Effect of Emitted Power on Waveform Intensity in Transcranial Doppler

In 1991 your journal published my comments1 on an article by Halsey2 that reported the effects of increased emitted power on the quality of transcranial Doppler (TCD) signal intensity. What could have been an important contribution to solving what is still an unfamiliarity with the basic concepts of acoustic measurement demonstrated in this article makes it clear that this was not the case. Such drugs are likely to act mainly by eliminating heat production. It is possible that these agents may also augment convection by, for example, increasing evaporation. However, it is probable that additional cooling techniques will be required to produce effective temperature reduction in a reasonable length of time.

I think it has already been proved that hypothermia decreases the rate of ischemic tissue damage, but that is not the key issue. Strokes generally occur with little or no warning. The patient must be transported to the hospital, a diagnosis must be made, and then treatment can be initiated. It has been shown that the time required to initiate therapy can be reduced quite remarkably to less than 90 minutes. However, if pharmacologic therapy is then started along with active cooling techniques, there will be a further delay until adequate hypothermia is attained. Most stroke patients are elderly and have concomitant medical problems, most notably cardiac disorders. Thus, treatment should be induced gently to delay until adequate hypothermia is attained. Consequently, the muscular responses to lowered core temperature are abolished, and the central regulatory responses to hypothermia may be blunted or lost. Furthermore, in many experiments the test animals have skin removed and are otherwise manipulated to increase the rate of convective losses.

Menon and Young point out that hypothermia can be induced pharmacologically, and this may be a useful way to treat stroke victims. Such drugs are likely to act mainly by eliminating heat production. It is possible that these agents may also augment convection by, for example, increasing evaporation. However, it is probable that additional cooling techniques will be required to produce effective temperature reduction in a reasonable length of time.

It is stated that the first series of TCD examinations was carried out with a TC2-64 instrument (Eden Medical Electronics, Uberlingen, FRG) "at the standard transducer power of 100 mW/cm²." These are units of intensity, not power. However, the instrument to which they refer has no such "standard transducer power" or intensity. It seems likely that the setting of the intensity control at 100% has been mistakenly assumed to be 100 mW/cm².

The purpose of this control is to allow a percentage reduction of the acoustic output in order to use the lowest intensity required to obtain usable Doppler signals. It does so by regulating the amount of electrical power to the transducer crystal, which converts it to acoustic energy. But this is only one of three variables that determine the acoustic intensity, the others being the pulse repetition frequency (PRF), which is increased with the scale setting, and the configuration of the transducer itself, especially the depth of focus. A setting of acoustic intensity in mW/cm² is therefore not possible but is only an adjustment to a percentage of the maximum at a given combination of these parameters.

Even at the lowest PRF, used to measure flow velocities within the normal range, 100% emitted intensity is of the order of 250 mW/cm² and as high as 550 mW/cm² at the highest PRF, which is necessary to record velocities of up to 400 cm/sec. These are, of course, measurements of spatial peak-temporal average (SPTA) made in water, and the maximum value is reduced to 240 mW/cm² by the application of the Food and Drug Administration's formula originally used for estimating in situ intensity for fetal insonation to the application of the Food and Drug Administration's formula originally used for estimating in situ intensity for fetal insonation and to 51 mW/cm² by using the unit of estimated intracranial intensity (EII) proposed by me as being more realistic for TCD applications.

Throughout the second series of studies made with the Transpact TCD instrument (Medasonics, Freemont, Calif) repeated reference is made to results obtained at an emitted power [sic] of 400 mW/cm². Closer examination of the methodology reveals that this is not the case, but that this is intended to mean an emitted intensity that results in an in situ intensity of 400 mW/cm², although we are not told what this emitted value is. Calculations based on information provided in the instrument handbook show that it would be 676 mW/cm². This means that the limit of 50 J/cm² recommended by the American Institute of Ultrasound in Medicine, which Dr Itoh and his colleagues sensibly sought to observe for reasons of safety by limiting periods of emission to less than 120 seconds, was in fact being exceeded by over 60%

However, as with the instrument used for the first series, this TCD system also provides control of acoustic output only as a percentage of maximum, which is adjustable in the software and displayed on the monitor. This software provides for an "override" of the maximum output, as apparently used by Itoh and colleagues in this study, but the percentage displayed then becomes inoperative, remaining fixed at 100%. The reporting of values in mW/cm² therefore implies a precision which is by no means justified unless the two systems used were calibrated in a water bath. The unfamiliarity with the basic concepts of acoustic measurement demonstrated in this article makes it clear that this was not the case. Such calibrations require expensive equipment and are time consuming, but they are essential if we are to solve the important problems of efficacy and safety of TCD.

Although acoustic intensity obviously plays a role in the ability to obtain good Doppler signals in transcranial examination—just as it does in extracranial examination—this is not such an important factor as was first thought. The compact bone that forms the inner and outer tables of the skull has an attenuation coefficient of 5.6 dB/cm at the frequency of 2 MHz used in TCD instruments, which can therefore theoretically penetrate bone at thicknesses greater than those encountered in the temporal bones even of black subjects.
There are two main reasons that this is not always so in practice. The first is that the skull also has considerable effects on the geometry of the ultrasound beam, varying from a simple shortening of the focal distance (due to refraction and refocusing) to complete disintegration of the sample volume caused by irregularities of the inner bony surface ("split beam"). The first of these may possibly be compensated for by increasing the acoustic output of the instrument, but this will have no beneficial effect in the second case. The second reason is that attenuation in the cancellous issue of the diopole, where this is present, is between 10 and 30 times as great, and which has an even more destructive effect on the beam geometry. Osteoporotic changes in postmenopausal females cause a similar effect, and neither can be rectified by increasing ultrasonic intensity.

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References

Response
It is thought that the transcranial Doppler examination is useful for monitoring real-time changes of blood flow signals and screening for cerebrovascular diseases. Therefore, in our recent study, we checked a success rate for recording Doppler signals from the middle cerebral artery (MCA) in Japanese subjects, and by increasing emitted power we attempted to overcome the problem of obtaining no signals in elderly females.

For the adjustment of the emitted intensity, by learning Halsey’s method from a specialist at Medasonics who was in communication with Prof Halsey, we modulated only the amount of the electrical power to the transducer crystal to obtain the desired emitted power. As described in his reply to the Eden letter, the term "power of N mW/cm²" used throughout our article should be understood to mean "that power that would result in an estimated in situ intensity of N mW/cm²." As pointed out by Dr Eden, we mistakenly calculated the product of time and intensity; for safe recording, the exposure time at an emitted intensity that results in an in situ intensity of 400 mW/cm² should be limited to 71 seconds. In the majority of the subjects examined at higher emitted power, we finished the examination within 60 seconds, and fortunately any subjected to longer exposure time (from 70 to 120 seconds) did not complain of side effects.

From our results, increasing the emitted intensity undoubtedly improved the successful recording rate to some degree; however, as described in Dr Eden’s letter, there are still some problems to overcome for successful recording of MCA flow signals in elderly female patients. I agree that the problem of the recording failure would not be solved by increasing only the ultrasonic intensity. I think that increasing the sensitivity of the transducer as well as the emitted intensity would be necessary to improve the successful recording rate. I hope that a safe and accurate MCA detective instrument for elderly female subjects would be developed in the near future.

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Hyperglycemia in the Acute Phase of Stroke and Stress Response

We read with interest the article published in the August issue of Stroke by van Kooten et al. We have carried out similar work, the results of which have been published recently. We studied the relationship between blood glucose and the stress response in the acute phase of stroke. Plasma catecholamine levels were used as the sole measure of stress response. The authors found that there was significant hyperglycemia in acute stroke and that both hyperglycemia and plasma catecholamines were related to the severity of the acute illness but were not related to each other. The absence of a relationship between catecholamines and blood glucose was interpreted as evidence that glycemia is unrelated to stress response. This may not be true. Our work suggests that there is a significant relationship between plasma glucose levels in the acute phase of stroke and the stress response at that time.

We recruited 68 patients with acute stroke who had fasting blood samples taken within 24 hours of stroke onset. Fasting levels of plasma glucose, serum cortisol, plasma insulin, noradrenaline, glucagon, and hemoglobin A1c were determined. Those with known diabetes mellitus or taking drugs likely to cause hyperglycemia were excluded, as were those with coexisting acute illness that could have caused a spuriously high stress response. Blood sampling was carried out at a fixed time of day, using a standardized procedure. Outcome was assessed as 3-month mortality.

Fasting plasma glucose was higher in those who died (P = .04). Noradrenaline was elevated in this group, but the difference did not reach statistical significance. Using multivariate analysis, with plasma glucose as the dependent variable, we found that serum cortisol (r = .494, P < .001), plasma insulin (r = .475, P < .001), and hemoglobin A1c (r = .40, P < .001) were significantly and independently related to plasma glucose. Noradrenaline levels did not correlate with plasma glucose either univariately or multivariately.

The significant relationship between plasma glucose and serum cortisol suggests a direct relationship between glycemia and stress response. The positive correlation between insulin and plasma glucose suggests insulin resistance, another feature of stress response. The relationship between hemoglobin A1 and plasma glucose is not surprising, as preexisting levels of glycemia are related to current levels of blood glucose.

References
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