between a small bubble and a large formed center, and this ambiguity has been a drawback of TCD sonography for some time. The challenge is to distinguish not simply between microemboli and artifacts but between different types of emboli by exploiting some signal genuinely characteristic of their composition.

Patrick D. Hill, MSc
Department of Medical Physics and Engineering
The Middlesex Hospital
London, England

References


Response

We thank Dr Hill for his interest in our article.

The initial FFT broad spectrum analyses were performed on the TCD audio outputs digitized at an 8192-Hz sampling rate. According to the Nyquist theorem, a maximum measurable frequency of around 4000 Hz should be possible. The lack of visible frequency features above 500 Hz is more of a scaling problem. The amplitude of the frequencies between 250 and 500 Hz was at least a magnitude or more greater than the amplitude of the 700- to 2000-Hz shifts associated with diastolic/systolic velocities. Ordinate scaling was adjusted to fit the entire amplitude of the lower harmonics onto the graph, resulting in the higher harmonics appearing as a thickened baseline in the graph. Reduction of the graph to publishable dimensions further obscured their presence.

The choice of a 1024-Hz sampling frequency for detection and quantification of the air emboli was based on the attempt to key on the frequencies between 250 and 500 Hz. Therefore, a sampling rate in excess of the Nyquist requirement was deemed unnecessary.

The sampling interval duration was chosen to minimize data loss in the event of prolonged flurries of emboli. The algorithm used in these experiments processed the digitized audio signal immediately after acquisition, storing a single value representing the estimated volume for each triggered event. Processing, however, required a period of time, approximately 20 milliseconds, during which time the Doppler audio signal was not monitored. To minimize errors in the volume estimates during long flurries of air emboli, a 4-second acquisition window was chosen as a compromise between narrower windows that would require shorter processing times and occur more frequently and wider windows requiring longer processing periods that would occur less often.

We agree with Dr Hill that an additional factor that may have contributed to the 10-fold difference between the in vivo and in vitro calibration slopes would be the attenuation of the signal by overlying tissues. Additional studies would certainly be necessary to evaluate the extent and variability of this attenuation before this approach for estimating air bubble volume could be reliably used in the general patient population.

The smallest detectable volume will depend on a variety of factors. Ultimately, instrument power, transducer position, and ultrasound attenuation by tissue and bone will all play a role in determining what is the smallest discernable volume.

We appreciate Dr Hill’s pointing out our obvious error in identifying the correlation between the TCD power and embolus volume in Fig 6 as negative instead of positive.

The basic premise of our work was that the reflected ultrasound signal from air emboli contains a unique signature by which they could be identified. The "hollow metallic chirp" that has been described by some investigators as characteristic of air emboli appears to be due to frequencies within the 250- to 450-Hz envelope. Our experiences with particulate emboli, both in vivo and in vitro, suggest a somewhat different identifying sound. Rarely, if ever, has a "hollow metallic" chirp been associated with particulate emboli in our experience. Higher ultrasound energies, coupled with longer pulse lengths, and higher-resolution spectral analysis may identify harmonics that can be used to discriminate between particulates and air. The methodology presented in this article admittedly requires a great deal of refinement before it can be used reliably in a clinical environment. However, we would like to think that our work represents an approach that might stimulate other ideas and encourage continued investigation in detection and identification of vascular emboli using Doppler technology.

Leonid Bunegin
Department of Anesthesiology
University of Texas Health Science Center
San Antonio, Tex

Reference


Risk Factors for Extracranial Internal Carotid Artery Disease

To the Editor:

Nagao et al1 reported a well-researched and interesting study about changing patterns of carotid artery disease in the Japanese population. In particular, they cite the rising incidence of extracranial internal carotid artery disease. They reviewed the risk factors of sex, age, transient ischemic attack, diabetes mellitus, hypertension, ischemic heart disease, and hypercholesterolemia. Because of the increase in prevalence of diabetes mellitus, they suggest that there is a causal effect with regard to the increased rate of extracranial carotid artery stenosis. The westernization of the Japanese diet is implicated as a likely etiology for the increase in diabetes mellitus.

Unfortunately, there is a glaring omission from this article: smoking as a risk factor was totally ignored. The most important predictor for extracranial carotid artery stenosis in the United States is smoking. Whisnant et al2 in a study using angiography, compared the risk factors of smoking, age, hypertension, diabetes mellitus, gender, and systolic blood pressure. Subjects who smoked for 40 years had a rate of severe extracranial internal carotid artery stenosis 3.5 times higher than that of never-smokers. In another article of particular interest, 800 men of Japanese ancestry living in Honolulu were followed up in a prospective study in which the focus was not on the anatomy of carotid artery disease but on the unequivocally increased rate of hemorrhagic or nonhemorrhagic stroke in smokers. Cessation of smoking in this and another study was accompanied by a decreasing stroke rate. The Framingham study also supports this conclusion: the stroke rate increased with the number of cigarettes smoked per day, and it was reduced to the level seen in nonsmokers by about 5 years after cessation of smoking.

I do not believe that any conclusions about risk factors can be drawn from the report by Nagao et al.1 The changing patterns of carotid disease in the Japanese population should be further investigated with regard to the change in smoking habits.

Jack N. Alpert, MD
Department of Neurology
St Luke's Episcopal Hospital
Houston, Tex

References

1. Nagao N, Sadoshima S, Ibayashi S, Takeya Y, Fujishima M. Increase in extracranial atherosclerotic carotid lesions in patients...
Risk factors for extracranial internal carotid artery disease.

J N Alpert

Stroke. 1994;25:1883-1884
doi: 10.1161/01.STR.25.9.1883

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/25/9/1883.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/