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Chest compressions during ventilation in out-of-hospital cardiopulmonary resuscitation cause fragmentation of the airflow

1. Introduction

Out-of-hospital cardiac arrests (OHCA) are a leading cause of death in the developed world and survival rates from OHCA vary greatly among studies and regions [1,2]. The quality of the cardiopulmonary resuscitation (CPR) is an important factor in improving survival rates of OHCA patients [3]. Two main aspects in the CPR sequence are ventilation and chest compressions, which are often provided simultaneously when the orotracheal tube is in situ [4]. While the need for continuous, uninterrupted chest compressions has been established [3], numerous studies are questioning the need for external ventilation during CPR. Notwithstanding all major guidelines are recommending alternating chest compressions and ventilation at a 30:2 ratio, the use of continuous chest compressions only as standard BLS procedure in adults has been recommended by multiple studies, based on a better willingness to start CPR by bystanders, the low quality of mouth-to-mouth ventilation and too long interruptions of chest compressions during ventilation [5–8].

Chest compressions have been suggested to provide passive ventilation. Animal research has shown that there can exist a sufficient amount of passive ventilation generated by chest compressions, in particular during BLS [9]. Whether this can be translated to human resuscitation, remains questionable. Numerous studies have been conducted regarding this matter, with divergent results [10–14]. While most of these studies were conducted in the emergency department, a recent study by Vanwulpen et al. quantified the chest compression-generated ventilation in an out-of-hospital setting using a flow sensor, concluding that the generated tidal volumes were not sufficient enough for adequate alveolar ventilation [15]. A study by Duchatelet et al. observed the phenomenon of so-called “reversed airflow” (RF), i.e. chest compressions causing an expulsion of air out of the lungs during the inspiratory phase of the ventilation in patients receiving prehospital CPR. Secondary, alleged fragmentation of the airflow, caused by chest compressions, was observed.

No significant conclusions could be formulated regarding the RF and alleged fragmentation due to small sample size and the cumbersome and possibly inaccurate way of measuring the airflow [16]. Both studies of Vanwulpen et al. and Duchatelet et al. are part of a project aiming to gather different parameters regarding the ventilation during resuscitation of OHCA patients, using a custom-built flow sensor. This study is also part of the same project and focusses on the occurrence, quantification and importance of the fragmentation of the airflow caused by manual chest compressions during manual ventilation, performed according to current guidelines, during prehospital CPR.

1 2. Materials and Methods

2 2.1. Methods

3 This is a single-center, prospective observational study, held at Ghent University Hospital (tertiary
4 referral hospital). The emergency department takes care of more than 40.000 patients and deals with
5 over 100 OHCA at yearly basis. All included patients were older than 18 years of age and were
6 orotracheally intubated and resuscitated in an out-of-hospital setting by a prehospital Emergency
7 medical team (EMT) of Ghent University Hospital. The prehospital medical team consisted of an
8 emergency physician, a nurse and an emergency medical technician. All patients were treated
9 according to the 2015 European Resuscitation Council guidelines [4] and received simultaneous
10 manual ventilation (Bag Valve Mask) and manual chest compressions performed by members of the
11 prehospital medical team. Permission of the Ethical Committee of Ghent University Hospital was
12 obtained (B67020083371) as well as deferred informed consent.

13 Patients were excluded from analysis if no flow measurements were available or if there was failed or
14 invalid registration (cf. infra). Other exclusion criteria were technical malfunction or solely
15 spontaneous ventilation. A registration was approved valid when a clear curve could be identified on
16 the registered ventilation. When a registration failed, the flow graph showed a flatline during
17 ventilation or a curve with no apparent ventilation peaks.

18 2.2. Materials

19 A custom-built bidirectional flow sensor was used to quantify the volumes of ventilation. Immediately
20 following the intubation, the sensor was connected to the endotracheal tube (Hi-Contour, Covidien,
21 Ireland) using a low resistance, single use antimicrobial filter (Dar AdultPediatric Electrostatic Filter
22 HME, Covidien, Ireland). The inlet of the sensor was connected directly to a single use Bag Valve Mask
23 resuscitator (Spur II Adult, Ambu, Denmark), filling with 100% oxygen at a rate of 15 L/min.

24 The flow sensor consisted of a SFM3000 (Sensirion AG, Swiss) mass flow sensor (flow range: +/- 200
25 slm, accuracy: 1.5% of the measured value, update rate: 2 kHz (0.5ms)) connected via a USB 2.0 cable
26 to a microcomputer (Intel Compute stick, Intel Corporation, Santa Clare, USA) running Windows 10
27 (Microsoft Corporation, Redmond, USA), powered by a 26,000 mAh portable battery (Intocircuit
28 Powercastle). The default sampling time of 0.5 ms (i.e. 2 kHz) was adjusted to a sampling time of 20
29 ms (i.e. 50 Hz) to reduce the size of the log files. This reduction had no significant impact on the
30 accuracy of the data. The microcomputer had a wireless connection with the hospital Wi-Fi network,
31 and could remotely be monitored and controlled via a TeamViewer (TeamViewer GmbH, Germany)
32 connection. The data from the sensor was collected continually and was manually saved daily using
33 the TeamViewer connection.

34 Chest compression data (depth, quality, frequency) and ECG was measured using defibrillator pads
35 (CPR stat-padz multifunction, Zoll Medical Company, USA) and an accelerometer (Zoll Medical
36 Company, USA) – placed on the patient's sternum – connected to a Zoll X-Series defibrillator (Zoll
37 Medical Company, USA). Data from the Zoll defibrillator was collected every week using a USB-stick
38 and decompiled using Zoll Data Retriever and RescueNet Code Review software (Zoll Medical
39 Company, USA).

1 Patient demographic and treatment data (i.e. age, sex, circumstances, initial rhythm, duration of CPR,
2 ROSC) were collected from the hospital electronic medical records system.

3 All collected data was stored in a private database. At the end of the data collection phase, the
4 database was converted to a database in SPSS Statistics 25 (IBM Corporation, Endicott, USA) to
5 facilitate data analysis.

6 2.3. Data analysis

7 Python based software (Python Software Foundation, Delaware, USA) was custom developed by
8 engineering students at Ghent University to visualize the data recorded by the flow sensor as well as
9 the chest compression data. A Plotly plugin (Plotly, Montreal, Canada) inside the Python environment
10 was used to visualize both types of data. Synchronization was done visually, guided by timestamps
11 included in both types of data.

12 For every patient included in the study, the first minute of ventilation with simultaneous chest
13 compressions was analyzed. After visualizing the flow file and the compression information in graphs
14 using the Python based software, the first complete minute of the registration that was of sufficient
15 quality to allow an analysis, and was supported by continuous chest compressions, was isolated. Solely
16 for the purpose of a visual comparison between ventilation with and without continuous chest
17 compressions, in one patient, one extra minute of ventilation was analyzed. This minute was isolated
18 where only ventilation could be visualized after return of spontaneous circulation (ROSC). In this
19 minute no chest compressions were given. This minute after ROSC was graphically visualized using the
20 Python based software next to a minute with before ROSC, therefore with continuous chest
21 compressions (Fig. 1.1 and Fig 1.2). The respectively absence and presence of fragmentation of the
22 flow is clearly distinct.

23 The beginning and ending points of every inspiration during the minute of registered ventilation were
24 manually identified, based on the visualization of the flow in Python (Fig. 2). Subsequently the volume
25 and duration of each inspiration were calculated, as well as the number of fragments each inspiration
26 was divided into. To quantify the volumes of each inspiration, the volume of each fragment of that
27 inspiration was calculated using an integral function to calculate the area under the curve (AUC). The
28 total number of episodes of RF during the analyzed minute of ventilation were also noted.

29 All data in this study is reported as mean (standard deviation; range).

1 3. Results

2 During the study period, data from the flow sensor was obtained in 17 cases, of which 10 cases could
3 be included in the study. 5 cases were excluded due to an invalid registration by the flow sensor, 2
4 cases were excluded because the flow sensor was only connected after ROSC was achieved (therefore
5 no chest compressions were given during the registered period).

6 All 10 patients were intubated in a prehospital setting and had a presumed cardiac cause as the
7 underlying reason for cardiac arrest. The mean age was 71 years (14;51-87), 7 patients were male.
8 Initial rhythm was asystole in 6 patients, pulseless electrical activity in 3 patients and ventricular
9 fibrillation in 1 patient. ROSC was achieved in 4 patients, of which 2 could be discharged from the
10 hospital at a later stage.

11 The mean ventilation rate was 12/min (2;9-16). The measured mean minute volumes and tidal
12 volumes (TV) were respectively 6.21 L (1.51;3.79-8.15) and 514 mL (99;422-682).

13 Fragmentation of the airflow was observed in all patients, with an average of 3 fragments per
14 inspiration (1;2-5) and a calculated mean volume of 214 mL per fragment (65;112-341). Reversed
15 airflow was observed in 6 patients, with a mean of 4 episodes per minute (5;0-16).

16 4. Discussion

17 In this pilot study, a custom-built flow sensor was used to quantify the fragmentation of the airflow
18 caused by chest compressions during ventilation in out-of-hospital cardiopulmonary resuscitation. The
19 objective of our study was to analyze this fragmentation and the potential impact on the volumes of
20 air passing through the endotracheal tube. While this phenomenon of fragmentation had been briefly
21 observed in a study by Duchatelet et al., no further data could be provided since this was not the focus
22 of the study [16].

23 To our best knowledge, our study is the first focusing on the fragmentation of the airflow. Our
24 objective was to visualize and quantify this fragmentation. While Duchatelet et al. calculated the
25 ventilation volumes by measuring the pressure gradient over the ETT, we calculated the volumes
26 directly measuring the airflow using a flow sensor. The method using the pressure gradient was very
27 cumbersome and was of unclear accuracy, our flow sensor was more straightforward and easier to
28 use by our EMT.

29 Our results show that in OHCA patients undergoing manual ventilation, chest compressions generated
30 a fragmentation of the flow in all cases. Secondly, one or more episodes of RF were observed in most
31 of the patients.

32 When we look at the ventilation rate in our study, our patients were ventilated with 12 breaths per
33 minute on average, no patient was hyperventilated [17]. Duchatelet et al. observed a mean ventilation
34 frequency of 18 ventilations per minute. The mean TV measured by our flow sensor (514 mL (99;422-
35 682)) comply with the recommended TV of 500 – 600 mL [17]. Mean TV in the study of Duchatelet et
36 al. were more than 600 mL in 75% of their patients, while in our study only 20% had mean TV higher
37 than 600 mL. Similar to our study, all patients in the study of Duchatelet et al. were manually
38 ventilated. Hyperventilation, mostly through high respiratory rates rather than excessive TV, is a
39 common thread in most of the studies regarding ventilation during out-of-hospital CPR. The
40 persistently high airway pressures created by hyperventilation are likely to have a detrimental effect

1 on blood flow during CPR [18]. In hyperventilated patients, fragmentation of the ventilation flow may
2 compromise gas exchange: when the fragments become smaller than the dead space, this might lead
3 to carbon dioxide retention. For example, when a ventilation of 500 mL is split into five equal airflow
4 fragments, then each fragment should have a volume of 100 mL. If the RF episodes also have a volume
5 of around 100 ml, then only dead space ventilation would occur [16]. A larger patient population could
6 possibly show differences in fragmentation between patients who were either hypoventilated or
7 hyperventilated.

8 Although TV in this study may seem adequate, they were calculated as the sum of the individual
9 inspiratory airflow fragments and do possibly not accurately reflect true TV. We are currently working
10 on an updated version of the program, which could possibly be used not only to visualize the airflow,
11 but also to calculate the sum of the volumes, which would make the analysis of the data easier and
12 possibly more accurate.

13 The major limitation in this project is the small patient population. This study was a pilot study, using
14 a custom-made flow sensor. The flow sensor had several parts which were vulnerable, resulting in
15 infectivity or lack of analysis unabling us to use it and this sometimes for multiple days. Additionally,
16 the EMT personnel was not carrying the device all time because of the scarce number of resuscitated
17 patients. An OHCA can be a very stressful occasion, even for trained rescuers, therefore the sensor
18 was not a priority and was overlooked regularly. In addition to the small number of patients included,
19 the data may not be representative for all patients.

20 A follow-up to this study, conducted in a larger patient population, might help to determine the effect
21 of fragmentation of the airflow during resuscitation on the efficacy of the ventilation and on clinical
22 outcome.

23 5. Conclusion

24 Chest compressions have a significant effect on the ventilation flow during CPR, as chest compressions
25 during ventilation induce a fragmentation of the airflow. While fragmentation was observed in all
26 patients, there was wide variation in the number and volume of the fragments between patients. This
27 fragmentation may result in incorrect measured values of the delivered tidal volumes. The
28 quantification of airflow volumes using a flow sensor and the importance of the fragmentation of the
29 airflow caused by chest compressions can be essential in improving cardiopulmonary resuscitation
30 techniques. The effect of this fragmentation on the efficacy of ventilation during resuscitation and on
31 clinical outcome remains unknown and needs further investigation.

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43 Appendix

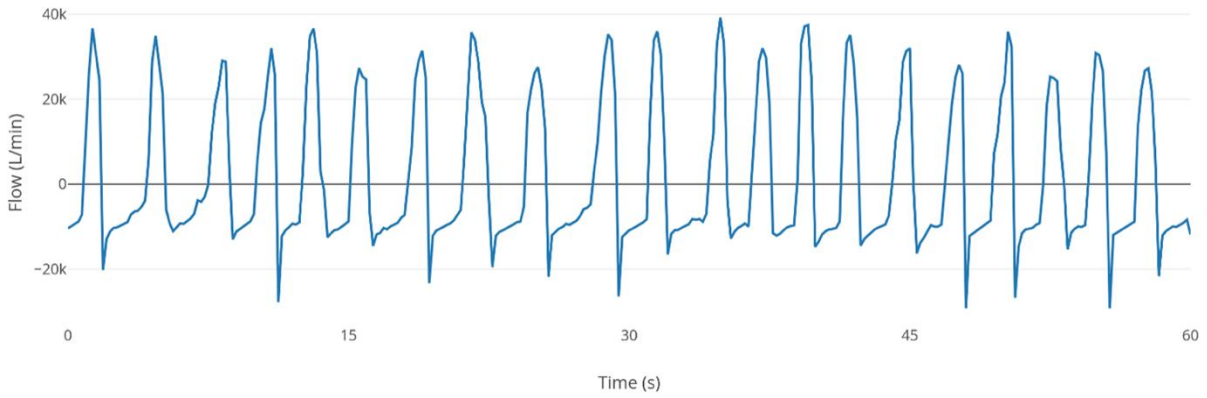
44 Figures

45 **Figure 1.1** One minute of ventilation without chest compressions in patient who achieved ROSC
46 following an OHCA

1 **Figure 1.2** One minute of ventilation before ROSC with simultaneous chest compressions on the same
2 patient

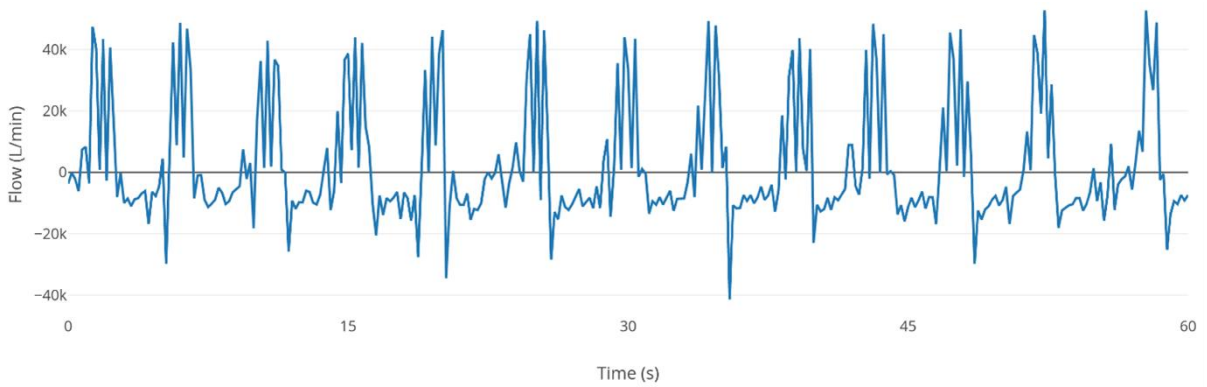
3 **Figure 2** Fragmentation of the inspiratory flow with periods of Reversed Airflow

4



5

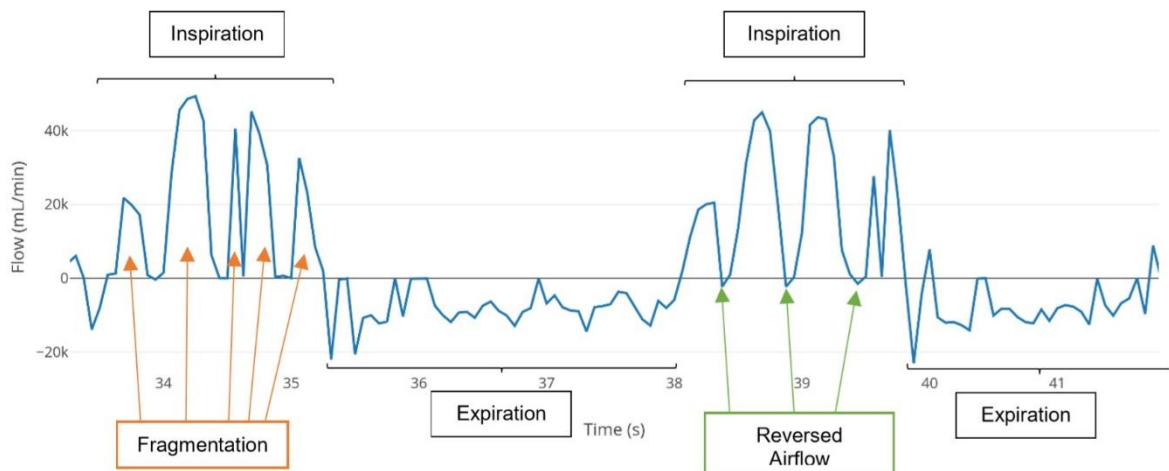
6 **Figure 1.1**



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8

9 **Figure 1.2**



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Figure 2

