Epilepsy is a disorder of spontaneously recurrent seizures which are caused by abnormal electrical discharges in the brain. It affects between 0.5% and 1% of the world’s population [1]. Seizures may be divided into those that begin with a local discharge of epileptic activity and which appear focal on electroencephalograms (EEGs), termed partial seizures, and those that are initiated simultaneously throughout the brain, called generalized seizures [2]. The most common focus for a partial seizure is the temporal lobe. Complex partial seizures are a subset of partial seizures which are characterized by impairment of consciousness or memory; they most frequently originate from the temporal lobe. Although only a small percentage of patients with seizures are refractory to medical treatment, complex partial seizures are responsible for the majority of such cases [3,4]. Surgical treatment of epileptogenic structural disorders such as mesial temporal sclerosis, tumours and vascular malformations may eliminate seizures in these patients with medically intractable epilepsy. Such surgery is targeted at the ‘epileptogenic zone’ which is that part of the cortex which must be resected to eliminate seizures [5] but does not necessarily correspond to the ‘epileptogenic lesion’ seen on structural imaging [6,7].

The role of imaging is to detect and characterize the structural basis of focal seizures. Computed tomography (CT) is able to identify large structural abnormalities and remains adequate in the emergency or perioperative setting. CT is also more frequently used than MRI for the investigation of recent onset seizures in adults in the U.K., despite MRI being more sensitive to the detection of early disease. A specific cause for seizures, usually either cerebrovascular disease, primary or secondary brain tumour, is identified in fewer than 50% of patients with such recent onset seizures [8]. However, the superiority of MRI for the identification of hippocampal sclerosis, cortical abnormalities and other surgically correctable epileptogenic lesions means that it is generally preferred for the assessment of chronic epilepsy, and particularly when complex partial seizures are present. Up to 80% of patients with chronic temporal lobe epilepsy have structural lesions identified by MRI [9,10]. The MRI assessment of chronic epilepsy will be emphasized and illustrated in this review since most radiologists will be familiar with the MRI appearances of tumour, infection and inflammation which result in new onset seizures in adulthood [8].

**THE ROLE AND INDICATIONS FOR MRI IN CHRONIC EPILEPSY**

The demonstration of epileptogenic lesions by MRI is important for the treatment and prognosis of individual cases and helps select those patients with medically intractable seizures who are surgical candidates [8,10–14]. Physiological imaging investigations such as single photon emission computed tomography (SPECT), positron emission tomography (PET) and functional MRI all
provide complementary information in those patients considered for epilepsy surgery, both to help delineate the ‘epileptogenic zone’ and to identify functionally eloquent cortex [7]; however, they are inadequate for the assessment of brain structure. MRI alone is currently recommended for the neuro-imaging evaluation of patients with chronic epilepsy [14].

MRI should, ideally, be performed on all patients with an electroclinical diagnosis of partial seizures or partial seizures evolving to secondary generalized seizures since partial epilepsy is often associated with a structural abnormality of the brain [15]. Some seizures that appear generalized from the start on clinical and EEG criteria are actually rapidly spreading partial seizures [16,17] and so MRI studies are also warranted when there are unclassified or apparently generalized seizures in the first year of life (when seizure type is particularly difficult to differentiate) or in adulthood [14,18]. MRI is essential when seizures are poorly controlled with medication or are associated with progressive neurological or neuropsychological deficit [14,19]. CT is only indicated in the patient with chronic epilepsy if MRI is not readily available, if it is contraindicated, or when it may provide complementary information, for instance to detect calcification in patients with a history of congenital infection or stigmata of tuberous sclerosis.

MRI PROTOCOLS

Patients with chronic epilepsy should be examined with a specific imaging protocol which best demonstrates the likely abnormalities. Because most partial seizures and medically intractable seizures arise from the temporal lobe, and in particular from the hippocampus, oblique coronal imaging, orthogonal to the hippocampal structures, is most useful. The hippocampus lies in a plane which is seen on a midline sagittal section as the line joining the splenium of the corpus callosum to the postero-inferior frontal lobe [11]. The optimum protocol includes an oblique coronal high resolution T1-weighted volume data set through the whole brain which allows reformating in any plane, measurement of hippocampal volumes and co-registration with functional data. A spoiled gradient recalled echo acquisition with a 1.5 mm partition size is one such sequence. An oblique coronal T2-weighted sequence, typically using 3 mm thin sections, should also be obtained with either a fast spin echo or conventional spin echo technique in order to detect hippocampal signal abnormalities [20,21]. A coronal fluid attenuated inversion recovery (FLAIR) sequence is helpful to increase conspicuity of high T2 signal cortical lesions adjacent to the cerebrospinal fluid (CSF) spaces [22] although its increased yield of abnormalities in epilepsy relative to standard sequences is disputed [23–25]. Gadolinium-DTPA enhanced T1-weighted images are required to look for primary or secondary tumours, infection or inflammation in the presence of recent onset epilepsy [8]. However, intravenous gadolinium does not increase the detection of structural abnormalities in chronic epilepsy and it is not routinely administered [26] although it is useful for the characterization of abnormalities.

STRUCTURAL LESIONS IN CHRONIC EPILEPSY

The histological findings in patients undergoing surgery for temporal lobe epilepsy reveal mesial temporal sclerosis (50–70%) to be the commonest abnormality, with tumours, developmental abnormalities of neuronal migration and cortical organization, vascular malformations and post-traumatic, inflammatory or ischaemic gliosis being found less frequently [27–30]. A non-specific or normal histological examination is found in 10–25%, while dual abnormalities, which are usually due to a combination of hippocampal sclerosis and either glialoma, heterotopia or vascular abnormalities [31], are present in 8–22% of patients. A similar distribution of structural abnormalities is diagnosed in patients undergoing MRI for temporal lobe epilepsy or medically refractory epilepsy [9,10,32].

In patients with extratemporal refractory epilepsy, coexistent hippocampal pathology is rare [33], and MRI reveals tumours and vascular malformations to be the commonest lesions in the frontal lobe, tumours and cortical dysgenesis in the parietal lobe, and cortical dysgenesis and vascular malformations in the occipital lobe [32]. Lesions are less commonly identified in the presence of extratemporal seizures [34].

Mesial Temporal Sclerosis

Mesial temporal sclerosis (MTS) refers to neuronal loss and gliosis of the hippocampus which leads to reorganization of neuronal pathways and the formation of an epileptogenic focus [35]. It may be a consequence of childhood febrile seizures, encephalitis, pre- and perinatal insults or may represent a pathological response to repeated seizures [35–37]. It is the commonest abnormality in medically intractable epilepsy and surgical resection of the hippocampus and anterior temporal lobe renders up to 90% of these patients seizure-free [3,38].

The MRI assessment of MTS requires some knowledge of hippocampal anatomy. The macroscopic anatomy and internal architecture, together with imaging correlation, of the hippocampus has been extensively reviewed [39,40]. Coronal sections demonstrate the normal grey matter of the hippocampus to be isointense to cortex with T1-weighting [41] and slightly hyperintense to cortex with FLAIR sequences [42]. The hippocampal head (also called pes or foot) is bulbous and is seen in the same coronal plane as the interpeduncular cistern (Fig. 1a). It lies postero-inferior to the amygdala from which it is separated by the uncal recess of the temporal horn and the alveus, which is a thin layer of white matter. Prominent interdigitations are seen on its superior aspect at the lateral ventricular surface (Fig. 1b). The body of the hippocampus (Fig. 1c) is seen at the level of the midbrain. It is ovoid in shape and is the most uniform portion. It lies inferior to the choroidal fissure and sits on the subiculum of the parahippocampal gyrus from which it is separated by the hippocampal fissure (which may be obliterated or only partially visualized). The tail of the hippocampus is located (Fig. 1d) at or behind the midbrain where it is seen adjacent to the crura of the fornices.

The two primary MRI findings of mesial temporal sclerosis are hippocampal atrophy (usually recognized by
Fig. 1 – Normal hippocampal anatomy. (a) T2-weighted coronal image at the level of the interpeduncular cistern demonstrates the hippocampal head (arrowhead) separated from the amygdala (star) by the uncal recess of the temporal horn (curved arrow). (b) T1-weighted coronal image demonstrates the interdigitations on the ventricular surface of the hippocampal head. (c) T2-weighted coronal image at the level of the midbrain demonstrates the ovoid hippocampal body (curved arrow) inferior to the choroidal fissure. (d) T2-weighted coronal image posterior to the midbrain shows the hippocampal tail (arrowhead) adjacent to the crus of the fornix (curved arrows).
asymmetry in the case of unilateral atrophy) and increased signal intensity of the hippocampus on T2-weighted imaging (Figs 2a–d) [38,43–45]. The most recent MR investigations using visual inspection of these features have demonstrated sensitivities of 87–100% [12,44,46,47].

In analysing these features it must be appreciated that minor asymmetry of hippocampal volumes is normal [41]; however, significant asymmetry is specific for MTS and is not prevalent in normal patients [48,49]. Hyperintensity on T2-weighted sections may also be seen in the proximity of the hippocampus owing to partial volume averaging of the CSF, tumour, oedema, blood products, flow artifact [50] and developmental cysts [41]. In addition, mesial temporal hyperintensity on FLAIR sequences is mimicked by
temporal horn choroid plexus or incomplete CSF signal suppression [11,51]. However, if the high T2 signal is truly localized to the hippocampus, it has been shown to be a highly specific finding for MTS [12,20,52] and may occur in the absence of atrophy [12,53]. The whole hippocampus must be carefully studied for atrophy and signal abnormality since the changes are non-uniform in 44% of patients, most frequently being localized to the body [54]. Visual assessment of hippocampal asymmetry may be hampered by head rotation. The position of the internal auditory canals on T2 weighting and the ventricular atria [41] or middle cerebellar peduncles on T1 weighting are useful landmarks to ensure that the same coronal anatomical sections are being compared for each hippocampus.

Visual assessment may reliably detect hippocampal asymmetry of more than 20%; however, quantitative analysis is
required to assess smaller hippocampal volume ratios [55]. When hippocampal volumetric analysis is compared to qualitative analysis, the sensitivity for MTS is slightly increased [3,56]. However, the commonly used methodology of manual outlining of the hippocampus on contiguous thin section T1-weighted images (usually over 20 images) is demanding and time-consuming, and automated methods are still in their infancy [50]. It is therefore impractical in routine clinical practice and is generally only used in the pre-operative assessment of selected cases. Measurements of the T2 relaxation time may also be quantified and this improves the sensitivity for hippocampal abnormalities [57].

There are numerous secondary MR features which support a diagnosis of MTS. These include temporal horn dilatation (Fig. 2d) [58], loss of hippocampal internal architecture [59], decreased hippocampal signal on T1-weighted images and poor parahippocampal grey–white matter definition (Fig. 2d). Other findings, such as
ipsilateral atrophy of the temporal lobe [60], thalamus (Fig. 2b) [61], fornix (Fig. 2c) and mamillary body (Fig. 2a) [62], are related to the afferent and efferent pathways of the hippocampus (Fig. 3). These secondary features are present in 40–60% of patients with MTS [9,38] and help improve the diagnostic accuracy when used with the primary findings, but they are unreliable signs in their own right. Loss of hippocampal interdigitations has recently been proposed as a further major criterion for the MR diagnosis of MTS [63].

Bilateral, roughly symmetrical hippocampal atrophy is present on MRI and pathological studies in 10–15% of patients with MTS [31,64]. Assessment of bilateral abnormalities is difficult, both visually and with volumetric techniques. It is best studied by measuring absolute hippocampal volumes [62] which have a defined normative range [65], should be calculated for each centre and may be corrected for total intracranial volume [66].

The visual search for MTS must continue even in the presence of another focal lesion on MRI since dual pathology is not uncommon (Fig. 3). If an extrahippocampal lesion is surgically resected, but coexistent sclerosed hippocampus remains, there is a poor prognosis [11,67]. Similarly, undetected subtle extrahippocampal pathology is responsible for poor outcome following surgery for MTS [68].

Disorders of Neuronal Migration and Cortical Organization

Disorders of neuronal migration and cortical organization are being identified more often in patients undergoing MRI for seizure disorders [69]. An incidence of 4–7% in patients with epilepsy referred for MRI has been reported [10,70] and it is the most common presentation of these disorders [71]. They are the most common underlying lesion in infants and young children with epilepsy, accounting for up to 40% of children with infantile spasms [72]. The range of abnormalities includes cortical dysplasia (agyria, pachygyria, polymicrogyria), abnormal location of grey matter (band, laminar or nodular heterotopias) (Figs 4 & 5), schizencephaly, hemimegalencephaly, tuberous sclerosis (Fig. 6) and dysembryoplastic neuroepithelial tumour (DNET; a mixed glioneuronal neoplasm with evidence of mild dysplasia in the adjacent cortex). The MRI features of these conditions are well described [73–77]. The interpretation of these MR findings, requires particular attention to the analysis of cortical grey matter, grey–white matter

Fig. 6 – Thirteen-year-old girl with complex partial seizures and tuberous sclerosis. (a) T2-weighted axial and (b) FLAIR axial images reveal multiple high signal areas on T2-weighted sequence (arrowheads) and FLAIR sequence, within the cortex and subcortical white matter of both frontal lobes. These represent hamartomata (tubers).
boundary, white matter and the periventricular region. Minor derangements may only be detected when the volumetric data is reformatted as a tangential slice or as a surface display of the 3D reconstruction [69].

Focal cortical dysplasia (Fig. 7) is the most common major malformation and is the most frequently considered for surgical resection [78,79]. It is often located in the central and pre-central cortex [71,79]. The MRI features are of broad gyri with thick cortex (greater than 4 mm), indistinct grey–white matter junction and abnormal signal in the underlying subcortical white matter [11,72,77]. Focal polymicrogyria [80], a forme fruste of tuberous sclerosis, DNETs [81] and other low grade tumours [82] may have similar MRI appearances.

Some clinical syndromes due to disorders of neuronal migration and cortical organization have characteristic MRI appearances described. Pseudobulbar palsy and cognitive impairment are associated with bilateral perisylvian and perirolandic malformation [83] whilst gelastic epilepsy, precocious puberty and cognitive impairment are the typical clinical features of a hypothalamic hamartoma (Fig. 8) [84].

**Tumours**

Tumours are the principal structural abnormality in 12% of patients with medically intractable epilepsy referred for MRI [32]. Most of the tumours for which epilepsy surgery
is performed are located in the temporal lobe cortex [3,85].
These tumours tend to be low grade and very indolent.
There was a mean pre-operative history of 14 years of
chronic seizures in one series [85]. Gangliogliomas are the
tumours most commonly associated with an epileptogenic
focus, and there is a good prognosis following resection
[85,86]. These lesions have a low signal on T1-weighted and
high signal on T2-weighted sequences, and may demon-
strate gadolinium enhancement and mass effect [87]. Other
frequent tumours in epileptic patients are pilocytic and

Fig. 8 – Thirty-seven-year-old man with a 20 year history of gelastic
seizures and a hypothalamic hamartoma. (a) T1- and (b) T2-weighted
coronal images demonstrate a pedunculated lesion which is isointense
on the T1-weighted image (arrow), and hyperintense on the T2-
weighted image, which arises from the left side of the hypothalamus.

Fig. 9 – Thirty-two-year-old woman with longstanding complex
partial seizures secondary to a histologically proven oligoastrocytoma.
(a) T1-weighted coronal image post-gadolinium and (b) T2-weighted
axial image reveal a non-enhancing lesion which is hypointense on the
T1-weighted image, and hyperintense on the T2-weighted image (arrows),
within the right superior frontal gyrus.
fibrillary astrocytomas, DNETs and oligodendrogliomas (Fig. 9) [3,85,88].

Vascular Malformations

Vascular malformations are the cause of intractable epilepsy in 2–3% of patients in surgical series [18,29,30]. Patients have an 18% risk of developing a seizure disorder when managed conservatively over 20 years [89]. Arteriovenous malformations (AVMs) have a distinctive MR appearance owing to the cluster of round and linear signal voids (Fig. 10). Cavernous haemangiomas (Fig. 11) are considered more epileptogenic than AVMs [90], possibly owing to the greater reactive gliosis. Cavernous haemangiomas are seen as circumscribed lesions on MRI with a central area of heterogeneity corresponding to methaemoglobin, deoxyhaemoglobin and calcification and a haemosiderin ring giving a low signal on T2-weighted imaging [91]. Epileptogenic vascular malformations are more often superficial and associated with surrounding T2 hyperintensity than their non-epileptogenic counterparts [92]. Local resection of vascular malformations carries the best prognosis of all epileptogenic lesions, with an excellent chance of seizure remission [11,79].

Other MR Features Associated with Epilepsy

Epileptogenic cortical encephalomalacia and gliosis secondary to trauma, infarction or infection are well demonstrated with MRI. Destructive lesions are an important cause of childhood seizures [72]. Cerebral insults during the first 6 months of gestation result in smooth porencephalic cavities not lined by gliosis [93], while late gestational, perinatal or post-natal injury leads to focal or generalized encephalomalacia (Fig. 12) [72]. At the opposite end of the age spectrum, MRI has proved useful in the demonstration of ischaemic lesions associated with late onset epilepsy [94]. MRI has recently shown subcortical plaques to be responsible for seizure activity in the 4% of multiple sclerosis patients with epilepsy [95]. Tuberculomas and cysticercosis (Fig. 13) are the most commonly identified causes of epilepsy in developing countries and MRI may demonstrate the various stages in the development of the non-calciﬁed cortical cysticercosis lesion [96].

Complex partial and generalized status epilepticus may result in reversible hyperintense lesions on T2-weighted images in the supratentorial grey matter [97,98] and diffuse hippocampal and gyral high signal on T2-weighted sequences with additional swelling [99,100]. Focal lesions in the splenium of the corpus callosum have also been noted.

Fig. 10 – Twenty-five-year-old man with complex partial seizures secondary to an arteriovenous malformation. (a) T2-weighted axial and (b) coronal images demonstrate multiple vascular flow voids within the left parietal lobe with a dilated branch of the right middle cerebral artery (arrow in a) feeding the nidus and a dilated cortical vein (arrow in b) draining the nidus and ultimately communicating with the superior sagittal sinus (not shown).
Fig. 11 – Fifty-year-old man with complex partial seizures and a cavernous angioma. T2-weighted axial image shows ‘popcorn’ central hyperintensity surrounded by a rim of hypointensity (curved arrows) within the right uncus.

Fig. 12 – Thirty-two-year-old man with complex partial seizures secondary to ischaemic damage in early life. (a) T2-weighted axial image shows hyperintense encephalomalacia within the left frontal lobe secondary to a branch middle cerebral artery infarct. The left cerebral hemisphere is smaller than the right and there is enlargement of the left frontal paranasal sinus (arrow). (b) FLAIR coronal image reveals much of the left frontal encephalomalacia to be isointense to CSF with a rim of hyperintensity corresponding to gliosis.
Fig. 13 – Thirty-six-year-old Indian man, who last visited India 4 years previously, developed focal motor seizures secondary to cerebral cysticercosis. (a) T1-weighted post gadolinium sagittal image and (b) T2-weighted axial image at clinical presentation show a superficial ring enhancing lesion within the left frontal lobe which has a hypointense rim (arrow) and surrounding hyperintensity on the T2-weighted image. The enhancing rim of the fibrous capsule and the pericystic oedema result from death of the larva. The patient was treated with antiepileptic drugs alone. Eight weeks later (c) T2-weighted axial image demonstrates resolution of the pericystic oedema.
in epileptic patients and may be the result of antiepileptic
drug toxicity [101].

MRI FOR EPILEPSY SURGERY

Epilepsy surgery requires an MRI examination [11]. This
may be supplemented by a variety of functional imaging
investigations (functional MRI, MR spectroscopy, PET,
ictal SPECT), although the minimal imaging requirements
and cost-effectiveness of these examinations have not been
established [34]. The role of these other techniques in
epilepsy imaging is well reviewed elsewhere [7,11,50].
Functional imaging techniques can be particularly helpful
when there is discordance between the EEG focus and the
lesion or when no lesion is evident on high quality MRI
[102]. Cortical dysplasia is the abnormality most frequently
detected with PET but not with MRI in patients with
[102]. MRI also allows accurate assessment of location and
employed to place intracranial depth recording electrodes
into the mesial temporal lobes or deep frontal structures
depending on EEG surface recording, and subdural mats,
which are surgically implanted onto the surface of the
brain. MRI based stereotactic procedures are commonly
employed to place intracranial depth recording electrodes
[103]. MRI also allows accurate assessment of location and
detection of complications, when depth electrodes [104] and
subdural electrode grids [105] are placed freehand.

MRI based surgical guidance systems have also been
developed which allow three-dimensional imaging to be
presented to the surgeon with superimposed real time infor-
mation concerning the position of the surgeons ‘pointer’
[106] and this has proved useful in epilepsy surgery.

Post-operative MRI performed a minimum of 3 months
following surgery is useful, if seizures have not remitted [7].
If MRI demonstrates surgery to be incomplete then a
second operation will allow a more extensive resection and
consequently a better prognosis [7,110]. The MRI appear-
ances of cortical resection [107], corpus callosotomy [108]
and hemispherectomy [109] have all been defined.

FUTURE DEVELOPMENTS IN MR EPILEPSY
IMAGING

The future relevant technical improvements in MRI
include increased spatial resolution and improved post-
processing of data. Phased array surface coils [111] have
aided detection and characterization of seizure foci and
high field strength systems may improve resolution.
Three-dimensional surface displays or curvilinear reconstruc-
tions may be used to display the gyral morphology and aid the
visual interpretation [69]. Advances in quantitative MRI
have helped assess the relative volume and infolding of
cortical grey matter and may overcome the limitations of
subjective visual assessment [112]. Such increased sophisti-
cation will mean improved diagnostic yield and there will
likely be a reduction in the requirement for invasive EEG
recording and in the number of patients diagnosed with
cryptogenic epilepsy [71]. Functional MRI has not been
discussed but may become more important both in
detecting the location of seizure foci and in delineating
eloquent regions of the brain for pre-operative planning
[113,114].

MRI has had a huge impact on the imaging of epilepsy
over the past 10 years. MRI can identify specific abnormal-
ities, helps determine treatment and predicts prognosis.
However, there remain MRI occult epileptogenic lesions
even with optimum imaging protocols. Furthermore,
structural abnormalities on MRI are not synonymous with the
‘epileptogenic zone’ so close correlation with EEG
findings [115] and other investigations is necessary.

REFERENCES

1 Sander JWA, Shorvon SD. Incidence and prevalence studies in
epilepsy and their methodological problems: a review. J Neurol
2 Commission on Classification and Terminology of the
International League Against Epilepsy. Proposal for the classifica-
3 Jack CR Jr. Epilepsy: surgery and imaging. Radiology 1993;189:
635–646.
4 Semah F, Picot MC, Adam C, et al. Is the underlying cause of
epilepsy a major prognostic factor for recurrence? Neurology
The Treatment of Epilepsy: Principles and Practice. Philadelphia,
6 Gloor P. Approaches to the localization of the epileptogenic
lesion. In: Engel JJ, ed. Surgical Treatment of Epilepsy. New
7 Spencer SS. The relative contributions of MRI, SPECT and PET
imaging in epilepsy. Epilepsia 1994;35(Suppl. 6): S72–S89.
8 Bradley WG, Shey RB. MR imaging evaluation of seizures.
imaging findings in temporal lobe epilepsy. Int J Neuroradiol
with varying severity: MRI study of 222 patients. Neuroradiology
12 Lee DH, Gao F-Q, Rogers JM, et al. MR in temporal lobe
epilepsy: analysis with pathological confirmation. Am J Neuroradiol
13 Commission on Neuroimaging of the International League
Against Epilepsy. Guidelines for neuroimaging evaluation of
patients with uncontrolled epilepsy considered for surgery.
14 Commission on Neuroimaging of the International League
Against Epilepsy. Recommendations for neuroimaging evaluation
of patients with epilepsy. Epilepsia 1997;38:1255–1256.
16 Fisher RS, Stein A, Karis J. Epilepsy for the neuroradiologist.
17 Kobayashi K, Ohkutsu Y, Oka E, Ohtahara S. Primary and
secondary bilateral synchrony in epilepsy: differentiation by
estimation of interhemispheric small time differences during
short spike wave activity. Electroencephalogr Clin Neurophysiol
1989;23:103.
18 Bronen RA. Epilepsy: the role of MR imaging. Am J Roentgenol
19 Bronen RA, Fulbright RK, Spencer SS, Spencer DD, Kim JH,
Lange RC. Economic impact of replacing CT with MR imaging
seizures and mesial temporal sclerosis: evaluation with fast spin
23 Bergin PS, Fish DR, Shorvon SD, Oatridge A, de Souza NM, Bydder GM. Magnetic resonance imaging in partial epilepsy: additional abnormalities shown with the fluid attenuated inversion recovery (FLAIR) pulse sequence. J Neurol Neurosurg Psychiatry 1995;58:439–443.
68 Sisodiya SM, Moran M, Free SL, et al. Correlation of widespread preoperative magnetic resonance imaging changes with...