

Aneurysm surgery after ISAT

Aneurysm surgery after the International Subarachnoid Aneurysm Trial (ISAT)

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Every so often in the history of medicine a major technical or pharmaceutical innovation leads to a sudden and fundamental shift in practice. 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 carbamazepine 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 aneurysm surgery. Even before the publication 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 overestimated. 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 proportion of that part of cranial neurosurgery

that needed the operating microscope and much of higher neurosurgical training 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 A 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 coiling 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 occlusion by coiling is achieved in only about 70% of aneurysms and it is known that even fully coiled aneurysms may reform 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 better surgical results might have been achieved by experienced and specialised vascular surgeons, and they also argue for the development of specialised centres 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.^{3,4} 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. Furthermore, 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.³

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 microtechnology of endovascular techniques is improving and evolving all the time. This steady improvement is apparent to those of us who have access to interventionalists with experience and ability. 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 believing 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 experience shrinks as a consequence of retirement and death.

Some further points need to be made. First, as the active treatment of aneurysms 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 treatment at all. The celebrated study of Alvord *et al* showed that patients who survived between one and three days in grade I after a ruptured aneurysm had

an 80% chance of being alive at two years while for those who survived between seven and 21 days the chance rose to 95%.⁵ Other studies have shown that the natural history of ruptured aneurysms treated conservatively is relatively benign. Sharr and Kelvin found that the great majority of ruptured vertebral-basilar aneurysms treated conservatively remained well many years later⁶ and the Cooperative Study of the mid 1960s found that of 34 patients who refused operation for a ruptured aneurysm 82% remained alive and well at follow up of up to six years.⁷ If so many patients who have no treatment at all do well it seems likely that they would do even better after incomplete coiling which at least provides some protection. In any case, aneurysms which are partially coiled or where the aneurysm reforms can always be re-treated with coiling in the future when it may well be that new and improved endovascular technology will be available. Even surgery is not completely foolproof. Probably only a minority of surgeons carry out routine check angiography, and without it the surgeon cannot be sure that the aneurysm they have "clipped" is indeed fully occluded.

The second point about surgery is emphasised by the results of a recent series of surgical results published in this journal.⁸ These were the results achieved by an experienced vascular surgeon in the period immediately prior to the introduction of coiling. At first sight the one year surgical results appear good with an overall mortality of 2.6% for all patients regardless of clinical grade with 83.7% in the best category of the Glasgow Outcome Score. However, these overall results disguise the fact that a high proportion of patients experienced complications and subsidiary procedures that would have

been largely avoided with endovascular treatment. Faced with the choice it does not seem likely that many neurosurgeons would choose surgery as opposed to coiling for themselves if they had a ruptured aneurysm any more than they would choose surgery as opposed to stereotactic radiotherapy if they had an acoustic neuroma.

Some vascular neurosurgeons believe that there will still be a role for them in treating aneurysms that are not amenable to endovascular treatment or where the latter has failed. The present author believes that they are deluding themselves. It is becoming apparent that the overwhelming majority of aneurysms, whatever their situation, are now amenable to coiling and the scope of endovascular treatment is bound to enlarge as experience and technology improve. While it is true that there may remain a residue of large and/or complex aneurysms that cannot be so treated it is likely that these will present such formidable surgical problems that it would be difficult to justify a surgical attack even today when we still have very experienced aneurysm surgeons. How much more difficult it will be to justify surgery as opposed to conservative management when there are no longer neurosurgeons with the day to day experience of the technical difficulties of routine aneurysm surgery.

The consequences of ISAT are extremely serious not only for vascular neurosurgeons but also for neurosurgery as a whole. At one blow a major part of neurosurgery is under threat of extinction. For those who have specialised in vascular neurosurgery this situation can be fairly described as catastrophic. A worrying feature is that as neurosurgery breaks down into subspecialties, many neurosurgeons will be exposed to the possibility that technical advances will

make their experience and expertise redundant. As far as aneurysm surgery is concerned there is no escaping the fact that the writing is on the wall much as it was for the stagecoach in the late 1820s. The ostlers and postilions and even some of the passengers may have attempted to point out the problems associated with the new steam engines but for the detached observer there could be no doubt about the way that the future was going to develop.

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The international classification of headache disorders

The international classification of headache disorders, 2nd edn (ICDH-II)

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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 research that has produced better characterisation 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 Health¹ and another, quite similar, from the Research Group on Migraine and Headache of the World Federation of Neurology.² 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”.³

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,⁴⁻⁶ 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 the validity of the criteria coupled with, each defined disorder. It is on the basis of a large number of such studies that the second Classification Subcommittee of the IHS has, over the past four years, developed “The International Classification of Headache Disorders, 2nd edition” (ICHD-II).⁷

WHAT HAS CHANGED

When widely used classification systems and their diagnostic criteria markedly change, 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.

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 subform 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 fulfil 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 in 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.

Table 1 Classification of primary headaches (first level, with selected disorders at second and third levels)

ICHD-II code	ICD-10NA code	Diagnosis
1.	[G43]	Migraine
1.1	[G43.0]	Migraine without aura
1.2	[G43.1]	Migraine with aura
1.5.1	[G43.3]	Chronic migraine
2.	[G44.2]	Tension-type headache (TTH)
2.1	[G44.2]	Infrequent episodic tension-type headache
2.2	[G44.2]	Frequent episodic tension-type headache
2.3	[G44.2]	Chronic tension-type headache
3.	[G44.0]	Cluster headache and other trigeminal autonomic cephalalgias
3.1	[G44.0]	Cluster headache
3.2	[G44.03]	Paroxysmal hemicrania
4.	[G44.80]	Other primary headaches
4.5	[G44.80]	Hypnic headache
4.6	[G44.80]	Primary thunderclap headache
4.7	[G44.80]	Hemicrania continua
4.8	[G44.2]	New daily-persistent headache (NDPH)

Table 2 Classification of secondary headaches (first level, with selected disorders at second and third levels)

ICHD-II code	ICD-10NA code	Diagnosis [and aetiological ICD-10 code]
5.	[G44.88]	Headache attributed to head and/or neck trauma
5.1	[G44.880]	Acute post-traumatic headache
5.2	[G44.3]	Chronic post-traumatic headache
5.3	[G44.841]	Acute headache attributed to whiplash injury[S13.4]
5.4	[G44.841]	Chronic headache attributed to whiplash injury[S13.4]
6.	[G44.81]	Headache attributed to cranial or cervical vascular disorder
6.2.2	[G44.810]	Headache attributed to subarachnoid haemorrhage (SAH)[I60]
6.4.1	[G44.812]	Headache attributed to giant cell arteritis (GCA)[M31.6]
7.	[G44.82]	Headache attributed to non-vascular intracranial disorder
7.1.1	[G44.820]	Headache attributed to idiopathic intracranial hypertension (IHH)[G93.2]
7.2.1	[G44.820]	Post-dural puncture headache[G97.0]
7.4	[G44.822]	Headache attributed to intracranial neoplasm[C00-D48]
7.6.2	[G44.82]	Post-seizure (post-ictal) headache[G40.x or G41.x to specify seizure type]
8.	[G44.4 or G44.83]	Headache attributed to a substance or its withdrawal
8.2	[G44.41 or G44.83]	Medication-overuse headache (MOH)
9.		Headache attributed to infection
9.1.1	[G44.821]	Headache attributed to bacterial meningitis[G00.9]
9.4.1	[G44.821]	Chronic post-bacterial meningitis headache[G00.9]
10.	[G44.882]	Headache attributed to disorder of homeostasis
11.	[G44.84]	Headache or facial pain attributed to disorder of cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cranial structures
11.2.1	[G44.841]	Cervicogenic headache[M99]
12.	[R51]	Headache attributed to psychiatric disorder
12.1	[R51]	Headache attributed to somatisation disorder[F45.0]
12.2	[R51]	Headache attributed to psychotic disorder[code to specify aetiology]
13.	[G44.847, G44.848 or G44.85]	Cranial neuralgias and central causes of facial pain
13.1	[G44.847]	Trigeminal neuralgia
14.	[R51]	Other headache, cranial neuralgia, central or primary facial pain
14.1	[R51]	Headache not elsewhere classified
14.2	[R51]	Headache unspecified

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 a recognised cause of headache, was recognised 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 recognises *headache attributed to psychiatric disorder*. In the 1988 edition, psychiatric diseases were recognised 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, new 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 ischemic 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*¹ (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 favorable 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.² Other studies have shown that small diffusion-weighted imaging lesions predict a favourable outcome.³ 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 definite 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 oligoemia; (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.⁴ 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.³ 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 acute basilar artery occlusion treated with intra-arterial thrombolysis, and assessment of the scale in further studies with large numbers of patients.

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