Congestive Heart Failure and Central Sleep Apnea

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KEYWORDS

• Congestive heart failure • Central sleep apnea • Cheyne stokes respiration • Loop gain

KEY POINTS

- Congestive heart failure (CHF) is a common clinical syndrome among patients in the intensive care unit (ICU), who frequently require noninvasive or mechanical ventilation.
- CHF affects breathing control by increasing chemosensitivity and circulatory delay, predisposing to central sleep apnea, classically in a crescendo–decrescendo pattern of respiration known as Cheyne–Stokes respiration (CSR).
- Few data are available to determine prevalence of CSR in the ICU, or how CSR might affect clinical management and weaning from mechanical ventilation.

CLINICAL CONSIDERATIONS

Historical Perspective

An abnormal respiratory pattern has long been recognized as an ominous sign of congestive heart failure (CHF). The observations of 3 physicians who have lent their names to the pattern remain informative:

> *His breathing was very particular: he would cease breathing for twenty or thirty seconds, and then begin to breathe softly, which increased until he breathed extremely strong, or rather with violent strength, which gradually died away till we could not observe that he breathed at all. He could not lie down without running the risk of being suffocated, therefore he was obliged to sit up in his chair.*
>
> *—John Hunter, 1781*[1]

> *The patient suddenly developed palpitations and displayed signs of severe congestive heart failure. The only particularity in the last period of his illness, which lasted eight or nine days, was in the state of respiration. For several days his breathing was irregular; it would entirely cease for a quarter of a minute, then it would become perceptible, though very low, then by degrees it became heaving and quick, and then it would gradually cease again. This revolution in the state of his breathing occupied about a minute, during which there were about thirty acts of respiration.*
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> *—John Cheyne, 1818*

> *This symptom [periodic breathing], as occurring in its highest degree, I have only seen*

This article originally appeared in Critical Care Clinics, Volume 31, Issue 3, July 2015.
Disclosure Statement: Dr S.A. Sands is supported by a National Health and Medical Research Council of Australia Early Career Fellowship and R.G. Menzies award (1053201). Dr R.L. Owens is supported by the National Institutes of Health K23 HL105542. He has previously consulted for Philips Respironics.
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Sleep Med Clin 11 (2016) 127–142
http://dx.doi.org/10.1016/j.jsmc.2015.10.003
1556-407X/16/$ – see front matter

during a few weeks previous to the death of the patient.

—William Stokes, 1854

The initial observations by Hunter, Cheyne, and Stokes were made in patients close to death. They were the first to note the characteristic waxing and waning respiratory pattern of "Cheyne–Stokes respiration" (CSR), a common pattern of central sleep apnea in patients with CHF. CSR is characterized by complete cessation of respiratory effort and airflow (apnea phase) alternating with profound hyperventilation (hyperventilation phase). Such patterns occur during wakefulness, but are typically more prominent during sleep (**Fig. 1**). The apnea phase of CSR causes arterial hypoxemia, and the hyperventilation phase produces surges in blood pressure, arousal from sleep, and dyspnea (**Fig. 2**).[2–5] Typically, CSR has a periodicity of 45 to 90 seconds, and occurs during non–rapid eye movement (REM) sleep stages 1 and 2. CSR severity is typically measured by quantifying the percent of total sleep time in CSR, and by the number of apneas and central hypopneas per hour of sleep (apnea–hypopnea index).[6] Despite advances in the treatment of CHF (eg, β-blockade, spironolactone), untreated CSR remains highly prevalent during sleep and retains its association with increased morbidity and mortality independent of the severity of heart failure (**Boxes 1–4**).[7–10]

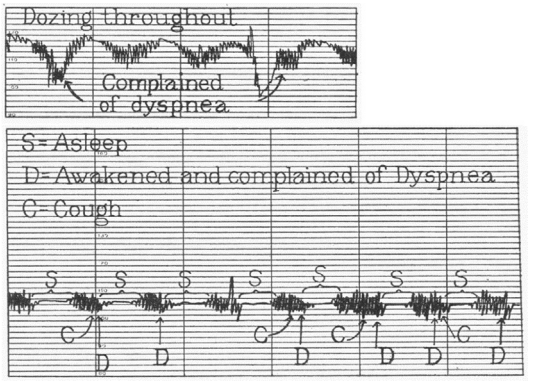

Fig. 2. Increased ventilatory drive during the hyperventilation phase of Cheyne–Stokes respiration results in dyspnea in a patient with heart failure. (*Adapted from* Harrison TR, King CE, Calhoun JA, et al. Congestive heart failure: Xx. Cheyne-Stokes respiration as the cause of paroxysmal dyspnea at the onset of sleep. Arch Intern Med 1934;53(6):897; with permission.)

Epidemiology in Stable Congestive Heart Failure

One of the first rigorous studies to use polysomnography by Javaheri and colleagues[11] found a high prevalence (40%) of CSR in patients with systolic heart failure. This prevalence has been a relatively consistent finding depending on the population studied (with increased prevalence with worsening heart failure) and the threshold and technology used to diagnosis sleep disordered breathing.[12–15] Although early epidemiologic studies predated the widespread use of advanced heart failure therapies, even the most recent studies continue to show a consistently high prevalence of CSR.[14,16] CSR is not limited to systolic heart failure; CSR is common in patients with symptomatic heart failure with preserved

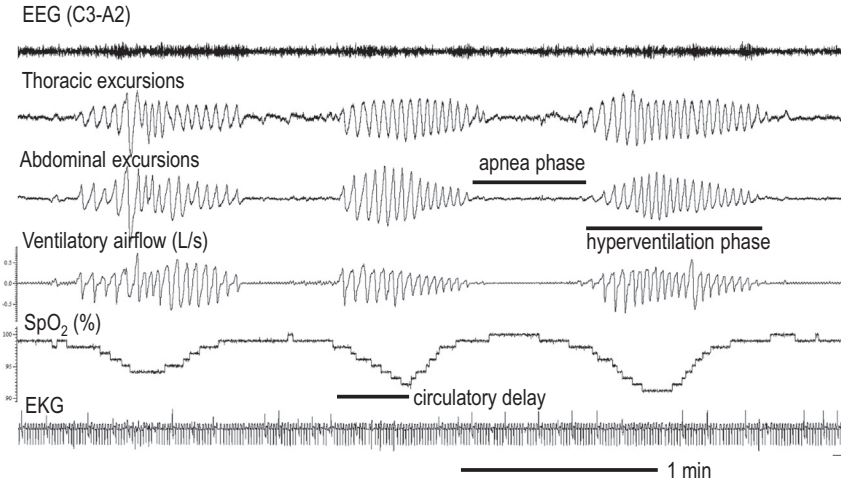

Fig. 1. Cheyne–Stokes respiration in a patient with congestive heart failure. Note the crescendo–decrescendo pattern of respiratory effort and airflow. The lung-to-ear circulatory delay can be approximated by the time from resumption of airflow to the start of the increase in oxygen saturation. EEG, electroencephalogram; EKG, electrocardiogram; SpO_2, oxygen saturation.