PROCEEDINGS OF THE SOCIETY OF BRITISH NEUROLOGICAL SURGEONS: 59th MEETING

The 59th Meeting of the Society of British Neurological Surgeons was held at the Royal Society of Medicine, London, as a joint meeting with the Canadian Neurological Society and the Association of British Neurologists on July 14-15, 1959.

The Chair was occupied in rotation by the Presidents of the three Societies, G. S. Clark-Maxwell (Derby), Dr. Allan Walters (Toronto), and Professor F. J. Nattrass (Newcastle). Dr. Earl Walker (Baltimore) attended as the special guest of the Society.

Neuraxial Congenital Dermal Sinuses

W. S. KEITH (Toronto) reported eight cases of lumbo-sacral dermal sinus. Three cases were infected, of which two recovered and one died. He noted that in spite of many publications on this subject during the past 30 years these insignificant lesions were usually overlooked until infection had gained access to the central nervous system. Experience at the Hospital for Sick Children, Toronto, showed that death from this cause still occurred and that there was still a high incidence of damage to the brain, spinal cord, and cauda equina and also severe complications in the urinary tract. All these complications were preventable and a greater awareness of the existence and significance of these lesions was desirable.

Colloid Cysts of the Cervical Canal

R. T. JOHNSTON (Manchester) described in detail two cases of intrathecal cyst of the cervical canal causing cord compression. The cysts were attached to the anterior aspect of the cord by a narrow pedicle, contained colloid, and were pathologically indistinguishable from the colloid cysts of the third ventricle. Their relationship to congenital cysts of the central nervous system in general was discussed, and the question raised as to whether colloid cysts favour predominantly the third ventricle because they originate only in a vestige of a specialized structure such as the parahypophysis, or because an error of development at a stage which produces a colloid cyst is more likely to occur in the third ventricle than elsewhere.

Radioactive Encephalography: Automatic Brain Scanning Using Radioactive Iodinated Albumin

WILLIAM FEINDEL, JOSEPH STRATFORD, G. A. B. COWAN, and S. FEDORUL (Saskatoon) reported on the method and results of using an automatic brain scanner in localizing various lesions in the brain.

They noted that a number of radioactive substances had been used recently as indicators of local changes in the blood-brain barrier. Local variations in the uptake of the substances in the brain could then be mapped out by radiation counting devices applied externally to the head. Rectilinear automatic scanning had been developed, particularly by Sweet and Brownell, and detailed studies of collimation had been made by Shy and others. The present report was a review of 70 neuro-surgical cases with verified intracranial lesions in which automatic scanning was carried out with twin scintillation counters mapping the head in a spherical pattern. This apparatus, which had been designed by Reid and Johns at the Saskatoon Cancer Clinic, had been in use for over the past three years and the advantages and limitations of the method were described.

Aspects of the Behaviour of Human Gliomas in Tissue Culture

T. P. MORLEY (Toronto) reported on the results of tissue culture of human gliomas.

As a basis for future study of gliomas in vitro, 60 tumours had been grown in culture and their appearance and behaviour recorded. Certain features might be seen which were characteristic of the group, even though the various types of tumour did not give rise to predictable patterns in culture. Attempts—at last unsuccessful—at transplanting these tumours into rats and mice were reported briefly and information concerning the shedding of glioma cells into venous blood was presented.

The Evolution and Dissolution of Convulsive Phenomena following Head Injuries

A. EARL WALKER (Baltimore) presented a report on the course of patients having post-traumatic convulsive disorders over an eight- to 10-year period. He noted that the incidence of post-traumatic epilepsy did not seem to have lowered, compared with First War figures, in spite of better methods of wound therapy, less infection, and improved treatment for the severely head-injured patient. He wondered, however, whether the populations of the surviving head-injured from the two world wars was the same. Was it not possible that better methods of treatment had saved the lives of severely injured patients, a group in which the frequency of epilepsy was almost 50%?

He stressed the difficulty of making a clear diagnosis in many cases and this was especially true of sudden.
attacks of “dizziness”. These attacks were commonly ascribed to vasomotor instability and although commonly transient might persist for weeks or months. He suggested that inclining the patient on a tilt table whilst taking E.E.G. recordings or suddenly compressing the ocular globes might help in diagnosis. The appearance of slow waves associated with syncope favoured a vasomotor origin of the attacks.

Even when attacks could be identified as epileptic it was not easy to decide whether the patient should be called an epileptic or not. Such a designation involved important courses of action by the patient, by his medical attendant, his employers, and his lawyer. It was of the utmost importance to know, therefore, if a patient had had one or more attacks which were epileptic, what were the chances of further attacks. In an attempt to answer this question he had studied two series of patients. The first was a series of 244 men seen and studied in detail in 1945-46. All had sustained head injuries, most of them penetrating the brain, and had had one or more convulsive seizures. These had been followed for a period of 10 years. The second series was 732 head-injured men in World War II examined six to nine years after injury. Of these, 207 developed epileptic manifestations. The period of follow-up varied from two to 10 years. The total followed up of the two groups was 435; 16 had died in the interim period.

In the first series of those living, 46.1% had had no attacks for more than two years and 35.6% had had no attacks from the fifth to the tenth year after injury; 14.6% had experienced only one or two attacks per year in the last five years and the remainder were having frequent attacks. This freedom from attacks was not related to medication. Of the second series, 21.1% had had no attacks for five years, 12.1% for two years, 22% had rare attacks, and 43.9% were having several attacks a year.

The frequency of major attacks was less than that of attacks of all types. Many of the patients having minor seizures were so little affected by them that they might be considered as “normal”. This spontaneous regression of attacks after head injury had been mentioned by most writers on the subject but had been little emphasized.

The time of cessation of attacks was of interest and in all categories the highest incidence of cessation was in the second and third years. For all types of attack, 20% stopped in this period and about 5% per year thereafter. The incidence of freedom from major seizures was almost double this figure in the second and third years.

Patients with attacks occurring early after a head injury were generally considered to have a better prognosis than those with late attacks. In the first series, patients who had early attacks fared no better than those starting late. This series, however, was probably a selected one and patients who had had early seizures in evacuation units or base hospitals might not be referred to the epilepsy centre. In the second series, however, only 15% of patients who had an attack within a week of wounding were having attacks six years later; this might be compared with 50% of patients having their initial seizure one to four weeks and 65% of those having an initial attack more than four weeks later.

In the first five years, the cessation or absence of any type of attack for one year was a good omen, for such patients had about four chances out of five of having no more attacks in the next five to eight years. If the patient was free of attacks for two years, the chances of recurrence of multiple attacks was quite slight. Even if the patient had occasional attacks in the first five years after an injury, however, there was still one chance in four that there would be no recurrence of attacks in the subsequent five years.

Frequency of attacks was of prognostic value, those having few attacks in the first five years being more likely to become free of attacks. Although the number of attacks might remain the same, improvement could take place by a shift from major to minor seizures.

Many people had hoped that E.E.G. recordings would give reliable information as to the possibility of epilepsy. From the evidence available he could only conclude, however, that brain waves might denote cerebral damage but they did not reliably indicate or forecast the occurrence of convulsive complications. Hence an abnormal E.E.G. did not mean that a patient had or would have post-traumatic epilepsy. In fact, men having comparable severity of injury, with or without epilepsy, had E.E.G.s of practically identical degrees of abnormality. This was true of records made six months after injury and many years later. In head injuries, therefore, the E.E.G. findings correlated with the degree of severity of wounding which was only one of the determining factors in the genesis of a post-traumatic epilepsy.

Where there was reasonable suspicion of attacks having occurred and a fear that these might recur, he felt it would be better to award the man an insurance policy against recurrence rather than compensation. In closed injuries, the premium should be not more than 1% and in open wounds not more than 10% of the face value of the policy. If he did develop seizures, he would be compensated, if he did not he would not have been stigmatized and the compensating agent would not have paid a penalty.

Even if seizures did develop, it should be clearly understood that they might not chronically recur. In fact, if within the first year or two after a head injury, the patient had had only one or two attacks, he would question the advisability of labelling the condition post-traumatic epilepsy. There was excellent evidence that such patients had a good chance of living a normal life not punctuated by convulsions.

**Psychogenic Regional Pain Alias “Hysterical” Pain**

**Allan Walters (Toronto)** delivered the Presidential address of the Canadian Neurological Society on this subject. He noted that hysterical pain had traditionally meant pain in a bodily part, often with functional tenderness and sensory deficit and with no source of afferent stimulation. When it persisted unrecognized, it might elude diagnosis and defy treatment. A clinical
series of hysterical pain problems from a neuro-psychiatric consulting stream had been reviewed. The range of psychiatric backgrounds extended well beyond hysteria to include most of the neurotic, psychotic, and organic types of reaction. Hysterical pain, then, did not signify hysteria and theories of hysteria might provide only a partial explanation. It was suggested that the term psychogenic regional pain be brought forward into widest usage in place of the term hysterical pain.

This kind of pain was psychogenic in that it might vary independently of the afferent sensory stream, whereas it appeared consonant and appropriate to the way the patient was feeling or behaving. It was regional in that it was perceived remotely from mental activity and localized in a region of contiguous physical tissue. The boundaries of this region, be it area or volume, could not be explained by present knowledge of anatomy or physiology, whereas they seemed to have the attributes of form and symbol in the process of perception. This pain appeared to be a symbol of mental life remotely localized in the somatic perceptual field.

This emphasis on the process of perceptual symbolization as a source of pain raised a context of general problems which were discussed in detail. He noted that such an emphasis under the term psychogenic regional pain might be a more useful basis for the study and treatment of this type of pain in view of our new knowledge and fresh appreciation of the complex processes which were involved in our perception of our pains.

**Peripheral Nerve Conduction in Neurological Patients**

R. W. Gilliatt (London) said that motor nerve conduction was known to be slow in regenerating nerve fibres (Hodes, Larrabee, and German, 1948), in peripheral neuritis (Lambert, 1956), and in localized compressive lesions such as the carpal tunnel syndrome (Simpson, 1956). Comparable disturbances of sensory nerve conduction had been demonstrated by direct recording from the median and ulnar nerve trunks (Gilliatt and Sears, 1958).

These techniques had now been used to study the long-term effects of operation on the carpal tunnel syndrome. Post-operative examinations on patients with slowing of median nerve conduction at the wrist had been carried out at regular intervals for periods up to two years. Another series of carpal tunnel syndromes treated by splinting of the wrist had been investigated in the same way and a further group treated by local injection of hydrocortisone was being studied in collaboration with Dr. J. B. Foster.

In patients with lesions of the deep branch of the ulnar nerve, Simpson's original observation of a local conduction delay in the hand had been confirmed (Ebeling, Gilliatt, and Thomas, 1960). It was of interest that a lesion in the hand might cause slight slowing of conduction in the proximal portion of the ulnar trunk as high as the axilla: this phenomenon had also been observed in regenerating nerves after suture at the wrist. In mild ulnar lesions at the elbow, motor nerve conduction studies had sometimes been unhelpful, significant abnormalities being found only when recording directly from the ulnar nerve above the elbow during stimulation at the wrist. In such cases, attempts had been made to define the upper level of the lesion by stimulating the nerve trunk above the elbow and recording the arrival of afferent volleys at axillary level.

In the lower limbs it was possible to record afferent volleys from the peroneal nerve and this technique might prove of value in the investigation of patients with mild sensory loss and paraesthesiae in the feet.

In patients with long-standing polyneuritis marked slowing of nerve conduction had been the rule but in Charcot-Marie-Tooth disease the results had been variable and some of his patients had shown normal conduction velocity in spite of gross peripheral wasting.

**References**


**Broca's Contribution, Reviewed a Century Later**

Macdonald Critchley (London) discussed Broca's contribution to aphasiology. He noted that until the beginning of the nineteenth century scant attention had been paid to this subject. Articulatory defects were not distinguished from dysphasia and both were confused with incoherence of speech and mutism associated with states of semicoma. Later, physicians began to realize that speech impairment could arise independently of lingual paralysis and that loss of memory for words and names could exist without any concomitant loss of general memory.

The ideas of Gall that mental processes were divisible into rigid compartments and that each aspect of brain function could be correlated with a circumscribed area of the brain were a further stimulus to precise thinking.

Within the interregnum of indecision which these phrenological notions evoked came the classical demonstration by Broca.

He then described the events surrounding this demonstration and the discussions which followed it. He stressed the fact that the part played by Dr. Ernest Auburtin in this important event was often overlooked.

He noted that Broca's contribution triggered off a belief that speech disorder was a focal symptom in brain disease, and by implication that the normal faculty of speech possessed a cerebral "representation" of a limited punctate character.

Although many neurologists were utterly unimpressed by such over-simplification, they did not make a direct attack upon the materialists' notions and it was only within the last decade or so that more general dissatisfaction had been expressed. Not even had a rigid ascription of speech function to purely left-brain activity and speech dysfunction to left-brain disease been wholly accepted and the role of the minor or non-dominant hemisphere was no longer inadmissible.

A final remark was necessary as to the problem of...
Recent interest in the brain-stem in relation to consciousness had led to a belief that damage here was the chief cause of coma, but it should be pointed out that the hemispheres were much more vulnerable and if widely concussed were just as likely to inactivate the brain-stem centres as the other way round. The application of hypothermia to severe injuries was of great interest but should be watched very critically. A reduction of, say, the degree of decerebrate rigidity, meant that the nerve cells involved were being inactivated by cold, but when exactly this was advantageous must be very difficult to judge.

In recent years his special interest had been concerned with the rehabilitation of cases of head injury. Wartime experience demonstrated the importance of a vigorous programme of physical training, and it was regrettable that the standard of rehabilitation of civilian injuries was in many places so low. Good rehabilitation prevented the post-traumatic neurosis from developing, but it must be realistic; there was no such thing as complete recovery from severe head injury, and the patient must understand this.

G. F. Rowbotham (Newcastle) said that it was well known that the clinical picture of a head injury resulted from a complex of interacting factors that arose both within and outside the head. He was concerned with an attempt to break up this complex and to focus attention on those factors that arose directly from the injury to the brain itself. Such a study was important in the context of this discussion since, given adequate treatment, the final outcome of any head injury must depend on the nature, the site, and the extent of the intracranial injury.

He was concerned on this occasion with post-mortem findings but mentioned operative findings, clinical features, neurological adductions, and animal experiments as other important methods of investigation.

His material consisted of a series of 30 brain specimens from patients under his care who had died from head injury. The brains had been removed and the extent and size of surface haemorrhages noted. They were then cut in coronal section and lesions photographed. Histological examination was made of the brain-stem and post-mortem findings correlated with clinical signs.

(An extensive series of brain sections was then demonstrated.)

He realized that these sections demonstrated extreme degrees of injury but stressed that they probably represented in kind similar injuries which did not come to necropsy. It could be concluded from this material that the cerebral hemispheres were more likely to be damaged than the brain-stem and that lasting physical morbidity was largely hemispheral in origin. He thought that brain-stem syndromes were due, in the majority of cases, to hemispheral damage or swelling and not to intrinsic brain-stem damage. Brain-stem syndromes due to

dysarthria versus dysphasia. Originally regarded as more or less identical, they became sharply distinguished by Jackson and his contemporaries. After several changes of view in the intervening years, contemporary ideas once again tended to swing away from too rigid a cleavage between disorders of language. It was now recognized that in aphasic utterance there might be a considerable upset in phonemic patterns, but furthermore, cases of inborn articulatory defect might lead to a stunting in the free growth of the faculty of language. Lastly, the notion of articulatory apraxia—once enthusiastically acclaimed, later discredited—had once again become mooted as a factor in acquired affections of speech.

A Symposium on the Results of Modern Treatment of Head Injuries

C. E. G. Gould (Vancouver) stated that once the severity of the damage, in terms of depth and duration of altered consciousness, length of retrograde amnesia, degree and duration of confusional state, condition of the spinal fluid, and precise information as to local damage had been appraised, a prognosis could be formulated. He wished to elaborate, however, on certain factors that had no relationship to the above criteria but which might exert a profound effect on prognosis.

Amongst these factors could be mentioned the premorbid personality of the patient and the litigation factor. Frequently a potent influence was exerted on a patient by a marital partner, members of the family, or friends. These influences often "froze" a patient into a pattern of reaction to injury where function was below the level predicted on a solely organic basis.

A particular problem in Canada was the immigrant who had not been successfully assimilated into the country of adoption. In such cases one frequently saw a paranoid reaction directed towards the insuring body.

In older patients, where head injury had been incurred at work, and especially when the work was of a hazardous nature, the motivation to return to work was weak.

When assessment of the rôle these factors might be playing in a given case was added to the assessment of severity of injury in organic terms, the picture could become complex to the point where prognosis was difficult and frequently inaccurate.

W. Ritchie Russell (Oxford) said that every new discovery in cerebral physiology became applied to the study of brain injuries and the experiences of the past 50 years reflected many changing attitudes. One could recall the old emphasis on microscopic lesions in the brain-stem, the concept of acute compressive anaemia of the brain, the period of treatment by intensive dehydration or by routine craniotomy in relation to fractures. It was generally agreed now that the chief effect of head injury in acceleration concussion was a physical disturbance of neurones caused by shearing and distorting waves of force throughout the brain. Strich's recent demonstration of widespread tract destruction in cases of traumatic dementia was most significant in this regard.

The most important advance in treatment in recent years had been concerned with protecting the lungs from inflation during coma. First aid workers now transported these patients in the semi-prone position and when difficulties persisted tracheotomies were done in hospital.

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brain-stem damage were invariably fatal. He stressed that damage to the corpus callosum was common and probably played a significant rôle in the clinical picture and morbidity. He noted also that acute subdural haemorrhages were common and often of sufficient size to compress the brain, and could lead to severe and long-lasting head pains. The incidence of damage to large arteries with infarction was small but when it did occur gave rise to long-lasting morbidity.

Discussing the mechanisms of unconsciousness in head injury, he agreed that there was, as yet, no proof that the cerebral hemispheres were essential for consciousness and that injury to the brain-stem could cause unconsciousness. He thought, however, that it was possible that paralysis of function in both hemispheres could cause loss of consciousness and if this were so, then in head injuries he considered that the seat of unconsciousness was hemispherical and not brain-stem.

E. Bruce Hendrick (Toronto) reviewed a series of 1,997 cases of cranio-cerebral injury admitted to the Neurosurgical Service of the Hospital for Sick Children, Toronto, between January, 1955, and December, 1958. The major forms of injury were discussed briefly. Special attention was paid to the treatment of severe head injuries with hypothermia and to the diagnosis and handling of extradural haemorrhage in children.

A. N. Guthkelch (Manchester) considered some of the special problems related to head injuries in children and arising from a group of 590 cases admitted to the Royal Manchester Children's Hospital. He stressed the fact that in the mild case diagnosis was difficult and in 43 cases there was no record of head trauma, the only presenting feature being that the child had vomited after an accident. In another group of cases the child fell over and then had a generalized convulsion. Undoubtedly, some of these cases were of simple epilepsy and the head injury secondary to the fit.

The incidence of epilepsy was 5%, including cases with a single fit, the incidence of persistent post-traumatic epilepsy was 1.2%. The incidence was higher, 7% in cases of compound depressed fracture. From this series, however, it would seem that widespread brain damage was more important than a focal lesion, for in the group of cases with more than seven days' disturbance of consciousness, the incidence of epilepsy was 25%

Post-concussional symptoms were not often a problem and its frequency varied with the severity of the injury. Contrary to usual belief intracranial haematomas were not uncommon in children, and in this series 12 extradural, 13 subdural, and five intracerebral haematomas were treated. All but one case of acute subdural haematoma made good recoveries.

He drew special attention to a group of injuries around the orbit; these might be more serious than was at first suspected. In four cases sharp objects had penetrated the cranial cavity through the orbit. This penetration was unsuspected at first with the result that one case developed clot compression, one meningitis, and two cases developed brain abscesses. In two cases injury to the soft tissues around the orbit resulted in cerebral thrombophlebitis and epilepsy. In four cases a haematoma in the orbital area without demonstrable external injury or fracture led to intracranial infection.

There were 11 deaths in the series (1.9%) most being due to primary brain damage. In three, necropsy revealed widespread anoxic changes; tracheostomy might have saved them. Two deaths followed air encephalography.

Joe Pennybacker (Oxford) reported the results of a questionnaire circulated by the Society amongst its members in January, 1959. The object of the enquiry was to determine what proportion of head injuries in the United Kingdom were "covered" by neurosurgeons and to assess the value of special methods of treatment such as tracheostomy, hypothermia, and dehydration. The present report was a preliminary analysis of the returns which appeared to indicate that, with a few exceptions, neurosurgical cover was inadequate and facilities for convalescence and rehabilitation needed expansion. The rôle of the general surgeon in head injuries was discussed and it appeared that the most important practical step which could be taken now was to train young general surgeons in the few special techniques applicable to head injuries.

The replies as to special methods of treatment indicated general agreement that tracheostomy was valuable, but there was less certainty as to the value of hypothermia and dehydration. The prognosis in cases of prolonged coma was discussed, and although it appeared that modern methods of treatment were saving more lives in the acute stage to be followed by prolonged coma, the numbers had not yet reached serious proportions.

Intervertebral Disc Protrusions in Childhood and Adolescence

J. E. A. O'Connell (London) said that experience of intervertebral disc protrusions in childhood and adolescence had been confined to those occurring in the lumbar region. The records of 38 patients with such protrusions and aged 17 years or less were reviewed and the findings compared with those of an earlier analysis of 500 cases of lumbar disc protrusion unselected as regards age.

While the aetiological factors in adolescence were similar to those in later life, it was pointed out that the maximum age incidence of adolescent protrusions differed in the two sexes and corresponded to the period of most rapid development in each. The stresses occasioned by growth may thus be of aetiological significance.

The symptomatology in the young patients resembled that in adults, but adolescents might complain less of pain although their symptoms tend to be more persistent. No case in the series developed a severe subjective defect of function. The spinal signs and those of tension in the roots of the sciatic nerve were frequently more severe in the young patient, while the neurological ones were less frequent and less severe than in adults. Radiological abnormality was frequently absent.

Other lesions in the lower portion of the spinal canal or involving the lumbo-sacral portion of the spine were
important in differential diagnosis. Of the former, intraspinal neoplasms were the most important, but meningoceles of the extradural nerve produced a clinical picture identical with that of disc protrusions. Of the vertebral lesions, spondylolisthesis and ankylosing spondylitis were the most important. Arthritis of the apophyseal joints occasioned a picture almost identical with that of a disc protrusion in one patient. Although it was at one time always suspected in adolescents exhibiting the clinical picture which has been described, no case of tuberculous spondylitis had been encountered.

At operation, the last intervertebral disc or the penultimate one was involved in every case. The protrusions were composed of soft elastic disc tissue with a high fluid content, and on the average were larger than in adults.

The results of surgical treatment were good, 91% being either cured or greatly improved. Recurrence of symptoms required re-operation in 6% of cases.

The different clinical picture in adolescents might be due to the mobility of the spine in early life. Muscle spasm due to the stimulation of the hyperirritable extradural nerve by the large soft protrusion might induce severe deformity and limitation of spinal movement and straight leg raising. This protected the extradural nerve from injury and neurological signs were therefore mild or absent.

**Therapy in Carotid Artery Disease**

H. J. M. Barnett and W. H. Lougheed (Toronto) presented a study of a substantially large series of patients proven arteriographically to be afflicted with carotid thrombosis or stenosis. The therapeutic problems posed by this group were discussed.

The most impressive result of the study was the need that it pointed to for early diagnosis. The difficulty encountered in distinguishing carotid artery disease from middle cerebral artery disease on a clinical basis had led them to conclude that one could not with any certainty discuss “carotid artery syndromes” and their therapy unless referred to pathologically or radiologically proven cases. On the other hand, the inefficiency of arteriography in determining clinically significant collateral and contralateral blood flow to the brain was remarked and illustrated.

Their experience with anticoagulant and surgical management of these cases was outlined. The hazards of embarking on surgery without an arteriographic survey of the total problem were presented. The complicating factor of greatest importance was the coincident occurrence of intracerebral carotid as well as basilar-vertebral disease.

**A Controlled Clinical Trial of Anticoagulant Therapy in Cerebrovascular Disease**

John Marshall (London) described a clinical trial of anticoagulant therapy in cerebrovascular disease. The aim of the trial was to see if long-term anticoagulant therapy reduced mortality, prevented further strokes, and improved the functional capacity of the patients. One hundred and forty-two patients were in the trial, 71 on anticoagulant therapy and 71 as controls. There was no significant difference in the incidence of further non-fatal cerebrovascular accidents, but the incidence of fatal cerebral haemorrhage was significantly higher in the treated group, hence the trial was stopped.

**The Surgical Treatment of Primary Intracerebral Haemorrhage**

Wylie McKeith (London) presented a survey of 240 patients who had suffered a primary intracerebral haemorrhage, presumably due to rupture of an atherosclerotic vessel, and had been treated surgically. The very variable modes of onset were described and the difficulties of purely clinical diagnosis emphasised. Angiography was used in 167 cases but proved diagnostic in only 70% so that recourse to Burr holes, often followed by ventriculography, was often necessary to establish the diagnosis. The absence of bleeding into the ventricular system or subarachnoid space in 20% of cases was commented upon.

Patients were treated by burr hole and aspiration (104), aspiration and later craniotomy (73), and craniotomy alone (31). Of the 208 patients treated, 106 died within three weeks. Follow-up of the 102 survivors for periods of one to 10 years revealed 31 deaths. Ten were from progression of the disease in periods of one to three months and five from further cerebrovascular accidents. In the remainder the cause of death was either unknown or from some other disease. Of the 63 survivors, 25 were well and leading a normal life, 14 had minimal disability, 18 were moderately disabled, and six totally disabled.

Age did not influence the chance of survival but situation of haemorrhage did. Capsular haemorrhages rupturing into the ventricular system and basal ganglia carried an almost 100% death rate. Those patients in coma had a 100% mortality whilst in those who were alert the death rate was 23%.

Cerebellar lesions formed some 10% of these cases and although they posed difficult diagnostic problems they were amenable to surgical treatment.

He stressed the fact that a controlled trial of surgical and medical methods of treatment was necessary to establish the correct course of action in this pathological condition.