

sensitive to ischemia and is generally delineates the infarcted brain. Perfusion weighted imaging (PWI) defines the areas of poor cerebral perfusion. Thus comparing the DWI and PWI images, it is possible to identify areas of ischemic brain that is at risk of irreversible infarction (Diffusion-perfusion mismatch). Several new MR techniques such as susceptibility weighted imaging have been developed to exclude hematoma, hemorrhagic conversion of infarcts and presence of microbleeds, which would represent contraindications to thrombolytic therapy. The versatility of MRI may soon see this modality playing the role of a "Brain clock" to decide whether to initiate thrombolytic therapy rather than the "Epidemiological time clock" that is in use today^{12,13}. The role of various clinical, laboratory and imaging parameters in decision regarding institution of thrombolytic therapy were summarized by Higashida et al.⁹

The procedure of thrombolysis starts with a diagnostic angiogram of the cranio-cerebral circulation to document the site of occlusion, status of potential collateral pathways, and to exclude other contraindications to thrombolysis. The target vessel is catheterized using a micro-catheter, which is deployed co-axially through a guiding catheter. Systemic heparinization is carried out with administration of 5000 IU bolus of heparin followed by hourly administration of 1000 IU. Most experience of intra-arterial thrombolysis has been obtained with urokinase as the thrombolytic agent. After the clot is gently macerated using the micro-guidewire, Urokinase is infused through the microcatheter into the clot. End points of infusion are complete recanalization, infusion of 1 million units of urokinase, or 6 hours elapsed since onset of symptoms. Periodic check angiograms are obtained and the micro-catheter is repositioned as required.

LIMITATIONS OF INTRA-ARTERIAL THROMBOLYSIS

The major problem with intra-arterial thrombolysis is that this mode of therapy requires ready access to an interventional radiologist and other ancillary staff, trained in intra-arterial thrombolysis, at all times. This is a major limitation and such availability is limited to a few academic institutions. This mode of therapy also requires additional time for catheterization of the cranio-cerebral vessels and accessing the site of occlusion. Though the hemorrhagic complications are commoner with intra-arterial thrombolysis, there was no significant difference in the outcome.

PATIENT EDUCATION

Perhaps the greatest impediment in the emergency management of acute stroke is the lack of awareness amongst the general public regarding the importance of early treatment. Most members of the general public fail to correctly recognize the symptoms of stroke or are unaware that stroke is a medical emergency. Thus most stroke patients present for medical care outside the therapeutic "window period" where thrombolytic therapy can reverse the neurological deficits. Lack of accessibility to specialized stroke centers with facilities and expertise for rapid imaging and endovascular recanalization is another issue that needs to be addressed by health care administrators.^{14,15}

REFERENCES

1. American Heart Association. 1997 Heart and Stroke statistical Update 1997; 13–14.
2. Chambers BR, Norris JW, Shurvell BL, et al. Prognosis of acute stroke. *Neurology* 1987; 37: 221–225.
3. International Stroke Trial Collaborative Group. The International Stroke Trial (IST): A randomized trial of aspirin, subcutaneous heparin, both, or neither among 19435 patients with acute ischemic stroke. *Lancet* 1997; 349: 1569–1581.
4. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med* 1995; 333: 1581–1587.
5. Ringleb PA, Schellinger PD, Schranz C, et al. Thrombolytic therapy within 3 to 6 hours after onset of ischemic stroke: Useful or Harmful? *Stroke* 2002; 33: 1437–1441.
6. Katzan IL, Furlan AJ, Lloyd LE, et al. Use of tissue-type plasminogen activator for acute ischemic stroke: the Cleveland area experience. *JAMA* 2000; 283: 1151–1158.
7. Zeumer H, Hacke W, Ringelstein EB. Local intra-arterial thrombolysis in vertebro-basilar thrombo-embolic disease. *AJNR Am J Neuroradiol* 1983; 4: 401–404.
8. Furlan A, Higashida R, Wechsler L, et al. Intra-arterial prourokinase for acute ischemic stroke. The PROACT II study: a randomized controlled trial. *Prolyse in Acute Cerebral Thromboembolism*. *JAMA* 1999; 282: 2003–2011.
9. Higashida RT, Furlan AJ. Trial design and reporting standards for intraarterial cerebral thrombolysis for acute ischemic stroke. *Stroke* 2003; 34: e109–e137.
10. Brandt T, von Kummer R, Muller Kuppers M, Hacke W. Thrombolytic therapy of acute basilar artery occlusion: variables affecting recanalization and outcome. *Stroke* 1996; 27: 875–881.
11. Hacke W, Zeumer H, Ferbert A, Brückmann H, Del Zoppo G. Intraarterial thrombolytic therapy improves outcome in patients with acute vertebrobasilar occlusive disease. *Stroke* 1988; 19: 1216–1222.
12. Schellinger PD, Fiebach JB, Hacke W. Imaging-based decision making in thrombolytic therapy for ischemic stroke - Present status. *Stroke*. 2003; 34: 575–587.
13. Hjørt N, Butcher K, Davis SM, et al. Magnetic resonance imaging criteria for thrombolysis in acute cerebral infarct. *Stroke*. 2005; 36: 388–397
14. Nandigam K, Narayan SK, Elangovan S, et al. Feasibility of acute thrombolytic therapy for stroke. *Neurology India* 2003; 51: 470–473.
15. Pandian JD. Feasibility of acute thrombolytic therapy for stroke: Comments. *Neurology India* 2004; 52: 126–127.

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