especially in low-income and middle-income countries, which have 80% of the burden of non-communicable diseases.\textsuperscript{3} Governments have the primary responsibility for ensuring that appropriate institutional, legal, and financial arrangements are provided. WHO will work with Ministries of Health, UN agencies, and international and national partners—including civil society—to implement the plan. Data collected from all countries to track national, regional, and global progress will be presented to the World Health Assembly in 2016, 2021, and 2026.

For all countries, the cost of inaction far outweighs the cost of action. However, effective implementation of the plan faces many challenges: reliable data on risk factors and mortality are needed, human and financial resources are often inadequate, and some health systems are weak. For countries with limited resources, phased scale-up of the most cost-effective interventions could be prioritised. Such interventions can greatly reduce the burden of non-communicable diseases and yet can be afforded by all countries. To implement such interventions, current health spending needs to increase by 4% in low-income countries, 2% in lower middle-income countries, and less than 1% in upper middle-income countries.\textsuperscript{4,5} For low-income countries, national spending on health might need to be supplemented with funding from international partners and development agencies. WHO will strive to assist countries to overcome the challenges and provide technical support to launch a sustainable and pragmatic national response to realise the vision of the action plan: “a world free of the avoidable burden of non-communicable diseases”.

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We declare that we have no conflicts of interest.


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**An optimum blood pressure target after lacunar stroke?**

Sustained lowering of blood pressure by 10 mm Hg systolic and 5 mm Hg diastolic lessens the risk of recurrent stroke by about one-third (relative risk 0·66, 95% CI 0·56–0·79).\textsuperscript{1,2} Larger reductions in blood pressure are associated with greater reductions in risk. This effect is consistent in most subtypes of stroke because hypertension-induced intracranial aneurysms and intracranial small-artery disease are major risk factors for haemorrhagic stroke, and hypertension-induced atrial fibrillation, atherosclerosis, and intracranial small-artery disease are major risk factors for ischaemic stroke.\textsuperscript{3} The blood-pressure range within which the association with stroke risk remains linear, however, is uncertain.

In some stroke patients, such as those with severe, bilateral carotid or vertebrobasilar occlusive disease, lowering of systolic blood pressure to less than 150 mm Hg raises the risk of stroke\textsuperscript{4} owing to loss of autoregulation of intracranial arteries and thus the maintenance of cerebral perfusion below a certain pressure threshold.\textsuperscript{6} For each stroke patient there is likely to be a lower range of blood pressure in which the benefits of lowering pressure in the prevention of recurrent stroke are maximised before the risks of haemodynamic stroke increase. Whether an optimum target range exists is unknown; stroke guidelines merely state that an absolute target for blood pressure reduction is uncertain and should be managed on an individual basis.\textsuperscript{7,8}

In *The Lancet*, the SPS3 Study Group\textsuperscript{9} report on a randomised trial in which they investigated whether a target systolic blood pressure of less than 130 mm Hg is safe and more effective than a target range of 130-149 mm Hg in patients with recent but non-acute stroke.

Among 3020 patients with subcortical lacunar ischaemic stroke in the preceding 2 weeks to 6 months, 95% in each treatment group achieved their allocated target systolic blood pressure at least once, as measured...
by a standardised automated electronic device. After 1 year, the mean systolic blood pressure was 127 mm Hg (95% CI 126–128) in the lower-target group and 138 mm Hg (137–139) in the higher-target group. This mean 11 mm Hg difference was sustained until the end of the study (mean follow-up 3.7 years) and was associated with a non-significant reduction in all recurrent stroke in the lower-target group (annualised stroke rate 2.25% vs 2.77% per year; hazard ratio [HR] 0.81, 95% CI 0.64–1.03, p=0.08). This result was consistent across various subgroups, including baseline systolic blood pressure. Intracerebral haemorrhagic stroke was reduced by about two-thirds (0.11% vs 0.29% per year; HR 0.37, 95% CI 0.15–0.95, p=0.03). Mortality and the rate of serious treatment-related adverse events did not differ significantly between groups. Thus, the SPS3 trial shows that reduction of systolic blood pressure to a target of less than 130 mm Hg in the weeks after lacunar stroke is well tolerated, safe, and effective in reducing the rate of recurrent stroke, particularly haemorrhagic stroke.

The magnitude of the reduction in rate of recurrent stroke in SPS3 was less than the 34% (95% CI 21–44) reduction seen in previous trials of blood-pressure lowering, despite a similar degree of reduction in systolic blood pressure (11 mm Hg in SPS3 vs 10 mm Hg in previous trials). Several factors might explain this difference. First, lowering of systolic blood pressure could be less effective in the prevention of recurrent stroke among patients with lacunar ischaemic stroke than among those with all types of ischaemic stroke. Limited data from previous trials, however, suggest that lowering of blood pressure has a consistent effect for all subtypes of ischaemic stroke. Second, the SPS3 estimate could be confounded by greater use of β blockers in the lower-target group than in the higher-target group. These drugs increase visit-to-visit variability in systolic blood pressure, which might raise stroke risk at any blood pressure. More patients, though, also used calcium-channel blockers and diuretics in the lower-target group, which lessen variability in systolic blood pressure.

Third, the association between systolic blood pressure and stroke risk might be weaker with systolic blood pressures lower than 130 mm Hg than with higher systolic blood pressures, such as the mean of 138 mm Hg achieved in the PROGRESS trial. Fourth, the difference in estimated stroke rate reduction in SPS3 and previous trials could simply be due to chance. The 95% CIs for both estimates of recurrent-stroke reduction are wide and overlap (SPS3 19%, 95% CI –3 to 36 vs other trials 34%, 21 to 44), which suggests consistency of the results. Furthermore, the 63% reduction in haemorrhagic stroke with a mean difference of 11 mm Hg between groups in SPS3 is consistent with the 50% (26–67) reduction in haemorrhagic stroke with a mean 9 mm Hg difference in the PROGRESS trial.

When the SPS3 results are viewed in the context of all evidence for lowering of blood pressure in individuals with previous stroke, the 19% rate reduction for all recurrent stroke seems likely to be real rather than a chance observation. The failure to reach conventional significance at the p=0.05 level is probably because the trial was underpowered to identify or exclude reliably a relative-risk reduction of 19% at the p=0.05 level. The observed rate of recurrent stroke was only half of what was anticipated (about 11% over 3.7 years vs 21% over 3.0 years), probably because of expert secondary prevention throughout the trial. The observed annual rate of serious complications of hypotension, however, was also low (0.40% and 0.26% in the lower-target and higher-target groups, respectively). Consequently, the SPS3 trial was underpowered to identify or exclude confidently a slight, but nevertheless clinically important, increase in serious complications of hypotension with systolic blood pressure lower than 130 mm Hg.

The implications of SPS3 are that clinicians should endeavour to achieve and maintain systolic blood pressures lower than 130 mm Hg in patients who have...
Intermittent pneumatic compression in patients with stroke

A patient with acute stroke has just been admitted who is immobile, and cannot walk to the bathroom without help. Looking at the patient’s unmoving legs the risk of thrombosis is clear, but a low molecular weight heparin (LMWH) might lead to bleeding, and elastic compression stockings cause skin problems. So you settle on intermittent pneumatic compression devices (IPCs)—but do they actually prevent blood clots?

Decades after IPCs were first reported to prevent venous thromboembolism (VTE) in surgical patients, Martin Dennis and colleagues1 present the results of the randomised CLOTS 3 trial in The Lancet, showing that IPCs reduce the risk of VTE in immobilised medical inpatients who have had a stroke.

VTE is among the most significant complications associated with hospital stay.2 Several entities recommend strategies to prevent hospital-acquired VTE;3,4 indeed, the US Center for Medicare Services does not compensate hospitals for treating VTEs acquired in hospital or diagnosed in the month after discharge. Yet VTE prevention measures are persistently underused, and their use is substantially lower in medical inpatients than in surgical inpatients.5 Is it surprising that three-quarters of hospital-acquired VTEs occur in medical patients?6

There are several reasons doctors fail to provide prophylaxis. One could simply forget; or hesitate to use an anticoagulant because of a fear of excessive bleeding. Medical patients often have disorders that place them at high risk of bleeding and can require invasive procedures, sometimes at short notice. Although regulatory bodies dictate use of pharmacological prophylaxis in ever-larger populations of medical inpatients, recent trials have shown a very tight balance

Comment

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I declare that I have no conflicts of interest.


