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EXPERIMENTAL PAPER

Spontaneous gasping produces carotid blood flow during untreated cardiac arrest☆

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Summary
Objectives: Coincident with ''agonal'' gasping during cardiac arrest, there are prominent increases in stroke volumes even in the absence of chest compression. In the present study, we tested the hypothesis that gasps also increase carotid blood flow (CBF) during untreated cardiac arrest.
Materials and methods: The tracheas of nine domestic male pigs, weighing 39 ± 2 kg, were intubated and animals were ventilated mechanically. Ventricular fibrillation (VF) was induced electrically and untreated for 5 min. Coincident with the onset of VF, mechanical ventilation was discontinued. The right femoral artery and vein were cannulated. Intrathoracic pressure (ITP) was measured with the aid of a balloon tipped catheter advanced into the esophagus for a distance of 35 cm. A transonic flowprobe was placed around the right common carotid artery for measurement of CBF.
Results: Gasps increased in frequency during the first 4 min of untreated VF together with increases in CBF. The CBF produced by gasping averaged 220 ± 102 mL/min, which represented approximately 59% of a pre-cardiac arrest CBF. Significant increases in CBF were highly correlated with the decreases in ITP during the inspiratory phase of the gaspings ($r = 0.78$) and with the increases in aortic pressure during the expiratory phase of the gaspings ($r = 0.76$).
Conclusions: Spontaneous gasps produce significant increases in CBF during untreated cardiac arrest. The present study therefore confirmed beneficial effects of gasping during cardiac arrest.

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Introduction

Gasping is a striking ''agonal'' respiratory phenomenon characterized by forceful inspirations, which are often observed in the dramatic conditions of sudden cardiac arrest.[1,2] In both experimental and clinical reports the presence and the frequency of gasping were predictive of the success of resuscitation.[2,3]

Accordingly, gasping is considered an ''autoresuscitative'' phenomenon triggered by hypoxia,[4,5] which persists until respiratory centers located in the caudal portion of the medulla oblongata are disabled.[6,7] In experimental models of induced apnea, gasping appeared after an interval of approximately 2 min, when the arterial oxygen tension (PaO_2) decreased below 5 mmHg. This agonal breathing was able to improve PaO_2 to over 30 mmHg promptly and thereby resuming regular respiration.[5]

Gasping promotes entry of the air into the lungs, securing greater oxygen and CO_2 exchange. In a porcine model of cardiac arrest, spontaneous gasping was able to generate more than 4 L/min of ventilation.[8] Moreover, gasping has been shown to provide another and probably important source of pulmonary gas exchange during CPR.[8–10] More frequent gaspings accounted for greater PaO_2 and lower arterial CO_2 tensions, when precordial compression, combined with oxygen supplied at the port of the tracheal tube, was the only resuscitative intervention.[10]

We have documented previously the hemodynamic effects of gasping during untreated cardiac arrest. The expiratory phase of gasping is associated with increases in arterial pressure and in coronary perfusion pressure (CPP).[1] Nevertheless, earlier reports have demonstrated that gasping improves not only ventilation but generates cardiac output during untreated cardiac arrest.[10,11] Based on the favourable hemodynamic effects which follow each gasp, we therefore previously anticipated that gasping would facilitate increased cerebral perfusion.[1]

In the present study we investigated the effects of gasping on generation of carotid blood flow (CBF). We hypothesized that gasping would be able to produce increases in CBF during untreated cardiac arrest.

Materials and methods

The experiments were performed in an established porcine model of cardiac arrest.[12–14] All animals received humane care in compliance with the ''Principles of Laboratory Animal Care'' formulated by the National Society for Medical Research and the *Guide for the Care and Use of Laboratory Animals* prepared by the Institute of Laboratory Animal Resources and published by the National Institutes of Health (NIH publication 86–32, revised 1985). The protocol was approved by the Institutional Animal Care and Use Committee of the Weil Institute of Critical Care Medicine. The animal laboratories of the Institute are fully accredited by American Association for Accreditation of Laboratory Animal Care (AAALAC) International.

Nine Yorkshire-cross male domestic pigs (*Sus scrofa*) weighing 39 ± 2 kg were fasted overnight except for free access to water. Anesthesia was initiated by intramuscular injection of ketamine (20 mg/kg) and completed by ear vein injection of sodium pentobarbital (30 mg/kg). Additional doses of sodium pentobarbital (8 mg/kg) were injected at intervals of approximately 1 h to maintain anesthesia. A cuffed tracheal tube was advanced into the trachea and animals were mechanically ventilated with a volume-controlled ventilator (Model MA-1, Puritan-Bennett, Carlsbad, CA), with a tidal volume of 15 mL/kg, peak flow of 40 L/min, and FiO_2 of 0.21. End-tidal PCO_2 ($EtPCO_2$) was monitored with an infrared capnometer (Model NPB-75, Nellcor Puritan Bennett Inc., Pleasanton, CA). Respiratory frequency was adjusted to maintain $EtPCO_2$ between 35 and 40 mmHg.

For measurement of mean aortic pressure (MAP), a fluid filled 8-Fr angiographic catheter (model 6523; USCI C.R. Bard, Inc., Salt Lake City, UT) was advanced from the right femoral artery into the thoracic aorta. For the measurements of right atrial pressure (RAP) a 7-Fr balloon tipped catheter (Abbott Critical Care 41216) with an atrial port was advanced from the right femoral vein and flow directed into pulmonary artery. Conventional external pressure transducers were used (Transpac IV, Abbott Critical Care Systems, North Chicago, IL). For measurement of intrathoracic pressure (ITP) a balloon tipped catheter, connected to a pressure transducer, was advanced from the incisor teeth into the esophagus for a distance of 35 cm.[15] CBF was measured continuously with the aid of a flowprobe (Ultrasonic Blood Flow Meter, T101, Transonic Systems Inc., Ithaca, NY) positioned around the right common carotid artery. To induce VF, a 5-Fr pacing catheter (EP Technologies, Inc., Mountain View, CA) was advanced from the surgically exposed right cephalic vein into the right ventricle. The catheter subsequently was advanced into the apex of the right ventricle with the aid of an image intensifier. To measure the scalar