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Do manual chest compressions provide substantial ventilation during prehospital cardiopulmonary resuscitation?

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Abstract

Introduction

Chest compressions have been suggested to provide passive ventilation during cardiopulmonary resuscitation. Measurements of this passive ventilatory mechanism have only been performed upon arrival of out-of-hospital cardiac arrest patients in the emergency department. Lung and thoracic characteristics rapidly change following cardiac arrest, possibly limiting the effectiveness of this mechanism after prolonged resuscitation efforts. Goal of this study was to quantify passive inspiratory tidal volumes generated by manual chest compressions during prehospital cardiopulmonary resuscitation.

Materials and methods

A flowsensor was used during adult out-of-hospital cardiac arrest cases attended by a prehospital medical team. Adult, endotracheally intubated, non-traumatic cardiac arrest patients were eligible for inclusion. Immediately following intubation, the sensor was connected to the endotracheal tube. The passive inspiratory tidal volumes generated by the first thirty manual chest compressions performed following intubation (without simultaneous manual ventilation) were calculated.

Results

10 patients (5 female) were included. Median age was 64 years (IQR 56, 77 years). The median compression frequency was 111 compression per minute (IQR 107, 116 compressions per minute). The median compression depth was 5.6 cm (IQR 5.4 cm, 6.1 cm). The median inspiratory tidal volume generated by manual chest compressions was 20 mL (IQR 13, 28 mL).

Conclusion

Using a flowsensor, passive inspiratory tidal volumes generated by manual chest compressions during prehospital cardiopulmonary resuscitation were quantified. Chest compressions alone appear unable to provide adequate alveolar ventilation during prehospital treatment of cardiac arrest.

Keywords: “cardiopulmonary resuscitation”, “advanced cardiac life support”, “artificial respiration”, “out-of-hospital cardiac arrest”

1. Introduction

Early, high-quality cardiopulmonary resuscitation (CPR) has been shown to improve survival following out-of-hospital cardiac arrest (OHCA).¹ The importance of performing minimally interrupted chest compressions of adequate depth and rate has been established.² The need for providing external ventilations, especially during early resuscitation efforts, has remained debated.^{3,4} Some studies have shown lay rescuer compression-only CPR to be associated with improved outcomes.⁵ Increased willingness to perform compression-only CPR, minimizing interruptions of bloodflow to vital organs and avoiding potential harmful effects of hyperventilation have been proposed as possible reasons.³

Additionally, chest compressions have been suggested to provide passive ventilation.^{6,7}

This mechanism was first reported in 1961 by Peter Safar, who observed that chest compressions generated an average tidal volume of 156 mL in curarised, intubated healthy subjects.⁸ In animal models of cardiac arrest, manual chest compressions have been shown to generate limited alveolar

ventilation.⁹⁻¹¹ In humans, measurements of ventilation generated by manual and mechanical chest compressions have only been performed after arrival of OHCA patients in the emergency department (ED). Mechanical and manual chest compressions were reported to generate limited inspiratory tidal volumes, unable to provide adequate alveolar ventilation.^{12,13} Importantly, measurements took place after prolonged resuscitation efforts, when chest molding, atelectasis and intra-thoracic airway closure could have limited the effectiveness of this mechanism.^{6,7} In a swine model of cardiac arrest, tidal volumes generated by mechanical chest compressions were shown to gradually decline, failing to provide meaningful ventilation after 10 minutes of CPR following 6 minutes of untreated ventricular fibrillation.¹¹

Goal of this study was to quantify inspiratory tidal volumes generated by manual chest compressions, performed according to current guidelines, during prehospital CPR.

2. Material and methods

2.1 Methods

This was a single-center, prospective observational study. A convenience sample of adult, endotracheally intubated, non-traumatic OHCA patients, treated by the prehospital medical team of Ghent University Hospital (tertiary referral hospital, annual census of 45 000 patients in the emergency department) was recruited. This study was approved by the Ethical Committee of Ghent University Hospital (B67020083371). Deferred informed consent was obtained.

The prehospital medical team consisted of an emergency physician, nurse and emergency medical technician. Patients were treated according to 2015 European Resuscitation Council guidelines.¹⁴ Chest compressions and ventilations were performed manually, by different members of the team. Immediately following endotracheal intubation, a flow sensor was connected to the endotracheal tube. Realtime CPR feedback was available.

2.2 Materials

A SFM3200 (Sensirion, Swiss) mass flow sensor was used to measure bidirectional airflow (update time: 8ms, accuracy: 2.5% of the measured value) through the endotracheal tube (Hi-Contour, Covidien, Ireland). A low resistance, single use antimicrobial filter (Dar Adult-Pediatric Electrostatic Filter HME, Covidien, Ireland) separated the endotracheal tube from the sensor. The inlet of the sensor was connected directly to a manual resuscitator (Spur II Adult, Ambu, Denmark), filling with 100% oxygen at a rate of 15 L/min.

To control the sensor, a battery powered microcomputer was used, enabling continuous registration of airflow through the sensor and wireless transfer of this data to a remote server. All data received on this server was automatically analysed in order to identify when the sensor had been used. This process eliminated the need for users to manually activate the sensor.

A Zoll X-series defibrillator (Zoll Medical Company, USA) was used to monitor the patient and to provide realtime CPR feedback.

2.3 Data analysis

Flow data was visualised using DataGraph (Visual Data Tools, USA). Changes in airflow due to manual ventilations and chest compressions were manually identified (Figure 1). In each patient, the first thirty chest compressions performed after connection of the flowsensor to the endotracheal tube (without simultaneous manual ventilation), were analysed. Inspiratory airflow generated by each chest compression was integrated using a custom script, producing the generated inspiratory tidal volume. RescueNet Code Review (Zoll Medical Company, USA) was used to gather and review chest compression quality data. Patient demographic and treatment data were collected from the hospital electronic medical records system. Results are reported as median and interquartile range (IQR).

3. Results

The sensor was available for a total period of 11 months. During this time the prehospital medical team responded to 104 cases of cardiac arrest. Endotracheal intubation was performed in 94 cases.

The sensor was used in 10 cases, data from all 10 cases was analysed.

The median age was 64 years (IQR 56, 77 years), 5 patients were female. In 7 cases the cardiac arrest was witnessed, bystander CPR was performed in 4 cases. The median response time of the prehospital medical team (time between the team being alerted and arrival on scene) was 9 minutes (IQR 8, 10 minutes). Initial rhythm was asystole in 5 patients, pulseless electrical activity in 3 patients, ventricular fibrillation in 1 patient and pulseless ventricular tachycardia in 1 patient. Return of spontaneous circulation was achieved in 5 patients.

The median compression frequency was 111 compression per minute (IQR 107, 116 compressions per minute). The median compression depth was 5.6 cm (IQR 5.4 cm, 6.1 cm). The median duration of CPR before measurements were started, was 14 minutes (IQR 13, 16 minutes). The median inspiratory tidal volume generated by manual chest compressions was 20 mL (IQR 13, 28 mL).

4. Discussion

In this study, inspiratory tidal volumes generated by manual chest compressions were measured during prehospital treatment of OHCA. In previous studies, evaluation of this passive ventilatory mechanism took place only after admission of the patient to the ED, following more prolonged resuscitation efforts.^{12,13} Authors suggested failure to detect clinically relevant passive ventilation in these ED based studies could be due to changes in thoracic and lung characteristics following ongoing resuscitation efforts, limiting the amount of passive ventilation generated by chest compressions.^{6,7}

Our results show manual chest compressions, performed according to current guidelines, to be unable to provide adequate alveolar ventilation during prehospital treatment of OHCA. Calculated inspiratory tidal volumes were consistently smaller than average adult anatomical dead space (150 mL). Recently, Chang et al. reported an association between ventilation in >50% of pauses in chest compressions during prehospital treatment and return of spontaneous circulation and survival.¹⁵ At the time of prehospital medical personnel arriving, any oxygen stored in the body is likely to be consumed, whilst our study shows chest compressions alone are unable to provide adequate ventilation.

Tidal volumes reported in our study are larger than tidal volumes previously reported for manual chest compressions performed in the emergency department (7.5mL, IQR 3.5 – 12.6 mL), and smaller than tidal volumes reported for mechanical chest compressions in the emergency department (41.5 mL, range 33.0—62.1 mL).^{12,13} Further research is needed to analyse potential associations between patient and chest compression characteristics and the amount of passive ventilation generated.

The amount of passive ventilation generated by manual chest compressions during the first minutes following cardiac arrest, when bystander CPR is performed, remains uncertain. Measurements were started after a considerable amount of time had already passed, following a median period of 14 minutes of CPR. At this time, thoracic and lung characteristics could have already changed substantially. In a laboratory swine model of cardiac arrest, chest compressions generated adequate tidal volumes in the first minutes following cardiac arrest but failed to provide ventilation after 16 minutes of cardiac arrest.¹¹ Performing measurements during the initial moments following cardiac arrest, remains logistically challenging.

Other limitations existed in this study. Care was taken to use components which generated as little resistance as possible. Still our research setup might have generated additional airway resistance. Adjusting measured flow for the resistance generated, would have required adding pressure sensors to the device, increasing its size and limiting ease of use in the prehospital setting. A small convenience sample of patients was included. In a large number of cases, despite being available, the sensor was not used. Forgetting to use the device was the major reason reported by members of the prehospital medical team.

This study is the first to quantify inspiratory tidal volumes generated by manual chest compressions during prehospital CPR. Ongoing use of the sensor deployed in this study, will allow further research of ventilation during cardiopulmonary resuscitation to be performed.

5. Conclusion

Using a flowsensor, passive inspiratory tidal volumes generated by manual chest compressions during prehospital CPR, were quantified. Manual chest compressions alone appear unable to provide adequate

alveolar ventilation during prehospital treatment of OHCA.

Declarations of interest: none

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Legends to figures

Figure 1. Visualised flow data

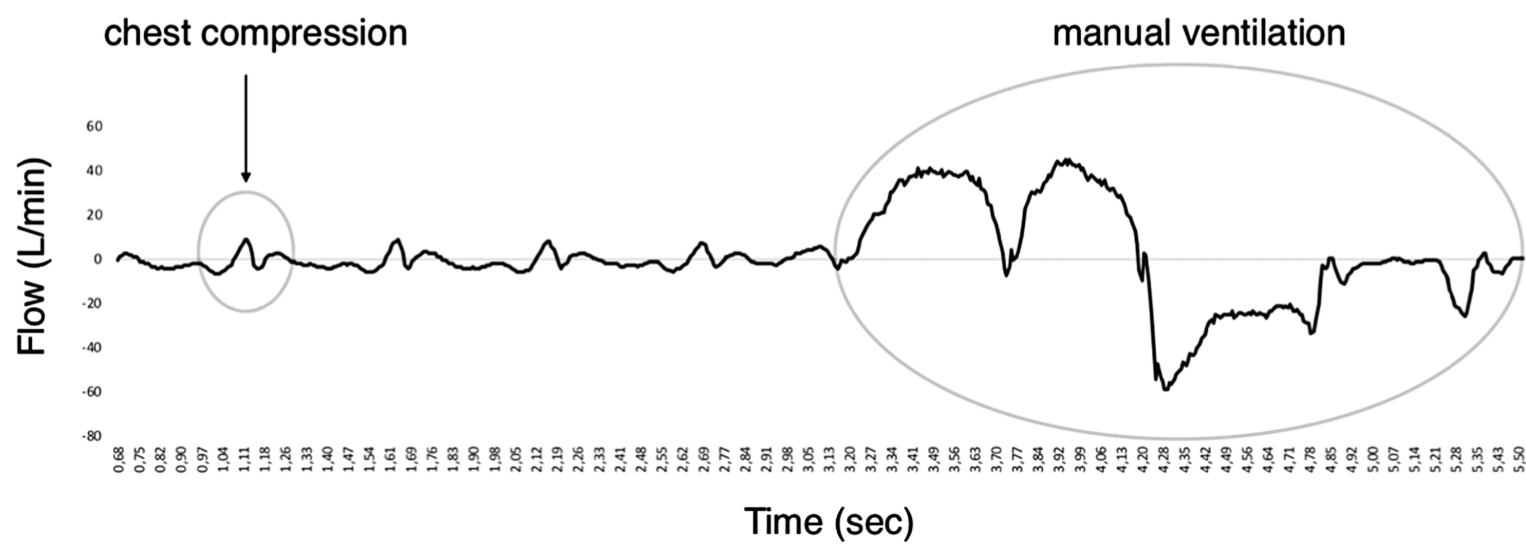


Figure 1