Short Communication

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Quality of Mechanical Ventilation and Pacemaker Setting

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ABSTRACT

Increasingly, patients who require mechanical ventilator therapy already have implanted cardiac devices such as pacemakers, CRTs, or ICDs. These devices influence ventilation quality by setting the lower limit heartrate. Because gas transport capacity in the blood is partly determined by heartrate, the setting of the lower limit heartrate of these devices can limit the transport of CO_2 to the lungs and the transport of O2 to the tissue. At the Fachkrankenhaus Kloster Grafschaft hospital, several cases were observed in which the lower heartrate limit of the devices was left at the factory setting of 60 bpm, or a nighttime lowering of the lower heartrate limit was set too low. Reprogramming the devices to higher lower heartrate limits always resulted in normocapnic blood gas values.

Abbreviations: PM: Pacemaker; CRT: Cardiac Resynchronization Device; ICD: Cardiac Defibrillator; BGA: Blood Gas Analyses; NIV: Noninvasive Ventilation

Introduction

Increasingly, patients requiring mechanical ventilation are experiencing cardiac comorbidities requiring the implantation of a pacemaker (PM), cardiac resynchronization device (CRT), or cardiac defibrillator (ICD). Germany leads the European cardiac device implantation rate, with a steadily rising trend [1-3]. While there were 1,264 implantations per million inhabitants (pMI) in 2010, this number rose to 1,314 pMI in 2016 up to 1,816 pMI in 2024. Since the cardiac device therapy, once initiated, is usually continued lifelong, more and more of these devices are accumulating in the population. This device can play a crucial role in ventilation settings, as the CO_2 transport capacity to the lungs depends, among other parameters, on the heartrate [4,5]. When initiating intermittend mechanical ventilation only at night, the pulse rate critical for normocapnic gas transport may be undercut in cases where the devices are either left at the usual factory setting of 60/min or a nighttime lowering of the lower limit heartrate is programmed. In these cases, the achieved cardiac output is so low that, despite optimal ventilator settings, not enough CO_2 is transported to the lungs and thus exhaled. Even in normocapnic patients, the lower limit heartrate of the cardiac device can be problematic if the heartrate limits O_2 transport from the lungs to the tissue (Figure 1). Bassett, et al. [6] have extensively demonstrated that cardiac output, as the product of stroke volume and pulse rate, is crucial for O_2 uptake [6].

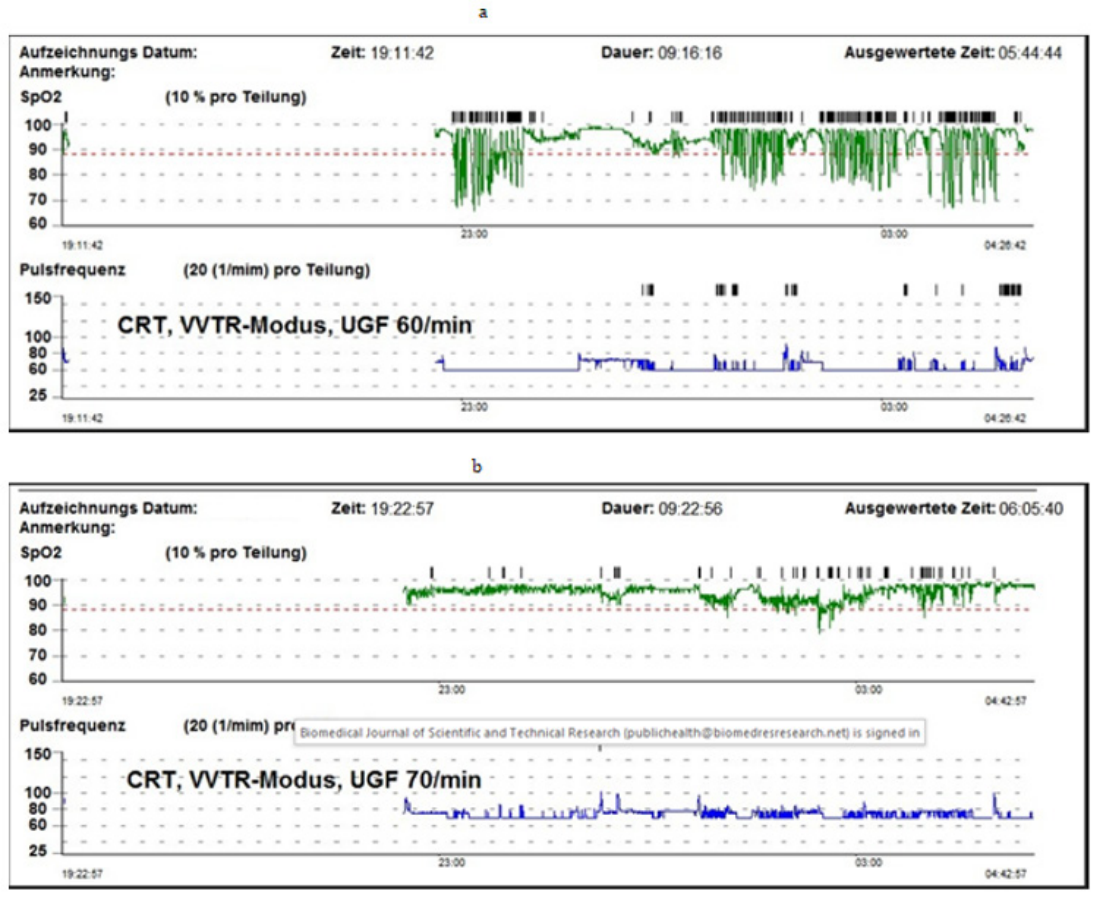

Figure 1:

a. Pulse oximetry of a patient with early childhood hypoxic brain injury. Deep, long-term desaturations are observed with the lower limit heartrate set to 60/min during normocapnia.

b. Pulse oximetry of the same patient as shown in Figure 1a, but after increasing the pacemaker's backup heartrate to 70/min. This resulted in stable saturation with values predominantly above 90%. No mecssshanical ventilation was necessary.

Methods

As part of routine controls for home ventilated patients or those in which machanical ventilation therapy is started, transcutaneous pCO_2 measurements (tCO_2) and blood gas analyses (BGA) are performed very regularly to monitor the target parameter pCO_2, in accordance with the S_2 guideline of the German Society for Pneumology and Respiratory Medicine (DGP) [7]. TOSCA 500 devices (Radiometer, Copenhagen, Denmark) and SenTec SDM monitors (SenTec AG, Therwil, Switzerland) are available for tCO_2 measurements. BGAs are performed using ABL 90 Flex devices (Radiometer medical ApS, Bronshoj, Denmark), and pulse oximetry is performed using Nonin Wrist Ox_2 3150 devices (Nonin Medical, USA). Pulse oximetry is performed simultaneously with the TOSCA 500 and SenTec SDM devices, so that, just like with the Nonin pulse oximeters, pulse rate curves are available for the entire measurement night. In cases of persistent hypercapnia after successful ventilation initiation, invariate heartrate curves were observed in some cases, preferably at even-numbered 10s (50/min or, most frequently 60/min). In other cases, sudden nocturnal changes (by exact amounts of 5/min or 10/min) in the heartrate were observed on the background of an invariate heartrate graph. Upon closer examination, it turned out that these were patients with PM, CRT, or ICD devices, in whom the lower heartrate limit of the cardiac device had remained at the factory setting, or in whom an evening heartrate reduction or a morning heartrate increase of the backup heartrate had been programmed.

Results

An influence of the cardiac device setting on ventilation quality was not observed in all cases. However, in some cases, it was observed that the set lower limit heartrate or the nighttime heartrate reduction

impaired blood gas transport to such an extent that hypercapnic tCO_2 values occured, which fell significantly either during the programmed morning increase of the pacemaker backup rate (Figure 2) or upon awakening of the patient. In critical cases, the cardiac devices could be reprogrammed accordingly to allow and ensure improved nighttime CO_2 exhalation within the framework of the ventilation setting. The last case of a patient with COPD stage GOLD IV and concurrent three-vessel coronary disease, a history of myocardial infarction, ischemic cardiomyopathy, first-degree AV block, and ICD implantation eight years previously was successfully initially placed on noninvasive ventilation (NIV). However, the pCO_2 values remained in the hypercapnic range (BGA values: at 11:30 p.m. pCO_2 = 54.3 mmHg, at 2:34 a.m. pCO_2 = 56.7 mmHg). The ICD device was set to a lower heartrate limit of 50/min, which the patient consistently achieved during the night. With the assistance of the cardiology department, the ICD device was reprogrammed to a lower heartrate limit of 60/min, by which normocapnic pCO_2 values could be achieved (BGA values: at 1:49 a.m. pCO_2 = 48.7 mmHg, at 4:25 a.m. pCO_2 = 39.4 mmHg). This example, as well as those in Figures 1 & 2, are part of a collection of cases that began in 2012 with the first case of this type.

Since then, a steady stream of cases has been added in which, after successful NIV adjustment, only reprogramming the respective cardiac device enabled ventilation quality within the range of normocapnic BGA values or a significant reduction in pCO_2 values.

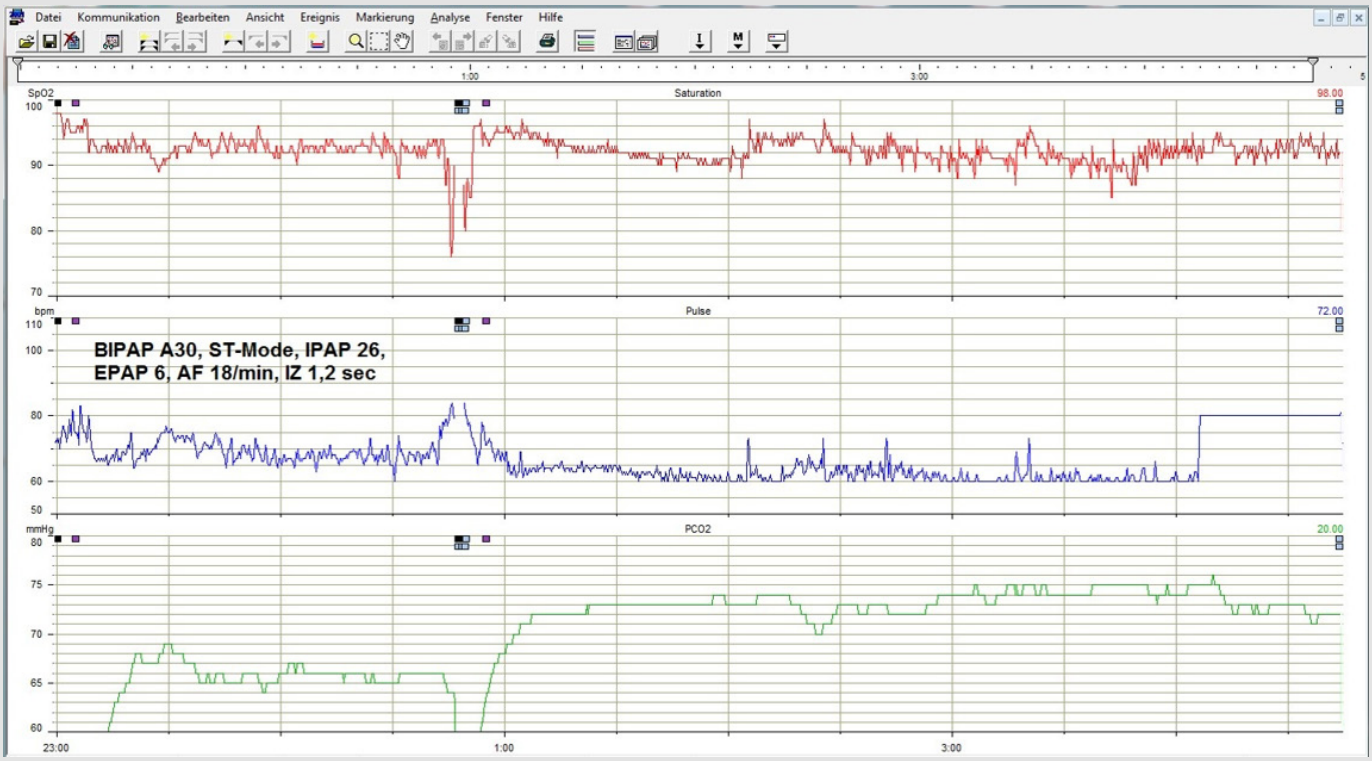

Figure 2: Transcutaneous pCO_2 measurement of a patient undergoing noninvasive ventilation with the implanted pacemaker's night mode activated (programmed reduction of the nighttime heart rate). From 11:00 p.m. to 0:50 a.m., the patient was still awake, then fell asleep after using the toilet with tCO_2 values raising up to 75 mmHg. In the morning, around 4:07 a.m., tCO_2 dropped after the programmed end of the nighttime reduction of the lower limit heart rate (60/min) and increased to the daytime backup level of 80/min.

Discussion

A growing number of patients requiring mechanical ventilation are already fitted with electronic cardiac devices (SM, CRT, ICD) when acute or chronic respiratory insufficiency occurs. The setting of these dardiac devices regarding the lower heartrate limit, or the setting of a nighttime lowering of the backup heartrate limit, is evidently based primarily on standard or factory settings. In a study of 140 pacemaker patients, Kruse [8] found that in 103 (73.6%) patients, the lower frequency limit remained at the factory setting of 60 beats/min. Only in 37 (26.4%) patients was the lower heartrate limit actively adjusted downwards (n=8) or upwards (n=29) [8]. In various cardiac diseases (e.g. hypertension, coronary heart disease, heart failure), a reduction in pulse rate (usually with beta-blockers) is pursued as a cardiac ther-

apy strategy, as this prolongs the diastolic time in which the blood flow to the heart muscle itself occurs [9,10]. A low heartrate is often considered beneficial for cardiology patients. Therefore, the setting of the lower heartrate limit on implanted devices appears to receive relatively little attention. The question of the heartrate range within which the lower heartrate limit of such a device can be easily varied remains unanswered. Chew, et al. [11] conducted a study in which the nocturnal heartrate of dual-chamber pacemakers was set to 50/min for three weeks and then to 80/min for three weeks. The various parameters of systolic and diastolic cardiac function were each worse at 80/min than at 50/min. However, the study only examined nine patients with impaired cardiac function [11].

Ideally, cardiac function could be measured individually in each individual case, depending on the lower heartrate limit set on the cardiac device. Nighttime impedance cardiography could be used for this. Using impedance cardiography, the heartbeat volume could be recorded continuously, beat by beat, throughout the entire night. If night mode is activated on the devices, two different backup heartrates can be set within a single measurement night and their effect on stroke volume determined. A statistical intra-individual comparison of the mean stroke volume values of approximately 14,000 cardiac cycles per heartrate setting would allow an assessment based on a very solid data population. This technology is now available at the Kloster Grafschaft hospital. Since the pulse rate determines blood gas transport capacity (CO_2 transport to the lungs and O_2 transport from the lungs to the tissue), the setting of the lower heartrate limit on the PM/CRT/ICD devices influences ventilation quality. The lower heartrate limit of the cardiac device can be set so low that, despite correctly set ventilation parameters, not enough CO_2 is transported to the lungs and adequate ventilation quality is not achieved. In such cases, in addition to IPAP, EPAP, respiratory rate, and inspiratory time on the ventilator, the dardiac device, as the fifth element of the ventilation setting, must be adjusted as required. In normocapnic patients (Figure 1) without a ventilator, O_2 transport may be limited.

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