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ast night the team in the emer-
gency department tried to re-
suscitate a frail 54-yr-old man in cardiac arrest. He had been a passenger in a stationary car that was hit from behind by a van. Return of spontaneous circulation was achieved in the emergency department, but he died a few hours later from pulmonary hemorrhage and a C2/C3 fracture of his ankylosed cervical spine. At postmortem examination he was also found to have multiple bilateral rib fractures and a fractured sternum. The pathologist also noted small hemorrhages, resembling petechiae, in the endocardium of the left ventricle below the aortic valve. A familiar finding, she noted, following both accidental chest trauma and cardiopulmonary resuscitation (CPR). And one that is impossible to distinguish, with confidence, from the petechiae often seen in this site following hypoxia or asphyxia or even the larger subendocardial hemorrhages that occasionally follow hypovolemic or circulatory shock (1).

Were the rib fractures, fractured sternum, and subendocardial hemorrhages the result of the accident, or were they the result of vigorous attempts at cardiopulmonary resuscitation? And therein lies the problem.

In this issue of Critical Care Medicine, Dr. Nishida and colleagues (2) report a series of postmortem examinations in 80 patients who received cardiopulmonary resuscitation, none of whom had any gross evidence of trauma. In seven (9%) of them the authors found evidence of a cardiac conducting system injury. They have attributed this to the CPR. In corroboration they cite 30 postmortems conducted in patients who did not receive CPR at the time of death. No cardiac conducting system injury was found in any of them. However, 73 patients receiving CPR showed no evidence of conducting system injury either, and both groups had mean durations of CPR of >40 mins. And we do not, of course, know the incidence of conducting tissue injury in those who survive CPR.

This interesting study raises many questions. Can it ever be proved, by postmortem examination alone, that such an injury was caused in the way the authors suggest? If there really is such a link, then how important is it clinically? Did the conduction system injury contribute to the inability to resuscitate? Or was it an epiphenomenon? What would have been its significance if return of spontaneous circulation had been achieved? How would such an injury have manifested itself? Would pacing have been required to maintain cardiac output? Are Dr. Nishida and colleagues correct when they imply that conducting tissue injury reflects suboptimal CPR technique? Or could it be possible that occasional conducting tissue injury is an inevitable consequence of effective cardiac compression?

Is there any circumstantial evidence to suggest how significant any conduction system injury might be? In their 2002 study, the Hypothermia After Cardiac Arrest Study Group found a 32% incidence of lethal or long-lasting arrhythmia in their normothermic patients (3). A similar incidence was found in the hypothermia group. One might speculate that some of these could have been caused by a conducting system injury.

How common are chest injuries in patients subjected to cardiopulmonary resuscitation? Hoke and Chamberlain (4) recently reviewed 15 studies of CPR in adults. Rib fractures were reported in 12–96% of cases, whereas sternal fractures were reported in 1–43% of cases. The results from a single center may be more helpful. In their series of 1,823 postmortems of patients who died after CPR, Black et al. (5) found rib fractures in 29% of cases and sternal fractures in 14% of cases.

The incidence of rib or sternum fractures reported by Dr. Nishida and colleagues—one in seven (14%) for the patients with conducting system injury and 14 in 73 (19%) in the others—is at the low end of the spectrum. Thus, it seems

*See also p. 363.

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unlikely that CPR in the series reported in this issue was any more traumatic than usual. Indeed, it has been suggested that in some cases, “serial rib fractures may even be a prerequisite for adequate cardiac output during CPR” (4).

Is there any evidence in the literature to suggest that all the conducting system injuries described by Dr. Nishida and colleagues might have occurred before CPR was started? There are reports of cases of spontaneous coronary artery dissection causing death (6, 7). But there is also a report of coronary artery dissection following blunt cardiac trauma (8). Pathologists take considerable interest in petechial hemorrhages because of their potential forensic significance as a sign (albeit of very low sensitivity and specificity indeed) of possible asphyxia, poring over mucosal, serosal, and endothelial surfaces—but rarely, if ever, searching for them on the cut surfaces of internal organs.

All this is to some extent speculation. What we do know is that the outcome from out-of-hospital cardiac arrest is poor. Survival to hospital discharge in nonnursing home residents of Baltimore in the 1980s was 11% (9), whereas a review of 113 studies suggested a survival rate of 15.2% (10). An earlier review of 39 different emergency medical services found discharge rates ranging from 2–25% for all cardiac rhythms and from 3–33% for ventricular fibrillation (11). One-year survival rates tended to be much lower, for example 5% after an initial resuscitation rate of 44% (10). Three recent studies have confirmed that survival rates for ventricular fibrillation are higher than for other rhythms: 32% (12), 44% (at 6 months) (3), and 20% (to discharge) (13). In the latter study the overall survival to discharge for all out-of-hospital cardiac arrests was 6.2%.

The key to surviving out-of-hospital cardiac arrest remains early defibrillation. The exponential decline in the rate of survival as the time to defibrillation increases is well known (14). The mean duration of CPR in the seven cases showing conduction system injury was nearly 48 mins (range 15–80 mins), so the chances of survival would have been low.

Given the varied nature of the underlying pathology in these patients it is not unreasonable to suggest that the pathogenesis of their conduction system injuries was the physical trauma of CPR. It is reasonable to assume that local hemorrhage into the conducting tissue might have an effect on its function, but to what degree, and for how long? This might have been a concomitant of injury to the conducting fibers, but we cannot know that. Experience with myocardial injury from ischemia indicates that it takes between 6 and 12 hrs for histologic changes to become visible, so we cannot tell whether the red cell extravasation seen by Dr. Nishida and colleagues was accompanied by significant myocyte injury.

This study poses more questions than it answers. Without further information, no practical implications can be drawn from it, except that there may be a link between CPR and conducting system injury. Clinicians looking after patients who have survived a cardiac arrest with more than a few minutes of associated CPR should be alert to the possibility of such an injury—but it will not change their management.

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