Management of Hypertension in stroke

Introduction

Blood pressure measurements fall into several categories. This classification can change time to time with regular guidelines from JNC.

- **Normal blood pressure.** Your blood pressure is normal if it's below 120/80 mm Hg.(JNC-7)
- **Elevated blood pressure.** Elevated blood pressure is a systolic pressure ranging from 120 to 129 mm Hg and a diastolic pressure below (not above) 80 mm Hg. Elevated blood pressure tends to get worse over time unless steps are taken to control blood pressure. Elevated blood pressure may also be called *prehypertension*.
- **Stage 1 hypertension.** Stage 1 hypertension is a systolic pressure ranging from 130 to 139 mm Hg or a diastolic pressure ranging from 80 to 89 mm Hg.
- **Stage 2 hypertension.** More-severe hypertension, stage 2 hypertension is a systolic pressure of 140 mm Hg or higher or a diastolic pressure of 90 mm Hg or higher.
- **Severe Hypertension**

Severe HPT is defined as BP ?180/110 mmHg (persistent elevation after 30 minutes bed rest). The most common cause of this condition is still longstanding poorly controlled essential HPT. The evaluation of these patients should include a thorough history and physical examination, particularly looking for signs of acute TOD and causes of secondary HPT. Patients are categorised as having:

- asymptomatic severe hypertension,
- hypertensive urgencies, or
- hypertensive emergencies
- **Hypertensive crisis.** A blood pressure measurement higher than 180/120 mm Hg is an emergency situation that requires urgent medical care.

  If you get this result when you take your blood pressure at home, wait five minutes and retest. If your blood pressure is still this high,medical help is mandatory immediately.

If you also have chest pain, vision problems, numbness or weakness, breathing difficulty, or any other signs and symptoms of a stroke or heart attack, contact your local emergency medical number.

The aim of drug therapy in patients with severe HPT is to reduce BP in a controlled, in a safe manner in order to avoid acute coronary, cerebral or renal ischaemia,or if ischaemia is already present, to avoid aggravating the situation.

- *Rapid reduction of BP (within minutes to hours) in asymptomatic hypertension or hypertensive urgencies is best avoided as it may precipitate ischaemic events.*

Both numbers in a blood pressure reading are important.*But after age 50, the systolic reading is even more important*

Isolated systolic hypertension is a condition in which the diastolic pressure is normal (less than 80 mm Hg) but systolic pressure is high (greater than or equal to 130 mm Hg). This is a common type of high blood pressure among people older than 65.

Home monitoring is an important way to confirm if you have high blood pressure, to check if your blood pressure treatment is working or to diagnose worsening high blood pressure.

History of CVD, chronic kidney disease (CKD), heart failure, it is appropriate t tae measurements at intervals, and repeated over further months to estimate the effect of treatment.

Management

Points to be discussed:

- **Non pharmacological measures**
- **Treatment targets**
- **Pharmacological measures**
- **Goals**
- **Treatment of special situations – in this article, Strokes of different types will be discussed**

Initial non-pharmacological methods

1. Eat healthy foods. Eat a heart-healthy diet. ...
2. Decrease the salt in your diet. Aim to limit sodium to less than 2,300 milligrams (mg) a day or less.
3. Maintain a healthy weight.
4. Increase physical activity.
5. Limit alcohol.
6. Don't smoke.
7. Manage stress.
8. Monitor your blood pressure at home.

Treatment targets

Before understanding treatment targets, an idea must be formed about nature of manifestations how high BP causes target organ damage (TOD)

Manifestations of TOD (target organ damage) /(target organ complication) –TOC

Organ system	**Manifestations**
Cardiac	Left ventricular hypertrophy (LVH), coronary heart disease (CHD), heart failure
Cerebrovascular	Transient ischaemic attack (TIA), stroke
Peripheral vasculature	Absence of one or more major pulses in extremities (*except dorsalis pedis*) with or without intermittent claudication, presence of carotid bruit.
Renal	GFR <60 ml/min /1.73m^2, proteinuria (≥1+), microalbuminuria (2 out of 3 positive tests over a period of 4-6 months)
Retinopathy	Haemorrhages or exudates, with or without papilloedema

Cardiovascular Risk Stratification

Co-existing condition BP levels(mmHg) DBP 100 – 109 SBP 120 – 139 and/or DBP 80 – 89	No RF No TOC No TOC Low	TOD or RF (1 - 2), No TOD Medium	TOD or RF (≥3) or Clinical atherosclerosis High	Previous MI or Previous Stroke or Diabetes Mellitus(DM) Very High
SBP 140 – 159 and/or DBP 90 – 99	Low	Medium	High	Very High
SBP 160 – 179 and/or	Medium	High	Very High	Very High
SBP 180 – 209 and/or	High	High	Very High	Very High
SBP >210 and/or DBP >120	Very High	Very High	Very High	Very High

Goals

Before understanding treatment targets, an idea must be formed about nature of manifestations how high BP should be diagnosed and investigated first and categorise patients accordingly.

Diagnosis

Methods of preliminary testing

- **Ambulatory monitoring.** This 24-hour blood pressure monitoring test is used to confirm if you have high blood pressure.

  The device used for this test measures your blood pressure at regular intervals over a 24-hour period and provides a more accurate picture of blood pressure changes over an

average day and night. However, these devices aren't available in all medical centres, and they may not be reimbursed.

- **Lab tests.** Your doctor may recommend a urine test (urinalysis) and blood tests, including a cholesterol test.
- **Electrocardiogram (ECG or EKG).** This quick and painless test measures your heart's electrical activity.
- **Echocardiogram.** Depending on your signs and symptoms and test results, your doctor may order an echocardiogram to check for more signs of heart disease. An echocardiogram uses sound waves to produce images of the heart.

Further line of investigations

HPT is a silent disease; 64% of cases remain undiagnosed.

Therefore, BP should be measured at every chance encounter. Evaluation of newly diagnosed hypertensive patients has three main objectives i.e.:

1. To exclude secondary causes of HPT.
2. To ascertain the presence or absence of target organ damage (TOD).
3. To assess lifestyle and identify other cardiovascular risk factors and/or concomitant disorders that affect treatment and prognosis.

The baseline investigations should include the following:

- Full blood count (FBC)
- Fasting lipid profile
- Urine albumin excretion or albumin/creatinine ratio
- Fasting blood sugar (FBS)
- Urinalysis
- Electrocardiogram (ECG)
- Renal profile and serum uric acid
- Chest x-ray (if clinically indicated)

Category	**Target Blood Pressure (mmHg)**	
Uncomplicated HPT	<140/90	Once target BP is achieved,
HPT in high risk groups: Diabetes Mellitus, history	<130/80	follow up at 3-6

Effective Antihypertensive Combinations

Effective combination	**Comments**
β-blockers + diuretics	Benefits proven in the elderly, cost-effective. However, may increase the risk of new onset DM
β-blockers + CCBs	Relatively cheap, appropriate for concurrent CHD
CCBs + ACEIs/ARBs	Appropriate for concurrent dyslipidaemias and DM
ACEIs + diuretics	Appropriate for concurrent heart failure, DM and stroke
ARBs + diuretics	Appropriate for concurrent heart failure and DM

Oral Treatment for Hypertensive Urgencies

Drug	Dose	Onset of Action (hr)	Duration (hr)	Frequency (prn)
Captopril	25 mg	0.5	6	1-2 hours
Nifedipine	10-20 mg	0.5	3-5	1-2 hours
Labetalol	200-400 mg	2.0	6	4 hours

Most patients can be effectively managed by their own family practitioners. Patients with the following conditions should be referred to the appropriate specialist for further assessment:

- hypertensive urgency or emergency
- suspected secondary hypertension
- resistant hypertension
- recent onset of TOC/TOD
- pregnancy
- children <18 years' old

Strokes –A TOD

Strokes can be broadly defined in 2 categories and the management of Hypertension varies accordingly.

A] Ischaemic stroke

Fundamental knowledge of anatomical changes prior to management of ischaemic stroke**.**

Auto regulatory mechanisms

Autoregulation is the intrinsic capacity of resistance vessels in end organs, such as heart, kidney, and brain, to dilate and constrict in response to dynamic perfusion pressure changes, maintaining blood flow relatively constant

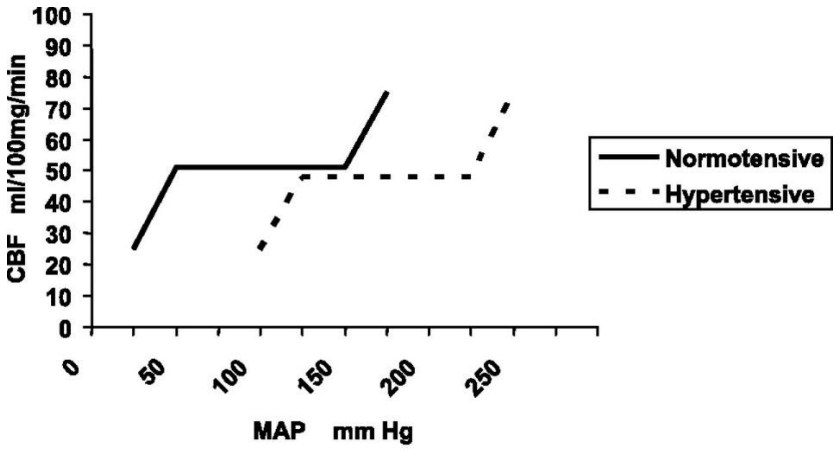
Brain

Despite its comparatively small size, the brain receives a disproportionate amount of blood flow compared with most other organ systems. Cerebral blood flow is closely coupled to brain metabolism and can be affected by respiratory-induced CO_2 changes and arterial blood pressure.

Autoregulation is the intrinsic capacity of resistance vessels in end organs, such as heart, kidney, and brain, to dilate and constrict in response to dynamic perfusion pressure changes, maintaining blood flow relatively constant.

This rapid vascular response occurs within seconds of arterial pressure fluctuations. The exact mediators of cerebral autoregulation are not completely understood.

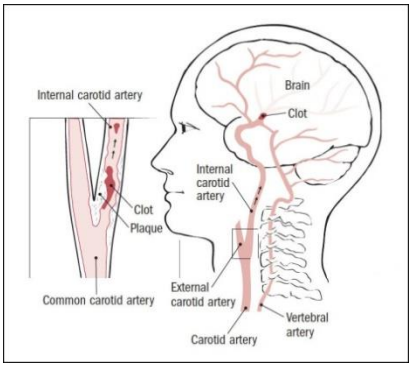
However, neurogenic stimuli; metabolic factors, such as adenosine accumulation during low perfusion; and direct intravascular pressure effects on smooth muscle or mediated via endothelial-derived relaxation factor (i.e., NO) and constriction factor (ie, endothelin-1) have been implicated.



CBF=cerebral blood flow; MAP=mean arterial pressure

Already damaged carotids

Carotid artery disease occurs that deliver blood to your brain and head (carotid arteries). The blockage increases your risk of stroke, a medical emergency that occurs when the blood supply to the brain is interrupted or seriously reduced.



This picture demonstrates that due to stenosis, the ICA is already compromised, further reduction in BP, except in certain absolute conditions(Heat failure, Aortic dissection etc), in a stroke setting will reduce blood flow to the brain further.

Estimation of dynamic cerebral autoregulation from spontaneous fluctuations of arterial blood pressure (ABP) and cerebral blood flow velocity (CBFV) is an attractive monitoring option for cerebral hemodynamic impairment.

Patients with carotid artery stenotic disease and poor cerebral haemodynamic reserve are in increased risk of stroke. Haemodynamic reserve can be estimated by measuring cerebrovascular reactivity induced by breathing CO2 and pressure-autoregulation by analyzing spontaneous slow fluctuation in arterial pressure and MCA blood flow velocity.

The correlation indexes Dx and Mx were significantly higher ipsilateral to stenosis and increased with degree of stenosis, indicating increasing dependence of CBFV on ABP and thus impairment of cerebral auto regulation.

When calculating CO2 reactivity in patients with carotid artery disease, changes in arterial pressure should be considered. Both CO2 reactivity and pressure-autoregulation describe the magnitude of haemodynamic deficit caused by stenosis, pulsatility index expresses the asymmetry of stenosis. Thus maintaining a MAP of 110 mm Hg is advisable in ischemic stroke setting.

B] Haemorrhagic stroke

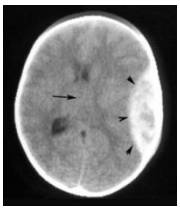
The different types of intracranial haemorrhages in brain are:

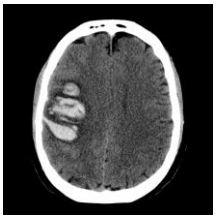
- Subdural
- Intracerebral (ICH) & Intraventricular(IVH)
- Subarachnid
- Epidural.

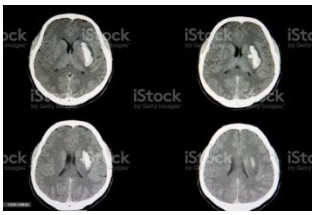
Of these the ICH & IVH are important as regards haemorrhagic strokes.

The common sites of ICH are:

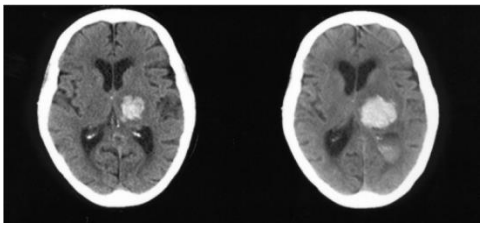
- Putaminal or basal gangionic
- Thalamo - capsular
- Cerebellar
- Brain - stem
- Lobar (related to amyloid angiopathy and ethanolism commonly)

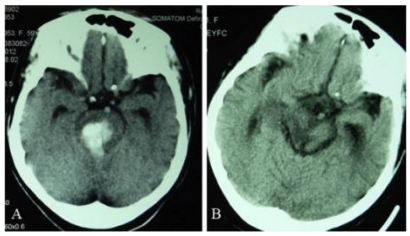






Subdural & Epidual Lobar Basal ganglionic & capsular





Thalamic bleed Brain stem bleed

Management of HTN in ICH/IVH

An initial CT with CT angio to rule out aneurismal bleed is to be done.

Then if there is an established cause the management should proceed.

Clinical staging:

- Is there is history of HTN and if so s there any drug default
- Categorizing whether the HTN is Accelerated or Malignant (with papilloedema
- GCS scoring to assess the state of damage
- Judging the age and co morbidities to decide what is the expected life span as well as quality if the patient recovers,
- Discussing the facts with relatives and knowing how much aggressive the treatment should be.

Approach Considerations

The treatment and management of patients with acute intracerebral hemorrhage depends on the cause and severity of the bleeding. Basic life support, as well as control of bleeding, seizures, blood pressure (BP), and intracranial pressure, are critical.

The presence of chronic hypertension after ICH has often been discussed as a modifiable risk factor for recurrent events. Clinical evidence is relatively lacking for clinicians to understand the extent of blood pressure lowering and the optimal agents to use in this setting.

While management of hypertension and coagulopathy are generally considered basic tenets of ICH management, a variety of measures for surgical hematoma evacuation, intracranial pressure control, and intraventricular haemorrhage can be further pursued in the emergent setting for selected patients.

Hypertensive strokes

The major risk factors for ICH include chronic arterial hypertension and oral anticoagulation. After the initial hemorrhage, hematoma expansion and perihematoma edema result in secondary brain damage and worsened outcome.

ICH is a medical emergency and initial management should focus on urgent stabilization of cardiorespiratory variables and treatment of intracranial complications. More than 90% of patients present with acute hypertension, and there is some evidence that acute arterial blood pressure reduction is safe and associated with slowed hematoma growth and reduced risk of early neurological deterioration.

However, early optimism that outcome might be improved by the early administration of recombinant factor VIIa (rFVIIa) has not been substantiated by a large phase III study.

More than 85% of ICH occurs as a primary (spontaneous) event related to rupture of small penetrating arteries and arterioles that have been damaged by chronic arterial hypertension or amyloid angiopathy. Sixty percent to 70% of primary ICH is hypertension related and, in the elderly, amyloid angiopathy accounts for up to one-third of the cases.Secondary ICH can be related to multiple causes

- ICH was previously considered a single hemorrhagic event, but it is now known that it is a complex, dynamic process with 3 distinct phases:
- Initial hemorrhage,
- Hematoma expansion,
- Perihematoma edema.[20–22] Disease progression and outcome are primarily influenced by 2 of these factors: hematoma expansion and perihematoma brain edema.
- In up to 40% of cases, the hemorrhage extends into the ventricles (intraventricular hemorrhage [IVH]) and this is associated with obstructive hydrocephalus and worsened prognosis.
- Other factors associated with poor outcome include large hematoma volume (>30 mL), posterior fossa location, older age, and admission mean arterial blood pressure (MAP) >130 mm Hg.
- The mechanisms of early hematoma growth are unclear but likely to be related to sudden increases in intracranial pressure (ICP), causing local tissue distortion and disruption, vascular engorgement secondary to obstructed venous outflow, blood-brain barrier disruption, and a local coagulopathy secondary to release of tissue thromboplastin.
- Hematoma expansion is an important cause of early neurological deterioration, the severity of which depends on original hematoma size and subsequent expansion rate.

- There is an exponential increase in mortality when the hematoma volume exceeds 30 mL.
- The 30-day mortality of patients with hematoma volume >60 mL in association with a Glasgow Coma Scale (GCS) score <8 is >90% compared with 19% for those with a hematoma volume <30 mL and a GCS score >9.
- Perihematoma brain edema develops early, evolves over many days, and is the primary cause of neurological deterioration after the first day.
- Although the presence of an ischemic penumbra around the area of the ICH was previously a concern, recent evidence does not confirm the presence of perihematoma tissue ischemia unless the hematoma is massive.

Anticoagulation induced strokes

ICH is the most feared complication of warfarin anticoagulation, and the need to arrest intracranial bleeding outweighs all other considerations.

Treatment options for warfarin reversal include vitamin K, fresh frozen plasma, prothrombin complex concentrates, and rFVIIa.

Warfarin-related ICH has a very high mortality, with reported rates up to 67%.

The need to arrest intracranial bleeding outweighs all other considerations and, although there is often a reluctance to reverse anticoagulation in patients considered to be at high risk of thrombotic complications (e.g., those with mechanical heart valves), the evidence overwhelmingly supports the correction of coagulopathy in all patients.

There is a relatively short time window for treatment and options include vitamin K, fresh frozen plasma (FFP), prothrombin complex concentrates (PCCs), and rFVIIa.

Intravenous vitamin K (5–10 mg), which supports endogenous synthesis of clotting factors, should be administered to all warfarin anticoagulated patients with ICH.

Vitamin K takes approximately 6 hours to reach therapeutic levels but has an effect that lasts beyond the relatively short half-lives of FFP and PCCs. FFP contains factors II, VII, IX, and X and is an effective way of correcting the INR acutely. However, it has a short duration of

action and, because large volumes (20–40 mL/kg) may be required, there is a risk of intravascular volume overload and heart failure.

Restarting oral anticoagulation –in patients who needs as mandatory(post CABG,PTCA)

The optimal time for resumption of oral anticoagulation is unresolved.

However, in the acute phase, the risk of recurrent ICH from restarting warfarin exceeds the risks of systemic thromboembolism from withholding it.

In patients with prosthetic heart valves, the risk of valve thrombosis in the absence of warfarin has been estimated at 1.8% per year and of ischemic stroke at 4% per year, producing an overall risk of valve-related thromboembolism of 0.2% to 0.4% over a 2-week period.

Oral anticoagulation is usually withheld for between 7 and 10 days after ICH.

Beyond this time, the risk of thromboembolic events in the absence of anticoagulation outweighs that of recurrent ICH after its reintroduction.

However, survivors of lobar ICH with atrial fibrillation should not be offered long-term anticoagulation because the risks of recurrent hemorrhage outweigh the potential benefits.

The role of IV heparin, or subcutaneous low-molecular-weight heparin (LMWH), as temporary therapy prior to reinstitution of warfarin is unclear.

Antiplatelet induced strokes

There is no evidence to guide the specific management of antiplatelet therapy–related ICH.

With an increasingly elderly population, there has been a dramatic increase in the number of patients receiving long-term antiplatelet medication.

Aspirin is associated with an absolute risk increase in ICH of 12 events per 10,000 persons, although this must be put into the context of an overall benefit of aspirin in terms of reduced risk of myocardial infarction and ischemic stroke.

High-dose aspirin increases the risk of ICH further in the elderly, particularly in association with untreated hypertension.

The risk of ICH is increased even more by the combination of aspirin and clopidogrel.

Antiplatelet therapy is also an independent predictor of hematoma expansion.

Role of neurosurgical intervention

With the exceptions of placement of a ventricular drain in patients with hydrocephalus and evacuation of a large posterior fossa hematoma, the timing and nature of other neurosurgical interventions is also controversial.

There is substantial evidence that management of patients with ICH in a specialist neurointensive care unit, where treatment is directed toward monitoring and managing cardiorespiratory variables and intracranial pressure, is associated with improved outcomes. The value of placement of a ventricular drain in patients with hydrocephalus is undisputed, but the timing and nature of other neurosurgical interventions are more controversial.

One meta-analysis failed to show a statistically significant reduction in the odds of death with surgical intervention (odds ratio, 0.84; 95% confidence interval, 0.67–1.07) compared with standard medical therapy.

The Surgical Trial in Intracerebral Hemorrhage (STICH) randomized 1033 patients with supratentorial ICH to surgery within 72 hours or conservative management; no outcome benefit of hematoma evacuation compared with standard medical therapy was demonstrated.

Because the mean time to surgery was >24 hours, STICH also does not exclude the possibility that earlier surgery might have been beneficial in some patients. However, there is evidence from other sources that ultra early surgery (within 4 hours of ictus) is associated with an increased risk of re bleeding and higher mortality (>75%).

In contrast to supratentorial lesions, there is better evidence that patients with a posterior fossa hematoma benefit from early surgical evacuation because of the high risk of deterioration.

The place of decompressive craniectomy after ICH is not established, although in a small series, 6 of 11 patients (54.5%) treated with hemicraniectomy had a good functional outcome.These findings suggest that a randomized controlled trial of decompressive craniectomy after ICH is warranted.

Hematoma aspiration via minimally invasive surgery (MIS) offers some advantages over conventional surgery, including the possibility for local anesthesia, reduced operating time, and reduced tissue trauma.

Thrombolysis, with or without clot aspiration, can also be performed using MIS, but one meta-analysis concluded that, although intraventricular thrombolysis is safe, there is no definite evidence of efficacy.

However, preliminary data from the Minimally Invasive Surgery plus rtPA for Intracerebral hemorrhage Evacuation (MISTIE) trial suggest that MIS plus recombinant tissue plasminogen activator (rtPA) offers greater clot resolution than conventional medical therapy.

A recent preliminary report of the Clot Lysis Evaluating Accelerated Resolution on Intraventricular Hemorrhage (CLEAR-IVH) trial also confirms that low-dose rtPA can be safely administered to stable IVH clots and may increase lysis rates.

BP monitoring and management is critical after ICH, but the targets for treatment remain controversial. Even in previously normotensive patients, hypertension is a very common finding and associated with worse outcome, probably because excessive hypertension is a cause of hematoma expansion.[37,38] In a recent multicenter study, systolic BP (SBP) >140 to 150 mm Hg after ICH doubled the risk of subsequent death or dependency.[39]

The risks of a sudden therapeutic reduction in BP after ischemic stroke are well known,[40] but it is possible that these same concepts may not apply after ICH because of the absence of an ischemic penumbra around small-volume ICHs.[27] A small, single-center study suggested that BP reduction in patients with acute ICH is safe and that aggressive reduction might reduce the risk of neurological deterioration in the first 24 hours after admission.[41] Two recently completed multicenter studies have provided more robust preliminary data on BP control after ICH. In the INTEnsive blood pressure Reduction in Acute Cerebral hemorrhage Trial (INTERACT), 203 patients were randomized to a low-target SBP of 140 mm Hg to be

achieved within 1 hour and maintained for at least 24 hours after ICH, and 201 were randomized to a more conservative SBP target of 180 mm Hg

The Antihypertensive Treatment in Acute Cerebral Hemorrhage (ATACH) study evaluated the feasibility and safety of 3 escalating levels of antihypertensive treatment with IV nicardipine in patients with ICH-related acute hypertension.

Preliminary data from this study suggest that reduction of SBP to 110 to 140 mm Hg in the first 24 hours after ICH is well tolerated and associated with a reduced risk of hematoma expansion, neurological deterioration, and in-hospital mortality.

Only patients with a presentation GCS score >8 and hematoma volume <60 mL are being recruited into the ATACH study, so its results will be relevant only to the less severe end of the ICH spectrum.

Haemostatic therapy

There has been interest in the application of hemostatic therapy to minimize hematoma expansion and improve outcome after ICH.

In 2005, a phase IIb placebo-controlled study showed that treatment with recombinant factor VII (rFVIIa), a potent initiator of hemostasis, within 4 hours of ICH significantly reduced hematoma growth in association with reduced mortality and improved functional outcome in survivors at 3 months.

This improvement was seen despite a small increase in thromboembolic complications in the rFVIIa-treated patients (7% vs 2% for rFVIIa and placebo, respectively, $P = 0.12$).

However, a subsequent phase III trial in 841 patients, the Factor VII for Acute Hemorrhagic Stroke (FAST) study, failed to replicate these clinical outcomes.

In this 2-dose study (rFVIIa 20 and 80 μg/kg), the dose-related reduction in hematoma expansion did not translate into a beneficial effect on the risk of death or severe disability. Post hoc analysis of the FAST data suggests that rFVIIa might be effective in a subgroup of younger patients (<70 years) with baseline ICH volume <60 mL if administered within 2.5

hours of the onset of symptoms. On balance, current evidence suggests that any potential benefit of rFVIIa is offset by a modest increase in the risk of thromboembolic complications.

Management of hypertension in haemorrhagic strokes

In up to 40% of cases, the hemorrhage extends into the ventricles (intraventricular hemorrhage [IVH]) and this is associated with obstructive hydrocephalus and worsened prognosis.

Other factors associated with poor outcome include large hematoma volume (>30 mL), posterior fossa location, older age, and admission mean arterial blood pressure (MAP) >130 mm Hg.

The mechanisms of early hematoma growth are unclear but likely to be related to sudden increases in intracranial pressure (ICP), causing local tissue distortion and disruption, vascular engorgement secondary to obstructed venous outflow, blood-brain barrier disruption, and a local coagulopathy secondary to release of tissue thromboplastin.

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Although the presence of an ischemic penumbra around the area of the ICH was previously a concern, recent evidence does not confirm the presence of perihematoma tissue ischemia unless the hematoma is massive.

Control of ICP

There is a high risk of increased ICP after large-volume ICH, particularly in the presence of IVH.

Although there is limited evidence for the monitoring and management of ICP after ICH, many neurocritical care units continuously monitor ICP in all sedated ICH patients requiring mechanical ventilation.

Anticonvulsant Therapy

Approximately 8% of patients with ICH develop clinical seizures within 30 days of the ictus, and continuous electroencephalographic monitoring demonstrates subclinical seizure activity in up to 25%.

Seizures are more likely to occur in the presence of a lobar hematoma.

The use of prophylactic anticonvulsant medication after ICH is controversial, although one small study showed that it does reduce the risk of early seizures.

Current guidance does not recommend universal prophylaxis, but that therapy should be considered in selected patients with lobar ICH.If seizures do occur, they should be treated aggressively in the usual manner.

Glycemic Control

Hyperglycaemia worsens cerebral ischemic injury, and admission hyperglycaemia is associated with increased 30-day mortality after ICH.

However, the targets for glycemic control are unclear, and there is increasing evidence that "tight" glycemic control with insulin infusion can be associated with a critically low cerebral extracellular glucose concentration after brain injury.

Until further data become available, systemic glucose levels should not be treated in the acute phase after ICH unless >10.0 mmol/L (180 mg/dL).

General Therapy

General measures, including fluid management, fever control, provision of enteral nutrition, and prevention of aspiration pneumonia or neurogenic lung injury and bedsores, are the same as for patients with ischemic stroke.

Thrombo-embolic prophylaxis with compression stockings and intermittent pneumatic compression is recommended in all patients from admission. *Subcutaneous low-molecular-weight heparin should be considered after 24 to 48 hours, when it does not seem to result in an increased risk of recurrent hemorrhage.*

Fever, is common after ICH, it can raise ICP (if exceeds 38 degrees celcius), infections should be sought, like chest (including ventilator-acquired pneumonia).Urosepsis and bed sores.

These factors are associated with increased intensive care unit and hospital length of stay and worsened outcome.

There is substantial evidence that management in a specialist neurointensive care unit results in improved outcomes after ICH.

Summary

- Hypertension is a silent killer and people should be aware of it, especially those with strong family history or those having other metabolic diseases.
- The period in which hypertension is detected is important. In young age, all means should be taken to see for secondary causes.
- If a diagnosis of essential hypertension is established, all means, pharmacological and non pharmacological are to be taken, as at every duration gaps JNC guidelines may change and classification may change. So best is to be in regular touch with a family physician and regular BP monitoring before TOD occurs.
- In this article the only TOD discussed is effect of hypertension on brain catastrophes.
- Both Ischemic and haemorrhagic strokes can occur but managing hypertension differs.

- The principle of already damaged blood vessels should be kept in mind and accordingly collaterals and auto regulation should be remembered by treating consultant.
- The management accordingly changes for lowering BP during a stroke thus varies in ischemic and haemorrhagic types.
- It is to be remembered that often a specialised Neuro-ICU is not available, except in big set ups , hence regular training of staff is required to monitor general conditions like infections, bed and bladder care, DVT prophylaxis as these makes a lot of difference in affected patients.
- Also a good surgical team is essential to be along with neurologists, physicians, intensivists as all together it's a joint effort to take care of affected patients.
- Finally it has to be remembered that the family should be in touch regularly and explained the risks and if the patient is not recoverable completely, depending on extent of lesions, then the future outcome and managing permanent neurodeficits at home should be guided.
- Finally, although hypertension is a silent killer, it should be kept in mind at all ages but if there is TOD, like in this article, strokes, a team approach can improve both quality of life of patient and reduce the mental stress of family members.
- It's a danger, but reaching the peak of Everest, was dangerous too, but was achieved by proper planning, training, knowledge and skill.