“EEG HAPPY FAMILIES”: THE FUN WAY TO LEARN ABOUT COMMON EEG ABNORMALITIES

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The electroencephalogram (EEG) is an important test for neurologists. However, many neurological trainees only have limited exposure to EEG and most will not have directly reported EEGs. When you report EEGs rather than look at illustrative examples you concentrate much harder recognising the importance that your interpretation will have; you look for what information you can from the request form; you try and get as much out of the technician’s report – and as a result you learn much more.

To attempt to recreate this level of concentration we have devised this simple game. Below there are seven fragments of EEGs, eight sets of clinical information, eight technician reports, and eight EEG conclusions. Your job is to match them up—to make the sets of happy families—and a table has been provided to make this easier. You will appreciate that you have one “spare” request form, technician report, and conclusion to make it a little harder. For the sake of the game the technician reports and conclusions have been edited to separate the description from the conclusion.

All the EEGs are on the same montage for the sake of consistency. The montage is colour coordinated and is given in fig 1. Some of the abnormalities would be better seen on different montages and with digital recordings these would normally be switched, but that would disrupt the game.

The correct answers are given in a table on page ii46.

The game aims to help you identify common EEG abnormalities but also to help you appreciate the importance of filling in an EEG request form to provide the neurophysiologist with useful information that will make their report more useful to you.

Request form

- A: 65 year old man. Memory loss over last six weeks. Confused.
- B: 19 year old man. Single generalised seizure without warning. History of brief jumps in arm for 2–3 years.
- C: 44 year old woman. Two episodes of unwitnessed loss of consciousness—vision bit blurred before hand.
- D: 56 year old man. Three generalised seizures in last three weeks.
- E: 24 year old man. Admitted with sudden onset seizures, fever, confusion, and memory loss.
- F: 26 year old woman; episodes of déjà vu and dizziness over the last six months. No loss of consciousness (LOC).
- G: 12 year old girl. Always been a daydreamer. Blanking out for periods? epilepsy?
- H: 44 year old man: frequent episodes of flashing in the right visual field—last about 30 seconds—noted to be dazed afterwards.

Technician’s report

- i) There is pronounced slowing over the left hemisphere. The right hemisphere is normal.
- ii) There is nearly continuous biphasic/triphasic sharp wave discharges of varying interburst interval over the left hemisphere. Intermittently there is spread to involve the right hemisphere. There is no clear evolution in frequency or amplitude. Between the discharges the background is disorganised and slow.
- iii) There are frequent focal sharp waves arising in the left temporal region. The background EEG is normal.
- iv) Two episodes were observed during the video-EEG recording of sudden jerking of the head. This was associated with a spike/polyspike and wave discharges (associated with some occipital muscle artefact).
- v) There is 10–12 Hz α waves. No response to photic stimulation or hyperventilation.
- vi) There is pronounced slowing over the right hemisphere. The left hemisphere is normal.

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Blank table for you to fill in:

EEG number	Request form (A-H)	Technician's report (i-viii)	Conclusion (a-f)
1			
2			
3			
4			
5			
6			
7			

- vii) A grossly abnormal record with fairly frequent bursts of high amplitude irregular delta over both hemispheres with anterior preponderance R>L. The background is disorganised with excessive slowing.
- viii) The resting record was normal. During hyperventilation there were frequent runs of 3 Hz spike/wave discharges associated with brief cessation of hyperventilation and momentary unresponsiveness.

Conclusion

- a) Normal record. A normal record does not rule out epilepsy.
- b) A normal sleep record.
- c) This is likely to be prion disease, though non-convulsive status should be considered. A repeat EEG may help.
- d) This EEG indicates an increased liability to focal onset seizures arising from the left temporal lobe. A structural trigger needs to be considered.
- e) This is childhood absence epilepsy.

Figure 1 The montage used in the EEG segments. The right side (even numbers) is purple, the left side (odd numbers) green, and the central electrodes are red.

- f) This record indicates this is a primary generalised epilepsy, most likely juvenile myoclonic epilepsy.
- g) A structural lesion in the left hemisphere needs to be considered.
- h) This is likely to represent a viral encephalitis.

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EEG 1

EEG 2

EEG 3

EEG 4

EEG 5

EEG 6

EEG 7

EEGs 1–7.

THE NEUROLOGIST IN THE DGH: HOW TO SURVIVE WITHOUT ON SITE NEURORADIOLOGY

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E Teasdale

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What would be my advice to a young neurologist, boldly birthing from the womb of the neurosciences ivory tower training ground to practice in the reality of medicine that is found in the district general hospital (DGH)? How could it be possible to survive without the skills of the ever available super-expert neuroradiologist and the safety net of the all embracing specialist centre?

The basic principles are of course simple common sense, and the specifics depend upon knowing and understanding radiologically useful neuroanatomy and the range of normal appearances. My remarks are directed primarily to the newly appointed consultant as she (or he) takes up a post which involves a mixed neurological practice of the hub and spoke variety or one based mainly in a district hospital. Established consultants will have either resolved the problems or alienated the local general department by insisting that all of her (or his) important examinations are referred up to the neuroradiology department for a “proper report”. This is not the way forward that I would recommend! Instead I suggest you follow my simple survival course.

FUND RADIOLOGICAL SUPPORT

At your consultant interview, and before you accept your consultant post, make sure that the employing authority has implemented the Royal College of Radiologists guideline for ensuring that extra funds are made available to support your imaging needs. This is equivalent to two sessions of radiology time per new consultant neurologist and one additional session for a replacement post. In this way it will be possible to benefit directly the local radiology service (make a friend for life) or to expand the specialist service in the neuro-centre so that a neuroradiologist can regularly visit the local hospital and forge links benefiting both services. This gives the local radiologist someone with whom they can discuss difficult neurological cases, and allows the neuroradiologist to visit the local hospital to help and advise on cases or scanning protocols and to take part in multidisciplinary meetings. We have found this a great benefit, especially for the neuroradiologists, as the food supplied at the local interdisciplinary meeting far exceeds that at the “ivory tower”!

MAKE FRIENDS WITH THE LOCAL RADIOLOGISTS

This may sound too obvious but it is possible to make working relationships more difficult than they need be if you remain known to your colleagues only as an almost illegible signature on a request card. On your first day at the hospital go to the radiology department, introduce yourself to the consultants, and set up a meeting where you can discuss the type of service they have to offer and the type of service you require. Single out one or two you think you will best be able to work with, and groom them to be your local specialist neuro-imager (see later). Consider making a donation to their library of such excellent neuroradiology textbooks as Anne Osborn¹ or Scott Atlas² to stimulate interest and give support.

CHECK OUT THE LOCAL IMAGING TECHNOLOGY

Most district hospitals will have access to multidetector computed tomography (CT) and 1.5 Tesla magnetic resonance (MR) imaging, and they may be more modern than you have been familiar with. The radiologists will know very well what the scanners are capable of—for example, diffusion MR or submillimetre CT angiography—so ensure that you are familiar with any technical limitations and work around them. The vast majority of your imaging needs can be met with basic MR and multidetector CT. It is unlikely that you will require anything (except catheter angiography and neuro-interventional treatments) that cannot be provided by a general radiology department.

ACCESS TO EXAMINATIONS

Waiting times differ from hospital to hospital, with many general departments having stringent demands placed upon them for the investigation of patients with possible cancer. If you do have a

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patient who really requires an emergency examination you are likely to be more successful in achieving this if you contact one of the radiologists directly to discuss the case. Once they learn that you do request urgently only for a true emergency they will accommodate such requests readily in the future. It is also helpful if there is an imaging abnormality on these first urgent cases as most radiologists fail to get the same clinical satisfaction as neurologists from a normal scan!

HELP THE GENERAL RADIOLOGIST

The general radiologist will feel at a great disadvantage to you because she/he has not had the same training in neurological disease and will not be so familiar with detailed neuroanatomy. Make sure that you are! To ensure that you request imaging of the correct area and to improve your ability to read a scan, it is more important to know radiological neuroanatomy—for example, the site of the cerebral peduncles, the aqueduct, or the columns of the fornix are more important than to know the ultrastructure of the axons within them. Learn exactly where anatomical structures are on images in all three planes. This will help you to interpret the images for yourself and sharing this information with the radiologist will greatly enhance her/his confidence and interest in neuroimaging. It may be prudent to form a closer working relationship with one or two of the local radiologists so that by sharing experiences, cases, and clinical and anatomical information you will have a local colleague who has adopted neuroimaging as their subspeciality interest in addition to, or in place of, for example, gastroenterology.

HOW TO MAKE A REQUEST

It is necessary to be specific when you write the request. Do not use neurological jargon or unusual technical words—for example, “visual obscurations”, “man in a barrel syndrome”—or eponyms—for example, Miller-Fisher syndrome; Guillain Barré. These are likely to be unfamiliar to the general radiologist, and may not help to choose the correct protocol. Even worse it smacks more than a little of one upmanship! Clearly, legibly, and concisely state the clinical problem, the possible diagnosis you want to exclude/confirm, and identify a specific anatomical region—for example, parasellar, periaqueductal—if you want to be sure that imaging is correctly targeted.

IMAGE TRANSFER

A system may well already be in place and will mostly be used for the transfer of images for patients in whom the local physicians want admission to or advice from the neurocentre. However, it also works for keeping you in touch with the general physicians when you are not at the local hospital or if there are images on which you want a specialist neuroradiological opinion. In the absence of a comprehensive area wide picture archiving and communication system (PACS) this is essential. If you are aware that no such link exists you should make it a condition of your appointment that one is set up. It is also an excellent means of encouraging general and neuroradiological cross fertilisation.

MR OR CT: WHICH TO CHOOSE?

To many neurologists this may seem like a non-question. Who in their right mind would do a CT scan if MR is available? General radiologists tend to agree with that view; however, that is in part because they are not routinely performing CT adequately for the nervous system and they

Figure 1 Routine axial computed tomography (CT) demonstrates heterotopic grey matter (arrow) along the ventricular surface in this patient with seizures.

are not accustomed to using CT to its full potential for the nervous system, and moreover believe that any abnormality will be so obvious on MR that they could not miss it even if they did not know what it was.

There is no doubt that it is easier to perform good MR than good CT, and with less direct interaction for the radiologist. MR is a superb technology when it is available, but this is part of the problem. All patients expect and deserve appropriate prompt investigations. It is essential to use the technology available as efficiently as possible—that is, if CT and MR are equivalent in a particular situation, the one that gives the quickest result should be used. We should not create waiting lists for MR made up of people who could be examined adequately by CT; this disadvantages some patients by denying them an early CT, and delays an essential MR to others. The current Scottish Intercollegiate Guideline Network (SIGN) guideline for epilepsy³ and the current article on imaging in epilepsy⁴ are arguably examples of a specialist centre approach to a common clinical suspected presentation.

Complex specialised detailed targeted MR is very helpful in the (very) small number of people in whom surgery is being considered. But is MR mandatory in the majority of persons with suspected or established epilepsy? The yield in children, from such a policy, of management altering lesions absent on CT is exceedingly low.⁵ Much of the support for the use of MR over CT is based upon indirect comparison with superseded CT technology and the assumption that if MR is more sensitive for subtle parenchyma change, then it must be more useful than CT in epilepsy management, which may not be altered by the imaging. Even if high level evidence of superiority of MR over CT was available, the lack of access to it and the detailed imaging protocols makes such an investigative approach, in the confines of a DGH, a less than pragmatic one.

Surely in an adult with evidence of progressive neurological deficit and intractable focal seizures it is better to have an early CT scan and commence appropriate treatment as

Figure 2 (A) A small calcified focus is clear even in the cerebellum and is associated with a venous anomaly (B) commonly associated with a cavernoma.

early as possible rather than wait 6–9 months (UK average) for MR. Similarly early reassurance of normality is better than the anxiety of a long wait for a similar result. I would recommend a sensible case by case approach to the selection of the imaging and its urgency. This should be done in discussion with the local radiologist rather than blindly following guidelines that are often based on low levels of evidence.

All that radiologists ask is that you will consider carefully the overall optimal examination for your patient before automatically requesting MR. With the possible exception of acute demyelinating conditions, a well done modern CT scan will demonstrate any lesion requiring urgent or emergency intervention and the vast majority of any other structural pathology (figs 1 and 2).

WHAT NEUROLOGISTS NEED TO KNOW ABOUT IONISING RADIATION HAZARDS

The diagnoses likely to be under consideration by the neurologist tend to make them request MR and not CT. This is often a mistake because CT can offer a better option. The usual reason for not using CT is that it involves “dangerous” radiation, but this does not stand up to scrutiny.

The recent Ionising Radiation (Medical Exposure) Regulations (IRMER)⁶ state that the radiation dose must be as low as reasonably achievable to provide diagnostic information, or, if “comparable information can be achieved using an alternative that avoids ionising radiation, that

technology should be used instead”.⁶ Rigid implementation of IRMER would mean that no patient should ever receive a CT scan of brain or spine because not to use MR may be a criminal offence. Does CT have any future in neuroradiology if it is effectively illegal? In reality it is impractical, undesirable, and illogical to consider withdrawal of CT as a neuroradiological tool.

It is impractical because of the massive financial investment that would be necessary to replace it with an equivalent MR availability. It is undesirable because CT is an extremely accurate, effective, and readily available examination that can be used in any patient. There are no exclusions for CT, and it remains the examination of choice in an urgent situation. In a patient with visual failure it would be inappropriate, unnecessary, and illogical to withhold a CT examination and await MR because the radiation concerns are merely theoretical.

It is now vitally important to acknowledge that there is no direct evidence that low dose diagnostic radiation will cause a fatal cancer in an adult. Indeed there is evidence that low dose radiation confers protection against cancer. The original concern was based on extrapolation of the effects of the atomic bomb dropped on Hiroshima. This led physicists to conclude that there could be a theoretical 1% increase in the lifetime risk of cancer, in the UK, that would equate to a theoretical increase of 0.2% from the current level of 20–25% lifetime risk. Today the radiation dose for a routine multi-detector head CT is approximately 50 mGy which is equivalent to nine months background radiation. Survivors of the Hiroshima bombing who received less than the equivalent of 60 years background radiation do not show an increased rate of cancer and are even living longer than their unexposed compatriots.⁷ There is a growing evidence base which demonstrates that low dose ionising radiation is beneficial rather than harmful.⁸ We can be reassured and reassuring that the benefits of a CT scan in being able to provide an immediate diagnosis outweigh any theoretical risk and perhaps may even improve the patient’s health! Regularly repeated scans and those done on young children do require to be considered more carefully.

IMAGING PROTOCOLS FOR CT/MR

This is an area where the neurologist can make a real contribution to the efficiency of the examinations for her/his patients. General radiologists usually use only one protocol for all brain imaging in CT—that is, thin section for the posterior fossa (for example, 2.5 mm) and larger for the supratentorial compartment (for example, 8–10 mm). In practice optimal brain and skull base imaging is achieved by a very thin (≤ 1 mm) slice helical acquisition then displaying the images as thicker sections (3 mm post fossa, 5 mm supratentorial) to increase spatial resolution and minimise partial volume artefact. Such scanning also allows routine multiplanar reformations that are of benefit when the scan actually shows structural pathology.

You should suggest diplomatically that the detailed clinically driven protocols used in the neuro-centre could be adopted where appropriate for all your targeted imaging. You must be persuasive and supportive if you want to meld with your new imaging providers. (The CT and MR protocols used in the Institute of Neurological Sciences are available on the NHS Greater Glasgow website: www.nhsgg.org.uk)

Figure 3 This is typical herpes encephalitis. Note the low attenuation in both insular regions, more extensive on the right. Note also the low attenuation in the affected right cingulate gyrus. Can you identify the hippocampus? Are they affected radiologically?

WHAT NEUROLOGISTS NEED TO KNOW ABOUT CT IMAGING

Now you know that CT is safe and efficient and obviously much preferred by patients to MR, you also need to know that the development of multidetector CT (MDCT) technology led it to undergo a revival as an imaging method which is challenging (even overtaking) MR as a diagnostic tool for structural pathology. Modern scanners with submillimetre slices can cover the whole head in less than five seconds and the entire spine in 25 seconds. This is a boon to the claustrophobic or restless patient, especially those in pain.

Be clear about the question you are asking. Do you want to exclude structural pathology or learn more about subtle changes in the parenchyma of the brain? The spatial and temporal resolution of CT exceeds that of MR, which does gain in its ability to show soft tissue differentiation. MR is optimal in many of the non-structural diseases of the brain but CT is best when you are considering skull base, parasellar, cavernous sinus, and orbital pathology.⁹ MDCT is entirely adequate for the primary investigation of headache, epilepsy, acute stroke, trauma, dementia, multisystem atrophy, structural posterior fossa disease, movement disorders, spinal claudication, and lumbar disc disease, and in excluding acute spinal cord compression. The improved resolution from MDCT scanning versus the outmoded 10 mm slices used originally to compare CT with MR mean that these results are no longer valid. If the local radiologists have not improved their scanning techniques with their new MDCT scanners then you must encourage them to do so.

For reasons which I fail to comprehend, general radiologists feel comfortable performing and reporting MR angiography of the cervical and cranial vessels, yet are not comfortable performing comparable CT angiography. MDCT angiography of the circle of Willis is now routine for the detection of ruptured and unruptured aneurysms and is optimal in assessing the aortic arch, carotid, vertebral, and intracranial vessels in patients with stroke and transient ischaemic attacks (TIAs).¹⁰ The local radiologist may at first not feel comfortable exploring such CT angiography, but this

Figure 4 High attenuation material replaces the normal low attenuation cerebrospinal fluid (CSF) in the right sylvian fissure. Look at the normal left sided fissure. Acute subarachnoid haemorrhage (SAH).

is another situation when the image transfer facility comes into its own. Base CT angiographic data can be sent to the neuro-centre and processed there, allowing radiologists in both sites to compare their results. It is, however, such an easy technology to use that there should be no difficulty for the radiologist to learn the skills, should they so wish to do so. CT venography is an effective and easily understood examination in venous thrombosis and benign intracranial hypertension, providing timely diagnosis,¹¹ and is now rapidly becoming more widely accepted as the optimal investigation.

Figure 5 The temporal horns are notably dilated. This is the first sign of altered CSF dynamics and can be seen in meningitis of any cause and may indicate a late referral with SAH. In this case the history was of acute infective meningitis. It is safe to perform lumbar puncture in this situation as there is no focal mass.

HOW TO READ A CT SCAN

So what else do neurologists need to know to be able to read the CT scans of patients they have been referred or on whom they request CT? This depends a little on whether it is a sheet of film or interactive PACS.

In any event do not look at one image in isolation on PACS. Image interpretation depends upon pattern recognition and this is best with no less than four images on the screen at one time.

- ▶ Reading any scan is an **active** not a passive process.
- ▶ If it is a sheet of film then read the scan top down and then bottom up again, using the cerebrospinal fluid (CSF) spaces as pointers to local structural pathology. They are easier to see than subtle changes in attenuation which should be looked for secondarily.
- ▶ Always check that the midline structures are midline, especially the fourth ventricle. Everybody should have a patent third ventricle and basal cisterns (are you sure you know where they are?); if not, consider that the intracranial pressure might be raised.
- ▶ Do not forget to look at the top cuts of the hemispheres for subtle obliteration of the sulci signalling an early tumour mass effect, swelling from infarction, or an extra-axial collection.
- ▶ Areas to review include: the orbits and parasellar structures (the pituitary should not extend into the suprasellar cistern); the cranio-cervical junction—if necessary look at it in sagittal reconstruction; and the upper-most cortical cuts—sulci present both sides?
- ▶ Only now can you look at the parenchyma of the brain itself to ensure the basal ganglia and thalami are all of their normal higher than white matter attenuation and are well and clearly defined. Is the cortical grey matter equal over the hemispheres? What about the cerebellar peduncles and hemispheres? The brain stem and mid brain?
- ▶ It should be possible to identify the major cerebral arteries and veins, with an attenuation of about 40–60 Hounsfield units as they are usually slightly brighter than grey matter. Higher attenuation than this (around 80 Hounsfield units) implies acute thrombosis (hyperdense middle cerebral artery in acute stroke or the cord sign in acute venous thrombosis) or a higher than normal haemoglobin concentration in the blood.
- ▶ Know the basic structural anatomy of the brain and spine and be familiar with the age related changes and the common normal variants—for example, asymmetrical ventricular size, fat deposits in dural folds, pineal cysts.
- ▶ Learning reading technique and normal variants can be facilitated by sitting through CT reporting sessions with a neuroradiologist which, I assume, is all part of the basic neurological training programme.

COMMONLY MISSED PATHOLOGY

Remember, CT is the examination of the acutely ill patient and so you must personally be able to read such scans in the emergency situation.

There are only six conditions which fall into the above category and they can be misinterpreted on either CT or MR. The correct diagnosis is usually suggested by the history and clinical findings, with the imaging being used to confirm the suspected clinical scenario.

Herpes encephalitis

There may be no mass effect or swelling but there will be reduced attenuation in the insular region(s). You

Figure 6 The high attenuation in the right posterior temporal lobe is typical of haemorrhage. The surrounding low attenuation indicates that it is not immediately recent or that it is part of the primary disease. This is a typical site for venous infarction secondary to thrombosis and you will note the linear blood density lesion just deep to the occipital bone on the right. This is fresh thrombosis within the right transverse sinus which has precipitated thrombosis of the vein of Labbe causing local haemorrhagic venous infarction.

must actively look out and assess the attenuation in the hippocampus and the cingulate gyrus (can you recognise these on a CT/MR? Make sure you can). Reading the scan is not equivalent to simply gazing at it and looking for inspiration; it is an active process, positively looking for evidence of a disease you understand and believe to be present, or need to exclude (fig 3).

Subarachnoid haemorrhage (SAH)

A stereotyped classical clinical situation, so examine the CT scans carefully. Is the CSF density correct in the inferior interhemispheric fissure/the pre-pontine cistern/the foramen magnum? Is there subtle early hydrocephalus with slight dilatation of the temporal horns? This is a most useful sign to recognise for early delay in CSF absorption. It will give you a “get out of jail free card” in several situations—for example, SAH and meningitis. Also remember that the presentation of SAH may be delayed and the patient presents with the secondary ischaemic effects which are obviously related to the sites of rupture of the common aneurysms, peri-sylvian ischaemic low attenuation, inferior frontal and pericallosal interfrontal ischaemia; these are uncommon sites for primary occlusive vascular disease. Also check the dependent posterior horns of the lateral ventricles, a common place for a small blood/fluid level to lurk unnoticed (fig 4).

Cerebral infection

Meningitis is usually clinically obvious and treatment is proactive. There remains the vexed question of whether to perform a CT scan before lumbar puncture (LP) or not. The answer is simple: clinical confusion (in the patient, not the doctor) is a common sign in meningitis and alone does not contraindicate LP. Unless there are signs of a focal abnormality and/or papilloedema, or a definite reduction in consciousness as judged by the Glasgow coma scale (GCS),

Figure 7 (A) Low density is present in the right cerebellar hemisphere and there is early hydrocephalus evidenced by the dilated temporal horns. (B) In another patient the basilar artery is hyperdense reflecting acute thrombosis. (C) This shows a “tiger’s eye” (arrow) infarction on the left secondary to basilar perforator ischaemia.

CT scan is not required before LP. UK wide direction and agreement upon this point would go a long way to improving the frequent fraught out-of-hours interactions between junior, inexperienced clinicians and radiologists. A SIGN/ National Institute for Health and Clinical Excellence (NICE) guideline is required and further discussion is outwith the scope of this presentation. Patients with cerebritis or subdural empyema frequently present with seizures and a reduced GCS, so may be confused with those with herpes encephalitis. This is where it is important that you carefully check that the midline is central; it will not be so with an empyema as the purulent collection in the subdural space, although small in lateral measurement, will be large in overall volume as it lies over the hemisphere, displacing the anterior horn of the lateral ventricle posteriorly, and the posterior horn of the lateral ventricle anteriorly. Cerebritis is evidenced by ill defined loss of normal attenuation (usually) in the inferior temporal regions adjacent to infected fluid filled mastoids. This needs to be looked for positively by changing the windowing on the scan to see inside the bone. The scan will only tell you the truth if you actively seek it. You are in control; don’t expect the scan spontaneously to answer the question (fig 5).

Acute cerebral venous thrombosis

This is the great imitator clinically and radiologically, from subtle loss of density in all of one or both thalami (there is no arterial or other cause for this), pronounced oedema and petechial haemorrhage in the posterior temporal region, to large haematomas based in the white matter of the brain. Whenever any of these are evident or there is subtle SAH over the cortex, consider acute venous thrombosis. In the acute situation there will almost always be evidence of hyperdensity in the veins or venous sinuses on a properly conducted CT scan. A simple CT venogram will confirm the diagnosis to you and the radiologist (fig 6).

Posterior circulation ischaemia

This clinical presentation frequently encompasses an intermittent but persistent and progressive development of cerebellar and/or brain stem symptoms and signs. CT is often

Figure 8 This patient presented with sudden visual loss. Note the small central and right sided mass in the suprasellar cistern which is normally a CSF filled, five pointed star. This presents the suprasellar extension of the previously unknown pituitary adenoma which has undergone infarction or spontaneous haemorrhage—that is, pituitary apoplexy.

thought to be normal or to suggest tumour, but without a focal enhancing mass. Frequently MR is requested and while the patient waits for this there is a rapid clinical decline often associated with progressive basilar occlusion or malignant swelling of the cerebellar infarction and life threatening hydrocephalus. Intra-arterial thrombolysis can be effective even up to 48 hours after the onset; early diagnosis and treatment are required. Keep the clinical scenario in mind as you assess the CT scan. It may be normal, or may show low attenuation in part of the cerebellum, small “tiger’s eye” infarctions in the central thalamus of terminal basilar perforator ischaemia, or even hyperdensity in the vertebral or basilar artery; this may well be the first sign so always look

Figure 9 (A) The typical lesions of demyelination are seen on this T2 weighted image involving the corpus callosum and lying at right angles to the ventricular wall. (B) Axial FLAIR sequence shows the typical non-specific white lesions at the junction of the grey and white matter typical of age related (presumed) ischaemia.

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for it. CT angiography with detailed vascular imaging from the level of the fifth cervical vertebra to include the circle of Willis will show any vessel occlusion or dissection (fig 7).

Acute visual failure ($\pm$ 3, 4, 6, nerve palsy)

Clearly this requires a careful examination of the orbits, cavernous sinuses, and parasellar region. CT, as the usual first examination, will show a pituitary infarction or haemorrhage into an (unknown) adenoma of pituitary apoplexy or enlargement of the cavernous sinuses from cavernous sinus thrombosis which can be proved with CT venography. Always check the visual cortex in case there has been bilateral occipital ischaemia (fig 8).

WHAT NEUROLOGISTS NEED TO KNOW ABOUT MR IMAGING

The nature of the technology means that good quality MR imaging is easily achieved with much less effort by the radiographer or radiologist than it is to get it “right” with CT. Frequently general radiologists will be comfortable performing neuro MR but will be less confident in reporting it than CT. MR is optimal for the identification of inflammatory/ischaemic diseases of the brain and spine parenchyma and degenerative/malignant spinal disease, and in these it excels. Some mass lesions, haemorrhage/haemorrhagic lesions, and skull base disease can often be much more confusing with MR and it may be simpler to do a CT scan than to perform more and more different MR sequences in an effort to resolve the confusion.

Remember, MR will not be routinely available out-of-hours in the DGH. What will you do then? Use CT or refer up to the neuro-centre?

In most situations this should be to perform CT locally.

MR brain imaging technique

- ▶ The usual routine brain MR comprises a sagittal T1 weighted sequence with axial FLAIR (fluid attenuated inversion recovery) and T2 weighted scans. These are selected to optimise the demonstration of parenchymal lesions as most pathological lesions are wetter than normal brain and so are bright on T2 and FLAIR scans.
- ▶ FLAIR shows free fluid as dark (for example, CSF), but focal parenchymal pathology as bright. Both are bright on T2.

- ▶ If you are concerned about the possibility of acute or chronic haemorrhage then you should request a gradient echo T2 sequence which exaggerates the paramagnetic effect of deoxyhaemoglobin (acute) and haemosiderin (chronic) and makes either very black.
 - ▶ To differentiate fat from subacute haemorrhage (methaemoglobin) as both are bright on T1, repeating the T1 sequence with fat saturation will obliterate the bright signal of fat but not blood.
 - ▶ If the lesion is dark on T2 then it is either air, calcified, or fibrous tissue with little water, or acute haemorrhage.
 - ▶ Enhancement with gadolinium will be done at the discretion of the radiologist but you should request it automatically if sarcoidosis or granulomatous disease is suspected.
 - ▶ If it is important to confirm acute infarction is present and CT is normal then it can be confirmed if the tissue is bright on diffusion weighted scans and dark on the accompanying apparent diffusion coefficient (ADC) map (this technique may not be routinely available).
- MR can, and should be, as simple as that!

Reading an MR brain: similar to reading a CT, but easier

- ▶ The issue with MR is not usually in the identification of the lesions but in the interpretation of the cause of that lesion. The differentiation between grey and white matter is superb so the brain anatomy is excellently displayed and there is no need to use the ventricular system to infer pathology (but you may then miss raised pressure) as pathology will be obviously “bright” within the tissues on T2. A good knowledge of brain anatomy is even more essential with MR so make sure you have it.
- ▶ Non-specific “white spot disease” is ubiquitous and reflects the oversensitivity of MR to normal age related changes in the brain. This causes most confusion for general radiologists who are keen not to miss demyelination or infarction. As a general rule ischaemic/granulomatous lesions are scattered in the hemispheres at the grey/white matter junctions and commonly present centrally within the pons. Demyelinating plaque lies at

Figure 10 (A) The sagittal T2 shows a lesion with slight mass effect and mixed dark and iso-intense signal in the dorsal half of the cord at the level of C2. The T1 image (B) shows some slight bright signal within the lesion. Yes, haemorrhage until proven otherwise in this cavernoma!

right angles to the walls of the lateral ventricles, affects the corpus callosum and also specifically affects the cerebellar peduncles and periaqueductal grey matter, and may have an associated halo of less bright tissue surrounding the lesions (fig 9).

- ▶ The FLAIR sequence is most sensitive for lesions above the tentorium but may not show lesions in the posterior fossa.
- ▶ MR angiography is not required in the investigation of possible arteriovenous malformation; they are seen on standard imaging.
- ▶ I always report brain MR with set words which ensure that I examine all the structures as I dictate the report. If you do this mentally each time you should not overlook pathology.

"There is no evidence of any structural or signal abnormality within the supra or infra tentorial compartments" (*look at each image in turn, do not just flick over the middle ones only*). "The cranio-cervical junction is normal (*check here on the midline sagittal images the parasellar region and aqueduct also*) and there is no hydrocephalus" (*recheck the temporal horns and all ventricles*). "Normal flow voids are present in the major cerebral arteries and veins" (*all arteries should be black in the standard sequences so check the circle of Willis and ensure both carotid flow voids are present in the skull base/cavernous sinus on T2; veins are variable but the sagittal sinus is black in all sequences except the top slices of the FLAIR*).

Spinal imaging

General radiologists will be well capable of assessing structural spine disease including intradural extramedullary

lesions, extradural masses, bone and disc degeneration, and malignant and infective disorders, and will usually ensure that these are referred directly to neurosurgery. Intradural pathology may be very subtle on the commonly used sagittal fast spin echo T2 sequence. It is possible to optimise your chances of detecting intramedullary pathology by asking the radiologist to include a sagittal stir T2 in all cases where spondylosis is *not* the likely diagnosis. The generalist may need help in assessing cord disease but fortunately there are only seven patterns to work out.

- ▶ **Demyelination**—plaques tend to affect the posterolateral white matter.
- ▶ **Acute transverse myelitis**—usually causes diffuse cord swelling and high signal on T2 sequences over several cord segments.
- ▶ **Ischaemia**—tends to involve mainly central grey matter. Always look for any associated black vessel flow voids—you'll be a star if you pick up a dural fistula!
- ▶ **Meningitis: infective (for example, tuberculosis) or malignant**—affects the pial surface but may cause secondary ischaemia. Surface enhancement may be seen.
- ▶ **B12 deficiency (and HIV)**—the posterior columns will be bright on T2.
- ▶ **Tumour**—this will always cause unequivocal cord expansion, unlike the slight expansion possible with most of the above conditions. It will usually be bright on T2 sequences and show enhancement with gadolinium. It may be associated with a syrinx or tumour cyst/s.
- ▶ **Haemorrhage**—a full consideration of haemorrhage in MR would require a chapter of its own, so only the very basics will be considered here. If there is any lesion which is—or contains some tissue which is—bright on T1 and very dark on T2 call it blood until a neuroradiologist says its not! This will be subacute haemorrhage and be more likely to occur in a disease process referred subacutely to a neurologist. Haemorrhage is also quite commonly seen in acute arterial cord ischaemia but not in chronic venous ischaemia associated with a dural fistula. It may be seen in tumours but you should also remember that it is (almost) universal in cavernomas which may be multiple, and it is always a cache if they are also present in the brain MR which you have subsequently requested (fig 10).

Out-of-hours spinal MR imaging

This will usually require patient transfer to the neuro-centre. If it is clinically likely to be caused by tumour or infection and not inflammatory or ischaemic disease, then in most cases MDCT will prove to be adequate, so it is well worth doing this locally first as it may save the patient an unnecessary journey.

CONCLUSIONS

I know many of you reading this may be outraged that your particular interest has been ignored, or worse still apparently belittled, but I am giving a *survival* guide not a comprehensive neurology/neuroradiology course. Remember that is why you donated the neuroradiology books to the library in the first instance and it takes six years to train a neuroradiologist!

I hope you will enjoy interacting positively with your local radiological support—you both have so much to give to each other to improve the care of your patients. Carefully select the examination you request, actively interrogate all images, and keep interest and "colour" in your approach to imaging. That way you will be more often right than wrong. Images may be monochrome but no one likes to be wrong and "sing the

blues in black and white”; “life *should* be stereo each day” otherwise we are not optimising the imaging technology for the benefit of our patients (apologies to Robbie Williams; “Escapology”).

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► Old but still the best! Layout easier to follow than more recent edition. Get it on-line.

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CORRECTION

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Fuller G, O’Beirne M, Murphy P, Oware A. “EEG happy families”: the fun way to learn about common EEG abnormalities. *J Neurol Neurosurg Psychiatry* 2005;**76**(suppl II):ii13–15. It is with mixed feelings that we report a correction to the answer table for “EEG happy families”. Mixed, because our regret for our error is tempered by our delight that so many people have taken the time to do the exercise and to let us know there is a mistake. Here, with apologies, is the correct table, with the change in bold.

EEG number	Request form	Technician’s report	Conclusion
1	B	iv	f
2	C	v	a
3	D	i	g
4	E	vii	h
5	F	iii	d
6	A	ii	c
7	G	viii	e