Arterial Gas Emboli After Blast Injury[1] (35469)

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(Introduced by U. C. Luft)

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German investigators (1), basing their conclusions on experimental and clinical observations made during World War II, first emphasized the importance of arterial gas emboli in blast injuries to mammals. Subsequent investigations (2, 3) confirmed that arterial gas emboli were common sequelae of blast injury to the chest, at least in animals that died rapidly from severe injuries. Post mortem evidence suggested that the bubbles entered the bloodstream soon after injury from alveolar–venous fistulas in damaged lungs. Arterial gas emboli have been found in an animal autopsied after 80 min of post-blast survival (4) although the time of bubble formation could not be determined precisely. The exact time distribution of bubble generation, as well as the incidence and pathologic significance of arterial bubbles in non-fatal cases, remained obscure.

Methods. Modifications of previously described ultrasonic techniques (5–7) and a 3-mm i.d. cuff around a carotid artery were used to telemeter blood velocity (Doppler) and embolus detector signals as an unanesthetized adult beagle was subjected to LD_{50} air blast in a shock tube (8). The procedure, for which the side-effects of chemical anesthesia would have been undesirable, was ancillary to broad-based investigations of acute and chronic blast pathophysiology. In addition, previous experience has corroborated the findings of earlier investigators who observed that animals and humans exposed to severe blast display a sudden and dramatic decrease in the level of consciousness without any indications of being in pain (1, 9–13).

Results. The time distribution of embolus detector deflections, along with maxima and minima of the blood velocity trace, are shown in Fig. 1 for the time of blast and for the first half hour thereafter. Figure 2 shows traces made before, and 5 and 12 min after the blast. The embolus detector trace of Fig. 2c shows rhythmic bursts of deflections; these bursts correlated in time with the dog's breathing cycle, which was recorded separately. All deflections were accompanied by "chirping" sounds on the audio channel of the telemetry system. The blood velocity trace of Fig. 2c shows a tendency to fall toward zero after each burst of deflections on the embolus detector trace; this decrease may be an indication of reduced velocity due to distal occlusion.

The dog recovered after displaying severe respiratory distress but no gross neurologic defects. Repeat tracings were made during the 5 days after blast and were similar to Fig. 2a, even with the dog coughing (first day) or trotting (fifth day). The dog was sacrificed and subjected to postmortem examination on the fifth day. The lungs showed bilateral residual hemorrhage typical of early resolution after severe blast injury. The brain, heart, kidneys, and other abdominal organs showed no infarction, hemorrhage, edema, or other gross evidence of embolic damage. Confirmation of the presence or absence of microscopic pathology was not carried out.

Discussion. Simulated thrombotic and fat emboli, as well as air bubbles, caused deflections on the embolus detector trace during *in vitro* tests, but the deflections during the dog

[1] This investigation was supported by General Research Support Grant FR-05531-06 from the National Institutes of Health and by contract with the Defense Atomic Support Agency. The research was carried out in accordance with the "Principles of Laboratory Animal Care" as established by the National Society for Medical Research.

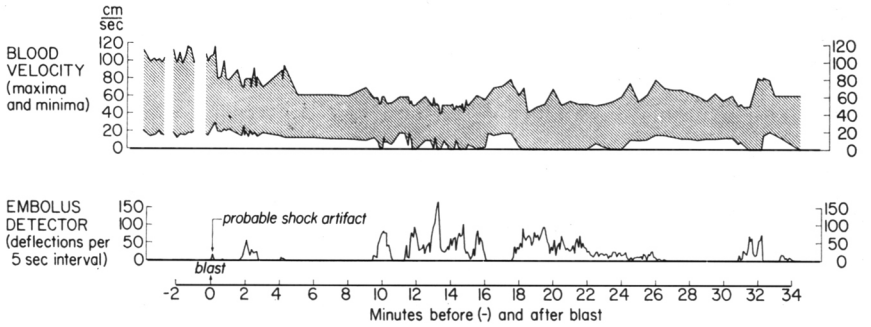

FIG. 1. Timing of embolus detector deflections with maxima and minima of the blood velocity trace. Note the tendency of blood velocities to fall when large numbers of deflections are recorded, with prompt recovery as the deflections decrease or cease, suggesting a transient increase in peripheral resistance.

study were probably caused by air bubbles entering the arterial system from damaged lungs because: (i) arterial gas emboli are common after blast injury to the lungs (1–3); (ii) the "chirping" sounds reportedly characteristic of gas bubbles passing through a Doppler ultrasonic cuff (6–7) were present in the audio presentation of the telemetry signal; (iii) the rhythmic bursts of deflections correlated with breathing; (iv) at autopsy, gross emboli were not found persisting in the vessels downstream from the detector; and (v) autopsy showed no gross evidence of embolic tissue damage, suggesting that the deflections had been caused by short-lived and well-tolerated emboli. The large numbers of separate embolic events recorded from the carotid artery during brief intervals would suggest that the bubbles tended to be quite small.

If the above reasoning is correct, blast-induced arterial gas embolization occurs in

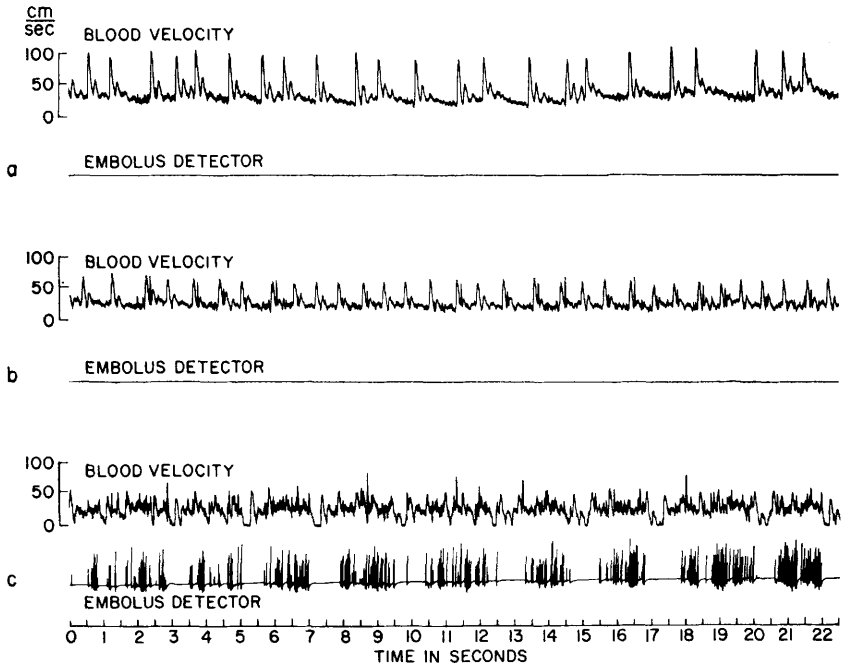

FIG. 2. Blood velocity and embolus detector traces: (a) before blast; (b) 5 min after blast; (c) 12 min after blast.

bursts related to breathing and can continue at least 0.5 hr after injury, even in an animal that later appeared to be well on the way to complete clinical recovery. These observations suggest that: (i) arterial gas emboli are more common and more often "silent" than had been suspected after blast injury, and perhaps after other nonpenetrating chest injuries that result in severely contused lungs; and (ii) enforced rest and a hyperoxic breathing mixture (without intermittent positive pressure) continued for hours after lung contusion would be rational therapy to suppress generation and enhance resorption of arterial gas emboli.

Summary. An implanted ultrasonic cuff has been used to monitor arterial emboli in a dog subjected to near-lethal air blast. Large numbers of emboli, apparently small air bubbles entering the blood from severely contused lungs, passed through a carotid artery at times during 0.5 hr after injury, the period during which such events were monitored during the present study. The dog recovered and showed no gross clinical or pathologic evidence of embolic damage, suggesting that arterial gas emboli are likely after sublethal lung contusion and that appropriate therapy could help to keep the bubbles "silent."

The authors thank Dr. Clayton S. White for suggesting the basic approach used in this experiment.

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Received Sept. 1, 1970. P.S.E.B.M., 1971, Vol. 136.