleton. E. & S. Livingstone. P. 135, 1951.
10. Helmholz and Harrington.: Am. Jour. Dis. Child. XLI: 793, 1931.
11. Hunter, C.: Proc. Roy. Soc. Med. 10: 104, 1917.
12. Hurler, G.: Zeitzchrift fur Kinder heilkunde. 24: 229, 1919.
13. Jerris, G. A.: Arch. Neurol. Psychiat. 63: 681-712, 1950.
14. Kresseler, R. J. and Aegester, E. E.: Journal of Paediatrics. 12: 579, 1938.
15. Lindsay, et al.: Amer. J. Dis. Child. 76: 239, 1948.
16. Potter, E. L.: Pathology of the fetus and the new born. Year Book Publications. P. 455, 1952.
17. Putnam and Pekan.: Am. Jour. Dis. Child. XXIX: 51, 1925.
18. Rochat, G. F.: Ophthalmologica. Basel. 103: 353, 1942.
19. Strauss.: New York State J. Med. 47: 157, 1947.
20. Tuthill, C. R.: Arch. Neurol. Psychiat. Chicago. 32: 198, 1934.

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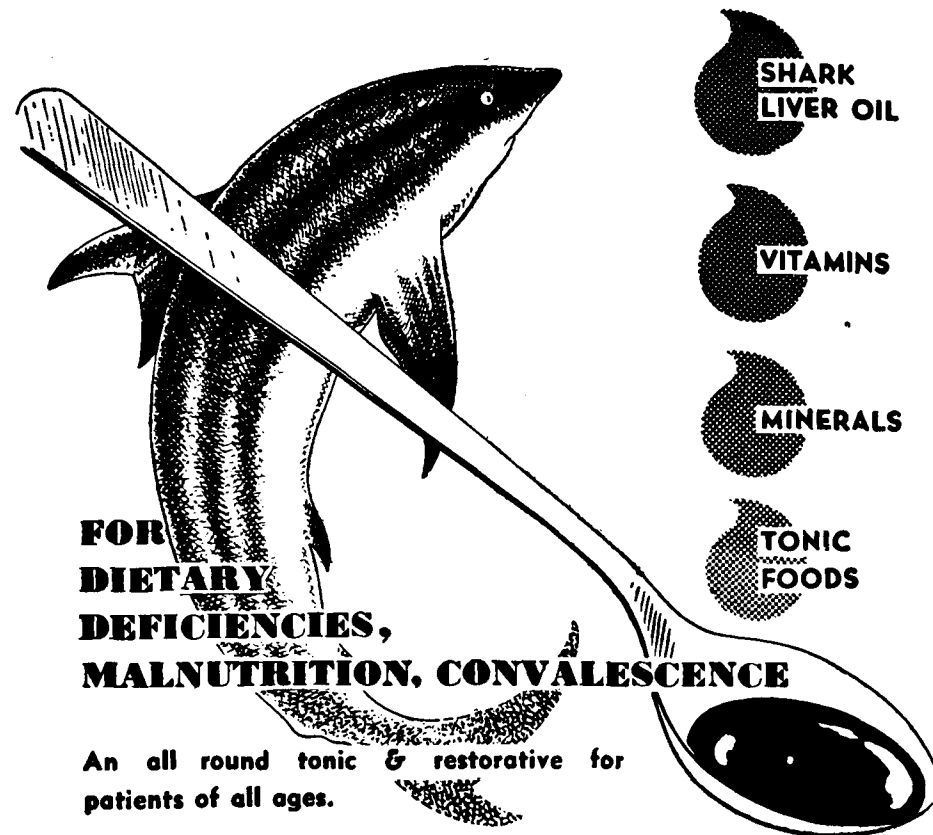
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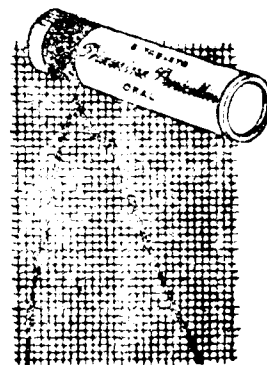
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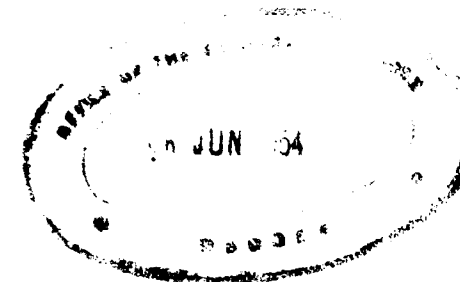


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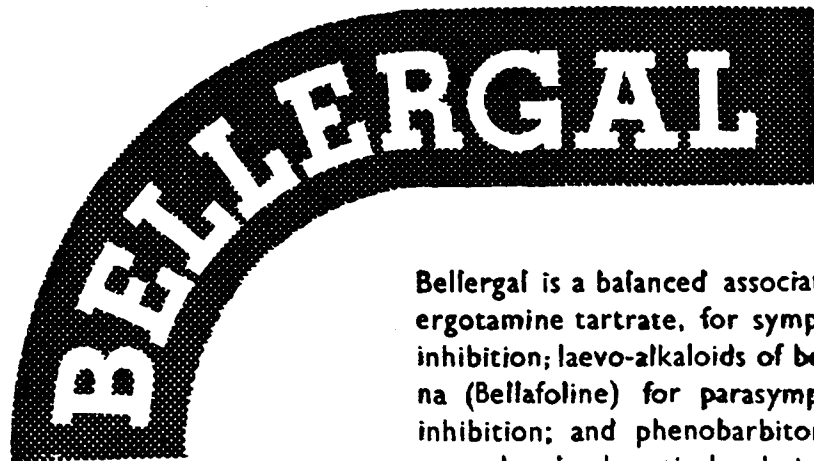
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Vol. II

No. 1

CONTENTS APRIL 1954

Creative Neurology - - -	T. B. Ghosh	1
Glioma of the Hypophysis - - -	R. G. Ginde and C. G. S. Iyer	5
An unusual effect of a Cerebral Epidermoid (Cholesteatoma) - - -	Darab K. Dastur	9
Electro-Encephalographic Study of Focal Seizures	Baldev Singh and Jacob Chandy	13
Association Notes - - -		21



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2. Frederick P. Moersch, M.D., & George P. Sayre, M.D., Neurologic Manifestations associated with dissecting aortic aneurysm. The Journal of the American Medical Assn., Vol. 144, No. 14: December, 1950. 1145-1146.

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CREATIVE NEUROLOGY*

by T. K. GHOSH, Calcutta.

Rt. Hon. Dr. Jayakar, Dr. Ranade, Dr. Coehlo, Dr. Chaudhuri, Members and Guests of the Conference, Ladies and Gentlemen,

Neurological Society of India is grateful to the medical fraternity of Poona for their kind invitation to hold our Annual Conference here, in the midst of such a representative and distinguished gathering.

The occasion evokes in our mind an atmosphere of gratitude, veneration and devotion to the noble souls who sprang from this part of our motherland. A sense of kinship and mutual understanding for you specially pervades our hearts in the distant State of Bengal. Perhaps, the genesis and historical reasons for this well-known fact needs no re-telling.

I am personally grateful to my esteemed friends who asked me to take charge of the office of President. I have accepted it as a command of my colleagues.

It is a great honour, perhaps the highest that a learned Society can confer. Probably, I am not wise or old enough to muse over it. But it will certainly strengthen my steps to the road to Neurology, which, alas, is so uneven and lonely.

This post of Presidentship makes me reflective and reminiscent. I have a vague impression of a President as an aged person—if not in years, certainly in mind—who on a glorious day—delivers a long learned address; and then sinks in solitude, quiet and contented, to read and brood over the same all through the year. This address is so much valued by the President that he stuffs it with all available information and then adds to it his own special views and own wisdom. It is some-

times so lengthy that not many stay to the end. And very few can follow the trend of his thought in that fast and long speech.

This is not a comforting thought for a speaker. But any way, I, as the President, must speak—if not for the sake of convention, at least not to create a precedent.

Neurological Society of India is a young Society of Neurological specialists of our country. Its members are not too many but they are ardent and enterprising. Friends may be asking what is the good of this newly formed Society. May I quote Benjamin Franklin's inimitable answer. "What is the good of a new born baby?" It is for our members to justify the existence of this specialized body devoted to Neurology by richly contributing to the health and happiness of our people.

This brings us to the much discussed topic of Specialization in Medicine. In 1921, Harvey Cushing, the pioneer in Neurosurgery addressed the Endocrinologists in the form of an allegory. While commenting on the birth of this new speciality, he said:

"We find ourselves embarked on the fog-bound and poorly chartered sea of Endocrinology. It is easy to lose our bearings for we have, most of us, little knowledge of sea-faring and a vague idea of our destination. Our motives are varied. Some unquestionably follow the lure of discovery; some are earnest colonizers; some have the spirit of missionaries and would spread the gospel; some are attracted merely by prospect of gain and are running full sail before the trade wind".

These incisive and prophetic expressions should deeply interest workers in all spe-

*Presidential Address read at the Annual Conference of the Neurological Society of India

There is specialization beginning with A for Allergy to U for Urology. Sometimes it is based on anatomy e.g. Cardiology. Others are built on diseases e.g. Tuberculosis. Some have limited themselves to age groups e.g. Pediatrics.

Specialization is as old as Medicine. The points at issue are not about the principle but in details: When does a speciality make its emergence, what does it want to achieve, and how should it function?

When the corpus of knowledge was small, it was within the intellectual embrace of an individual and the question of specialization was not looming large. In a perplexing age as ours, it is beyond the capacity of even the most gifted to master every bit of knowledge. So, there are branches and sub-branches whether in Arts or Science. Why should our medical men feel sore on this point? We have long ago divided ourselves as Physicians, Surgeons, Obstetricians, and Gynaecologists. If we now limit ourselves—not our vision but our energy—to certain particular episodes, or body systems, there is nothing wrong or derogatory about it. For, we want to finally integrate our services and provide every person as far as possible with the highest quality of health care.

Specialization does not want to thrive at the expense of the general practitioner. It desires to contribute the important information gained by special ability, experience and technical skill to the heavily worked Family Physician whose duty is to make full use of them for the benefit of his patient. A person who wants to specialize must have aptitude and love for his speciality and should have years of training as a general Physician and Surgeon. He must draw inspiration repeatedly from his original field.

With this rambling preamble on specialization, may we now have a look at the unique and distinguishing features of Neurology?

Neurology confines itself to the study of the Nervous System in health and disease. It is not an individual enquiry but a team organization. In Neurology, we have an

arena where many meet to form this dynamic speciality. Here is a merger of men and ideas from Medicine, Surgery, Pathology, Radiology, Ophthalmology, Anatomy, Physiology, Physics and Chemistry. Each has its roots in the basic subjects but ultimately flowers as what I may be permitted to call Creative Neurology.

From general medicine comes the Neurologist; Neurosurgeon from general surgery. Neuroophthalmologist, Neuroradiologist and Neuropathologist from their respective branches join the team. The Clinical Neurologist trained in Electronics functions as the Electroencephalographer. The Biochemist and the Nuclear Physicist come from Chemistry and Physics. Neuro-anatomists and Neurophysiologists from Anatomy and Physiology from important elements for investigative works. Physical Medicine, Physiotherapist, Psychologist and social workers join for better therapeutic results. In a Neurological Hospital, House Officers, Nursing Staff and Anaesthetist are essential to this group. And it is a matter of convenience, not of competition, who should be the chief of this dynamic group, a Neurologist or a Neurosurgeon. Such Hospitals should be established at places where there is a sufficient and varied neurological problem to keep them engaged and not rusted.

Perhaps, it needs a reminder that the neurological specialist should not be pathetically attached to sterile tract Neurology. He should not constantly dream for dramatic Brain Surgery. He should not take pride for spot diagnosis of the neurologically afflicted. He should be interested in the pattern of evolution of neurological diseases. In co-operation with fellow workers, he can then elucidate the structure and function of the Nervous System. Strangely enough, many leading scientists and men of letters at certain periods of their life make innocent excursions on the nervous structures for building their theories on the physical basis of mind and thought. Neurology should provide such persons and the psychologists a more stable foundation. Workers in the neurological field, should effectively contribute in matters which pertain so intimately to

human development and a more profitable kind of human existence.

May we now try to definite the role of Neurology in Teaching, Practice and Research?

Teaching of Neurology to a medical student at the undergraduate level has a profound and stabilising influence in his career. It trains him in general principles of observation and inference and in the systematic use of Anatomy and Physiology. It stimulates and enshrines faith in the basic concepts that he learnt in his pre-clinical course. We admit however there is a vast amount of rusty and useless information catered to our students, who luckily forget them in no time. It will be our prime task to disown and dismantle the concretions of our past errors which successive text-books of Anatomy and Physiology have accumulated. At the Post Graduate level, training and care of the candidate should be left to the Neurological specialists. It is desirable to have regional qualifying boards rather than diplomas.

The practice and place of Neurology in relation to the remainder of modern medicine is so little understood even by medical men that an appraisal seems compelling. The Neurologist in the past was primarily concerned with the diseases of nervous system. In accord with rapid development in medicine, the sphere of neurologist changed—many bacterial and protozoal diseases of nervous system showed a decline. The rise of Neurosurgery as an independent speciality and the mounting aggressiveness of Psychiatry soon encroached on the Neurologist's field, which constricted to contain the Polio, Epilepsies, Degenerative and Demyelinating diseases. Dereck Denny Brown—himself a noted neurologist—puts it testily when he says that “from the beginning of the budding process, the Neurologist has occupied a position that is somewhat academic—the impractical collector of static curiosities and geographer of abstruse anatomic networks, with more than the usual specialist's ritual of cabalistic signs.” There was and is considerable truth in the above evaluation. The Neurologist lived as a poor relation—a dependent

diagnostician and a match maker to the Neurosurgeon vis-a-vis the Brain Tumour patients. Sometimes, the Neurologist took to paying Psychiatric practice with a lingering look at his lean Neurological diary. A few years back, Neurology was almost withering in isolation, when it became infused with new ideas and strength by recruiting fellow workers from different departments of Medicine, Surgery and other branches of science as already alluded to. The Neurologist, to-day, functions in Neurological Medicine in solving different problems. He co-operates with his Medical and Surgical Consultant colleagues in the management of neurologic complication of diseases of other systems of the body. He acts as a consultant to the General practitioner who seeks his aid. He is prepared to undertake the full responsibility for the management of those conditions that need constant, neurologic supervision. The Neurologist shares the responsibility and anxieties of Neurosurgery. And a Neurosurgeon should not compete but co-operate with him. Pre- and Post-operative care would be better managed and checked by combined supervision. More time will be made available to a Neurosurgeon for his arduous task if he is relieved of these duties by his Neurologist friend. Neurosurgeon and Neurologist must work in the same unit. Neurosurgeon should learn neurological medicine to be a better Neurosurgeon. Neurologist should know Neurosurgery for better management of Neurosurgical problems. Harvey Cushing, the great Neurosurgeon was elected President of American Neurological Association in 1921. He made a far-reaching comment on the bonds of the Neurologist and the Neurosurgeon and said, “The fusion will be of benefit, if for no other reason than the balance sheets of Surgery should periodically be audited by those not actually engaged in its practice.”

Research in Neurology is a fertile field for pioneering spirit. Knowledge of neural and cortical function is far from complete. Neurological basis of consciousness remains to be clarified with a vertical orientation and a search on the depth. The genesis of electrical waves—its biochemical nature—is an ever elusive but attractive field. The nature of epilepsy still remains a mystery.

I have devoted considerable time to clear current thinking on Neurology. For, we face an unfortunate fact that Neurologists are almost non-existent in our country and young physicians are not eager entrants for they feel they have no future in Neurology. We want to clear up this misunderstanding. For Neurology is now entering on a new era of growth and productivity. Israel Wechsler—the learned Neurologist—has aptly said that “much as neurology has already contributed its future holds even greater promise.”

Neurology has undergone a slow and sometimes painful re-orientation. It dealt with diseases of the Nervous System. Now in addition, it embraces disturbances in neural control of the body systems—especially its complex reaction to stress—a direct interest in the mechanism of personality and behaviour.

We have attempted to clear up recurring misunderstanding concerning specialization in general and Neurology in particular. Discussion on specialization is now for historians of medicine, for, it has come to stay. Narrowness of outlook or atrophy of inbreeding which besets many a speciality, does not concern Neurology. For, it draws inspiration from many fields and functions as a team. There is no boredom but considerable joy of exploring uncharted regions.

Here is something new and fascinating in this speciality, where we integrate the differing aspects. Here is unity in diversity that our sages have taught us. We do not want to create boundaries or barriers. We do not want to carve out a Province of our own. We want to raise a banner of Creative Neurology.

It is a rallying point for fellow workers from different lands to come together under its unifying and self-effacing influence. Each focusses its special light to solve the problem. Some carry blue light and others are of red, green and many tints and, in fact, all colours. But their combined effect produces an unique white light that floodlights the forgotten and sunshines the neglected ills of mankind.

We realise that we in our country have not been much interested in Neurology in the past. I feel an impelling necessity to share with you a profound and magnificent utterance of Ramon Y. Cajal, the greatest scientist of Spain and the greatest Neuro-pathologist. He was tormented by the mental slavery of the young men of Spain and wanted to jolt their drowsiness. He wrote: “When the dark sea looks tranquil at night, a mere stirring of the water is enough to make clouds of shining noctilucas kindle their light and gleam like stars. The same thing happens in the ocean of Society. One need only shake the thicket of sleeping neurons, make them vibrate with the excitement of novelty, and inspire them with noble restlessness”.

What a stirring call from a Savant! Is it not a privilege to respond?

I come to the end of my talk; I realise this has been a sketch rather than a speech. I regret I have been ‘neuro-sophical’ in my thinking. But I could not help looking at the limitless landscape in Neurology with its widening horizon.

Wider Penfield, a foremost Neurosurgeon in his address to the American Academy of Neurosurgeons in 1948 has made this significant statement:

“I believe that a new day will dawn tomorrow and that in its light will be found an understanding of the nervous system. Mental as well as physical activity will then be recognized as a function of the brain and Neurosurgery and Psychiatry will lose the separate authority conferred upon them by ignorance until there remains only Neurology. Neurology will then stand forth as a single discipline to which Internists, Psychologists, Surgeons, Chemists and Physiologists will contribute”.

To-day, we want your permission to prefix “Creative” before the word “Neurology” and invite you all to unite under God around the banner of Creative Neurology for that bright tomorrow.

GLIOMA OF THE HYPOPHYSIS*

(A Case Report)

by R. G. GINDE and C. G. S. IYER, Bombay.

From statistics based on a large series of cases it would appear that the incidence of Hypophyseal neoplasms varies between 5—20 per cent of all intra-cranial tumours. While the tumours arising from the parenchymal cells of the anterior hypophysis and certain embryonal remnants have been the subject of a large number of reports, there are a few less frequently occurring tumours in the hypophyseal region that have not attracted much attention, but have to be borne in mind. This is particularly so, as they mimic the clinical neurologic symptomatology of the parenchymal pituitary tumours, viz., the adenomas.

This communication records an instance of a space-occupying lesion of the hypophysis presenting the clinical symptomatology of a chromophobe adenoma—but which on microscopic examination was found to be a glioma.

CASE REPORT

J.J.C., No. 537649, a 39 year old man was admitted to the K.E.M. Hospital, Bombay, in May 1953 for progressive failure of vision since 9 months prior to admission. Visual impairment commencing in the right eye, was progressive and in the following 3 months involved the left eye so that on admission he was unable to read or write and was just able to recognise people from a very close distance. During the course of these 9 months the patient had also experienced occipital headaches, and a sensation of heaviness all over the head with occasionally stiffness of the neck. There was no other complaint referable either to the nervous system or to the rest of the body.

He was a well built, moderately nourished adult male. The positive physical findings were restricted to the nervous system.

Neurological examination revealed retention of normal mental faculties. The visual acuity of the right eye was reduced to perception of light and that too, only when the light was projected from below and the nasal side. With the left eye the patient could read large letters again only when

*Paper read at the Third Annual Conference of the Neurological Society of India held at Calcutta on 23-2-54.

placed on the nasal side. Visual field Charting revealed a temporal hemianopsia on the left (Fig. 3-a). (The right was not charted as the visual acuity was reduced to perception of light). Both the pupils were dilated and reacted sluggishly to light. Ophthalmoscopic examination revealed primary optic atrophy on both sides—more marked on the right. The rest of the nervous system was normal, except for diminution of all the deep tendon reflexes.

Laboratory investigations were not contributory. A plain X-Ray of the skull (Fig. 1) showed generalised beaten-silver appearance of the skull bone, and marked widening and flattening of the Sella Turcica.



Fig. 1.

Lateral X-Ray of the skull showing marked deepening and widening of the Sella Turcica with undercutting of the anterior clinoid processes and encroachment of the sphenoid sinus. Pineal is calcified and seen in normal position.

With a pre-operative diagnosis of a chromophobe adenoma of the pituitary a right frontal osteo-plastic craniotomy was performed (R.G.G.) under N₂O and O₂ supplemented by Intravenous Sodium Pentothal. The dura was under great tension to relieve which the ventricle was tapped. On opening the dura and retracting the frontal lobe a solid intrasellar encapsulated tumour, maeon-coloured, and about 3 cms. in diameter was seen. Both the optic nerves were pressed upon and flattened by the tumour. With the aid of biopsy punch and suction a large part of the tumour was removed thereby relieving the pressure on the optic nerves and exposing the floor of the Pituitary fossa anteriorly. Immediately fol-



Fig. 2-a.

Microscopic structure of the neoplasm seen under (a) low, and (b) high magnification. (Sections stained with Haematoxylin and Eosin)



Fig. 2-b.

Following the operation his temperature rose to 103°F and then remained at 101.5° to 101° for the next few days, becoming finally normal.

One month following operation the patient still showed left temporal hemianopsia but with a smaller white object than before.

Since then he has been seen periodically. There has been improvement of vision in the left eye (Fig. 3-b). The acuity of vision in the right also was better. His other symptoms, particularly headaches had disappeared.

Pathology Report:—The tissue received at operation was fixed in 10 per cent neutral formalin and sections stained with Haematoxylin and Eosin, Phosphotungstic Acid Haematoxylin, and Gomori's Silver technique for Reticulum.

Microscopic examination (Figs. 2-a and 2-b) revealed blood clot and a loosely cellular tissue with a number of blood vessels. The constituent cells were elongated with indefinite outlines, oval, deep staining or vesicular nuclei and thin cytoplasmic processes. Occasional cells had a rim of acidophilic cytoplasm around them. A very occasional cell was binucleated. No mitoses were seen. The cells showed in some places a tendency to be loosely dispersed around blood vessels.

Some of the blood vessels of the tissue contained thrombi in various stages of organisation — there was in addition a fair amount of inflammatory exudate around such vessels. Small areas containing hemosiderin — filled macrophages were probably indicative of old haemorrhages. Scattered areas of calcification were seen.

Sections stained with Phosphotungstic Acid Haematoxylin showed a number of fine blue fibrils in the tissue.

Sections stained for reticulum (Gomori) revealed vascular reticulum only. There was no reticulum in the rest of the tissue. Histological findings were consistent with the diagnosis of an astrocytic glioma.

COMMENT

The clinical and radiological features presented by this patient were considered to be consistent with those of a slow-growing chromophobe adenoma of the Hypophysis. The pathological findings were a surprise. The architectural pattern of the tumour was quite different from that of any adenomas, and the type cells with the



Fig. 3-a.

Preoperative perimetric chart showing almost complete temporal hemianopsia in the left eye.

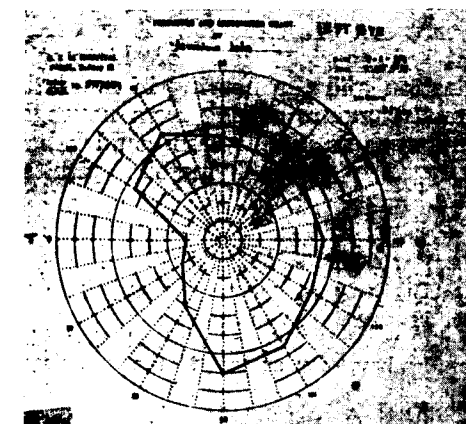


Fig. 3-b.

Further improvement in the visual field.

fibrillary cytoplasmic processes and uniform oval vesicular nuclei were reminiscent of the glial elements that occur normally in the infundibulum and neural hypophysis. The demonstration of glial fibrils by special stains and the absence of any reticulum in the substance of the tumour were additional features supporting the diagnosis.

From the scanty mention in the literature it is difficult to be certain about the frequency of occurrence of these tumours. The large series of Pituitary neoplasms (those of Cushing² and Courville³) fail to mention glioma, and a review by Kraus⁵ in 1945 makes only a passing reference to a few cases quoted in the literature without giving any further details. Of particular interest in this connection is a glial tumour of neurohypophyseal derivation first described by Globus⁴ in 1942. To the two original cases described by him was added a third by Scheinker⁶; all these three have been in children. The cellular and architectural feature of these tumours was reminiscent of the normal infundibular region and they had been called infundibulomas. It is felt that the structure of the tumour reported in this communication corresponds very closely to that des-

cribed by Globus and Scheinker in their reports.

In a recent report published in 1950 Antoni¹ has recorded his interesting experience with the microscopic study of a series of 10 Pituitary neoplasms clinically diagnosed as chromophobe adenomas. 8 of these fulfilled the histologic criteria of ependyomas, 1 was a glioma and the last was not classified histologically. On the basis of this study Antoni is of the view that "the opinion that chromophobe adenomas constitute the bulk of the suprasellar tumours connected to the Hypophysis needs revision". If his hypothesis viz., that a proportion of these tumours are in reality glial in origin is accepted Antoni feels that it would much better explain some of the puzzling topographic and clinical features of these cases particularly their spread and pressure effects and relative resistance to radiation.

While the findings of Antoni¹ are exceptional and have to be confirmed by others it is instructive to keep them in mind particularly when faced with pituitary neoplasms that extend above the Sella Turcica. Whether a careful re-study of these tumours will alter materially the pattern of the data that has accumulated on pitui-

tary neoplasms remains to be seen. The least that can be expected is a certain amount of reorientation of histological classification and the bearing in mind that occasional tumours arising from neuroglial elements such as in this case could behave clinically like paranchymatous pituitary tumours.

ACKNOWLEDGMENT

We are thankful to the Dean, K.E.M. Hospital, Bombay, for permission to report this case and to the Professor of Pathology, Seth G. S. Medical College, Bombay, for access to the pathological material.

REFERENCES

1. Antoni Nils.: Gliomas of the Neurohypophysis and Hypophyseal Stalk, A preliminary report. *J. Neurosurg.* 7: 521-531, 1950.
2. Courville, Cyril B.: *Pathology of the Central Nervous System — A study based upon a survey of lesions found in a series of thirty thousand autopsies.* Pacific Press Publishing Association, Mount View, California. 2nd edn. Pp. 450. 1942.
3. Cushing, H.: *Intra-cranial Tumours. Notes upon a series of two thousand verified cases with Surgical Mortality Percentage pertaining thereto.* Charles C. Thomas, Springfield, Illinois. 9: 150, 1932.
4. Globus, J. H.: A newly recognised tumour of neurohypophysial derivation with a note on the Aocous vasculosus. *J. Neuro-path. & Exp. Neurol.* (Quoted by⁶ below).
5. Kraus, J. E.: Neoplastic Diseases of the human Hypophysis. *Arch. Path.* 39: 343-349, 1945.
6. Scheinker, I. M.: *Neurosurgical Pathology.* Charles C. Thomas, Publishers, Springfield, Illinois. 15: 370,

AN UNUSUAL EFFECT OF A CEREBRAL EPIDERMOID (CHOLESTEATOMA)*

by DARAB K. DASTUR, Bombay.

The purpose of this communication is to place on record an instance of a cerebral epidermoid, which by virtue of its uncommon location gave rise to an interesting clinical effect.

CASE REPORT

Clinical Features: S.V., a Hindu male aged 60, was admitted to the surgical wards of the J. J. Hospital at Bombay on 21st November, 1953. He was brought immediately after being injured in a motor-car accident. There was a contused and lacerated wound on the dorsum of his left foot. The patient was conscious but did not answer questions relevantly and no history could be obtained from him.

He remained in the wards for nearly five weeks as his wound did not heal. During this time it was noted that he exhibited a certain paucity of speech and peculiarity of behaviour. The latter consisted of provoked outbursts of laughter and a tendency to suddenly run away into the compound and occasionally to climb up a tree there.

Routine clinical examination revealed nothing of note. He looked his age and was very thin, but — as deduced from his performance — his motor power and coordination were quite normal. No laboratory or radiologic investigations were conducted. He is not reported to have complained about his foot, even though a large ulcer persisted at the site of injury.

A week before his death, he had mild diarrhoea for a couple of days. He was constipated for the last three days of his life. At no stage of his illness did he complain of any gastro-intestinal symptoms. On 25th December, he had his lunch and a glass of milk in the evening, as usual, and was seen sitting up in his bed at 6-30 P.M. At 9 P.M. he was found dead.

Post-Mortem Findings: The weight of the body at the time of autopsy was 38 kg. Dorsum of the left foot showed a clean ulcer, about 4" x 3" in size, in the floor of which the extensor tendons were seen. The pertinent findings were restricted to the abdomen and the brain.

A right sided inguinal hernia was present. On opening the abdomen, marked congestion of the peritoneal surface of the small and large intestines was noted. Coils of the ileum, about a foot in length, were found strangulated in the hernial

sac. About 6" of this bowel was greenish-black with gangrene, and showed one large and two small perforations. Through these, faecal matter had poured out with resultant peritonitis. The stomach was found distended with fluid with a faecal odour. The liver weighed only 770 G and the spleen 60 G, and both appeared congested.

The scalp and skull were normal. On incising the dura, a large firm swelling was seen, presenting itself between the frontal lobes. It was in the region of the falx cerebri, and appeared to arise from the genu of the corpus-callosum. It had invaded both the frontal lobes, through the thinned out cortex of which, it could be palpated. Its surface was pearly-grey, glistening and nodular. Coronal sections through the brain revealed the tumour to extend from the anterior portion of the thalami—behind, to the frontal poles—in front, measuring about 3" across and 2½" vertically (Fig. 1).

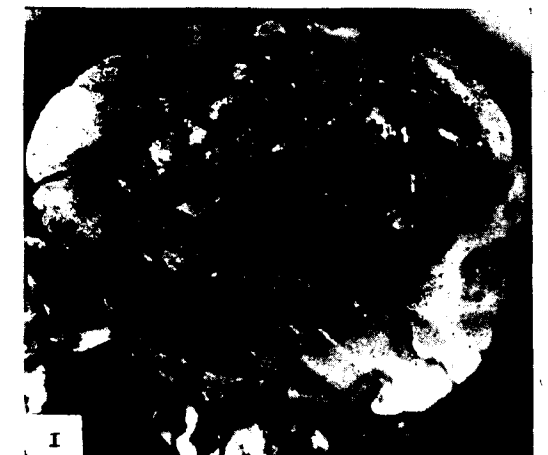


Fig. 1.

A coronal section through the middle of the frontal lobes showing the extent and gross appearance of the tumour.

The tumour had a capsule all round. The cut surface was greyish-white and glistening and presented a laminated appearance. It was soft and friable. The brain with the tumour weighed 1230 G.

Paraffin sections of the tumour and the surrounding brain were stained with Haematoxylin and Eosin and modified Masson's trichrome. Histologically the tumour was seen to consist of

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Fig. 2.

A low-power picture showing the laminated structure of stratum fibrosum, and the homogeneous appearance of stratum durum (in the centre of the field).



Fig. 4.

High power view of the cells of stratum granulosum, showing keratohyaline granules.

four characteristic layers. The outermost was stratum durum which was discernible only in parts (central, pale area in Fig. 2). It was made up of structureless material resulting from the cornification of epithelial cells. Stratum granulosum — the most distinctive part of the tumour — was seen in all the sections as a single or double layer of squamous cells. This layer was reminiscent of the squamous epithelium of normal epidermis (Fig. 3), and many of its constituent cells contained basophilic keratohyaline granules (Fig. 4).



Fig. 3.

Medium power view of the typical stratum granulosum resembling squamous epithelium. Adjacent part of the brain shows glial reaction.



Fig. 5.

Medium power view of a cluster of polygonal cells of the stratum cellulosum, surrounded by stratum fibrosum.

The bulk of the tumour was made up of stratum fibrosum, consisting of concentric layers of wavy homogeneous eosinophilic material, looking like the peels of an onion (Figs. 2 and 5). The highly friable core of this epidermoid constituted the stratum cellulosum (or 4th layer of Bailey), made up of a mass of polyhedral cells with very finely granular cytoplasm (Fig. 5). The cerebral cortex surrounding the tumour showed a mild astrocytic gliosis (Fig. 3). Hairs, blood vessels,

or glandular elements were not encountered anywhere.

An examination of other areas of the brain only showed up a small old haemorrhage in the hippocampal cortex: a zone of rarefaction containing many astrocytes, prominent capillaries, and basophilic deposits of iron.

Microscopic examination of the gangrenous bowel revealed almost complete sloughing off of the mucosa, with necrosis and infiltration by polymorphs and histiocytes of the muscle coats and the serosa.

DISCUSSION

In the course of embryogenesis, portions of the primitive ectodermal layer occasionally get included and buried in the flexures of the foetal brain. That these inclusions may, in post-natal life, give rise to neoplasms in the cranial cavity, has been known since the time of Hemak (1854). These tumours, which are best called Epidermoids (Bostroem—1897), were studied in detail by Bailey (1920). The tumour here reported fulfills all the criteria of a cerebral epidermoid.

From among the several accounts of this tumour, may be mentioned those of Critchley and Ferguson (1928), Love and Kernohan (1936), Munro and Wegner (1937), Rand and Reeves (1943), and Grant and Austin (1950). The latter authors consider the over-all incidence of epidermoids to be 1% of all brain tumours. It appears that only a little over 200 cases have been reported so far.

The commonest sites of occurrence for intradural (cerebral) epidermoids are the cerebello-pontile and parapituitary regions, though they have been encountered in the frontal and parietal lobes, and in the intraventricular, supracallosal, pineal and suprasellar areas (Jefferson and Smalley, 1938). To the best of our knowledge, not a single case of a strictly sagittally situated epidermoid with bilateral invasion of the frontal lobes, has been reported till now.

The great majority of cerebral epidermoids described in the literature, had produced symptoms of a space-occupying lesion, the most frequent of which were

headaches, mental disturbances, and visual defects. But, on account of the extremely slow growth of the tumour and the consequent absence of signs and symptoms of raised intracranial pressure, it is often difficult to arrive at a correct diagnosis or even to suspect this tumour (Northfield and Russell, 1951). Peyton and Baker (1942) have commented on the high incidence of epidermoids in the necropsies in mental hospitals.

The tumour reported here had extended from the basal ganglia behind to the frontal poles in front, thereby destroying a considerable proportion of the white matter of the frontal lobes. This naturally produced bilateral frontal lobotomy might explain why the patient never appeared to suffer from any pain, the lacerated wound of the foot, the strangulation and ultimate perforation of the bowel, as also the large space-occupying lesion in the brain having been tolerated without any complaint whatsoever. As Dynes and Poppen (1949) have pointed out, by the interruption of thalamo-cortical projections, the patients are divorced from their suffering. In the present case, this was achieved spontaneously and with fatal results.

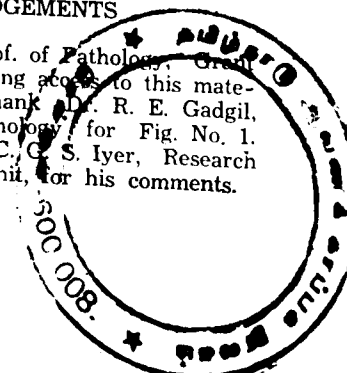
The mental apathy, inattentiveness, and euphoria which the patient exhibited were suggestive of the frontal-lobe syndrome (Denny-Brown, 1951), but, unfortunately, their significance was not realized, and the diagnosis was only made post-mortem.

SUMMARY

The case of a male aged 60, with a large silent mid-line cerebral epidermoid, is reported. A natural, bilateral, frontal lobotomy was produced. The patient died of an asymptomatic strangulated, perforated hernia.

ACKNOWLEDGEMENTS

I am grateful to the Prof. of Pathology, Grant Medical College, for allowing access to this material. I would like to thank Dr. R. E. Gadgil, Assistant Professor of Pathology, for Fig. No. 1. I am also grateful to Dr. C. S. Iyer, Research Officer, Neuropathology Unit, for his comments.



REFERENCES

1. Bailey, P.: Curvillier's "Tumour Perlees", S.G.O. 31: 390, 1920.
2. Bostroem, E.: (1897). Quoted by Critchley and Ferguson.
3. Critchley, M. and Ferguson, F. R.: The cerebrospinal Epidermoids (Cholesteatomata) Brain. 51: 334, 1928.
4. Denny-Brown, D.: The frontal lobes and their functions. In — "Modern Trends in Neurology", edited by Feiling, A. Butterworth and Co., Ltd., London. P. 13, 1951.
5. Dynes, J. B. and Poppen, J. L.: Lobotomy for Intractable Pain. J.A.M.A. 140: 15, 1949.
6. Grant, F. G. and Austin, G. M.: Epidermoids: Clinical Evaluation and Surgical results. J. Neurosurg. 7: 190, 1950.
7. Jefferson, G. and Smalley, A. A.: Progressive facial palsy produced by intratemporal epidermoids. J. Laryng. & Otol. 53: 417, 1938.
8. Love, J. G. and Kernohan, J. W.: Dermoid and Epidermoid Tumours (Cholesteatomas) of the Central Nervous System. J.A.M.A. 107: 1876, 1936.
9. Munro, D. and Wegner, W.: Primary Cranial and Intra-cranial Epidermoids and Dermoids. New Eng. J. Med. 216: 273, 1937.
10. Northfield, W. C. and Russell, D. S.: Intra-cranial Tumours. In — "Modern Trends in Neurology", edited by Feiling, A. Butterworth and Co., Ltd., London. P. 291, 1951.
11. Peyton, W. T. and Baker, A. R.: Epidermoid, Dermoid and Teratomatous tumours of Central Nervous System. Arch. Neurol. & Psychiat. 47: 890, 1942.
12. Rand, C. W. and Reeves, D. L.: Dermoid and Epidermoid Tumours (Cholesteatomas) of the Central Nervous System. Arch. Surg. (Chicago). 46: 350, 1943.
13. von Remak, R.: 1854). Quoted by Rand and Reeves.

ELECTRO-ENCEPHALOGRAPHIC STUDY OF FOCAL SEIZURES

by BALDEV SINGH and JACOB CHANDY, Vellore.

Seizures which do not have a Jacksonian march, but remain confined to a localized region of the brain are referred to as focal. In this communication we do not propose to consider a discharge focal when it spreads to other zones after having begun at one locus. The convulsions in focal fits remain confined to, a group of muscles one limb or one side of the body. In a series of 358 consecutive E.E.G. studies in the department of Neurology and Neurosurgery in the Christian Medical College Hospital, Vellore, focal discharges were found in 58 cases, i.e. 16%. In contrast, the figures given by Gibbs et al is 5%. This is because the E.E.G. studies in our cases have been selective and not routine for all patients suffering from fits. In all these cases the recording has been monopolar with Grass 8 channel machine. Electro-Corticography was done with the Grass Cortical Electrode assembly and depth electrode in the operation theatre.

TYPES

In this series there were found five types of focal discharges:—

(1) Delta waves:— (Fig. 1) is a typical representative of delta wave discharge. It consists of high voltage waves with a frequency of from 3 to 4 waves per second. This is the most frequent type of wave pattern found in this series being present in 26 out of 58 cases. This is because many of the suspected brain tumour cases are referred to this clinic. In addition to tumours, near the cerebral surface, other lesions giving this type of electrical disturbances are, demyelinating diseases, subdural haematomas and encephalitis, respectively. It may be emphasized here that in contradiction to the general belief, demyelinating diseases are a fairly common cause of fits and neurological disorders in this country. Central area, which consists of Parietal lobe with other lobes verging on it, is the commonest site of damage. Twenty one out of twenty six cases had lesion in this zone. The other five had pathology in the occipital and temporal regions respectively. The age incidence ranges from 3 to 53 years, the maximum number of cases being from 20 to 35 years of age. The sex incidence was 15 males and 11 females.

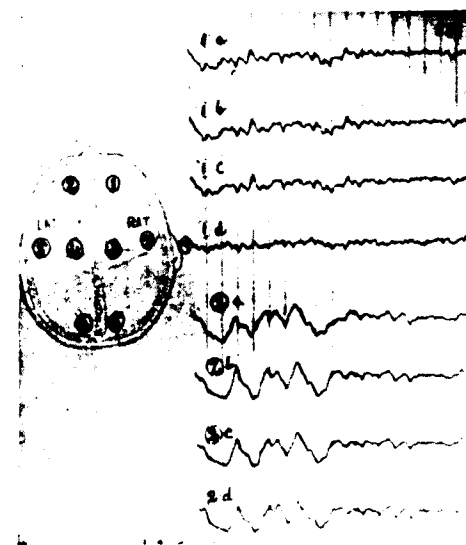


Fig. 1.

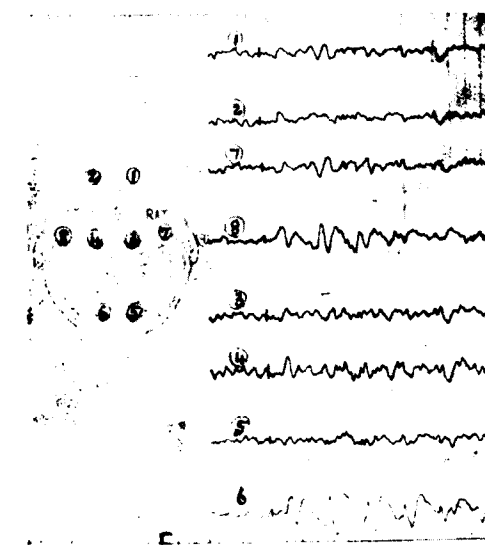


Fig. 2.

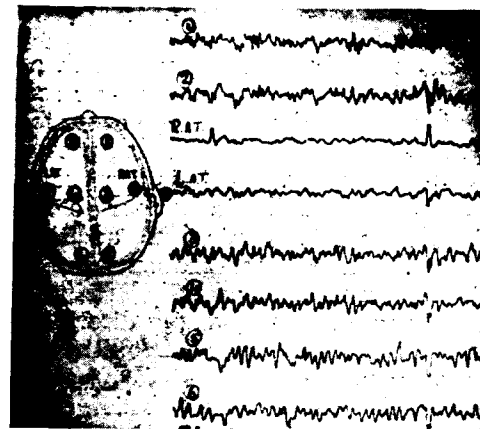


Fig. 3.

(2) Random spikes and sharp waves (Fig. 2). This type of electrical discharge was found in 21 out of 58 cases in the present series. The most usual site was the midtemporal zone which consists of Parietal and Frontal opercula as well as the whole of Temporal lobe, except the Temporal tip. Eighteen out of twenty one cases showed damage here. The rest of the three had an occipital focus. The age incidence was from 7 months to 42 years, the maximum being between 12 to 20 years. There were 19 males and 2 females. The most usual pathological lesion was recovering encephalitis and birth trauma, respectively. Tumours and degenerative lesions were least common in this group.

(3) Psychomotor spikes (Fig. 3) were found in 7 cases, all males, between the ages from 11 to 21 years. The right and the left anterior temporal zones were the sites of damage. There was a high incidence of birth trauma in these cases. Penfield et al⁸ have demonstrated recently that the lesion is due to vascular obstruction, either venous or arterial or both at the hiatus tentorii produced during parturition at the time when temporal lobe is in a stage of development most vulnerable to anoxaemia. Isaiah² et al have found

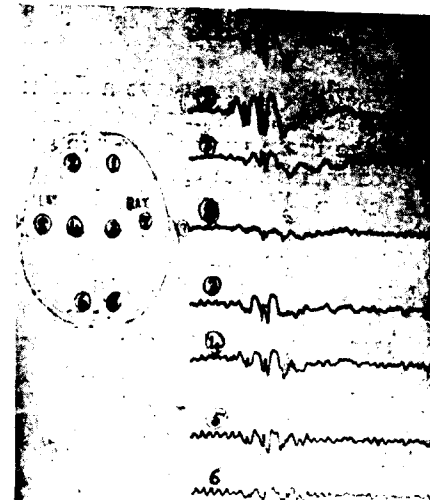


Fig. 4.

further that such a damage is likely to be maximum if the emissary vein shunt fails during the moulding of the head in the birth canal.

(4) Petit mal variant type of discharge is represented in Fig. 4. It was present in 3 cases only, all male, between the ages from 7 to 17 years. The focus was extensive involving the central zone in all cases. Encephalitis and trauma were the only two aetiological factors detected in these patients.

(5) Petit mal type of discharge (Fig. 5) was found only in one male 16 years of age with a history of having had some type of Encephalitis at the age of 8 years. The E.E.G. showed a right frontal zone focus.

Electro-Corticography was done in all cases who were chosen for ablation. Fig. 6 is one of the records of this investigation. Although usually the wave pattern by surface electrodes tallied in frequency and form with the one recorded by Electro-corticography this was not invariably so. Evidently, the tissues intervening between the brain and the surface have an effect on the conduction and voltage of the discharge. All the Electro-corticography was done under I.V. Pentothal. It has been detected

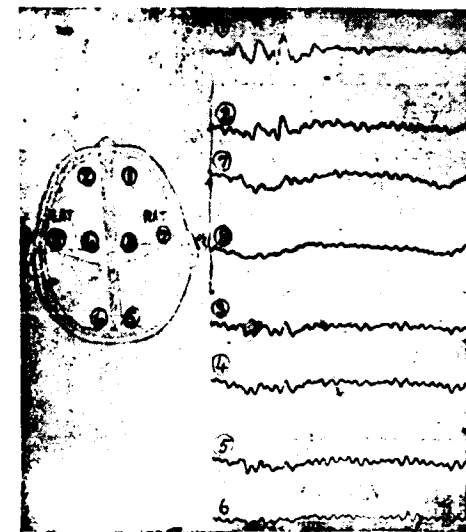


Fig. 5.

that there is an optimum rate of I.V. drip which varies with each case to give the best electrical activity of the region of brain under investigation.

Anatomical localisation of focal seizures indicates that (i) the central and mid-temporal zones were most frequently involved in giving Delta waves, random spikes and petitmal variant type of discharge, (ii) occipital zone usually showed random spikes, (iii) the frontal zone random spikes and petitmal type of discharge, and (iv) the anterior temporal region the psychomotor type of spikes.

Some other features noticed in regard to the anatomical localization are as follows:—

(1) The central area:— The negative spikes from this region usually spread to the opposite parietal area as low voltage negative spikes. Auras of Paraesthesia involving one side of face, a limb or one side of the body were present only in about 28% of our cases. Neurological signs, however, are very frequently met with.

(2) Mid-temporal zone:— Negative spikes from this region spread as positive

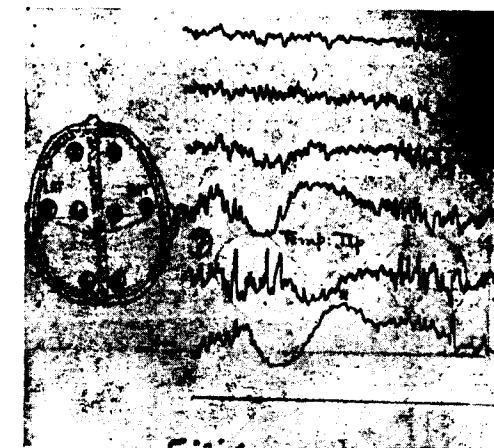


Fig. 6.

spikes to both the frontal areas, to both the parietal and rarely to the opposite mid-temporal zone. A focus here, if it starts in childhood, disappears spontaneously in some of the cases. Aura is present only in about 32% cases and consists of inability to talk, fear or nausea respectively. Some of these patients never learn to talk, are fearful, hypermotile and are behaviour problems. Impaired hearing when present is easily detected by diminished or absent arousal response to a loud noise made when the patient's E.E.G. record is being taken during sleep.

(3) Occipital zone:— Negative spikes from this area usually spread as negative spikes to the opposite homologous symmetrical zone. Spread to other zones is in the form of positive spikes. Aura of visual hallucinations was present in 15% of our cases. Strabismus is present in some cases. History of premature birth was noticed in 2 of our 4 cases. It has been surmised that the occipital cortex, not yet fully developed in a premature child, may get damaged by photic stimulation to give temporary seizure activity. Occipital foci found in children sometimes are noticed to disappear spontaneously in subsequent life.

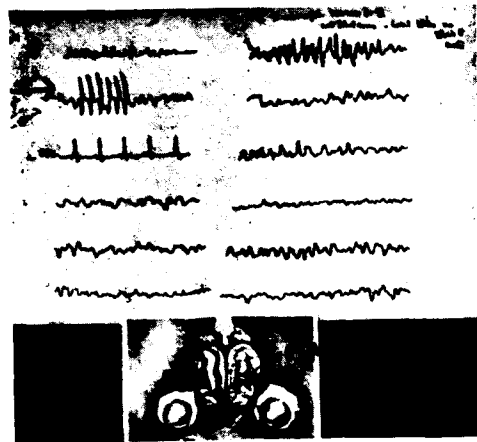


Fig. 7.

(4) Frontal zone:— Negative frontal spikes spread as low voltage negative spikes to the opposite frontal zone or positive spikes to both occipital zones. When they spread to one occipital region, it usually is the opposite one. Only 3% of cases in this series had aura, mostly gustatory.

(5) Anterior temporal zone:—The negative spikes when they spread to the opposite side are negative in sign. Spread to frontal zones is in the form of positive spikes. Aura of fear was present in 55% of our cases and one case had typical De-ja veu type of feeling.

DISCUSSION

The data on focal seizures enumerated so far needs elaboration on the following points:—

(1) The neurophysiology of various types of discharges and the factors responsible for their spread to other areas.

(2) The correlation of these discharges to auras, clinical features and treatment of the disease.

1. Normal cerebral excitation during awake period depends upon the process named desynchronization due to peripheral afferent impulses constantly stimulating the various regions of the brain. During epileptiform seizure this highly differential

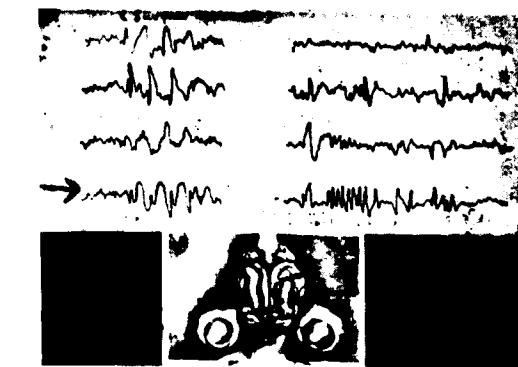


Fig. 8.

activity gives way to large numbers of nerve units becoming organised into a massive synchronised mechanism which becomes self activating and dysrhythmic. Synchronisation depends primarily on the spatial regulation of energy release in the aggregate of neurones. Inadequate block between cells permit them to fire in unison. Once locked in step the neuron mass develops excess of power and begins to give abnormal focal discharges which increase and spread as more and more neurons get involved. Pacella⁷ et al using local surface application and intracortical injection of aluminum cream in monkeys have produced a primary focus of Electro-encephalographic abnormality which spreads locally and is projected at distance in due course. Early ablation of such a focus stops progress of the lesion. Even in advanced stages such an ablation is helpful to overcome the disease partially.

The pattern of abnormal discharges, i.e. spikes, slow waves, and spikes alternating with slow waves, depend upon the size of neuron mass effected. By surface application and intracortical metrazol injection, Singh⁹ et al have demonstrated in cats that the various types of focal electrical discharges are determined by the extent of neuron area or neuropile affected by the drug. Fig. 7 represents the focal discharge

from the sigmoid gyrus where metrazol in 2.5% strength was applied on 2 mm. area. It shows focal spikes without any spread. When the same concentration of drug was applied to the whole of one hemisphere as in Fig. 8, the pattern of discharge changed to slow waves of various dimensions. Intracortical injection as in Fig. 9, gives wide spread of spikes in all zones of the cortex. This applies equally well to the human brain.

Gibbs¹ and Haynes have attempted to assess the localisation value of electrical sign in electro-encephalography. Their conclusion is that negativity indicates a local and positivity a distant disturbance when the sign is referred to an inactive area. The spread of negative sign to the homologous area is due to the fact that every time the primary area is stimulated, the secondary area gets synchronously excitable till by summation it begins to discharge an identical electrical wave pattern. This happens only if neurons of two areas have an identical form and function and are interconnected with one another. Positive wave transmission is an indication of the fact that although neural pathway exists between the two neuron aggregates, yet they are not identical in their disposition and special arrangement. Negative spikes therefore locate the focus which is active and is the primary seat of disease.

The electrical activity in its normal or abnormal form depends upon the bio-

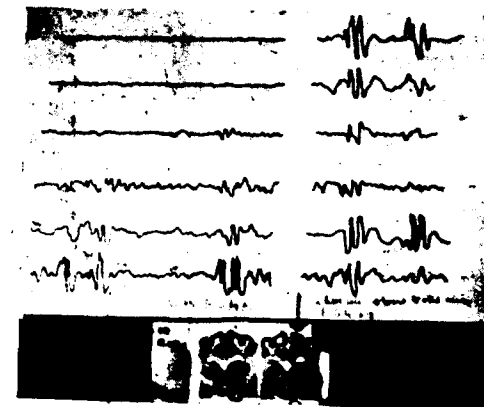


Fig. 9.

chemical changes in the metabolism of the cell. There are two types of systems described in this connection:—

- (1) The acetyl-choline system described by Nachmansohan⁶.
- (2) The Glutamic Acid system emphasised by Tower¹¹.

According to Nachmansohan, the sodium and potassium concentration gradient between inside and outside of cell determines electric potential change in the cell. When a stimulus reaches the cell surface it releases acetyl-choline from its bound form with the proteins which acts upon the protein of the cell membrane to break its resistance and allows rapid to and fro movement of Na & K ions. This process generates the electric current. The liberated acetyl-choline is rapidly hydrolysed by cholinesterase which allows proteins to assume the original resting position. The restitution of ion balance and reformation of acetyl-choline is a slow process which depends on the energy rich phosphates.

The Glutamic Acid system has more than one function in the metabolism of nervous tissue¹⁰. (1) Glutamic acid neutralizes ammonia liberated during neural activity. If this ammonia is allowed to accumulate it leads to convulsions. Glutamic acid is changed to Glutamine in this chemical reaction. (2) Its change to alpha-glutamic acid in a reversible combination makes it possible for the neurons to utilise Krebs' cycle, which is a universal chemical reaction in the body, for getting nutrition from proteins and carbohydrates. (3) Its association with the synthesis of acetyl-choline, especially its bound form is helpful to regulate the effect of free acetyl-choline in impulse generation. (4) It's effect to increase cell membrane permeability and absorption of K ions following the discharge of the impulse, is especially useful to control the neuron excitability. (5) Most of the anticonvulsants with the exception of the dione group help the energy rich phosphates to act on proteins to bind the free acetyl-choline. Glutamic acid helps as an adjuvant to this effect of the anticonvulsants.

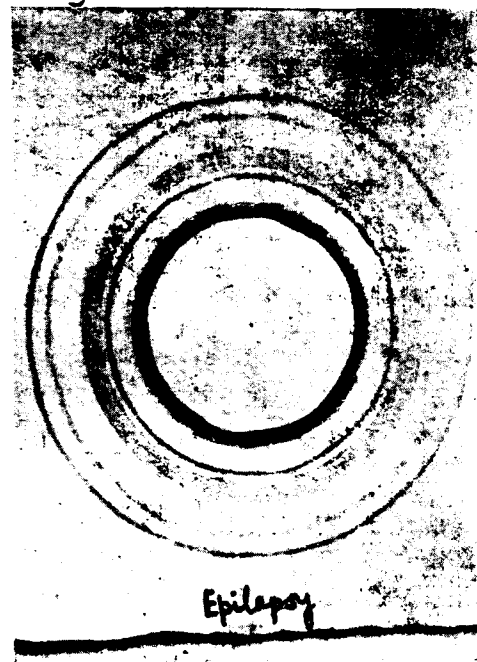


Fig. 10.

Figs. 10 and 11 give our findings regarding C.S.F. Amino acids in normal¹⁰ and epileptic individuals. The main feature of these findings is the diminution of Glutamic acid in vivo. The deficiency increases with the size of the focus and is markedly increased where the damage to the cortex is most advanced.

2. The correlation of focal discharges to auras and clinical features depends upon the anatomical substratum of the cortical region damaged and the extent of such a damage. Clinical manifestations are abnormalities in the normal function of the diseased area. A spike discharge which is produced by a smaller damage gives rise to a comparatively less marked functional derangement than Delta waves or spikes and slow waves. The quality of aura and intensity of convulsive manifestations differ with the difference in the type of the discharge.

The functions of frontal, parietal and occipital lobes are well known and the

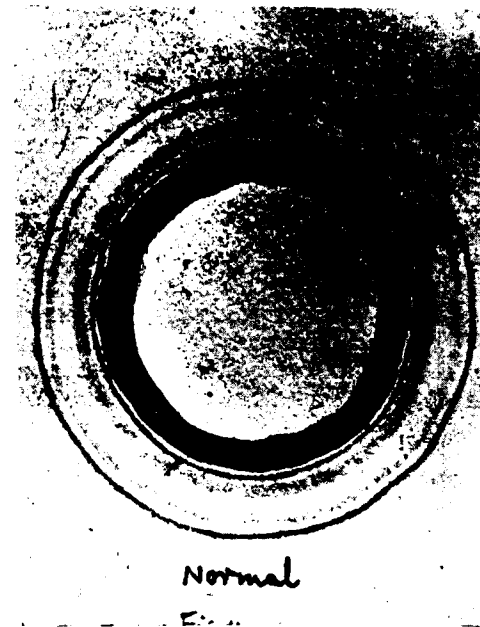


Fig. 11.

explanation for gustatory, paraesthetic and visual hallucination auras need not be entered into. However, the interpretation for the aura of fear in Temporal lobe fits has been possible recently due to the work done on the connections of this lobe to the hypothalamus by Marsan⁵ et al. The supplement of fear element in the aura and in the general personality of the patient seems to be due to the derangement in the projections between the hypothalamus and the Temporal lobe.

The E.E.G. study of focal discharges and the biochemical changes associated with them have given insight into the problems of medicinal and surgical treatment of focal fits.

The ammonia neutralizing and acetylcholine binding effect of glutamic acid and other supplementary amino-acids have increased the importance of these compounds in the therapy of fits. In our hands smaller doses of Glutamic acid, i.e. gr. 7½ two or three times a day are found to be

satisfactory as adjuvant to anticonvulsants. Intrathecal and intravenous administration of glutamic acid salts will be found more useful when they are made available.

Surgical treatment by ablation of epileptogenic foci is based on the following considerations:—

(1) All focal fits should first be given a full trial for treatment by medicinal means. This will not only exclude cases who cure spontaneously or by medication, but will also reduce the area for ablation in persistent disease.

(2) Pacella et al has shown that ablation of epileptogenic focus relieves the animal of chronic epileptic fits. Kopeloff et al⁴ have emphasized further that if a secondary epileptogenic focus has already developed, it is very important to distinguish it from the primary focus by E.E.G. studies before ablation is planned to deal with the latter. In case the mistake is made to remove the secondary instead of the primary focus, the animal becomes more vulnerable to the spread of the disease, after the operation. This localization is done by giving I.V. Pentothal drip. The secondary focus stops firing much earlier under influence of Pentothal as compared to the primary focus.

(3) The ablative surgery should be preceded and followed by anti-convulsant and adjuvant therapy for an adequate period.

(4) Ablation gives good results for spike foci, psychomotor discharges and Delta wave seizures. Spike and dome foci when ablated produce wide spread abnormal discharges in the rest of the cortex. Every case with ablation should continue with the medicinal treatment for an adequate period after the operation.

SUMMARY

1. Five types of focal electrical discharges in 58 cases suffering from focal fits have been described.

2. The neurophysiological and biochemical explanation of the focal dis-

charges and their spread has been given. It is pointed out that synchronization of neuron aggregates of various sizes determines the type of discharge, i.e. spikes, slow waves or spikes and slow waves. The conduction of spikes with the negative or positive sign depends on the identity in the structure of the primary and secondary loci. If the two sites are homologous and are symmetrical in structure the projection is negative although of lower voltage. If these conditions are not satisfied but the areas are interconnected otherwise, the projection is positive. High voltage negative spikes are interpreted to localise the lesion. Low voltage negative spikes or positive spikes are just projections from the area of damage. The normal electrical activity depends upon a balanced interaction of acetylcholine and glutamic acid systems. The degree of imbalance and consequent synchrony determines the abnormality of the discharge. By paper chromatography the deficiency of glutamic acid in c.c.f. of epileptics has been demonstrated. The role of glutamic acid in normal function of the neuron and its relation to Krebs's cycle has been stressed.

3. The clinical manifestations and auras produced by damage to the various regions of the brain have been given a brief mention. These features are derangements of the normal function of the region effected. The recent work on temporal lobe fits and auras and the neural connections of this lobe to hypothalamus have been described.

4. Treatment of focal fits depending upon the study of focal discharges has been clarified. All focal fits must first be treated with anti-convulsants and adjuvants especially glutamic acid as some of these cases have a reversible damage.

5. If there are two independent foci in homologous areas, one of them alone is usually primary as determined by I.V. Pentothal drip. For ablation primary focus should be dealt with. If the secondary focus is removed instead of the primary, the latter flares up and spreads more rapidly.

REFERENCES

1. Gibbs, F. A. and Hayne, R.: The localizing value of Electrical sign in Electroencephalography. *Dis. Nerv. System.* 9: 389-390, 1948.
2. Isaiah, P. and Chandy, J.: *Caput Succedenum* (In Press).
3. Jasper, H. H.: *Electroencephalography in Epilepsy* — 186. *Epilepsy* by Hoch & Knight, 1947 (Grune & Stratton, New York).
4. Kopeloff, L. M., Chusid, J. G., Pacella, B. L. and Kopeloff, N.: Experimental chronic Epilepsy in monkey. *Arch. Neurol. Psych.* 67: 336-342, 1952.
5. Marsan, C. A. and Stol, J.: Subcortical connections of the temporal pole in relation to temporal lobe seizures. *Arch. Neurol. & Psych.* 66: 669-686, 1951.
6. Nachmansohan, D.: *Energy Sources of Bio-electricity Phosphorus metabolism*, Vol. 1, edited by W. D. McElroy & B. Glass (The John Hopkins Press, Baltimore, 1951).
7. Pacella, B. L., Kopeloff, L. M. and Kopeloff, N.: Electroencephalographic studies on induced and excised epileptogenic foci in monkeys. *Arch. Neurol. & Psych.* 58: 695-703, 1947.
8. Penfield, W., Baldwin, M. and Earle, K. M.: Incisural sclerosis and temporal lobe seizures. *Arch. Neurol. & Psych.* 69: 27-42, 1953.
9. Singh, B., Amador, L., Senser, W. and Cranday, P.: Effect of metrazole on the electrical activity of cat's brain. Meeting of the American E.E.G. Soc. Symposium on Petimal, 1951.
10. Singh, B., Joshua, D. and Chandy, J.: Amino-acid content of C.S.F. (In Press).
11. Tower, D. B.: Mechanism of seizures, etc. *Epilepsia*, 3rd series. 1: 88-96, 1952.

ASSOCIATION NOTES

MINUTES OF THE EXECUTIVE COMMITTEE MEETING

(Held on 22nd Feb. 1954 at Calcutta.)

An Executive Committee Meeting of the Neurological Society of India was held on the 22nd February, 1954, in the Tropical School of Medicine, Calcutta. Dr. T. K. Ghosh, the President was in the chair and the following members were present:

Dr. Ram G. Ginde,
Dr. C. G. S. Iyer,
Dr. W. Grillmayr,
Dr. Jacob Chandy,
Dr. Baldev Singh,
Dr. S. T. Narasimhan, and,
Dr. B. Ramamurthi.

The following resolutions were passed:

1. Admission of New Members: The following were admitted as Full Members, as they are practicing full time neurology:

Dr. Ram G. Ginde, Bombay

Proposed by B. Ramamurthi.

Seconded by C. G. S. Iyer.

Dr. Bharucha, Bombay

Proposed by C. G. S. Iyer.

Seconded by S. T. Narasimhan.

2. It was decided to recommend to the General Body to admit the following as as Honorary Members:

Dr. Denny Brown

Proposed by C. G. S. Iyer.

Seconded by T. K. Ghosh.

Dr. I. S. Wechsler

Proposed by T. K. Ghosh.

Seconded by C. G. S. Iyer.

3. The Balance Sheet of the Society was presented and as accepted. The Balance Sheet shows a balance of Rs. 559-3-0 to the credit of the Society.

4. The Executive Committee passed a resolution, thanking Dr. Jacob Chandy for the donation of Rs. 201-13-0, towards the publication of the 1st issue of the Journal 'NEUROLOGY'.

5. The Executive Committee passed a resolution, accepting the proposal of the Treasurer to send Bank Order Forms to the various members for paying their subscriptions.

6. The Executive Committee authorised the Secretary to sign along with the Treasurer to withdraw money from the Bank whenever necessary.

7. The Executive Committee passed a resolution, thanking Dr. T. K. Ghosh for his very valuable services as President.

8. The Executive Committee passed a resolution, thanking the Reception Committee, at Calcutta, for the excellent arrangements they had made for the Third Annual Conference of the Neurological Society of India.

MINUTES OF THE GENERAL BODY MEETING

(Held on 22nd Feb. 1954 at Calcutta.)

A General Body Meeting of the Neurological Society of India was held on the 22nd February, 1954, in the Tropical School of Medicine, Calcutta. Dr. T. K. Ghosh was in the chair and the following members were present:

Dr. Ram G. Ginde,
Dr. C. G. S. Iyer,
Dr. W. Grillmayr,
Dr. Jacob Chandy,
Dr. Baldev Singh,
Dr. S. T. Narasimhan, and,
Dr. B. Ramamurthi.

1. The General Body approved the admission of the two New Full Members, viz., Dr. Ram G. Ginde, and Dr. Barucha, and the two Honorary Members, viz., Dr. Denny Brown, and Dr. I. S. Wechsler.

2. The Accounts as submitted by the Treasurer was accepted.

3. As there was another Journal with the name 'Neurology', it was decided to change the name of the Journal of the Society as Indian Journal of NEUROLOGY. The Secretary explained the difficulties in getting advertisements for the Journal and the finances of the Journal and requested greater co-operation from the members in the matter of procuring advertisements.

4. The General Body endorsed the resolution of the Executive Committee, authorising the Secretary to sign along with the Treasurer to withdraw money from the Bank whenever necessary.

5. It was resolved to hold the next annual meeting of the Society is to be held along with that of Association of Physicians of India, and it was resolved to request the Railway Board to consider giving half concession traveller form for members attending the annual conference as this annual conference is being held along with two other Associations, and as the policy of the railway board is to encourage railway traffic by issuing concessions.

6. The following office-bearers were then elected:

Dr. Ram G. Ginde, — President

Proposed by C. G. S. Iyer.

Seconded by S. T. Narasimhan.

Dr. B. Ramamurthi was elected to continue as the Secretary, and,

Dr. S. T. Narasimhan were elected to continue as the Treasurer, unanimously. (Proposed from the chair).

7. The General Body passed a resolution thanking Dr. T. K. Ghosh, the retiring president, for his services.

8. The General Body passed a resolution thanking the Reception Committee, at Calcutta, for the excellent arrangements they have made for the third annual conference of the Neurological Society of India.

BRIEF REPORT ON THE THIRD ANNUAL CONFERENCE OF NEUROLOGICAL SOCIETY OF INDIA

(Held on 20th to 23rd Feb. 1954 at Calcutta.)

The Third Annual Conference of the Neurological Society of India was held at Calcutta from 20th to 23rd February, 1954, along with the Association of Physicians of India, Association of Pediatricians of India, and Cardiological Society of India. Dr. B. C. Roy, Chief Minister of West Bengal, inaugurated the conference. Dr. T. K. Ghosh, delivered a very inspiring Presidential Address.

On the morning of the 21st February, a combined meeting of the Neurological Society of India along with other associations was held in which the following papers were presented:

Pediatric Aspects of Intracranial Injuries

by Dr. P. M. Udani, Bombay.

Agenesis of Corpus Callosum—A Case Report

by Dr. P. M. Udani, Bombay.

The following papers were presented on the 22nd February:

Tubercular Granuloma

by Dr. R. G. Ginde, Bombay.

Incidence of Disseminated Sclerosis

by Dr. B. Ramamurthi, Madras.

Latent Syphilitic Diseases and their diagnosis

by Dr. W. Grillmayr, Colombo.

The Barbiturate Electroencephalogram

by Dr. R. B. Davis, Ranchi.

Muscular Dystrophy

by Dr. C. G. S. Iyer, Bombay.

Glioma of Hypophysis

by Dr. R. G. Ginde & Dr. C. G. S. Iyer, Bombay.

A Symposium on Epilepsy was held in the afternoon and was largely attended by the members of the four associations. The following speakers participated in the symposium:

Fits in Children

by Dr. Baldev Singh, Vellore.

E.E.G. in Epilepsy

by Dr. S. T. Narasimhan, Madras.

Medical Treatment of Fits

by Dr. R. B. Davis, Ranchi.

Surgery of Epilepsy

by Dr. Jacob Chandy, Vellore.

Social Problems in Epilepsy

by Dr. B. Ramamurthi, Madras.

This was followed by a number of questions, which were answered by the speakers.

The following papers were presented on the 23rd February:

Autonomic Influence in Health and Disease

by Dr. V. K. Pillai, Trivandrum.

Electro Encephalographic Study of Focal Discharges
by Dr. Baldev Singh, Vellore.

Migraine—Its Dissimilarities of seizures with Epilepsy
by Dr. N. N. Das, Calcutta.

Surgery of Epilepsy by Ablation & Hemispherectomy
by Dr. Jacob Chandy, Vellore.

Cerebral Angiography in Intracranial Lesions
by Dr. B. Ramamurthi, Madras.

MINUTES OF THE EXECUTIVE COMMITTEE MEETING

(Held on 23rd February 1954 at Calcutta.)

An Executive Committee Meeting of the Neurological Society of India was held on the 23rd February 1954 in the Tropical School of Medicine, Calcutta. Dr. R. G. Ginde, the newly elected president, was in the chair and the following members were present:

Dr. T. K. Ghosh,
Dr. W. Grillmayr,
Dr. Jacob Chandy,
Dr. Baldev Singh,
Dr. N. N. Das,
Dr. R. B. Davis,
Dr. S. T. Narasimhan, and,
Dr. B. Ramamurthi.

1. The Subject of Paraplegias was accepted for the symposium for the year 1955, and the following speakers were accepted:

1. Neuro-physiological Aspects
by Dr. Baldev Singh, Vellore.
2. Medical Aspects
by Dr. Barucha, Bombay.
3. Intra Spinal Tumours
by Dr. B. Ramamurthi, Madras.
4. Surgical Aspects (other than tumours)
by Dr. Jacob Chandy, Vellore.
5. Control of Bladder and bed sores
by Dr. R. G. Ginde, Bombay.

20 minutes each paper.

2. Dr. T. K. Ghosh suggested that the Neurological Society of India must give lead to the various Universities in India, regarding the teaching of undergraduates and postgraduates 'NEUROLOGY'. After some discussion, the committee accepted the suggestion from Dr. Ramamurthi that data of the methods of teaching 'Neurology' 'Neuro-surgery' to the undergraduates and postgraduates in the various universities to be collected and the matter may be discussed again next year.