GUEST EDITORIAL

Open-chest cardiac massage for non-traumatic cardiac arrest

Two generations of physicians have routinely used closed-chest cardiac compression together with assisted ventilation as the cornerstones of basic and advanced life support following non-traumatic cardiac arrest. One hundred years since the first successful use of closed-chest cardiac massage to resuscitate a human (Osborn, 1987), it is appropriate to reconsider open-chest techniques and their relevance to current practice and guidelines in this country. Following the first successful report in 1898 (Osborn, 1987), open-chest cardiac massage became the standard technique for resuscitation from cardiac arrest until the 1950s, but the patients on whom the technique was employed were almost exclusively those undergoing surgery.

In 1960, Kowenhoven demonstrated that closed chest compression resulted in elevations in blood pressure in fibrillating dogs and that the animals could be subsequently successfully defibrillated (Jude *et al.*, 1964; Kowenhoven *et al.*, 1960). The conclusion that blood was being squeezed out of the heart by direct pressure was true for this animal model, although further investigation in the late 1970s has indicated that, during closed-chest compression and ventilation, cerebral and myocardial perfusion in man is primarily achieved by an increase in intrathoracic pressure rather than by direct myocardial compression. While refinements of techniques of closed-chest compression, together with pharmacological adjuncts and mechanical devices, may further improve blood flow, it is apparent that closed-chest compression is at best a 'holding manoeuvre' to permit electrical defibrillation to be achieved.

During closed-chest compression, cardiac output rarely approaches 25% of normal values (Del Guercio *et al.*, 1963), while myocardial perfusion may be less than 5% of normal values (Ditchley *et al.*, 1982; Nieman *et al.*, 1982). Both cerebral and myocardial blood flow decrease with time during closed-chest techniques (Krause *et al.*, 1986; Jackson & Freeman, 1983) and, as a consequence, survival following cardiac arrest of more than 15 min duration is dramatically reduced and, after 30 min, exceptional (Bedell *et al.*, 1983; Jeresaty *et al.*, 1969; MacKay *et al.*, 1987). Overall, successful resuscitation following closed-chest techniques for cardiac arrest occurs in 4–26% of patients in an accident and emergency setting (Piganol, 1981; Robertson & Little, 1984) while, for hospital in-patients, approximately 15% survive to be discharged (Bedell *et al.*, 1983).

In comparison, two studies have reported on outcome following open-chest cardiac massage for cardiac arrest with recovery rates of 28–58% (Stevenson *et al.*, 1953; Briggs *et al.*, 1956). At first glance, these survival rates contrast dramatically with those for closed-chest compression. It is, however, important to recognize that the underlying disease process contributed significantly to eventual outcome. The majority of the arrests studied occurred during anaesthesia or surgery. Cardiac arrest in these circumstances occurs as a consequence of hypoxia and myocardial irritability under conditions

of sympathetic and vagal overactivity aggravated by the direct effects of anaesthetic agents. This is a markedly different pathophysiological situation from that following acute myocardial ischaemia or infarction.

Nevertheless, there are a number of theoretical and clinical reasons for the potential superiority of open cardiac compression. Preliminary animal studies suggested that during open-chest CPR, carotid blood flow, blood pressure and the ability to achieve successful defibrillation were similar to standard CPR (Redding & Cozine, 1961). However, for the animal model then employed, direct cardiac compression occurred during closed chest compression, thus rendering comparison invalid. Subsequently, cardiac output values 2.5 times greater than that produced by closed techniques have been reported (Weiser *et al.*, 1962), with levels of cerebral perfusion at levels of 63–94% of normal for periods of up to 1 h. During this time, neuronal viability (as assessed by EEG activity and pupillary responses) was apparently unaffected (Byrne *et al.*, 1980; Yashon *et al.*, 1971; Alifimoff *et al.*, 1980). Furthermore, during prolonged resuscitation, cerebral blood flow is maintained at levels of 71% of control (Stadjuhar *et al.*, 1983).

In addition to the two large human series, case reports confirm that survival is possible following prolonged open-chest massage (Russell, 1962; Shocker & Rosenblum, 1967).

With this background, it is surprising that there is only one reported study comparing cardiac output in man during open and closed cardiac massage. In the small number of patients studied, direct cardiac massage more than doubled the closed chest cardiac output (Del Guercio *et al.*, 1965), while mean circulation time fell significantly.

Emergency thoracotomy for penetrating cardiac injury is now a well-described procedure for which clear indications are present (Ivatury & Rohman, 1987). For non-traumatic cardiac arrest, such issues are less clear-cut. A considerable body of evidence exists to show that open-chest cardiac massage is more haemodynamically efficient in providing myocardial and cerebral perfusion than closed techniques. Specific complications such as infection and direct cardiac injury are uncommon (Altermeier & Todd, 1963). The procedure itself and the subsequent aftercare is neither technically demanding, nor expensive. However, it is increasingly apparent that all grades of medical staff have difficulty in performing even conventional closed-chest basic or advanced life support (Skinner *et al.*, 1985; Casey, 1984). To add further confusion by recommending open-chest massage in the routine management of cardiac arrest would, thus, be inappropriate at present. What is required is further research to define those situations where open-chest massage is clearly indicated and to confirm its superiority for these carefully determined events.

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