Structure and Function
Brain stem evoked responses and Radiological imaging of the brain stem

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Abstract
20 patients with small, well defined intra-axial lesions within the pons or low midbrain were examined. All lesions were demonstrated by CT scan and/or MRI. Based on the anatomical sites of the lesions, patients were classified into two main groups. Group (A) with posteriorly placed lesions in the immediate peri-4th ventricular or periaquiductal regions of the brain stem. Group (B) with anteriorly placed lesions in the basis pontis or crus cerebri. All patients were subjected to brain stem evoked study. The brain stem evoked studies were abnormal in all group (A) patients and were within normal in all group (B) patients. The brain stem lesions were asymptomatic in 30% of group (A) patients, while all group (B) patients had symptomatic brain stem lesions. The clinical picture of group (A) patients was in the form of cranial nerve palsy, Gaze palsy, vertigo, cerebellar manifestations and increased intracranial pressure with no long tracts affection. The clinical picture of group (B) patients were exclusively in the form of long tract affection. Accordingly it was concluded that the existence of brain stem evoked abnormalities should point to either fuctional disturbances or structural pathology in the immediate peri-4th ventricular or peri-aquiductal regions of the brain stem and not elsewhere; and unless these two regions are involved by the brain stem lesions, either as primary sites or by secondary extension, the brain stem evoked response will be normal.

Introduction: If one could record directly from several different levels of the subcortical auditory pathway in man, one would see in the first 10 milliseconds following an appropriate acoustic stimulus a series of potentials corresponding to the sequential activation of the peripheral, ponto medulary, pontine and mid brain portion of the pathway. Jewett and Willistone (1971) were the first to describe the human brain stem auditory evoked potentials (BAEP). Feinmesser and Szabo (1974) were the first to report selective alterations of different BAEPs by various types and loci of posterior fossa pathology. Over the following few years this clinicopathological correlations with specific BAEP abnormalities were extended by others, (Star and Achor 1975, Starr 1976, Starr and Hamilton, 1976, Stockard and Rossiter, 1977, Gillroy et al., 1977) on the basis of such studies, it appears that waves I, III, V represent volume conduction of electrical activity through the acoustic nerve, pons and mid brain respectively, and that the latencies in between these potentials represent neural conduction in the corresponding segments of the central auditory pathway. The aim of this study is to define which lesioned anatomical sites within the brain stem are likely to be associated with BAEP abnormalities and which are not.

Subjects and Method: 20 patients with strictly focal brain stem lesions that could be demonstrated by CT scan and/

or MRI were examined. All patients were subjected to
1. Clinical examination.
2. Cr scan and/or MRI with special focus on the brain stem.
3. Brain stem evoked response

Only patients with small, well-defined brain stem lesions were included in the study. Patients with more diffuse lesions were excluded.

Results: Based on the results of the radiological imaging of the brain stem, patients were subclassified into two main groups. Subclassification was based on the anatomical localization of the lesions within the brain stem as follows:

Group (A). Comprized 10 patients. The lesions were anatomically localised in the immediate peri-4th ventricular or peri-aquiductal regions of the pone and low mid brain respectively.

Group (B). Comprized 10 patients; the brain stem focal lesions were anteriorly localised in either the basis pontis or the crus cerebri of the low midbrain.

The clinical picture results of radiological brain stem imaging and results of brain stem evoked studies of group (A) patients are summarised in (Tables 1, 2,3).

The clinical picture and results of radiological imaging of the brain stem in group (B) patients are summarized in table (3). Brain stem evoked studies were within normal in all group (B) patients with anteriorly placed brain stem lesions

The clinical picture of group (A) patients was characterised by:

1. Complete absence of long tract affection (100%).
2. Anilateral or bilateral cranial nerve palsies (40%).
3. Cerebellar manifestations (50%).
4. Asymptomatic (40%).
5. Vertigo (30%).
6. Gaze palsy (20%).
7. Increased intracranial pressure (20%).

In group (B) patients, the clinical picture was exclusively limited to long tract affection (left or right sided hemiplegia).

It is interesting to note that whereas cases no. 8, 9, 10 (group A) had completely asymptomatic (subclinical) brain stem demyelinating patches, the brain stem evoked studies were abnormal, while in patients no. 8, 9, group (B), the brain stem demyelinating patches were symptomatic, and yet the brain stem evoked response studies were within normal.

In general, focal brain stem lesions exclusively localised posteriorly in the immediate peri-4th ventricular or peri aquiductal regions of the brain stem were associated with abnormal brain stem evoked responses. These lesions were symptomatic in 70% of group (A) patients and asymptomatic in 30% of these patients.

On the other hand, focal lesions exclusively localised anteriorly within the brain stem were associated with normal brain stem evoked responses. These lesions were symptomatic in 100% of group (B) patients.

These finding sspoint to the existence of an electro-clinical dissociation in brain stem evoked responses. It also points to the fact that only posteriorly placed focal lesions within the immediate peri-4th ventricular or peri-aquiductal regions of the brain stem, whether symptomatic or asymptomatic, are likely to induce evoked response ab-

normalities. While more anteriorly placed lesions in the basis pontis or the crus cerebri of the low midbrain, though associated with higher incidence of long tract affection, are unlikely to induce brain stem evoked response abnormalities. i.e the closer the focal lesions to the peri-4th ventricular or the peri-aquiductal regions of the brain stem, the higher the probability of inducing brain stem evoked abnormalities.

Discussion: The only difference between group (A) patients and group (B) was the anatomical sites of the lesions. Anteriorly placed lesions within the brain stem were associated with normal evoked responses studies in all patients, while posteriorly placed lesions were associated with abnormal evoked response studies in all patients.

The anatomical structures that are localised in the immediate peri-4th ventricular or peri-aquiductal regions of the brain stem are summarised in table (4) and Figure (14). Also the ventral and dorsal cochlear nuclei, the trapezoid body, the lateral lemniscus and the inferior coliuli are among the structure that are posteriorly localised within the brain stem. They constitute the main intra-axial generators of the brain stem evoked response waves Stockard, et al., (1980).

By putting things together, it is possible to state that only the posteriorly localised focal brain stem lesions that are more likely to disrupt the function of the intra-axial generators of the brain stem evoked response waves. These focal lesions are commonly associated clinically with gaze palsy, vertigo, cranial nerve palsies, internuclear ophthalmoplegia or cerebellar manifestations see (Table 4).

On the other hand, a more anteriorly placed lesions in the brain stem, being away from the intra-axial generators of the brain stem evoked waves, are unlikely to induce brain stem evoked abnormalities. These lesions, being situated in the basis pontis or crus cerebri, were associated with higher incidence of long tract affections.

Accordingly, abnormalities of the brain stem evoked responses are mainly due to involvement of the immediate peri-4th ventricular of the peri-aquiductal regions of the brain stem, and unless these regions are selectively involved by the lesions, the brain stem evoked response study will be normal regardless of whether the brain stem lesion, that is situated elsewhere, are symptomatic or not.

From the clinical point of view, the probabilities of encountering brain stem evoked abnormalities are increased if the clinical picture is dominated by vertigo, gaze palsy, cranial nerve palsies, or cerebellar manifestations as these signs point to a more posterior brain stem involvement. Absence of these signs probably will lower the incidence of encountering brain stem evoked abnormalities.

Functional lesions of the brain stem (such as vertebro-basilar TIAs with vertigo) could also be associated with evoked response abnormalities, though these functional lesions may not reach a sufficient magnitude to induce structural pathology that could be demonstrated radiologically. By analogy, these functional lesions, unless situated in the immediate peri-4th ventricular or peri-aquiductal regions of the brain stem, are unlikely to induce evoked response abnormalities.

To end up, the existence of brain stem evoked abnormalities should point to either functional disturbances or structural pathology in the irnmediate peri-4th ventricular or peri-aquiductal regions of the

brain stem and not elsewhere. Even with more diffuse pathology of the brain stem (like diffuse brain stem glioma for example) the presence of brain stem evoked abnormalities should be regarded as due to extension of the patholgy to the peri-4th ventricular or peri-aquiductal zones of the brain stem.

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Cross sections at the level of high medulla, pons and low midbrain.

Table (1) Clinical picture of group (a)patients

Patients number	Clinical Picture
1	Vertigo, horizontal gaze palsy, trunkal ataxia, no long tract affection
2	Bilateral 7th nerve palsy, vertigo, Rt 6th nerve palsy, turnkalataxia, no long tract affection.
3	Asymptomatic
4	Bilateral vertigo, horizontal gaze palsy, bilateral 7th nerve palsy, bilateral cerebella manifestation, bilateral r bulber manifestations, vertigo, no long tract affection.
5	Asymptomatic
6	Increased intracranial presure, 7 th nerve palsy, bulber manifestation, turnkalataxia, no long tract affection.
7	
8	Asymptomatic
9	
10	

Table (2)

Patients number	Sex	Age	Radiological description of the lesion	Result of brain stem evoked response
1	M	4.2	Posteriorly placed focal brain stem glioma (Dorsal exophytic brain stem glioma). The lesion is mainly pontine, with extension to the low mid brain (See Fig. 1)	Bilateral pontine, low mid brain dysfunctin (See Fig. 8)
2	M	7	Posteriorly placed focal pontine glioma (Dorsal exophytic focal pontine glioma (See Fig.2)	Bilateral pontine lesions, more on the left side
3	M	9	Rt posterior haemorrhage secondary to lateral sinus thrombosis (See Fig. 3).	Rt pontine lesion
4	M	39	Extensive subependymal lymphoma extending to the periaquiductal zone and around the 4th ventricle forming by butterfly lesions (See Fig.4).	Bilateral pontine, low mid brain lesions more on the Rt side(See Fig. 9)
5	F	40	Multiple encephalitic patches around the 4th ventrical and the aquiduct (See Fig.5).	Bilateral pontine,low mid brain lesions more on the left side(See Fig. 10)
6	M	12	Middle linw medulloblastoma compressing the posterior aspect of the pons (See Fig.6)	Bilateral pontine lesions more on the left side
7	F	9		
8	M	20	Demyelinating patches around the 4th ventricle (See Fig.7).	Unilateral or ilateral pontine dysfunction
9	M	25		
10	M	19		

Table (3)

Patient number	Radiological description of the pathology	Clinical Picture
1 2 3 4 5 6 7	Anteriorly placed pontine lacunar infrarctions (See Fig. 11)	Right or left sided motor hemiplegia
8 9	Anteriorly placed right sided demyelinating patch (See Fig. 12)	Left sided hemiplegia
10	Atrophy of the right crus cerebri (See Fig. 13)	Left sided hemiplegia

Table (4)

Anatomical structure	Clinical results of lesions
Cranial nerves 3,4,5,6,7,9,10,12	Cranial nerves palsies
Gaze centres	Horizontal and vertical gaze palsies
Medical longtudinal bundle	Internuclear ophthalmoplegia
Vestibular nuclie	Vertigo
Cerebellar peduncles	Cerebellar manifestations

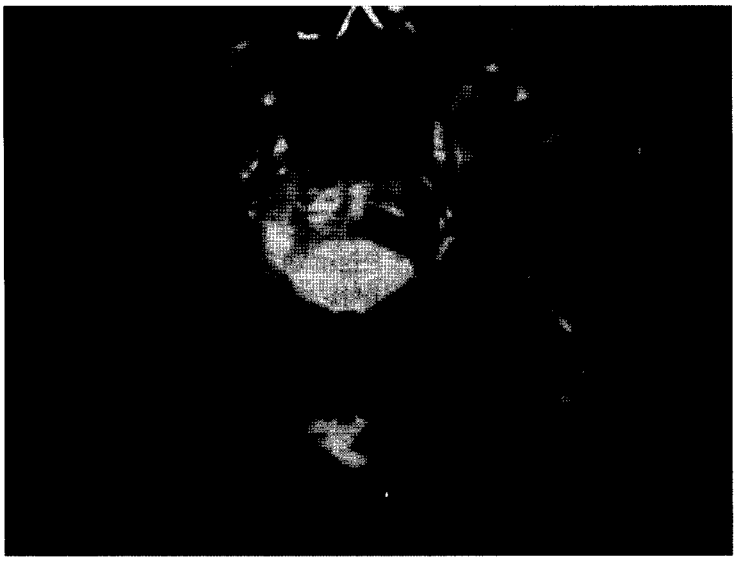
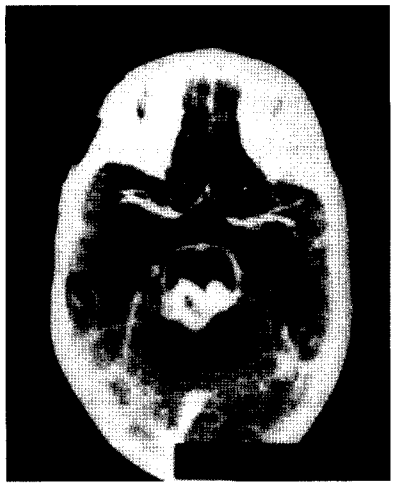


Figure (1)



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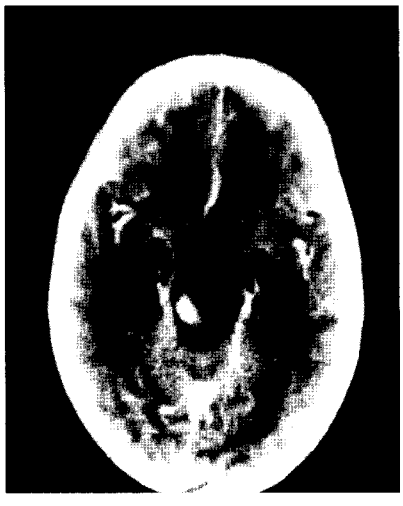

Figure (3)

Figure (4)

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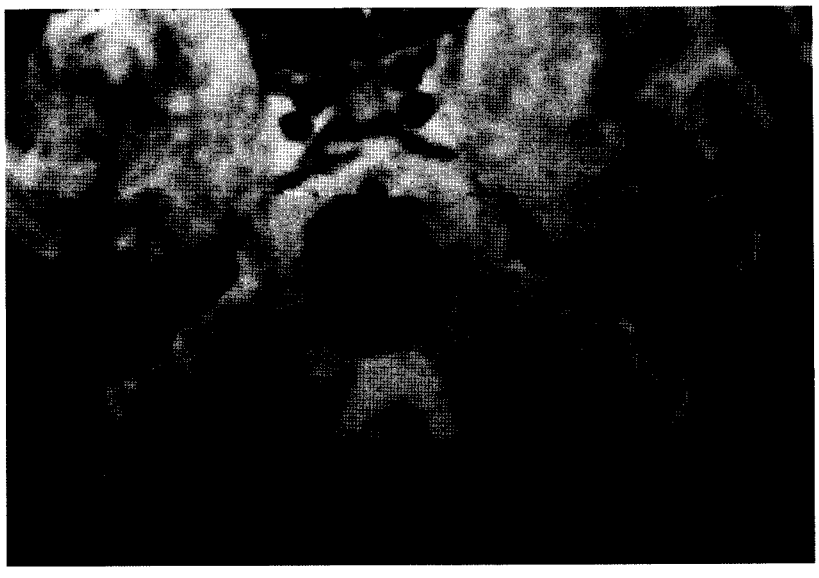
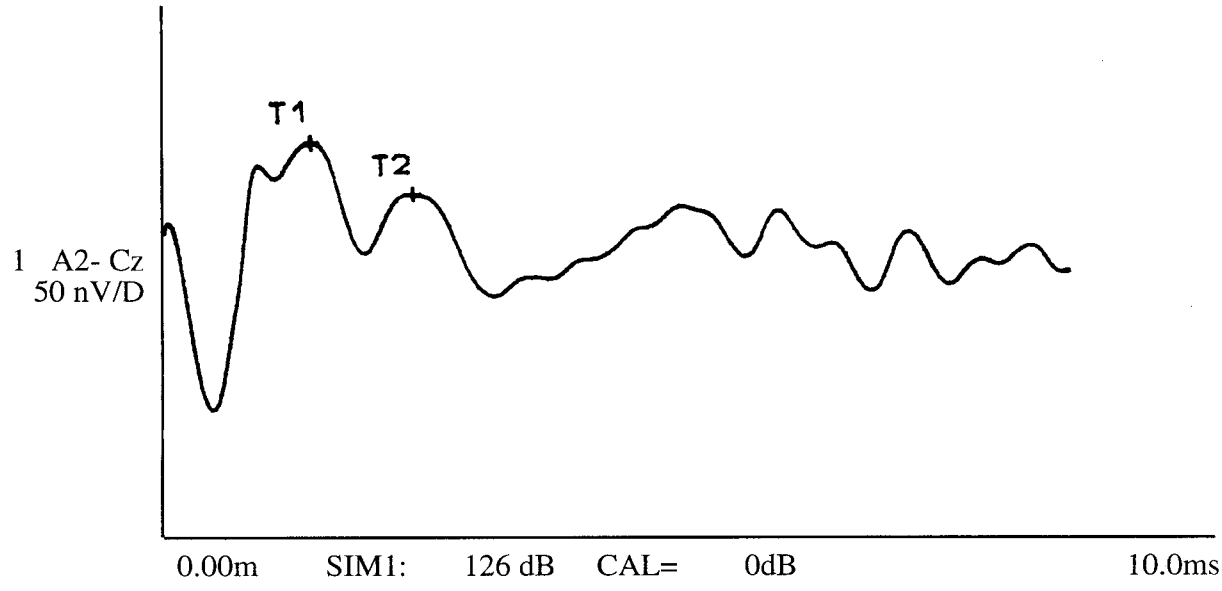


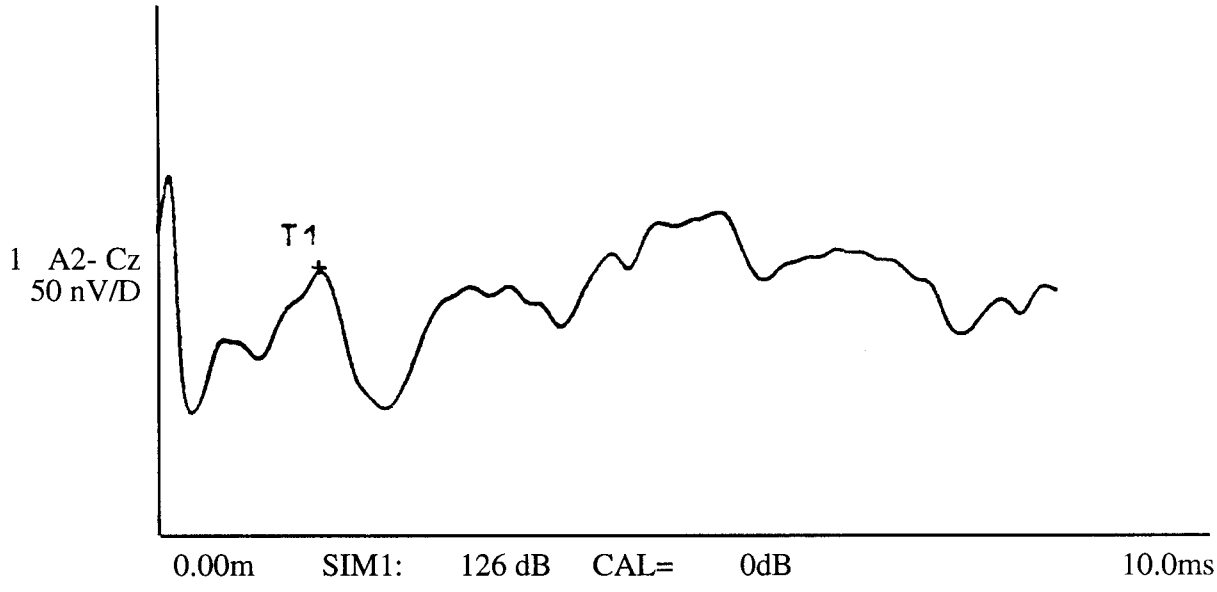
Figure (7)

SAFWAT ABDEL DAEIM
BAEP left ear, 12 Hz 18.4.93
T1 : (1.70 ms, -171 nV T2 : (2.82 ms -93.7nV)

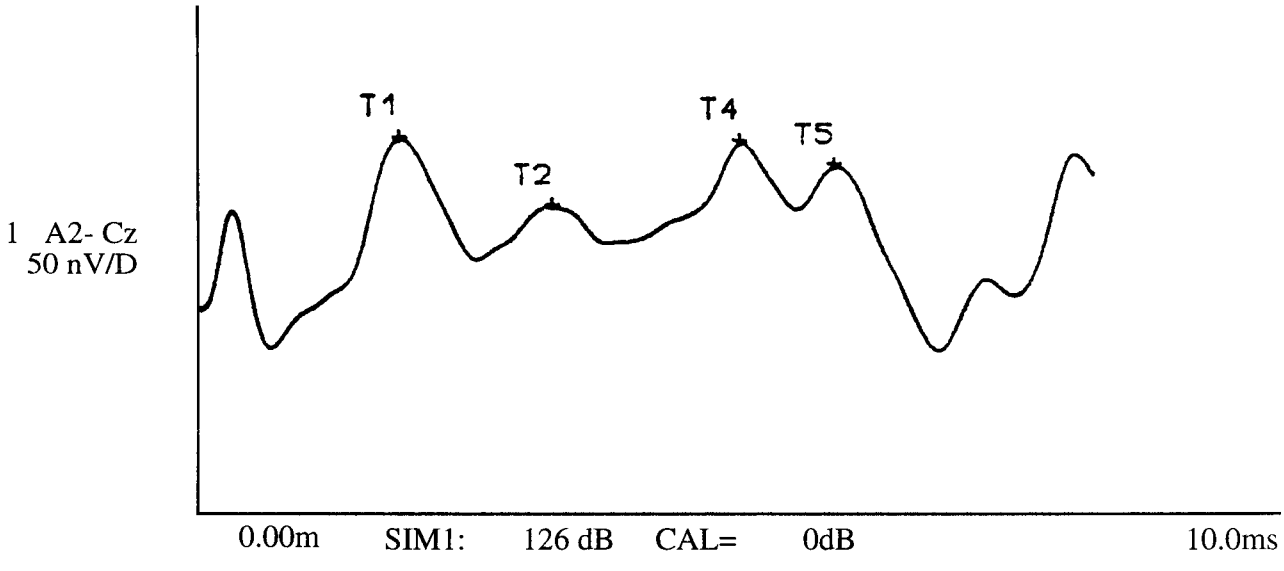


SAFWAT ABDEL DAEIM
BAEP, right ear, 12 Hz 18.4.93
T1 : (1.74 ms, -3.1 nV T2 : (3.00 ms, --171nV)

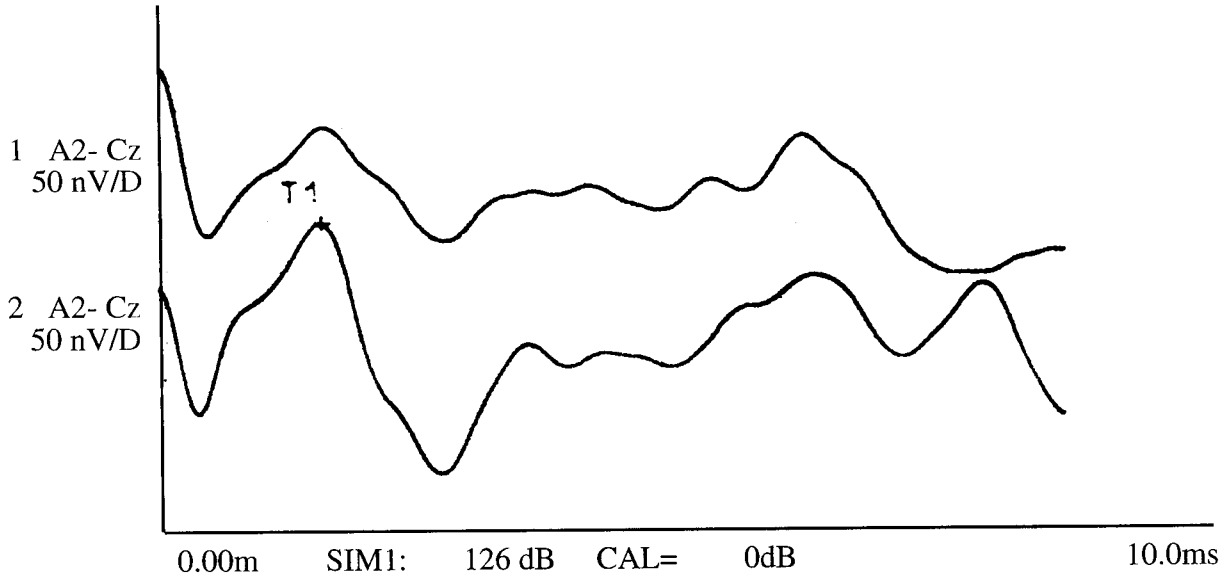
MARZOOK EL SAIED HASAN
BAEP left ear, 12 Hz 14.2.93
T1 : (1.84 ms, --6.2 nV)



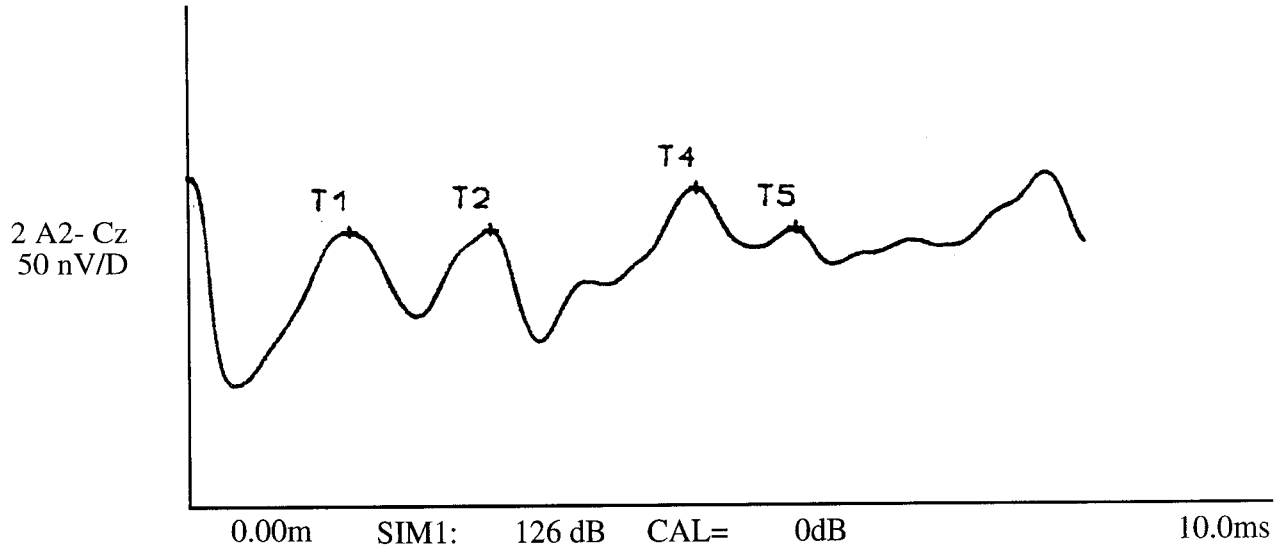
MARZOOK EL SAIED HASAN
BAEP, right ear, 12 Hz 14.2.93
T1 : (2.28 ms, -162 nV T2 : (4.02 ms, -65.6nV)
T4: (6.10 ms, -156 nV T2 : (7.14 ms, -121 nV)



WAFAA KHALEEL
BAEP left ear, 12 Hz 6.2.93
T1 : (1.78 ms, -168 nV)



WAFAA KHALEEL
BAEP, right earr, 12 Hz 6.2.93
T1 : (1.82 ms, -25.0 nV T2 : (3.38 ms, -34.3nV)
T4: (5.66 ms, -100 nV T2 : (6.78 ms, -50.0 nV)



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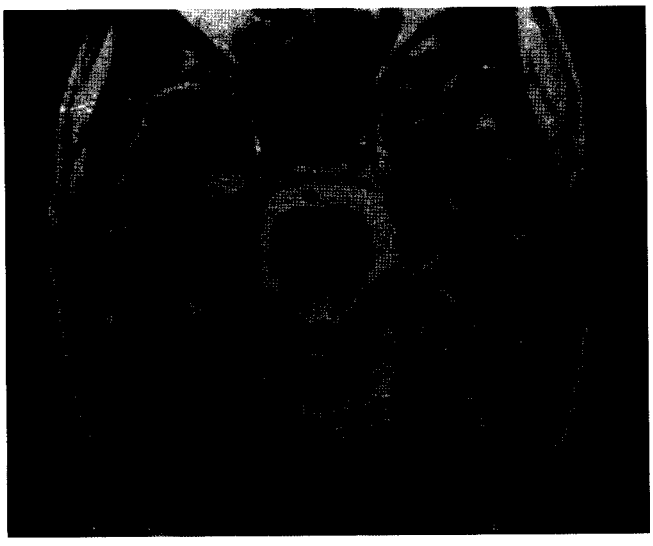

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Figure (13)

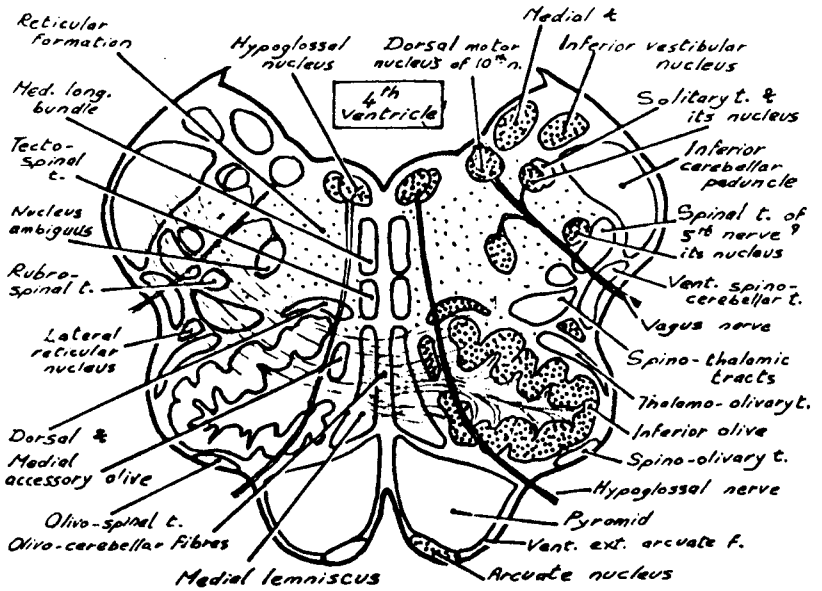

DIAGRAM OF A T.S. OF OPEN MEDULLA

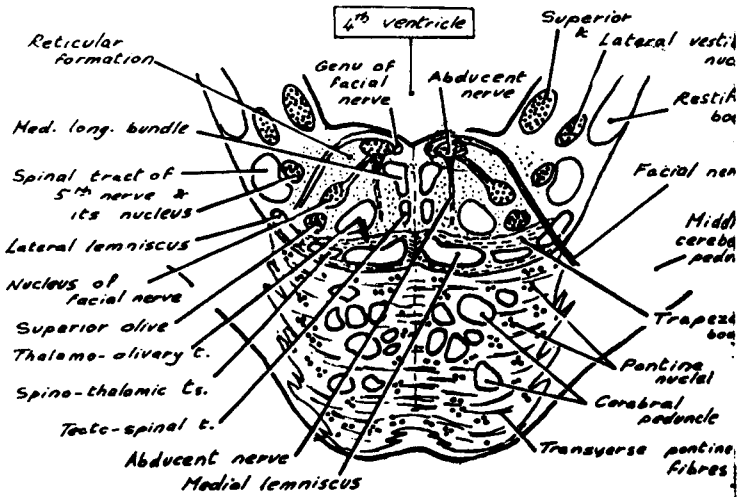

DIAGRAM OF A T.S. OF PONS "FACIAL COLLICULUS"

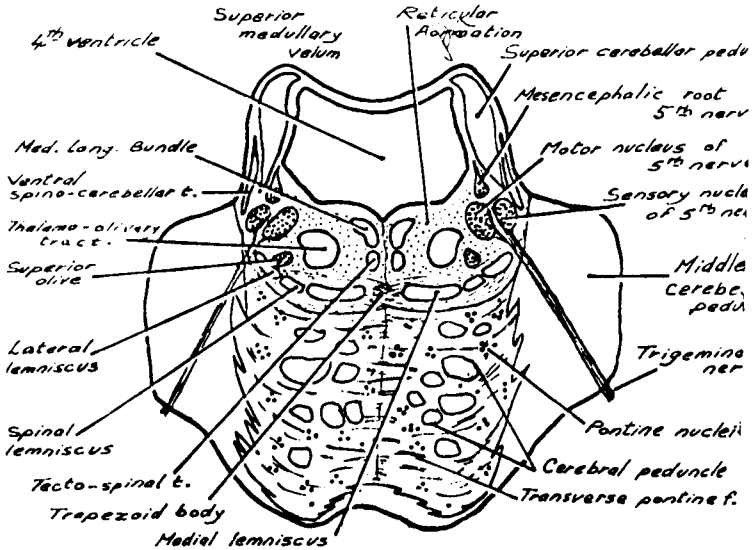

DIAGRAM OF A T.S. OF PONS

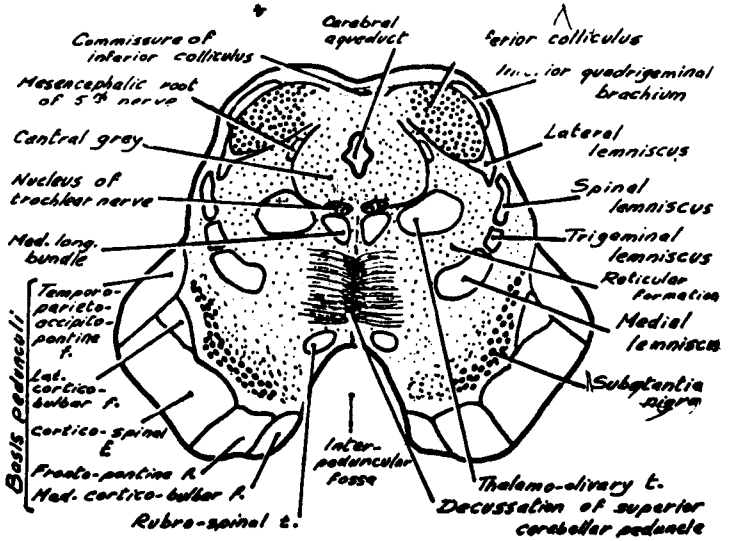

DIAGRAM OF A T.S. OF MIDBRAIN
"INFERIOR COLLICULUS"

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البنية و الوظيفة:
الاستجابات المستثارة فى جذع المخ و صور الأشعة لها

فحص ٢٠ مريضا لديهم تلف صغير محدد فيما بين المحاور داخل القنطرة أو الجزء الأسفل من المخ الأوسط. و أخذت صور لكافة أماكن التلف و قسم المرضى إلى مجموعتين(ا ،ب) على اساس الموقع التشريحى للتلف. و عرضوا جميعا لاستثارة فى جذع المخ. و كانت الاستجابات غير عادية فى المجموعة (ا) بينما كانت الاستجابات عادية فى المجموعة (ب) و كانت الصور الاكلينيكية للمجموعة الأولى فى شكل شلل للأعصاب أما الثانية فقد أخذت شكل تأثير طويل المدى.

و الخلاصة أن وجود استجابات غير عادية فى جذع المخ تشير إما إلى اضطرابات وظيفية أو اضطرابات بنيوية.