Observational studies have shown a linear relationship between systolic blood pressure (SBP) and cardiovascular disease (CVD) risk rising from 115 mm Hg. Randomised controlled trials have shown that lowering the SBP by approximately 10 mm Hg reduces the risk of stroke by 35-40%, myocardial infarction by 15-25% and heart failure by up to 65%. The target for blood pressure lowering has, however, been uncertain. Previously available data have clearly shown the benefits of lowering SBP to below 150 mm Hg, whilst most hypertension guidelines have recommended lowering SBP to below 140 mm Hg (below 150 mm Hg in patients aged 80 years or above).

The much awaited results of the Systolic Blood Pressure Intervention Trial (SPRINT) have recently been published\(^1\). In this landmark study, 9361 high risk patients without diabetes mellitus or a previous stroke (mean age 68, 65% men), with a SBP of 130 mm Hg or higher, were randomised to intensive (SBP <120 mm Hg) or standard (SBP <140 mm Hg) blood pressure control.

More than 90% in both randomised groups were receiving blood pressure lowering drug treatment at baseline. The mean number of drugs was 1.8 at baseline, increasing to 2.8 in the intensive-treatment group during the study period. Medications for patients in the intensive-treatment group were adjusted on a monthly basis to a SBP target of less than 120 mm Hg. For those in the standard-treatment group, medications were adjusted to a SBP target of 135-139 mm Hg; the dose was reduced if SBP was less than 130 mm Hg at a single visit. Mean SBP at baseline was 140 (SD 16) mm Hg in both groups. Throughout the follow-up this dropped to 121 mm Hg in the intensive-treatment group compared with 135 mm Hg in the standard-treatment group - a 14 mm Hg difference. Importantly, blood pressure was recorded with an automated device, (Model 9070, Omron Healthcare) after five minutes rest in an office free from staff. The mean of three recordings was calculated and used in the analyses.

SPRINT was stopped ahead of time after a median follow-up of 3.3 years, owing to a significantly lower rate of CVD events (primary outcome – a composite of myocardial infarction, acute coronary syndrome, stroke, heart failure or cardiovascular death) and all cause mortality in the intensive-treatment group. CVD events were confirmed in 562 participants – 243 in the intensive-treatment group and 319 in the standard-treatment group. In total, 365 deaths occurred – 155 in the intensive-treatment group and 210 in the standard-treatment group. The relative risk reductions in the primary endpoint and all-cause mortality associated with intensive treatment were 25% (95% CI: 11 to 36) and 27% (95% CI: 10 to 40), respectively. The numbers needed to treat during the study period were 61 for a CVD event and 90 for a death of any cause. Some serious adverse events including hypotension (but not orthostatic hypotension or injurious falls) and acute kidney failure were higher in the intensive-treatment group than in the standard-treatment group, but the rates were low and were clearly outweighed by the morbidity and mortality benefits. It should be underlined that patients with diabetes mellitus or a previous stroke were not included in SPRINT and that we therefore cannot necessarily extrapolate the results to those groups of patients.

However, these SPRINT results make it necessary to reassess SBP goals for high risk patients for whom a lower systolic goal now seems appropriate, which is in keeping with a recent report on the benefit of intensive blood pressure lowering in high risk patients \(^2\). Nevertheless, one major problem is how to translate the SBP values achieved in SPRINT to informal clinical practice around the world given widely variable standards in blood pressure measurement.

The white coat effect on recorded blood pressure levels is variable and may be large and other conditions of measurement also impact greatly on the level recorded. For example, it has been shown that blood pressures recorded whilst strictly following guideline-recommended methods are significantly lower than when measured in usual clinical practice \(^3\). The level of blood pressure recorded is also affected by the presence or absence of clinical staff although some patients are “cuff” rather than just “white coat” responders \(^4\).

So – it seems likely that automated recordings after 5 minutes rest with no medical staff present are likely to be lower than "usual" clinic or office measurements (as frequently practiced) and may equate more closely to home and/or ambulatory blood pressure levels \(^5\). Consequently automated office blood pressure recordings, as performed in SPRINT, may be approximately 10/5 mm Hg lower than those obtained in more usual clinical practice \(^3\).

How then do we interpret the SPRINT findings in the context of less rigorous clinical practice, which is perhaps “the norm”? Ideally clinic blood pressures should be measured as in SPRINT, and if so, the findings appear robust in suggesting we should aim for an SBP of <120 mm Hg. With less rigorous measurement techniques – as most commonly practiced – the SBP values achieved in the intensive treatment arm (121 mm Hg) may well be equivalent to 131 mm Hg!

Proponents of home and/or ambulatory blood pressure monitoring will no doubt argue that these difficulties in interpretation and extrapolation to typical clinical practice would have been largely overcome had home or ambulatory blood pressure recordings been used in SPRINT. Meanwhile, whilst these difficulties are debated, and assuming it is agreed that SBP targets are lowered at least for high risk patients – this has clear implications for lowering treatment thresholds!

Finally, whilst SPRINT undoubtedly challenges us to rethink blood pressure targets, at least in high risk patients, as pointed out at the first presentation in Orlando, don’t expect the patients to thank you when you discuss adding an extra tablet!
REFERENCES

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