Augmentation of Artificial Circulation During Cardiopulmonary Resuscitation

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Experiments in dogs with ventricular fibrillation were carried out to determine the effects of epinephrine, plasma volume expansion and manual pressure over the abdomen upon carotid flow and arterial pressure during external cardiac compressions. Epinephrine given intravenously produced a significant increase in systolic arterial pressure but no significant change in carotid flow. Epinephrine given subcutaneously over the sternum in 2 mg. increments had no effect on either systolic pressure or carotid flow. Both intravenously and intra-arterially administered dextran 75 improved carotid flow during external cardiac compressions, both routes being equally effective. Volume expansion, therefore, is indicated during external cardiac compressions even in normovolemic subjects, provided there is no pulmonary edema. Constant manual pressure exerted over the upper abdomen during external cardiac compressions produced a significant increase in carotid flow, but is not recommended because it promoted rupture of the liver.

Cardiopulmonary resuscitation (CPR), intermittent positive pressure ventilation (IPPV) plus external cardiac compression (ECC), has been accepted as the best method to provide artificial circulation in cardiac arrest until adequate spontaneous cardiac action is restored. Carotid flow rates measured in dogs with ventricular fibrillation, and cardiac output measured in patients with proven cardiac arrest during ECC were only about 10–30 per cent of the values obtained during normal spontaneous circulation. Clinical experience indicates that this borderline flow produced by ECC, even under ideal circumstances, limits the chance for complete recovery of vital organ function following prolonged periods of ECC. Besides stressing the importance of the earliest possible restoration of spontaneous circulation (by defibrillation or reversal of asystole), additional means of augmenting blood flow during ECC would be desirable. The following measures were studied: (1) epinephrine administration; (2) blood volume expansion; (3) compression over the abdomen.

Experimental Methods

Twenty-six mongrel dogs, weighing from 8 to 12 kg., were used in these experiments. Anesthesia was induced with intravenous pentobarbital sodium (25 mg./kg.) and a cuffed tracheal tube inserted. During the first 45 minutes the animal was allowed to breathe room air spontaneously while a femoral vein and artery were cannulated for the administration of drugs or fluids. Preparation for the following measurements were made: (1) carotid blood flow by a calibrated differential transformer rotameter; (2) arterial pressure using a Statham P23D transducer; (3) ECG, lead 2; and (4) tidal volumes by a Wright ventilator meter. All transducers were connected to appropriate preamplifiers and parameters 1–3 were recorded continuously on a Grass Model 5 Polygraph.

Forty-five minutes following administration of the anesthetic, ventricular fibrillation was produced by an external electric shock (140 volts, 60 cycles a.c.). Three minutes prior to the production of ventricular fibrillation control measurements were taken. Thirty seconds following ventricular fibrillation ECC was