

LETTER TO THE EDITOR

To the Editor:

I wish to commend Demierre and coworkers for their excellent presentation "ECG Sources of Gaseous Microemboli," *J.E.C.T.* 17(1):20, 1985. Obviously, microemboli, whatever their origin, are dangerous in the extracorporeal circuit. Avoiding them is basic to a safe, adequate and rewarding perfusion.

Although the authors limited their investigation to the sources of gaseous microemboli, my opinion is that, due to the importance of the problem, the fate of bubbles reaching the patient should have been discussed—particularly, in view of provided graphical information that bubble oxygenators concurrently with associated procedures produce and release into the patient showers of microbubbles (Figures 1 and 2). In these circumstances large quantities of gaseous microemboli are expected to accumulate and to constitute a clinical hazard.

In our practice, however, we have not observed signs of cerebral impairment in patients perfused with bubblers. Moreover, use of bubblers continues to dominate in cardiac surgery in a majority of hospitals. Therefore, if the authors' ultrasonic recordings of microbubbles are not artifacts of noise generated by bubbling, defoaming, pumping, mechanical interfer-

ence, and moving blood particles, then my question is: What happens to the avalanche of bubbles in the patient? In this regard, my belief is that microbubbles passing to the patient—in spite of control techniques—are readily diffused, absorbed and equilibrated in the tissues. Usually, these are oxygen bubbles and unarmful, as long as they are not combined with air and saturated with nitrogen (65%) and carbon dioxide (15%).

As an associate in research in this institution, I studied the rate of absorption of oxygen in the tissues of animals by injecting subcutaneously a large bubble (0.5ml) of 100% O₂ into the thigh muscle in the vicinity of the implanted tissue oxygen micro-electrode¹. Continuous recording of data showed a rapid rise (in 60 seconds) of pO₂ in tissue from a baseline of 25±5 mm Hg to a level of 92±12 mm Hg followed by a slow decline to 29±6 mm Hg, in 10 minutes. Urschel and associates² achieved a 3000% change in the myocardial pO₂ in less than 90 seconds after direct epicardial application of diluted (0.36%) hydrogen peroxide (H₂O₂).

Additionally, we measured simultaneously pO₂ in tissues and blood of anesthetized rabbits while breathing alternately room air and 100% O₂³. The results summarized in Table 1 correlate with previous discus-

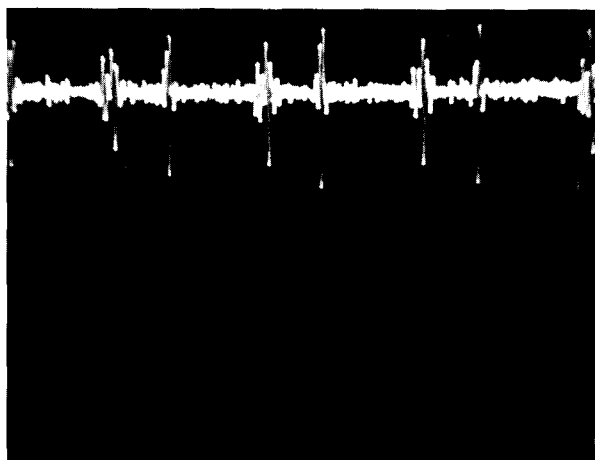


Figure 1: LEFT CAROTID PHONOANGIOGRAM (PCA) Recording of noise due to blood flow and cardiac beating.

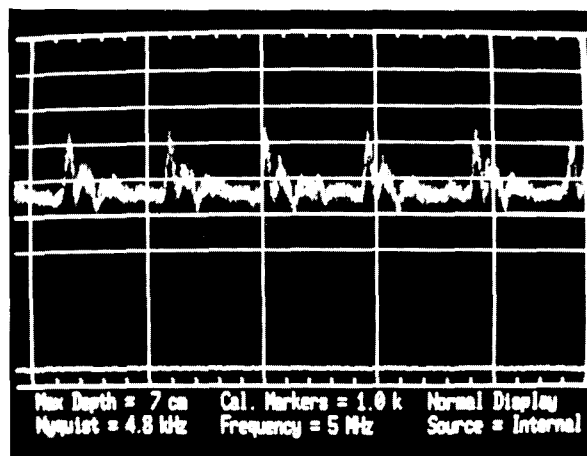


Figure 2: TRACING OF RIGHT CAROTID BLOOD FLOW Recording of noise of circulating blood during systolic and diastolic cycles. (ATL/MK 450PV Ultrasound System)

Direct communications to: Frank C. Cieslak, Ph.D., C.C.P., Columbus Hospital, 2520 N. Lakeview Avenue, Chicago, IL 60614

sion and indicate that oxygen, regardless of its form, source, and concentration is rapidly dispersed and absorbed by the tissues.

On the other hand, if we assume that the authors' ultrasonic recordings reflect other events than moving bubbles, then we have to insist on further research in order to establish clearly and accurately that blood particles moving along as illustrated in Figure 1 and 2, are not recordable.

Sincerely,

Frank C. Cieslak, Ph.D., C.C.P.

From the Department of Surgery, Columbus Hospital, and Northwestern University School of Medicine, Chicago, IL.

Table 1
Blood and Tissue pO₂ Recording in Rabbits
Breathing Room Air and 100% O₂

	ROOM AIR	100% O ₂
ARTERIAL pO ₂	100 = 10 mm Hg	560 = 72 mm Hg
TISSUE pO ₂	30 = 4 mm Hg	79 = 14 mm Hg

References

1. Nora, P.F., Bransfield, J., Cieslak, F. and Laufman, H.: HPO in Clostridial Toxicity and Strangulation Obstruction. *Arch. Surg.* 93:236-244, 1966.
2. Urschel, H.C., Jr., Finney, J.W., Morales, A.R., Balla, G.A., Race, G.J. and Mallams, J.T.: Effects of Hydrogen Peroxide on the Cardiovascular System. *Proc. 3rd Int. Conf. on Hyperbaric Medicine*. Ivan W. Brown, Jr. and Barbara G. Cox, ed., National Academy of Sciences, National Research Council, Washington, D.C. 1966.
3. Bransfield, J.J., O'Brien, P.H. and Nora, P.F.: Observations on Measurements of Oxygen Tissue Tensions in Experimental Animal Tumor Systems. *Proc. 4th Int. Congress on Hyperbaric Medicine*, Juro Wada and Takashi Iwa, ed., Igaku Shoin Ltd, Tokyo, 1970.