Aneurysm surgery after ISAT

R S Maurice-Williams

E
very so often in the history of medicine a major technical or pha
taceutical innovation leads to a sudden and fundamental shift in prac
tice. Such events as the introduction of general anaesthesia, the use of anti-
sepsis in surgery, and the introduction of streptomycin immediately spring to
mind. Similar events have also occurred in the narrow world of neurology and
neurosurgery. The appearance of carba-
mazepine and l-dopa had an immediate
and dramatic effect upon the extent to
which surgery was used in trigeminal
neuralgia and Parkinson’s disease. The
computed tomography (CT) scan quickly
led to the disappearance of lumbar air
encephalography and ventriculography
and the redundancy of the radiological
expertise that these older forms of
investigation required.

It seems likely that the International
Subarachnoid Aneurysm Trial (ISAT)
will have a comparable effect on aneur-
ysm surgery. Even before the publica-
tion of the results of the interrupted trial
in The Lancet in October 20021 there had
been a progressive shift from surgery to
coiling in the treatment of aneurysms
whether these had ruptured or not.
Since the publication of ISAT it is clear
that many units in the UK have almost
abandoned direct surgery. This change
may have been more marked in the UK
than in other countries for, in this
country, few patients with aneurysms
receive private treatment—meaning
that any change in policy has no
economic disadvantage for the surgeon.

The gravity of this development for
neurosurgery cannot be overstated.
A ruptured aneurysm is the most
common neurosurgical condition which
requires the highest degree of surgical
skill. The condition affects relatively
young patients who are otherwise in
good health, and treatment can make
the difference between a full cure with a
normal life expectancy on the one hand
and death or hopeless disability on the
other. Aneurysm surgery constitutes, or
at any rate constituted, a major propor-
tion of that part of cranial neurosurgery
that needed the operating microscope
and much higher neurosurgical train-
ing was dependent upon it. For many
neurosurgeons aneurysm surgery was
their life, and it is perhaps no great
surprise that some of them have been
reluctant to accept the implications of
ISAT and have argued that the need for
aneurysm surgery will persist despite it.

The main findings of ISAT may be
summarised as follows.1 At total of 2143
patients with ruptured intracranial
aneurysms were randomly assigned to
clipping (n = 1070) or coiling (n = 1073).
At one year the outcome was assessed by
a modified Rankin score of 36—that is,
patients who were dependent or dead.
A significant difference was found
between the groups and the trial was
abandoned: 22.7% of coiled patients
were dependent or dead compared with
30.6% of those subjected to surgery. The
annualised risk of rebleeding after coil-
ing was 0.16% (2 cases per 1276 patient
years) and zero for clipping.

Detailed objections to the findings of
the trial have been made both in the UK
and in the USA. In an editorial in the
British Journal of Neurosurgery Kirkpatrick
and colleagues have made a number of
cogent points.2 First, only a minority of
the patients admitted to the centres
participating in the trial were eventually
randomised, and they point out that the
results obtained may not be applicable
to the non-randomised majority.
Secondly, they state quite correctly that
clipping provides a permanent cure for
the overwhelming majority of treated
patients although if neck remnants are
left these may regrow into aneurysms
and rebleed. In contrast, complete occlu-
sion by coiling is achieved in only about
70% of aneurysms and it is known that
even fully coiled aneurysms may re-
form and bleed. A third point that they
make is that the results of the surgically
treated group do not seem to be
especially good, and that while a high
proportion of the coiling may have been
carried out by radiologists who were
very experienced in this field the same
may not have been true of the surgeons
concerned who may have had a greater
mix of ability. They argue that much
to better surgical results might have been
achieved by experienced and specialised
vascular surgeons, and they also argue
for the development of specialised cen-
tres for both surgery and endovascular
treatment.

At about the same time, a lead
editorial in the Journal of Neurosurgery
by Dr R C Heros, a leading American
vascular neurosurgeon, addressed the
data accumulated both by ISAT and in
a large series of coiled patients reported
by Murayama and colleagues.3 Heros
points out that even in the best hands
only about 60% of aneurysms can be
fully coiled at the first attempt—the
proportion being somewhat greater in small
aneurysms with narrow necks. Further-
more, recanalisation of completely coiled
aneurysms occurs in between 2% and
37% of aneurysms and the risk of this is
greatest in the largest aneurysm sacs.4

These arguments against the long
term usefulness of coiling are well
thought out but fail to take account of
certain key factors. The results of coiling
are not static—they are constantly
improving. Not only are interventional
radiologists gaining more experience
but, more importantly, the microtech-
nology of endovascular techniques is
improving and evolving all the time.
This steady improvement is apparent to
those of us who have access to inter-
ventionalists with experience and abil-
ity. In the author’s own unit there has
been a clear improvement in the results
of coiling during the past two to three
years and it now appears that virtually
all aneurysms can be successfully coiled
with minimal mortality and morbidity.
In contrast, the technology of surgery
has been static since the introduction of
the operating microscope into aneurysm
surgery in the late 1970s and early
1980s. Indeed it is difficult to see how
aneurysm surgery could be improved
further and there are reasons for believ-
ing that the results of surgery are
worsening as surgery is spread amongst
a greater number of surgeons, many of
them in training. This process is bound
to accelerate as the small number of
surgeons with a huge aneurysm experi-
ence shrinks as a consequence of retire-
ment and death.

Some further points need to be made.
First, as the active treatment of aneur-
ysms has become so universal it has
perhaps been forgotten that a very high
proportion of patients who survive in
good condition after the rupture of an
aneurysm do well without any treat-
ment at all. The celebrated study of
Alvord et al showed that patients who
survived between one and three days in
grade 1 after a ruptured aneurysm had
The international classification of headache disorders, 2nd edn (ICDH-II)

J Olesen, T J Steiner

Better classification of headache disorders enables better headache research, understanding of headache, communication, and, ultimately, management of a set of disabling neurological disorders

In the past, many have viewed the primary headache disorders as a continuum of which each is part. Better headache classifications have prompted slowly developing awareness of the existence of many discrete entities amongst these disorders. This in turn has contributed not only to their better recognition in clinic (and therefore management) but also to nosological characterisation that has produced better classification of many headache disorders.

THE ORIGINS OF ICHD-II

The first proposals for the classification of headache disorders were put forward in the 1960s, one from an ad hoc committee of the US National Institutes of Health and another, quite similar, from the Research Group on Migraine and Headache of the World Federation of Neurology. Both proposals merely listed the few headache disorders that were accepted at that time, and gave short descriptions of them rather than diagnostic criteria. The major advance, and
the first internationally acceptable—and clinically useful—classification system, came in 1988 when the Headache Classification Committee of the International Headache Society (IHS) published what is now referred to as “The International Classification of Headache Disorders, 1st edition”.

Several years of work involving more than 100 international headache experts resulted in a much more comprehensive and hierarchical classification of headache types and subtypes, both primary and secondary, using up to four digits to designate the subordinate levels. Explicit diagnostic criteria for at least most major headache disorders were also set out for the first time. This classification became universally accepted, and was translated into more than 20 languages. No competing headache classifications have existed since that time.

Although the diagnostic criteria were, in many cases, based on the opinions of experts rather than on published and verifiable data, it is testimony to the quality of those opinions that subsequent research has shown most of the criteria to be both valid and reliable. No research can be done on a disease that is not defined but, conversely, it is difficult to define a disease on which no research has been done. This situation sounds impossible but may be managed by many and often small successive steps of improvement. Thus, the advent of criteria for many headache disorders in 1988 made it possible to undertake research that would confirm, dispute, or occasionally reject the existence of, and research that would confirm, dispute, or occasionally reject the existence of, and classify headaches encountered clinically that are not yet recognised as entities in order to stimulate research into them.

The most important changes to the classification and diagnostic criteria of migraine relate to migraine with aura. The revised criteria do not fundamentally change the way we diagnose aura but are, we believe, more easy to understand and apply. Furthermore, they allow a subdivision of migraine with aura into subtypes, all characterised by the occurrence of typical aura (which itself has detailed diagnostic criteria) but, respectively, followed by migraine headache, non-migraine headache, or no headache at all. Other changes to migraine with aura have considerably sharpened the diagnostic criteria for familial hemiplegic migraine (FHM), the dominantly inherited form of migraine for which two causative genes have now been found. Sporadic hemiplegic migraine (SHM) is a new entry, and both FHM and SHM are more distinctly separated from basilar-type migraine, a new term for what was previously basilar migraine.

An important new inclusion is chronic migraine (CM), classified under complications of migraine. This diagnosis is given to patients who fulfill the criteria for pain and associated symptoms of migraine without aura for 15 or more days per month over three months or longer, without medication overuse. By far the most common cause of migraine-like headaches occurring on 15 days or more per month is medication overuse, described below, giving rise to medication-overuse headache (MOH). CM, in which by definition this is absent for at least two months, is probably a rare syndrome and no good cases have been published in detail, but all experts have seen indisputable cases. If the criteria for CM are otherwise met but medication overuse is present or suspected, the diagnoses given should be probable MOH and probable CM. In addition, because a principle of the classification is that all headache disorders present in a patient are separately diagnosed and coded, the antecedent migraine subtype (almost invariably migraine without aura) is also diagnosed. If, after medication withdrawal, the patient improves, a definite diagnosis of MOH is made and, if not, the confirmed diagnosis should be CM.

In tension-type headache (TTH) the only significant change has been the subdivision of the episodic subtype into infrequent episodic TTH and frequent episodic TTH. The former is defined as TTH occurring less than once a month and was separated out because such a mild headache disorder can hardly be regarded as a health problem, more as a normal variant in the general population (although still requiring classification). TTH is a medical problem only if its frequent episodic and chronic variants.

The major change to cluster headache and other trigeminal autonomic cephalalgias is the inclusion of short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT). In addition, an episodic subtype of paroxysmal hemicrania has been newly recognised.

### WHAT HAS CHANGED

When widely used classification systems and their diagnostic criteria markedly changed, the knowledge acquired with the earlier criteria becomes of uncertain validity for, and may not be applicable to, disorders diagnosed with the revised criteria. A consequence is that much research would have to be repeated. Fortunately, the diagnostic criteria for the most important headache disorders have not been disputed over the years since the first edition of the classification and, consequently, the criteria for migraine, tension-type headache, cluster headache, chronic post-traumatic headache, and trigeminal neuralgia have been preserved in ICHD-II. Research into these diseases, where it has used the definitions of 1988, remains valid now and will still be in the future. Published evidence has, however, dictated many other important changes.

As before, the classification separates the primary headache disorders (Chapters 1–4) from the secondary headache disorders (Chapters 5–12) (Tables 1 and 2). The former include, most importantly, migraine, tension-type headache, and cluster headache. The latter, of which there is a large number, are attributed to some other causative disorder specified in the diagnostic criteria attached to them. The appendix, a new addition in ICHD-II, includes proposed criteria for headaches encountered clinically that are not yet recognised as entities in order to stimulate research into them.

### Table 1

**Classification of primary headaches (first level, with selected disorders at second and third levels)**

<table>
<thead>
<tr>
<th>ICHD-II code</th>
<th>ICD-10NA code</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>[G43]</td>
<td>Migraine</td>
</tr>
<tr>
<td>1.2</td>
<td>[G43.0]</td>
<td>Migraine without aura</td>
</tr>
<tr>
<td>1.3</td>
<td>[G43.1]</td>
<td>Migraine with aura</td>
</tr>
<tr>
<td>1.4</td>
<td>[G43.3]</td>
<td>Chronic migraine</td>
</tr>
<tr>
<td>2.1</td>
<td>[G44.2]</td>
<td>Tension-type headache (TTH)</td>
</tr>
<tr>
<td>2.2</td>
<td>[G44.2]</td>
<td>Infrequent episodic tension-type headache</td>
</tr>
<tr>
<td>2.3</td>
<td>[G44.2]</td>
<td>Frequent episodic tension-type headache</td>
</tr>
<tr>
<td>2.4</td>
<td>[G44.2]</td>
<td>Chronic tension-type headache</td>
</tr>
<tr>
<td>3.1</td>
<td>[G44.0]</td>
<td>Cluster headache and other trigeminal autonomic cephalalgias</td>
</tr>
<tr>
<td>3.2</td>
<td>[G44.03]</td>
<td>Cluster headache</td>
</tr>
<tr>
<td>3.3</td>
<td>[G44.03]</td>
<td>Paroxysmal hemicrania</td>
</tr>
<tr>
<td>4.1</td>
<td>[G44.80]</td>
<td>Other primary headaches</td>
</tr>
<tr>
<td>4.2</td>
<td>[G44.80]</td>
<td>Hypnic headache</td>
</tr>
<tr>
<td>4.3</td>
<td>[G44.80]</td>
<td>Primary thunderclap headache</td>
</tr>
<tr>
<td>4.4</td>
<td>[G44.80]</td>
<td>Hemihypoaesthesia</td>
</tr>
<tr>
<td>4.5</td>
<td>[G44.80]</td>
<td>Migraine without aura</td>
</tr>
<tr>
<td>4.6</td>
<td>[G44.80]</td>
<td>New daily-persistent headache (NDPH)</td>
</tr>
</tbody>
</table>
Table 2 Classification of secondary headaches (first level, with selected disorders at second and third levels)

<table>
<thead>
<tr>
<th>ICHD-II code</th>
<th>ICD-10NA code</th>
<th>Diagnosis [and aetiological ICD-10 code]</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.</td>
<td>[G44.88]</td>
<td>Headache attributed to head and/or neck trauma</td>
</tr>
<tr>
<td>5.1</td>
<td>[G44.880]</td>
<td>Acute post-traumatic headache</td>
</tr>
<tr>
<td>5.2</td>
<td>[G44.3]</td>
<td>Chronic post-traumatic headache</td>
</tr>
<tr>
<td>5.3</td>
<td>[G44.841]</td>
<td>Acute headache attributed to whiplash injury [S13.4.4]</td>
</tr>
<tr>
<td>5.4</td>
<td>[G44.841]</td>
<td>Chronic headache attributed to whiplash injury [S13.4.4]</td>
</tr>
<tr>
<td>6.</td>
<td>[G44.81]</td>
<td>Headache attributed to cranial or cervical vascular disorder</td>
</tr>
<tr>
<td>6.2.2</td>
<td>[G44.810]</td>
<td>Headache attributed to subarachnoid haemorrhage [SAH] [I60]</td>
</tr>
<tr>
<td>6.4.1</td>
<td>[G44.812]</td>
<td>Headache attributed to giant cell arteritis [GCA] [M31.6]</td>
</tr>
<tr>
<td>7.</td>
<td>[G44.82]</td>
<td>Headache attributed to non-vascular intracranial disorder</td>
</tr>
<tr>
<td>7.1.1</td>
<td>[G44.820]</td>
<td>Headache attributed to idiopathic intracranial hypertension (IIH) [G93.2]</td>
</tr>
<tr>
<td>7.2.1</td>
<td>[G44.820]</td>
<td>Post-dural puncture headache [G97.0]</td>
</tr>
<tr>
<td>7.4</td>
<td>[G44.822]</td>
<td>Headache attributed to intracranial neoplasm [C00-D48]</td>
</tr>
<tr>
<td>7.6.2</td>
<td>[G44.82]</td>
<td>Post-seizure (post-ictal) headache [G60.x or G41.x to specify seizure type]</td>
</tr>
<tr>
<td>8.</td>
<td>[G44.4 or G44.83]</td>
<td>Headache attributed to a substance or its withdrawal</td>
</tr>
<tr>
<td>8.2</td>
<td>[G44.41 or G44.83]</td>
<td>Medication-overuse headache (MOH)</td>
</tr>
<tr>
<td>9.</td>
<td>[G44.821]</td>
<td>Headache attributed to bacterial meningitis [G00.9]</td>
</tr>
<tr>
<td>9.4.1</td>
<td>[G44.21]</td>
<td>Chronic post-bacterial meningitis headache [G00.9]</td>
</tr>
<tr>
<td>10.</td>
<td>[G44.882]</td>
<td>Headache attributed to disorder of homeostasis</td>
</tr>
<tr>
<td>11.</td>
<td>[G44.84]</td>
<td>Headache or facial pain attributed to disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cranial structures</td>
</tr>
<tr>
<td>11.2.1</td>
<td>[G44.841]</td>
<td>Cervicogenic headache [M99]</td>
</tr>
<tr>
<td>12.</td>
<td>[R51]</td>
<td>Headache attributed to psychiatric disorder</td>
</tr>
<tr>
<td>12.1</td>
<td>[R51]</td>
<td>Headache attributed to somatisation disorder [F45.0]</td>
</tr>
<tr>
<td>12.2</td>
<td>[R51]</td>
<td>Headache attributed to psychiatric disorder [code to specify aetiology]</td>
</tr>
<tr>
<td>13.</td>
<td>[G44.847, G44.848 or G44.85]</td>
<td>Cranial neuralgias and central causes of facial pain</td>
</tr>
<tr>
<td>13.1</td>
<td>[G44.847]</td>
<td>Trigeminal neuralgia</td>
</tr>
<tr>
<td>14.</td>
<td>[R51]</td>
<td>Other headache, cranial neuralgia, central or primary facial pain</td>
</tr>
<tr>
<td>14.1</td>
<td>[R51]</td>
<td>Headache not elsewhere classified</td>
</tr>
<tr>
<td>14.2</td>
<td>[R51]</td>
<td>Headache unspecified</td>
</tr>
</tbody>
</table>

Under other primary headaches are a number of newly included headache disorders that are fairly rare but should be known to neurologists: hypnic headache, new daily persistent headache (NDPH), hemicrania continua, and primary thunderclap headache.

A clear improvement to the second section of the classification is that the criteria for all secondary headaches are now built over the same frame: criterion A specifies the headache characteristics, criterion B requires the presence of the causative disorder (sometimes, when this is important, with another set of diagnostic criteria), criterion C defines the causal relationship (often just a close temporal relation) and criterion D demands that the headache greatly improves or disappears after cure or remission of the causative disorder. When A–C are fulfilled but not D, it is generally recommended to diagnose headache probably attributed to [the presumed causative disorder]. There are exceptions, although only chronic post-traumatic headache, chronic headache attributed to whiplash injury, and chronic post-bacterial meningitis headache are currently recognised entities of this sort. Others are included in the appendix for lack of evidence to support their existence. This scheme allows a much better characterisation of each headache and will hopefully stimulate more nosological research into the secondary headaches.

Causation is the key issue in the classification of the secondary headaches. The terminology used for the secondary headaches has been strengthened in ICHD-II to reflect better evidence of causation. Previously these headaches were described as associated with the causative disorder but this term has been replaced throughout by attributed to. Nevertheless, causation is not always clinically apparent or certain. When, for example, a patient develops a headache for the first time with or shortly after head trauma and this headache persists for months or longer, few would disagree with a diagnosis of chronic post-traumatic headache. Such de novo headache, and only such, occurring in close temporal relation to another disorder that is recognized cause of headache, was recognized in the 1988 classification. This led to some unacceptable situations. A patient who had infrequent episodic TTH, but after head trauma, experienced frequent and severe headaches that phenomenologically still conformed to the definition of TTH could not in the past receive a diagnosis of post-traumatic headache, only of TTH. This problem has been solved by allowing (according to the circumstances) one or two diagnoses: the primary headache diagnosis—in this case infrequent episodic TTH, with or without a secondary headache diagnosis—in this case chronic post-traumatic headache. The following circumstances support the addition of the secondary headache diagnosis: (i) a close temporal relation exists between the suspected cause and the apparent aggravation of the previously present headache; (ii) the suspected cause is known to be able to cause headache such as is now present; (iii) the aggravation of the previously present headache is very marked; (iv) the headache greatly improves (returning to its previous pattern) or disappears within three months after cure or remission of the suspected causative disorder.

All chapters on the secondary headaches have benefited from much more careful review, and include better descriptions and are better referenced than in the first edition. Two new chapters have been added. One deals with headache attributed to disorder of homeostasis and includes headaches due to systemic disorders such as high blood pressure, hypoxia, and hypercarbia as well as hormonal, fluid, and other disturbances. The other new chapter recognizes headache attributed to psychiatric disorder. In the 1988 edition, psychiatric diseases were recognized only for their ability to be a cause of TTH. Now they are placed in line with other causative diseases. Unfortunately, very few research studies have focused on headache in psychiatric patients, and the chapter contains only two entities that were considered to be proved beyond doubt to cause (rather than be comorbid with) headache. Yet, in the opinion of the experts, psychiatric disorders can quite frequently be the cause of headache and a number of these have been set out, with carefully proposed diagnostic criteria, in the appendix. Hopefully, these will encourage more epidemiological and nosological research in this field.

A clinically very important entity, now in ICHD-II, is medication-overuse headache (MOH). Previously this was inadequately covered under headache associated with chronic use of a substance. It is now well documented that frequent and regular use over time of acute antimigraine medication and/or analgesics by people with migraine or TTH risks aggravation of the primary headache; this is the most common cause of a chronic migraine-like syndrome. The new term avoids the pejorative abuse or misuse since, in their traditional senses, these do not apply to the great majority of
patients. Overused medications are often prescribed by a doctor who is sadly unaware of the risks of over-frequent use, and used within limits specified in the prescription. The limits indicated are for triptans, ergotamine, opioids, or combination analgesics, use on 10 or more days per month, and, for simple analgesics, use on 15 or more days a month. Note that the quantity of medicine taken per month is no longer regarded as the main criterion of overuse.

CONCLUSION
IHCD-II is a major advance in the classification and diagnosis of headache disorders. Together with a growing body of evidence showing that different headache disorders often require quite different treatments, it should stimulate general practitioners (who manage most headaches) as well as neurologists and other specialists to make detailed as well as accurate headache diagnoses and tailor treatments accordingly. As in the first edition, the rule is still to diagnose and code all the distinct types of headache that a patient has. This multiple-diagnosis system has proved valuable in pointing out the various problems and thereby leading to appropriately differentiated treatments. We hope that the IHCD-II, like its predecessor, will have substantial impact upon clinical diagnosis of headache disorders and thereby improve patient management.


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Time to define a rapid scoring system

Clinical and radiological predictors of recanalisation: time to define a rapid scoring system

S Husain

Factors that predict basilar artery occlusion outcome

Acute basilar artery occlusion is a known cause of brain stem infarction that is often fatal. The most physiological way of treating this condition is recanalisation of the occluded artery to revascularise endangered ischaemic brain. Thrombolysis by intravenous recombinant tissue plasminogen activator (rtPA) brings substantial clinical benefit in acute stroke, however, a large number of patients do not respond to this treatment. Intra-arterial thrombolysis may improve overall outcome in patients with basilar artery occlusion but predicting benefit of therapy is still difficult in the individual patient. The crucial issue in acute basilar artery occlusion is defining the predictors for successful recanalisation and improved outcome.

Arnold et al. (pp 857) have tried to define clinical and radiological predictors of recanalisation and outcome in acute basilar artery occlusion. They found that a low National Institutes of Health Stroke Scale (NIHSS) score and vessel recanalisation (as seen on the angiogram) were associated with a favourable outcome. Complete recanalisation was seen in 8/40 (20%) cases whereas partial recanalisation was achieved in 24/40 (60%) cases, and a favourable outcome was seen in 14/40 (35%). This suggests that even partial recanalisation of basilar artery occlusion may bring about a favourable outcome. Angiographic recanalisation was seen more often in patients in whom intra-arterial thrombolysis was started within six hours of onset of symptoms and when the admission computed tomography (CT) scan showed a dense basilar artery occlusion sign, which represents a large diameter clot (embolic origin) in an artery with otherwise normal lumen. On the contrary, if the basilar artery is already stenosed due to atherosclerosis, it may not appear dense when occluded totally. This may be a possible explanation for the presence of

the dense basilar artery (BA) sign being favourable for recanalisation, since, in this situation, thrombolysis will cause normalisation of the arterial lumen. In this series, the predictors of poor outcome were presence of quadriplegia and coma suggesting extensive brainstem compromise. This is similar to the observation of poor outcome in middle cerebral artery (MCA) stroke patients where the perfusion-weighted CT lesion volume was more than 100 ml. Other studies have shown that small diffusion-weighted imaging lesions predict a favourable outcome. The length of the basilar artery occlusion and the state of the collaterals are other independent variables affecting survival.

The existence of the ischaemic penumbra for several hours following acute occlusion of the arterial blood supply to the brain is the basis for acute revascularisation treatment in ischaemic stroke. Depending on the location of the occlusion in a vessel and the degree of collateral blood flow, the duration of the ischaemic penumbra varies from individual to individual. It is worthwhile to identify patients in whom the parenchyma may be salvageable after longer time periods; these patients may benefit from late recanalisation treatments. The identification and quantification of the penumbra before the initiation of definitive revascularisation may be of immense benefit in identifying patients who may have a favourable outcome irrespective of the therapeutic window. The emergence of diffusion- and perfusion-weighted magnetic resonance imaging (MRI) techniques have
revolutionised the role of MRI in the evaluation of patients with acute stroke. Until recently, the diffusion–perfusion mismatch model provided a simple and feasible means to identify the ischaemic penumbra, but studies have now demonstrated that the simple diffusion–perfusion mismatch model is only a rough approximation of the ischaemic penumbra. There has been a paradigm shift towards a variety of novel concepts regarding MRI and the ischaemic penumbra. These include (i) diffusion–perfusion mismatch does not optimally define the penumbra; (ii) early diffusion lesions are in part reversible and often include both irreversibly infarcted tissue and penumbra; (iii) the visible zone of perfusion abnormality overestimates the penumbra by including regions of benign oligaemia; (iv) MRI is a very practical method for acute stroke imaging; and (v) therapeutic salvage of the ischaemic penumbra has been demonstrated in humans with the use of diffusion-perfusion MRI. The results of ongoing thrombolysis trials involving evaluation of stroke patients with MRI may contribute further to the prediction of outcome in cases of acute basilar artery occlusion.

The astroglial protein S100B is a marker of cerebral tissue damage. A single S100B value <0.4 µg/l obtained 48–96 hours after stroke onset indicates successful clot lysis <6 hours in MCA/M1 occlusion with a high degree of accuracy. A single S100B value may serve as a surrogate marker of early and sufficient recanalisation in large scale thrombolytic studies and thus help optimise doses of thrombolytic agents to reduce haemorrhagic complications.

Lastly, I think the time has come for the formulation of an outcome scale based on the evidence available from various studies on predictors of recanalisation and outcome of 40 patients with acute basilar artery occlusion treated with intra-arterial thrombolysis. J Neurol Neurosurg Psychiatry 2004;75:857–62.

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