

artificial ventilation and when to administer drugs to suppress spontaneous ventilation in case of apparently normal SpO₂ during gasping. Gasping is a well-known phenomenon occurring after cardiac arrest. It is reported in 33 to 60% of all OHCA patients and is common during the first minute in patients with ventricular fibrillation [1–3]. There is ample literature on the positive relationship between the presence of gasping and the probability of successful survival without neurological sequelae; on the other hand, there is remarkably little literature defining gasping and its difference from eupnoea. In the available literature, the main difference is described as a change in breathing pattern: very rapid inspiration followed by prolonged expiration. Ramirez and Lieske in their publication report the absence of expiratory activity and lack of involvement of expiratory muscles in breathing pattern as another characteristic feature of gasping [4]. The transition between normal respiration and gasping may not always be clear; pre-gasping activity – the transition between eupnoea and gasping similar to our case except of high frequency has also been described [4]. There are large differences concerning effective volume of gas exchange during gasping. Wolfskeil measured tidal volumes achieved by gasping and the average volume was about 350 ml with a wide range of 52–1280 ml [5]. Finally, scientific information regarding the frequency of gasping is completely missing. Only vague data can be found on the mostly slower frequency of agonal breathing compared to normal breathing, which slows down further over time until it spontaneously disappears or reappears when chest compressions are initiated.

In this case report, an unusual rapid frequency of gasping (24 breaths per minute) was observed. In our patient, gasping with unobstructed airways during CPR had an unquestionable positive effect on maintaining adequate oxygenation (SpO₂ was 100% with oxygen flow 8 l/min) even without artificial pulmonary ventilation (APV). The duration of gasping was prolonged to a full 12 minutes during CPR until ROSC, partly also because of good quality of chest compressions, and probably even several minutes after ROSC. It confirms the previous report that transition between normal respiration and gasping may not always be clear [4]. The different pressure ratios in the chest during spontaneous gasping and artificial ventilation with intermittent pressure with intermittent pressure should also be taken into account. The literature comparing, for example, active compression-decompression resuscitation and impedance threshold device for out-of-hospital cardiac arrest does not give clear results either [2]. Thus, our decision was based primarily on the clinical condition of the patient. Because of apparently good effect of gasping on oxygenation we decided not to start 30:2 compression-to-ventilation ratio recommended in ERC guidelines. The reason for this decision were published data mainly from animal experiments, that compared to APV gasping has several beneficial effects on circulation and brain functions. Gasping produces negative intrathoracic pressure and generates a pressure gradient between right heart and large vessels that promotes venous return. Minor increasing of intrathoracic pressure during expiratory phase potentiates aortic pressure. Gasping-induced changes in circulation improves coronary perfusion, but also brain circulation. Similar changes can be observed in humans using active compression-active decompression device [6].

Moreover, strenuous gasping would interfere with APV and general anaesthesia would be necessary.

The question remains as to whether gasping can be used in some cases as the only ventilation during chest compression without following the standard 30:2 compression-to-ventilation ratio recommendation (so-called “hands only CPR”). We found only three similar reports. Bunya et al. described a successful resuscitation of a man with asystole but with preserved breathing (no other data concerning ventilation are available) [7]. In their second publication, they mentioned a transport of patients after OHCA with continuous chest compression and gasping only to a hospital with good survival rate, but again without more detailed information concerning agonal breathing. [8] An extreme duration of gasping – 67 minutes – in a patient with ventricular fibrillation was reported by Okamoto et al. [9]. Gasping was preserved all the time during transport, repeated defibrillation and connecting to ECMO. Patient survived. The second patient of Okamoto et al. was also treated by ECMO and had preserved gasping, but no information concerning its duration was described. Here we come to the next question, namely whether we should perform early advanced airway management (e.g. tracheal intubation and artificial lung ventilation) or prefer gasping during extended CPR in patients with gasping. The differences in available literature and lack of sufficient data does not allow us to give a clear answer. On the one hand, there are data from animal studies of beneficial effect of gasping on brain circulation and cardiac output compared to APV [6]. On the other hand, artificial pulmonary ventilation eliminates the patient's breathing effort and may reduce lactic acidosis in tissues. The results of the acid-base balance examination indicated that the body made a maximal effort to compensate for metabolic acidosis by hyperventilation, suggesting that the patient had a preserved respiratory response to the low pH even during cardiac arrest and resuscitation. We can only speculate whether elimination of oxygen consumption using muscle relaxation would have led to a better outcome. Next benefit of the use of general anaesthesia with advanced airway strategy is our concern that the patient might perceive subjectively unpleasant and painful procedures during CPR (mainly chest compressions and defibrillation) as well as risk of regurgitation and aspiration. On the other hand, cardio-depressive effect of anaesthetics and muscle relaxants and well-known risk of failed tracheal intubation may result in unfavourable result [10]. Surprisingly, even large-scale studies do not give a clear answer. The latest European Resuscitation Council guidelines stated that: “Advanced life support providers should initiate artificial ventilation as soon as possible in any patient in whom spontaneous ventilation is inadequate or absent” [10]. Does this mean that in some cases of gasping with apparently large gas exchange volumes and high rates, where patients' respiratory effort prevents manual face and bag breathing, oxygen administration alone is sufficient? In our opinion, it is necessary to proceed individually in atypical situations using all clinical knowledge.

Conclusion

In this case report, we described an abnormal agonal breathing pattern during CPR in a patient with ventricular fibrillation and demonstrated that in some cases gasping may be effective as a single method of