CASE REPORT

Stabilizing Rib Cage Destabilizes Breathing!!

Rahul Kumar[1], Anshul Jain[2], Dipti Gothi[3], Mahismita Patro[4], Sunil Kumar[5]

Abstract

Central sleep apnea (CSA) is characterized by intermittent episodes of decreased ventilation due to lack of drive to breathe while asleep. It is due to an abnormal carbon dioxide control of ventilation which may be due to blunted or enhanced ventilatory response to carbon dioxide. An increased CO_2 sensitivity, an important factor in the development of central sleep apnea, precipitates it when associated with unstable breathing, i.e., high loop gain. The combination of speed of feedback gain, plant gain, and controller gain leads to high loop gain. We hereby report a case of central sleep apnea precipitated after coronary artery bypass grafting (CABG) performed via midline sternotomy. Factors responsible for sleep apnea in this case were cardiac failure, postsurgery low cardiac output syndrome, and decreased thoracic cage movement due to sternotomy. Normally these postsurgical cases resolve on their own, but our patient had very severe symptoms and hence he reported.

Keywords: Central sleep apnea, Loop gain, Pulmonary mechanics.

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Introduction

Central sleep apnea (CSA) is characterized by intermittent episodes of decreased ventilation due to lack of drive to breathe while asleep. It is diagnosed as CSA syndrome when the apnea-hypopnea index (AHI) is ≥5/hour and >50% of the events are central with evidence of daytime sleepiness.[1] Sternotomy suture may alter pulmonary mechanics as seen with the abnormality of the chest wall and hence may lead to CSA. We hereby report a case of CSA precipitated after coronary artery bypass grafting (CABG) performed via midline sternotomy. Only one such case is described in online literature to the best of our knowledge.[2]

Case Report

A 63-year-old man had undergone CABG for triple vessel disease following myocardial infarction in December, 2019. He developed severe choking episodes after the surgery for which the relatives were so scared that they kept awake in the night observing him helplessly! He had excessive daytime sleepiness and poor sleep quality after the CABG. He had no other comorbidities. On evaluation, the Epworth Sleepiness Score was 8/24, the STOP Bang score was 4/8, while the perioperative sleep apnea prediction score was 3/9. The body mass index was 19.5 kg/m^2. Clinical and laboratory examination revealed no abnormality. Serum N-terminal pro-brain natriuretic peptide was normal. Arterial blood gas showed pH:7.43, $PaCO_2$:34 mm Hg, PaO_2:79.1 mm Hg, HCO_3^-:18.3, SaO_2:95.9%. The chest radiograph showed cardiomegaly with sternotomy sutures. The 2-D echocardiography showed regional wall motion abnormality with left ventricular ejection fraction of 45–50%. Overnight polysomnography suggested presence of CSA with AHI of 45/hour (Fig. 1). Continuous positive airway pressure (CPAP) at 10 cm of water was prescribed after titration study. On follow-up, the symptoms of sleep disorder had resolved. Polysomnography repeated after 6 months showed AHI of 10/hour. There was evidence of unstable breathing with persistent high loop gain (Fig. 2).

Discussion

The CSA is due to an abnormal carbon dioxide (CO_2) control of ventilation. Abnormal CO_2 control of ventilation may be due to blunted or enhanced ventilatory response to CO_2. The blunted response is exhibited by hypoventilation and is associated with hypercapnia. It is seen in neuromuscular disorders and drug overdose.[3] The enhanced ventilatory response to CO_2 leads to hypocapnia which can be best demonstrated by CO_2 ventilatory response curve.[4,5] The curve is a graphical depiction between $PaCO_2$ and alveolar ventilation, which is usually linear. The minute ventilation typically increases by 2–5 L/minute for every 1 mm Hg increase in $PaCO_2$. If the ventilatory drive is increased the curve shifts to left which means there is a heightened ventilatory response to CO_2 (Fig. 3). An increased CO_2 sensitivity is an important factor in the development of CSA and Cheyne–Stokes respiration. But it alone cannot lead to CSA unless there is an associated unstable breathing, i.e., high loop gain.

A high loop gain is commonly seen with heart failure. CSA occurs in 30–50% cases of heart failure and involves at least two of the three physiological abnormalities: (a) prolonged lungs to brain circulatory time due to the reduced cardiac output (speed of feedback gain); (b) inability of lung to respond to changes in PaO_2 and $PaCO_2$ due to pulmonary congestion (plant gain); and (c) enhanced ventilatory response to $PaCO_2$ (controller gain).[6–8] The combination of these factors leads to high loop gain. High loop gain fuelled by low apnea threshold (critical value of $PaCO_2$ below which the respiratory drive ceases) is the mechanism behind CSA in cardiac failure.[9] During daytime behavioral influences and neuro-compensatory responses strongly oppose apnea even in the presence of marked decreases in $PaCO_2$ but during night CSA develops easily as only chemosensitivity drives the

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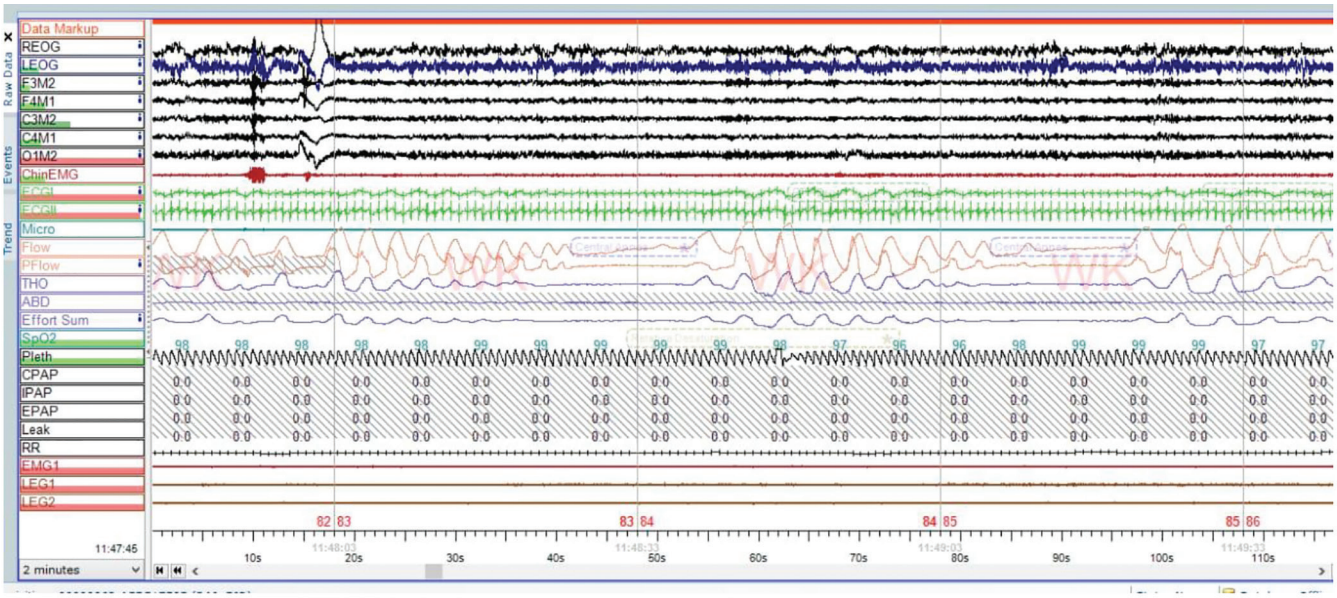

Fig. 1: Immediate postsurgery polysomnography recording. AHI was 45/hour as per AASM 2012 criteria

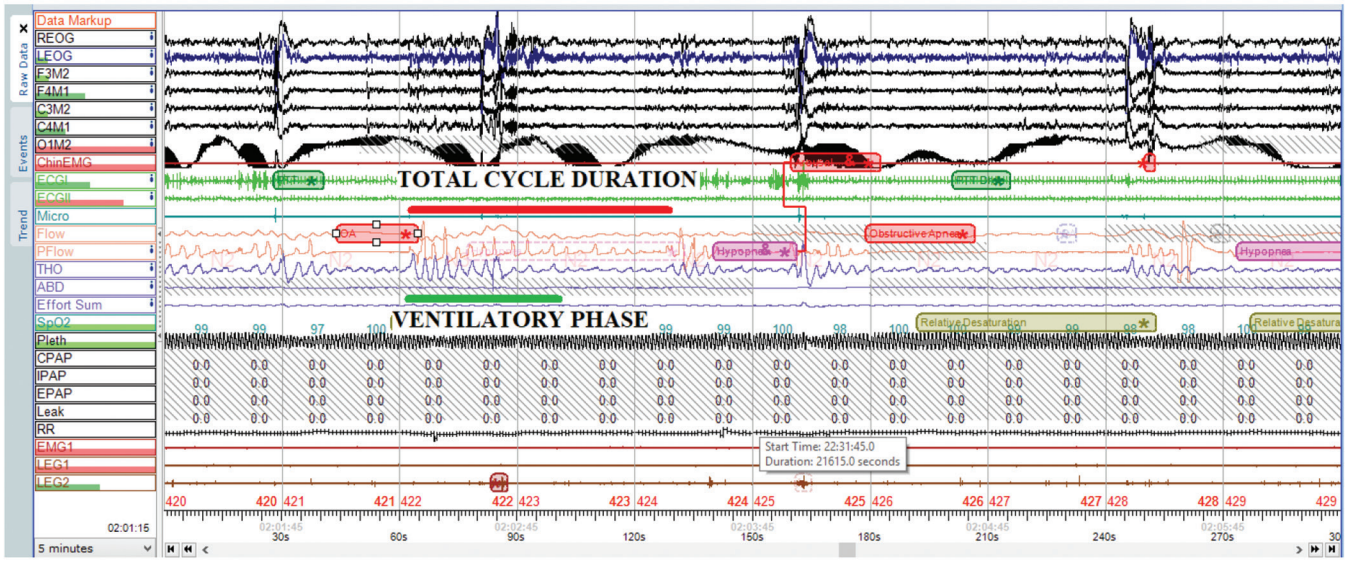

Fig. 2: Representative polygraph recording from PSG done after 6 months for calculation of loop gain. Loop gain is calculated by formula: $2\pi/[2\pi DR - \sin(2\pi\ DR)]$, where DR is the ratio of the duration of the ventilatory phase (time from the end of one apnea to the start of the next – indicated by green line) to the total cycle duration (time from the end of one apnea to the end of the next). Duration of ventilatory phase in this case was 37 seconds and the duration of the total cycle was 67 seconds. Hence, DR was 37/67 = 0.55. Loop gain was calculated using the formula $2\pi/[2\pi DR - \sin(2\pi\ DR)]$ and was found to be 1.85

ventilation.[3] Our patient had high loop gain demonstrated in polysomnography 6 months after the surgery even in absence of significant CSA indicating that high loop gain alone was not responsible for CSA in our case.

Thus, other than the low ejection fraction, factors responsible for CSA in this case could be (1) increased reduction in cardiac output as cardiac surgery is known to reduce the cardiac output[10] and (2) decreased thoracic cage movement due to sternotomy,[11] basically inability of chest wall (plant gain) to respond to the increased metabolic needs. Hence, CSA was due to alteration in cardiac and pulmonary mechanics following CABG. This rare cause of CSA should be kept in mind following CABG. Normally these postsurgical cases resolve on their own, so they do not get reported. One such case has been described in online format by Testelmans et al.[2] Since our patient had very severe symptoms and severe CSA, he required treatment with CPAP. If the symptoms are not severe, patients can be managed conservatively and should be reassessed after 3 months.

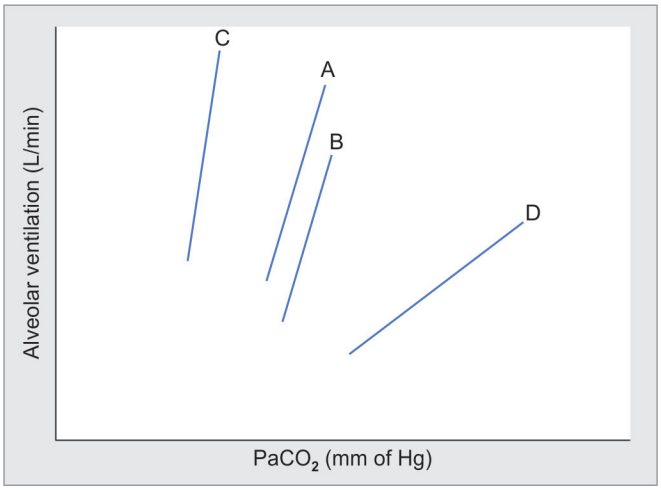

Fig. 3: The CO_2 response curve is a graphical depiction between $PaCO_2$ and alveolar ventilation. The CO_2 response curve A in awake and normal, B in sleep and normal, C in hyperventilation and shifted to left, and D in hypoventilation and shifted to right

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