A fifteen-year-old boy suspected with barotrauma after sCUBA diving was presented by the Mobile Medical Team (MMT) to the emergency department (ED) at a level one trauma centre. The patient had been diving for 20 minutes at a maximum depth of nine meters when he probably panicked and dislodged his regulator underwater. As a consequence, he surfaced too fast without exhaling air which could have resulted in the intrathoracic pressure rising above a critical point. He probably aspirated water as well. On arrival at the scene, the patient was hypoxic and unresponsive so that the MMT decided to intubate.

On arrival in the ED the patient was screened for injuries according to ATLS (Advanced Trauma Life Support) principles. On examination the patient was found to be intubated and the pulse oximetry reading was 100% on a ventilator using 100% oxygen. On auscultation of the lungs, rhonchi were noted on both sides. At this time, the patient's blood pressure was 117/70 mmHg, pulse 100 bpm, temperature 36.3 degrees Celsius, pupils equally responded to light, Glasgow Coma Scale was E1M3Vtube while still sedated with midazolam, and no signs of lateralization were present. Further physical examination revealed anterolateral crepitus by palpation of the neck.

A chest radiograph was obtained, which showed no evidence of a pneumothorax. It did show diffuse bilateral alveolar density - which are characteristic of the radiologic findings in pulmonary oedema and it showed some subcutaneous emphysema as well. A CT scan of the thorax was obtained which revealed a pneumomediastinum, minimal pneumopericardium, minimal pneumothorax on both sides, air in the pulmonary vein and evidence of aspiration (Figure 1). A CT scan of the neck and cerebrum revealed subcutaneous emphysema in the neck, and no evidence of the presence of intracranial air. The clinical presentation was assumed to be a combination of burst lung syndrome and near drowning.

The risk of AGE and decompression illness (DCI) was considered to be present. A hyperbaric centre in an academic hospital was consulted asking if recompression therapy was indicated. Given the risk of the patient developing expansion of the pneumomediastinum with compression on the heart and great vessels while being treated in the recompression tank - with limited resuscitation possibilities available - it was decided not to treat the patient with recompression therapy.

The patient was admitted to the Paediatric Intensive Care Unit (PICU). Despite the significant changes on the CT scan of

Abstract - A pneumomediastinum is a condition in which air is present in the mediastinum. It can result from a pulmonary barotrauma which occurs during SCUBA (self contained underwater breathing apparatus) diving. We report on a fifteen-year-old patient who suffered from a pulmonary barotrauma including a pneumomediastinum after SCUBA diving. The patient was admitted to the PICU (Paediatric Intensive Care Unit) and treated conservatively. Subsequently the patient developed neurological deficits resulting from an arterial gas embolism (AGE). This case report discusses some of the principles of the pathophysiology and clinical presentation of diving emergencies.

Keywords - diving accidents, pneumomediastinum and baro-trauma

Case report
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Figure 1. CT slice showing pneumomediastinum (see arrows)
the chest, the patient did not require any ventilator support after admission to the PICU and could be extubated the same day. The patient obtained a maximal Glasgow Coma Score however, the patient had a mild right-sided hemiparesis and experienced a generalized seizure a few hours after extubation. A subsequent MRI showed evidence of left frontoparietal ischaemic infarction due to an AGE or due to general hypoxia of the brain during near drowning. Five days after admission, the patient was discharged with slight residual neurological symptoms, consisting of a mild right sided hemiparesis, which gradually improved over time.

Discussion
SCUBA diving is a popular sport. Anyone who participates in this sport must obtain diving certificates. From the age of ten, children can participate in an ‘open water’ diving course. The Dutch Association for Diving Medicine (Nederlandse Verening voor Duikgeneeskunde, NVD) advises fourteen years as a minimal age for diving. Diving is not without risks; in 2009 three fatal diving injuries and 37 non fatal injuries were documented in the Netherlands alone [1]. This is probably an underestimated number, because not all diving accidents are reported to the diving association. The pathophysiology for these accidents is mostly related to the behaviour of gases under different conditions, causing barotraumas due to changes in the volumes of gas-containing spaces in the body, decompression illness and toxic effects of increased partial pressures of certain gases in the breathing mixture [2]. In fact, the most common cause of death in divers is drowning and near-drowning followed by pulmonary related illnesses [3]. Most physiological changes in a scuba diver’s body flow from the physics of the behaviour of breathing gas under increased pressure associated with increasing depth [4]. For every 10 meter of depth, the atmospheric pressure increases by one atmosphere. As a result, a scuba diver diving from sea level to 30 meters of depth experiences a four-fold pressure increase from one to four atmospheres. Boyle’s law states that at a constant temperature and mass, the volume of a gas is inversely proportional to the pressure exerted on that gas. When the pressure is doubled the volume is reduced to one half of the original volume. When the pressure is reduced by one half the volume doubles, which is therefore the other way around [5]. The increase of pressure with depth has implications for gas filled spaces in the body such as the lungs, bowel, middle ear and sinuses. In the case of the pulmonary system, air can escape through the upper airway. When the air gets trapped and can’t escape during the diver’s ascent, (e.g. because of a closed glottis) a pressure gradient between any isolated gas and the surrounding tissue may result in a barotrauma. When a diver holds his breath during ascent, this can cause pneumomediastinum, pneumothorax or an AGE. During the diver’s ascent, gas can be forced into the pulmonary capillaries causing an AGE. Rupture of the lung may cause pneumothorax which can expand during ascent. Gas then tracks via the pulmonary interstitium into the mediastinum (pneumomediastinum). From there it can track into subcutaneous tissue manifesting as subcutaneous emphysema [6]. An AGE is the most feared complication of all, because it can cause cerebral infarction. A barotrauma may occur within a few minutes and presents differently from decompression sickness described below where the symptoms can-not be immediately recognised. Barotrauma is not dependent on diving time and depth. It can occur during a dive at a depth of 1-2 meters. The main cause for this type of accident is a panic emergency ascent in relatively inexperienced young divers [7]. Little is known about the treatment of a pneumomediastinum.

Henry’s law states that exposure to increased pressures of gas results in proportionally increased quantities of gas being dissolved in body tissues [4]. The most common breathing gas for divers is pressurized air which contains a mix of oxygen and nitrogen. When a diver breathes air under high pressure for a certain amount of time, the concentration of nitrogen dissolved in body fluids will increase. Blood will then become saturated with nitrogen [6]. If the pressure is suddenly decreased, such as when a diver rapidly ascends, a delay occurs before nitrogen can diffuse back from blood to a non-fluid space e.g. air in the lungs. This mechanism causes the formation of nitrogen bubbles as dissolved nitrogen returns to its gas form while still in the blood [8]. Another effect of increased partial pressure of dissolved nitrogen in the blood is the effect on the cerebral cellular membrane, causing an anaesthetic effect called nitrogen narcosis. This can be compared with the effects of alcohol, every fifteen meters of depth is equivalent in its effect to one alcoholic drink. DCI results from the effect of the bubbles on organ systems. It may cause vascular compression or occlusion, increase in tissue pressure or tissue distraction [4,6]. Besides, DCI may cause increased blood viscosity, damage to the endothelium and activation of the complement system [2]. The symptoms of DCI are often summed up by ‘the bends,’ ‘the chokes,’ ‘the staggers’ and ‘the tingles’ representing the musculoskeletal, pulmonary, inner ear, skin and central nervous system involvement. DCI can be initially treated with 100% oxygen until hyperbaric therapy can be provided. The window of opportunity for recompression therapy is unknown. The sooner therapy is initiated, the higher the probability of a good outcome. Clinical experience shows that the prognosis can be improved when recompression therapy is initiated hours or even days after time of onset [6].

Effects of immersion also give rise to physiological changes. Especially in cold water vasoconstriction leads to a redistribution of blood flow from the extremities into the central circulation. This may precipitate pulmonary oedema. It is believed that immersion in water causes increased cardiac preload. In such a situation, left ventricular cardiac performance is decreased by high negative inspiratory pressures caused by resistance in the breathing regulator [6]. Water aspiration may also be a causative factor [9]. Due to water aspiration, divers can develop acute dyspnoea and a productive cough with pink frothy sputum. Fluid aspiration can impair gas exchange as a result of altered surfactant function. This results in injury to the alveoli, atelectasis and adult respiratory distress syndrome (ARDS).

Our patient suffered from a pulmonary barotrauma including a pneumomediastinum after SCUBA diving. During admission he developed neurological symptoms as a result of an AGE.
The question remains whether this could have been prevented if the patient had been treated with recompression therapy. However, in this case this therapy was judged to be too risky. A pneumomediastinum is sporadically mentioned in the literature, where it is stated that a pneumomediastinum does not need any specific treatment [3,6,10]. Both death as well as a successful outcome after recompression treatment of a patient suffering from a pneumomediastinum have been reported [7,11]. It is still not clear what the best treatment is for a patient - presenting to the ED - with a pneumomediastinum after scuba diving that would minimize the risk of a potentially life-threatening AGE. In general, it is recommended that a hyperbaric treatment facility is consulted or, a doctor specialized in diving injuries to discuss the best treatment option for your patient.

References