EFNA

European Federation of Neurological Associations

Working for people living with brain disorders

EFNA brings together pan-European federations of neurological patient groups in a "Partnership for Progress"

Our concept of 'Partnership' is the basis for all EFNA's activities. We work with the medical and healthcare professions, other patient organisations, the pharmaceutical, device and other industries, the European Commission and the European Parliament, to pursue the common aims of EFNA's members. EFNA's approach to any issue is taken solely in the interests of patients and is independent of possible external influence or conflict of interest.

EFNA members and affiliates

- Euro-Ataxia
- European Parkinson's Disease Association (EPDA)
- European Dystonia Federation (EDF)
- Stroke Alliance For Europe (SAFE)
- European Network for Research in Alternating Hemiplegia in Childhood (ENRAH)
- Retina Europe
- European Huntington Disease Association (EHDA)
- European Multiple Sclerosis Platform (EMSP)
- Motor Neurone Disease Association (MND) – Europe
- European Headache Alliance (EHA)
- Progressive Supranuclear Palsy (PSP) – Europe
- European Alliance of Neuromuscular Disorders Associations (EAMDA)
- International Brain Tumour Alliance
- Guillain-Barré Support Groups – Europe
- Neuropathic Pain Alliance

Among our many activities (see our website at www.efna.net) two recent events stand out. Both are held during the EFNS (European Federation of Neurological Societies) annual congress.

Awareness Day – Multi-disciplinary, allowing real dialogue on important matters of common interest among patients and carers, health professionals, scientists and policy makers. In partnership with EFNS Education Committee.

Special Congress Session: "The Good Life" - With the diagnosis of a severe neurological disease, life changes for ever in some respects. But that change need not be completely negative and some people find strengths they were unaware of before. This session invites you to walk with us through literature, art and music, as seen through the mirror of people living with a severe neurological disorder .

Dates for 2007 - at the EFNS Congress in Brussels

Awareness Day: *Friday 24 August*. The Good Life: *Sunday 26 August*.

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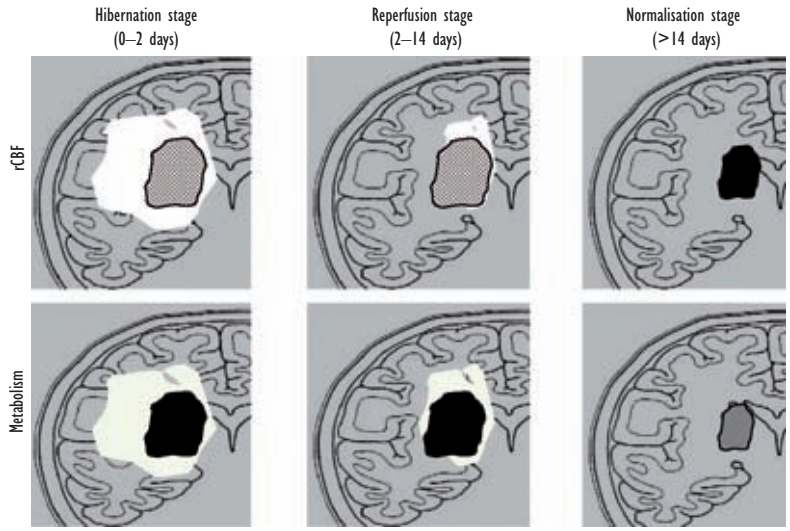
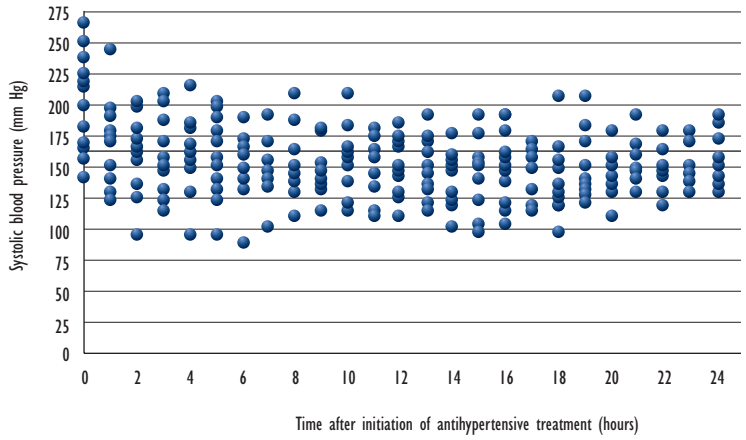
Figure 1: Different Phases of the Cerebral Blood Flow and Metabolism in the Perihaematoma



rCBF: regional cerebral blood flow

Reprinted with permission from publisher Elsevier. Qureshi AI, et al., Neurosurg Clin N Am *(2002);13: pp. 355–370.*

Figure 2: Showing Systolic Blood Pressure Control with Intravenous Labetolol and Hydralazine



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autoregulation in the perihaematoma and, as a result, sudden decrease in the blood pressure can lead to the vasodilatation which can increase the intracranial pressure (ICP) lowering the CPP.[23]

In an animal study performed by Qureshi et al.,[24] there was no change seen in the CBF, cerebral metabolic rate of oxygen ($CMRO_2$), and oxygen extraction fraction (OEF) in dogs with elevation of ICP and MAP after ICH. Subsequent authors with the help of radiological studies have come up with the similar conclusion. Hirano et al.[25] and Zazulia et al.[26] have shown no ischaemia in the periclot region by the use of positron emission tomography (PET), and Carhuapoma et al.[27] have shown the same results with diffusion weighted image (DWI) in patients with acute ICH. From the above studies it is clear that there is no ischaemia in the perihaematoma. The toxic effects of the blood and its products in the perihaematoma can lead to the decrease in the metabolism.[21,28,29]

The CBF and metabolic changes in the perihaematoma evolves in three different phases (see *Figure 1*):

- hibernation phase, which is seen during the first 48 hours, and is defined as a reduction in the CBF and metabolism in both ipsilateral and contralateral hemispheres;
- reperfusion phase, which is observed within 48 hours to 14 days and consist of heterogeneous pattern, including areas of normal, hypo- and hyperperfusion; and
- normalisation phase, which is seen after 14 days, and consists of normal blood flow except in non-viable tissue.[30]

The above theory, based on the careful laboratory and clinical evaluation, lays ground for the relative safety of decreasing blood pressure during the hibernation phase.

American Heart Association Guidelines

The American Heart Association (AHA) has put forward a set of guidelines with recommendation for the treatment of blood pressure in ICH. However, before the treatment, several different factors should be kept in mind; namely, chronic hypertension, ICP, age, mechanism of haemorrhage and time interval since onset. MAP should be maintained between 90 and 130mmHg.[31] In patients with elevated ICP who have an ICP monitor, CPP should be kept >70mmHg.

Pre-clinical and Clinical Studies

The current literature supports the fact the elevation of the blood pressure is associated with poor neurological outcome, haematoma expansion, with no associated perihaematoma ischaemia. Thus, treatment of hypertension in an acute clinical setting should be considered an option, although what we do not know at this point is the parameter of the blood pressure control in order to sustain an adequate CPP.

Meyer and Bauer[32] demonstrated the improvement in mortality in patients with ICH who were treated with antihypertensive medications; the results of this study were limited by the fact that the treated

Call for Papers

Deadline for abstract submission: **Sunday, 21 January 2007**
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Epidemiology of stroke ♦ Risk factors of stroke ♦ Stroke and diabetes ♦ Etiology of stroke ♦ Stroke and infections ♦ Stroke and lipids ♦ Acute stroke: Clinical patterns and practise ♦ Acute stroke: Early management and stroke units ♦ Acute stroke: Complications and early outcome ♦ Acute stroke: Treatment concepts ♦ Long-term outcome of stroke ♦ Chronic conditions and recurrences ♦ Vascular imaging ♦ Interventional neuroradiology ♦ Brain imaging ♦ Interesting cases ♦ Recovery and rehabilitation ♦ Management and economics ♦ Experimental studies ♦ Vascular surgery and neurosurgery ♦ Cerebral haemorrhage and SAH ♦ Venous diseases ♦ Heart & brain ♦ Cerebrovascular autoregulation ♦ Genetic disorders ♦ Ongoing trials ♦ Meta-analysis and review papers ♦ Large clinical trials (RCTs) ♦ Dementia / Cognition ♦ Behaviour and mood ♦ Stroke and movement disorders ♦ Intracranial aneurysms and vasospasm ♦ Vascular biology ♦ Stroke nursing

New: Nursing Symposium

Nursing and rehabilitation professions are invited to submit abstracts for a full-day teaching programme.

Submission

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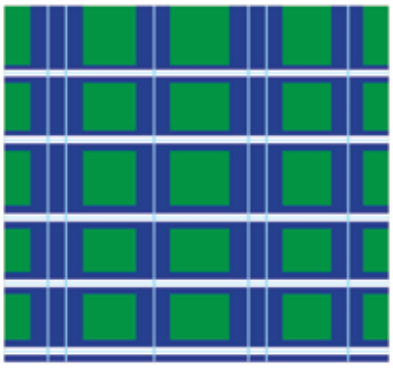

Figure 3: Showing Systolic Blood Pressure Control with Intravenous Nicardipine

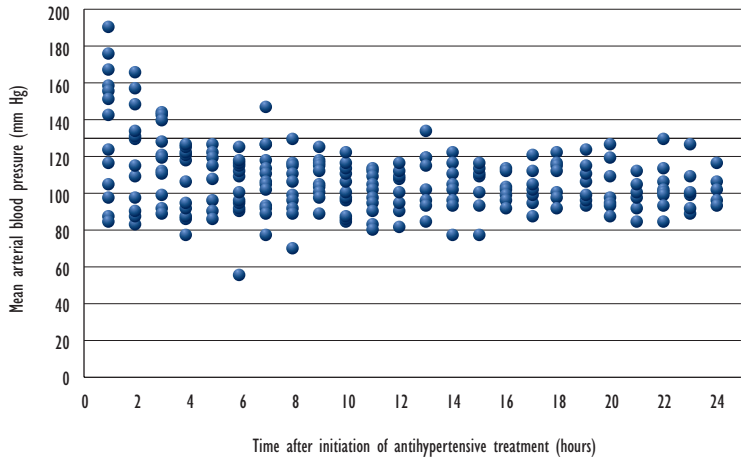

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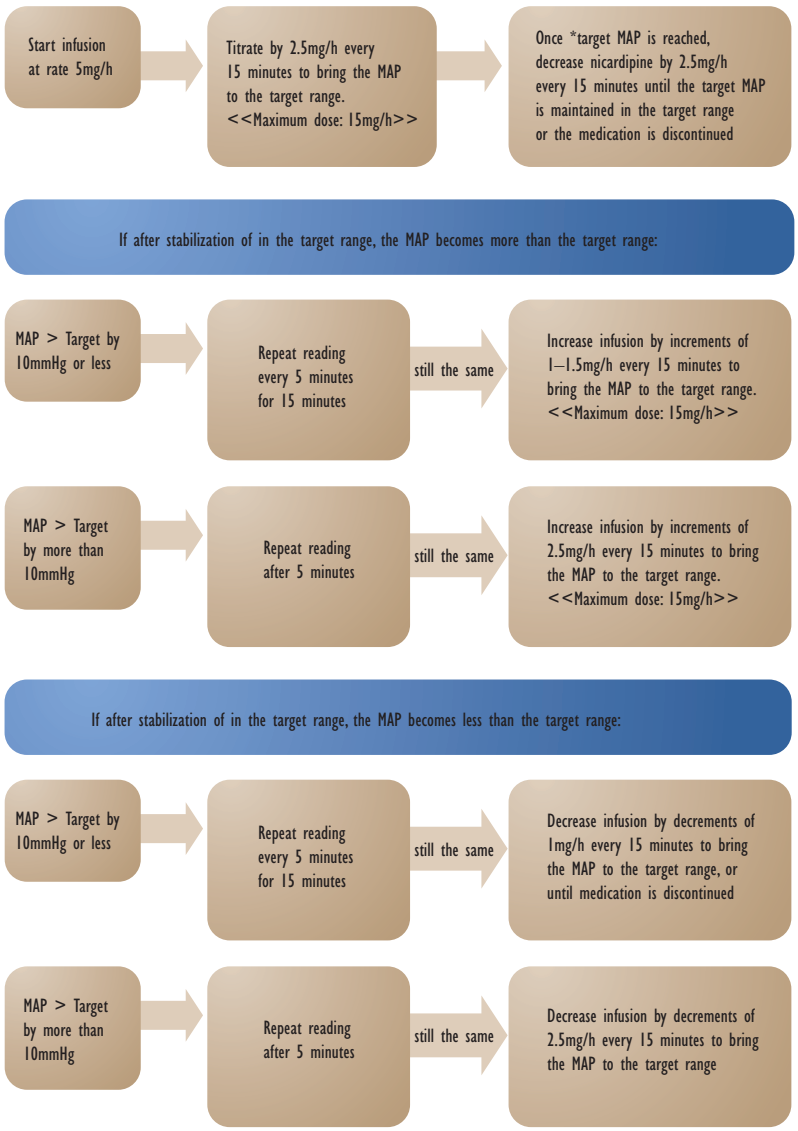
Figure 4: Nicardipine Infusion



*MAP: mean arterial pressure, *target MAP: 90–130mmHg*

group had less severe symptoms. Dandapani et al.,[12] have shown reduction in mortality and morbidity with the reduction of the blood pressure within 2–6 hours after ICH, but this study did not consider variables like ICH volume, ventricular blood, and initial Glasgow Coma Scale (GCS). Qureshi et al.,[33] showed that pharmacological reduction of MAP in normotensive experimental animals is not associated with changes in ICP and CBF around and distant to the region of the ICH. The study has its limitations, as the animals were normotensive as opposed to the majority of patients who develop ICH having chronic hypertension. Powers et al.,[34] looked at 14 patients and performed pharmacological reduction with the help of nicardipine and labetolol and showed that 15% reduction of MAP was not associated with any CBF changes in pre- and post-PET scan study. Qureshi et al.,[35] in 2004 in a multicenter prospective trial, treated patients within six hours and between six and 24 hours. This study clearly showed that patients who were treated within six hours were more likely to be independent at one month compared with patients who were treated between six and 24hours. Keeping the AHA guidelines (MAP <130mmHg) in mind, a study was designed in 2006 that recruited 29 patients.[36] These patients were treated with intravenous nicardipine, to maintain more even and effective reduction of blood pressure (See *Figures 2* and *3*). The study demonstrated that 86% of patients tolerated nicardipine, neurological deterioration was observed in 13% of patients and haematoma expansion was seen in 18% of patients.

Current Clinical Trial

The Antihypertensive Treatment of Acute Cerebral Haemorrhage (ATACH) trial[37] is designed to determine the tolerability of the treatment as assessed by achieving and maintaining three different SBP goals with intravenous nicardipine infusion for 18–24 hours post-ictus in subjects with ICH who present within six hours of symptom onset. The neurological deterioration during the treatment and any serious adverse events will also be monitored. The patients are being recruited if they have initial SBP greater than 200mmHg. The study is divided into three tiers: in the first tier the SBP is kept between 170 and 200mmHg; in the second tier between 140 and 170mmHg; and in the third tier between 110 and 140mmHg. The study will be the largest of its kind and will be able to help increase our understanding of the principles of blood pressure control in acute ICH. Currently, we use AHA guidelines to maintain MAP between 90 and 130mmHg at our institution with intravenous nicardipine infusion using our protocol (see *Figure 4*).

Initiation of Oral Antihypertensive Medications

Twenty four hours after ICH, patients should be maintained at intermediate levels (below 160/100) as high proportion of the ICH patients have chronic hypertension by the use of oral antihypertensive medications. *The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure* provides a new guideline for hypertension prevention and management. The following are the key messages:

Blood pressure elevation has been associated with poor clinical outcomes including death and dependency.

a) In persons older than 50 years, SBP of more than 140mmHg is a much more important cardiovascular disease (CVD) risk factor than diastolic BP.

b) The risk of CVD, beginning at 115/75mmHg, doubles with each increment of 20/10mmHg; individuals who are normotensive at 55 years of age have a 90% lifetime risk for developing hypertension.

c) Individuals with a SBP of 120–139mmHg or a diastolic BP of 80–89mmHg should be considered as prehypertensive and require health-promoting lifestyle modifications to prevent CVD.

d) Thiazide-type diuretics should be used in drug treatment for most patients with uncomplicated hypertension, either alone or combined with drugs from other classes. Certain high-risk conditions are compelling indications for the initial use of other antihypertensive drug classes (angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers, β-blockers, calcium channel blockers).

e) Most patients with hypertension will require two or more antihypertensive medications to achieve goal BP (<140/90mmHg, or <130/80mmHg for patients with diabetes or chronic kidney disease);

f) If BP is more than 20/10mmHg above goal BP, consideration should be given to initiating therapy with two agents, one of which usually should be a thiazide-type diuretic".[38] ■

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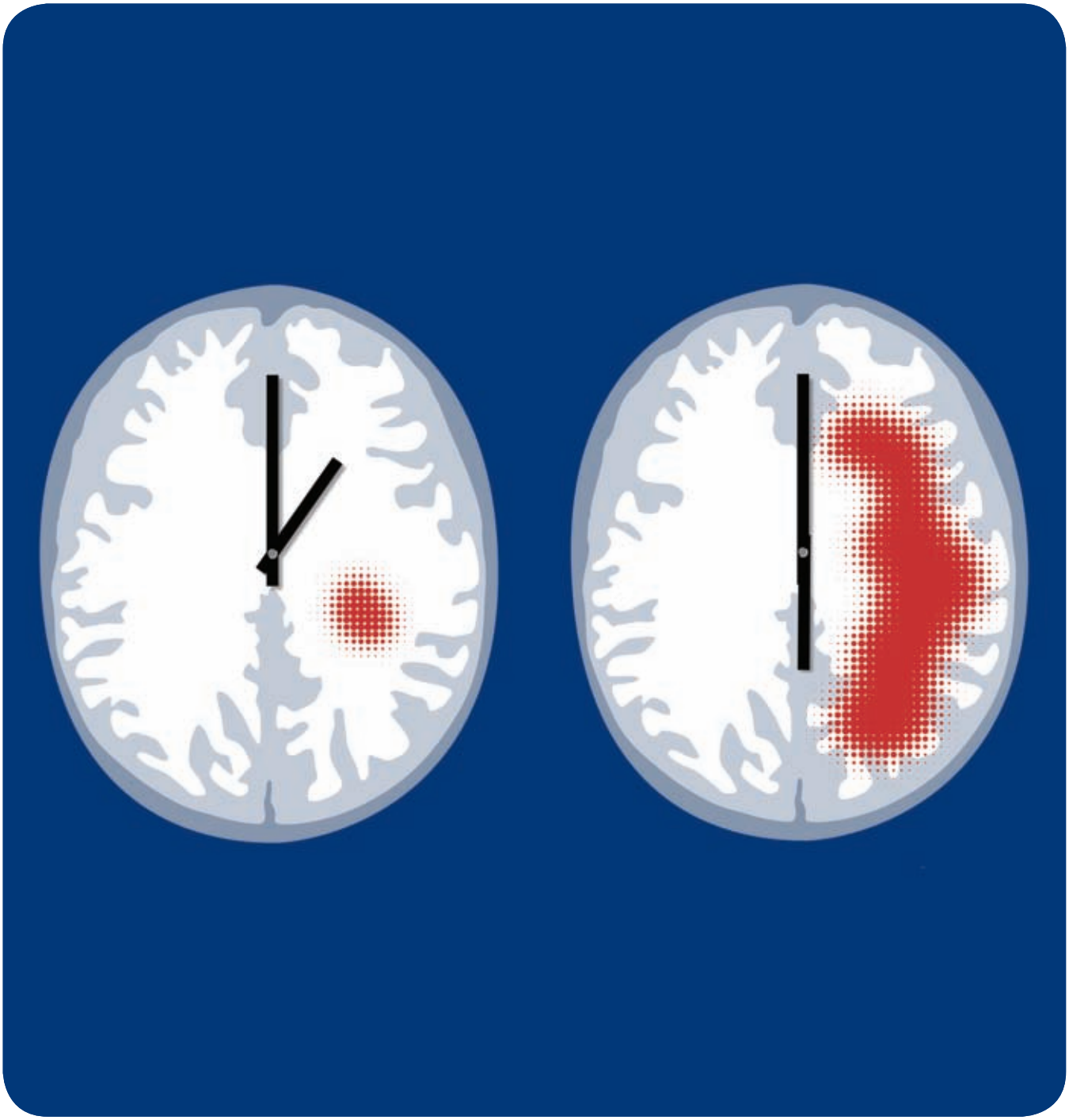

As time marches on, so does an Intracerebral Haemorrhage

Intracerebral Haemorrhage (ICH) is a dynamic process, and bleeding can continue for several hours after symptom onset.[1] Substantial early haemorrhage expansion is common in ICH patients,[1] and can have a large impact on patient outcome.[2]

Early Intracerebral Haemorrhage expansion – time to take action.

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