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159

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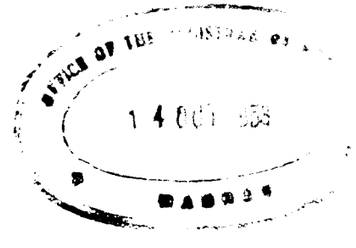

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CONTENTS JUNE 1953

NOTICE TO CONTRIBUTORS

Neurology, journal of the Neurological Society of India publishes original articles in Neurology and Allied Subjects. Papers for publication and all editorial communications should be addressed to the Secretary, Neurological Society of India, 43, Harris Road, Madras 2. Papers that are accepted are published on the understanding that they have not been and will not be published in whole or in part in any other journal.

Manuscripts: Manuscripts must be typewritten on one side on quarto or foolscap sheets, double space and with a margin of not less than two inches. They should bear the name and full address of the author to whom proofs are to be sent. The paper should conclude with a brief summary stating the conclusions described in the paper.

Tables and Illustrations: Tables should be numbered in Roman numerals and set out on sheets separate from the text. The illustrations should be kept separate from the text and should be numbered consecutively in Arabic numerals. Each figure should be referred to in the text and the descriptive legends should be typewritten on a separate sheet of paper. Nothing should be written or typed on the back of the illustrations. Photographs and photo-micrographs should be printed on glossy paper and should not be larger than 6.5 x 6.5 cms. If transmitted through post they should not be folded and should be protected by corrugated cardboard paper.

Illustrations will not be accepted unless they reach a certain standard of excellence technically, present an attractive appearance and add significantly to the value of the presentation.

References: These should be arranged alphabetically by authors. For citation of a book, give in order author's name, complete title, place of publication, name of publishers, year and page. For an article author's name, initials, full title of paper exactly as it appears, name of journal (abbreviated as in the Quarterly Cumulative Index Medicus), volume number (in heavy type Arabic numerals), inclusive page numbers and year. The following examples illustrate the desired punctuation and arrangement:

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.

All communications regarding membership of the Society, subscription to the Journal, and advertisements, may be addressed to the Secretary, Neurological Society of India, 43, Harris Road, Madras 2.

NEUROLOGY

Vol. I JUNE 1953 No. 1

CONSTITUTION & BYE-LAWS OF THE NEUROLOGICAL SOCIETY OF INDIA

Constitution

1. The name of the Society shall be "The Neurological Society of India.

2. The aims and objects for which the Society is set up are:

(a) To maintain close association and co-operation among those devoting full time to Neurology and its associated branches.

(b) To maintain the highest standard in the ethics and practice of this speciality.

(c) To maintain this high standard, every endeavour shall be made to give adequate training to those who are properly qualified and who intend to take up this speciality.

(d) To promote and encourage original research in the clinical and experimental work pertaining to this speciality.

3. In pursuit of these primary objects, there shall be Members, Associate Members, Corresponding Members and Honorary Members, and the Society shall undertake the appropriate activities.

4. The Member shall be one who is devoting his full time to the speciality.

5. The Associate Member shall be one who is interested in the speciality.

6. The Corresponding Member shall be a person from a foreign country, who is devoting his full time to Neurology and its associated branches and who may not be able to attend at least one meeting every three years.

7. The Honorary Members are those who by reason of professional and scientific merit are deemed worthy of such election to the Society.

8. For members, the subscription shall be Rs. 20/- per year and for associate members, Rs. 10/- per year.

9. The Officers of the Society shall be:

(a) President.

(b) Vice-President.

(c) Secretary.

(d) Treasurer.

They shall be elected annually by the Executive Committee.

10. For the present, the Executive Committee shall consist of all the full Members of the Society.

11. There shall be at least one General Meeting of the Society every year.

12. The Society shall be affiliated to the International Neurological Society.

Bye-Laws

Membership

1. A Full-Member shall be proposed by any Full-Member and the Executive Committee shall be the authority to admit Members.

2. Each Full-Member shall present a paper to the Society at least once in two years.

3. Any Full-Member who fails to attend one at least of three consecutive meetings of the Society shall forfeit his Membership, unless a reason acceptable to the Executive Committee is offered.

4. Corresponding and Honorary Members shall be proposed by one Full-Member and be seconded by another Full-Member. Their election shall be carried out by direct invitation from the Committee. Such Members shall be exempt from the payment of annual subscription.

5. The position of each Associate Member shall be reviewed every four years to see whether the interest they have shown warrants their continuation of Membership.

6. The Associate Members shall be entitled to attend all the meetings of the Society and may take part in all discussion and have the privilege of Membership, except that they shall not vote.

Officers

7. The officers of the Society shall hold office for one year and shall be eligible for re-election.

8. The Office Bearers of the Society will be elected at the General Body Meeting, which will be held during the Annual Conference of the Society.

9. The Presidential address will be delivered by the President at the end of his term.

10. The Executive Committee shall transact all the business of the Society.

11. The Treasurer shall operate the funds of the Society.

12. The audited accounts of the treasurer must be submitted to the Executive Committee.

Local Meetings

13. Local Meetings of the Neurological Society of India can be held in different parts of India, if the interest of the members warrants it.

Amendments

14. All Amendments to the constitution and bye-laws shall be submitted to the Secretary at least four months before General Body Meeting of Full-Members and copy of such proposal shall be forwarded to every full-member, at least two months before the meeting. On motion, the amendment shall be accepted by a vote of 2/3rd of the total number of full-members of the Society.

15. A Journal shall be published by the Society.

TRUE EXTRACT OF A LETTER FROM THE OFFICER ON SPECIAL DUTY
(All-India Medical Institute) MINISTRY OF HEALTH, NEW DELHI.

COPY OF LETTER

To the Secretary, N.S.I., Madras.
D.O. No. 8-2/AIMI 53.

Ministry of Health,
NEW DELHI.
20th May, 1953.

Dear Dr. Ramamurthi,

I have no doubt that there will be Departments of Neurology and Neuro-Surgery in the All-India Medical Institute, but it is too early to indicate, at the moment, the approximate time at which these departments will be developed. The importance of these branches is such that there is little doubt that they will be developed as integral parts of the Institute.

With kind regards,

Yours Sincerely,
(Sd.) Dr. K.C.K.E. RAJA.

To

B. Ramamurthi,
Secretary, Neurological Society of India,
43, Harris Road, Madras-2.

INAUGURAL ADDRESS TO THE NEUROLOGICAL SOCIETY OF INDIA

by Sir A. L. MUDALIAR, M.D., LL.D., D.Sc., D.C.L., Madras.

The inauguration of the Association of Neurologists and Neuro-surgeons is an event of great significance in the march of events in the field of medical science in this country. I had looked forward to this day and had hoped to be present in person to share with you the joy that such a function has been rendered possible and to share alike with you the anxiety that a great responsibility now rests on your shoulders to prove yourselves worthy founders of such an association. Within the last two decades, several associations of a similar nature have been founded and have been holding annual or biennial session in different cities, where members of the profession practising or interested in such specialities foregather to discuss the many problems with which they are confronted and to contribute to the sum total of knowledge gained in the practice of the specialities.

It is a matter for regret that many of the specialities in medicine were not taken note of in the past with the result that Indians have had to go to foreign countries to study and to gain some knowledge of such specialities; consequently, there were few who had such facilities and who could help to train their countrymen in such specialities. I hold to the belief that no college could satisfactorily fill the role of undergraduate teaching unless simultaneously there were developed facilities for post-graduate study and teaching and necessary provision was available for research being carried on.

It is with this object in view that the Government of India in the Ministry of Health appointed a special committee on whose recommendation several departments of study in the different medical colleges are being upgraded. The grateful thanks of the medical profession are due to the Hon'ble Rajkumari Amrit Kaur, Minister of Health, who has with a singular foresight and devotion made this possible as well as the establishment of an All-India Council of Post-graduate Medical Education. Despite criticisms and comments, I venture to state that the establishment of such a Council is calculated to foster the development of post-graduate education along right lines and to ensure that proper standards are maintained so that the products turned out prove themselves worthy of the qualifications conferred on them and worthy of the trust and confidence that will be reposed in them by their colleagues in the medical profession and by the public. I am sure that your association which, though so young in age, yet is concerned with so highly specialised a field in medical science will appreciate and value the need for setting up high standards in your speciality.

I have always set greater store on the practical training that is available than on the academic distinctions that can be gained under such an imperfect system of examination. In my opinion, a neurologist should be a good physician before he can take to the specialised study of neurology and a neuro-surgeon should be a good general surgeon before he takes to the study of neuro-surgery. If academic qualifications be required as a criterion of specialisation, I would not hesitate to suggest for instance that a neuro-surgeon should have taken the Master's degree in general surgery and then specialise for two or three years before he can be recognised as a neuro-surgeon. Such specialised study should be in all branches of applied science — neuro-chemistry, neuro-pathology and bacteriology, neuro-anatomy and physiology, and allied sciences. It is for these reasons that stress should be laid on the facilities that are available in the shape of equipment and teaching personnel in the several fields of applied knowledge before recognition can be given to an institute as a centre for training of post-graduates in the speciality.

It is a matter for regret that to-day, not many centres can hope to satisfy the minimum requirements needed as stated above and yet qualifications are being awarded with such meagre training as is available in the vain hope that, after such qualifications, the persons will improve on their own initiative.

It is because of such obvious defects that the Government of India has constituted a Council to advise Universities and other bodies and so to persuade them to adopt standards to improve the facilities available.

India needs to-day the best that is available in the world and it is a penny-wise policy to be content with the second best. The independence that the country achieved on the 15th August 1947 is not merely independence from foreign rule but, if its significance is appreciated in full, if it is to connote anything in reality and if it is to be for the good of the country, it is the independence that will lead to India standing on the secure pedestal with the best and most advanced of the nations on the intellectual and moril plane no less than on the political plane. It is in this hope and with this longing that I offer you my warmest felicitations at this happy event.

NEUROLOGY COMES TO LIFE*
OR
THE PRECEPTS AND CONCEPTS IN NEUROLOGY

by JACOB CHANDY, Vellore.

The introduction of a new speciality in medicine to a sub-continent, which not only had been enslaved in conservatism, but also revels in myths and mysteries and foster a dead unscientific system of medicine on the plea of nationalism is not the occasion for any welcome. A few leaders in the medical profession may say that there is room for well-trained persons and sit back as a sympathetic audience to watch the Act one unfold. Some physicians and surgeons appear to be singularly disinterested. They have hitherto not recognised any particular need for this speciality. They have always treated these cases with satisfaction and hold the conviction that what they could not treat were not worth treating at all. But we have yet few others who are willing to support and take pride in the development of this speciality. To these doctors, in particular, I address my remarks.

More than 2000 years before Christ, the Egyptians had many specialists. The historian Herodotus made note that some undertook " to cure diseases of eye, others of the head, others again of the teeth, others of the intestines, and some, those which are not local ". For two millenniums those specialists continued to practise and to collect fees without making the slightest advance. Indeed they steadily degenerated. The same is true of Indian medicine. The trouble was that the Egyptians and we made the same fatal mistake. The early scholars had written hermetic books. Not only we and the Egyptians believed that those books were correct but also that there was nothing that could be added into its contents. There, to depart in any way from the treatment prescribed in those hermetic books was to endanger one's own life. This was a remarkable experiment. Even the science of medicine was frozen by rules, which may have been the best at that time. This experiment demonstrated that standardization can halt advance, but that it does not in any way hinder retrogression. Scientific progress, whether it be advancement in our fundamental knowledge or improvement in the diagnosis and treatment of diseases, is often depending upon the introduction and development of new techniques and approaches. Scientists and physicians, who devoted their full time in the study of some branch of science, were able to contribute considerably to our knowledge in that branch, and we are greatly indebted to their original and pioneering work which has paved the way for great progress.

The study of the nervous system had been one of the most difficult subjects and the progress had been rather slow. Clinical pictures of various diseases of the Nervous System were described and studied from time to time by various workers mostly in England and Europe. Those observations are on record even from the time of Hippocrates. During 18th and 19th centuries, much advance has been made both in the fundamental knowledge of the physiology of nervous system and in the studies of its many diseases. But the most outstanding period in the history of Neurology is the latter half of the 19th century. First of all, Neurology was recognised as a special branch of Medicine. A few people scattered throughout the world, devoted full time to this specialisation. Many of the clinical neurological syndromes were recognised and described. Much of the fundamentals of neuro-physiology was understood at this period and steady progress was made from that time onwards. The electrical activity of nerve cells was observed. Histopathological studies of the diseases of nervous system was started, but more rapid progress in this field was made only after the turn of the century. Chemistry of brain was little known, but definite attempts were made towards its study about that time. Extensive anatomical studies also bore much fruit. We owe much to those pioneers for their untiring work in establishing some of the fundamentals of neuro-anatomy. The recognised developments in Neuro-physiology, Neuro-anatomy and Neuro-pathology began to show more light to explain some of the clinical syndromes of the diseases of Nervous system. Better understanding of the diseases of the mind also came about at that time by the work of Freud and his school.

Till that time the treatment of diseases of Nervous System was practically nil, but about the turn of the century, the establishment of Neurological Surgery gave great hope for treatment as well as added to the optimism for further rapid progress of the science of Neurology. Thus Neurology gave birth to Neuro-surgery some time after the conception of localization of function in the central nervous system by Jackson, Broca, Fritschi, Hitzig and Ferrier. But there were many technical difficulties in the Surgery of the Nervous System. Even as late in 1906, Horsely himself made the following statement : " In fact the advancement in technique of surgical treatment of diseases of brain and spinal cord has been relatively less than the improvement in our knowledge of the seat and nature of the disease for which surgical intervention is useful and necessary ". If this was true of Horsley's own work, it was even more so of the leading surgeons in Europe, particularly Bergmann, Kocher, Krause von Eiselsberg, Chipault, Broca and others all of whom had made significant contributions to neuro-surgery and had written extensively on this subject, even though their results were certainly not nearly so good as those of Horsely. In other words, at the turn of the twentieth century it was evident that a large new field of surgery was opening, indeed had been opened, but that in this new field no one had as yet appeared who fully appreciated and was able to develop a surgical technique which was sufficiently delicate and meticulous, to make operations particularly on the brain, produce results comparable to the results of operation elsewhere in the body. Though Horsley, Macewen and Godlee and others started developing the surgical technique of cranial operations in England and Europe, it was Harvey Cushing in the United States who really brought up this speciality to firmer footing. Neuro-surgery came of age under the leadership of Cushing, Dandy, Frazier, Scahs, Mixter and others. That was a period of real technical evolution.

With the march of neuro-surgery, neuro-physiology including neuro-chemistry and neuro-pathology kept pace. Electro-physiology has not only given a better understanding of the function of cerebral cortex, but has also made us understand the various anatomical relationships. Curative and palliative treatments for many disabling conditions of the nervous system have driven away the pessimistic attitude of the medical profession towards neurological diseases. The static neurology has become a dynamic neurology. Much more is done for the patients, and more knowledge is gained in physiology and pathology. There is yet much more to be learned.

What is the position of Neurology in India at the present time ?

The position of neurology in India is in no way different to any of the other special fields. We have to admire all those people who in the midst of very heavy work of general practice tried to keep up the progress in these special fields. It is no wonder that they were not able to do any original and pioneering work in any particular field. They were not able to give any time at all even to think of clinical or experimental research. Even to satisfy the demand of the public in desperate need of their service, itself was not possible. There are only very few trained and qualified persons to treat the millions of suffering humanity in our country. Besides, how can any physician advance or develop himself in the knowledge of Neurology without a constant challenge to his diagnosis by surgical verification or pathological confirmation. There is no need to talk about the difficulties in our country for obtaining

*Presidential address at the I Annual Conference of the Neurological Society of India—delivered at Hyderabad, March 1952.

autopsies and adequate pathological examinations. Nowhere in India was there any neurology with a neuro-surgical team working till 1949. A few general surgeons occasionally attempted to undertake a few neurological operations as in those days of late 19th century in England or Europe and this has in no way helped Neurology.

Politically we have been emancipated and we are making rapid strides in every facet of the political and economic aspect of our country. But, what is the situation in the medical field? No doubht that when medicine gets involved in politics there will follow the inevitable disaster of degeneration and regression. Is it not tragic that some politicians are pushing aside scientific medicine in favour of some system of Medicine which is obsolete and antique? Hence, we have to be more alert and must try to progress and develop rapidly the scientific medicine, especially in all its various special fields and participate in research both clinically and experimentally. We must attain the standard and place in the medical world equal to any other country.

I have chosen the title of this address as "Neurology comes to life or the Precepts and Concepts of Neurology" with the firm belief that the inauguration of the Neurological Society of India is a definite step in our history of medicine. Neurology is taking on a new life with all its idealistic concepts of advancement in every direction. We recognise our ancient heritage, we realise where we had fallen short, we are determined to set right our faults and we should attain the standard equal, if not better than any other country in the world, and should start contributing to the progress of this science of medicine.

The aims and objects of this Neurological Society of India, as given in the memorandum of its Constitution are:—

(a) To maintain close association and co-operation among those devoting full time to Neurology and its associated branches.

(b) To maintain the highest standard in the ethics and practice of this speciality.

(c) To maintain this high standard, every endeavour shall be made to give adequate training to those who are properly qualified and intend to take up this speciality, and

(d) To promote and encourage original research in the clinical and experimental work pertaining to this speciality.

No doubt, here we have set forth an ideal which we will all be proud of, but how can we obtain our ideal? Sir William Osler said that there is only one way, and to be in this fraternity, the master word, the "open sesame" is work, for work must precede and work must follow. Co-operation among the workers, exchanging ideas and experiences, imbibing enthusiasm and zeal, and giving encouragement to persevere for greater and more work is the keynote to achieve success. Some men think in anatomical terms, others neurologically, psychologically, pathologically, physiologically, or even chemically. There should be no room in our cult for those who think only financially, socially, or not at all and such men should be kept out. It is enough for us to learn to think about the various problems in terms of some basic science. There are many problems we have to face which are more commonly seen in our country, and we have to solve them. As Dr. Archibald said: "To gather knowledge and to find out new knowledge is the noblest occupation of the physician, to apply that knowledge with sympathy born of understanding, to the relief of human suffering in his lovliest occupation".

I hope you will forgive me if I indulge in a few of my experience since I started working in the Christian Medical College Hospital, three years ago as a Neurologist and Neurosurgeon.

Before I joined the staff of this Institution, many of my colleagues had the belief that a Neurosurgeon may not have much to do in this country. Outstanding physicians of long experience in our country had not seen more than a few brain tumours diagnosed and confirmed during their life time. Physicians and Surgeons throughout the country expressed the view that nothing very much can be done in any way, for any of the neurological cases. There is also a common belief even among eminent members of our profession in India that many of the diseases and syndromes described in the text-books of Neurology are rarities in this country. Yet, what was the real situation? These is so much work and demand by the public that it is not possible to do all one wants to do for these unfortunate patients. The demand for hospital accommodation is so great that even very seriously ill patients have to wait for weeks before they can get admitted. During the first year, we had 143 major neurological operations of which 28 were for brain tumours; during the 2nd year, 185 operations of which 48 were craniotomies. There is further increase of work in the third year. So far 218 operations were done of which 57 were craniotomies. Almost all pathological varieties of brain tumours were encountered. The varieties of operations were also amazingly extensive. We have seen almost all the so-called rare diseases of the nervous system. One of the most striking features is the variation in the clinical picture of many of these neurological conditions. They need extensive investigation and study. I am not venturing to say that there are many new neurological diseases, but only that the conditions seen in our country have to be studied in detail in their clinical and pathological aspects, and the factors in the etiology also have to be evaluated. They all need extensive investigation. So far, all the diseases of the Nervous System described in the text-books are due to studies done in other countries, and I am sure we have great opportunity to give additional knowledge, both in the basic principles and in the clinical picture.

I am certain that the experience of other neurologists and neurosurgeons may be the same. What are all these facts amounting to? We have all varieties of Neurological conditions. Neurosurgery promises effective help to some of these diseases entities either curative or palliative. Adequate training in the surgical technique of the special branch is needed for workers in this field. Something more-the alluring promise of new discovery. I have pointed out before that neurology has already left its pupa stage of static condition and entered into a more dynamic stage. This is effected by confirming the diagnosis by pathological verification. This shall help in giving adequate treatment as well as in making further advances in this field. There is a challenge to every explorer. It is difficult to explain, but powerful. Powerful enough to draw full time workers to this speciality. Kipling called it an "Everlasting whisper. Something hidden; go and find it. Go and look behind the ranges, something lost behind the ranges. Lost and waiting for you. Go!"

The promise of discovery may be found in other special fields, as well as in the nervous system, but nowhere is the future so full of promise, the possibility of advance so unlimited, and the results of research so important, to mankind.

We must have many centres in India where concentrated neurology can be done. Such a centre, for its basic necessities, should have at least a Neurologist, a Neurosurgeon, and a Psychiatrist. Adequate assistance in Neuro-radiology, Neuropathology and Neuro-physiology will enhance efficiency and add facilities for not only clinical work but also clinical and experimental research.

In our country, we have learned to depend on the Government to establish and maintain all such centres or institutions. Even a casual observer can very easily see in all the democratic countries of the world, that most of such institutions, if not all, are developed and maintained by private bodies of the public. In those countries, the public is conscious and eager to foster such work so that there is sociological improvement and scientific advancement. Our Government is doing more than it's share in public health and medical work and it is not fair for us to expect much more to be done and sit back and wait. It will be

detrimental for the Government in its many other projects for the progress of our country. How can we get our public interested in our own health and scientific standard ? Most of the extensive scientific researches which have produced brilliant results for the alleviation of human suffering, are sponsored in other countries by private bodies especially business concerns. How many of our industrialists and financiers are interested in these lines ? Very few indeed. Is it due to the fault of the medical profession ? Probably we have to work harder and the results may encourage our people to support such work. The public will have to gain confidence in us. If we work persistently and with determination and keep up the ideal, no doubt we will be trusted by the public. Let us dedicate and consecrate our work in the highest ideals for the public and then we can find support from every where.

NEUROSYPHILIS

by P. KUTUMBIAH, Vellore.

Incidence

The incidence of neurosyphilis has been variously estimated by different groups of workers at different periods in different countries. One of the most interesting statistics is that published by Prof. Brussgaardof, Oslo. During the period of 20 years from 1891 to 1910 at the Riks Hospital, Oslo, 2181 cases of primary and secondary syphilis were dealt with and disposed off by Prof. Boeck without any treatment. During the later years 1925—1927, Prof. Brussgaard, the successor of Prof. Boeck set out to determine the fate of these individuals ranging from 3 to 40 years after the infection. In addition he was able to determine the cause of death of 164 others. The percentage of patients showing at that time of the re-examination evidence of neurosyphilis was 9.5. The standard citation in this field is from the group of 4154 syphilitic Austrian officers observed by Mattauscheck and Plicz (1920) for over a period of 22 years (1890—1912). Of these 132 (3.2 per cent) developed cerebro-spinal syphilis ; 198 (4.7 per cent) developed G.P.I. ; 113 (2.77 per cent) developed tabes dorsalis. The total number developing neurosyphilis was 443, i.e. 10.67 per cent. The figures for the incidence of neuro-syphilis among patients treated by modern methods of treatment are those given by Lees. Among 13,191 cases of syphilis observed by him in 12 years were 603 cases of neurosyphilis, a percentage of 4.5.

Statistics with regard to the incidence of the various types of neurosyphilis are very scarce in medical literature. Vad and Kulkarni (1929) reported 16 cases of tabes dorsalis from Bombay. The nationality of the patients is not noted except in one instance of a Chinese. Manohar (1935) in the course of a detailed examination of 882 autopsies found evidence of central nervous system infection in 19 per cent of the syphilitics. The most recent statistics are those published by Rajam. In the period between 1931 and 1940 the total number of syphilis admissions in the General Hospital, Madras, were 24,999. Of this total the number of neurosyphilis cases were 851 (3.4 per cent). Of this number, males were 778 and females 73. Out of this number, 23 were cases of G.P.I., i.e. 2.7 per cent ; 28 were cases of tabes dorsalis, i.e. 3.2 per cent ; 21 were cases of primary optic atrophy, i.e. 2.4 per cent.

Classification : Head and Fearnsides (1914) proposed the division of cases of syphilitic disease of the C.N.S. into those of syphilis meningo-vascularis and syphilis centralis. We propose to follow this classification.

Asymptomatic Neurosyphilis : We owe the conception of asymptomatic neurosyphilis to Ravaut. If spinal fluid abnormalities are present in the absence of clinical evidence of neurosyphilis, the condition is known as asymptomatic neurosyphilis. This form of neurosyphilis may be present in early syphilis, falling gradually until it reaches an average of 15 per cent among those in whom the disease is of more than four year's duration. It is a highly important phase of syphilis since it may be the fore-runner of clinical neurosyphilis. It is a type of neurosyphilis which responds comparatively better to treatment and is thus a preventer of clinical neurosyphilis. The importance of this condition lies in the fact that the spinal fluid abnormalities ante-date the appearance of obvious clinical damage in the nervous system by many years. The potential paretic or tabetic patient is recognisable within the first year, rather than 15 years later, when irreparable damage may already have been done. The latent neurosyphilitic can only be discovered by means of routine spinal fluid examination. If this condition is recognised before the development of clinical symptoms and signs, they may be prevented by appropriate treatment.

Patients with early or late asymptomatic neurosyphilis are divided into 3 groups by

Moore. Group I includes patients with minimal serological abnormalities—increased cell count and|or globulin content, with other tests negative ; Group II those with fluids of an intermediate type. There is a steady rise in the early cases of infection and a sharp drop between secondary and late syphilis ; Group III those with maximal change (the paretic formulae). The abnormalities of Group I fluids tend to disappear rapidly after treatment, this being more apparent in early than late syphilis ; Group II fluids tend to increase after two or more treatments. Only after 3 or more courses of treatment there is a marked fall in 'Group II' fluids. Treatment has no effect on Group II fluids especially in early syphilis. The character of treatment is of great importance in influencing the spinal fluid abnormalities in early but not in late syphilis. In early syphilis, asymptomatic neurosyphilis is almost three times as frequent when treatment is irregular as when it is carried out without rest periods of any kind ; and Group III fluids are increased by tenfold by allowing treatment to lapse. In late syphilis, on the contrary, no such effect is apparent. The difference in this respect between early and late syphilis is notably accounted for by the fact that in early syphilis immunity is in the process of establishment and may be suddenly altered or temporarily abolished by anti-syphilitic drugs. Undestroyed foci of organisms in the nervous system, to which penetration of the arsphenamines is admittedly difficult, may then multiply without any hinderance from the patient's own defence mechanism. In late syphilis, on the other hand, immunity is so firmly established that the spinal fluid (and probably the tissue of the nervous system) undergoes slight, if any, alteration from the shock of the treatment. Although persistently positive serologic tests in early syphilis are frequently indicative of asymptomatic neurosyphilis, negative tests do not mean freedom from the involvement of the nervous system.

What is the significance of spinal fluid abnormalities in patients who are clinically normal ? Do they tend spontaneously to disappear ? What is their ultimate prognosis ? Of the patients with normal fluids, only 3.6 per cent developed definite or questionable evidence of neurosyphilis in all instances, meningeal or meningovascular in type. Of the patient with Group II asymptomatic neurosyphilis, the analogous figures are 32 per cent ; of those in Group III (paretic formula fluids), 73 per cent and in this latter group 30 per cent of the total number of patients have already developed parenchymatous neurosyphilis. Group I data is incomplete.

The prognosis of asymptomatic neurosyphilis is equally gloomy whether discovered early or late in the course of infection unless the development of clinical neurosyphilis is prevented by appropriate intensification or modification of treatment. Spontaneous repression of Group II and Group III spinal fluids to normal is a rare phenomenon. There are no figures available as to the incidence of asymptomatic neurosyphilis among the Indian population. Rajam in an investigation of about 120 cases of early syphilis, both primary and secondary, found about 26 per cent of abnormal spinal fluids of exclusively of the first and second groups. Paretic curves occurring in early syphilis are by no means uncommon. Statistics of the occurrence of asymptomatic neurosyphilis in all stages of syphilitic infection in India is an urgent necessicity.

Syphilis Meningovascularis : The manifestations of meningovascular syphilis may, for clinical purposes, be grouped according to the situation of the principal lesions into (1) Meningitis, (2) Encephalitis, (3) Myelitis, (4) Hemiplegia, (5) Muscular atrophy, (6) Combined Sclerosis. Only some interesting lesions will be discussed below :

Meningitis : The nervous system is more often attacked in the opening phases of syphilis than was once imagined ; even during the first few weeks, at the preroseolar or roseolar stage, a meningeal reaction in the form of increased cells and protein may occur ; disclosed clinically by rachialgia, headache, insomnia, paresthesia, muscular weakness and altered reflexes or an unmistakable acute meningitis. Merritt and Moore (1935) in an important study

of the literature and 80 cases of their own found, (1) acute syphilitic meningitis is a rare complication of syphilis as it occurs in less than 5 per cent of cases. It usually occurs in young adults but it may be a complication of congenital syphilis. The meningeal symptoms usually develop within one year of the primary infection, but it may develop at any time after the initial lesion and it may be a complication of 'parenchymatous' neurosyphilis. The symptoms have an acute or subacute development. The meningitis is not caused or precipitated by arsenical therapy. (2) Clinically the cases divide themselves into three groups according to the presence of symptoms indicating involvement of the meninges in various regions of the brain as follows :—

1. *Acute syphilitic meningitis* : Cases with headache, nausea, and vomiting, choked discs and meningeal signs (stiffness of the neck and Kernig's sign), indicating involvement of the meningies in the posterior fossa interfering with the circulation of the cerebrospinal fluid and causing the development of an acute hydrocephalus.

2. *Acute vertical meningitis* : Cases with headache, nausea, vomiting, convulsions or mental symptoms indicating involvement of the meningies over the vertex of the brain.

3. *Acute basilar meningitis* : Cases with cranial nerve palsies indicating involvement of the meningies at the base of the brain. In our series of 97 cases of neurosyphilis there were 7 cases of meningitis with cell counts over 60 and the W.R. of the blood and the C.S.F. were positive with increase of albumin and globulin. There was one case of acute vertical meningitis and one case of acute syphilitic meningitis complicating congenital syphilis.

CASE 1.

H.M. aged 32 years. No history of genital sore at any time. About 6 months prior to admission he had a traumatic sore on the right thumb which did not heal for a long time and become septic. At that stage section of the sore showed granulamatous tissue with numerous plasma cells and lymphocytes. He attended to all sorts of v.d. patients on ships in the routine execution of his duties. Three days prior to admission had increasing and diffuse headache with insomnia. On the afternoon of the third day he felt very bad and all of a sudden lost his consciousness and developed convulsions. This fit lasted for about 15 to 20 minutes. Later he was incoherent for a time and slowly recovered. Physical examination revealed clear fluid ; Protein 25 mgms. per cent ; globulin ; cells 90 per cent c.m.m. lymphocytes. Blood W.R. . Was given a seven-day course of 2.4 million units of Penicillin ; and is now four years after apparently all right without any recurrence of fits.

CASE 2.

H.M. Eleven years of age. Admitted for treatment of headache, progressive blindness and epileptiform fits. The nose was depressed but there was no deformities of the teeth. Headache was paroxysmal, severe and generalised. No vomiting. Except for the total blindness in the right eye and light perception only in the left eye, there was no other lesion discernable, in the central nervous system. Fits were epileptiform and occurred at intervals. The blood W.R. + + ; C.S.F. W.R. + . X-ray skull : sutures separated ; sella turcica slightly widened ; Glucose tolerance test :— normal. Fundus examination showed papilloedema. Patient was put on anti-syphilitic treatment with remarkable improvement. Headaches much less in duration and severity and fits infrequent after treatment. This was a case of acute syphilitic meningitis with hydrocephalus complicating congenital syphilis.

Cerebello-pontine angle syndrome in syphilis : One of the interesting forms of syphilitic meningitis is the cerebello-pontine angle syndrome. Minz (1931) reported four cases of this syndrome in syphilis of the central nervous system. In all four cases there were noted involvement of the V, VII and VIII cranial nerves (which originate at this angle), cerebral manifestions of various degrees and general brain symptoms—headache, vomiting, etc. There also have been observed some remote effects as paresis of the abdomen, of the IX and X or of the IX and XII nerves. Besides cranial nerve involvement there were also symptoms due to pressure on the pyramids (increased reflexes) on the opposite side. As a sequel to intra-cranial pressure eye-symptoms developed. The women patients complained of amenorrhoea of 4 or 5 months duration. The W.R. of the blood and C.S.F. were positive in all cases. From

a review of the literature the author concludes that cerebello-pontine angle syndrome in syphilitic involvement of the C. N. S. is not rare. In some instances this symptom complex occurs in an almost pure form. In others it is combined with other symptoms. The great practical importance of its early recognition should be emphasised. This is easy with the employment of serologic methods. In case in which the diagnosis is difficult one should institute anti-syphilitic therapy under which the patient improves rapidly.

CASE 3.

H.M. aged 35. Admitted for headache, giddiness, tinitus and vomiting. Examination revealed loss of sensation over right half of the face V (sensory). VII, VIII and IX cranial nerves on the right side were affected. C.S.F. W.R. + + ; 3 cells; Proteins: 60 mgms. per cent; globulin + ; Chloride: 670 mgms. per cent; Sugar: 52 mgms. per cent. Langes: 0000123333. He had a course of anti-syphilitic treatment. On 3-1-46 he reported for a check-up. He felt much better. Giddiness and vomiting had cleared up. Anaesthesia over the right half of the face still present. His hearing was much better. Still has some ringing in the ears. VII th nerve paresis had cleared up. Blood W.R. was negative. C.S.F. protein: 20 mgms. per cent; globulin: nil. On the other hand, Marburg, Neusberger, Winklemann and Eckel (1933) report cases presenting angle syndromes with a long fluctuating course, which were thought to be instances of multiple sclerosis but were later proved by operation or autopsy to be cases of angle tumour. We had one such case.

CASE 4.

H.M. Aged 46. Admitted with inability to walk and dimness of vision of five years duration. He gave venereal history & history of headache also. Clinical examination revealed nystagmus, altered speech, intention tremors and signs of pyramidal involvement of both sides. A diagnosis of cerebro-spinal syphilis-disseminated sclerosis type was made. Blood W.R. negative; C.S.F. slightly under tension; cells: normal; W.R. + ; Protein: 35 mgms. per cent; globulin: present; Langes: 3221110000. R.E.V. 6|10; L.E.V. 6|16. Optic neuritis of both eyes. Autopsy revealed a tumour in the cerebello-pontine angle.

Encephalitis: It is one of the rarer forms of meningo-vascular syphilis. There was one case of encephalitis in this series. This form is characterised by gross cerebral symptoms, consisting of dullness, loss of memory amounting in some cases to acute dementia. It may appear at any time from a few months to twenty years or more after infection. The symptoms depend on inflammatory changes in the meninges, with endarteritis of the vessels of the brain followed by a varying amount of secondary atrophy and sclerosis. In young men this form of syphilis occasionally evokes a condition distinguishable at first sight from dementia precox. The inhibition of mental processes, want of emotional expression and negativism that characterises the disease may be present, with increased cell count of the C.S.F. and may show remarkable improvement under anti-syphilitic treatment. This should not be confused with the haemorrhagic encephalopathy of arsenical reaction. The condition need not be necessarily acute but may run a sub-acute course.

Myelitis: Adams and Merritt (1944) observe that syphilis of the spinal cord is a clinical rarity. There were only 31 cases of spinal syphilis among 2231 syphilitic patients at the Boston City Hospital. There were 54 cases of meningeal and vascular and vascular syphilis of the spinal cord in our series of 97 cases. This is in marked contrast with the incidence of this lesion in western countries. Whereas in their series spinal cord cases from less than one per cent of total syphilitic patients, in our series they form 55 per cent. In Rajam's series, they formed 62 per cent. They classify meningo-vascular syphilis of the spinal cord into groups. (1) Syphilitic Meningomyelitis; (2) Spinal vascular syphilis; (3) Syphilitic spinal pachy-meningitis, including Gumma of the spinal cord and Syphilitic hypertrophic pachymeningitis; (4) Syphilitic Polyneuritis. In our series there were 37 cases of meningomyelitis; 16 cases of spinal vascular disease (transverse myelitis); and one case of hypertrophic pachymeningitis. Special mention must be made of a form of meningomyelitis called Erb's paralysis. King in his monograph on 'Syphilis of the spinal cord' summarises this syndrome as follows: This syndrome is not common. It occurs five times more frequently in males and is sometimes a late

complication of syphilis. Clinically, the patient presents a picture of a slowly developing weakness, fatigue, and stiffness of the legs with little or no pain. Bladder disturbances are frequent. Examination reveals spastic paraplegia with little or no sensory loss. At first there is a paraplegia in extention but this later may occasionally become paraplegia in flexion. There is no block in the sub-arachnoid space. The condition has to be differentiated from a cord tumour, multiple sclerosis, other forms of cord involvement due to syphilis and changes due to pernicious anaemia. The blood and spinal fluid W.R. are frequently positive but may be negative. The process is very resistant to treatment and frequently progresses in spite of it.

Syphilitic Spinal Pachymeningitis: There was one case of this type in this series. The earliest symptoms included pains and paresthesias in the arms and hands followed by atrophic paralysis of hand and arm muscles, segmentary sensory loss and spastic paraplegia. The C.S.F. showed Froin's syndrome. The only clinical distinction between gumma of the dura and hypertrophic pachymeningitis appears to be a slower evolution and greater tendency to segmental muscle atrophy and sensory loss in the latter.

CASE 5.

Hindu male aged 30 years. Admitted for pain and stiffness of the neck and paresis of all the limbs. No definite history of infection, lost two children soon after birth. Rigidity of the neck, both upper extremities paralysed and the lower extremities paretic, muscles of the forearms wasted, especially the extensors, slight wrist drop, the intrinsic muscles of the hand wasted, ulnar nerves on both sides not thickened. B.J. and T.J. absent, S.J. sluggish, K.J. brisk, ankle clonus present on both sides; pupils dilated, equal on both sides, react sluggishly to light; C.S.F. xanthochromic, 7 cells per c.m.m. albumin 600 mgms. per cent; globulin: normal amount; Sugar 94.3 mgm. per cent; Chlorides 66.8 mgms. per cent; W.R. negative, Blood W.R. negative, X-ray of cervico dorsal spine: no caries of the spine; no cervical ribs.

Muscular Atrophy: The nature of muscular atrophy in syphilitic persons has been the subject of much discussion: but Leri and others have amply demonstrated the existence of a progressive amyotrophy due to meningo-vascular changes. Martin (1925) in his paper on Amyotrophic Meningomyelitis (spinal progressive muscular atrophy of syphilitic origin) critically reviews the literature on the subject and discusses its symptomatology and pathogenesis. From an analysis of 60 cases the main clinical features are given as follows: atrophy of the atonic type beginning (a) in the small muscles of the hands, (b) in the shoulder muscle or (c) in the muscles on the outer side of the leg; it may be unilateral or bilateral; its onset is not infrequently preceded by pain; bulbar symptoms are uncommon; the vibration sense may be impaired; there may be difficulties of sphincter control. Argyll Robertson pupils were only present in 28.5 per cent of the cases. There was no sure case in his series in which it was negative in the C.S.F. before any anti-syphilitic treatment had been given. The following two cases illustrates the above observations:

CASE 4.

H.M. 30 years of age: Admitted for treatment of wasting and deformities of hands and feet of two years duration. Onset gradual; started in the muscles of hand and feet and slowly had spread up into fore-arm and leg. No subjective sensory phenomena noticed. He has observed twitching in the muscles. No history of trauma, syphilis, hereditary or familial incidence. Below the wrists and knees voluntary movements are greatly diminished. The thenar, hypothenar, lumbricals, and interossei muscles are completely wasted. There is a tendency to Griffin's paw symmetrically present in both hands. Similar wasting of muscles of feet with gross contractures seen. Walks on toes which are extended on the metatarso-phalangeal joints and flexed at the interphalangeal joints, and also there is medial rotation of the feet. Deep reflexes all lost. Plantars not elicited, cremastric and abdominals present and equal on both sides. Sphincters normally acting. Sensory system Spino-thalamic and posterior column sensations are normal throughout the body including the wasted areas. Cranial nerves normal. No nystagmus; speech normal no signs pointing to the affection of the cerebellum. Blood W.R. negative. C.S.F. clear; normal pressure; proteins 25 mgms. per cent; globulin negative; cells 3 per c.m.m. W.R. — —; Lange's curve: 0011110000. Muscles: React both to Galvanic as well as Faradic stimulation. Fibrillations are seen in the fore-arm and leg muscles. A diagnosis of Amyotrophic Meningo-Vascular syphilis was made. The resemblance to Peroneal muscular atrophy is very close.

CASE 5.

Hindu, female, 14 years of age: Admitted for inability to use her limbs and impairment of speech. Duration three months and slowly progressive. Headache not prominent. No stigmata of congenital syphilis. Clinical examination showed it to be a case of amyotrophic lateral sclerosis with bulbar symptoms, the nerves involved being bilateral XII, and later Vagus as evidenced by bilateral abductor paralysis resulting in dyspoea and stridor for which a tracheotomy was done. Fibrillation present in muscles. Blood W.R. — —, C.S.F. protein 15 mgms. per cent; globulin nil; and W.R. — —. The patient was given antisyphilitic treatment and began to improve rapidly in weight, strength and colour and later on was able to move about with some support. She was under observation for six months and finally was discharged at request with the tracheotomy tube on. A diagnosis of Amyotrophic (parenchymatous) form of congenital neuro-syphilis was made.

Syphilis Centralis: This group comprises of G.P.I. Tabes Dorsalis; Muscular Atrophy; Optic Atrophy; Gastric Crises; and Epileptic Manifestations. The existance of these types of neurosyphilis in oriental countries is very much doubted and two reasons have been given for its supposed rarity:

(1) The prevalence of malaria in oriental countries is thought to be antagonistic to the prevalence of these forms of neurosyphilis. Induced malaria is now the recognised treatment of G.P.I., tabes dorsalis and to a limited extent meningovascular syphilis. We have not enough evidence to dogmatise as to whether the development of tabes dorsalis and G.P.I. are prevented in a malarial country. The available evidence on the other hand, points to this assumption being not correct. Vizagapatam District is notoriously malarial and the cases of this type included in this series are all from this district. We have already quoted enough evidence to show that cases of G.P.I. and Tabes Dorsalis occur in India. For European countries the figures given by Mattuscheck and Plicz (1920) are tabes dorsalis. 2.77 per cent; and for the G.P.I. 4.7 per cent. The figures for India are tabes dorsalis including Optic Atrophy 5.6 per cent; and G.P.I. 2.7 per cent. Even these figures do not in any manner indicate its true incidence. A large percentage of mental disease due to syphilis find their place in mental hospitals. A fair number of tabes dorsalis cases are to be found seeking relief for progressive blindness in an eye hospital.

(2) The neurotropic strain of treponema pallidum is not common in oriental countries. Before this assumption is taken for granted the existence of a neurotropic strain of Treponema pallidum should be proved. Most authorities doubt the existence of the so-called neurotropic strain. Unless the existence of the neurotropic strain is proved beyond all controversy it is futile to assume that supposed rarity of neurosyphilis is due to the rarity of that hypothetical strain in oriental countries.

There were four cases of tabes dorsalis in this series. Definite history of syphilis was available only in one case. The age of the patients varied from 27 to 40 years. In the case of the patient aged 27 years a history of syphilis 12 years back was definitely given. In all the four casss there was marked ataxy; the knee jerks and the ankle jerks were lost; there was loss of vibratory sense and impairment of joint sense, deep muscle sense and tendon sense in the lower extremities; loss of sensation to light touch over the nose and round the mouth and dulling of sensation to pin prick over the same area; a band of anaesthesia across the chest about the level of the nipple. The true Argyl Robertson pupils with fixity and miosis were not noted. But the reaction of the pupils was sluggish to light in two cases. There was no optic atrophy in any case. The C.S.F. was normal with regard to the protein, globulin and cell content in three cases and in one case the protein was 50 mgms. per cent; globulin was present and cells were 24 per cmm. The W.R. of the blood and the C.S.F. was strongly positive in all the four cases.

CASE 6.

Hindu. Male. Age 27 years. Admitted for inability to walk for the past two years. Gave a history of syphilis 12 years back. Had a course of N.A.B. injections. Four years ago noticed weakness and pain in the joints. Romberg's sign positive. Marked in-coordination present in the lower extremities. No tremors or twitchings of the muscles. Muscular power good. Deep reflexes lost in the lower extremities. Abdominal reflexes lost in both upper quadrants and brisk in the lower ones. Cremastric reflex lost on both sides. Plantar reflex not elicited. Sphincters normal; sensation: light touch lost over the toes of the right foot and the dorsum of both feet; a band of anaesthesia at the level of the nipple across the chest, joint sense lost over the lower extremities. Cranial nerves normal. C.S.F. protein 50 mgm. per cent, globulin nil, Chlorides 702.5 mgms. per cent, sugar 54 mgms. per cent, Langes curve: 000233210. Cells 24 per cmm. W.R. + + ; Blood W.R. + + ; Fundi normal. Test meal: nothing abnormal. There were two cases of G.P.I. in this series one was aged 30 and the other 47. In both cases the mental condition of the patients was one of apathy and depression. In both there was history of syphilis but the exact date of infection was not ascertainable. C.S.F. showed characteristic changes. Protein was increased to 35 mgms. per cent in one case and to 40 mgms. per cent in the other. Globulin was present in both cases. The Lange's curve reading in one case was 155532000 and in the other 4543332110. W.R. of the blood and of the C.S.F. were strongly positive in both cases.

CASE 7.

H.M. 47 years of age. Tremors of the upper extremities. History of venereal disease years back. Addicted to drink. Well nourished individual. Oedema of the ankles and the dorsum of the feet. Tremors of the tongue present. Speech tremulous and hesitating. Fine tremors of the hand and fingers, worse on exertion. Slight paresis of the muscles of the left side of the face. Pupils unequal, react to light. Sphincters of both rectum and bladder involved. Mental condition: cerebration slow, speech occasionally incoherent, misses one or two letters of his name in writing. Behaviour queer-apathetic in the day and boisterous at times in the nights. Memory poor. C.S.F. protein 35 mgms. per cent, globulin nil. Langes curve 15553200. W.R. + + ; Blood W.R. + + ; Patient rapidly became demented.

Optic Atrophy: Primary optic atrophy occurs so frequently as a complication of Tabes dorsalis that most observers have looked upon it as a manifestation of 'parasyphilis'. Thus Nonne goes so far as to say that primary optic atrophy is always a sign that the case is one of tabes dorsalis, even if the knee jerks are present and he adds that it may exist alone for a long period before the appearance of any other signs. Thus it would seem that syphilis can produce degeneration of the optic nerve exactly analogous to the destruction of the posterior columns of the spinal cord in tabes dorsalis. Moore and Woods (1940) in a critical review of the pathogenisis of syphilitic optic atrophy discuss the recent theories put forward to account for it. They conclude that it seems probable that the pathogenesis of syphilitic optic atrophy, so often (perhaps always) associated with tabes dorsalis, is identical with that of tabes dorsalis itself. This in turn is as yet unknown, though recent studies, especially in the field of nutrition, offer hope of its elucidation. There was no case of primary optic atrophy in this series but Rajam has reported 21 cases of primary optic atrophy in his series of 851 cases of neurosyphilis.

Conclusions

1. Neurosyphilis is not so rare in India as it is commonly made out in current medical literature.

2. Meningo-vascular syphilis forms the great bulk of the cases of neurosyphilis seen in India.

3. Spinal syphilis is extremely common, forming about 55%—62% cases of neurosyphilis.

4. Cases of tabes dorsalis and G.P.I. occur but they are not so common as in the western countries.

Summary

1. A brief summary of the incidence of neurosyphilis in India and other European countries is presented.

2. An analysis of 97 cases of neurosyphilis in Indians classifying them under various types with illustrative cases is given.

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ELECTRO-ENCEPHALOGRAPHY IN INTRACRANIAL LESIONS

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Electro-Encephalography is in use since 1929, in Germany and since 1935 in England, United States of America and other Western Countries, to help in the diagnosis and in determining the prognosis of cerebral lesions. [1]This technic has passed the stage of pure research and has become a dependable and an almost indispensable adjunct to the diagnostic armamentarium of the Neurologists, Neurosurgeons and Neuropsychiatrists. Berger has described the three types of waves Alpha, Beta and Delta. Another later addition is Theta waves, by Walter. The shift from Delta rhythm to Alpha, from birth to adolescence is the process of normal evolution.

This paper is particularly devoted to the pathological variations in cases of increased intracranial tensions and tumours. [6]At the instance of F. L. Golla, Grey Walter and his associates undertook the localisation of cerebral focal pathology within a year after Adrian confirmed Berger's work and predicted a great future for this aspect of study.

Berger had reported, during his life time the localisation of at least three intracranial neoplasms which have been verified at the operation.

Studies on the electrical potential discharges have been made under different conditions to explain the variations in the record when neoplasms are present on or near the cerebral cortex. [6]Workers have done electroencephalography from the skull and the area of the cortex, just underneath that point. No particular variation was found except that the recording from the skull was of lower amplitude due, probably, to resistances offered by the skin and the underlying tissues. Tonnies has pointed out that the "spread" of the potentials is increased by the complex resistance of net-work of the dura, skull and scalp.

Ether anaesthesia was found to produce 3 to 4 per second waves of 100 uv. Increase in the intracranial pressure was also found to produce waves similar to that produced by ether anaesthesia but of a lower amplitude. If intravenous injection of hypertonic saline was given, in such cases, the alpha rhythm reappeared. In cases of intracranial tumors, in which the cerebral cortex was affected by invasion of proximity and where the pressure was still moderate or had been reduced, slow potentials (of the order of 10—20 uv in size and occurring at the rate of 2 to 3 per second) have been led off from the skull immediately over the place where the tumour was subsequently found at the operation or at autopsy: If two leads were placed on the site of the neoplasm, a bipolar recording from these leads produced only a suppression of all electrical activity. Such waves were not present either in acoustic nerve tumors or cerebellar tumors indicating that the efficacy of this technic has not yet been perfected for localisation of these basal lesions. For the same reason Aneurysms which mostly involve circle of Willis and from which leakage is possible, at the base, are difficult to diagnose with this technic. This also proves that slow waves appear only when the tumor affects cerebral convexity (at or near the surface). It cannot be definitely stated that the "slow" waves are due to tissue damage.

Foester and Altenberger have shown that a Glioma was electrically much less active by itself than the cerebral cortex. Similarity of the tumor wave to "anaesthesia" and "pressure" waves suggests that the potentials are generated by the cortical tissue in some phase of functional disturbance.

Abnormalities of Alpha rhythm:

[7]Alpha rhythm is suppressed during function, e.g., intense concentration on a problem. This is the most striking difference between the alpha and the delta rhythms as the latter is unaffected by any such functions. The alpha rhythm of 8 to 13 per second itself can vary within limits and symmetry to indicate a pathological condition of the cerebrum. A difference of over 50% in amplitude between the two hemispheres can be considered pathological. Extension of alpha beyond the parieto-occipital region can also be pathological. Also a restriction to less than 50% of the legitimate area of appearance indicate abnormality. A synchronous alpha is also indicative of brain pathology. Alpha consistently showing a difference of 0.5 c|s between the two hemispheres, frequent unilateral bursts, and total persistance are all indicative of abnormality.

The Delta waves:

0.5 to 3.5 c|s waves are the Delta waves. Larger the amplitude, the greater the area involved. A lower frequency indicates a more acute and|or rapidly progressive lesion. "Sub-Delta" waves which are 0.2 to 1 c|s are characteristic of abscess. These resemble waves of deep sleep or infancy; they may also suggest raised intracranial pressure or toxic conditions. If focal, this shows evidence of focal pathology in all cases, but the lesion is often posterior to the focus; if it is polyrhythmic and persistant, then it indicates cortical involvement. It is worth noting, also, that in addition to delta activity from the neighbourhood of the region of a cortical lesion, more rhythmic delta waves are often seen from remote regions. This type of disturbance often resembles the normal spontaneous rhythm in being responsive to physiological stimulation and seems to follow normal anatomic pathways. This is called "Quasi Physiological" rhythm.

Theta activity:

Theta activity, as described by Walter, was once called "slow alpha rhythm". Records containing "Theta" rhythm are much more difficult to interpret than records containing "Deltas". It is almost impossible to be certain of any pathology when "Theta" rhythm is encountered in children below 12 years, if only a single record is taken. The French workers have devoted much attention to what they call the "Anatomo-clinical" evaluation of electroencephalography and all workers would agree that changes in the picture from day to day or from week to week are of greater importance, both from distinguishing the organic from the functional and epileptic disorders and in helping to establish the differential diagnosis as between static, recovering and progressive lesions. "Theta" activity is definitely correlated with a subcortical disturbance and particularly with the perturbation of relations between cortex and basal structures.

The relation between the amplitude and the extent of "Delta" and "Theta" activity both topographically and temporally, can be used to infer the extent and the rapidity of the progress or decline of a pathological process. For example, cases in which the first abnormal feature was diffused but lateralised "Theta" discharges which were followed after weeks or months by more focal "Delta" rhythm on the same side, have been found P.M. to have a tumor arising near the 3rd ventricle and growing outwards. [8]An inverse relationship in order of time suggests a lesion arising superficially invading the deeper structures.

"Theta" waves are of 4 to 7 c|s waves. In psychopathy they have an amplitude over 20 uv. Their common frequency is between 5 to 6 c|s. They have simple forms. When they are persistent it is indicative of disease of the deeper structures. In juveniles and in psychopathy they appear in paroxysms and are responsive to physical and emotional stimulii. If they are focal, it is suggestive of underlying deep lesions and, if general, involvement of Thalamo-hypothalamic circuits.

Spikes:

The other pathological wave are "spikey" waves. These are signs of irritation and are localised in case of trauma, particularly birth trauma. They arise a little distance away from the apparently traumatised area and indicate the site for surgery, even better than do the obvious signs.

The other pathological wave is " spike & wave " which is different from " wave & spike " of Petit mal ; the significance of the same is not clear.

Electroencephalography has made rapid progress in many ways. The technical progress has been achieved with the use of the multi-channel instrument and also with the advent of the wave analyser which is able to give precisely the proportion of the combination of waves of different amplitude and frequency, with case, and which otherwise would have been a very laborious and impossible task.

The combination of recording blood pressure, electro-cardiogram, respiration, electro-myogram and skin resistance, all in a simultaneous recording is another advance. In the technic of localising, apart from the scalp leads, nasopharyngeal and tympanic leads are useful in diagnosing basal lesions. It is important when doing localisation that the inter-electrodal distance is kept constant when comparing homologous areas. It is also important that the inter-electrodal distance is kept within the limit of 4 cms. and 6 cms. for narrow localisations.

[5]An expanding lesion or cerebral trauma destroys nerve cells at or around it and as these die they apparently burst into low electrical oscillations. They do not confine their affects to any particular pathways or system. Electrical changes are often also the result of wide spread destruction, cerebral oedema or raised pressure in the ventricles. Electroencephalography tends to be more abnormal in inflamatory than in degenerative cases. [3]Unilateral vascular lesions embolic or thrombotic cause electroencephalographic abnormality on the same side for a few days or weeks and later tend to return to normal. This is more true in younger patients. In patients over forty years it takes a longer time to return to normality. Even when the destruction of the brain cells was extensive, resulting from large infarcts, at times early electric " recovery " took place, apparently because the total number of sick cells was reduced. The infarct then acted as good electric conductor of the potential generated by the adjascent neurons. Hence if the prime generator was intact and almost normal electroencephalographic pattern resulted often within two or three weeks. In some case the rhythm does not return to normal for a long time, probably, due either to the fact that the lesion was not stabilised or that the generators of " alpha " rhythm were destroyed.

Lesions in the occipital region often first, or concomitantly, give rise to slow waves in the ipsilateral frontal region. Only later does the more posterior, slow activity become dominant. This observation assists in the differential diagnosis of neoplastic lesions in the posterior fossa, since the later condition is not infrequently associated with contralateral frontal and occipital slow activity.

Suppression of rhythm and silent areas :

Another pathological condition is the suppression of " alpha " or the presence of " silent areas " in the brain. [5]In head injuries the degree of suppression of the normal " alpha " frequencies are often inversely related to the degree of prominence of abnormal slow waves and when these have subsided, return of the normal frequency constitutes the last stage of recovery of the electroencephalographic records from the injured brain. A " silent area " persisting for six months and more after head injury is due to complete loss of an extensive area of the cortex.

[4]It has been observed that lesions in the posterior parietal region causes complete suppression of alpha rhythm in the whole hemisphere. But occipital, temporal and frontal areas produce the suppression only locally. So inhibition of normal frequencies causing large silent areas do not indicate extensive brain damage. Porencephalic cyst and subdural haematoma cause the post-parietal cortex which is supposed " silent areas " locally if they are not near to be the point of origin of alpha.

The degree of suppression of " alpha " rhythm depends partly on the severity and the extent and partly on the proximity to

the point of origin of alpha rhythm. Interruption of the physiological continuity of the cortex causes the interruption of propagation of the normal " alpha " rhythm. This is in accordance with the view that the spread of the potential changes is by the neurons and not by simple electrical conduction. The abnormal suppression of cortical electrical activity as a result of trauma is a phenomenon distinct from the production of abnormally slow waves although they often co-exist. Suppression of abnormal waves in contrast to that of normal waves, occurs only at the site of complete destruction, displacement or inactivity of the damaged brain.

In severe concussion slow waves often appear. As they disappear there is a stage of suppression of all normal waves is seen without the appearance of slow waves, it indicates minor injury.

In subdural haematoma suppression is seen on the haematoma and slow waves around it. Meningioma growing slowly may not give rise to any pathological waves but if present in the posterior parietal cortex may give rise to general suppression of " alpha " rhythm on that side of the cortex. Glioma or other tumors do not give rise to a " silent area " unless a very large intracranial cyst is present.

In aneurysms the finding is the suppression of normal frequencies in the same way as in tumors but as they are so often found in the parietal or the parieto-occipital regions, suppression of normal waves is relatively common, and as they do not cause rapid progressive damage to the brain tissue, the suppression of normal frequencies is accompanied by little or no abnormally slow wave activity.

Porencephaly causes suppression of activity or normal frequencies with surrounding slow waves. Abscess produces high voltage slow activity.

In embolic phenomena it is possible to have a " silent area " in the hemisphere. Subsequent appearance of slow activity may indicate recovery. The suppression is due to destruction of brain tissue, but it also results from temporary suspension of activity as a result of concussion. In the former it is irreversible and in the latter full recovery is possible. If a portion of the damaged brain causing slow abnormal activity is removed, an area of suppression of alpha is left behind.

In a recent publication, Aird and his associates who worked on 329 patients, had studied electroencephalograph and co-related it with pneumoencephalograph. They have been able to localise to the extent of 96% in Gliomas and slightly less in other focal lesions. On an average they have localised successfully in over 80% of cases. According to them it is possible to localise a lesion by the difference seen in homologous potentials alone. This has been reasonably accurate except that when found alone, without differences in the form and frequency of homologous tracings, one is unable to decide which side is involved. In such instances, it is necessary to correlate the electroencephalographic findings with the clinical data in order to determine the question of lateralisation. When used in conjunction with such findings, however, it is of considerable value in determining whether the lesion under consideration is of " inactive " (low voltage area on the side of the lesion) or " irritative " (high voltage area on the side of the lesion) character. A demonstration of high as opposed to low voltage areas by electroencephalography has been of particular value in brain tumor suspects and Jacksonian convulsive states, while the finding of an " inactive " focus on the side of the lesion is strongly indicative of a degenerative process (except for angiomas and subdural abscess) rather than a tumor. No Gliomas and but a few meningeomas in their series have been associated with an " inactive " focus. Decision as to further study and treatment, may be influenced by such findings, as a patient is much more likely to be a candidate for surgery, if the suspected area is associated with a high voltage area. The incidence of cerebral pathology was found to be low when no electroencephalographic focus is found. In their series homologous potential inequali-

iies occurred in 100% of focal electroencephalography. A synchronism of hemispheric discharges occured in 52% of focal electroencephalograph. So inequalities of homologous potentials and synchronism of hemispheric discharges are primarily of localising value but alone do not indicate the side of the lesion. Differences of wave form and frequency, on the other hand are chiefly of value in showing the side of the lesion and verifying a focal diagnosis.

Scope of Electroencephalograph :

What further information can the electroencephalograph give about the nature of a lesion of the hemisphere, apart from its approximate site ? It seems that it may give evidence upon six of its qualities.

(i) *Extent* : Electroencephalography merely records the electrical activity of damaged brain tissue, but the area of this can be mapped out within the limitations of a technic which includes recording through the thickness of the scalp and cranium. The sharpness of the definition of abnormality may also be judged.

(ii) *Severity* : The degree of damage which is taking place at the time of recording may be judged by the period, voltage and persistance of the abnormal waves.

Absolute destruction may be recognised by a " silent area " in which there is absence of reduction of electrical activity ; but it can be definitely confirmed only by correlating the electroencephalography and radiological changes.

(iii) *The Depth* : The depth of the lesion in the hemisphere may be surmised, at least, to a consideration of whether it lies in the cortex, the white-matter or the basal ganglia, by relating the clinical observations, to the voltage and period of abnormal waves and to the extent of their diffusion over the scalp. Three per second Delta on one side with mixed seven per second wave bilaterally indicates deep lesion.

(iv) *Secondary changes* : Long standing lesions give rise to characteristic changes in the electroencephalograph which have many features by which they can be distinguished from immediate and direct effects of brain damage. These secondary changes often produced are associated with epilepsy.

(v) *Associated changes* : Physical disorders which may be associated with cerebral lesion give wide spread changes in the electroencephalograph which often are symmetrical and fairly easily recognised. Such changes are seen with high intracranial pressure, changes in consciousness or in toxic states. For their interpretation, knowledge of the clinical picture is essential.

If in a patient with a history of hemiplegia of slow onset indicative of an expanding neoplasm, the electroencephalograph is found to be normal, it indicates, whatever the neoplasm is, it has not yet caused damage to the brain tissue and assures that it could be only benign lesion and not a glioma. It is clear that in each of these instances the electroencephalograph can only be interpreted reasonably if the interpreter has full knowledge of other aspects of the clinical problem. That this is essential is made evident by the fact that there is usually more than one possible explanation for any isolated phenomenon, which may occur in the electroencephalograph. Even a sequence of related phenomena in the electroencephalograph may be difficult to interpret if considered alone.

All these lead on to the conclusion that the future progress in applied electroencephalography, will depend more upon intimate knowledge of the functional anatomy of the nervous system and rather less upon technical contrivances. It has been proved beyond doubt that electroencephalography alone gives a more correct diagnosis than neurological examinations alone or ventriculography alone, with reference to the localisation of intracranial lesions. It is also less annoying to the patient and requires less co-operation but it is the whole picture that is important and these methods are complimentary and confirmative.

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MALONONITRILE IN THE TREATMENT OF MENTAL ILLNESS

by N. S. VAHIA and V. P. MONDKAR, Bombay.

Introduction

At present the value of amino acids in the treatment of mental illness is not very clear. Hyden and Hartelius found that the nerve cells subjected to heavy stimulation showed decrease in nucleo proteins and that the nitrile compounds administered to experimental animals hastened the regeneration of nucleo proteins. They considered that there was reduction in cellular protein in the specimen of the prefrontal cortex of psychotic patients removed during leucotomy, and believed that nitrile compounds had beneficial effects in psychotics, which was often permanent. On the other hand, MacKinnon and others, while publishing the results of nine psychotic patients treated with Malononitrile reported unpleasant toxic reactions, and concluded that Malononitrile had almost no therapeutic value. According to Hartelius, MacKinnon's results were due to faulty technique. He again confirmed his original impression that the drug has definite therapeutic activity. Mayer and Mayer criticised the work of Hyden and Hartelius on theoretical grounds. They believed that the nerve cell abnormalities, which Hyden and Hartelius considered to be characteristic of mental disease, were in their opinion, probably artefects and they described similar cell abnormalities in the mentally normal patients and normal animals. Delay and other found Malononitrile very toxic and therefore used Dinitrile Succinate, and reported good results in depression. Harris used dinitrile in 30 cases of depression, and found that there was no clear evidence of its therapeutic value.

In view of the difference of opinion regarding the value of Malononitrile, an attempt was made at the K.E.M. Hospital, Bombay, to study the value of this amino acid. By kind cooperation of Prof. Hyden, it was possible to get a trial sample of Malononitrile, prepared in their laboratory.

Twelve cases, selected from the outpatient department were admitted for this purpose, out of which, two discontinued the treatment. The results of the remaining ten cases, that took the whole course of treatment have been described in the chart. The age of patients varied from 20 to 44 years. There were six males and four female cases. The diagnosis in these ten cases were, schizophrenia (catatonic type) three cases, schizophrenia (paranoid type) two cases, involutional depression one case, reactive depression two cases, and Hypochondriasis two cases. The duration of illness varied from two months to three years. 2 c.c. of 5% Malononitrile solution, diluted with 38 c.c. normal saline, was injected very slowly. 45 minutes were taken in giving the injection. The injections were given on alternate days. Ten injections were given in every case. Sodium Thiosulphate was given in nine cases, one hour after the administration of Malononitrile.

One patient, a case of reactive depression of three years duration, that had not responded to electro convulsive treatment showed satisfactory response, that had been maintained till the time of publication. Four other patients showed fairly good immediate response, but relapsed after some time.

The following side effects were noted. Vomiting in eight cases, flushing of the face in seven cases, palpitation in four cases, twitching and tremors in two cases, headache in one case, giddiness in one case, sleeplessness in one case. Sodium Thiosulphate injection was used as an antidote, in every case, with immediate relief from these side effects.

Discussion

The number of cases in this series is small, because a trial sample of about 50 c.c. of 5% solution was available to us. A large variety of mental illness was given

CHART No. 1

MELONONITRILE IN THE TREATMENT OF MENTAL DISEASES

Date	22—3—52	20—3—52	6—6—52	26—6—52	24—7—52	22—3—52	30—8—52
Name	N. M.	H. D.	D. P.	K. V. K.	R. R.	P. D.	S. M.
Age	26	44	20	26	27	22	30
Sex	Male	Female	Male	Male	female	Male	female
Diagnosis	Hypochondriasis	Involutional psychosis	Schizophrenia catatonic	Schizophrenia Paranoid	Schizophrenia catatonic	Schizophrenia paranoid	Acute Depression
Duration	6 mths.	1 year	3 months	3 years	2 months	2 years	2 years
Previous treatment	20 insulin subshock	Nil	Nil	Psychotheraphy	Nil	Nil	Nil
Dose of Malononitrile each time	2 c. c.	2 c. c.	2 c. c.	2 c. c. 38 normal saline	2 c. c. 38 normal saline	2 c. c. 38 normal saline	2 c. c.
No. of injections	10	10	10	10	10	10	10
Side effects	Nil	Vomitting palpitation twitching numbness	redness of face. palpitation, vomiting	redness of face, palpitation, severe vomitting, headache	palpitation redness of face, vomiting	redness of face, palpitation, restlessness	vomitting
Sodium Thiasal phate administration	Nil	yes	yes	yes	yes	yes	yes
Response	45% imp.	40% imp.	became worse	80% imp.	no imp.	no imp.	no imp.
Follow up	recurrence of the symptoms 2 mths. after discharge	symptoms reoccurred after 6 days	...	symptom s reoccurred after 8 days	...	...	...
Other treatment	...	10 E. C. T. given	5 E. C. T. given	...	Insulin subshock	18 E. C. T.	40 insulin subshock 10 comas 20 E. C. T.
Remarks	...	60% imp. maintained	admitted in a mental Hospital	Condition same as it was before admission	admitted in a mental hospital	maintained for 4 months	Admitted in a mental hospital

the treatment, as this was a preliminary investigatian, to find out in what kind of psychological disturbance the drug would be useful. It was decided that a bigger quantity of the drug might be asked for or prepared here, for use in that type of illness, in which it was found to be of some benefit. Incidently it might be mentioned that there was no chance of change in the composition of the amino acid, as it was manufactured in Prof. Hyden's laboratory, and was preserved in ice all along till the time of administration.

Although the data presented above is of debatable value, because of very small number of each variety of illness, the impression was that the drug had produced no significant improvement in nine out of ten. The only case that responded was a case of depression of three years duration. Whether the improvement in this case was due to Malononitrile or a natural remission remains undecided.

These findings gave us an impression that there was no definite evidence of therapeutic efficacy of Malononitrile in the small series of mentally ill patients of different types treated by us.

Summary

Ten cases of mental illness of different types were treated with Malononitrile. The trial sample of the amino acid was obtained from Prof. Hyden's laboratory and the technique was similar to one used by them. Nausea, vomiting, flushing of the face, and other side effects were noted, but all of them were controlled easily with Sodium Thiosulphate. Only one out of ten cases, a case of depression showed satisfactory response, that was well maintained.

The impression was that Malononitrile in our small series had no marked therapeutic action.

We thank Prof. Hyden for his kind cooperation for supplying to us Malononitrile solution from his laboratory in a refrigerated condition.

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DERANGEMENTS OF CONSCIOUSNESS IN POSTERIOR FOSSA SPACE OCCUPYING LESIONS

by BALDEV SINGH, Vellore.

There has been a great deal written about consciousness, but the fundamental neurological basis for it has been demonstrated experimentally only recently by Magoun et al.[1]. There have been difficulties even to define consciousness. In this paper, the word is proposed to mean awareness of environments and of self. It is a function of an organism in action, a physiological performance of a very active tissue, the central nervous system. Unlike some other functions of the neurone this one does not follow the all or none law. Its intensity or "acuteness" varies greatly even in normal health.

Central nervous system is a hierarchy of more and more complex neural mechanisms. In it there is an integration of complexity at its various levels as to be useful to the animal for healthy living. There are at least 6 levels (Fig. 1) described:—

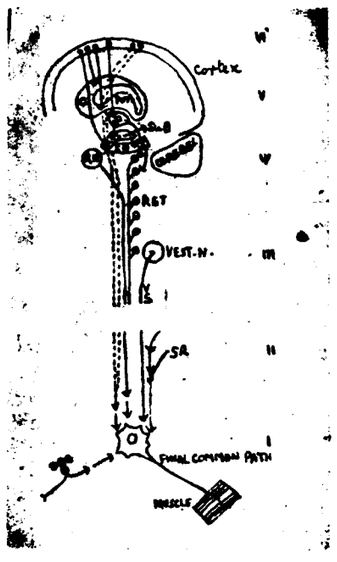

Fig. 1.

(1) The neuro-muscular, (2) the spinal, i.e., the final common path, (3) the hind brain with postural centres, (4) the midbrain, (5) the Thalamus and Basal Ganglia and finally (6) the cerebral Cortex itself. (Fig. 1).

The graded function of consciousness also is integrated at many levels like other important functions of the central nervous system. At every stage it is a state of awareness of the stream of afferent impulses that reaches the highest level in a given organism. In man and higher mammals, the experimental evidence seems to hold that this function is related to the levels 4, 5 and 6, i.e., the upper part of mid-brain, (the region round about the aqueduct of Sylvius), the Thalamus and the Cerebral Cortex, all acting in association with one another (Fig. 2). This mechanism was originally described by Forbes[2] and the concept has since been elaborated and postulated by several investigators. The essential basis is an arrangement of neurones which can keep up a sustained state of reverberating circuit so as to perpetuate an un-abated activity following a few intermittent stimuli. As illustrated in Fig. 2,

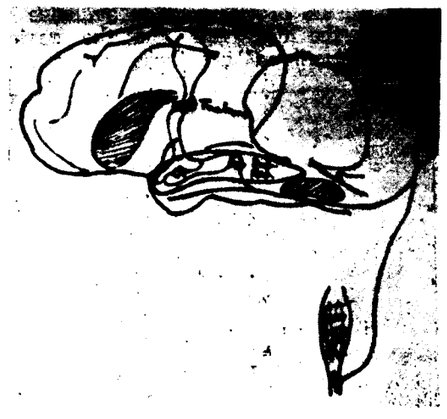

Fig. 2.

the afferent impulses received at the peri-aqueductal reticular tissue from the lower levels establish neuronal circuits through the Thalamus to cortex and back supplying mechanism for sustaining a stimulus by reverberation. Damage anywhere in this circuit is likely to produce effects which give rise to symptoms of various types and grades of unconsciousness. This is very adequately explained in Fig. 2, which illustrates the fact that the afferent fibre when it reaches the mid-brain gives a collateral that enters the reticular formation whilst the continuation of this axon passes to the Thalamus and ends there. The impulses conveyed to the former go to the Thalamus and from there finally on to the sensory cortex. The functional difference between these two pathways is that in case of the first, i.e., via the substantia reticularis, just a threshold stimulus is enough to keep the animal alert; whilst in the second pathway, i.e., when stimulus ends in Thalamus more intense stimulation is required to achieve the same purpose and then too only partially. Ranson[3] therefore very adequately suggested the name of "wakefulness centre" for this first circuit. This mechanism also offers an explanation as to why when an animal is semi-conscious a more intensive stimulation is needed to arouse it.

Embryological dveelopment of the nervous system provides more evidence to support the hypothesis that the regions already mentioned are those concerned in

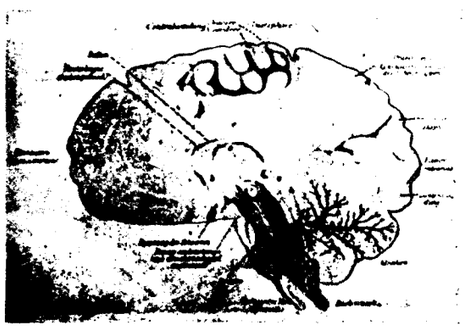

Fig. 3.

maintaining consciousness. Fleschsig has shown as illustrated in Fig. 3, that during the foetal life and afterwards in infancy the first areas to be myelinated are the ones in the brain stem. This is closely associated with the appearance of consciousness in the growing organism.

Finally phylogenetically too it is possible to support this contention because animals such as fishes[4] in which Brain stem level is the highest seat of the nervous integration, a state of wakefulness is an incessant process. Such animals never show clouding of consciousness or sleep. It is also concluded from this that both the phenomenon of sleep and certain states of discriminative and incisive consciousness in man as stimulated by impulses from the special senses requiring intense attention are associted with the development of the cortical tissue. Birds and reptiles[5] possess only a rudimentary cortex and so they sleep very little, and have a very limited range of discrimination.

Anatomically quite a large part of the brain stem, medulla, Pons and mid-brain, is situated in the posterior fossa of the human skull. Since this is the region which is concerned so much with the physiology of consciousness, it is just logical that under certain circumstances of disease in the posterior fossa the patient should show symptoms related to the derangements of awareness. In this paper, we shall deal with the expanding lesions of the posterior fossa and how they produce the symptomatology of deranged consciousness.

The posterior fossa is bounded above by a tough dural sheet, tentorium cerebelli with an exit hole called the 'incisura'. This hiatus transmits the brain stem, vessels like Basilar artery and vein and pontine, interpeduncular, and ambient cisterns. The vein of galen opens into the anterior end of the straight sinus whilst the veins of Rosenthal lie one on either side of the mid-brain as is illustrated in Figs. 4 & 5. The floor of the fossa contains foramen magnum the basioccipit and clivus and the foramina which transmit the hypo-

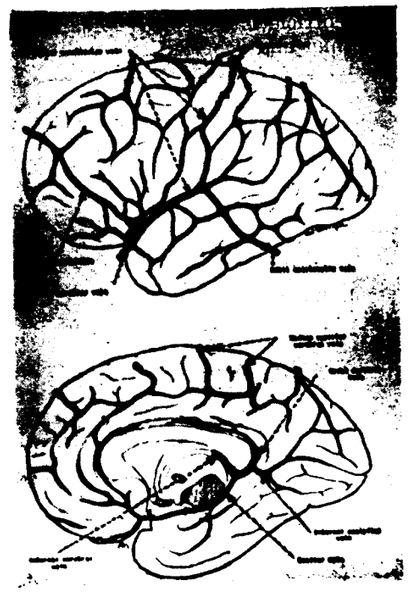

Fig. 4.

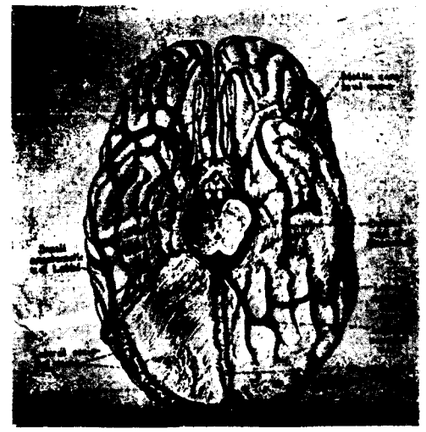

Fig. 5.

glossal and glossopharyngeal nerves with associated blood vessels. Anteriorly the fossa is limited by the petrous pyramids one on either side, which through the internal auditory meatus transmit the facial and auditory nerves. The posterior wall is formed by the occipital bone below the Torcular herophile and the Transverse sinuses where the tentorium cerebelli is attached to the bone. The Transverse sinuses end at the Jugular foramina to give rise to the Internal Jugular veins. The main contents of the fossa are cerebellum, Brain stem with the fourth ventricle and its choroid plexus, the cranial nerves already enumerated, the cisterns particularly the cerebello-medullaris and the basilar, the meninges and the blood vessels. It accommodates part of the aqueduct of sylvius. Tentorium cerebelli is a tough membrance and allows only a very limited expansion of the posterior fossa contents. The most vital point is the incisura tentorii which allows communication between the supra and the infra tentorial regions. Whenever there is increased intracranial tension in either of these two compartments the brain stem gets displaced up or down as the case may be and gets pressed by the edge of the incisura or any part of the brain which gets pushed into this tentorial hiatus. The subject of Tentorial coning has been very extensively discussed in the literature and the effects and dangers of coming through the foramen magnum are well known. In this paper, the effects of Infratentorial coning, in so far as consciousness is concerned, is presented.

There has been 21 cases of expanding posterior fossa lesions seen and operated on during the last 2½ years in the Neurosurgical service of the Christian Medical College Hospital, Vellore. Every one of these 21 cases was investigated by ventriculography and the nature of the lesions determined by pathological studies. This group contains 8 cerebellar Tuberculomas, 2 of which were found to be high up near the incisura tentorie, 4 medulloblastomas, 2 astrocytomas, 5 acoustic neuro-fibromas,

one meningioma and one ependymoblastoma. Out of these 21 cases, there were six which either gave history of derangements of consciousness, i.e., drowsiness, semi-consciousness or complete unconsciousness lasting for a few minutes or upto several days. 3 of these 6 cases on admission were in a state of marked drowsiness. In everyone of these 6 cases, ventriculography showed some common features. In a composite form this is illustrated in Fig. 6. The 3rd ventricle gets enlarged deformed, and displaced and aqueduct is either seen only in its proximal part or not at all. In all these cases the lateral ventricles are markedly dilated and very often on operation, the various cisterns in the posterior fossa are found to be abnormally tense because of the blocked cerebrospinal fluid in them. In two out of these 6 cases the angle of Wilson and Lutz is found to be less than 140°. It, in fact, is 120° and 110° respectively. This is an angle which subtends between the line drawn at the base of the anterior fossa reaching the tip of the anterior clinoid process and another line from the anterior clinoid process to the supra-pineal recess. This angle is diminished if brain stem is displaced upward and increased if the stem is displaced downwards. Dyke and Davidoff have demonstrated by Pneumoencephalography

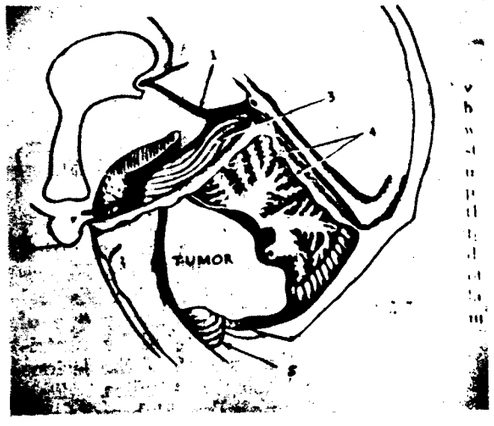

Fig. 6.

in cerebellar tumours that the floor of the fourth ventricle is closer to Dorsum Sellae than normal and roof of the fourth ventricle is further from the posterior fossa than normal. In none of our six cases was Pneumoencephalography done and hence this statement cannot be corroborated.

When the tentorium cerebelli is considerably elevated the posterior horns of the lateral ventricles do not fill well. Although in two of the six cases quoted this could be demonstrated, it is not taken to be very reliable as such filling defects are present even when there is no tentorial elevation.

The brow up view sometimes shows that the tip of the temporal horn as it is viewed in the orbit is more laterally displaced on one side than the other. This is an indication of transtentorial displacement upwards of the cerebellum on the side of the displaced horn. This has been detected only in one of these 6 cases.

The whole of this picture gives an impression of a block at the incisura tentorii due to the upward displacement of brain stem or infratentorial coning produced by cerebellar herniation or herniation of the tumour situated near the hiatus. As to how this takes place in cerebellar and acoustic nerve tumours is diagramatically represented in Figs. 7 & 8. The effect of all these factors is that the reticular tissue suffers a damage and gives rise to the alteration in the consciousness found in these patients. In the clinical examination of these patients it is not enough just to make a general statement about the awareness. In order to know whether it is the mid-brain, Thalamic, cerebral, or involvement of all three simultaneously, it is required to investigate the symptom more extensively. A patient may be totally unconscious though still alive. In that case he won't react to even a violent stimulus as burning. This is an example of the damage being maximum in the mid-brain and diencephalic region blocking both the channels already described. If he is conscious to painful stimuli of some intensity Thalamus is functioning. If he is aware of sti-

Fig. 7.

Fig. 8.

muli through special senses the cortex and Thalamus are both intact and there is very minor damage to the mid-brain and diencephalic regions producing drowsiness.

The pathology of the lesion is very well explained by Scheinker.[5] He found that this type of Transtentorial herniation was associated with haemorrhages and oedema of the brain stem the macroscopic and the microscopic appearances of which are represented in Figs. 9 & 10 respectively. The brain stem on naked eye examination shows tremendous swelling of the left half of it and the right peduncle shows signs of compression and downward displacement. There are coalescent small haemorrhages within the swollen left tegment. Section taken from the haemorrhagic area and its surroundings shows the congested veins with all degrees of disintegration of the vessel wall. In some of these perivenous haemorrhages only the shadows of the vessel wall was to be seen in the centre. In the vicinity of the haemorrhages the enlarged veins are surrounded by large masses of extravasated red blood cells extending into the distended perivascular spaces. The neural parenchyma shows a high degree of rarefaction, and oedema especially pronounced in the vicinity of the haemorrhages. There is no glial or mesodermal reaction to be seen.

Such a situation as this arises not because the arterial supply of the brain stem has been pressed upon but because the venous supply has been obstructed. This fact has already been made clear in connection with the description and course of the veins of Rosenthal which drain the mid-brain.

Although the displacement of the brain stem and the damage done thereby to the rostral reticular tissue due to venous block plays the chief role in the symptomatology of unconsciousness, the same damage can also be brought about by the direct pressure of the tumour thereby producing a local pressure necrosis or Ischaemic necrosis as was originally surmised by Cushing in Mutter lecture. He had by putting a rubber bag subdurally demonstrated that the ill-effects of increasing intracranial pressure in the posterior fossa were due to the displacement of the medulla oblongata downwards into the spinal canal and coning there which damaged the vital centres. This is really only half the truth for even though the dilated 3rd and lateral ventricles act as a deterrent to Infratentorial coning, this occurs all the same in due course as the disease advances. This fact,

Fig. 9.

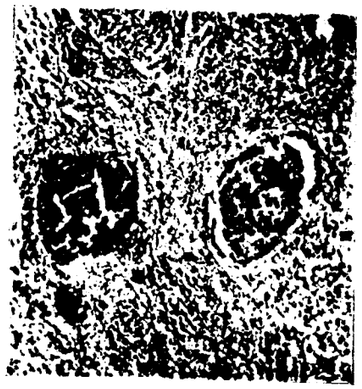

Fig. 10.

however, emphasises that in cases in which infra-tentorial coning is suspected, ventricular puncture must always be followed immediately or simultaneously by lumbar puncture. This would avoid further impaction of the mid-brain when ventricular puncture diminishes the pressure in the supra-tentorial space.

The conception of Respiratory, cardio-vascular and vomiting centres in the medulla is being more and more clarified by Wang and his associates by putting into the medulla very small radon seeds. It is becoming increasingly evident that these centres have a diffuse boundary and are a part of the general reticular tissue in the region. As has been put by Jefferson, one would be hard put to it to say at what point the pressure of posterior fossa tumours is most important since it must be exerted over the whole length of the pons and medulla. It is known, of course, that the hind brain may recover from a degree of physiological block which would have proved irreversible in the mid-brain or the inter-brain. Evidently the reticular tissue subserving the function of consciousness is much more sensitive to injury than the reticular tissue which is concerned with functions of circulation, heart-beat, blood pressure and respiration.

Consciousness therefore is a very delicate process. It is when the consciousness is dulled that there is case for alarm for this is indeed an alarm signal. It is particularly true of the cases who are to undergo surgical operation. It is always an advantage to improve the state of the patient in so far as damage to the brain stem is concerned before starting the anaesthesia. In some of these cases if proper precautions are not taken the anoxia produced by anaesthesia fatally disintegrates the neurones already under heavy stress. It is ever so much safe to remember that the greater the drowsiness the patient has, the worse he is as an operative risk and also the deeper in coma he is the worse is the prognosis so far as his recovery is concerned. The depth of unconsciousness is an indication that the disease is advanced beyond reversibility.

Summary

It has been attempted in this paper to explain that consciousness depends on integration of neurone function at various levels of the nervous system. Physiological, Embryological and Phylogenetic evidences prove that in man and in higher mammals consciousness is due to association and correlation of physiological activities in the region of the upper part of brain stem, diencephalon, Thalamus and the cerebral cortex. Derangements of consciousness are due to lesions in these various zones.

It has been further demonstrated by clinical and special investigations that in posterior fossa space occupying lesions, it is the damage eto the brain stem which is th essential feature to produce defects in consciousness. This damage to the brain stem is due to its displacement producing venous compression.

Finally, the deeper the patient is comatosed the worse the prognosis. When ventricular puncture is contemplated, it is safe to do simultaneously lumbar puncture also to prevent further transtentorial impaction. To prevent any ill-effects of anoxia which engers life in these cases, all precautions should be taken to ensure adequate oxygen supply to the brain in general and brain stem in particular before anaesthesia is administered.

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NEUROLOGY IN INDIA AND THE NEUROLOGICAL SOCIETY OF INDIA

by B. RAMAMURTHI, Madras.

The aims and objects of the Neurological Society of India are explained in the Constitution of the Society, published elsewhere in this Journal.

Neurology and Neuro-Surgery, as a speciality, are just now springing up in our country, though they achieved this status in other countries more than a quarter of a century ago. Due to various reasons, this delay in the development of this speciality was inevitable. But now with the achievement of political independence and with the unbounded energy and enthusiasm of the younger members of the Nation, it is inevitable that these specialities grow from strength to strength. Prejudiced and disinterested men may delay the progress, but not stop it.

As has been pointed out in his address by the previous president, the need for these specialities is apparent. To give an instance to substantiate this statement — In the 2½ years in which the Neuro-Surgical Unit of the Government General Hospital, Madras, has been functioning, more than 120 Craniotomies have been done. The existence of these special units have proved that

(a) Neuro-Surgery can be done in our country, and,

(b) that there is more than enough clinical material in the country needing relief from this speciality.

Time is past when Neurology was a mere academic exercise. With the advancement of therapeutics and the advent of Neurosurgery, Neurology has become a subject where therapy is possible and cure very often effected.

Hence we feel that every medical college in this country must develop its speciality of Neurology and Neuro-Surgery and thus contribute to the relief of suffering and also the advancement of knowledge.

Sir A. L. Mudaliar, in his Inaugural Address, has stated: "It is a matter for regret that many of the specialities in medicine were not taken note of in the past with the result that Indians have had to go to foreign countries to study and to gain some knowledge of such specialities; consequently, there were few who had such facilities and who could help to train their countrymen in such specialities. I hold to the belief that no college could satisfactorily fill the role of undergraduate teaching unless simultaneously there were developed facilities for post-graduate study and teaching and necessary provision was available for research being carried on."

It needs men with courage, broad vision and a youthful mind to achieve the above and place Medicine in India in the proper pedestal. Shall we hope that this will come to pass?

Development of any new speciality has been beset with innumerable obstacles. Local prejudices, indifference, apathy and vested interests have to be overcome: men with singular devotion to the ideal are needed. It is the aim of the Neurological Society of India to draw strength from all such men and see that the speciality of Neurology advances in this country. To this aim is this Journal devoted and on this endeavour, we offer our prayers for the Almighty.

ASSOCIATION NOTES

MINUTES OF THE EXECUTIVE COMMITTEE MEETING, HELD AT HYDERABAD, ON MONDAY, 3RD MARCH, 1952

The members present on the occasion were as follows:

1. Dr. Jacob Chandy (President),
2. Dr. B. Ramamurthi (Secretary),
3. Dr. S. T. Narasimhan (Treasurer),
4. Dr. T. K. Ghosh, of Calcutta
5. Dr. N. S. Wahia, of Bombay and,
6. Dr. Baldev Singh, of Vellore.

1. The Secretary read the minutes of the previous Executive Committee meeting, held at Madras, and this was approved.

2. The Secretary was authorised to register the Neurological Scoiety of India.

3. The Committee placed on records the permission granted by the Government of Madras to Dr. B. Ramamurthi, to be the Secretary of the Neurological Society of India.

4. The bye-laws of the Society were then discussed, and the following bye-laws adopted.

Membership

1. A full member shall be proposed by any full member and the Executive Committee shall be the authority to admit members.

2. Each full member shall present a paper to the Society at least once in two years.

3. Any full member who fails to attend at least one of three consecutive meetings of the Society shall forfeit his membership, unless a reason acceptable to the Executive Committee is offered.

4. Corresponding and honorary members shall be proposed by one full member and be seconded by another full member. Their election shall be carried out by direct invitation from the Committee. Such members shall be exempt from the payment of annual subscription.

5. The position of each Associate Member shall be reviewed every four years to see whether the interest they have shown warrants their continuation of membership.

6. The Associate Members shall be entitled to attend all the meetings of the Society and may take part in all discussion and have the full previleges of membership, except that they shall not vote.

Officers

7. The officers of the Society shall hold office for one year and shall be eligible for re-election.

8. The Office Bearers of the Society will be elected at the General Body Meeting, which will be held during the Annual Conference of the Society.

9. The Presidential address will be delivered by the President at the end of his term.

10. The Executive Committee shall transact all the business of the Society.

11. The Treasurer shall operate funds of the Society.

12. The audited accounts of the treasurer must be submitted to the Executive Committee.

Local Meetings

13. Local meetings of the Neurological Society of India can be held in different parts of India, if the interest of the members warrants it.

Amendments

14. All amendments to the constitution and bye-laws shall be submitted to the Secretary at least four months before General Meeting of Full Members and copy of such proposal shall be forwarded to every full member, at least two months before the meeting. On motion, the amendment shall be accepted by a vote of 2|3rd of the total number of full members of the Society.

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5. It was unanimously agreed that an Indian Journal of Neurology is necessary Dr. T. K. Ghosh, of Calcutta, is requested to send the report in about two months' time regarding the details of publishing the Journal.

6. The Treasurer present a statement of accounts to the Committee for information. He was authorised to incur any necessary expenditure for printing letter-papers, to get envelopes, for postage and stationery, and other contingent expenditure.

7. The Executive Committee placed on its records, vote of thanks for the following :

1. Sir A. Lakshmanaswamy Mudaliar,
2. Major Gen. Bhatia,
3. Dr. Pargaonker, and,
4. Members of the Reception Committee.

MINUTES OF THE GENERAL BODY MEETING

The first General Body Meeting of the Neurological Society of India was held at 5 p.m. on 3rd March, 1952.

2. The Secretary explained to the meeting, how the notion of the Society was conceived and the Society was inaugurated. He said that the response from medical men all over India to the Neurological Society was very enthusiastic.

3. The President requested full co-operation from all members to make the society a useful and successful one.

4. It was decided to have the next annual meeting of the Neurological Society along with the Association of Physicians, at Poona, in March, 1955.

5. There was a general discussion and it was agreed that the papers to be read must be received earlier and the programme fixed sufficiently early, so that there is no last minute disturbance.

6. It was also decided to correspond with other Societies, so that a symposium may be held.

7. A resolution of vote of thanks was passed for the following :

1. Sir A. Lakshmanaswamy Mudaliar,
2. Major Gen. Bhatia,
3. Dr. Pargaonkar, and the,
4. Members of the Reception Committee.

MINUTES OF THE EXECUTIVE COMMITTEE MEETING HELD AT VELLORE IN DECEMBER 1952

After a considerable discussion on the proposal of starting a journal, it was

Resolved to use Rs. 225 from the Society's Funds towards the publication of the Bulletin of the Society.

Dr. Chandy kindly agreed to bear any extra expenditure that might be incurred in the publication of the Bulletin. One hundred copies of this Bulletin are to be printed. It is to be more or less in the style of the 'Indian Journal of Surgery'.

2. The nominations for election of the President and the Vice-President for the year 1953-54 were discussed and it was

Resolved that a request should be made to Dr. T. K. Ghosh of Calcutta, to stand for the Presidency and Dr. N. S. Vahia of Bombay for the Vice-Presidency of the Society for the year 1953-54, at the election of office-bearers of the Society in the Executive Meeting of March, 1953.

3. The proposal of membership of Dr. R. B. Davis of Ranchi was presented by the Secretary and it was

Resolved that he may be accepted as a member and that he should be informed to read a paper before the Society for confirmation of his membership.

4. There had been questions regarding full time neurological work of Dr. Das of Calcutta, and his eligibility for membership in the Society. It was, therefore,

Resolved to ask the Secretary to write to Dr. Das whether he is eligible for membership or only for associate membership.

5. It was reported by the Secretary that Dr. C. C. Saha has already sent in Rs. 20 as membership fee to the Society requesting admission as member. In view of the fact that Dr. Saha is practicing now as a General Consultant, and it is in the constitution of the Society that members who are not full time workers in Neurology or its associate branches, could not be admitted as members of the Society, it was

Resolved that Dr. Saha be informed of the constitution of the Society and to inform him that we regret our inability to accept him as a member and to request him to be an associate member.

6. It was also

Resolved to ask the Secretary to send letters to all members and associate members requesting them to present papers at the forthcoming Second Annual Conference of the Neurological Society of India : also to write to the Society for confirmation of their membership, to avail themselves of this opportunity for the same.

MINUTES OF THE EXECUTIVE MEETING HELD AT POONA ON THE 26TH FEBRUARY, 1953

Dr. S. T. Narasimhan, was proposed to the chair unanimously.

The following were declared elected as Full-Members:

1. Dr. Leo Krainer, (Lt. Col.)
   Armed Forces Medical College, Poona.
2. Dr. C. G. Subramania Iyer,
   Neuro-Pathological Unit, Tata Memorial Hospital, Bombay.
3. Dr. Darab K. Dastur,
   Neuro-Pathological Unit, Tata Memorial Hospital, Bombay.

Dr. Tarit Kumar Ghosh, M.D., Neurologist, of Calcutta, was unanimously elected President of the Society, for the year, 1953-54.

Dr. N. S. Vahia, of Bombay, was elected as Vice-President.

THE SECOND ANNUAL CONFERENCE

The Second Annual Conference of the Neurological Society of India was inaugurated at the Auditorium, in B. J. Medical College, Poona, along with the Association of Physicians and Paediatricians.

The Welcome Address was read by Dr. Ranade, and the inaugural address by Dr. M. R. Jayaker, Vice-Chancellor, Poona University.

Messages

Messages for the Conference of the Neurological Society were received from the following:

Hon'ble Ministers for Health
of
Government of India, New Delhi,
Government of Madras, Madras,
Government of Bihar, Patna,
Government of Punjab (East), Jullunder,
Government of Travancore-Cochin, Trivandrum,
Government of Saurashtra, Rajkot,
and,
Dr. B. V. Mulay, President, Indian Medical Assn., Sholapur.

Presidential Address

In an inspiring Presidential Address, Dr. T. K. Ghosh, emphasised the wide basis from which Neurology has sprung and the great future that awaits it. Many problems in the structure and function of the nervous system are yet to be unravelled. There were too few neurologists and neuro-surgeons in the country. He emphasised that with wide co-operation from all sections of the Medical World, the cause of Neurology will be furthered.

Scientific Papers

The following papers were presented at the Conference:

1. Melononitrile in the Treatment of Mental Diseases
   by Dr. N. S. Vahia, Bombay.

2. Electrical Discharges of Epileptic Brain
   by Dr. Baldev Singh, Vellore.
3. Ophthalmoplegia (Clinical Study)
   by Dr. Jacob Chandy, Vellore.
4. Treatment of Parkinsonism
   by Dr. P. Isaiah, Vellore.
5. Cysticercosis in India
   by Dr. Menimo De Souza, Bombay.
6. Association of Acute Anterior Poliomyelitis & Tuberculous Meningitis
   by Dr. Leo Krainer, Poona.
7. Macroscopical Demonstration of the Fiber Anatomy of the Brain
   by Dr. Leo Krainer, Poona.
8. Cutaneous Sensations with special reference to Leprosy
   by Dr. K. Dastur, Bombay.
9. Objective & Subjective Electro Encephalography
   by Dr. S. T. Narasimhan, Madras.
10. Pituitary Appoplexy
    by Dr. B. Ramamurthi, Madras.
11. Stellate Ganglion Block in Cerebral Thrombosis
    by Dr. Siva Saran Misra, Lucknow.

THE SECOND GENERAL BODY MEETING

The Second General Body Meeting of the Neurological Society of India was held on the 28th February, and 1st March, 1953, in two sessions.

Dr. T. K. Ghosh, the President, was in the chair.

Minutes of the previous Executive Committee meeting

The Secretary read the minutes of the previous Executive Committee Meeting, which was adopted.

Annual Report by the Secretary

The Secretary read the report of the last year's activities. He said that the Membership of the Neurological Society of India has shown an increase from 32 to 58. Of these, there are 13 Full Members and 45 Associate Members. During the year under review, 4 Full Members, and 16 Associate Members were admitted. The Secretary requested, that Dr. M. V. Govindaswami, and Dr. Grillmayr, may be admitted as Full-Members.

The Secretary explained that he has remitted the amount equivalent of $12 to the International Neurological Congress. The Society is affiliated to the International Congress, and according to the rules of the Congress, all National Societies have to contribute at $2 per full member, towards the Congress. The Congress will be held at Lisbon in the month of September (7th to 12th), this year.

The Secretary reported that Local Clinical Meetings were held at Madras and Vellore, under the auspices of the Society.

The Secretary explained the efforts taken to publish the Bulletin 'Neurology' the official publication of the Society. The Executive Committee, which met at Vellore, in December last, decided to spend Rs. 225 for the publication of the bulletin. Dr. Jacob Chandy has agreed to contribute any amount that may be in excess of this amount. The Secretary thanked Dr. Chandy for this contribution.

The Secretary closed the Annual Report by emphasising that the Neurological Society of India was purely a Scientific Body and that the Members were co-operating in that spirit. He requested that such co-operation should continue to come in greater measure.

Discussion on Full Membership & Associate Membership

There was a discussion on this subject. Dr. M. De Souza of Bombay wanted rules to be relaxed. It was explained to him that any one, who declares himself to be a Neurologist is entitled for admission to the Society, as a Full Member. It is the spirit of the rules and not the letters of the rules which is important.

Election of Office Bearers

There was a general discussion about the rules for the election of office bearers and the little discrepancies that arose during the preceding years, was explained. After the discussion, the following resolution was passed unanimously:

"It was resolved that the Office Bearers of the Society will be elected at the General Body Meeting, which will be held during the Annual Conference of the Society. The Presidential address will be delivered by the President at the end of his term"

Proposed by Dr. S. T. Narasimhan,

Seconded by Dr. D. K. Dastur.

The resolution was then adopted.

In accordance with the above resolution, the following were declared elected:

Dr. B. Ramamurthi, was elected as Secretary

Proposed by Dr. N. S. Vahia,

Seconded by Dr. Leo Krainer.

Dr. S. T. Narasimhan, was elected as Treasurer

Proposed by Dr. N. S. Vahia,

Seconded by Dr. D. K. Dastur.

Annual Accounts

The Treasurer then presented the Accounts for the year 1952-53 which was adopted.

Vote of thanks for messages received

A Resolution of vote of thanks was adopted for the Messages received and it was decided to communicate same to them.

Vote of thanks for the Reception Committee

It was moved from the chair to thank the President and the Members of the Reception Committee at Poona, for their hospitality and the very fine arrangements that they had made both for the Scientific and the Social aspect of the Conference.

Resolution of thanks to Dr. Chandy

It was resolved to thank Dr. Jacob Chandy, for the promise of contribution to the Journal of the Society.

Discussion on All-India Medical Institute

There was a discussion about the All-India Medical Institute and the facilities to be given for Neurology and Neuro-Surgery. It was also decided to request Dr. D. K. Dastur, to contact Dr. R. Ginde and Dr. Kahnolkar, and know from them about the exact state of affairs on this matter.

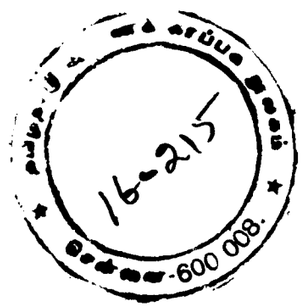
Admission of Honorary Members

It was moved from the chair that the following be admitted as Honorary Members of the Neurological Society of India and it was adopted:

1. Professor Wilder Penfield,
   Montreal Neurological Institute,
   MONTREAL, (Canada).

2. Professor Sir G. Jefferson,
   Professor of Neuro-Surgery,
   University of Manchester, MANCHESTER (U.K.).

3. Professor Hans Hoff,
   Professor of Neurology,
   Neurological Clinic, University of Vienna,
   VIENNA (Austria).



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