A Spectrum of Knock-Type Doppler Signals in the Intracranial Vessels

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Background and Purpose—Knock-type Doppler signals (KTDS) are detectable by transcranial Doppler, and it has been hypothesized that they are related to an occlusion of a small perforating artery and microvascular ischemia. However, the nature of KTDS has not been prospectively defined. We aimed at describing the spectral and power motion Doppler characteristics of KTDS and ultrasound exposure conditions that lead to their appearance.

Methods—Consecutive patients referred with symptoms of stroke or transient ischemic attacks to our cerebrovascular ultrasound laboratory were screened for the presence of KTDS. The presence of microvascular ischemia was assessed using brain MRI.

Results—Among 327 patients with cerebrovascular symptoms, 46 (14%) had KTDS. KTDS were found more frequently in posterior circulation vessels (55% vertebral artery, 21.5% basilar artery, and 6% posterior cerebral artery). There was no association between ultrasound identification of KTDS and the presence of brain ischemia in the distribution of any vessel (OR, 0.37; 95% CI, 0.09–1.53; P=0.171) on univariate logistic regression analyses. KTDS was not related to the presence of microvascular ischemia on brain MRI (OR, 1.12; 95% CI, 0.55–2.29; P=0.761). We described the range of spectral and power motion Doppler appearances of KTDS and experimentally demonstrated the most likely underlying mechanism being a large vessel wall movement artifact.

Conclusions—Although KTDS can be distinguished from other spectral flow signals, they can be found in normal vessels, they do not seem to be associated with the vessel affected by ischemia, and they should not be overinterpreted. (Stroke. 2009;40:644-647.)

Key Words: knock-type Doppler signals ■ small vessel knock ■ transcranial Doppler

Knock-type Doppler signal (KTDS) can be detected by transcranial Doppler (TCD) as transient short-duration signals, typically appearing in systole with a characteristic “knock” sound.1 Syme1 proposed that this is attributable to an occlusion of a small perforating artery and microvascular ischemia; he published intriguing case reports2 of complete clinical recovery with continuous monitoring of the knock signals with TCD. However, the spectrum of knock-type Doppler signal appearance, location, and their nature still remain undetermined. Our study aimed at describing the spectral and power motion Doppler characteristics of KTDS and ultrasound exposure conditions that lead to their appearance. In a cross-sectional study, we also evaluated the potential association of KTDS with the vessel affected by ischemia and the presence of microvascular ischemia on brain MRI.

Subjects and Methods

Neurosonology Evaluation

We evaluated consecutive TCD examinations with the power motion Doppler (PMD) system (PMD 100; Spencer Technologies) in patients with symptoms of stroke or TIA who were referred to our cerebrovascular ultrasound laboratory. PMD-TCD combines a multi-depth PMD display with a single-depth conventional TCD spectral analysis. The 3 TCD windows were used for insonation following a standard protocol.3 PMD TCD provides a simultaneous multi-depth, multi-vessel display of flow direction and intensity.4 PMD serves as a window-finding tool leading to more complete intracranial vessel assessment with spectral Doppler.5 The waveform is displayed using a 3- to 5-sec sweep speed at a specific depth (cm). In addition to power-motion multi-depth display, the spectral waveform, mean flow velocity in cm/sec estimated by mean Doppler frequency,6 and Gosling-King pulsatility indexes were recorded.6 We screened the following vessels for the presence of KTDS: anterior cerebral artery, middle cerebral artery (MCA), terminal internal carotid artery, posterior cerebral artery, vertebral artery, and basilar artery. High-pass filter setting was set at 100 Hz according to the consensus on microembolus detection criteria.7 We analyzed the following characteristics of KTDS: timing during the cardiac cycle when the signal appeared, its intensity (dB), direction, duration (ms), amplitude (cm/sec), rhythm, audibility to a human observer, appearance on the PMD image, and vessel involved. An additional experiment was performed on 5 volunteers using a duplex scanner (Micromaxx; Sonosite) to position the

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Doppler gate on a pulsating wall of extracranial arteries as determined by the gray-scale imaging (common carotid artery, internal carotid artery, and external carotid artery) to replicate insonation conditions that can lead to KTDS appearance. In each vessel, 3 separate spectral Doppler placements were performed.

**Neuroimaging Evaluation**

All patients referred to our laboratory had previously undergone neuroimaging studies including brain CT in all cases and brain MRI according to the discretion of the referring physician (in patients with no evidence of brain ischemia on baseline brain CT and no contraindication for MRI). An attending-level staff neuroradiologist and stroke neurologist (study investigator) evaluated neurodiagnostic imaging studies independently of TCD results. The neuroradiological interpretation in the medical records that standardly notes the vessel affected by ischemia and the presence of microvascular ischemia (lacunar infarction) on brain MRI was considered the official reading for the purposes of the study, because all neuroradiologists were blinded to TCD findings. More specifically, a lacune was defined as a hyperintense signal shown in T2 and fluid attenuation inversion recovery (FLAIR) sequences with sharp margins, <2.5 cm in diameter, located in the deeper structures and irrigated by penetrating branches. The study was conducted as part of a broader Institutional Review Board-approved TCD validation study against other imaging modalities at our neurovascular ultrasound laboratory with waiver of informed consent for participation in the study.

**Statistical Analyses**

The data were expressed as mean, median, range, and standard deviations using descriptive statistics. The prevalence of KTDS between anterior and posterior circulation vessels was compared using $\chi^2$ test. The potential association of KTDS with the vessel affected by ischemia and the presence of microvascular ischemia on brain MRI were evaluated using $\chi^2$ test and logistic regression models. The Statistical Package for Social Science (version 10.0 for Windows; SPSS Inc) was used for statistical analyses.

**Results**

We have reviewed real-time digital PMD-spectral TCD recordings of 327 patients. There were 279 strokes and 48 TIA. KTDS was found in 46 (14%) patients and, among them, 5 patients have KTDS in 2 different intracranial vessels. KTDS were found in the vertebral artery (55%), basilar artery (21.5%), posterior cerebral artery (6%), and MCA (17.5%). KTDS was detected more frequently in with nonflowing fluid. The study was conducted as part of a broader Institutional Review Board-approved TCD validation study against other imaging modalities at our neurovascular ultrasound laboratory with waiver of informed consent for participation in the study.
The characteristics of KTDS are summarized in Figure 1 and Table 1. The frequency of KTDS did not differ between patients with or without brain ischemia in the distribution of vertebral artery (P=0.607), basilar artery (P=0.429), posterior cerebral artery (P=0.457), and MCA (P=1.000; Table 2). A total of 248 patients underwent brain MRI. The frequency of KTDS was similar in patients with stroke (13%) and TIA (19%; P=0.312).

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We also conducted an experiment by placing the spectral Doppler gate over the common carotid artery of 5 healthy volunteers undergoing direct tissue visualization with diagnostic B-mode duplex ultrasound. KTDS were found during early systole with the vessel wall distension in all patients (Figure 2). No KTDS were detected when the Doppler gate was placed either within the patent vessel or over surrounding microvasculature. Furthermore, we observed that the characteristics of KTDS changes with adjustment of the transducer position during routine TCD examination (Figure 2). We repeated the same experiment in both ICA and external carotid artery of the same 5 volunteers and obtained identical results.

### Discussion

Our study showed that KTDS are a relatively common TCD finding occurring mostly in systole, but they could be found in diastole. KTDS could be unidirectional, bidirectional, or alternating. KTDS are always repetitive, nonrandom, and are often heard as a thump, click, or knock. KTDS can be visually recognized as they often are subtracted from the power M-mode display. When they appear high-intensity and bidirectional on spectral display, PMD algorithms recognize this signature as an artifact and eliminate it from the display. Furthermore, increasing the high-pass filter setting may eliminate the KTDS from the spectral display. We did not identify any association between ultrasound detection of KTDS and the location of the vessel affected by ischemia or the presence of microvascular ischemia on brain MRI.

We described the spectra of the knock signals in patients with stroke and TIA, and an independently conducted experimental study by another group9 further explores the likely origins of these signals. From our observations on duplex imaging, it appears that the most likely underlying mechanism of KTDS is related to reflected ultrasound waves from a relatively large-artery wall motion. Chung et al9 also determined that vessel wall pulsation can produce knock signals. It is further supported by the fact that characteristics of KTDS change with transducer repositioning during TCD examination, ie, changing intercept of a proximal intracranial artery. No KTDS were observed when Doppler gate was positioned away from a relatively large artery. Therefore, KTDS is a common artifact that appears when a relatively large blood vessel wall acts as a bright reflector to create high-intensity knock-type signals and, like spectral waveforms,9 it is also affected by the angle of vessel intercept. This is important because knock signals are being attributed to a small vessel ischemia, and therapeutic decisions are advocated based on this finding.1,2

### Table 2. Distribution of KTDS According to the Location of the Vessel Affected by Ischemia

<table>
<thead>
<tr>
<th>Vessel</th>
<th>KTDS (+)</th>
<th>KTDS (-)</th>
<th>KTDS (+)</th>
<th>KTDS (-)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>VA</td>
<td>1/21 (4.8%)</td>
<td>20/21 (95.2%)</td>
<td>27/633 (4.3%)</td>
<td>606/633 (95.7%)</td>
<td>0.607</td>
</tr>
<tr>
<td>BA</td>
<td>1/16 (6.3%)</td>
<td>15/16 (93.8%)</td>
<td>10/311 (3.2%)</td>
<td>301/311 (96.8%)</td>
<td>0.429</td>
</tr>
<tr>
<td>MCA</td>
<td>1/182 (0.5%)</td>
<td>181/182 (99.5%)</td>
<td>8/472 (1.7%)</td>
<td>464/472 (98.3%)</td>
<td>0.457</td>
</tr>
<tr>
<td>PCA</td>
<td>0/9 (0%)</td>
<td>9/9 (100%)</td>
<td>3/645 (0.5%)</td>
<td>642/645 (99.5%)</td>
<td>1.000</td>
</tr>
</tbody>
</table>

*Fisher exact test.

BA indicates basilar artery; PCA, posterior cerebral artery; VA, vertebral artery.
Certain limitations of the present report need to be acknowledged. First, brain MRI was not performed in all patients and therefore selection bias cannot be ruled out. Second, the prevalence of knock signals was not evaluated in the nonpathological cerebral circulation of an age-matched subgroup of control subjects. This issue will be investigated by our group in a future study. Third, the conclusion that the KTDS signal is not related to lacunar stroke may be an overstatement, because this is a retrospective study in which TCD was not performed specifically to evaluate the vessels in proximity to the lacunar infarct in question (e.g., proximal MCA in the setting of basal ganglia lacune or basilar artery in the setting of pontine lacune). A prospective TCD study painstakingly evaluating the vicinity of the MCA or basilar artery at perforator origin would be needed to further clarify this issue. Finally, we performed the experiment using Duplex imaging on healthy volunteers only in extracranial vessels. Further studies are needed to replicate our findings in intracranial vessels using TCD.

In conclusion, KTDS are detectable by TCD in a substantial number of patients with symptoms of stroke and TIA. It is important for physicians to be aware that KTDS are likely ultrasound artifacts that can be found in both posterior and anterior circulation and seem not to be associated with small vessel ischemia. Knock-type signals should not be overinterpreted because data on clinical localization are limited and, given their random and potentially artificial nature, any treatment application should be subjected to a rigorous, prospective, and controlled trial.

I. Adjustment of the transducer position during routine TCD examination

KTDS were found in the left vertebral artery (a) of a patient with dizziness and no ischemic lesion on MRI. The signals became weaker during adjustment of the transducer position to obtain stronger flow signals (b) and KTDS disappeared after the transducer passed the best intercept of the vertebral artery (c).

II. Insonation of the vessel wall of a large artery visualized by B-mode can create knock-type signals with Doppler gate placed at zero degree angle.

KTDS were detected on spectral Doppler during early systole corresponding to the vessel wall distension on B-mode imaging.

Disclosures
None.

References
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