T I T L E

POSTERIOR CRANIAL FOSSA SPACE OCCUPYING LESIONS AT

KENYATTA NATIONAL HOSPITAL.

(1981 - 1989)

This thesis is submitted in part fulfillment for the Degree of Master of Medicine (Surgery) in the University of Nairobi, April 1990.

DECLARATION:

I hereby certify that this DISSERTATION is my own original work and has not been presented for a degree in any other University.

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THIS DISSERTATION HAS BEEN SUBMITTED FOR EXAMINATION WITH MY APPROVAL AS A UNIVERSITY SUPERVISOR.

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"An ailment which I will treat.
An ailment which I will contend.
An ailment not to be treated".

Ancient Egyptian medicine
(Papyrus).

A B S T R A C T.

A review of the clinical presentation, management and outcome of 46 patients, over a 9 year period who presented with posterior fossa space occupying lesions is presented.

Cerebellar space occupying lesions are the commonest in the posterior fossa. However, Brainstem gliomas are the most common specific lesion in the region (35% cases).

It is interesting to note that the youngest child diagnosed is 2 years as compared to other studies where much younger patients exist. The arguements for this anomaly and other age and sex characteristics are discussed.

17% of the patients presented with total blindness as compared to Nil in other recent reviews. This calls for corrective remedial measures urgently.

Tuberculomas have become rare and only one case over the last six years was seen. Generally, infective space occupying lesions account for 13% of cases.

Followup is disappointing. 50% (N=38) of the patients who left hospital were not heard from again. Their fate remains mysterious. Further, in those who turned up no quantifiable measure is made of their recovery.

It is recommended strongly that there is a need to have a standard, reproducible and repeatable measure that expresses the degree of recovery at a glance. Other suggestions for followup are presented.

C O N T E N T S

INTRODUCTION:

PREFACE:

I could see that the medical staff despaired and lost hope whenever the neurosurgeons diagnosed posterior fossa tumours in patients. Space occupying lesions in this part of the brain are dreaded by all, surgeons and medical staff alike. This was part of my short but very rewarding experience in the neurosurgery department. From it I got the inspiration and motivation for this study.

I have always believed that situations are not as gloomy as they might appear. Minor remedial measures may on occasion make a significant difference. For instance posterior fossa tuberculomas when diagnosed in good time are curable. Further, even if the situation was unfavourable it is only by carrying out further studies that it might become favourable.

How lethal are these tumours in our set up? Are these tumours really lethal as it is presumed? What about the trends in other set ups? These were some of the questions that really needed answers.

Basically, this was the framework and background that led to this study.

ACKNOWLEDGEMENTS

I wish to thank most sincerely Prof. G. M. Sande for his useful advise right from the start of this project. I am certain that without his invaluable advise this project might not have been a reality. To him I can only say that words cannot express my appreciation.

I wish to thank all the others involved in different but very important ways. Mrs Susan Thuo for the secretarial work. Mr Mwai, Medical Illustrations, for assisting with the diagrams, Records Department for the files and last but not least my family members for bearing with me during this write up. For the others who were involved but not mentioned, particularly the Nursing staff of Ward 13, I equally thank them all.

BACKGROUND INFORMATION

Neurosurgery is relatively recent not only in Kenyatta National Hospital but in Kenya. In 1969, Prof Ruberti, Consultant Neurosurgeon from the University of Padua in Italy, settled at the Nairobi Hospital and founded the first private neurosurgical practise in Nairobi. Concomitantly he served as an honorary neurosurgeon at the Kenyatta N. Hospital. Prior to this time Mr Peter Clifford looked after neurosurgery patients but in the Head and Neck Surgery sections.

In 1971, the neurosurgery section was officially inaugurated as a twelve bed unit in the Rhahimtula Wing of the hospital. Prof. Paxton, from the Department of Neurosurgery, University of Oragon U.S.A., worked for one year in the section on a full time basis while on sabatical leave. In the mid 70s' Mr Dar, neurosurgeon from India was engaged by the University. Hence becoming the first permanent neurosurgeon. He left behind 2 trained neurosurgeons when he departed for India in 1983. Unfortunately, one passed away in a road traffic acccident.

Presently the section has 32 beds and reviews all other neurosurgical cases in the hospital.

There are two full time neurosurgeons including a Professor of Neurosurgery. Two other Consultants are under training and it is hoped the number will go up.Supporting staff are adequate.

Presently about 162 minor cases and 101 major cases are operated yearly.

Ancillary services are in existent and operational with the occassional constraint of lack of supplies. Though Kenyatta N. Hospital has no CT Scan it has been available in the private sector since the mid 198os'.

Latest neurosurgical techniques are unavailable for instance equipment for stereotactic surgery, operating microscopes and carbondioxide laser systems

HISTORY OF NEUROSURGERY.

Mordern Neurosurgery is relatively recent having been developed mainly over the last hundred or so years(3).

However, early in life, in the Neolithic period (New stone age) there is archeological evidence that craniotomies were done(54). This age which dates back to about ten thousand years ago appears to have been a time for almost world wide trephination (primitive craniotomies). Evidence for this has been mainly from the recovered skulls which show large deliberate holes and the instruments that were used.

There is evidence that trephination was practiced in Africa including Kenya and Uganda(54). In Kenya it was prevalent mainly among the Kisii, oldest finding from Africa are from Sesebi in Sudan dating back to 1200 B.C.(54).

Indications for this surgery are not well understood but it appears to have been mainly used for chronic headaches, treatment of epilepsy and other convulsive disorders and to evacuate clots and debris following skull fractures. Another main indication was to allow the "Evil air to breathe out". In such cases the piece of bone removed was used by others as amulets(51). Hence it appears that magic and superstition were predominant.

In the intervening period upto around hippocrates time. (460-355 B.C.) it is evident that not much took place. Even the earliest medical papers (Smith papyrus) at about the 17th Century B.C. does not mention trephination. The first recorded writtings come to be seen in hippocrates treatises (460-355 B.C.). In addition he described certain neurosurgical conditions including head injury. Hence, it can be said that he was one of the pioneers of neurosurgery.

It was not until over the last Century that modern neurosurgery started evolving. Developments of other disciplines in particular Anaesthesia, X-rays, Physiological Anatomy and Microbiology, all of which evolved in the 19th Century, were crucial to this development.

General Anaesthesia was first employed by a dentist, Dr. Elijah pope of Rochester New York, in 1842 for Dental extractions. He used ether. In the same year Dr.Crawford Long used it successfully for the removal of a neck tumour. From 1846 fowards following its use and publicity by Morton and Warren in Havard Medical School its use spread widely (51). At around this time the major constraint in neurosurgery was functional anatomy because of the misconcept by many that the brain worked as a whole unit. It was not until the 1860's through the work of Pierre Paul Broca, a general surgeon, that the concept of division of functions in the brain

became clear. In 1870 two Germans Gustar and Eduard elicited motor activity in the limbs of dogs after electri-- cally stimulating parts of the brain. Few years later Professor Barthalow in Ohio proved the same in man. In 1891 the advent of antisepsis was added to these developments. In the late 19th Century neurosurgery became popular but soon this popularity waned because of high mortality and subsequent sequale. In 1893 starr reported that fifteen cases of posterior fossa tumours had been operated upon and only one patient recovered (55) oppenheim classified tumours of the cerebellum as inoperable and in 1902 reported a mortality of 71%.

Over the next few years prominent and dedicated surgeons continued to refine the art. Worthy of mention is Harvey cushinges (1869-1939) a surgeon at John Hopkins Hospital. He introduced several concepts and techniques which have withstood the test of time. For instance ventricular tap and the insertion of a Brain cannula into the ventricle during operation was advocated by cushinge By 1915 his neurosurgical operative mortality was 8.4%. DeMartel in 1931 insisted that the upright chair position with the head flexed was the most satisfact- ory position (55).

Advent of the x-ray in 1895 by Wilheim conrad Roentgen (1845-1923) perhaps was and still remains one of the greatest investigative discoveries (52). Today

skull x-rays are still important. In addition it widened the scope of neuro imaging research. In 1919 Dandy Edward (1886-1946) a surgeon at John Hopkins introduced pneumoven - triculography and pneumoencephalography. These were to be the backbone of neuro-imaging for many years to come. The 1920s saw the era of contrast neuroimaging. Jean sicard a french clinician developed myelography using lipiodol after accidentally discovering it was radio opaque. Cerebral angiography came about through the significant efforts of Antonio Caetano (1927). Despite the initial mortality and morbidity he perfected it well.

Recently, over the last few decades both investigative and operative neurosurgery have seen tremendous growth.

By 1972 computerised Axial tomography (C.A.T.) Scanning was being used in a few centres having been developed a little earlier (25). C.A.T. Scan has been replaced by the more refined computerised scans (C.T.Scans). Lesions are seen in it as alterations of normal density and interprated as Density Change (25). Modern scanners can discern differences in tissue density of less than 0.3%. Presently, CT scans have become the backbone of investigations in Neurosurgery. Nuclear magnetic resonance (N.M.R.) is becoming increasingly important. It is based on the concept of the behaviour of atomic nuclei in a static magnetic field and exposure of this to radio waves (9). Though the scans taken are similar to C.T. scans in

some instances delineation is more lucid. Other scans available are emission scans in which the source of irradiation is placed within the body and the detector is externally based. These rely on the radio isotope uptake of the particular tissues being investigated. Illustrations of these include the positron Emission Tomography (P.E.T). P.E.T. is based on the principal of positron emitting radionuclides being taken in by the organ under study and the distribution is externally monitored and computerised. Emission scans are mainly useful in investigating physiological functions rather than Anatomy.

Use of the operating microscope, microneurosurgery, is increasingly being used. Trauma is minimised and neurovascular structures visualised more accurately and efficiently (43).

More recently, the combination of stereotactic surgery has reduced mortality for deep intracranial space occupying lesions surgery to 0-2% (7,12,30,41). Intracranial tumours can now be wholly vaporised by a carbondioxide laser beam through a stereotactic approach under C.T. Scan guidance (41). Deep biopsies no longer need to be taken by the open method. Closed stereotactic approach enables biopsies to be taken accurately with almost no mortality (30). These newer Techniques besides

the reduced mortality have increased accuracy and because of the three dimensional orientation allow for complete removal of tumours.

FUNCTIONAL ANATOMY AND PHYSIOLOGY OF THE POSTERIOR FOSSA CONTENTS.

The contents of the cranium can be divided into two by the tentorium cerebelli. The Anterior and middle cranial fossa above and the posterior fossa below.

The posterior cranial fossa is the largest and deepest (46). Superiorly it is bounded by the tentorium cerebelli which anteriorly has a free margin and oval shaped aperture called the tentorium incisurae (46). Structurally the tentorium is a lamina of Duramater. Below, the floor is formed by parts of the occipital bone and is interrupted by another aperture, the foramen magnum. Laterally, it is bounded by bony relations mainly the occipital, parts of the temporal and parietal bones. Like elsewhere in the skull all this bone is lined on the interior surface by Duramater.

The parts of the brain in the fossa are the Brainstem (Mid brain medulla and pons) and the cerebellum (Fig.1). The superior colliculi of the Mid brain lie at the edge of the incisurae and hence the part of the Midrain below this lies in the posterior fossa.

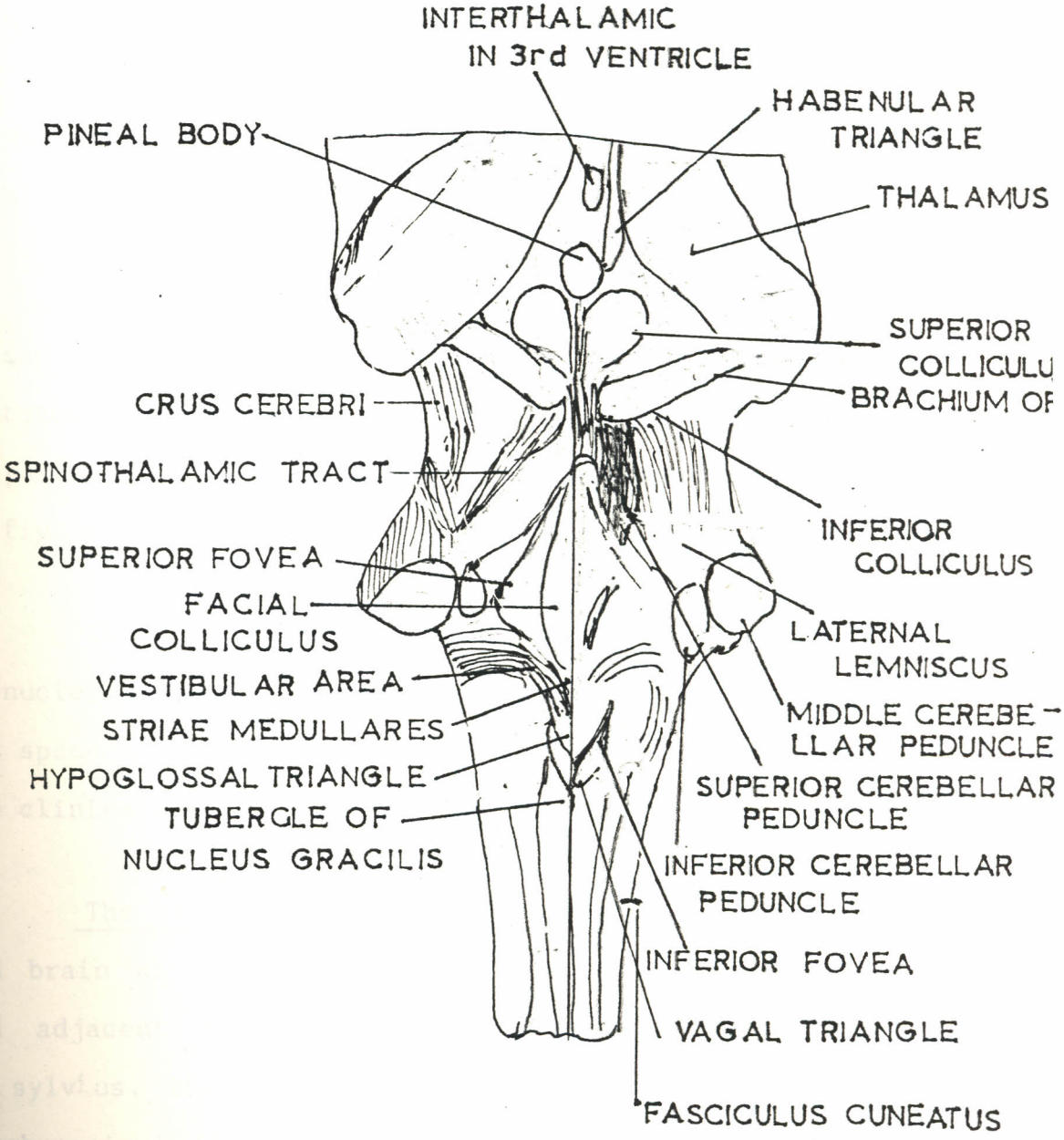

Fig. 1: Posterior view of the brainstem .

The anterior part of the superior surface of the vermis also lie in the region of the incisurae and other sturcturs lying in this region are the posterior cerebral artery and anterior choirodal artery (36). The Medulla exits through the foramen magnum where it becomes continous with the spinal cord.

Besides, the coverings of the brain (Duramater, arachnoid and piamater the external surface of the brain contains blood vessels which can also be afflicted by pathology. The distribution of the vessels is depicted in fig. 2.

It is important to appreciate the close relationship of nuclei and tracts in this region because when afflicted by space occupying lesions they form the basis of the clinical signs and symptoms of the lesion.

The third cranial nerve nucleus lie in the Mid brain at about the level of the superior colliculi and adjacent to the ventral aspect of the aqueduct of sylvius. the aqueduct being the canal through which cerebrospinal fluid is conveyed from the third ventricle to the fourth ventricle. Nuclear and infranuclear lesions are manifested ipsilaterally. Except for the levator muscles of both eyes which are innervated by one subnucleus and the third Nerve parasympathetic centre (Edinger-Westphal

subnucleus) which innervates bilaterally. The superior rectus muscle is innervated contralaterally. After emerging from the Mid brain the third nerve is closely related to the tentorial incisurae. It can be compressed against the edge of the incisurae or become ischaemic following the compression of its blood supply. (The posterior cerebral artery). In the brain stem there is a gaze centre which relays to the 3rd Nerve and sixth nerve its main role is to enable maintance of gaze in a particular direction (40). When afflicted the eyes may be unable to sustain a gaze (gaze paresis) and this may appear as irregular Jerky movements when trying to maintain gaze (gaze paretic Nystagmus) .

Paresis or paralysis of any of the ocular motor muscles (except the lateral rectus and superior oblique muscles) implies a third nerve lesion. When the parasympathetic centre is also involved then pupillary accommodation and light reflex responses are affected. Normally, this nucleus causes pupillary miosis and is inhibited actively by the reticular activating system (Pons & Medulla). In lesions of the latter e.g. pontine haemorrhage disinhibition occurs and pinpoint pupils are seen .

The fourth cranial nerve nucleus lies below the third nerve nucleus at the level of the inferior colliculi. It decussates while still in the Midbrain

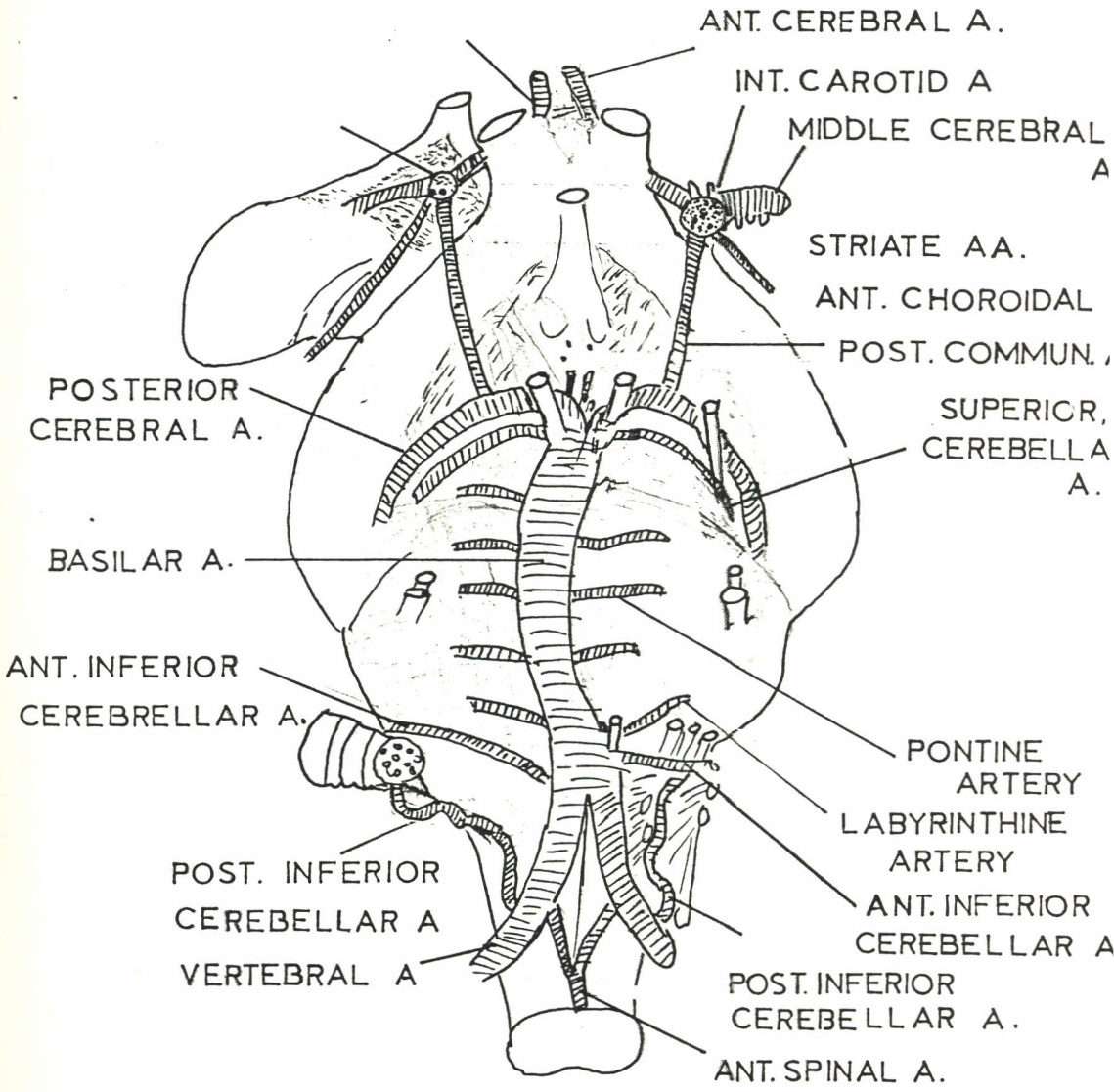

Fig. 2: Circle of willis and arteries of the brainstem.

and therefore innervates (superior oblique muscle) contralaterally (Fig.3).

The fifth nerve nucleus is long and is located along the entire brainstem. The mid brain part coordinates facial proprioception, pontine part ordinary sensation and motor, while the medullary part is concerned with pain. All the three exit together from the pons (Fig.2). The individual components may be affected alone. For instance medullary nucleus lesions cause only pain and temperature sensory deficit which is of the "onion-skin" pattern on the face. Recognition of sensory deficits are discussed elsewhere. However the corneal reflex as elicited by gentle stroking of the cornea with a wisp of moistened cotton is a sensitive index of trigeminal nerve impairment (Setti, 1985).

The six nerve nucleus lie in the pons near the midline and dorsally. It forms a visible swelling on the floor of the fourth ventricle. The facial colliculus (fig 1). The fascicular part of the seventh nerve pass over this nucleus in the region of the colliculus. Both nerves can be affected in lesions around this region. The nerve has a relatively long intracranial course and is stretched and distorted when intracranial pressure is increased. It is afflicted by several other lesions

and when the lateral rectus muscle it innervates is paretic, it is taken as more of a false localising sign.

The Seventh Nerve has three components (Motor, secreto motor and taste). The Motor nucleus lie deep in the floor of the ventricle in the inferior part of the pons. The nervus intermidius which moves with the seventh nerve for parts of its course carries fibres from the other two nuclei. The secreto-motor (floor of the mouth, palate, nasopharynx, paranasal sinuses, nasal cavity and lacrimal gland) has its nucleus lateral to the seventh nerve nucleus, the superior salvatory nucleus. Lying further lateral to this nucleus is the tractus solitarius which is sensory to the anterior $\frac{2}{3}$ tongue for taste. The descending cortico-bulbar fibres innervating the fascial musculature innervate it bilaterally for the forehead musculature. Hence, unilateral supranuclei lesions only affect the contra-lateral lower fascial muscles. Ipsilateral fascial paresis or paralysis imply nuclei or infranuclei involvement. Loss of taste in the anterior $\frac{2}{3}$ tongue and reduced tear secretion (Schirmers test positive) imply that the superior salivatory nucleus and tractus solitarius are also involved or the seventh nerve could be involved more peripherally e.g. cerebellopontine angle lesions. Hyperacusis (paresis of stapedius) occurs when fascial nerve is afflicted proximal to the geniculate ganglion.

The eight nerve cochlear nuclei responsible for sound reception lie in the inferior cerebellar peduncle..

The vestibular nucleus concerned with balance lies partly in the pons and medulla at the lateral angle of the fourth ventricle.

The ninth nerve is mainly secreto motor and its nuclei are inferior and continous with the secreto motor of the seventh nerve as the inferior salivatory nucleus and tractus solitarius respectively. Its motor nuclei (Nucleus Ambiguus) innervates only one muscle of which lesions are difficult to detect clinically. Some of its afferent fibres mediate gag reflex but the reflex can be intact even when the nerve is affected.

The tenth nerve has its secreto-motor nuclei continous with those of the ninth and seventh nuclei secreto-motor components. In addition the tenth nerve nucleus contains cell bodies that are motor efferent to smooth muscles of the respiratory and alimentary systems (dorsal nucleus). This nerve supplies the striated muscles of the soft palate, pharynx, oesophagus and larynx. The nucleus for striated muscles is the nucleus ambiguus situated in the region of the inferior cerebellar peduncle. The nerve fibres from this nucleus exit through the eleventh nerve but later join the tenth nerve. Paresis of these muscles explain the regurgitation of swallowed fluids through the nose and in addition the absent gag reflex in lesions of the nerve. Gag reflex is a satisfactory indicator of the tenth nerve function.

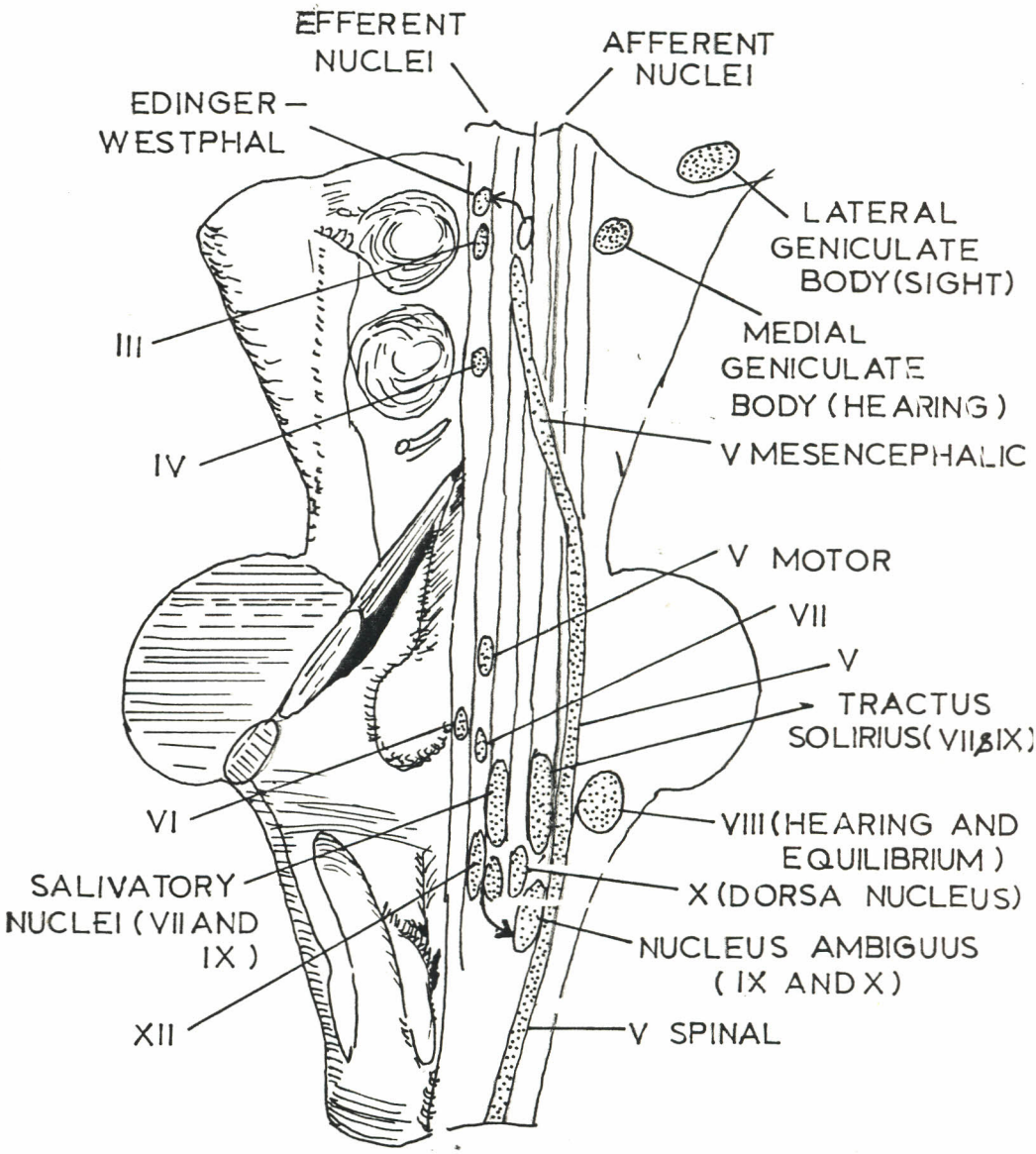

Fig. 3: Dorsal View of Brainstem Depicting Cranial nerves Nuclei

The eleventh nerve cranial fibres once outside the skull are all taken by the vagus as above, the spinal roots (C_2 C_3 C_4) goes on to supply the levator scapulae and sternomastoid muscles.

The twelfth nerve nucleus lies in the lower medulla in the inferior angle of the fourth ventricle just lateral to the median sulcus of that ventricle and emerge from the medulla as a vertical line of rootlets. It goes on to supply the ipsilateral tongue muscles both extrinsic and intrinsic except one extrinsic Fig. 3 depicts the distribution of these cranial nerves.

Cranial nerve nuclei and their fasciculi only form part of the structure of this region. In addition there are other nuclei and tracts that traverse the brainstem.

The corticospinal fibres (pyramidal fibres) are descening fibres from the motor cortex (pre-central gyrus). They enter the midbrain ventrally as the cerebral peduncles (crus cerebri) and are edged here on the outside by the incisurae tentorium borders. The fibres traverse the rest of the midbrain in the central aspect. In the pons the fibres are interspersed as small bundles and emerge out of the pons as the pyramids in the Medulla (Ventral aspect). About 80%

of the fibres decussate at the lower end of the medulla to enter the spinal cord as the corticospinal tracts. Hence, corticospinal lesions above this level manifest contralaterally. Whereas when most cranial nerve nuclei are inflicted the manifestation is ipsilateral. This is the basis of crossed paralysis an important feature of brainstem lesions. Unilateral pyramidal tract lesions are seen as hyperreflexia and hyptertonia. Sustained clonus which is the rythmic involuntary contractions of muscle induced by sudden stretching of a spastic muscle may be present. Together with an upgoing plantar response (Babinski's sign) they are diagnostic of pyramidal tract lesions.

Ascending fibres through the brain stem mainly comprise of sensory afferents. The posterior spinal column fibres (fasciculus gracilis and fasciculus cuneatus) at the lower medulla (just below inferior angle of 4th ventricle) form two swellings dorsally. The cuneate tubercle and gracile tubercle respectively. The fibres ascend from this nucleus after decussating ventrally. These fibres are afferent for vibration sense, muscle and joint position and appreciation of

position in space. The other afferent tract, the spinothalmic tract, responsible for crude pain, temprature and touch traverse the brainstem as the medial leminiscus in the lateral aspects of the medulla having decussated earlier in the spinal cord. The two tracts join in the mid brain to terminate in the thalamus. Lesions of the posterior column tracts manifest as loss of position sense, loss of vibration sense. and While those of the spinothalamic are seen as loss of sensation for pain and temprature differences. Depending on the level of the lesion the fascial involvement might be contralateral or ipsilateral. For instance in Tegmentum of medulla lesions, crossed sensory disturbance is seen, facial is ipsilateralto the lesion and the rest of the body it is contralateral. This is because fifth nerve nuclear and infranulcear lesions are ipsilateral. When the lesion is above the medulla when the fifth nerve fibres have decussated then the manifestion will be contralateral just like the rest of the body.

The reticular formation or reticular activating system is more of a physiological rather than antomical description). It comprises of cell bodies dispersed in the brainstem with some

forming discrete nuclei. They receive impules from sensory afferents and have both ascending and descending efferents. It is believed to be responsible for conciousness and Alertness. In addition the centres for respiration and cardiovascular regulation are lodged in here. In vegetative states where cortical pathways are destroyed by disease but the brainstem remains intact vital functions can continue and the body assumes characteristic postures, decerebrate rigidity or decorticate rigidity respectively (Patton, 1976) Decerebrate rigidity occurs when lesions affect cortical fibres at the upper border of pons and causes sustained contractions of all the extensor muscles (The antigravity muscles). Hence, legs, arms and neck are hyperextended. When the lesions are higher, e.g. the internal capsule, then decorticate rigidity results in which extensor rigidity occurs in the legs but with moderate flexion (Patton,1976). Asymmetric decorticate or decerebrate situations are more likely when transtentorial herniations (rostral-caudal herniations) are present. In addition decerebrate rigidity can occur transiently in increased intracranial pressure (see below).

There are other nuclei and fibres within the brainstem concerned with motor movement. this is the extrapyramidal system.Its nuclei include the red nucleus in the midbrain, olivary nucleus which forms a swelling lateral to the pyramids in the medulla and the vestibular nucleus. This group of nuclei and fibres along with other nuclei out of the brainstem help to ensure fine movements. Lesions often result in tremors and incoordination .

Dorsal to the brainstem in the posterior fossa is the cerebellum. It comprises of two hemispheres connected in the midline by the vermis. The cerebellum is connected by three preduncles to the brainstem. The superior peduncle (comprising mainly of efferent fibres enters the midbrain). The middle peduncle consists mainly of ponto-cerebellar fibres from pontine nuclei of the opposite side and is therefore connected to the pons. The inferior peduncle mainly containing afferent fibres joins with the medulla. The cerebellum is the central intergrative organ that controls muscle coordination. The vermis is principally concerned with maintenance of posture and equilibrium therefore it influences mainly the truncal musculature. Lesions

of the vermis are characterised by truncal ataxia.. The cardinal signs and symptoms of cerebellar disease are related to muscular incoordination, disturbance in posture and gait. Muscular hypotonia and disordered equilibrium. There is impairment of judgement of distance, range, speed and force of movement. For instance in the finger to nose test overshooting occurs and is followed by overcorrection. This is manifested as intentional tremors . Such incoordination affects speech giving rise to ataxic dysarthria. Ocular muscles are equally affected resulting in cerebellar nystagmus (ocular dysmetria). These manifestations are ipsilateral to the side of the lesion.

The fourth ventricle is the ventricle of the posterior fossa. From the midbrain the aqueduct opens on the dorsal surface of the pons and upper medulla as a cavity of which the roof has no brain substance and is only lined by ependyma and piameter. Ependymal cells are the cells which line the cavities of the brain and the spinal cord and are bathed in cerebrospinal fluid (c.s.f.) (31). In the lower medulla the ventricle closes in as the central canal which is continous with that of the spinal cord. The roof is covered by the cerebellum. The cavity

is diamond shaped and in its lateral recess which is around the inferior cerebellar peduncle has an opening, the foramen of luschka. This foramen opens just behind the eighth nerve into the cisterna pontis (subarachnoid space just ventral to the pons and medulla). The lower margin of the cavity has another median slit opening, the foramen of magendie. This one opens into the cisterna magna (cisterna-cerebello-medullaries) which is located between the undersurface of the cerebellum and the posterior surface of the medulla. These apertures are the only normal exit for c.s.f. from the cavities into the subarachnoid space where its absorption occurs in the arachnoid villi. Obstruction of these apertures results in increased accumulation of c.s.f. a condition referred to as hydrocephalus (14). An important content of the fourth ventricle is the choroid plexus (fig.2) which secretes c.s.f. The plexus consists of invaginated folds of piamater that penetrate into the interior of the ventricles and is rich in capillaries (31).

Cerebello-pontine angle is the name given to the area where the cerebellum adjoins the pons. It is of particular interest because the fifth and seventh to eleventh nerves pass around this region.

Eighth and seventh nerves along with the nervous intermidius pass into the internal a coustic meatus. Depending on the size of a lesion they are affected to varying degrees and this forms a basis of diagnosis of the lesions.

THE PATHOLOGY OF INTRACRANIAL SPACE OCCUPYING LESIONS IN THE POSTERIOR FOSSA

Perhaps, the most important concept in intracranial space occupying lesions is the monroe-kellie doctrine which emphasizes that because the cranium is rigid and therefore of constant volume, Any increase in volume of its contents results in reduction of volume of other intracranial compartments. Initially the fluids (C.S.F. and blood) and later the displacement of the solids (14).

Besides the actual involvement of the neuronal tissue in the pathologic process which will result in signs and symptoms of deficits of that particular tissue other effects are seen. Most important being increased intracranial pressure, brain oedema and ischaemia.

INTRACRANIAL PRESSURE

Space occupying lesions (S.O.L.s) in the posterior fossa will cause increased I.C.P. when they block the C.S.F. flow (aqueduct and 4th ventricle) pathways. Depending on the site rather than nature or size obstruction can occur early or late. For instance vermis S.O.L.s because of the close relationship

to the aqueduct will cause early whereas endophytic brainstem S.O.L.s will cause late obstruction.

Normally, any increase in I.C.P. when the subarchnoid pathways are patent is transmitted equally and rapidly throughout the spaces (Pascal's law). Pascal's Law states that a force applied to any portion of the surface of a contained volume of liquid is exerted unchanged on all other portions within the container (26).

When C.S.F. outflow is blocked in the posterior fossa the initial impact is in the supratentorial space because the C.S.F. produced there has no outlet. The lateral ventricles are inflated by C.S.F. Such increased accummulation of C.S.F. is reffered to as hydrocephalus and when secondary to obstruction as in this case, obstructive hydrocephalus. In the uniform distribution of this pressure supratentorially the vaginal sheaths surrounding the optic nerve are also affected (40) Venous congestion, abnormal axoplasmic flow and axonal swellings result. This manifests initially as papilloedema and later as optic atrophy, hence total blindness. This is one of the most important causes of blindness in increased I.C.P.

Further increases in I.C.P. result in displacement of brain tissue (herniations). The typical pattern of herniations in posterior fossa S.O.L.s is transtentorial (herniations) in which the brain is forced through the central incisurae cerebelli. It is central because there is no lateralisation of mass effects in the supratentorial space by posterior fossa S.O.L.s (Howell D.A., 1961). Lessly, Bitemporal Uncus herniations may also occur.

Central herniation features as the downward movement of the diencephalon through the incisurae. Diencephalon is the supratentorial part of the brain excluding the cerebral hemispheres. Howell D.A. showed that such herniations result in longitudinal brainstem compression and buckling. He stressed that the lower brainstem is unable to move downwards freely as the upper brainstem because the upper cervical dentate ligaments are unyielding. This results in longitudinal compression and backward angulation or buckling of the brainstem. The final outcome is paralysis of the mid brain tegmentum. This phenomena is responsible for the signs and symptoms seen in brainstem compression in these cases.

Cardinal features of this syndrome as reviewed by Donald O. Quest (1985) and Howell D.A. include

dilated pupils, progressive impairment of consciousness decerebrate rigidity spontaneously and commonest during peak I.C.P. increases or in response to painful stimuli. Deterioration in consciousness is progressive with herniation terminating in coma and death.

Vital signs are variably affected partly because of brainstem distortion and buckling and partly because of the herniating cerebellar tonsils compressing the medulla. Commonly increased blood pressure, bradycardia and cheyne-stoke breathing occurs. Exact inter-relationship of vital signs to herniation is still unclear according to Donald Quest.

It can be appreciated that the presence of the S.O.L. in the posterior fossa results in further unyielding during downward displacement in particular large cerebellar tumours by impacting in the foramen magnum. This presumably auguments the buckling.

ISCHAEMIA AND OEDEMA

During herniations compression of blood vessels occurs. For instance the posterior cerebral artery and lessly anterior choirodal artery may be compressed at the edge of the incisurae if it is complete

infarction of the occipital cortex and hence total blindness may result (Donald Quest 1985)

Rapidly expanding S.O.L.s compress adjacent blood vessels and ischaemia of adjacent brain tissue could occur. James D. W. has stressed that in such lesions local tissue pressure is higher than the overall intracranial compartment but he states that the clinical significance of this is unclear. Such ischaemia at least results in further deterioration.

The presence of the S.O.L. in particular neoplasms gives rise to surrounding oedema. Nerve tissue respond to any insult by oedema. Such oedema play a significant role by augumenting the compressive effects of S.O.L. Ischaemia when it occurs also causes surrounding oedema and therefore additive effect.Treatment of this oedema induced by neoplasms is steriod therapy. This is an important part of the management.

AIMS

To study the clinical presentation, management and outcome of posterior fossa space occupying lesions as seen at the Kenyatta National Hospital.

OBJECTIVES

The main objectives of this study were to:-

1. Study the age and sex distribution of the patients.

2. Study the signs and symptoms at the time of presentation.

3. Study the histopathological diagnosis of the space occupying lesions.

4. Study the treatment and outcome.

METHODOLOGY AND MATERIALS

A questionnaire was devised bearing the objectives in mind.

The neurosurgery operating records were used to identify operated cases from 1981. The records

department also identified posterior fossa lesions according to their filing system. The files were perused and data collected. In 1989 the patients were also examined and when possible seen in the wards and clinic.

Standard methods were used to elicit clinical signs whenever this was possible. At the end of the study data collected was analysed and interprated.

RESULTS

1. Period covered 1981 - 1989

2. Sample size = 46 patients.

3. Patients excluded from study (lost records or other vital information)= 13 patients.

AGE AND SEX CHARACTERISTICS

Of the 46 patients seen just slightly less than a half were females. Their ages ranged from 2 years to 71 years (Table 1). The peak incidence was in the first decade of life. After 14 years of age they are less common accounting for less than 50% of cases (Fig. 5).

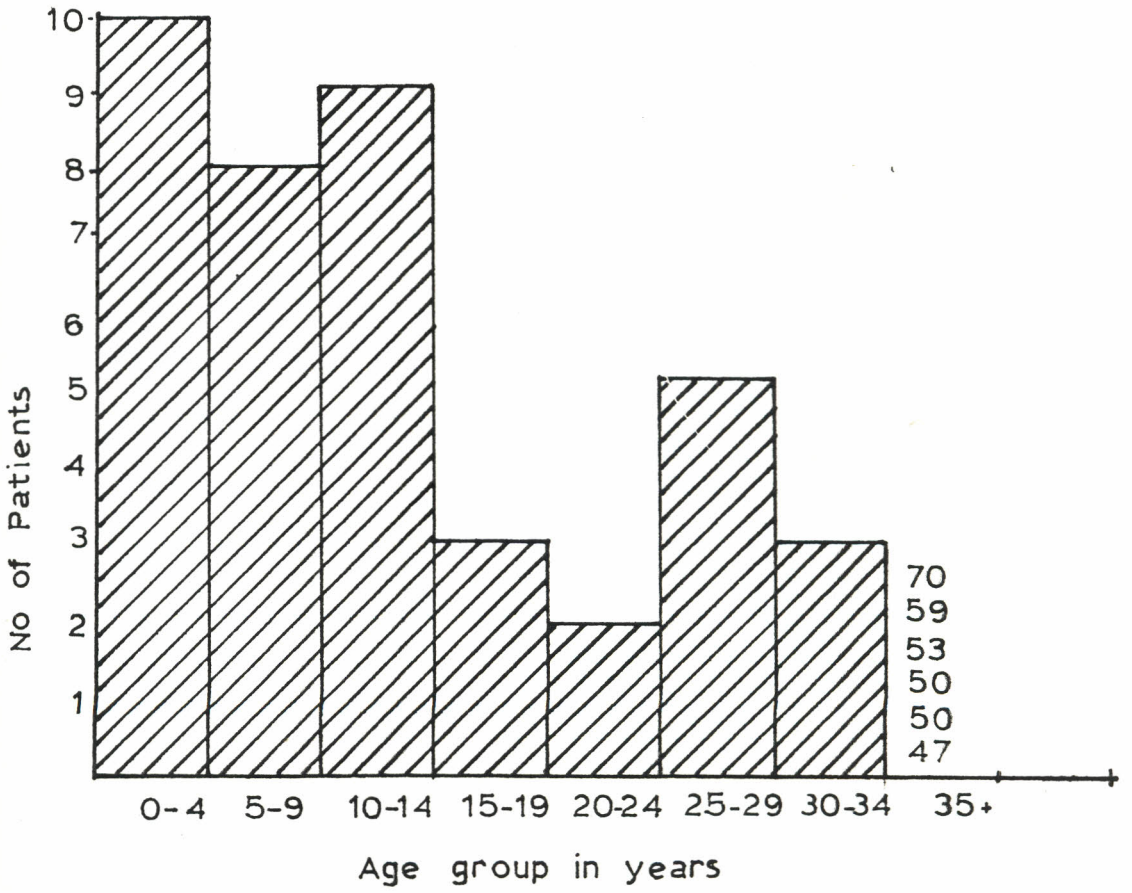

Fig 5: Frequency distribution by age group and sex (>35yrs by age only) of Posterior fossa S.O.Ls (N=46)

TABLE 1

DISTRIBUTION OF SPACE OCCUPYING LESIONS (S.O.L.s) IN THE POSTERIOR FOSSA BY AGE AND SEX. (N=46)

AGE GROUP (Years)	SEX		TOTAL
	Males	Females	
0-4	8	2	10
5-9	4	4	8
10-14	8	1	9
15-19	2	1	3
20-24	2	0	2
25-29	2	3	5
30-34	2	1	3
35+	3	3	6
TOTAL	31	15	46

The space occupying lesions were commonest in the cerebellum (Table 2) and all occurred before 29 years of age except one. The one patient was a 50 year old man with a simple arachnoid cyst. Brain-stem S.O.L.s were commonest before 39 years and only 2 were above 50 years. Cerebellopontine (C-P) angle S.O.L.s appear to be adulthood lesions. The age patterns are depicted in Figure 6.

TABLE 2

AGE GROUP DISTRIBUTION OF POSTERIOR FOSSA S.O.Ls BY SITE (N=46)

AGE GROUP (Years)	SITE			TOTAL
	Cerebellum	Brainstem	Cerebello-pontine angle	
0-9	12	6	0	18
10-19	7	5	0	12
20-29	4	1	2	7
30-39	0	2	1	3
40-49	0	0	1	1
50-59	1	2	1	4
60+	0	0	1	1
TOTAL	24	16	6	46

Overall male to female ratio was about 2:1. Male preponderance was particularly marked in cerebellar astrocytomas 5:1. In brainstem S.O.L. it was 3:1. Medulloblastomas 2:1. C-P angle was 1:6.

CLINICAL PRESENTATION

Majority of the patients presented with symptoms noticed over the last few months. 40% over 2-3 months and 80% less than 7 months. This excludes

cerebellopontine angle in which patients had had symptoms for greater then 2 years. There was no significant difference on the duration of symptoms at presentation between cerebellar and brainstem S.O.L.s (Table 3).

TABLE 3

DURATION OF SYMPTOMS IN WEEKS OF PATIENTS PRESENTING WITH BRAINSTEM AND CEREBELLAR S.O.L.s (N=34)

SITE	DURATION OF SYMPTOMS (WEEKS)				TOTAL
	<9	10-19	20-29	30+	
Brainstem	4	4	3	2	13
Cerebellum	6	5	7	3	21
TOTAL	10	9	10	5	34

$X^2 = 0.42$ $P > 0.05$

df = 3 Level of significance = 0.05

Difficulty in walking or deteriorating milestones was the commonest complaint at the time of presentation (Table 4). Only one patient with cerebellopontine angle S.O.L. did not have difficulty in walking. Visual disturbance was common and out of the 24 patients 8 had no bilateral perception of light on admission. This implies that 17% (N=46) of the patients presented with total blindness amongst other complaints. Of these 8, 2 each had astrocytoma and medulloblastoma, one cerebellar tuberculoma and one a developmental abnormality in the cerebellum. The remaining 2 had brainstem S.O.L.s. Other complaints of visual disturbance were mainly blurring of vision (mistiness) and seeing double. Of the 4 patients with vertigo only 2 had cerebellopontine S.O.L.s (Table 4). Vomitting was not common in brainstem S.O.L.s as in cerebellar S.O.L.s, (Table 5). Commonest mental change was apathy seen in 6 patients. In the other 2 with mental change one had features of irritability and the other difficulty in mental recall.

TABLE 4

DISTRIBUTION OF MAIN OBJECTIVE SYMPTOMS AT THE TIME OF PRESENTATION IN POSTERIOR FOSSA S.O.L. PATIENTS (N=46)

SYMPTOMS	NUMBER OF PATIENTS	% TOTAL
1. Difficulty in walking/ Deteriorating mile-stones	32	70
2. Headache	29	63
3. Visual disturbance	24	52
4. Vomitting	23	50
5. Mental changes	8	17
6. Fits	7	15
7. Altered level of consciousness	7	15
8. Faecal/urinary incontinence	5	11
9. Increased head circumference	6	13
10. Vertigo	4	9

TABLE 5

DISTRIBUTION OF THE COMMONEST OBJECTIVE SYMPTOMS ACCORDING TO SITE AND BY PERCENTAGE IN SITE IN PATIENTS WITH POSTERIOR FOSSA S.O.L.s

SYMPTOMS	BRAINSTEM (N=16)	CEREBELLUM (N=24)	CEREBELLO PONTINE (N=6)
1. Difficulty in walking/ deteriorating milestones	8(50%)	12(50%)	5
2. Headache	12(75%)	17(71%)	4
3. Visual Disturbance	6(38%)	18(75%)	6
4. Vomitting	4(25%)	17(71%)	2

10 patients of those examined for increased intracranial pressure had no evidence of increased pressure. Slightly less than a half of the patients with increased pressure had neck stiffness (Table 6). Scalp tenderness occurred but it was less common. 5 patients with features of increased pressure had no headache and 18 had no vomitting.

TABLE 6

DISTRIBUTION OF SIGNS OF INCREASED INTRACRANIAL PRESSURE BY SITE IN PATIENTS WITH POSTERIOR FOSSA S.O.L.s (N=41)

Sign	SITE			Total Number of Patients	% Total
	Brainstem	Cerebelum	C-P Angle		
1. Papilloedema	7	20	5	31	76
2. Reduced Consciousness Glasgow coma scale					
10-12	2	2	-	7	17
9-7	1	2	-		
3. Neck stiffness	4	13	3	19	46
4. Scalp Tenderness	2	4	1	7	17

The characteristc cross paralysis of posterior fossa S.O.L.s was seen only in 3 patients. All patients with cranial nerve involvement had at least an ipsilateral lesion with or without a contralateral cranial nerve lesion. Pyramidal tract and sensory involvement was more inconsistent either being ipsilateral or bilateral.

This being contrary to the expected contra-lateral lesion. In this study partial or complete cranial nerve involvement were categorised as one (Table 7)

TABLE 7

DISTRIBUTION OF CRANIAL NERVE INVOLVEMENT IN PATIENTS WITH POSTERIOR FOSSA S.O.L.s BY SITE (N=41)

Cranial Nerve	SITE Brainstem	Cerebellum	C-P Angle	Number of Patients	% Total
1. Oculomotor Nerve					
Unilateral	3	2	-	5	12
2. Trochlear nerve					
unilateral	1	1	-	3	7
Bilateral	-	1			
3. Trigeminal Nerve					
Unilateral	7	7	3	18	44
Bilateral	-	1	-		
4. Abducent Nerve					
Unilateral	7	7	3	17	41
5. Facial Nerve					
Unilateral	8	6	5	19	46
6. Vestibulo Cochlea Nerve					
Unilateral	1	2	5	8	20
7. Vagus Nerve	2	2	1	5	12
8. Hypoglossal Nerve					
Unilateral	2	2	-	4	10
9. Accessory Nerve	-	1	-	1	2

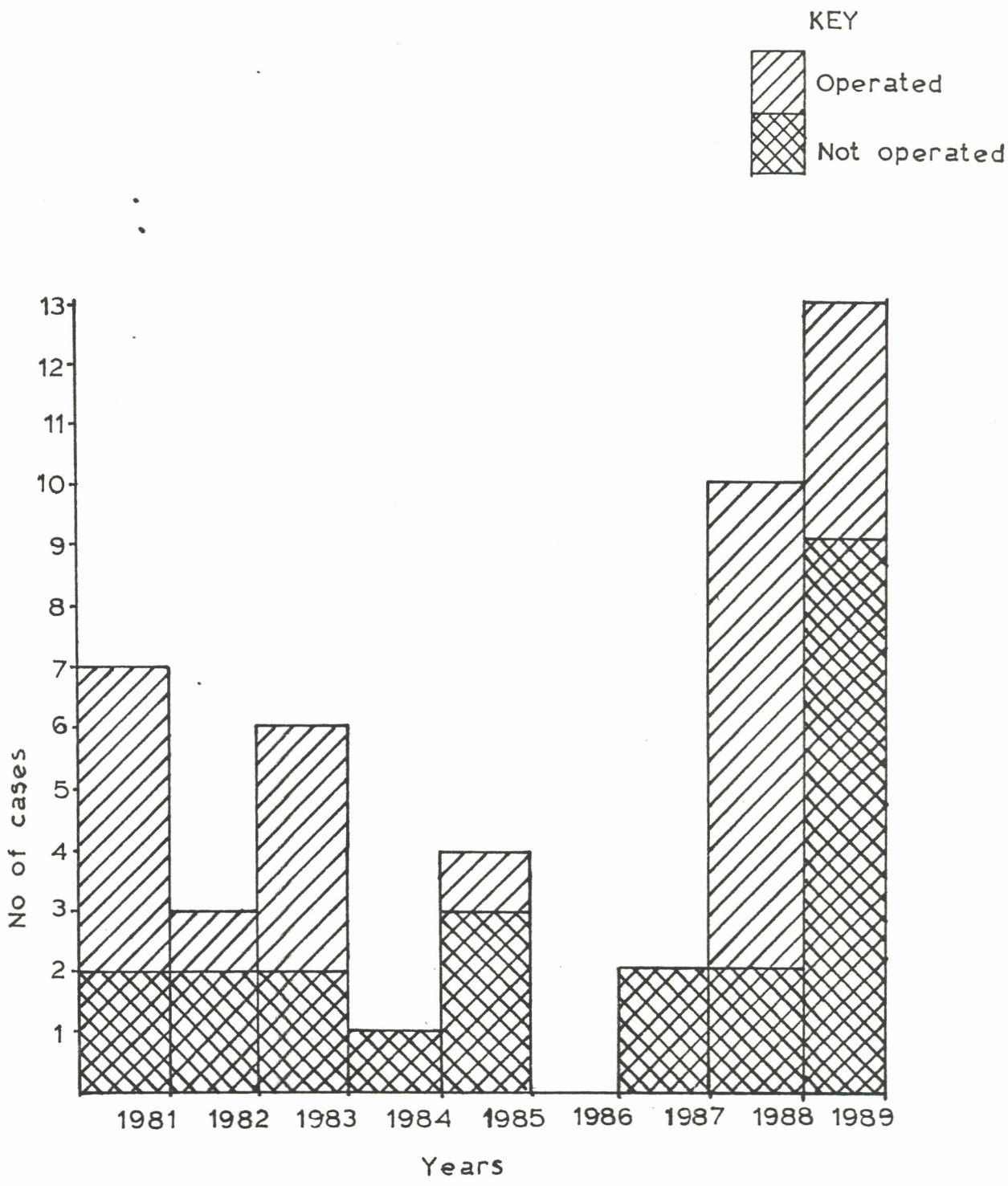

Fig. 7: Yearly distribution of Posterior fossa S.O.Ls according to age group and site (N= 46)

TABLE 8

DISTRIBUTION OF PYRAMIDAL TRACT (MOTOR), SENSORY AND CEREBELLAR SIGNS IN PATIENTS WITH POSTERIOR FOSSA S.O.L.s BY SITE (N=39)

Sign	SITE			Total Number of Patients	% Total
	Brain stem	Cere-bellum	C-P Angle		
1. Pyramidal Tract					
Extremity parésis with hyperreflexia.					
Hemiparesis	5	1	3		
Quadraparesis	3	-	1	16	41
Paraparesis	2	1	-		
Extremity paresis with hyporeflexia					
Hemiparesis	1	3	-		
Quadraparesis	1	2	-	9	23
Paraparesis	1	1			
Positive Babinski's sign					
Unilateral	8	7	4		
Bilateral	2	1	1	24	62
2. Sensory Tract					
Hemisensory loss	3	-	1	4	10
Disturbed postural sensibility (unilateral)	2	-	2	4	10
3. Ataxia					
Extremities	4	13	4	32	82
Truncal	2	8	1		
4. Nystagnus (All Types)	5	20	3	28	72

65% of the patients had cranial nerve involvement with one or more cranial nerves involved. It is interesting to note that one patient had only bilateral trochlear nerve involvement at the time of presentation. 34% of patients had 3 or more cranial nerves involved. V, VI and VII nerve were most commonly afflicted.

Incoordination as manifested by ataxia was the commonest peripheral sign (Table 8). It was not restricted to cerebellar lesions only. Positive Babinski's sign was present in over half the patients including those with no pyramidal tract deficiency. Sensory deficit was less common. 8 patients including one with C-P angle S.O.L. had neither pyramidal and sensory tract nor cerebellar signs. None of the patients with cerebellar S.O.L.s had any sensory deficit.

DIAGNOSIS

In this study 19 patients were diagnosed by C.T. scan beginning from 1988. Since then there is a definite increase in the number of patients diagnosed. (Fig 7) of the 13 patients diagnosed by C.T. Scan who had histology results only 5 correlated. One with astrocytoma and 4 with medulloblastoma. In the rest the diagnosis was wrong. Because most of the time some

of the films were missing and because of the small sample size (only 13 with histology) the scans were not analysed in further detail.

Ventriculography was performed in 16 patients using conray 280. 8 of the patients were subsequently operated and in all a space occupying lesion was found as suspected.

Carotid angiography was performed in 3 patients In.2 of them who were subsequently operated it had given misleading results. In one it had given the impression of a suprasellar S.O.L. and in one detected no abnormality but in both infratentorial tumours were found in theatre.

Vertebral angiography was done in one patient in whom it showed features of brainstem tumour.

TREATMENT AND OUTCOME

The main stay of treatment comprised of surgery (either excisional or diagnostic biopsies), V-P shunt, radiotherapy, chemotherapy or various combinations of these. Hence about 11 different modes of therapy were available. (See appendix 1). The poor follow up in this study (Table 10) makes correlation of treatment and outcome unreliable.

All S.O.L.s that were found to be arising from the brainstem were not operated. Either radiotherapy alone or with concomitant insertion of a V-P shunt. 7 patients with brainstem tumours were not given any definative treatment. Because of their poor general condition. All patients on admission were given any supportive treatment necessary. In all, 27 patients were operated and 2 of them had no histology results.

Like seen in Appendix one the number of procedures were numerous and numbers small and hence no further analysis was carried out.

The most common histological type was medulloblastoma. 17% (N=46) of all patients (Table 9). Since 1981 only 4 cases of confirmed tuberculomas have been seen at Kenyatta National Hospital. 3 of them between 1981 and 1983 and one in 1988.

TABLE 9

DISTRIBUTION OF POSTERIOR FOSSA S.O.L.s BY HISTOLOGY (N=25)

HISTOLOGICAL TYPE	NUMBER OF PATIENTS
1. Medulloblastoma	8
2. Astrocytoma	6
3. Tuberculomas	4
4. C-P angle neuromas	2
5. Developmental abnormality cysts	2
6. Others	3

Of the others one was hydatid cyst found in a patient from Lodwar, one a non-specific inflammatory lesion and one a simple arachnoid cyst in the cerebellar region. Out of the 6 patients with confirmed astrocytoma 4 were grade I and the rest grade II.

All the patients with medulloblastoma and astrocytoma except 2 were under 14 years of age of the 2 one was 18 years (medulloblastoma) and the other 25 years (astrocytoma). 3 of the 4 patients with tuberculoma were between 11-14 years and one was 29 years old.

17% of the total number of patients died while still in the hospital (Table 10). About 50% of the patients did not attend the clinic even once. Their fate is therefore unknown. Majority were those with brainstem tumours. Of the two patients followed up longest one had tuberculoma and had completed treatment 4 years earlier during the last follow up. The other had astrocytoma grade I and was inserted a V-P shunt, partial resection done, chemotherapy and radiotherapy administered.

5 patients died soon after operation and hence the operative mortality was about 19% (N=27). 2 of the patients had brainstem tumour, with increased

intracranial pressure and had been fixed V-P shunts for decompression. One patient had excisional biopsy of a cerebellopontine tumour and the other 2 had cerebellar S.O.L.s.

6 of all the posterior fossa S.O.L.s were of infective origin. This includes the one of non-specific inflammation which might not have been infective. It signifies that about 13% of the posterior cranial fossa S.O.L.s are infective.

3 patients developed post-operative complications One 50 year old man after successful excision of an arachnoid cyst developed post-operative seizures. One patient operated for cerebellopontine angle tumour developed an immediate facial nerve palsy. One patient inserted a V-P shunt got infection severe enough warranting the removal of the shunt.

TABLE 10

DISTRIBUTION OF POSTERIOR FOSSA S.O.L.s BY SITE AND OUTCOME (N=46)

	DIED IN HOSPITAL	NOT SEEN IN CLINIC	CLINIC FOLLOW UP (YEARS)		
			Yr.1	Yr.2	Yr.3
1. Cerebellum	3	5	7	7	2
2. Brainstem	4	9	1	2	-
3. C-P Angle	1	5	-	-	-

D I S C U S S I O N.

It appears that Brainstem and cerebellar posterior fossa s.o.ls are commonest around the first decade of life at Kenyatta National Hospital. The youngest patient in this study was 2 years with a diagnosis of brainstem tumour. Younger patients have been diagnosed elsewhere for instance Alvisi.c. et al (2) in their review had patients of one year of age. The youngest patient with a cerebellar s.o.l. was 2½ years with medulloblastoma in contrast to Donald Q.O. et al who had 5 patients aged one year in their series (15). It is possible that younger patients exist in Kenya but are either diagnosed late because of the poorer availability of health services or the diagnosis is missed because of the lack of availability of diagnostic techniques and facilities. Besides this early diangosis the age distribution is consistent with studies elsewhere (2,15,22,48,50,57). In this series 75% of the brainstem tumuors presented before 20 yrs of age this compares favourably to 77% in the review by Rusell et al.

C-P angle tumuors are rare before 20 yrs of age, in this institution, None were below 20 yrs. This

tendency to occur in adulthood is also common elsewhere (56,59).

Male preponderance in cerebellar astrocytomas, 5:1,is unusual. In other series the sex ratio is similar (18,11). In other studies Brainstem gliomas occur in equal frequency in males and females (38,57) in this series it was 3:1. This finding implies that males are more afflicted with these S.O.Ls unlike in other populations. The sex ratio M:F of 2:1 in medulloblastoma is classical in most literatures (35). C-P angle tumuors are commoner in females similar findings have been reported elsewhere (56,59).

Objective difficulty in walking or deteriorating milestones (failing motor system) is an important and common symptom its importance has been emphasized in other series(38). It is particularly important in our enviroment where headache, vomitting and convulsive seizures are common with infections. In particular meningitis and cerebral malaria which are prevalent. In addition headache and vomitting can be absent particularly in Brainstem tumuor, where increased intracranial pressure (I.C.P.) can occur late or be absent (2,22,57). Increased I.C.P. might be present in the absence of headache and vomitting (14). This stresses the importance of

reviewing the symptoms in totality. In this series several patients had been treated earlier for malaria and meningitis.

Any visual disturbance especially deteriorating vision should be taken seriously. 17% blindness at presentation is high and preventable. The principal cause of this blindness is increased I.C.P. (see above). This is exemplified by the fact that it occurred even in Benign cerebellar s.o.ls. The single most important sign of increased I.C.P. is papilloedema (14). Other associated signs like fixed dilated pupil, Transient motor disturbances and reduced level of conciousness imply herniation and in particular the latter has a poor prognosis (14,16,24). In a recent review of cerebellar astrocytomas (11)., though 83% of the patients had papilloedema none was blind. In 1931 Cushinge reported that 29% of cerebellar astrocytoma patients were blind at admission(10). This signifies that early diagnoses can minimise this blindness. It must be stressed that increased I.C.P. is not limited to cerebellar s.o.ls. In this study 44% (N=16) of the patients with Brainstem and 5 out of 6 with C.P. angle lesions had features of I.C.P. 46% of the patients appear to have heard severe intracranial hypertension as illustrated by Neck stiffness and 7 patients with reduced conciousness were most probably herniating. Alvisi et

in there review of 16 patients with Brainstem gliomas found that 69% had intracranial hypertension.

The occurance of mental changes (17%) is less then in other series. Strange et al found that 48% (N=31) of his patients had this deficit. Alvisi et al elicited 31% (N=16) in his series. It is possible that our figures could be higher especilly taking into consideration that in some of the patients with diminished conciousness this symptom could not be elicited accurately. None the less it is an important feature of Brainstem lesions that can result in mis diagnoses (as a functional disorder). Michael R.T. et al has stressed the importance of suspecting a brainstem lesion when Neuropyschiatric disturbance is coupled by abnormal eye movements. This is because the limbic, reticular and hypothalamic structures are closely related to the eye movement centres in the midbrain. In addition closed hydrocephalus can cause dementia like changes when it is long-standing.

Cranial nerve involvement is an important feature of posterior fossa s.o.ls in this study. Especially so for Brainstem s.o.l.s in which the

V, VI and VII nerves were affected in about ½ the patients. Alvisi et al in there review of Brainstem gliomas found 81% involvement of cranial nerves as compared to 65% in this study. Strange et al found VI, VII and V nerve most commonly afflicted, 65% and 48% respectively in the latter 2. This similarity of cranial nerve affliction with this series is noted. In one review IX and X nerve are said to be more common then V(38). In this study they were rare. V,VI and VII are most commonly afflicted because they have their nuclei in the pons the commonest site for brain stem glioma (38). In cerebellar s.o.Ls cranial nerve palsies signify advanced disease (11,38) and this again correlates with the high prevalence of blindness seen in this study. C.P. Angle S.O.L. cranial palsies show no unusual feature (60).

As stated earlier an objective symptom of difficulty in walking should be evaluated seriously. In addition to pyramidal tract involvement cerebellar pathways involvement is common in Brainstem tumuors and increases the incidence of gait disturbance. Alvisi et al in there review elicited

cerebellar signs in 81% and pyramidal tract signs in 63% of the patients. In this study overt pyramidal tract signs was elicited in 62% of the patients with Brainstem s.o.ls and in 53% (N=39) of all the patients with posterior fossa s.o.ls. Cerebellar signs was elicited in 50% (N=12) and 82% of all the patients with posterior fossa s.o.ls. As compared to Alvisi's review cerebellar signs are less common in brainstem s.o.ls. in this study. Strange et al elicited cerebellar signs in 74% patients. It is possible that cerebellar signs were missed in some patients in this study particularly those presenting with reduced conciousness. In addition in 4 of the patients with brainstem s.o.ls. there was no clue as to wether cerebellar signs were elicited or not. This finding is therefore questionable. However, the 2 signs are important and common.

The presence of quadraparesis, paraparesis or ipsilateral hemiparesis not consistent with the expected cross paralysis is Not unusual. It has been reported in other series (2,38,57). It must be appreciated that long standing increased I.C.P. contributes significantly to pyramidal tract signs and is principally secondary to herniation syndromes (14,16,24,26).

Sensory tract involvement is uncommon except in C-P angle tumuors. None of the patients with cerebellar s.o.ls had any peripheral sensory deficit (Not cranial). This is consistent with work done elsewhere (2,38,57).

Details of Nystagmus can be of localising value (40). Nystagmus is common and should be elicited whenever it is possible.

As noticed in the yearly trends it appears that the number of posterior fossa s.o.ls being detected is on the increase. Advent of C.T. scan appears to be the most important factor and several authors have observed this trend (4,5,20).

Brainstem S.O.Ls were not treated surgically. Histology is therefore a matter of logics based on the literature. Gliomas are the commonest s.o.ls in the brainstem (30,32,38). Kasili et al in their review of histopathology of intracranial neoplasms (97 cases) found only gliomas in the brainstem (3 cases). Chronic brain abscess can occur in the brainstem but are rare, less then 1% (49). Other s.o.ls that occur rarely include Tuberculomas, Haematomas, epidermoid cyst and secondaries (21,29,38). None of the patients had any significant history of previous infections esepcially otitis media, meningitis and T.B. None had confirmed tumuors at other sites.

It is therefore most likely that almost all the cases were gliomas. In studies where histology has been available most tend to be gliomas of the astrocytic type (21,34). Harold J.F.et al in there review found 62.7% (N=51) to be malignant (Grade III or IV astrocytomas. Derek T.J. et al found 48% (N=23) to be low grade (I or II) astrocytomas. Some authorities believe that Brainstem gliomas have a definate tendency to undergo anaplastic change (48). Other authors stress that because such gliomas arise in the brainstem substance and grow by infiltration they should all be regarded as malignant regardless of the histology(57).

The finding of cerebellar astrocytomas and medulloblastoma combined being lower than Brainstem gliomas is unusual. The 2 Neoplasms have for long been taken as being the commonest in the posterior fossa and Brainstem gliomas being third in prevalence (11,35,65). However, this trend is being questioned worldwide. After the advent of C.T.Scan more brainstem gliomas are being diagnosed (5,7,11). Bilaniuk L.T. et al have stated that the accuracy of C.T. scan in brainstem gliomas approaches 100%. In this study the largest number of Brainstem s.o.ls. was diagnosed in 1989. It is therefore possible that in this population

brainstem gliomas are more prevalent then astrocytomas and medulloblastoma. This statement takes into consideration the 13 patients excluded from the study. If a half of them had either of the 2 cerebellar neoplasms still the number of Brainstem gliomas (16) would be larger.

Other features of cerebellar astrocytomas and medulloblastoma were consistent with those seen elsewhere (11,18,35). None of the patients had cerebellar astrocytoma grade III -IV (cerebellar glioblastoma). This variety is rare and Gene kopelsen in his review of 745 patients with central nervous system astrocytoma found only 8 cerebellar cases of glioblastoma over an 19 year period.

In this series confirmed tuberculomas comprised 9% (N=46) of all the posterior fossa s.o.ls Sorour et al in their review found that it occured in 6% (N=86) of the surgically operated patients and 12.5% (N=16) in the postmortem cases in the posterior fossa. Figures are comparable. Poltera in the postmortem review of Ugandan cases found that 3% of all intranial s.o.ls were affected. It was not possible to calculate the percentage in the posterior fossa. Though in this study the cases of tuberculomas are few the age trend

deserves some mention. The earlier 3 cases were in children less than 14 yrs and the last one in 1988 was 29 yrs. Recently, it has been observed that there is a general decline in tuberculomas along with other types of T.B. mainly because of active treatment and prevention (23). It has been observed that with this decline the childhood variety which was commoner is becoming less common and the adulthood type commoner (33). This reversal of trend was evident in our few cases.

The finding of one patient with hydatid cyst from Turkana district is not unusual. Okello G.B.A. has stated that it is endemic in that District and that 10% of the people are infected. Studies have shown that where hydatid is endemic it comprises of 1-3% of all intracranial s.o.ls (6,17,44). Hydatid disease is a zoonosis caused by the larval stage of Echinococcus granulosus or Echinococcus Multilocularis. E.Multilocularis is invasive and is beyond the scope of this topic.E.granulosus infective host is the dog mainly and other carnivores. The larval stage may infest man accidentally if he ingests infested faeces. Because man is not definative host the larvae logdes in the body and forms a cyst in body organs which expands as its fluid content increases. This is the hydatid cyst commonest in the liver and lungs (6). Any other organ can be infested including

brain. The brain can be affected by the primary cysts or by embolization from the lungs or heart (57). C.T.scan has improved the diagnosis to near pathognomonic levels (44). On C.T. the lesions appear as cystic masses with densities almost similar to C.S.F. They show no ring enhancement with contrast usually but rarely this may be seen (3). Calcification may also occur rarely (17). Cysts might be multiple (58). It is important to suspect hydatid in patients coming from high risk areas because of the risk of anaphylaxis from the hydatid fluid spillage. During surgery of primary cysts when spillage occurs recurrence is inevitable (Okello, G.B.A. 1988). Okello G.B.A. in his review has stressed the importance of using scolicidal agents (e.g.1% cetrimide) and Albendazole to prevent recurrence after spillage.

It is possible that the C.P. angle neuromas that could be diagnosed by histology are more because one patient refused surgery and in 2 others no histology was available. One patient with a C-P angle had non-specific inflammation as the histology report infection can thus not be ruled out. It must be appreciated that other types of S.O.Ls besides neuromas can occur in the C-P angle. J.Thomsen in his review found 11 different tumuors other than neuromas. In this review meningiomas.

Epidermoid cysts and arachnoid cysts were the commonest. In our series none of the patients had features of Von Recklinghausen's disease and none of the lesions were bilateral. C-P angle neuromas in this study showed no unusual features worthy of mention (56,59).

Epidermoid and Dermoid tumuors are congenital lesions derived from developmental ectodermal inclusions secondary to imperfect embryogenesis (27) and attributed to the fact that the C.N.S. is of ectodermal origin. Dermoid tumuors are easily recognisable because of the presence of dermal appendages e.g. hair follicles,sweat glands and sebaceous glands. Epidermoid cysts are commoner and composed only of dermal epithelium and associated connective tissue (27). It is therefore most likely that the 2 cases of Developmental abnormality cysts were epidermoid cysts. Both patients are alive and well following excision.

In this study only one case of arachnoid cyst was found. Sorour et al in his review of brain tumuors in Egypt found only 2 patients with arachnoid cysts out of 377 but they do not mention the site. Kasili et al cited none. It appears that arachnoid cysts are uncommon especially in the posterior fossa in this population.

It is interesting to note that some histological patterns found in other populations in the posterior fossa were not found here. This includes meningiomas 2% in post fossa (55), Ependymomas 18% in post fossa (55). Kasili et al in their earlier review of patients in Kenya found 4 cases of Ependymomas but they did not state the site. Choroid plexus papilloma 0.4-0.6% of all intracranial s.o.ls (28), Metastases 19% of intracranial secondaries in Post fossa (29) and spontaneous cerebellar haemorrhages (20).

At least 67% of the patients with cerebellar s.o.ls were alive at the end of one year following treatment. Majority of patients from Kenyatta National Hospital are refferals from the peripheral hospitals. Some continue their follow up there. Other patients despair and resought to traditional medicine. Others change to private hospitals. It becomes very difficult to predict their final outocme. The period of risk as formulated by Collins et al for Wilms tumuor has been found fairly reasonable by Donald O.Q. et al. requires may years of follow up and even this could not be calculated. However, studies have shown that Benign cerebellar gliomas have a good survival rate of 94% - 25yrs cumulative survical rate (18.1) Medullo blastomas have a 25-70% 5 yr survival following treatment (35) comprising total excision and radiation.

This is before 2 yrs after 2 yrs an almost 100% Mortality is seen within 2-3 yrs. Tuberculomas are curable medically if diagnosed early and surgically if Advanced (4).

The high incidence of hospital mortality of patients with Brainstem gliomas, 25%, and the subsequent poor follow up are indicators of the poor prognosis of this neoplasm. Strange et al in his review of primary Brainstem tumuors found only 6% (N=31) surviving at the end of 2 yrs and none at the conclusion of his study. The patients were treated by surgery and radiotherapy and 35% of his patients recieved no treatment. Majority die within the first year and infrequent reports of 10 yr survival of 17-33% has been claimed by some workers (13). Recently, surgery has been recommended for a distinct group of Brainstem gliomas by some workers. Harold Hoffman et al found in there review that about 8.3% of the brainstem gliomas tended to grow into the 4th ventricle (exophytic) and therefore presented early with increased I.C.P. Such tumuors were solid, occured earlier in life then the others and upon resection prognosis was good. Histology revealed grade I-II astrocytoma longest survivor was alive 15 yrs after treatment. Alvisi et al in a similar study has reported similar success (mean 15 Yrs survival).

In these studies adjuvant post operative radiotherapy was recommended. It is therefore likely that at least in this group of benign brainstem gliomas the future prognosis will be much improved by surgery.

2 of the patients with C- P angle s.o.ls had been seen in other private hospitals earlier. It is likely that they continued their follow up there.

The post operative complication rate of 11% is not high. Only one patient had seizures. It is said that seizures alone can occur in upto 12% of patients in post fossa surgery (11). Immediate facial nerve palsy occured in one patient following C-P angle surgery. This patient has fully recovered since then. Such fascial nerve palsies may be transient or permanent(60). Transient when it is due to physiological disruption and permanent when the disruption is Anatomical. Only one patient had wound infection. This is a low infection rate.

CONCLUSIONS :

1. 17% blindness that can be prevented is high.

2. The high hospital mortality and the poor followup could be a reflection of high mortality as the final outcome especially in brainstem tumuors.

3. There is a definate need to step up followup because it is the only procedure that would allow for objective evaluation of treatment, otherwise evaluation of treatment is misleading and uninformative.

4. Follow up is unsatisfactory. Even mortality is vital information and essential part of follow up.

CONSTRAINTS

Finally, it is important to consider some of the major constraints which affected this study.

1. The numbers involved are small but most studies covering this area have dealt with small numbers. However, it must be stressed that larger numbers are more desirable because findings are more accurate.

2. As stated earlier poor follow-up was the principal constraint. It made detailed analysis of treatment and outcome impractical.

3. As noticed in some analysis tables the number of patients analysed are less than the total number of patients reviewed. This is because there was no clue as to whether the clinicians had those aspects in mind and hence they had to be excluded. Negative findings at least generally are important in records for retrospective analysis purposes.

4. Lack of journals in the library was significant. Relevant journals were often lacking and hence resort to textbooks was inevitable.

RECOMMENDATIONS:

1. To improve on the late diagnosis and therefore reduce the high percentage of blindness, perhaps communicating back to inform the refferal hospital and reffering doctors of the final diagnosis will be quite educative and polite. This could enhance early diagnosis and increase their suspicion index in the future. In addition awareness should be enhanced through seminars and other forums.

2. There is a need to identify the Benign group of Brainstem gliomas that are operable and operate them.

3. Reproducible, repeatable and quantifiable standard measures for recording degree of recovery is important. One such measure is the performance status karnofsky modified depicted in Table II (C.Alvisi et al, 1985).

Table II: Quantitative evaluation of quality of survival by the performance status Karnofsky Modified (PSKM)

GRADE I:	Able to carry out on normal activity. No special care is needed.
GRADE II:	Unable to work. Able to live at home and care for most personal needs. A varying amount of assistance needed.
GRADE III:	Unable to care for self. Requires equivalent of institutional or hospital care. Disease may be progressing rapidly.

4. Communication is 2 way and when we communicate about the final diagnoses those reffering should be encouraged to communicate about the final outcome of the patients for record purposes.

5. Importance of followup cannot be over emphasised. It is the basis of evaluation of treatment and outcome. It may eventually reduce costs because the most appropriate modes of therapy are timely executed.

Followup is generally unsatisfactory in most clinics in Kenyatta National Hospital. There is need to set up a followup unit. It should be charged with the task of followup through village visits, communication with the Administration and registrar of deaths and use of other viable methods.

6. This study has not exhausted treatment and outcome because of the small numbers involved and the poor followup. There is a need to carry out further studies in this area.

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APPENDIX 1

MODE OF TREATMENT AND OUTCOME IN PATIENTS WITH POSTERIOR FOSSA S.O.L.s (N=46)

MODE OF TREATMENT		DIED	FOLLOW UP			SEEN IN CLINIC
			1st YR	2nd YR	3rd YR	
1. Excisional/Diagnostic Biopsy only	CER	2	2	2	-	2
	C.P.	1	-	-	-	4
Preceded by V-P Shunt	CER	-	1	-	-	
Subsequent Chemotherapy	CER	-	-	1	1	1
Subsequent Radiotherapy	CER	-	1	2	-	1
Subsequent radio and Chemotherapy	CER	-	1	2	1	
With V-P Shunt and Radiotherapy	CER	-	1	-	-	1
2. V-P Shunt only	CER	1	-	-	-	
	B.S	2	1	-	-	1
With chemotherapy	CER	-	-	-	-	-
3. Radiotherapy only	B.S.	-	2	-	-	1
4. Chemotherapy only (empirical	CER	-	1	-	-	-
	CER	-	-	-	-	-
5. No treatment given	B.S.	2	-	-	-	7
	C.P.	-	-	-	-	1

KEY

B.S. - Brainstem

CER - Cerebellum

C.P. - Cerebellopontine angle.