Knock Signals in Ischemic Stroke

To the Editor:

I have described a new Transcranial doppler ultrasonography finding “small vessel knock (SVK)” and have proposed that this is an obstructive arterial signal.1,2 SVK is a high-intensity low-velocity signal found at the baseline occurring at aortic valve opening and closure with the signals moving in opposite directions. SVK has a characteristic appearance on single-gated and M-mode spectra and can be distinguished from wall movement, which is symmetrical.3

In the February edition of Stroke, Chung et al4 report the finding of an asymmetrical SVK-like intraluminal signal in vitro, at the site of obstruction, in both silastic and glass tubing. They found that the intensity of this signal increased as the concentration of ultrasound scatterers in the lumen increased. This shows that asymmetrical SVK-like signals are intraluminal obstructive ultrasound signals. Symmetrical “knock signals” were also found because of wall movement, and these increased in intensity toward the site of obstruction. The authors stated that the lumen diameter used in these models (4 mm main and 3 mm branch for silastic tubes, 3 mm main and 2 mm branch for glass tubes) does not represent the expected size of small arterial bifurcations found in vivo. However, SVK-like signals have recently been found in association with vascular occlusion ranging from likely small vessel occlusion up to internal carotid artery occlusion.5

In the same edition of Stroke, Tsivgoulis et al6 reported findings in 327 patients with stroke or TIA who were examined for “knock signals.” No SVK-like signals were identified or found at depths likely to be responsible for MRI abnormalities found in these patients and only symmetrical wall movement knock signals were identified in 14% of patients. The authors did not use DWL–Compumedics TCD machines, which have been used to detect SVK in all previously published cases. A filter of >100 Hz was used, which unfortunately in most cases is too high for SVK detection. This is illustrated in Figure 1, where the SVK signal is visible at a precise depth of 74 mm with a filter of 50 Hz and would be removed if the filter was increased to 150 Hz. The authors also describe diastolic “knock signals.” However, true SVK signals never occur in diastole and unlike emboli and wall movement are found only at one depth.3 The authors also report a lack of association between the “knock” signals they detected and MRI lesions. However, they do not mention that distal regions of all the intracerebral arteries are TCD invisible, and for small vessel disease this will always result in a poor correlation between TCD and MRI. Despite this, asymmetrical SVK signals have been found in stroke patients at appropriate depth and arterial TCD visible areas in association with both MRI positive and negative scans.1–3,5 Finally, the authors quote the Chung et al article but fail to acknowledge that an SVK-like signal was reproduced in vitro at the site of obstruction. They also showed symmetrical knock signals from wall movement in a nonoccluded internal carotid artery but did not show signals from an occluded internal carotid artery. If they had done this they would have found that both symmetrical wall movement and intraluminal SVK-like signals are found in...
obstructed internal carotid arteries\(^5\) (see Figure 2 courtesy of Professor Brian Chambers, NSRI, Melbourne).

Targeting SVK signals can result in clinical recovery including the return of an absent corneal reflex. Video evidence of this has been published.\(^2\) Clinical recovery is associated with changes to the SVK spectra, which include an initial broadening of the signals moving on to minimal flow signals and finally in some cases to open arteries (see Figure 3). The confirmation by Chung et al that the SVK signal is an occlusive waveform is an important step in supporting randomized clinical SVK targeting trials. However, filters should be set as low as possible to allow SVK detection, and because this increases spectral noise the spectral quality of the TCD machine needs to be high enough to distinguish the SVK signal from noise and wall movement at the baseline.

**Disclosures**

None.

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**Figure 2.** Shows a symmetrical signal obtained from the wall of an occluded internal carotid artery and an intraluminal asymmetrical SVK-like signal at the occlusion site.

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Figure 3. a-e, this shows the changes in the SVK signal (3a) during continuous insonation with broadening of the initial SVK signal (3b) to take up most of systole (3c) to a blunted waveform (3d) and finally a dampened waveform (3e). These changes occurred over a 5 minute period and were associated with full clinical recovery.
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Paul Syme

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