ROLE OF MRI IN EVALUATION CEREBRAL DEMYELINATING DISEASES

Thesis

Submitted for Partial Fulfillment of M.D. Degree

in *Radiodiagnosis*

Submitted By

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بسم الله الرحمن الرحيم

وقـــل رب زدنـــى علمـــا

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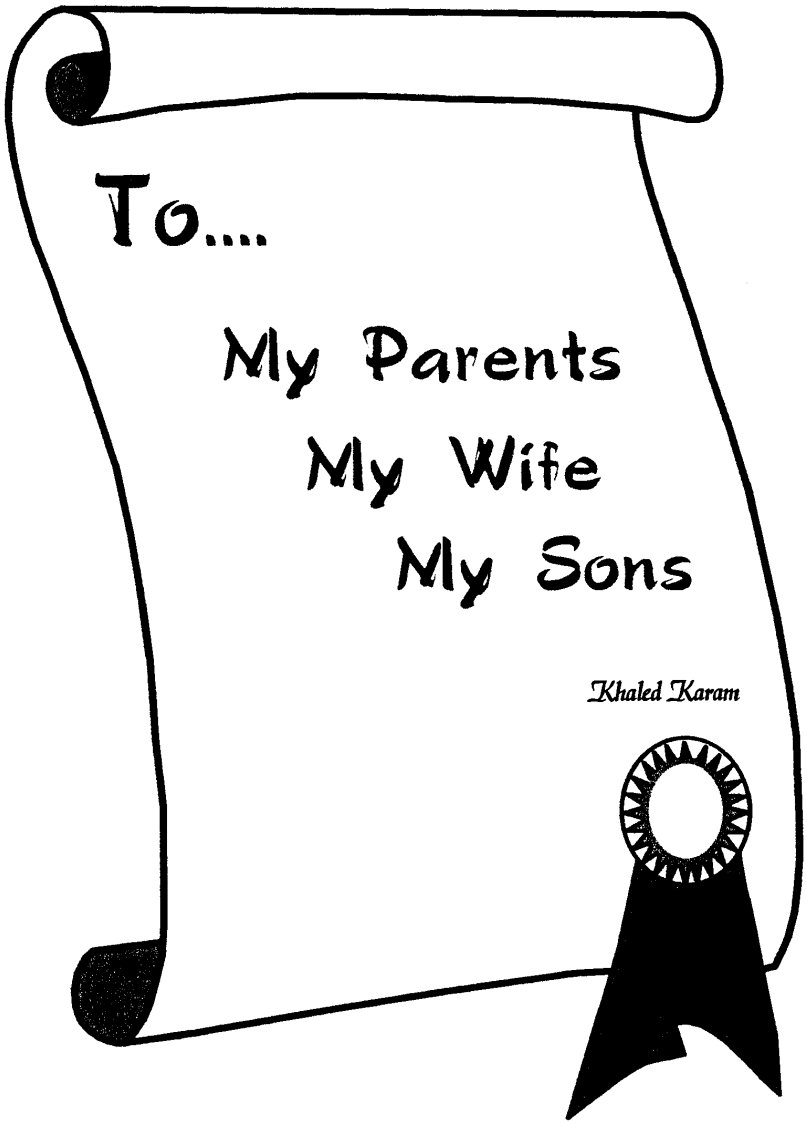
To....
My Parents
My Wife
My Sons
Khaled Karam

Acknowledgement

First and foremost, thanks to almighty ALLAH, to whom I relate any success in achieving any work in my life.

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INTRODUCTION AND AIM OF THE WORK

INTRODUCTION AND AIM OF WORK

Introduction:

The cerebral (and cerebellar) demyelinating lesions result from myriad causes that can be confusing and complex. To understand cerebral demyelinating lesion it is important to approach the abnormalities systematically and with a clear understanding of the clinical presentation and pathophysiology of the disease under consideration.

Magnetic Resonance Imaging (M.R.I.) is sensitive to the slight difference in tissue composition of normal gray and white matter and to subtle increase in water content associated with myelin diseases. Several studies *(Drayer et al., 1987)*, proved the superior capability of MRI over computed tomography (CT) in detecting white matter diseases.

Aim of The Work:

The role of M.R. imaging in the diagnosis, confirm the presence and to document the severity of cerebral demyelinating diseases.

EMBRYOLOGY OF THE WHITE MATTER OF THE BRAIN

The parts derived from cerebral vesicles are listed as follows:

A) Rhoben-cephalon (Hind brain):

1. Myelen-cephalon:
   a) Medulla oblongata.
   b) Caudal part of 4th ventricle.
2. Meten-cephalon:
   a) Pons.
   b) Cerebellum.
   c) Middle part of 4th ventricle.
3. Isthmus Rhomben-cephalon:
   a) Anterior medullary verum.
   b) Superior cerebellar peduncles.
   c) Cranial part of 4th ventricle.

B) Mesen-cephalon (mid brain):

1. Cerebral peduncles.
2. Tegmentum.
3. Tectum.
4. Aqueduct.

C) Prosen-cephalon:

1. Diencephalon:

a) Thalamus.

b) Meta-thalamus.

c) Caudal hypothalamus.

d) Caudal part of 3rd ventricle.

2. Telen-cephalon:

a) Cranial hypothalamus.

b) Cranial part of 3rd ventricle.

c) Cerebral hemisphere.

d) Lateral ventricles.

e) Inter-ventricular foramina.

Each cerebral hemisphere is formed of outer gray matter (cerebral cortex), underlying white matter (centrum semiovale) and small internal masses of gray matter (basal ganglia). In other words, the brain consists of gray matter (nerve cells) and white matter (myelinated nerve fibers).

Human brain is incomplete at birth, myelination of the nervous system proceeds rapidly in the peri-natal period occurring first in the peripheral nervous system, and last in the brain *(Dobbing et al., 1973)*.

The process of myelination is one in which the water content of the white matter of the brain decreases and its lipid content increases. Myelination of the brain begins during the 5th fetal month,

with myelination of the cranial nerves and continues throughout life *(Yakovelu and Lecour, 1976)*.

At birth, myelination is present in the medulla, dorsal mid-brain, cerebellar peduncle, posterior limb of the internal capsule and ventro-lateral thalamus.

Maturation proceeds from:

1- Central to peripheral.

2- Inferior to superior and posterior to anterior *(Barkovich and Jackson, 1989)*.

The cerebellum is myelinated at 3 months of age with an adult appearance on T_2 weighted images. The pre- and post central gyri are myelinated at 1 month and maturation of motor tracts is complete by 3 months. The pons matures from 3 → 6 months, with maturation proceeding rostorally along the cortico-spinal tracts, cerebral peduncles, through the posterior limb of internal capsule and central portion of the centrum semiovale *(Barkovich and Jackson, 1989)*.

The anterior limb of internal capsule by 2 → 3 months. The sub-cortical white matter matures starting at 3 months in the occipital region and proceeds rostorally to the frontal lobe, myelination in the corpus callosum can be a helpful landmark when estimating myelin development.

Myelination begins in the splenium (posterior) at 4 months and is complete involving the genu (anterior) at 6 months, the gradual myelination of the brain results in sequential major changes in its chemical composition, especially during the first year of life. Since myelin is rich in lipid and protein and poor in water, composed with the gray and white matter, myelination results in a decrease in brain water and increase in brain protein and lipid content. Water content decrease rapidly initially from 88 % at birth to 82 % at 6 months and then at a much slower rate through age 8→ 10 years. On the other hand, cholesterol level doubles with a more gradual steady rise through age 4→ 5 years and adults white matter has 3 times as much lipid as gray matter. After middle age, the fetal amount of the myelin lipid decreases and the cerebral hemisphere show increased water content.

Component	Gray matter	White matter	Myelin
Water	82 %	72 %	40 %
Lipid	6 %	16 %	30 %
Protein	11 %	11 %	29 %
Electrolytes	1 %	1 %	1 %

Also gray matter contains 8 % more oxygen and 8 % less carbon than dose white matter.

It is apparently these differences in chemical composition that account for the contrast between gray and white matter *(Brooks et al., 1980)*.

MRI ANATOMY OF THE BRAIN AND NORMAL WHITE MATTER APPEARANCE

Surface Anatomy Of The Cerebral Hemisphere

- Lateral Surface.
- Medial Surface.

1. Lateral Surface:

Lateral surface is convex in shape and has two prominent sulci, the central sulcus (Rolando's fissure) and the lateral sulcus (fissure & sylvius).

The course of the central sulcus on the lateral surface of the cerebral hemisphere begins at a point on the supero-medial margin, approximately halfway between the anterior and posterior end. From this point, the central sulcus courses down and forward separating the frontal and parietal lobes.

The lateral sulcus (fissure & sylvius) begins on the inferior surface, where it separates the frontal and temporal lobes, when it reaches the lateral surface. It sends on anterior horizontal ramus and on anterior ascending ramus and continues as the posterior ramus running a more or less horizontal course, separating the frontal lobe superiorly from the temporal lobe inferiorly.

As it courses posteriorly beyond the central sulcus, this lateral fissure separates the anterior part of the parietal lobe from the temporal lobe. Its terminal part turns dorsally into the parietal lobe.

On the lateral surface, the frontal lobe is well demarcated posteriorly by the central sulcus and inferiorly by the lateral sulcus on the lateral surface of the frontal lobe. The pre-central gyrus is outlined posteriorly by the central sulcus and anteriorly by the pre-central sulcus. The frontal lobe anterior to the pre-central sulcus is divided by a series of sulci into the superior, middle and inferior gyri.

The temporal lobe is outlined superiorly by the posterior ramus of the lateral sulcus. On the lateral surface of the temporal lobe there are two sulci that divide it into superior, middle and inferior temporal gyri. The parietal lobe is demarcated anteriorly by the central sulcus and posteriorly by an imaginary line. Inferiorly, it is separated from the temporal lobe by the posterior ramus of the central sulcus. The post central gyrus is the anterior part of the parietal lobe and abuts on the central sulcus, posterior to the post central gyrus. The parietal lobe is divided into the superior and inferior parietal lobules by the intra-parietal sulcus.

2. Medial Surface:

The medial surface is flat. In a sagittally bisected brain on obvious landmark on the medial surface, is cut surface of the corpus callosum. From front to back, it consists of the genu, body and

splenium. The corpus callosum is well demarcated by the callosal sulcus, which is continuous inferiorly with the hippocampal sulcus.

The cingulate sulcus follows a course more-or-less parallel to the callosal sulcus. The cingulate gyrus lies between the peri-callosal and cingulate sulci and therefore, encircles the corpus callosum. The cingulate sulcus is then continued as the sub-parietal sulcus outlining the isthmus, which is continuous with the para-hippocampal gyrus on the inferior surface of the temporal lobe. The precuneus is outlined posteriorly by the parieto-occipital sulcus, which is the anatomic border separating the parietal and occipital lobes.

The parieto-occipital sulcus originates superiorly at the supero-medial marginal of the cerebral hemisphere and courses down and forward. Near the isthmus of the cingulate gyrus it is joined by the calcarine sulcus, which courses backwards towards the occipital pole. The triangular part of the occipital lobe between the parieto-occipital and calcarine fissure is the cuneus. The part of the lobe inferior to the calcarine fissure is seen lingual gyrus, which extends to the inferior surface of the occipital lobe and is continuous anteriorly with the para-hippocampal gyrus of the inferior surface of the temporal lobe.

Normal M.R.I. Anatomy of the brain

1) Axial Section: (Fig. 1.1).

Six axial planes parallel to the canthomeatal line are presented. It is convenient to describe those planes in four levels:

1. The supra-ventricular level includes sections above the level of the lateral ventricles.
2. The high ventricular level includes those at the level of the body of the lateral ventricle.
3. The low ventricular level includes sections at the level of the basal ganglia and thalamus.
4. The infra-ventricular level includes those at the level below the thalamus through the inferior part of the frontal and temporal lobes.

(Gado et al., 1992)

a) Supra-ventricular level: (Figs. 1.2 and 1.3).

Examples of the supra-ventricular level include the sections situated 6 to 7 cm above the center of the external auditory meatus, these sections do not contained parts of the lateral ventricles.

Rather, each hemisphere is represented by a central core of white matter called the centrum semiovale and a peripheral array of convolutions of gray matter. Each convolution (gyrus) contains an extension of the white matter in its center.

A higher plane in the supra-ventricular level transects the para-central lobule on the medial surface of the cerebral hemisphere and a lower plane transects the cingulate gyrus.

On the lateral surface, both sections transect the frontal gyri, the pre-central and post-central gyri (lines I a and I b in fig 1.1).

More posteriorly, these supra-ventricular sections transect the parietal lobe at the level of the inferior parietal lobule or superior parietal lobule, depending on the level of the section.

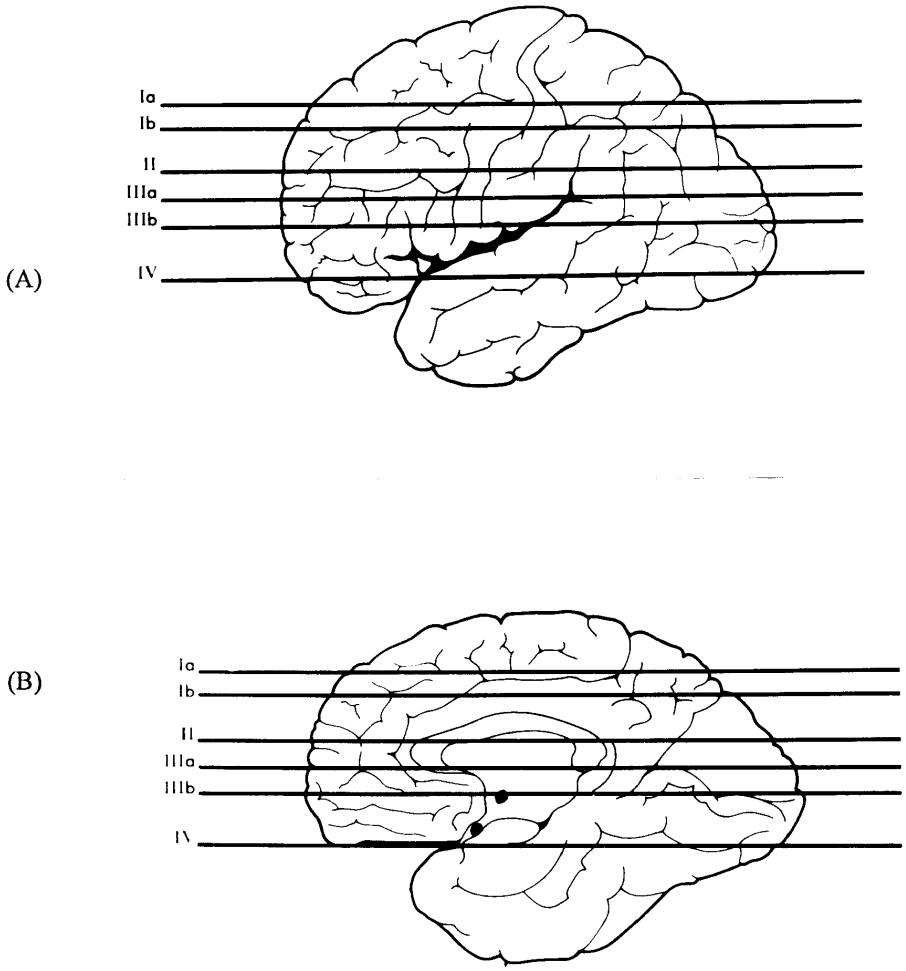
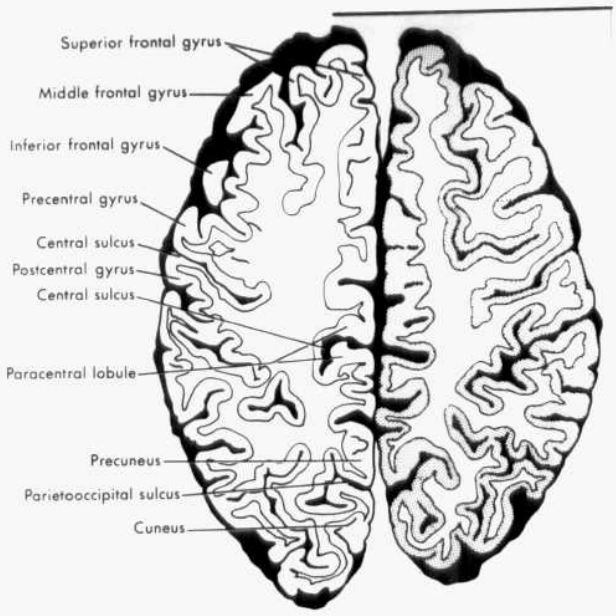


Fig. (1.1): Lines indicating levels of six selected axial sections in planes parallel to the canthomeatal line. Lines are drawn on a diagram of A) the lateral surface of the cerebral hemisphere and B) the medial surface of the cerebral hemisphere Ia, Ib, supraventricular levels; II, high ventricular level; IIIa, IIIb, low ventricular levels, IV, infraventricular levels. Adopted from *(Gado et al., 1992)*.

(A)



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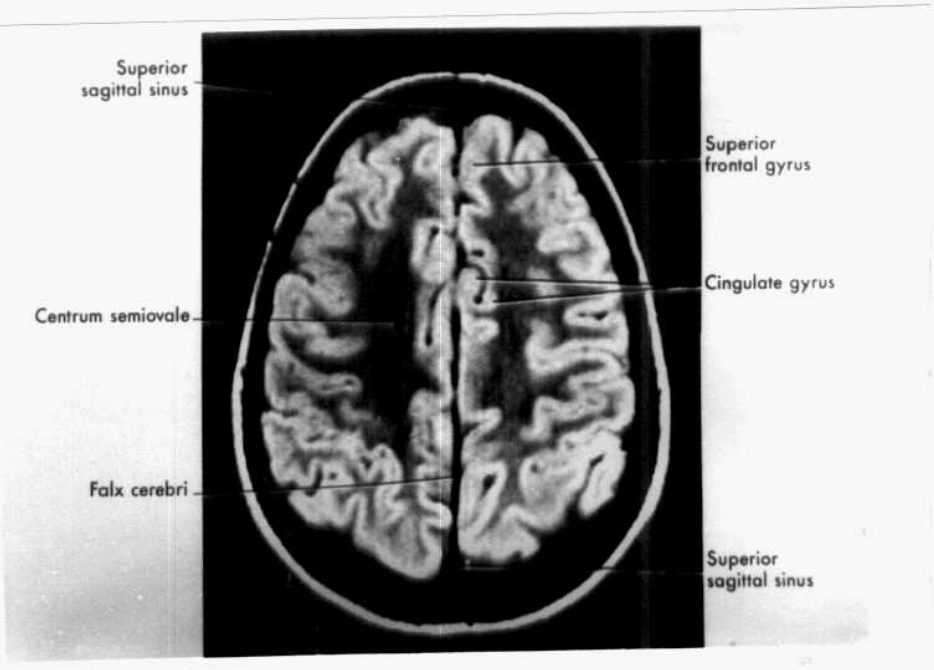
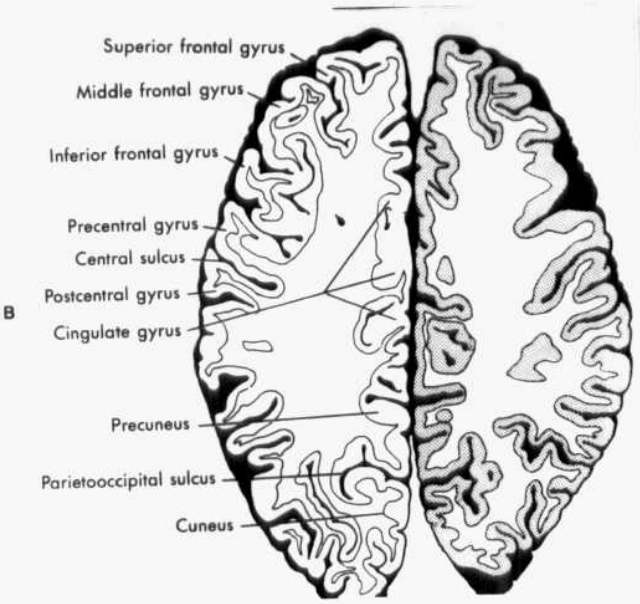


Fig. (1.2): Supraventricular level. A, line drawing of axial section of brain specimen made 6.8 cm above the canthomeatal line corresponding to line Ia, B, MR scan of a normal subject, obtained at a similar level. Adopted from *(Gado et al., 1992)*.

(A)



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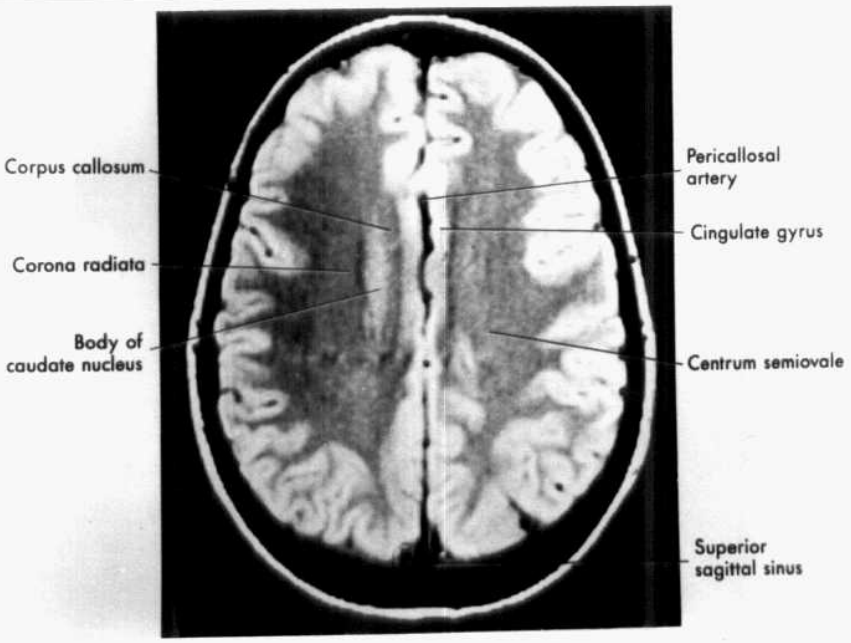


Fig. (1.3): Supraventricular level, A, line drawing of axial section of brain specimen made 6.1 cm above the canthomeatal line corresponding to line Ib, B, MR scan of a normal subject, obtained at a similar level. Adopted from *(Gado et al., 1992)*.

In the axial section, each cerebral hemisphere shows a curved lateral border and a flat medial border.

The central sulcus starts at the middle of the lateral border and dips into the hemisphere, taking a course medially and posteriorly and dividing the hemisphere into almost two equal halves.

In the higher supra-ventricular levels, the central sulcus is also identified on the medial surface, and it is surrounded by the para-central lobule.

The latter is delimited posteriorly by the marginal branch of the calloso marginal sulcus and anteriorly by the para-central sulcus. The lower supra-ventricular sections show the cingulate gyrus at the medial border, which is identified by the orientation of its cortex in an antero-posterior direction posterior to the para-central lobule (in the case of a higher section) or posterior to the cingulate gyrus (in the case of a lower-section). There is the precuneus outlined posteriorly by the deep parieto-occipital sulcus.

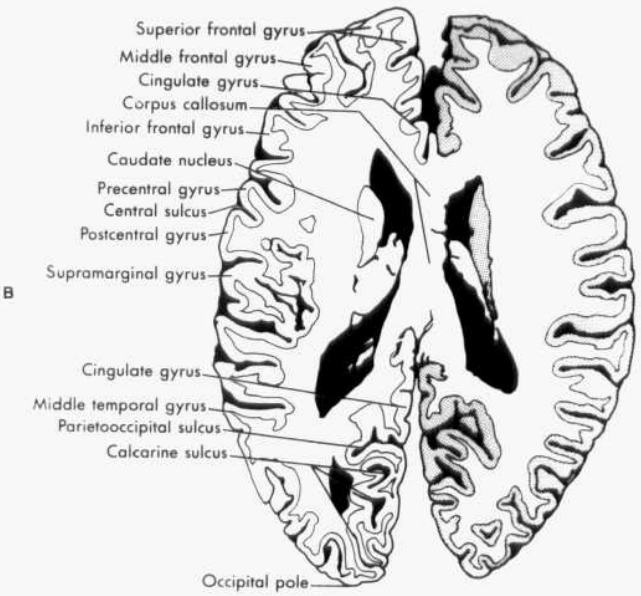
More posteriorly, the medial surface of the occipital lobe lead to the occipital pole. On the medial side of the hemisphere, the parietal lobe is well demarcated from the occipital lobe by an easily identified sulcus, on the lateral surface, no such anatomic demarcation lies between the two lobes.

b) High ventricular level: (Fig. 1.4).

Section in the high ventricular level transects the bodies of the lateral ventricles. An example is a section 4.7 cm above the external auditory meatus on the lateral surface of the cerebral hemisphere from front to back (line II in Fig. 1.1a).

The plane of the section transects the frontal gyri, the pre-central and post-central gyri, and the supra-marginal and angular gyri of the inferior parietal lobule. On the medial surface from front to back, the plane of the section transects the superior frontal gyrus, cingulate gyrus, corpus callosum, isthmus of the cingulate gyrus, precuneus of the parietal lobe and cuneus of the occipital lobe.

(A)



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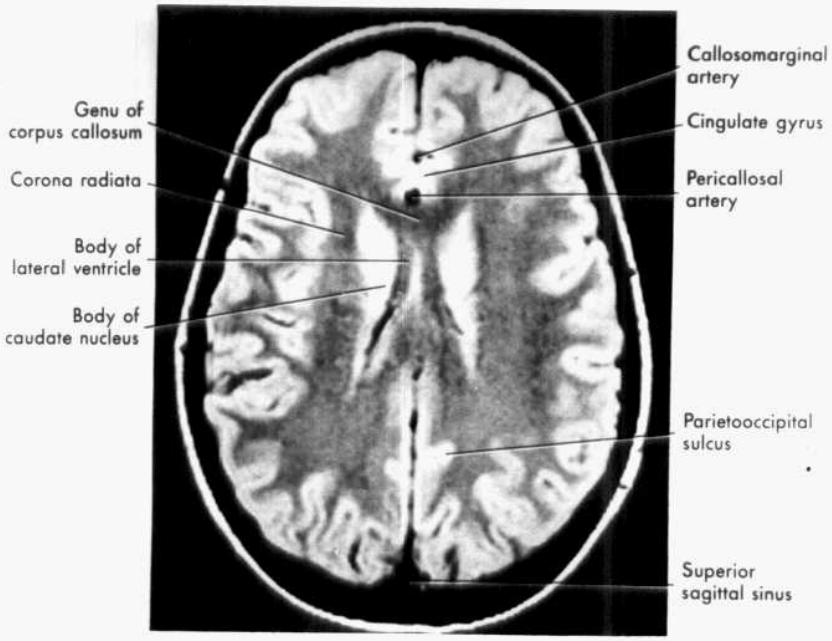


Fig. (1.4): High ventricular level. A, line drawing of axial section of brain specimen made 4.7 cm above the canthomeatal line corresponding to line II, B, MR scan of normal subject, obtained at a similar level. Adopted from *(Gado et al., 1992)*.

In the axial section, the inter-hemispheric fissure is interrupted in its middle third by the corpus callosum, which occupies the midline.

On each side of the corpus callosum, the body of the lateral ventricle appears an elongated C.S.F.-filled space oriented more-or-less parallel to the sagittal plane.

At its anterior and posterior ends, each lateral ventricle deviated slightly away from the midline, as the body of the ventricle joins the frontal horn and trigone respectively.

The tail of caudate nucleus forms a narrow strip of gray matter along the lateral wall of the body of the lateral ventricle.

Anterior to the corpus callosum, the medial border of each cerebral hemisphere consists of cingulate gyrus followed by the medial surface of the superior frontal gyrus, which extends onto the lateral surface. On the lateral side, the superior, middle and inferior frontal gyri and the pre-central gyrus form less than half of the border of the cerebral hemisphere.

The central sulcus is located on the lateral border of the section in approximately the same plane, in which the body of the lateral ventricles joins the frontal horn.

Behind the central sulcus is the post-central gyrus and the supra-marginal gyrus, both are parts of the parietal lobe. From this

point back, the lateral border of this section is indeed through the imaginary line joining the temporal, parietal lobes and the temporo-parietal junction.

More posteriorly, the lateral border of the section is formed by the temporo-parieto-occipital junction. From the occipital pole, the medial surface of the occipital lobe extends forward and is limited anteriorly by the deep parieto-occipital sulcus, which separates it from the lower end of the precuneus and from the isthmus of the cingulate gyrus. Thus the cingulate gyrus abuts on the corpus callosum anteriorly and posteriorly.

c) Low ventricular level: (Figs. 1.5 and 1.6).

Sections at the low ventricular level transect the lateral ventricles below the body. Therefore, they contain parts of the frontal horns and trigones of the lateral ventricles. Examples are sections made 3 to 4 cm above the external auditory meatus (lines III a and III b in Fig. 1.1) in these sections, the genu of the corpus callosum, frontal horns of the lateral ventricles and third ventricle form a prominent feature.

The lateral surface of each frontal horn is indented by the head of the caudate nucleus. Posterior to the third ventricle in the midline is the splenium of the corpus callosum or the tectum of the mid-brain, depending on the level of the section. On both sides, the third ventricle is surrounded by the thalami.

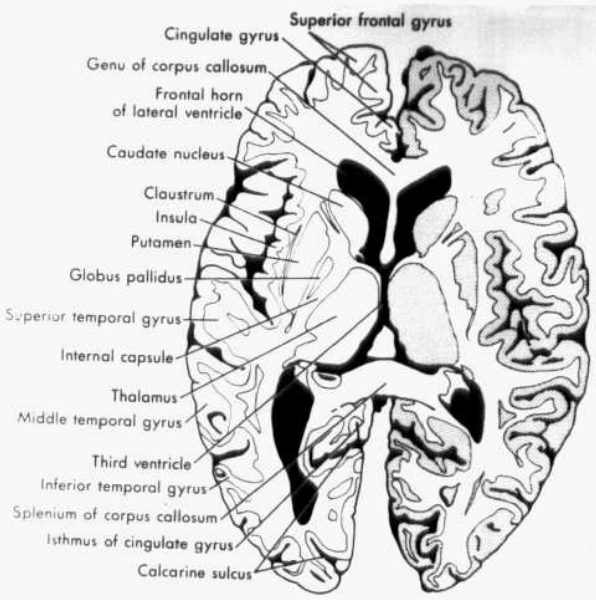
Lying lateral to the head of the caudate nucleus and the thalamus, the lentiform nucleus consists of a large mass of gray matter, called the putamen, and a smaller one on its medial side called the globus pallidus *(Gado et al., 1992)*.

Along the lateral side of the putamen is a strip of gray matter called the claustrum, which is the third part of the lentiform nucleus.

Another prominent feature in the low ventricular section is the insula, which is part of the cerebral cortex that has been buried by over-growth and the neighboring cortex of the frontal, parietal and temporal lobes, forming the frontal, parietal and temporal opercula respectively. The white matter between the putamen and the insula is divided by the claustrum into the external capsule on the medial side of the claustrum and the extreme capsule on its lateral side. The white matter on the medial side of the lentiform nucleus is the internal capsule. It is on L-shaped structure that consists of an anterior short limb and a posterior longer limb.

The anterior limb lies between the lentiform and caudate nuclei, the posterior limb between the lentiform nucleus and the thalamus.

(A)



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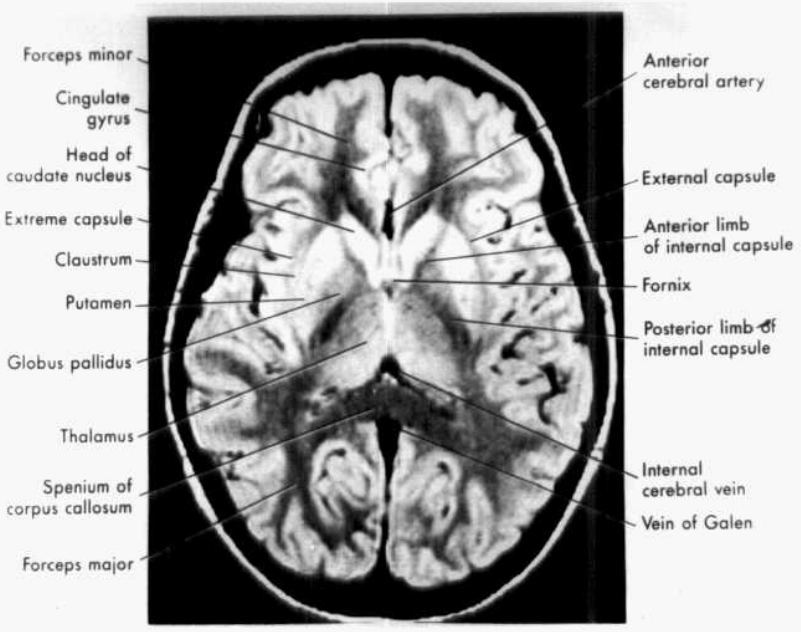


Fig. (1.5): Low ventricular level. A, line drawing of axial section of brain specimen made 4.1 cm above the canthomeatal line corresponding to line IIIa, B, MR scan of a normal subject, obtained at a similar level. Adopted from *(Gado et al., 1992)*.

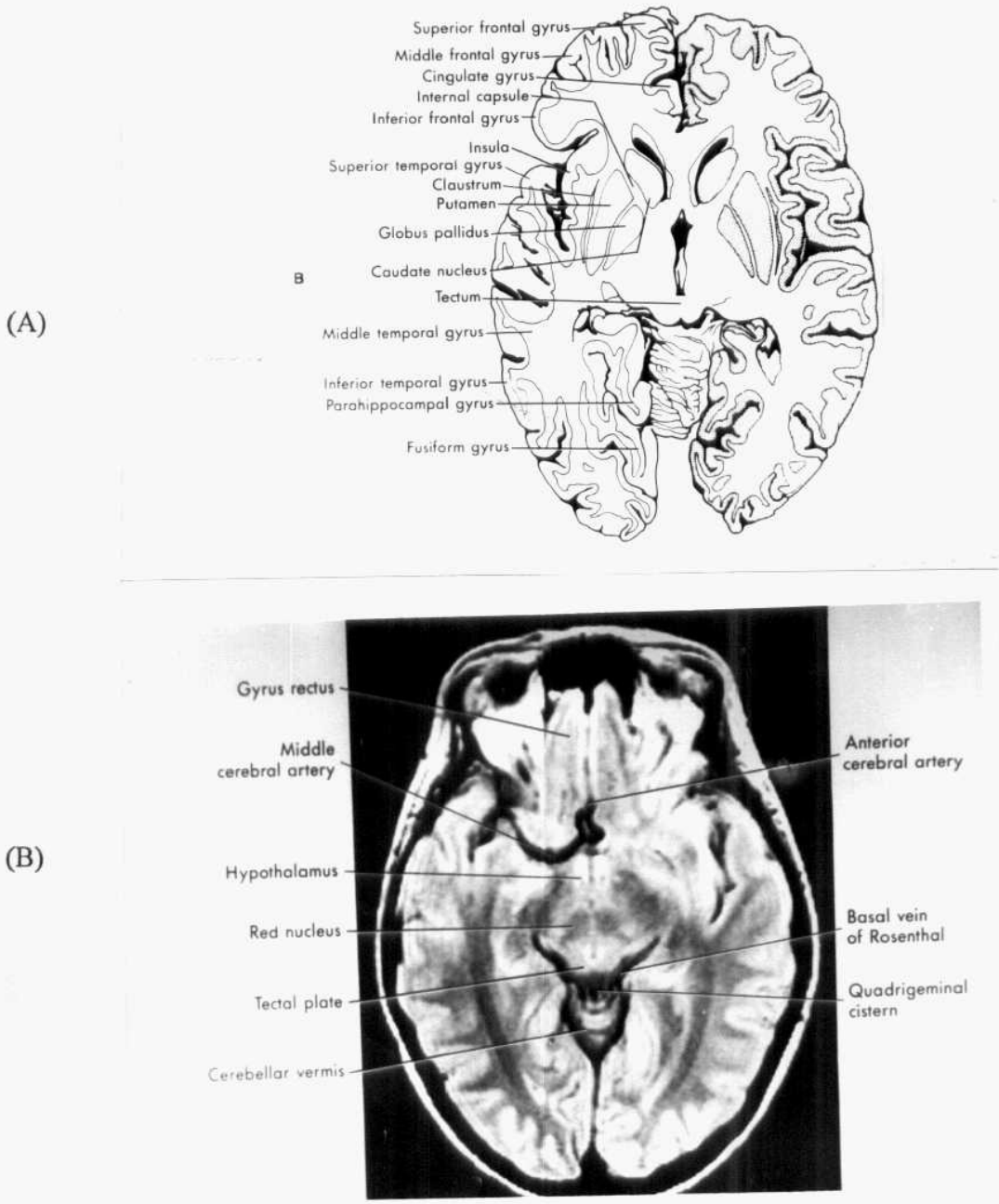


Fig. (1.6): Low ventricular level. A, line drawing of axial section of brain specimen made 3.4 cm above the canthomeatal line corresponding to line IIIb, B, MR scan of a normal subject, obtained at a similar level. Adopted from *(Gado et al., 1992)*.

The junction between the anterior and posterior limbs of the internal capsule is the knee or genu. On the lateral aspect of the section, the fissure of sylvius divided the lateral border into two almost equal halves. The anterior half is formed by the superior middle and inferior frontal gyri and the frontal and parietal opercula. Posterior to the fissure of sylvius, the lateral border of the section is formed by the superior, middle and inferior temporal gyri and more posteriorly by the occipital lobe from which it is separated by an ill-defined imaging line.

The inter-hemispheric fissure in the sections of the low ventricular level consists of a smaller anterior part and a larger posterior part, separated by several midline structures:- Genu of the corpus callosum, frontal horns, third ventricle and splenium of the corpus callosum (or tectum of the mid-brain depending on the level of the section). At the anterior part of the section, the medial border of the cerebral hemisphere is formed by the superior frontal gyrus and cingulate gyrus from front to back. At the posterior part of the section, the medial border of the cerebral hemisphere is formed by the occipital lobe separated from the splenium of the corpus callosum by the isthmus of the cingulate gyrus *(Stark et al., 1992)*.

d) Infra-ventricular level: (Fig. 1.7).

An example of the infra-ventricular level is a section 2 cm above the external auditory meatus (line I.V. in Fig. 1.1), this section

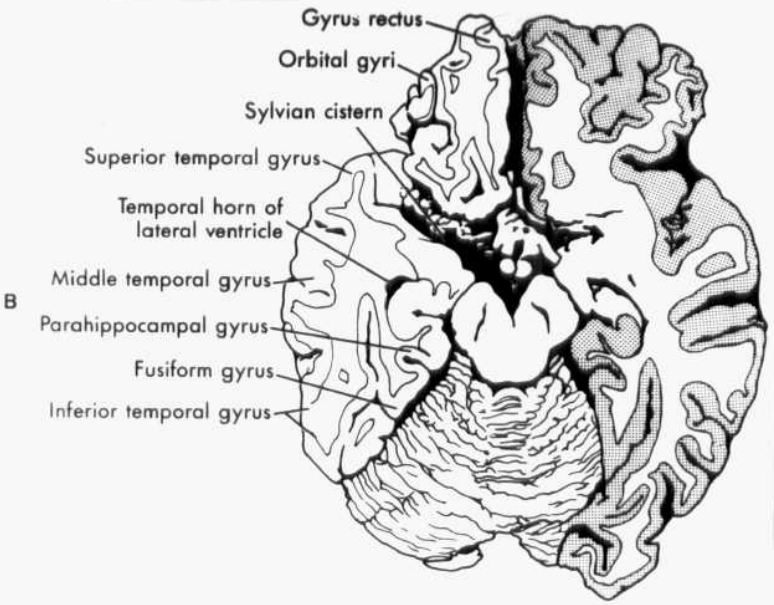
does not contain parts of the frontal horn, trigone, or body of the lateral ventricle. Rather, it may contain part of the temporal horn.

On each side, the lower parts of the frontal and temporal lobes are separated by the sylvian cistern, which is the stem of the lateral sulcus. The frontal lobe is formed by the gyrus rectus medially and the orbital gyri laterally. Farther laterally, the lower part of the inferior frontal gyrus may be included in the section.

The lateral surface of the superior, middle and inferior temporal gyri forms the lateral border of the section behind the frontal lobe. On the medial aspect, the inferior surface of the cerebral hemisphere courses forward from the occipital pole toward the midbrain. The inferior surface of the fusiform gurus and para-hippocampal gyrus form this border.

More anteriorly, the medial surface of the uncus abuts on the supra-sellar cistern of the optic tract, hypo-thalamic gray matter and the mammillary bodies.

(A)



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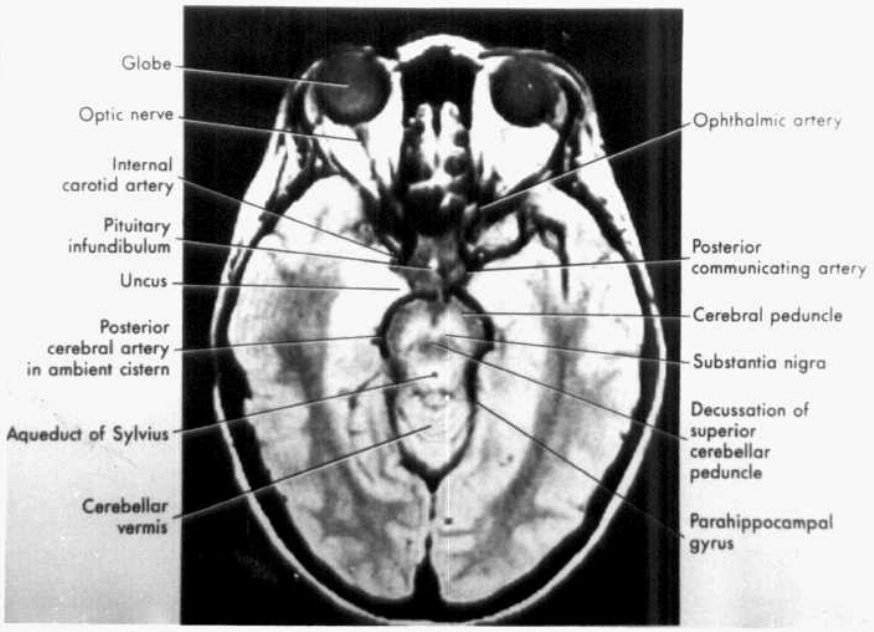


Fig. (1.7): Infraventricular level. A, line drawing of axial section of brain specimen made 2.1 cm above the canthomeatal line corresponding to line IV, B, MR scan of a normal subject, obtained at a similar level. Adopted from *(Gado et al., 1992)*.

2) Coronal Sections: (Fig. 1.8).

Coronal sections anatomy will be presented in four selected planes prependicular to the canthomeatal line (Reid's base line).

2. Mid-frontal plane.
3. Mid-thalamic plane.
4. Plane of the splenium and pineal body.
5. Plane of temporo-parieto-occipital junction.

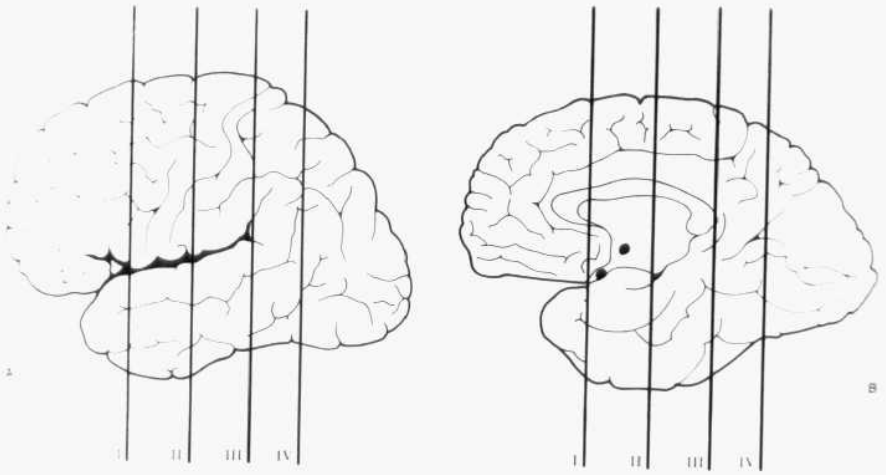
a) Mid-Frontal Plane: (Fig. 1.9).

The mid-frontal plans lies approximately 3 cm anterior to the external auditory meatus.

On the medial surface (line I in Fig. 1.8), the plane of this section transects the superior frontal gyrus, cingulate gyrus, corpus callosum and gyrus rectus. Thus in the cross section of the brain at this plane, the inter-hemispheric fissure is interrupted by the corpus callosum and the septum pellucidum between the frontal horns.

Above the corpus callosum, the inter-hemispheric fissure is bordered on both sides by the medial surfaces of the superior frontal and cingulate gyri. Below the septum pellucidum, the inter-hemispheric fissure is bordered on both sides by the medial surfaces of the gyri recti.

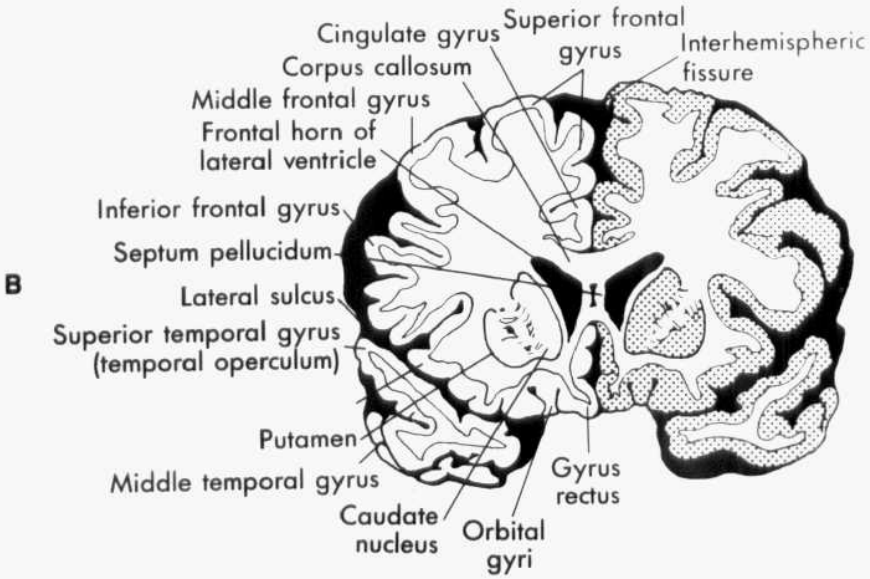
(A)



(B)

Fig. (1.8): Lines indicating four selected coronal planes perpendicular to Reid's baseline. Lines are drawn on a diagram of A, the lateral surface of cerebral hemisphere and B, the medial surface of cerebral hemisphere.
I, Midfrontal plane II. Midthalamic plane
III, plane of splenium and pineal body
IV, Plane of temporoparietooccipital junction
Adopted from *(Stark et al., 1992)*.

(A)



(B)

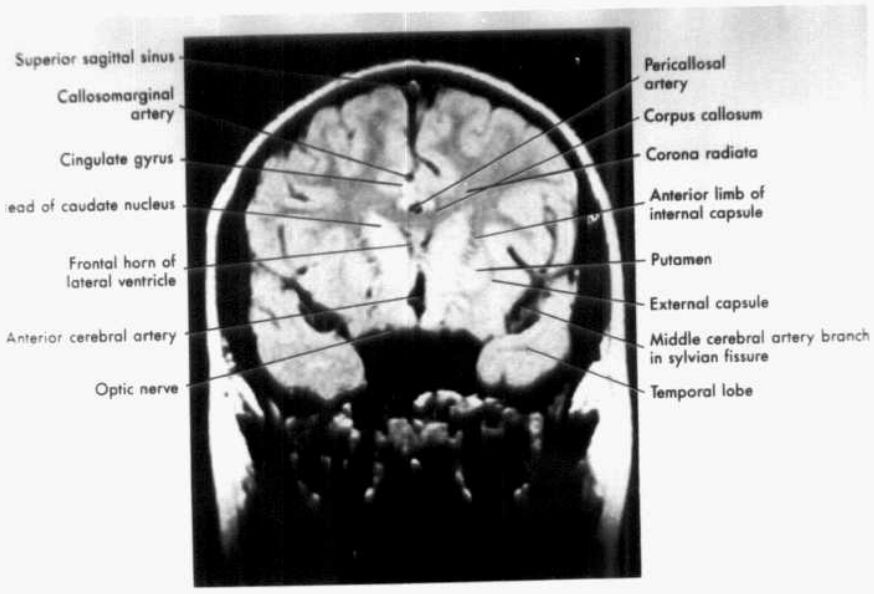


Figure 19-36

Fig. (1.9): Midfrontal plane. A, line drawing of coronal section of brain specimen made 2.9 cm in front of the external auditory meatus, corresponding to line I, B, MR scan of a normal subject, obtained at a similar plane. Adopted from *(Stark et al., 1992)*.

Whereas the frontal lobes abut on one another at the inter-hemispheric fissure, the temporal lobes are separated from one another by a gap occupied by the sella turcica and the cavernous sinuses.

On the lateral surface of the cerebral hemisphere, the plane of this section transects the superior, middle and inferior frontal gyri, the lateral sulcus and lateral surface of the anterior part of the temporal lobe.

In the cross section of the brain at this plane, the lateral sulcus is a prominent structure separating the hemisphere into a greater superior part formed by the frontal lobe and a lesser inferior part formed by the temporal lobe. The lateral surface of the inferior frontal gyrus forms the frontal operculum, whereas the lateral surface of the superior temporal gyrus forms the temporal operculum. The insula lies at a deeper plane.

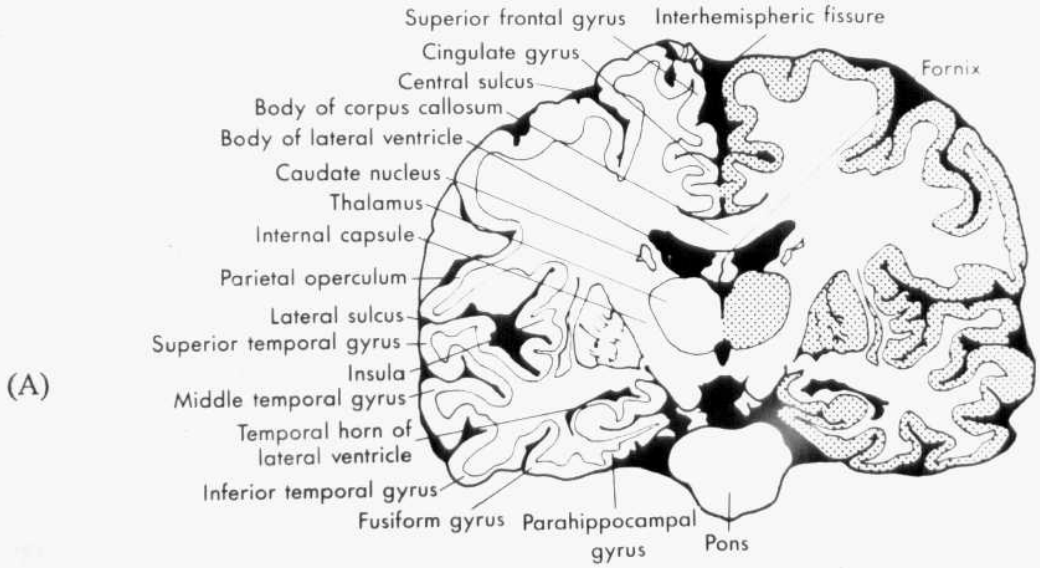
Between the insula and the frontal horns of the lateral ventricles, the caudate nucleus and the putamen form two masses of gray matter connected together by strands of gray matter and together are called the corpus striatum *(Gado et al.,1992)*.

b) Mid-Thalamic Plane: (Fig. 1.10).

An example of the mid-thalamic plane is a section of the brain approximately 5 cm anterior to the external auditory meatus. On the medial surface (line II in Fig. 1.8). The plane of this section

transects the posterior end of the superior frontal gyrus (or the anterior end of the para-central lobule), cingulate gyrus, corpus callosum and third ventricle.

Thus in the cross section of the brain at this plane, the inter-hemispheric fissure occupies only the upper part of the midline, where it is bordered by the medial surface of the superior frontal and cingulate gyri of the cerebral hemispheres. The corpus callosum lies at the bottom of the inter-hemispheric fissure.



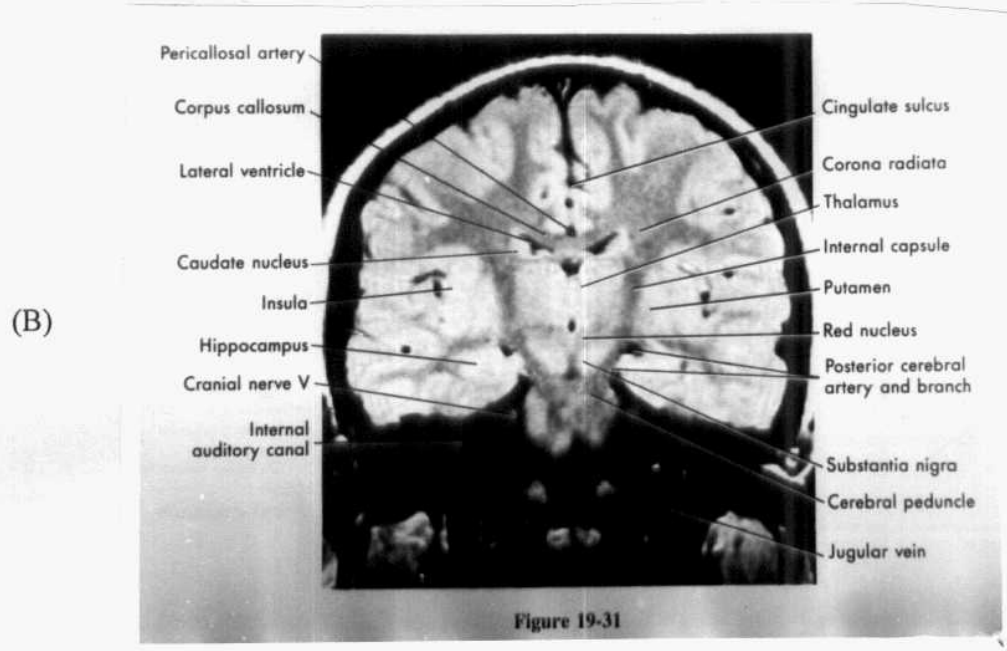


Fig. (1.10): Midfrontal plane. A, line drawing of coronal section of brain specimen made 0.5 cm in front of the external auditory meatus, corresponding to line II, B, MR scan of a normal subject, obtained at a similar plane. Adopted from *(Stark et al., 1992)*.

Below the corpus callosum, the bodies of the lateral ventricles lie above the third ventricle, which is surrounded on both sides by the two thalami.

Below that level, the gap between the temporal lobes is occupied by the anterior part of the brain stem.

On the lateral surface of the cerebral hemisphere, the plane of this section transects the lateral surfaces of the superior frontal gyrus, pre-central gyrus, post-central gyrus, fissure of sylvius and lateral surface of the temporal lobe. In the cross section of the brain at this plane, the lateral border of the section consists of the lateral surface of the frontal lobe in its upper third, the parietal lobe in the middle third and the temporal lobe in the lower third.

The central sulcus separates the upper, middle third and the fissure of sylvius separates the middle and lower thirds. The insula lies at a deep plane.

Unlike in the mid-frontal plane, where the head of the caudate nucleus forms a prominent part of the deep gray matter mass, in the mid-thalamic plane the tail of the caudate nucleus is a small structure at the lateral border of each ventricle. This is the part of the caudate nucleus that forms a narrow elongated strip along the lateral wall of the body of the lateral ventricle in the axial plane at the high ventricular level. In the cross section of the brain at the mid-thalamic level, the lentiform nucleus forms a large mass of gray

matter separated from the thalamus by the white matter of the posterior limb of the internal capsule. The temporal horn lies inferior to the lentiform nucleus in the medial inferior part of the temporal lobe.

The inferior surface of the temporal lobe courses medially and upward. It is formed by the inferior temporal gyrus lateral and by the fusiform and para-hippocampal gyri medially *(Stark et al., 1992)*.

c) Plane Of Splenium And Pineal Body: (Fig. 1. 11).

This plane lies approximately 2 cm posterior to the external auditory meatus. An example of this is a section taken 1.8 mm behind the external auditory meatus. On the medial surface of the cerebral hemisphere (line III in Fig. 1.8), the plane of this section transects the para-central lobule, cingulate gyrus and splenium of the corpus callosum.

Thus in the cross section of the brain at this plane, the inter-hemispheric fissure is seen in the upper part of the midline and is outlined by the medial surface of the para-central lobules and cingulate gyri. The splenium of the corpus callosum lies at the bottom of the inter-hemispheric fissure.

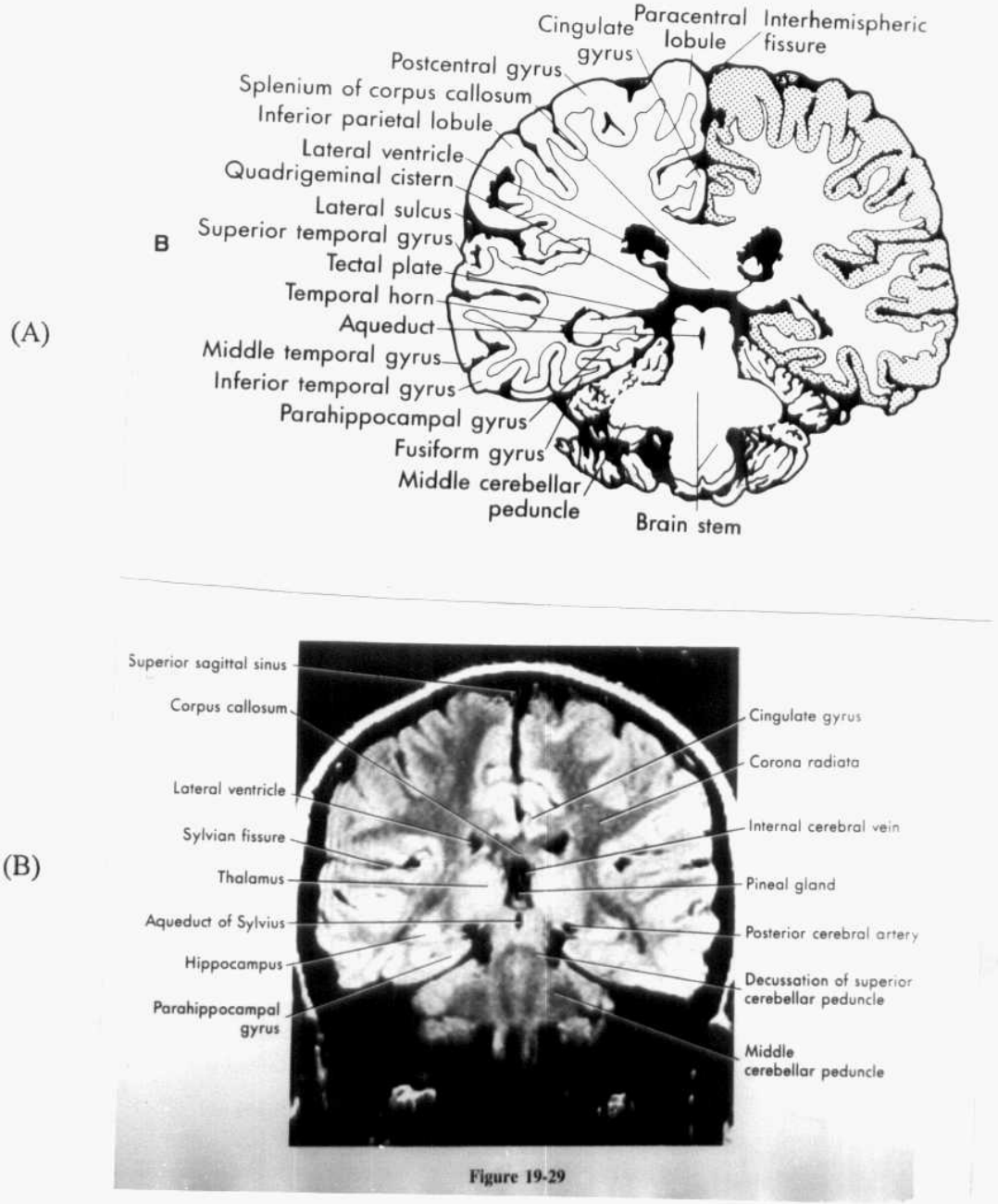


Fig. (1.11): Plane of splenium and pineal body. A, line drawing of a coronal section of a brain specimen made 1.8 cm behind the external auditory meatus, corresponding to line III, B, MR scan of a normal subject, obtained at a similar plane. Adopted from *(Stark et al., 1992)*.

Below the splenium, the C.S.F. space of the quadrigeminal cistern separates the splenium from the tectum of the mid-brain. The mid-brain appears as a column of brain tissue continuous with the remainder of the brain stem below with two lateral extensions formed by the middle cerebellar peduncles. The aqueduct is seen in the midline.

The relationship of the splenium and tectum corresponding to the appearance of these two structures in the low ventricular levels. The posterior parts of the lateral ventricles lie on each side of the splenium of the corpus callosum and are away from the midline, unlike the appearance in the mid-thalamic level. The diversion of the posterior parts of the lateral ventricles correlates with the appearance previously described in the axial section at the high ventricular level.

On the lateral surface of the cerebral hemisphere, the plane of this section transects the upper end of the central sulcus, post-central gyrus, inferior parietal lobule and lateral surface of the temporal lobe.

Thus in the cross section of the brain at this plane, the lateral border is formed almost entirely by the parietal and temporal lobes, but no part of the frontal lobe.

The lateral surface of the parietal lobe forms the larger part of the lateral border of the hemisphere in this section.

The fissure of sylvius, identified by its deep extension into the cerebral hemisphere, separates the parietal and temporal lobes on the lateral surface. The inferior surface of the temporal lobe extends medially and up and is formed by the inferior temporal fusiform and para-hippocampal gyri.

The temporal horn lies in the depth of the temporal lobe at a higher plane relative to the inferior surface compared to the mid-thalamic section.

d) Plane Of Temporo-Parieto-Occipital Junction: (Fig. 1.12).

The plane of the temporo-parieto-occipital junction lies approximately 3.5 cm behind the external auditory meatus.

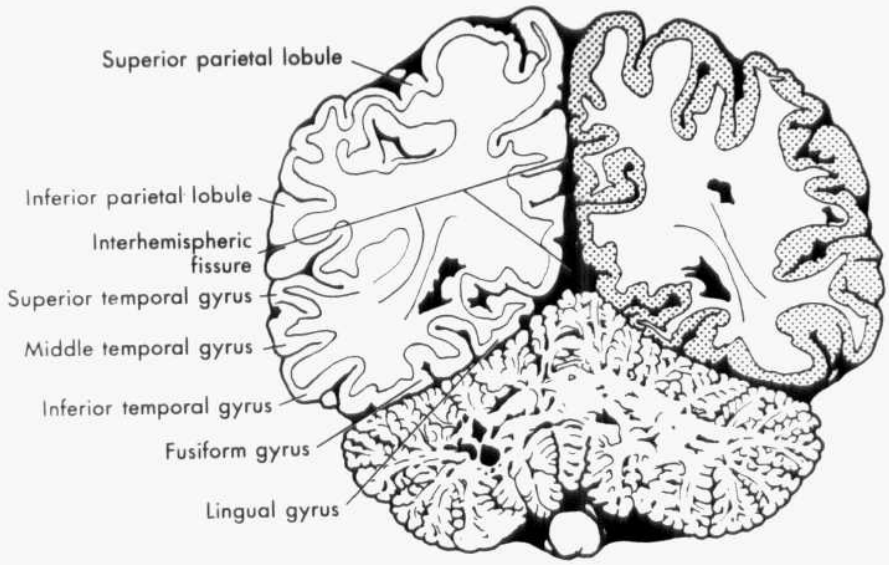
On the lateral surface of the cerebral hemisphere, the plane of this section (line IV in Fig. 1.8) transects the superior parietal lobule, temporo-parietal junction and lateral surface of the temporal lobe. Thus in the cross section of the brain at this level, two third of the lateral border of the cerebral hemisphere belongs to the parietal lobe and the lower third belongs to the temporal lobe, but demarcation between the two lobes is not as obvious as in the previous plane. On the medial surface of the cerebral hemisphere, the plane of this section transects the precuneus of the parietal lobe and the cuneus of the occipital lobe.

Thus in the cross section of the brain at this level, the medial border of the cerebral hemisphere is a continuous line, and the

inter-hemispheric fissure separates the two hemispheres completely with no crossing fibers of the corpus callosum in between. At this level, the falx cerebri and tentorium cerebelli are in continuity.

The inferior surface of the cerebral hemisphere is formed by the temporo-occipital junction from medial to lateral; therefore, this inferior surface is formed by the lingual gyrus, fusiform gyrus and inferior temporal gyrus. The occipital horn of the lateral ventricle lies in the depth of the cerebral hemisphere, slightly below its central *(Gado et al., 1992)*.

(A)



(B)

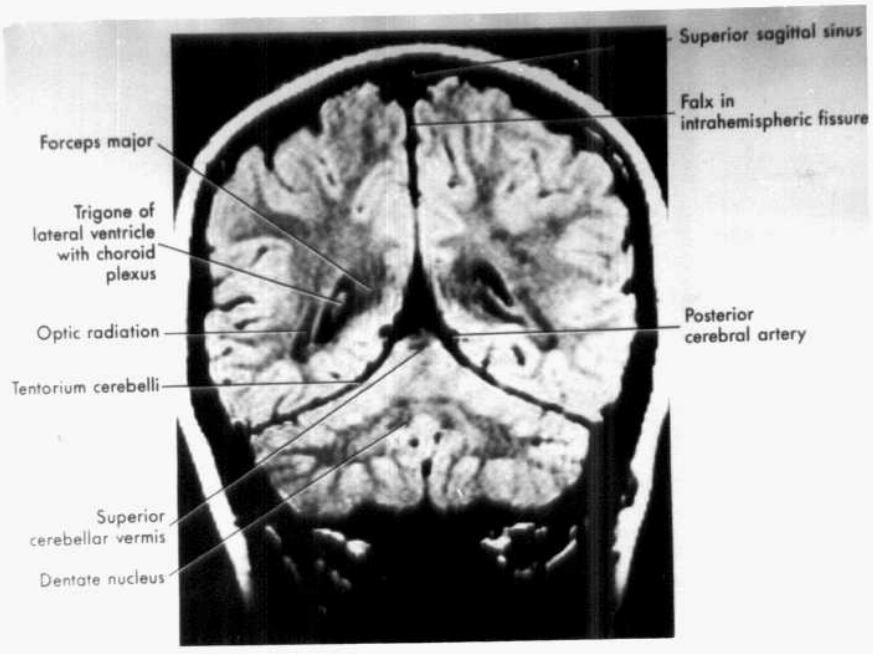


Fig. (1.12): Plane of the temporoparietooccipital junction. A, line drawing of a coronal section of brain specimen made 3.5 cm behind the external auditory meatus corresponding to line IV, B MR scan of normal subject, obtained at a similar plane. Adopted from *(Stark et al., 1992)*.

Anatomy and Normal MRI Appearance of White Matter

Functional Anatomy of White Matter:

The white matter consists of 3 kinds of myelinated nerve fibers:

A) Projection fibers.

B) Association fibers.

C) Commissural fibers.

A) The projection fibers pass into 2 opposite directions, to the cortex from the thalamus (thalamo-cortical radiation) and from the cortex to lower centers. The thalamo-cortical radiation includes sensory, visual and auditory radiation. The sensory fibers arise from the posterior ventral nucleus of the thalamus, pass through the posterior half of the posterior limb of the internal capsule to the sensory area of the parietal lobe. The optic fibers arise from the lateral geniculate body of the thalamus, passing through the retro-lentiform part of the internal capsule to the visual area in the occipital lobe. The auditory fibers arise from the medial geniculate body of the thalamus, passing through the sub-lentiform part of the internal capsule to the auditory area in the temporal lobe. The other types of the projection fibers are fibers that pass from the cerebral cortex to the lower centers and these include pyramidal and extra-

pyramidal tracts. The pyramidal tract is the motor pathway, which pass from the motor area of the brain to the motor nuclei of the cranial nerves and the anterior horn cells of the spinal cord. These fibers form pyramids in the medulla. In other words, the pyramidal tracts include the cortico-spinal tracts that pass through the genu and anterior half of the posterior limb of the internal capsule. The lateral cortico-bulbar tract to the motor nuclei of the cranial nerves 5,7,9,10,11 and 12 and the medial cortico-bulbar tract to the motor nuclei of the cranial nerves 3,7 and 6. The extra-pyramidal tracts include all motor connections whose fibers do not pass through the pyramids in the medulla. It arises from the pre-motor area of the cerebral cortex, basal ganglia, sub-thalamus and brain stem to the anterior horn cells. The pyramidal fibers are responsible for fine and precise movement, while the extra-pyramidal fibers are responsible for the gross synergistic movement.

B) The association fibers connect different cortical areas in the same hemisphere. They include the short association fibers (arcuate fibers) just deep to the cortex and the long association fibers.

C) The commissural fibers connect a part of the C.N.S. on one side with the same part on the other side. It includes the corpus callosum, the anterior, the middle, the posterior and the hippocampal commissures. The corpus callosum is the largest

and chief commissure, which connect all areas of the two cerebral hemispheres except the right and left temporal lobes. It consists of rostrum, genu (anterior part), body and splenium (posterior part). The anterior commissure, which pass anterior to the third ventricle, connect the olfactory bulbs and tubercles. The middle commissure passes through the third ventricle and connects the two thalami. The posterior commissure is the habenular commissure and pass posterior to the third ventricle. The hippocampal commissure connects the two fornices, which are formed by the axons of the cells of the hippocampus, and which end in the mammillary bodies of hypothalamus. The hippocampal gyrus and uncus are concerned with the sense of smell *(El Rakhawy, 1975)*.

Normal M.R.I. Appearance Of The White Matter

With the addition of Magnetic Resonance Imaging (M.R.I.) to the diagnosis armamentarium, we are able for the first time to closely follow maturation and myelination of the neonatal and infant brain.

An awarness of the chemical properties of myelin and the effect of these components on the T_1 and T_2 relaxation times of the water in the white matter of the brain can help us to understand the rate of myelination on the image produced. Myelin is composed of

double layer of lipids (cholesterol and glycolipidis) and large proteins *(Braung 1984)*.

Cholesterol (fat) has a short T_1 and proteins will also decrease the T_1 value of water. The result is a shortening of T_1 and therefore an increased intensity (increased brightness), on a T_1 weighted M.R. image, myelin lipids are hydrophobic, thus when myelination occurs; there is a loss of brain water. This results in a decreased in the T_2 value in the white matter and as a result a decreased intensity (darker) appearance on T_2 weighted image, thus myelination will be seen as an area of increased intensity (bright) on T_1 weighted image and decreased intensity (dark) on T_2 weighted images.

Tissue contrast in M.R.I. depends on differences in mobile hydrogen protons. Although lipids are rich source of hydrogen protons, the hydrogen protons in brain lipids are immobile.

Conversely water is rich in mobile hydrogen protons. In comparison to gray matter, white matter is relatively poor in water (a substance rich in mobile hydrogen protons) and relatively rich in myelin lipid (a substance lacking mobile hydrogen). White matter therefore has T_1 and T_2 relaxation times that are shorter than gray matter *(Doyle et al., 1981)*.

Milestones to look for on both T_1 and T_2 weighted images are presented in table (1) and table (2). Myelination of the frontal

white matter is seen as decreased intensity at 14 months of age and the appearance of brain in adult-like on T_2 weighted images at 18 months.

Table (1) milestones:

Normal myelination (1→ 6 months)

(T_1 weighted images at 1.5 T) from *(Barkovich, 1988)*.

Cerebellar white matter = 3 months

Splenium = 4 months

Genu = 6 months

Table (2) milestones:

Normal myelination (T_2 weighted images at 1.5 T) from *(Barkovich, 1988)*.

Splenium = 6 months

Genu = 8 months

Anterior limb of internal capsule = 11 months

Frontal white matter = 14 months

Adult pattern = 18 months

Summary of myelination finding

Age	Structures myelinated on T_2 WI	Gray - white matter differentiation	
		T_1 WI	T_2 WI
Birth	Dorsal mid-brain, superior and inferior cerebellar peduncles	Infantile	Infantile
1 month	Ventro-lateral thalamus, posterior limb of internal capsule	Infantile	Infantile
3 months	Optic chiasma and tract optic radiation, middle cerebellar peduncle	Iso-intense	Infantile
8 months	Centrum semiovale, anterior limb of internal capsule	Early adult	Iso-intense
10 months	Parieto-occipital white matter	Early adult	Iso-intense
1 year	Fronto-temporal white matter	Early adult	Early adult
2 years	Peripheral white matter	Adult	Adult

In the infant, there is a reversal of the usual adult pattern of gray matter - white matter contrast seen on the T_2 weighted M.R. scan.

Dietrich et al. (1988) remarked that it is important that radiologists are familiar with this pattern and recognize it is a normal phenomenon so as not to confuse this infantile appearance, with dysmyelinating or demyelinating disease, oedema or other pathologic processes. This appearance and its gradual reversion to the adult pattern correlated with an initially greater water content of unmyelinated white matter followed by subsequent water loss occurring during the myelination process as myelin is relatively hydrophobic.

The normal sequence and pattern of myelination has been the subject of a number of studies *(Dietrich et al., 1988),* described three pattern of gray - white matter differentiation on the M.R. scans, the infantile, the isointense and the early adult. On T_2 weighted spin-echo M.R. scan, they assigned grades of signal intensity from 0 to 10 (0 = the intensity of air, and 10 = intensity of subcutaneous fat) to various areas of known gray and white matter. Subject were normal infants and children ranging in the age from 4 days to 36 months. The infantile pattern was seen from 6 days to 6 months. In this pattern, white matter is two grades or more higher than gray matter. In the isointense pattern seen from 8 → 12 months of age, the gray and white matter differ in intensity by less than two grades.

In the 10 → 31 months of age groups, the early adult pattern prevails in which the gray matter is two or more grades higher than white matter. Another study by *(Barkovich et al., 1988)* determined that for any given location, myelination is generally recognizable on T_1 weighted sequences before it is seen on T_2 weighted images. They further noted that myelination proceeded from caudal to cephaled and from dorsal to ventral. This article lists myelination milestones on T_1 and T_2 weighted sequences for a number of brain locations using a 1.5. Tesla M.R. scanner.

Several examples are:

Cerebellar white matter is recognized as myelinated on T_1 WI at 4 months and on T_2 WI at 5 months, central occipital white matter is myelinated on T_1 WI at 5 months and on T_2 WI at 14 months, the genu of the corpus callosum shows myelination at 6 and 8 months for T_1 and T_2 weighted sequences respectively.

They further stated that the white matter in the area around the trigone of the lateral ventricles may not myelinate in some normal patients until the age of 10 years. Therefore, one must not necessary considers the isolated finding of high signal intensity in the peritrigonal areas as abnormal in young children.

The normal appearance on M.R.I. scan depends on the age of the patient and the pulse sequence used *(Barkovich et al., 1988)*.

In M.R.I. different radio frequency, pulse sequences may be used to produce images with varying dependence on proton density.

T_1 and T_2 images whose contrast largely depend on differences on proton density, T_2 show a low level of gray - white matter contrast in the brain and appear similar to x-ray and CT scan, although, there is very little signal from cortical bone and no artifact from bone. However, with strongly T_1 dependant images such as these produced with I.R. (Inversion Recovery) pulse sequence, a high level of differentiation can be seen between gray and white matter.

Since white matter appears white, and gray matter appears gray on these images and both sagittal and coronal scanning is readily achieved, the resemblance to gross anatomical sections is obvious *(Doyle et al., 1981)*.

The successful application of I.R. sequences has permitted the demonstration of gray – white matter contrast on a scale not possible with x-ray and CT scan. As a result anatomical structure such as the insular cortex, basal ganglia, internal capsule, brain stem can be accurately defined.

In addition, the gray – white matter interfaces of the brain provide a basis for localization of lesions *(Simmonds et al., 1983)*.

Arcuate "U" fibers are sub-cortical fibers at the marginal of gray and white matter. Occasionally they can be seen on T_2 WI at

1.5 Tesla in normal individuals as a ribbon of decreased signal intensity, owing to higher iron (ferritin) concentration in the arcuate fibers compared with cerebral gray or white matter. Their diagnostic importance concerns the sparing or involvement in various pathologic process and may improve diagnostic specificity in the evaluation of white matter disorders *(Drayer et al., 1987)*.

CLINICAL AND NEURO-PATHOLOGICAL FEATURE OF CEREBRAL DEMYELINATING DISEASES

1- Chronic Multiple Sclerosis (MS):

Is a demyelinating disorder that is characterized by inflammatory plaques of demyelination involving the white matter of central nervous system. Classically, it begins with a relapsing and remitting course. It is found predominately in Northen climates of Canada, Europe and United States. The incidence of M.S. is 40 to 80 individuals per 100.000 population and it is more prevalent in women than men 60 %: 40 %. More than 80 % of patients are between 18 and 50 years old *(Posers et al., 1989)*.

Early pathological stages of M.S. include the infiltration of lymphocytes and plasma cells around small vessels, the so-called peri-vascular cuff. This process follows a perivenous distribution, resulting in the peri-ventricular ovoid pattern. As the disease progress, these inflammatory cells are accompanied by damage to blood brain barrier and an influx of water and protein. Over time, fragmentation and lysis of myelin by hydrolytic enzyme in macrophages and ingestion of myelin by macrophages acceleration. This is followed by gliosis, which form asctrocystic plaques *(Dousset and Giang 1992)*.

Plaques are scattered through out the central nervous system, the presence of plasma cell in cerebro-spinal fluid implies

immuno-globulin secretion IgG. Cerebro-spinal fluid analysis are not specific for M.S. and many can be seen in other inflammatory diseases affecting the central nervous system *(Bornes et al., 1987)*.

Clinical Course of The Disease:

The disease usually begins between second and fifth decades. The patients might notice numbness dysethesia, burning sensation, there may be problems with balance, coordination or double vision. There may be difficulty walking or general motor weakness *(Kincaid et al., 1990)*.

Commonest sign impaired the visual acuity, nystagmus, asymmetric brisk tendons reflexes, positive babiniski sign and absent abdominal reflexes *(Hart & Sherman, 1982)*.

Variant of Multiple Sclerosis (MS):

A) Acute M.S. (Murburg type) is a variant of M.S. that resembles acute disseminated encephalomyelitis. Is usually associated with multiple foci of demyelination in central nervous system. The condition tends to follow a rapid course with death within a few months. Clinical manifestations are vomiting, headache, brain stem and optic nerve and spinal cord involvement *(Allen et al., 1992)*.

B) Concentric sclerosis (Balo type) is less common variant and it is characterized by alternating zones of myelinated and demyelinated tissue. Clinical feature includes early symptoms

suggesting a space occupying lesion and marked motor symptoms or psychic syndromes *(Allen et al., 1992)*.

2- Central Pontine Myelinolysis (CPM):

Is a demyelinating disease that affects the patient with history of alcoholism and liver disease. The disease has been associated with malnutrition, Addison's disease, renal failure and diuretic usage *(Adam et al., 1992)*. The affected patients suffered from alkalosis, hyponatremia and hypoxemia. Also rapid correction of hyponatremia cause pontine and extra-pontine myelinolysis. The osmotic changes of brain lead to alterations in water and electrolyte balance, this imbalance may injure oligodendrocyte, or damage myelin itself. Additionally, osmotic changes may lead to opening of the blood brain barrier, resulting in vascular injury. Injury that leads to oedema and demyelination *(Endo et al., 1981)*.

Earliest clinical finding associated with C.P.M. is actually a transient improvement in clinical symptoms associated with rapid treatment of the patient's hyponatremia, usually within 3 to 5 days after correction. Dysphagia, dysarthria, extra-ocular muscle weakness, seizure and mentality change ranging from a decreased in level of consciousness to coma. Shortly a spastic quadriparesis is seen. The recent report suggests an improved prognosis *(Wiederholt et al., 1977)*.

3- Viral and Post-Viral Cerebral Demyelinating Infection:

a) Progressive multifocal leuko-encephalopathy (PMLE):

Is a devastating demyelinating disease that affects immuno-compromised patient, in which a precipitate factor is immuno-suppression (irradiation, cytotoxic agents or adreno-corticosteroids or both and acquired immuno-deficiency syndrome (AIDS) *(Guilleux et al., 1986)*.

The disease occurs more commonly in males than female, between age 7 and 80 years of age *(Hseuh et al., 1988)*. The disease is caused by JC virus, a polyoma virus. It develops most frequently against a background of lympho-proliferative disease, although it is also linked both to acute and chronic myelocytic leukemia, to carcinomatosis and to benign disease of the reticulo-endothelial system. The virus infects the oligodendroglial cell leading to demyelination. The Jc virus is endemic in the United States *(Holman et al., 1991)*. Clinical presentation is variable. The disease is characterized by personality changes, confusion and decreased memory and may precede dementia. Motor and sensory deficits, ataxia, language disturbance and seizures. The prognosis is extremely poor. The death usually ensures within 6 months after becoming symptomatic *(Mark et al., 1989)*.

b) Lyme disease:

Is a demyelinating disease caused by Spirochete Borrelia Burgdorferi. The disease occurs world wise but is more commonly

in the New England States and the pacific coastal areas in the United States *(Finkel & Steere 1989)*. The pathogenesis of disease is complex by direct invasion by spirochete, a vasculitis or an immuno-complex mechanism *(Atlas et al., 1991)*. The brain may show axonal injury with peri-vascular inflammatory cell infiltration *(Steere et al., 1989)*.

Clinically, the patient may develop all or one of the following stages:

Stage I : Begins with a skin lesion like erythema.

Stage II : Consists with neuralgic and cardiac disorders.

Stage III : In which recurrent oligoarthritis develop, that is similar to juvenile rheumatoid arthritis.

c) Acute disseminated encephalomyelitis (ADEM):

Is a demyelinating disease that is of auto-immune origin. It usually occurs within 2 weeks after one of the childhood viral infection such as chicken pox or measles or after vaccination against rabies or small pox. The mechanism of white matter damage is believed to be vascular damage secondary to circulating immuno-complex deposition and complement activation *(Dunn et al., 1986)*. Prognosis is good and death occurs in 10 % to 20 % of clinical recognized *(Atlas SW et al., 1991)*.

The clinical course is monophasic and characterized by headache seizures and drowsiness may progress to lethargy even coma *(Atlas SW et al., 1986)*.

d) Viral encephalitis (VE):

Is a demyelinating disease caused by Herpes Simplex Virus (HSV-1). The lesion is due to reactivation at latent (HSV-1) in gasserian ganglia with subsequent spread to anterior and middle cranial fossa by branches of the trigeminal nerve *(Switzer et al., 1991)*. The virus causes a meningo-encephalitis with lympho-cystic infiltration. The prognosis is extremely fatal.

Clinical presentation is variable, the disease characterized by fever and headache are the most common early symptoms, other symptoms including seizures, meningeal signs, paraesthesia and aphasia *(Albertyn et al., 1990)*.

e) Cytomegalo virus encephalitis (CMVE):

The hallmark of CMV encephalitis (the owl's eye), which represent nuclear distension by viral inclusions with a surrounding halo. Their intra-nuclear inclusion may be present in ependymal cell, sub-ependymal astrocystes, oligodendrocyte and neurons course extensive destruction of white matter, gray matter and ependyma *(Post MJD et al., 1986)*. The infection with CMV may be sub-clinical in immuno-suppressed patients. Less often symptoms to develop over days to months with fever, seizure and altered mental status, dementia meningo-encephalitis vasculitis, cerebral hemorrhage and cranial neuropathy. Coma and death also be reported *(Dorfman et al., 1973)*.

f) AIDs – related white matter disease:

A demyelinating lesion is frequent finding of AIDs patient. The cause of this disorder a causative agent being human T – cell lymphotropic virus type III (HTLV III) known now as human immuno-deficiency virus (HIV) *(Sarngadharan et al., 1984)*.

HIV virus has been recognized to be neurotropic, replicating in multinucleated giant cells or macrophages. The polymorphic microglia are also frequently infected. The multinucleated giant cells are found scattered throughout the white matter or clustered within microglial nodules found in the cortex, basal ganglia and white matter. The brain stem and cerebellum may also include *(Petito et al., 1988)*.

Clinically, the patients have progressive cognitive impairment with behavior and motor disturbance, AIDs Dementia Complex (ADC) representing the clinical manifestation of subacute encephalitis. The subacute encephalitis is most common neurologic complication seen in AIDs *(Navia et al., 1986)*. After weeks to months, the patients were complaining from depression, a pathy, withdrawal, mild memory loss and dementia.

4- Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts And Leuko-Encephalopathy (CADASIL):

a) Age related:

A Virchow Robin Spaces (VR) or peri-vascular space: Is an invagination of subarachnoid space that accompanies the vessels as it courses from subarachnoid space into brain parenchyma. Small VR spaces is found in all age groups and represents a normal finding. Large VR space demonstrated 23 % under the age of 20 % and 33 % over the age of 60 *(Heier et al., 1989)*. VR spaces enlarge and become more numerous with increasing age, hypertension and dementia. VR spaces are found in the inferior one third of the basal ganglia. They are usually bilaterally symmetric and round. They can also be seen in the cortex and centrum semiovale.

b) Peri-ventricular hyperintensity:

Foci of increased signal intensity in the peri-ventricular white matter anterior to the frontal horns are a normal finding. Another age related normal finding seen in the peri-ventricular region is the terminal areas of non-myelination. They are found posterior and superior to the trigones. They are found normally in the first and second decades and occasionally does not stain for myelin, until the fourth decade *(Yakoklev et al., 1967)*.

Any extra-cellular water within the brain must be removed by ependyma of lateral ventricles and then excreted into ventricles via way of an active transport mechanism. Any pathological process

that would increase the extra-cellular fluid volume by disruption of the blood brain barrier, such as ischemia or infarct would increase the workload of the lateral ventricular ependyma. The maximum capacity of the transport system is exceeded, water begin to back up and create a band of hyperintensity around the lateral ventricles *(Boyko et al. 1989)*, demonstrates these areas to represent myelin pallor rather than demyelination.

c) Sub-cortical hyperintensity:

Fazekas et al. (1991) found hyperintensity in 11% of patents in 4th decade and 65% in the 7th decade. Most of these foci are unexpected or incidental. These lesions increase in frequency and size with age hypertension or a history of brain ischemia.

Bowen et al. (1990) analyzed peri-ventricular and sub-cortical hyperintensities in patients with dementia and found that the sub-cortical white matter lesion are more helpful than the peri-ventricular lesion in distinguishing vascular dementia from Alzheimer and normal aging. Peri-ventricular white matter lesions were present 100 % in both Alzheimer disease and vascular dementia, whereas sub-cortical lesion was found in 100 % of patients with multi-infarct dementia. With aging, a progressive decrease in cerebral blood floor and increase in arterio-sclerosis occurs, which are accentuated by hypertension. The resulting ischemia would affect the deep white matter, the affected white matter has a consistent triad of demyelination. Loss of axons and

fibrous thickening of the wall of arteries. The loss of myelin tends to the patchy with associated reactive gliosis *(Lotz et al., 1989)*.

d) Cerebral amyloid angiopathy (CAA):

CAA pathologically characterized by deposition of homogenous eosinophilic material in the media and adventia of the arterioles of the cortex and lepto-meninges *(Loes et al., 1990)*.

The distribution is focal and sporadic for unclear reasons. In addition, there is superimposed fibrinoid degeneration and micro-aneurysmal dilatations predispose the hemorrhage, with minor trauma or slight fluctuations in blood pressure *(Gray et al., 1985)*. The most common site involved were the centrum semiovale and deep peri-ventricular regions with sparing of corpus callosum and internal capsule.

e) Hyperintensive encephalopathy:

Is a syndrome that is characterized by a sudden sharp rise in blood pressure associated with headache, vomiting, nausea and altered mental status and can lead to stupor and coma. The rapidity with which the blood pressure rises and the degree of elevation relative to the base line blood pressure are two most important factor responsible for development of hypertensive encephalopathy *(Hause et al., 1988)*.

Pathologically, fibrinoid necrosis and thrombosis of arterioles along with parenchymal petechial hemorrhage (Chester et al., 1978).

f) Eclampsia:

Is a hypertensive disorder of pregnancy occurring after 20th weeks of gestation and is characterized by hypertension, peripheral oedema, protinuria and seizures. The neurologic complication includes headache, visual disturbance, focal neurological deficits and coma.

Richadr et al. (1988) suggests that there may be several mechanism, including vasculopathy, peri-vascular micro-hemorrhage and intra-cerebral hemorrhage.

Pathologically fibrinoid necrosis in the wall of arterioles and small arteries *(Lotz et al., 1989)*.

The most common area of involvement is cortical and sub-cortical white matter in occipital and posterior parietal lobes. They are bilateral and symmetric, other areas includes deep white matter and basal ganglia. They are not necessarily symmetric. Clinically, the lesion in deep white matter and basal ganglia correlate with mental status changes, whereas, sub-cortical lesions are frequently associated with visual disturbance *(Sanders et al., 1991)*.

g) Migraine:

There is initial vaso-constriction of intra- and extra-cranial arteries, which lead to ischemia, this ischemia is manifested clinically by the aura and neurologic deficit.

The lesions seen with migraine represent minute foci of ischemia owing to spasm or possibly break – through edema from loss of auto regulation following a migraine attack *(Osborn et al., 1991)*.

5- Iatrogenic Group of Cerebral Demyelinating Disease:

a) Radiation treatment:

The radiation treatment causes capillary endothelial damage that lead to breakdown of blood brain barrier and vasogenic edema. Cerebral blood flow may be reduced owing to heterogeneous endothelial hyperplasia, fibrinoid necrosis of penetrating arterioles and athero sclerotic-like changes in the large arteries *(Tsuruda et al., 1987)*.

Clinically, the patients may be a symptomatic or have signs of neurotoxicity characterized by varied degrees of irreversible dementia confusion, ataxia and psychomotor retardation. In general, the patients with the most severe form of neurologic compromise have the most extensive changes. Focal neurologic finding often correlate with region of focal signal changes with increasing age, the lesion are more common and widespread, this is probably due to existing age-related damage from radiation *(Tsuruda et al., 1987)*.

Sometimes, white matter develops small necrotic foci, vacuolation and petechial hemorrhage months to several years following radiotherapy. The focal abnormalities occur in areas that have received the highest dose of irradiation. Atrophy is commonly

depending on the severity of these changes, patient's age and total irradiation dose, these lesions may partially resolve, stabilize or progress over a protracted course of several months to several years. Eventually, this may lead to widespread, potentially fatal brain destruction characterized by coagulation necrosis *(Tsuruda et al., 1987)*.

b) FAHR's disease:

Is a familial disorder characterized by intra-cranial calcifications without abnormalities in the serum calcium or phosphate levels. The clinical presentation varies from a symptomatic to choreoathetoid movement in childhood and general progressive mental deterioration in adulthood. Pathologically, the calcification occurs in vessels wall and in the peri-vascular spaces of arterioles, capillaries and veins *(Scotti et al., 1985)*.

c) Sarcoidosis

The disease may causes patchy white matter lesions by invading the blood vessels wall and causing stenosis or occlusion of the lumen *(Greco and Steiner, 1987)*.

d) Toxic agents:

In chronic exposure or in acute larger doses related to industrial accidents or suicide attempts can cause leuko-encephalopathy. In same instances, the distribution is characteristic for the noxious agent e.g carbon monoxide, cyanide, hydrogen sulphide and ethylene glycol (Anti freeze) cause white matter

lesions. Methanol intoxication typically causes cortical blindness *(Dooms et al., 1987)*. In addition to cortical and cerebellar atrophy, multiple rounded white matter lesions have been documented in a group of chronic alcoholics in which other risk factors were excluded *(Gallucci et al., 1987)*.

TECHNIQUES OF MRI EXAMINATION

Soli et al. (1992) suggested the following techniques for routine examination of brain for white matter diseases:

The Mature Brain:

Screening the mature brain for white matter disease should start with T_1 weighted scout (T_1 WI) views in the midline sagittal projection, in order to quickly determine the level for further axial double-echo T_2 WI, which have proven to be most sensitive and the need to obtain a view of the anatomy in the sagittal plane *(Runge et al., 1984)*.

The sagittal view is of primary importance in the region of the foramen magnum, the 4th ventricle and the sylvian aqueduct. In addition to the axial images, coronal T_2 WI double echo-images should be obtained. They are helpful in determining whether small focal lesions are truly in the white matter. Also, precise peri-ventricular localization along the upper and lower surface of the ventricular system can be obtained. Often, subtle mass effect is best seen in the contour of the ventricles in the coronal view. Inversion Recovery (IR) T_1 WI are useful in special situations lesions in the brain stem are better seen and peri-ventricular and other lesions can be evaluated for T_1 WI morphology *(Young et al., 1986)*.

The Immature Brain:

T_2 WI of an immature brain should be obtained with sufficiently long repetition times to avoid effect on T_1 WI in the white matter decreasing its intensity and causing iso-intensity between gray and white matter. At higher field strengths (1.0 to 1.5 T) TR of 1.8 to 3.5 sec. Combined with sufficiently long echo delay (TE 80 to 160 m sec.) gives excellent gray matter differentiation. Exact scanning parameter for the time of physiologic cross over in the relaxation values of gray and white matter around eight months of age are not precisely known and may be slightly different for each individual, depending on the normal variation in the rate of development *(Nowell et al., 1987)*.

T_1 WI with short TE and TR spin-echo or IR sequence show progressive myelination and also give the best CNS to cerebro-spinal fluid (CSF) demarcation in coronal, axial and sagittal planes *(Johnson et al., 1987)*. MR relaxation times of gray and white matter can be reliably and reproducibly measured in vivo *(KJOS et al., 1985)*.

The T_1 WI value of cortical gray matter is approximately 701 m. sec. compared with 419 m sec. for sub-cortical white matter. The T_1 WI of (CSF) is approximately 2719 m. sec. The T_2 WI of cortical gray matter is 60 m. sec. and sub-cortical white matter has a T_2 WI of 53 m. sec., (CSF) has a T_2 WI of 166 m. sec. The proton density of gray matter is approximately 1.2 times that of white

matter. T_2 WI and proton density P.D sequences are the most useful spin-echo sequence in differentiating gray and white matter on the normal scan, and therefore most useful in detecting white matter abnormalities *(KJOS et al., 1985)*.

Drayer et al. (1986) and Curnes et al. (1988) point out the importance of recognizing that, on T2 WI sequences extremely low signal in the basal ganglia, which has been attributed to heavy iron concentration.

In the latter setting, the short T_2 WI reflects heavy myelination and fiber density and not iron deposition. These areas are distinguishable from areas of iron depositing by the fact that, unlike iron deposition, they appear as high signal on T_1 WI sequence and they can be seen with regularity in all normal patients over three years of age *(Curness et al., 1988)*.

Special Techniques:

- **Contrast enhancement:**

  The roles of contrast enhancement in evaluation of white matter abnormalities it assists in differential diagnosis and allows evaluation of activity and severity of white matter lesions, particularly in MS *(Grossman et al., 1986)*.

- **Motion Compensation Gradients (MCG):**

  (MCG) are used in high-field imaging to decrease artifacts from motion including C.S.F. pulsation, particularly in detection of

lesion at the level of basal cisterns. Contrast - to - noise ratio is improved between lesions and normal white matter and between gray and white matter, signal to noise ratio in white matter is also improved. The combined use of gradient and low bandwidth techniques will probably be routinely applied at high fields strength imaging in diagnosing white matter diseases *(Runge et al., 1987)*.

MRI manifestation of cerebral demyelinating diseases

1. Chronic Multiple Sclerosis (M.S.):

Often the appearance of M.S. in the brain can be described as multifocal lesion predominantly located in the peri-ventricular and deep in the white matter. *Ormerod et al. (1986)* found that peri-ventricular white matter adjacent to trigones and bodies at the lateral ventricles, most frequently affected (96% of patients), with a small decrease in frequency of lesions around the occipital 83% and frontal horns 73%. Lesions located around the third ventricle 34 % . Less frequently locations included the basal ganglia 25 % and internal capsule 11 %. Brain stem 68 %. White matter lesions are generally abundant in the centrum ovale and corpus callosum. However, the appearance of M.S. lesions on M.R. images is considerable variation in the size and shape of M.S. lesions. The lesions range from small and irregular to large and confluent *(Horowitz et al., 1989)*. The smallest lesions are punctuate in shape, when severe lesions are present, they may be round, angular, oblong, linear, confluent and lumpy. They extend from ventricular surface at

the right angle to ventricular surface (right angle demyelination lesion), and occupy in 86 % of M.S. cases *(Horowitz et al., 1989)*.

Composite feature of brain lesions characteristic for multiple sclerosis.

Paty et al., 1989	Three or more hyperintensities with one bordering the lateral ventricle.
Fazekas et al., 1991	Three or more hyperintensities and at least two of the following lesion characteristics. Size > 5 mm. Abutting the ventricular body. Infra-tentorial location
Barkhof et al., 1992	Communicative chance model for conversion to multiple sclerosis (80 % with all four features fulfilled): - Gadolinium enhancing lesion (at lest one). - Juxta cortical location (at least one). - Peri-ventricular location (at least 3). - Infra-tentorial location (at least one).

The plaques are hyperintense or intermediate (long TR., short TE) and T_2 WI (long TR ,TE) MR imaging series *(Wessbecher et al., 1992)*. The majority of MS lesions are hypointense, isointense on T_1 WI. Rarely the edge of T_1 WI lesions are hyperintense, these hyperintensities have been attributed to the presence of protein, lipid-laden macrophages and protein accumulation. There may be giant plaques of MS, which have the appearance of Tumor *(Davis et*

al., 1985). However, they may be differentiated from tumor by their lack of mass effect. Lymphoma may be difficult to differentiate from MS plaques because of its tendency to adjacent to ventricular surfaces and its prompt response to steroid *(Horowitz et al., 1989)*.

In more long-standing cases, cerebral atrophy develops. It is seen in up to 45 % of patients with clinically definite M.S. on the MR images, manifestation of atrophy are well demonstrated on T_1 WI *(Cobb and Mehringer, 1987)*. Change in the intensity of thalamus and putamen on T_2 WI in-patients with definite MS is an important observation recently published. This change is seen at high fields strength and correlates with increased ferritin, which causes local magnetic field and thus shortens T_2 WI *(Drayer et al., 1987)*. Plaques are also often seen within the body of the corpus callosum. In addition to demyelinating plaques within the corpus callosum, focal or diffuse corpus callosal atrophy may also be observed on the MR scan of patients with significant changes typical of MS *(Dietemann et al., 1988)*. Additionally, as pointed out by *Simon et al. (1986)*, this may be due to wallerian degeneration or the loss of axons within the corpus callosum secondary to demyelinating lesions in the peri-ventricular area of radiating white matter fibers.

Contrast Enhancement:

There is considerable difference of impression about the role of Gadolinium – DTPA in the images MS. Some feel that enhancement of MS plaques with para-magnetic contrast material

identifying active and subacute MS plaques *(Miller 1988 & Mattioli 1993)*. A chronic stable MS plaques do not enhance after intravenous administration of Gd – DTPA.

Grossman et al. (1989), showed that enhancement 10 minute post injection short TR short TE was most effective in detecting enhancement. The enhancement indicates blood brain barrier breakdown. The pattern of enhancement of lesion may be solid or ring enhancement. The lesion appear to extend to a maximum volume in approximately 4 weeks followed by a more gradual decline over 4 to 8 weeks, Over long interval 6 months to 2 years.

Cortico-steroid administration may have an effect on the size number or appearance of lesion. Many lesion continue to enlarge, other, may regress and disappears whereas most remain unchanged more over, severe report of decreased contrast enhancement after steroid therapy of MS *(Barkhof et al., 1992)*.

Variants of MS:

1. Acute MS: M.R.I. show scattered white matter lesions with a distribution comparable to that of chronic MS *(Davis and Robertson 1985)*.
2. Concentric sclerosis: The pattern of involvement is multifocal as in MS, but "U" fibers and gray matter spares *(Davis and Robertson, 1985)*.

2. Central pontine myelinolysis "CPM":

MRI demonstrated an area of decreased signal intensity on non-enhanced T_1 WI. The lesion however is much better identified on T_2 WI. The axial views demonstrated pontine lesions of various shape, may be triangular or trident shape. On sagittal views, the abnormality is usually oval in shape, and on coronal views, a curious bats wing configuration. A thin rim of normally preserved pontine tissue surrounding the focus of demyelination *(Miller et al., 1988)*.

Rippe et al. (1987) described the MR findings of extra-pontine myelinolysis, the thalamic regions appear to be the most common site of such extra-pontine involvement. The lesion appears hypointense on T_1 WI and hyperintense on T_2 WI. The lesion are well-defined symmetrical and not associated with mass effect. Gadolinium enhancement was identified around the periphery of the lesion, this finding is similar to acute multiple sclerosis *(Koch & Smith et al., 1989)*.

Enhancement of extra-pontine lesions has not been reported *(Rippe et al., 1987)*.

3. Viral and post-viral cerebral demyelinating infection

A) Progressive multifocal leuko-encephalopathy:

MRI demonstrated multiple asymmetric hyperintense lesions on long TR image. Initially, the lesions are small and either round or oval. Latter, they become confluent and large. The lesion may have

a characteristic scalloped lateral margin at the gray-white matter junction. The disease begins in the cerebral sub-cortical white matter, then spread to deep white matter, which occurs late in the disease process *(Hseuh et al., 1988 & Mark et al., 1989)*.

The posterior fossa disease is common, seen in approximately one third of PML cases. The basal ganglia, corpus callosum and brain stem are less commonly involved. The lesions typically do not enhance or cause mass effect Hemorrhage within the lesion also be reported *(Mark et al., 1989)*.

B) Lyme disease:

MRI shows multiple discrete hyperintense lesions on long TR image. Initially the lesion begins at peri-ventricular or sub-cortical white matter involvement of thalami or basal ganglia have been noted. Contrast enhancement has also been seen *(Lee et al., 1992)*.

C) Acute disseminated enchepalomyelitis:

MRI demonstrated multiple a symmetric foci of hyperintensity in both cerebrum and brain stem, the lesion are best seen on long TR images *(Atlas et al., 1991 & Kesselring et al., 1990)*. Segmental involvement of gray-white junction is often seen including the arcuate fibers. They are usually non-hemorrhagic and exhibit no mass effect. Enhancement of the lesion, probably is more uniform than in MS since all lesions in ADEM are active *(Atlas et al., 1986)*. There is often a dramatic response to steroid administration, steroid

therapy may decrease or ablate enhancement of the lesion *(Atlas et al., 1986)*.

D) Viral encephalitis:

MRI showed multiple coalescent hyperintensity in both white matter and cortex is seen in one or both temporal lobes *(Atlas SW et al., 1991)*. The hippocampus, para-hippocampus and amygdala are often involved *(Albertyn et al., 1990)*. There may also be extension into the frontal and parietal lobes. More over, marked atrophy and encephalomalacia also be noted. The lesion demonstrates parenchymal or lepto-meningeal contrast enhancement, but the non enhanced M.R. imaging is more sensitive for detection of early subacute hemorrhage *(Atlas et al., & Switzer et al., 1991)*.

E) Cytomegalovirus encephalitis:

MRI demonstrated a diffuse increase in signal on T_2 WI and P.D images without mass effect in the peri-ventricular white matter and in the centrum semiovale. Less extensive lesion including patchy and punctate lesions, usually in those who are less clinically symptomatic *(Olsen et al., 1988)*. Both cortical and central atrophy progresses on serial M.R. studies *(Chrysikopoulos et al., 1990)*. On post gadolinium contrast, these lesions do not enhance.

F) AIDs related disease:

MRI shown central and cortical atrophy, M.R. may reveal increased signal in the peri-ventricular white matter on T_2 WI. The lesion may appear broad and nodular. Gadolinium enhancement

may show ependymal or peri-ventricular enhancement mimicking lymphoma *(Sze et al., 1987)*.

4. Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts And Leuko-Encephalopathy (CADASIL) M.R.I. findings:

A) Age related:

MR imaging is isointense to cerebro-spinal fluid on all pulse sequences. The basal ganglia, VR spaces especially when asymmetric is to differentiated from lacunar infarcts. The infarct tend to be oval and asymmetric, occurring in the superior two third of the basal ganglia. The combination of prominent VR spaces and ischemic white matter disease produce an appearance termed etat crible *(Heier et al., 1989)*.

B) Peri-ventricular hyperintensity:

MR imaging revealed a thin continuous line to a thick irregular rind of high signal on T_2 WI and proton sequences *(Zimmerman et al., 1986)*.

C) Sub-cortical hyperintensity:

MR imaging, irregular areas of hyperintensity on T_2 WI usually are seen in the white matter antero-lateral to the frontal and occipital horn, the centrum semiovale, the basal ganglia and central portion of pons. The lesion does not extend into the corpus callosum and spare the medial sub-ependymal regions, along the trigones and occipital horn.

D) Cerebral amyloid angiopathy (CAA):

Extensive peri-ventricular hyperintensities that are bilateral and symmetric, more over, lobar hematoma also be noted.

E) Hyperintensive encephalopathy:

MR imaging revealed diffuse symmetric hyperintense signal along the peri-ventricular white matter and patchy small hypointense foci representing petechial hemorrhage.

F) Eclampsia:

Supra-tentorial and infra-tentorial high intensity lesions on T_2 WI are seen in 90 % of patients *(Kumor et al., 1987)*.

G) Migraine:

An ill-defined white matter lesion with prolonged T_2 WI relaxation time. The lesion commonly bilateral and involving the "U" fibers, gray matter and the posterior fossa. Mild atrophy may accompany these findings *(Sages et al., 1987)*.

5. Iatrogenic group of cerebral demyelinating diseases

A) Radiation treatment:

On T_2 WI M.R.I. show symmetric confluent, high signal foci in the deep peri-ventricular white matter within five to nine months from the time of treatment *(Tsuruda et al 1987)*. T_1 WI changes in the white matter are much less common *(Packer et al., 1986)*. The areas most often involved are adjacent to the frontal horn tip, occipital horn tip, lateral aspect of the ventricular body, lateral

aspect of the occipital and temporal horns, and centrum semiovale. The "U" fibers are frequently involved *(Drayer et al., 1987)*. There is relative sparing of the posterior fossa, basal ganglia and internal capsules. Areas of coarse calcification are seen as signal void. The involvement may be equally severe in the contra-lateral hemisphere and is seen remote from the tumour. Minimal mass effect is seen related to the area of abnormal signal. The appearance on M.R.I. varies, depending on the stage of the disease with long T_1 WI in necrotic areas and short T_1 WI or T_2 WI or both in areas of hemorrhagic in a characteristic pattern. Mass effect may be present and coagulative necrosis may mimic intra-cranial neoplasm *(Tsuruda et al., 1987)*.

B) FAHR's disease:

There is hyperintense signal on M.R. T_2 WI in the white matter of the centrum semiovale that arises from the non-calcified element. In the basal ganglia, low signal intensity is seen, probably owing to the heavier calcification displacing the signal giving element or para-magnetic effect of iron. The dentate nuclei show intermediate signal intensity *(Scotti et al .,1985)*.

C) Sarcoidosis:

High signal intensity of the basal meninges and intra-cerebral medullary nodular or supra-seller mass lesions in T_2 WI are seen *(Ketonen et al., 1987)*.

D) Toxic agent:

They are causing bilateral symmetric lesions in the globus pallidi with high intensity on T_2 WI. In addition, symmetric confluent areas of increased signal in the white matter of the temporal, occipital and posterior parietal has been described *(Herowitz et al., 1987)*. In addition, methanol intoxication the M.R.I. finding are bilateral symmetric involvement of the occipital lobes with that appears on both putamina of basal ganglia *(Dooms et al .,1987)*.

Material and Methods

Fifty-three patients were included in this study, 30 patients were female and 23 patients were male and their age's ranges between 6 years and 70 years.

They were referred to the radiological department at Al-Hussein University Hospital and El-Fatth radiological clinic through the period between April 1998 and Feb. 2000, preserved with different pathological lesions related to the cerebral demyelinating lesions.

These cases are taken at random with no specification for age, sex or type of cerebral demyelinating lesion.

MRI was performed to all patients, with magnitom 1.5 Tesla (T) and signa 0.5 T. All M.R.I. examinations were performed with the patients positioned to produce standard views. More than 90 % of the scan were created with 7 mm or 5 mm thick slices by 1.5 T and few were made by 0.5 T scanner with 10 mm thick slices.

The protocol used in examining these patients was as follows:

1. Axial T_1 WI:

TR	650 ms
TE	14 ms
Flip angle	70 degree

2. Axial T_2 WI:

TR	2570 ms
TE	102 ms
Flip angle	180 degree

3. Coronal T_2 WI:

TR	5400 ms
TE	99 ms
Flip angle	180 degree

4. Flair Axial

TR	9000 ms
TE	110 ms
Flip angle	180 degree

5. Axial T_1 WI with contrast

TR	650 ms
TE	14 ms
Flip angle	70 degree

Intra-venous contrast enhancement is done in some cases, (mainly cases with multiple sclerosis) using 20 ml of magnivist and injected in a dose of 0.1 mmol / kg body weight. The scan was obtained between 5 and 20 minutes post-injection.

During examination, we put certain points into consideration aiming at investigating them. These points differ according to the problem investigated.

As regard cases of cerebral demyelinating lesions, these points are located for:

1- Accurate localization of cerebral demyelinating lesions.
2- Diagnoses of cause of demyelination if possible.
3- Detection of any underlying active disease.
4- Diagnosis of extent of the inflammatory process.
5- Detection and diagnosis of associated tumors.
6- Tumour location and its extent.

In each case examined, we tried to list the diagnostic data got by M.R.I. scan. Then we tried to compare the results of this study with the results in other published studies as long as we can.

RESULTS

Group	Type of cerebral demyelination		No. of Cases	Sex		Age			Affected site
				Male	Female	From	To	Mean age	
1	M.S.		19	6	13	16y	50y	18y	Both cerebral Hemisphere
2	Central pontine myelination		2	2	0	35y	65y	50y	Pons
3	Viral & post viral cerebral demyelinating infection		11	7	4	6y	70y	21y	Both cerebral Hemisphere
4	CADASIL		8	5	3	20y	70y	55y	Mainly peri-ventricular & Centrum semiovale
5	Iatrogenic	Post irradiation	7	3	4	30y	65y	45y	Mainly supra-tentorial
		Other causes	6	3	3	8y	50y	27y	Mainly Centrum Semiovale

Patient with multiple sclerosis:

Nineteen cases with multiple sclerosis were included in this study and were assessed by clinical examination followed by M.R.I. scanning. Thirteen were female and 6 were male, with their age ranges between 16 and 50 years. Seventeen cases were referred for unsettled diagnosis. M.R.I. examination has revealed multiple variable sizes focal lesion of abnormal signal intensity scattered in the deep peri-ventricular white matter of both cerebral hemispheres. In addition, some area of abnormal high signal pattern appears perpendicular to the ventricular body. More over, some cases revealed M.R.I. signs of activity after gadolinium contrast enhancement in term of ring enhancement in some lesion.

Other two cases were female 20 old and her noticed left-sided hemiparesis and female patient aged 25 years were referred for M.R.I. evaluation. M.R.I. scanning revealed multiple focal lesion of abnormal signal intensity and best visualized on the F.L.A.I.R. pulse sequence.

Patient with central pontine myelinolysis:

Two cases aged 35 and 65 years presented with clinical manifestation related to central pontine demyelination were included in this study. All of them have history of alcoholism. In the first patient, a small area of abnormal intensity was seen at the right sided of the lower pons. Second patient presented by lack of concentration and mentality changes. M.R.I. scans revealed smaller ill-defined

area of signal alteration involving the central pons with no post-contrast pathological enhancement seen.

Patients viral and post viral cerebral demyelination infection:

Eleven cases with inflammatory lesion involving the white matter were included in this study. Their ages ranged from six and seventy years, seven were males and four were females. As regard cases of viral and post-viral cerebral demyelination infection, the first four cases were females aged 14 – 18 years presented with recurrent attacks of epileptic fits, M.R.I. scans shows diffuse ill-defined signal alteration involving the right deep temporal and also at both occipital white matter. 7 male patients with inflammatory changes were met with in this study, all of them has history of hemiparesis, dementia and mentally changes. M.R.I. scan shows an extensive inflammatory process supra- and infra tentorial in distribution. After I.V. contrast injection, these were small patchy areas of enhancement within such diffuse process and incomplete marginal enhancement of breaking down areas.

Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts And Leuko-Encephalopathy (CADASIL)

Eight cases were included in this study, 3 of them were females and 5 were males, with their ages ranging between 20 – 70 years. They were presented with headache, hypertension and some of neuro-logical deficit. M.R.I. examination revealed peri-ventricular line of abnormal signal pattern and multiple focal lesions

scattered through the white matter supra and infra tentorial in distribution.

Patients with Iatrogenic cerebral demyelinating lesions:

Seven cases subjected to radio or chemotherapy were included in this study, 3 males and 4 females and their ages ranging between 30 and 65 years, with mean age of 45 years.

The patients were referred for M.R.I. evaluation of neoplastic lesions after operative and / or chemo or radiotherapy. M.R.I. scanning revealed abnormal foci of signal alteration, mainly supra tentorial in distribution.

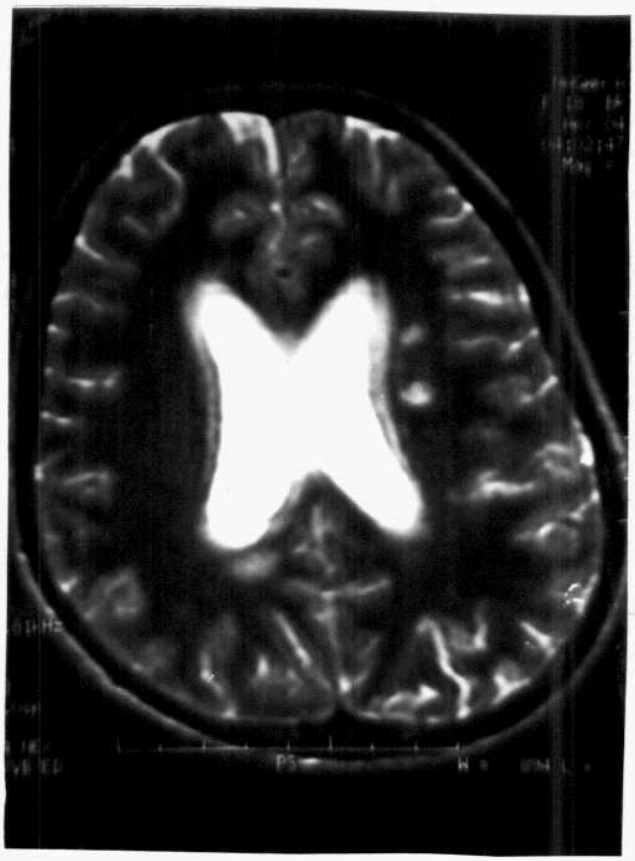
Another 6 cases with iatrogenic causes of cerebral demyelination were examined by M.R.I. scan. 4 cases with FAHR's disease were met in this study. The first 3 patients was male, they age ranged from 48 years to 27 years old. M.R.I. shows abnormal signal intensity, mainly at centrum semiovale, which appears bright on T_2 and low on T_1 WIs. The last 3 patients were female's age ranged between 15 and 23 years with established diagnosis by M.R.I. scanning.

SELECTED CASES

Case No. (1)

A 18 years old female patient, presented with history of severe headache of short duration.

Axial T_2-weighted image at the level of lateral ventricle: revealed multiple lesion, of high signal intensity are noted adjacent to superior lateral aspect of lateral ventricle and corpus callosum (right side) representing MS plaques.

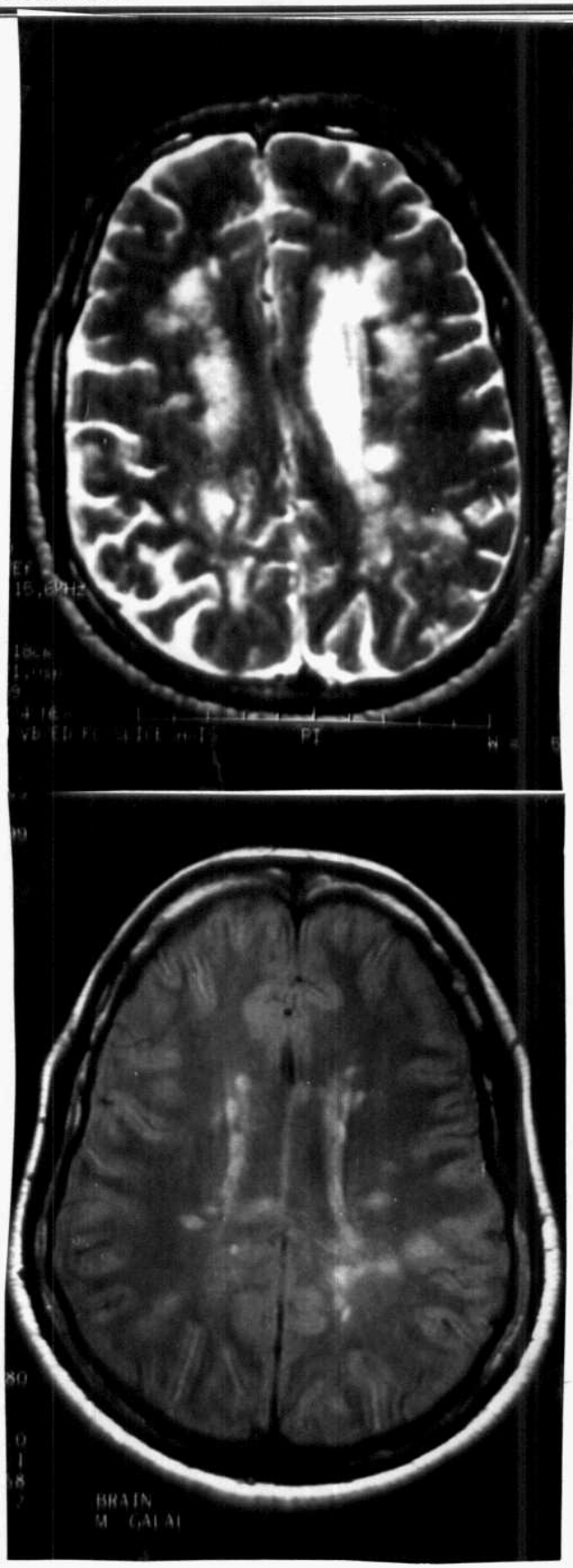


Case No. (2)

A 40 years old female patient, presented with left side hemiplagia.

A) Axial T_2-weighted image revealed multiple MS plaque within they white matter of the central semioval bilaterally.

B) Axial PD weighted image shows multiple additional plaques as area of increased signal intensity.

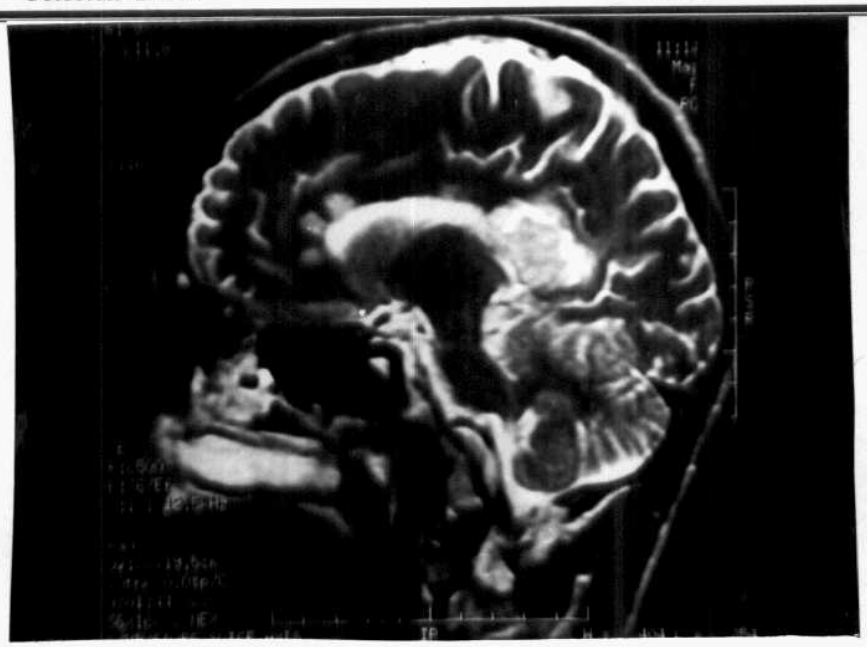


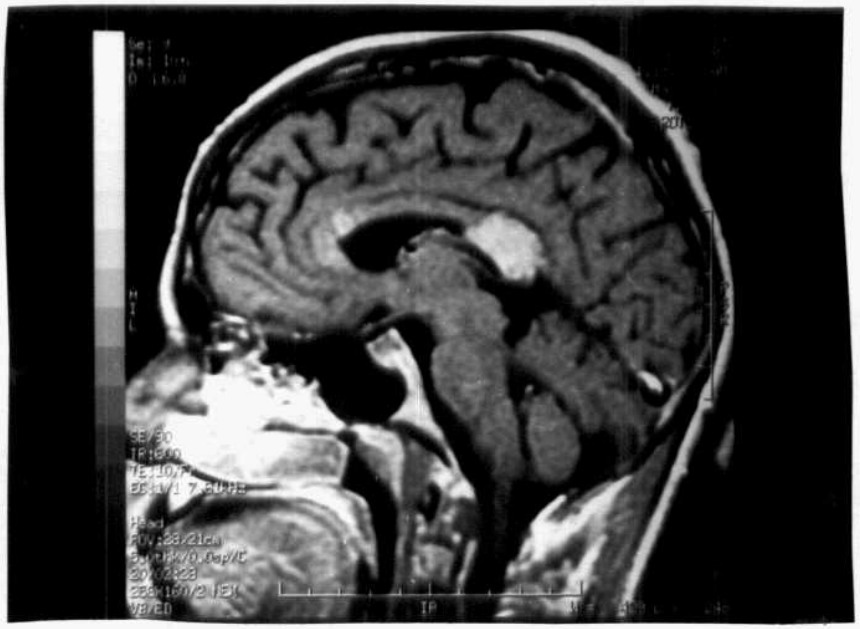
Case No. (3)

A 37 years old male patient, presented with history of headache blurring of vision and dementia.

A) Para median sagittal T_2 weighted image revealed glioma involving the splenium of corpus callosum. In addition multiple plaques of high signal intensity are seen in the white matter in the frontal lesion.

B) Para median sagittal T_1 weighted image post contrast enhancement revealed another enhancing plaques in the genu of corpus callosum, denoting recent activity.



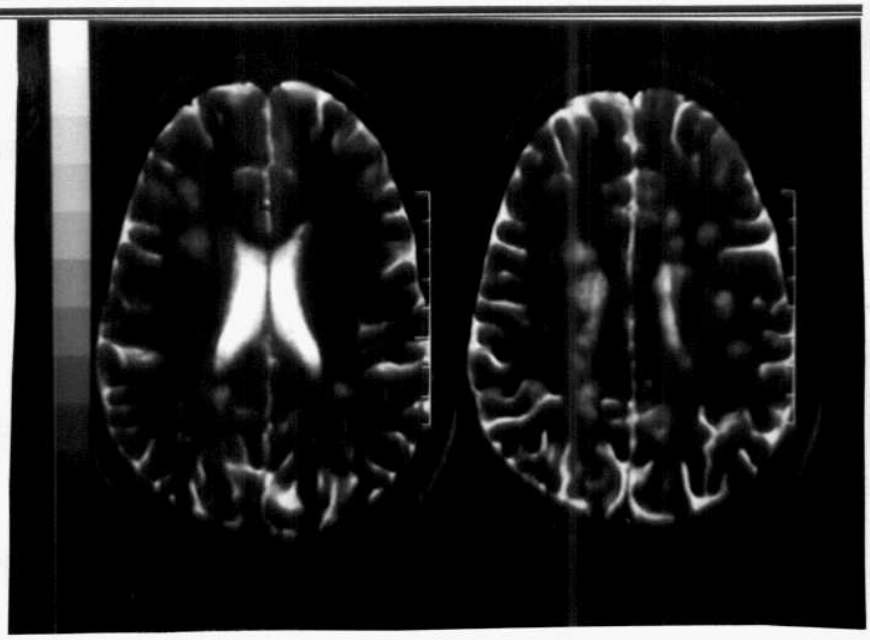


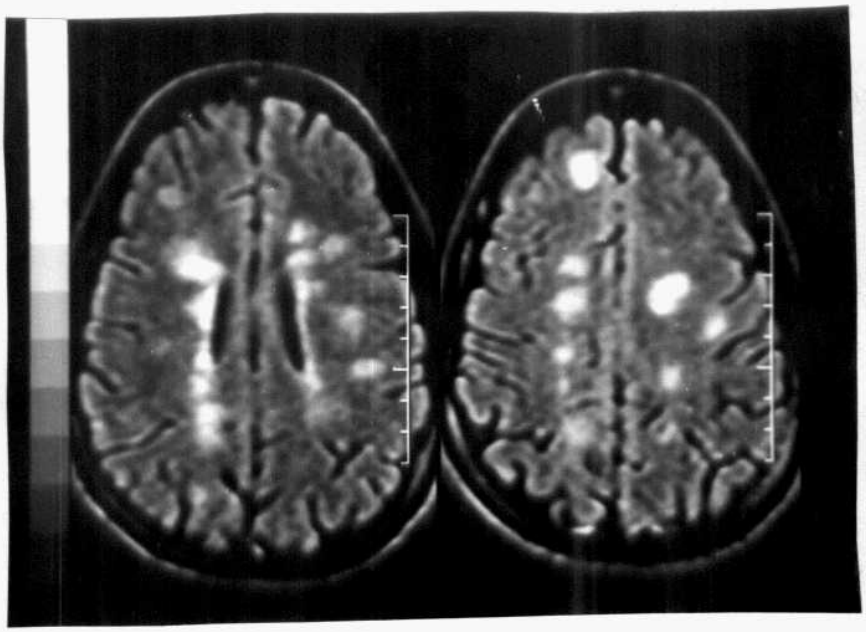
Case No. (4)

A 19 years old female patient.

A) Axial T_2-weighted image at the high and supraventricular level showing multiple plaques of white matter hyperintensities without associated significant mass effect.

B) Inversion recovery images at the same levels, showing additional plaques especially at the right frontal region.



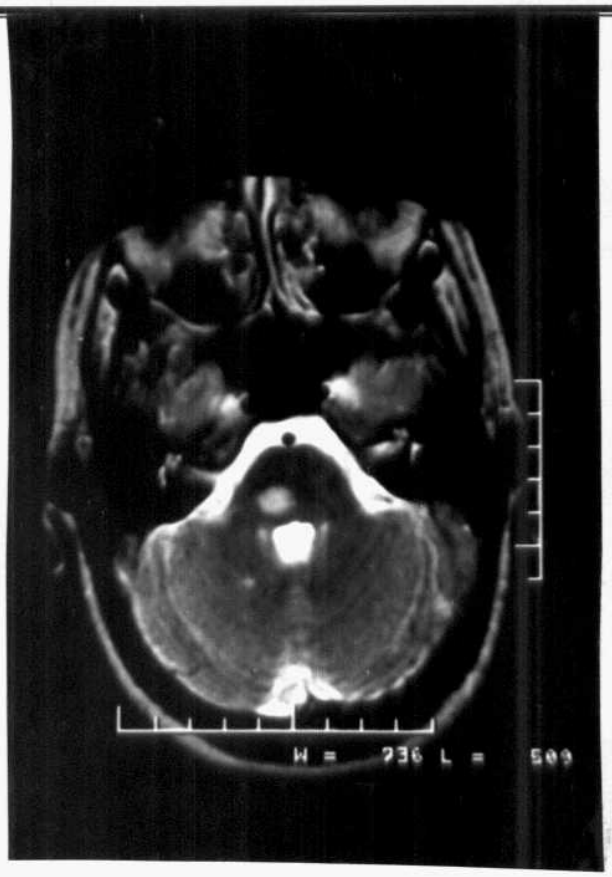


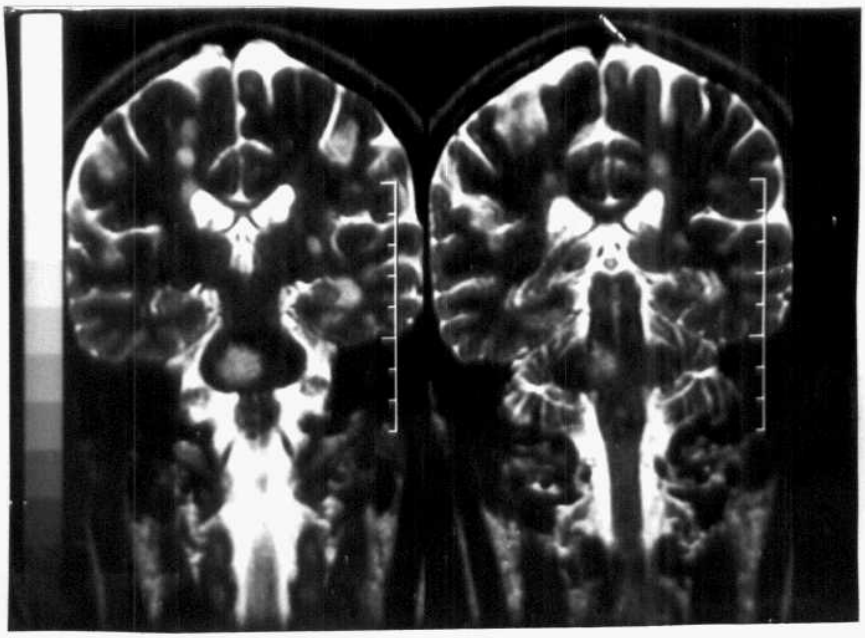
Case No. (5)

A 50 years old male patient presented with diplopia and diminished in visual acuity.

Axial and coronal T_2-weighted image showing high parietal multiple plaques of white matter hyperintensities bilaterally.

Another focal area of high signal intensity is seen in the right pontine region without significant mass effect. A case of central pontine myelinolysis.

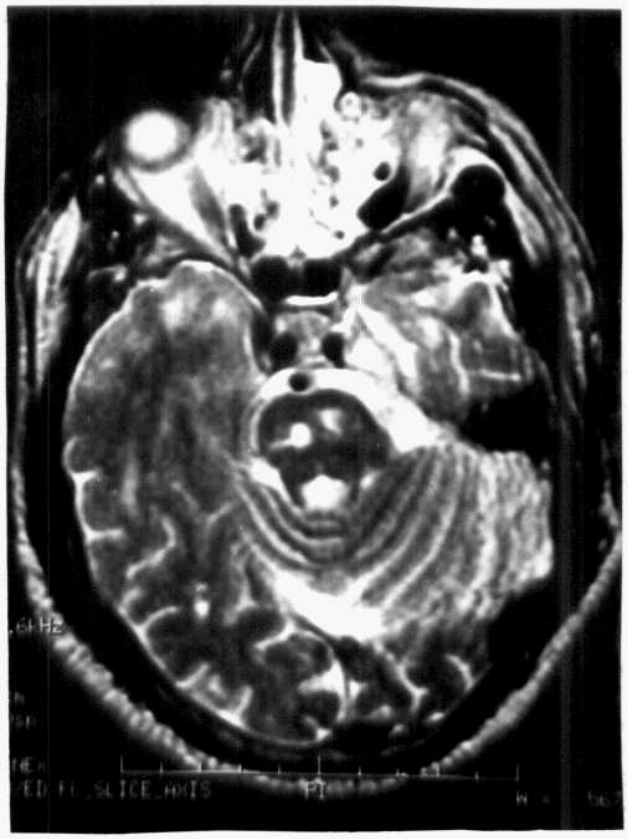




Case No. (6)

A 52 years old male patient presented with irritability, lack of concentration with history of alchoholism.

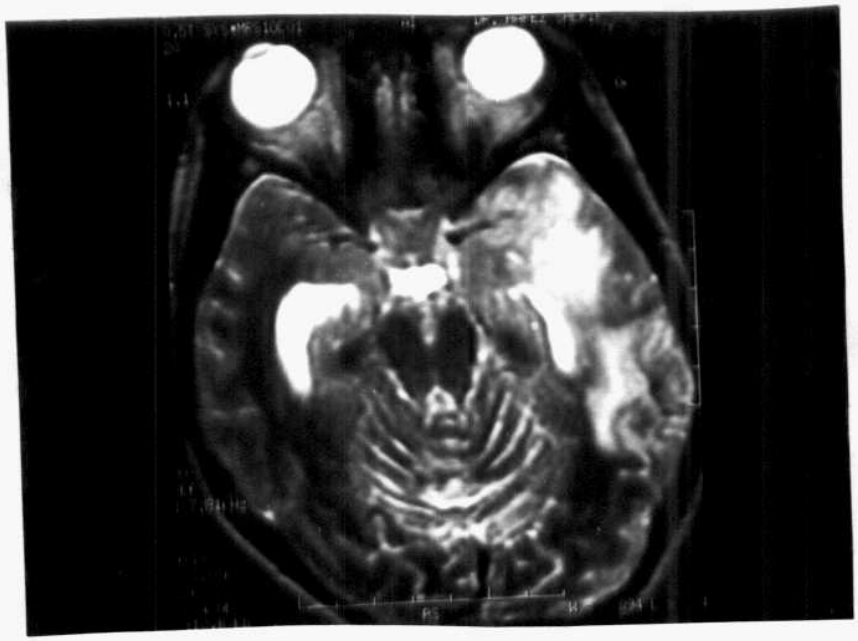
Axial T_2-weighted image revealed multiple plaques of high signal intensity in the pontine region without significant mass effect. A case of central pontine myelinolysis.



Case No. (7)

A 18 years old female patient presented with paraplagia.

Axial to T_2-weighted image shows poorly defined area of high signal intensity in the left temporal region with mild mass effect. There no obvious mass lesion identified. A case of viral encephalopathy.

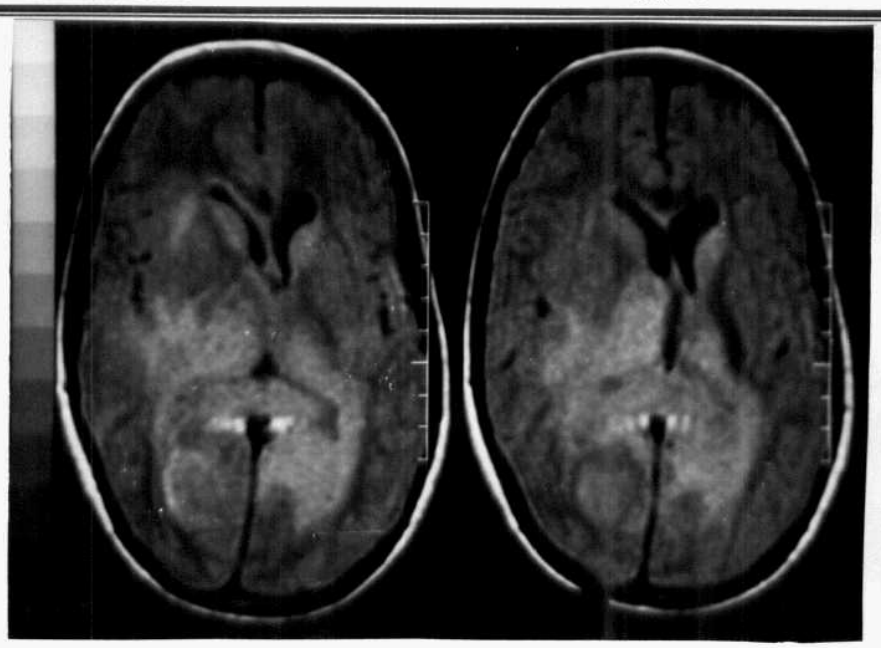


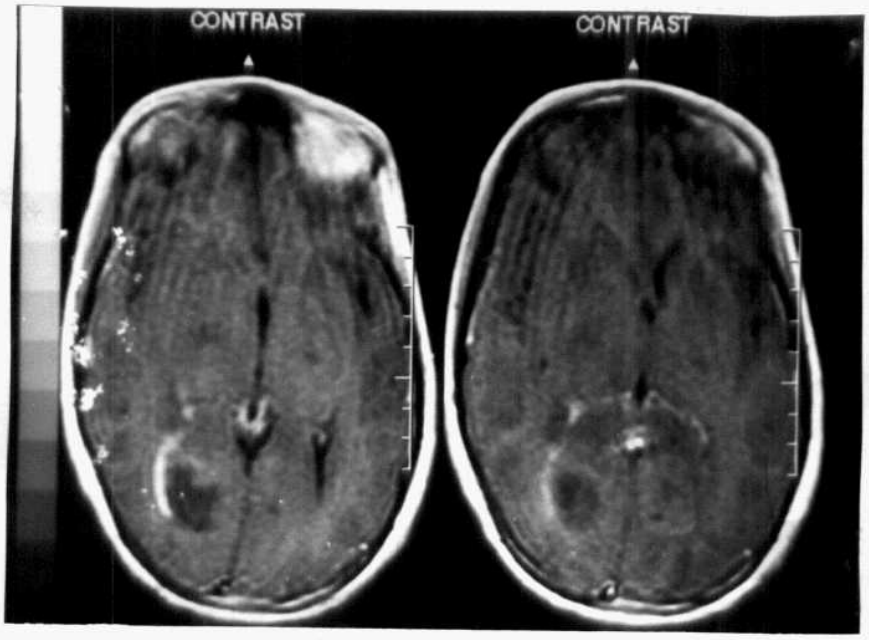
Case No. (8)

A 14 years old female patient presented by recurrent attacks of epileptic fits.

A) Inversion recovery weighted image shows diffuse ill defined signal alteration involving the right deep temporal region (mainly thalamic and both occipital regions with involvement of the splenium of corpus callosum.

B) Axial T_1 weighted imaged post contrast enhancement revealed area of contrast enhancement at right occipital region denoting active lesion. A case of viral encephalitis.

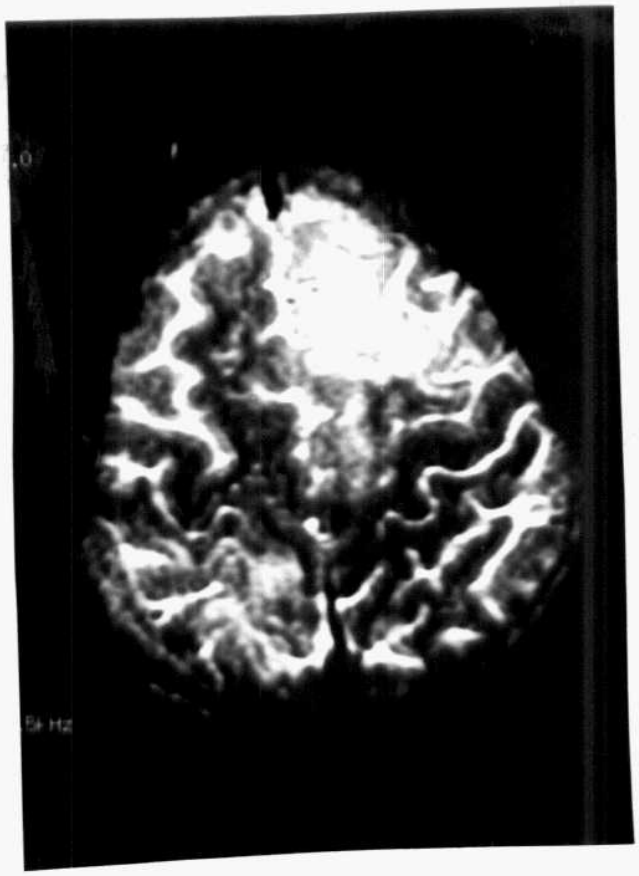




Case No. (9)

A 14 years old male patient presented by right side hemiplegia.

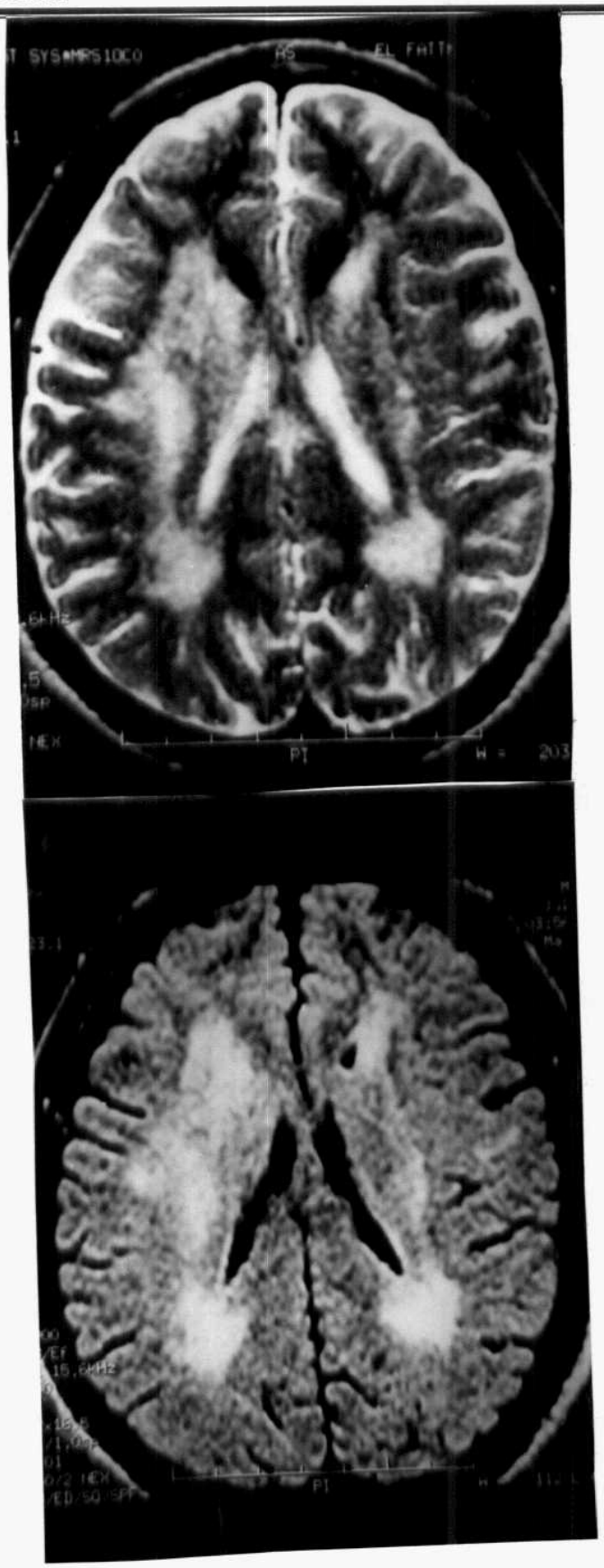
Axial T_2-weighted image shows left frontal focal area of white matter hyperintensity without significant mass effect no focal lesion identified. A case of progressive multifocal encephalopathy.



Case No. (10)

A 44 years old male patient presented by headache, blurring of vision and syncopal attacks.

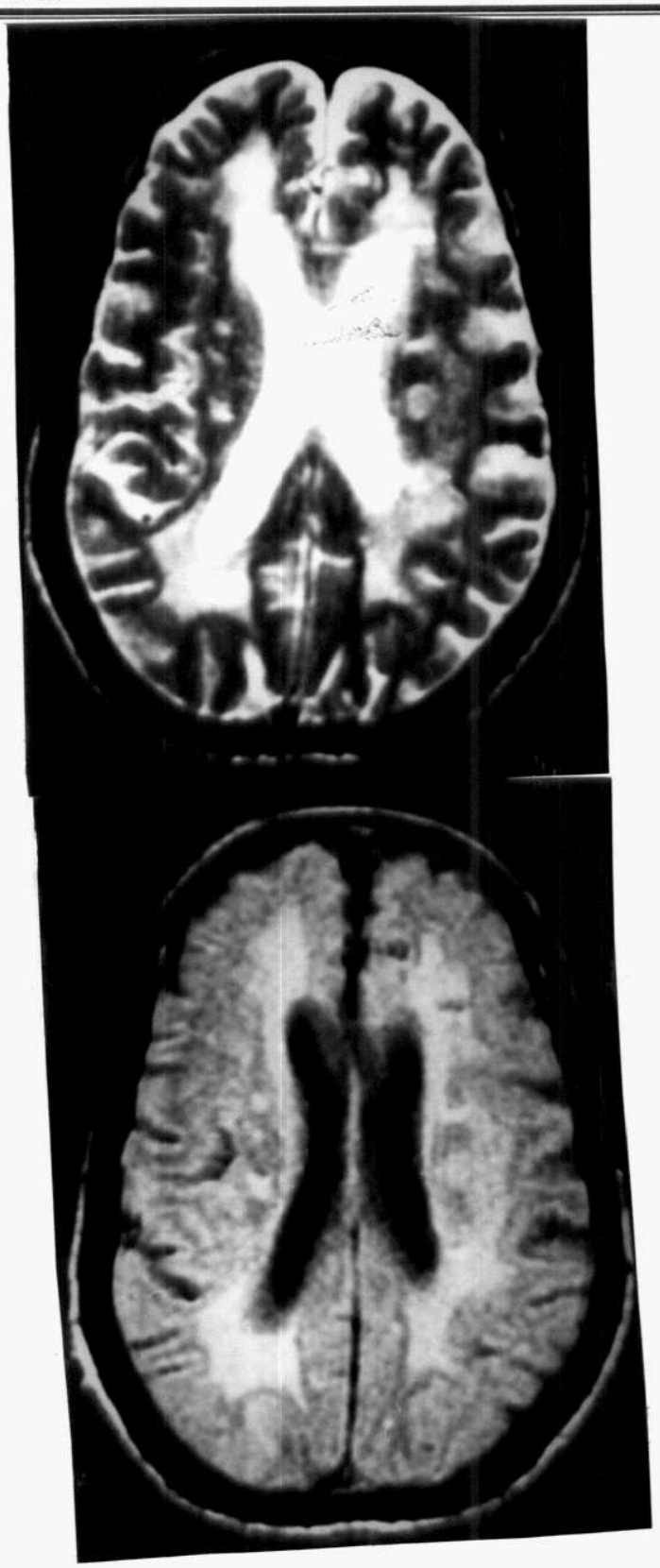
Axial T_2 and inversion recovery weighted images revealed multiple foci of increase signal intensity presented within the periventricular white matter. A case of ischemic leucoencephalopathy.



Case No. (11)

A 67 years old male patient presented by dementia lack of concentration and syncopal attack.

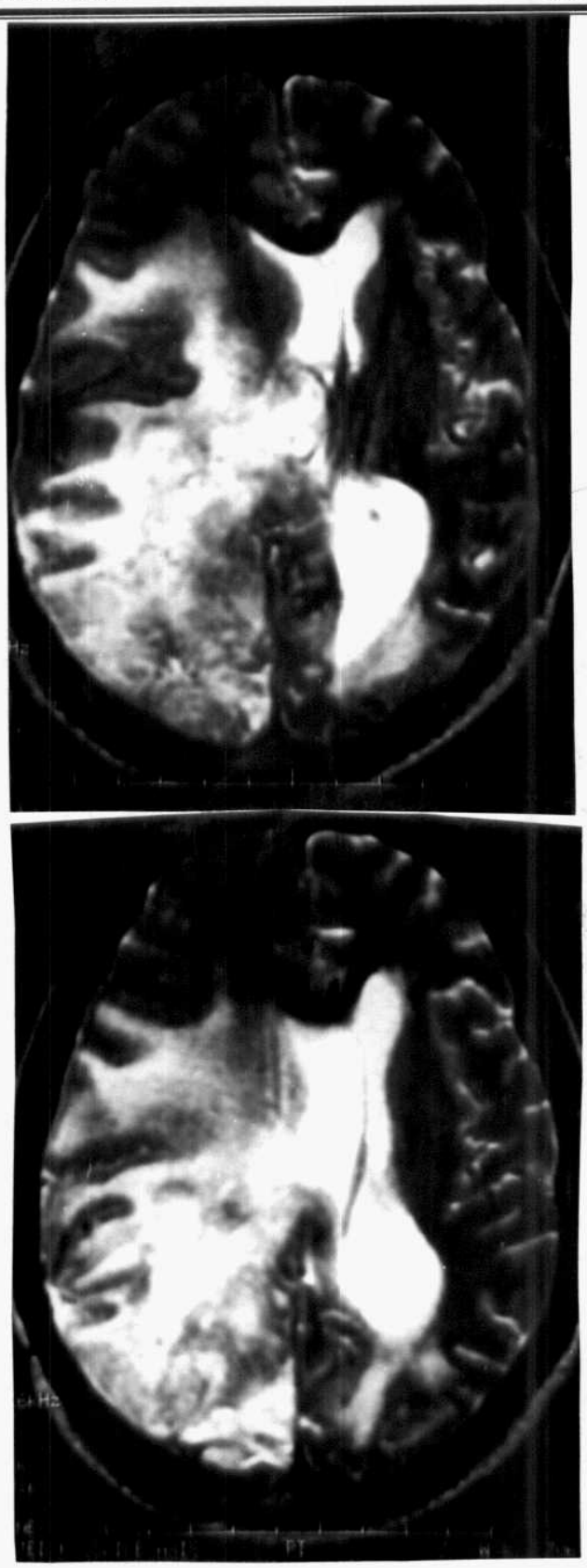
Axial T_2 and inversion recovery weighted images revealed multiple foci of high signal intensity involving periventricular white matter, and deep parietal region bilaterally. A case of hypertensive encephalopathy.



Case No. (12)

A 45 years old male patient presented by left side hemiplegia in addition follow up after cranioectomy and radiotherapy.

Axial T_2-weighted image in patient with previously resected right posterior parietal and occipital tumor with post irradiation leucoencephalopathy.



DISCUSSION

Multiple Sclerosis:

In spite of the number of cases of multiple sclerosis examined in this study, it is obvious that M.R.I. has allowed unique evaluation of anatomical structures.

From our results we can state that M.R.I. is of value in the diagnosis of multiple sclerosis, particularly in assessing the size, shape and position of plaques.

Ormerod et al. (1986) found that peri-ventricular white matter adjacent to the trigones and body at the lateral ventricle most frequently affected with a small decrease in frequency of lesion around the occipital and frontal horns, we think that might be true in some cases. Actually we found multiple plaques at deep peri-ventricular white matter zones of both cerebral hemispheres most aggregated at both parietal region.

Wessbecher et al. (1992) state that abnormality of MS plaques were detected more often by M.R.I., rather than laboratory investigation, they appear bright at T_2 WI and P.D. and best visualized on the FLAIR pulse sequence.

Nesbit et al. (1991) stated that the M.R.I. is particularly helpful in delineating the distribution and configuration of MS plaques, we agree with all of them as in our study we found that M.R.I. is

extremely useful in evaluating the position and distribution of MS plaques.

Also we found that M.R.I. is efficient in assessment of combined active and inactive lesions and this is quite obvious in demonstration of MS plaques. So we recommend to do M.R.I. in cases of MS lesions whenever possible especially when other investigation cannot give clear definition of the suspected or unproved MS lesions.

Central pontine myelinolysis:

Miller et al. (1988) have studied the value of M.R.I. in diagnosis of central pontine myelinolysis. They conclused that M.R.I. is superior in defining subtle change of pons and they found that lesion is much better identified on T_2 WI.

We agree with them as in our study, we found that M.R.I. changes of pontine demyelination are visible without overlap. M.R.I. was particularly variable for the depiction of central pontine myelinolysis.

Viral and post viral cerebral demyelinating infection:

As regard our cases of inflammatory cerebral demyelination we failed to found any M.R.I. features, which could specify. However, the patients with viral infection included in our study are examined in the early stage of the disease. This mean that we agree with *(Hseuh et al., 1988 & Mark et al., 1989)* who stated that there

are no definite M.R.I. feature characteristic of the type of the pathogenic organism.

In one of the patients with viral infection a diffuse ill-defined signal alteration was seen involving the right deep temporal, thalamic region, and also at both occipital white matter zones closely related to the trigones and occipital horns of both lateral ventricles. It appears faintly dark on T_1 WI and faintly bright on T_2 WI and P.D. images. After I.V. contrast injection, there were small patchy areas of enhancement within such diffuse process. However, M.R.I. scan led to the exclusion of neither benign nor malignant tumour because it excluded the presence of localised mass lesion of definite margin.

According to M.R.I. features seen in our patients with viral infection, the possibility of viral infection were considered. So we recommended to do M.R.I. examination to all cases with suspicious of inflammatory cerebral demyelination, where the diagnosis was not settled yet and also to patients with settled diagnosis of viral demyelination to detect signal alteration as well as soft tissue changes.

Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts And Leuko-Encephalopathy (CADASIL):

As regard our cases with (CADASIL) demyelination, M.R.I. was found to be of great value, it provides certain criteria (well-definition of the lesion, it exact location, signal pattern, presence or

absence of associated lesion), which are considered as an indication for the nature of the vascular demyelination. This means that we agree with *(Fazekas et al 1991)*, who stated that the previous criteria are evidence of vascular demyelination. However, we believe that considering the different mechanism by which pathological processes can result in vascular demyelination detected by M.R.I. scan.

Iatrogenic demyelinating lesions:

Tsuruda et al. (1987) stated that symmetric confluent, high signal foci in the deep peri-ventricular white matter within 5 to 9 months from the time of treatment.

We think that might be true in some cases. However, in our series, we did not see such a case. Actually we found a signal alteration as bright on T_2 WI and P.D. images. The area affected most often involved frontal horn centrum semiovale and lateral aspect of the ventricular body. Also we found that M.R.I. is efficient in assessment of iatrogenic groups and this is quite obvious in demonstration of demyelination.

SUMMARY AND CONCLUSION

MRI clearly has a major role in the diagnosis of cerebral demyelination disease. Advanced imaging techniques make it possible to image the entire C.N.S. and contrast enhancement delineates acute lesions.

A careful consideration of clinical and M.R.I. findings provides a high level of diagnosis accuracy.

The amount of imaging information needed will depend on the clinical and initial M.R.I. and other laboratory findings *(Barkhof et al., 1997)*.

Most importantly M.R.I. is not only capable of providing positive evidence for multiple sclerosis but simultaneously serves to exclude other pathological abnormalities, which may mimic multiple sclerosis, we therefore recommended that M.R.I. of the brain should be obtained at least once in every patient with suspected or even clinical definite cerebral demyelinating diseases.

Recommendation of examination protocols can only be general as they have to be optimized according to the machinery available and will require constant updating and refinement with further technical advances and an increasing understanding of the patho-physiology of cerebral demyelinating lesions.

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ARABIC SUMMARY

الملخص العربى

هناك عدة أمراض تصيب المادة البيضاء للمخ ، و قد ثبت أن التصوير بالرنين المغناطيسى ذو حساسية عالية فى إظهار التغيرات التى تحدث فى المادة المخية البيضاء فى هذه الأمراض. و الغرض من هذه الرسالة هو إلقاء الضوء على مظاهر أمراض المادة البيضاء للمخ عند استخدام الفحص بالرنين المغناطيسى. وقد اشتملت هذه الدراسة على الأبواب التالية:

الباب الأول: المقدمة و الغرض من البحث.

الباب الثانى: التطور الجينى للمادة البيضاء بالمخ.

الباب الثالث: الصفات التشريحية و المظاهر العينية للمادة البيضاء كما تبدو عند التصوير بالرنين المغناطيسى.

الباب الرابع: الصفات المرضية و المظاهر الإكلينيكية لأمراض المادة البيضاء بالمخ.

الباب الخامس: طرق الفحص بالرنين.

الباب السادس: المظاهر المرضية لأمراض المادة البيضاء كما تبدو عند التصوير بالرنين المغناطيسى.

و قد أجرى البحث على ثلاثة و خمسين مريضا ، و تم الفحص بعمل رنين مغناطيسى على المخ ، و تم تدوين النتائج بالتفصيل و مناقشتها و مقارنة المعلومات ، و تم عرض نماذج الفحص لبعض المرضى مع تقرير كل حالة و التشخيص النهائى لكل منهم ، و قد ثبت أن التصوير بالرنين المغناطيسى أعلى مكانة بكثير من الأشعة المقطعية بالكمبيوتر فى تشخيص أمراض المادة المخية البيضاء ، و ذلك لأنه يتميز بحساسيته العالية ووضوح أكثر للصورة و إمكانية التصوير فى عدة مقاطع مختلفة.

أمراض المادة البيضاء ككل تظهر فى صورة مساحات قليلة الاستضاءة فى مقاطع وقت الاسترخاء الأول ((T_1)) ، بينما تظهر فى صورة مساحات شديدة الاستضاءة فى مقاطع وقت الاسترخاء الثانى (T_2) ، كذلك فإن التصوير بالرنين المغناطيسى يساعد فى اكتشاف ما إذا كان المرض ثابت أم نشط:

أولا: بحساب الكم الإجمالى للأنسجة المريضة فى تصوير تتابعى للمادة البيضاء.
ثانيا: بحقن صبغة مادة الجادولينيم التى تتميز باتجاهها إلى مساحات الالتهابات النشطة.

أيضا أحد أهم الأغراض التى يستخدم فيها التصوير بالرنين المغناطيسى هى متابعة العلاج ، و بما أنه عالى الحساسية فى اكتشاف أمراض المادة البيضاء حتى التى ليس لها عوارض إكلينيكية ؛ فإنه يتيح إمكانية تقدير العلاج مبكرا عن إمكانية ذلك إكلينيكيا.

دور الرنين المغناطيسى
فى تقييم إصابات المادة المخية البيضاء

دراسة

توطئة للحصول على درجة الدكتوراه

فى الأشعة التشخيصية

مقدمة من

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القاهرة

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