A Peculiar Type of Dyspnea: Kussmaul, Cheyne-Stokes, and Biot Respiration

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Abstract:
Observations concerning respiratory rates and patterns date back to the time of Hippocrates and Galen, and there are many descriptive terms such as ataxic, agonal, and clustered. Among these terms are three well-known but often misunderstood and misused eponymous respiratory signs: Kussmaul respiration, Cheyne-Stokes respiration, and Biot respiration. In the 21st Century, in which roentgenograms and laboratory tests often serve as surrogates for physical examination, respiratory patterns – though frequently present – are often overlooked. Herein, to rejuvenate clinical interest and clarify misconceptions concerning their application and utility, we present clear descriptions of three useful clinical signs: Kussmaul respiration, Cheyne-Stokes respiration, and Biot respiration.

Keywords: Kussmaul, Cheyne-Stokes, Biot, dyspnea

Introduction
The most regularly overlooked and ignored vital sign in the clinic and wards today is the respiratory rate, and it has nearly become the standard to simply document twenty breaths per minute as the rate when we think of the patient as breathing normally (Figure 1). In the 21st century, when physicians frequently spend more time in front of a computer than at the bedside, it would be uncommon to merely observe a patient’s respiratory pattern for many minutes; however, important clinical information can be missed by our lack of observation. In an effort to revive clinical interest, increase awareness of these signs, and clarify historical dispute, we present the index cases1 and descriptions of three well-known, but often missed and misunderstood, eponymous respiratory patterns: Kussmaul respiration, Cheyne-Stokes respiration, and Biot respiration.

Kussmaul Respiration

A Peculiar Type of Dyspnea: In this type of dyspnea there is not the least suggestion as is so common in all other types that the passage of air to or from the lung has to combat obstruction in its path; to the contrary it passes in and out with the greatest of ease. The thorax expands noticeably in all directions without a pulling-in of the lower end of the sternum or intercostals spaces… and this complete inspiration is followed by a likewise complete expiration… Yet everything is indicative of extreme air hunger, such as the discomfort of angusti of which the

1 Index case refers to the clinical case in which a condition or sign was first described.
patient complains, the extreme activity of the respiratory muscles, and the loud noise that the powerful inspirations and more so the expirations make... A true stridor, however, never exists... To the contrary the noise of expiration often becomes a groan even when the patient lies unconscious in deep coma... The marked contrast between the extreme general weakness of the patient and the powerful respiratory movements is a striking peculiarity of this picture. (1)

This clinical scenario which depicts a diabetic patient was written by Adolf Kussmaul (1822-1902) in 1874. A year after his ground-breaking accounts of Kussmaul Sign and Pulsus Paradoxus, he published “A Peculiar Type of Dyspnea” wherein he first documents this respiratory pattern now known as Kussmaul Respirations. He had trained under the famous physicians Rudolf Virchow, Franz Näegele, Josef Skoda, Carl von Rokitansky, and Ferdinand von Hebra and was well-equipped by 1874 with the clinical acumen and pathologic skills to make such an observation.

In describing these respirations, his name has become synonymous with the term diabetic coma which was formerly referred to as “Kussmaul Coma”. Without understanding the acid-base physiology responsible for the “peculiar type of dyspnea”, he nonetheless suspected that a chemical imbalance due to the patient’s diabetes was responsible. He went on to study the effects of acetone, chloroform, ether, and alcohol on rabbits and humans which, when viewed with knowledge of the physiologic disturbances these substances cause today, suggest that he was on the right track. (2)

The present day clinical significance is that the air-hunger described by Kussmaul is secondary to extreme metabolic acidemia. Patients with this type of acidemia exhibit markedly low partial pressure of arterial carbon dioxide (pCO₂) as compensation for low bicarbonate. Figure 2 shows the respiratory tracing representative of a patient with Kussmaul respiration; as demonstrated in the figure, the respiratory rate is normal or slightly reduced. Although the rapid, shallow respirations that accompany less severe acidemia will often be described as Kussmaul respirations, the pattern Kussmaul described is more accurately a sign of severe acidemia when the rate of respiration has decreased dramatically.

Although the index case describes a diabetic coma, patients with advanced renal failure, sepsis, or intoxications such as ethylene glycol may present in a similar manner. Physicians should be alert to such possibilities, especially if they observe dyspnea distinguished by large tidal volumes without adventitial breath sounds or increased work of breathing.

Cheyne-Stokes Respiration

The only peculiarity in the last period of his illness... was in the state of the 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. (3)

Dr. John Cheyne (1777-1836) first noted this “peculiar” breathing pattern in 1818. In that year, he published a paper entitled “A
Figure 1: Normal Respiratory Tracing

Figure 2: Kussmaul Respiratory Tracing

Figure 3: Cheyne-Stokes Respiratory Tracing
Figure 4: Biot Respiratory Tracing

Figure 5: Cluster Breathing
Case of Apoplexy, in which the Fleshy Part of the Heart was converted into Fat” in which he described the index case of a 60-year-old man who eventually died from congestive heart failure and apoplexy (stroke). Figure 3 demonstrates the respiratory tracing for this breathing pattern; it is characterized by gradual increasing and decreasing tidal volumes followed by short periods of apnea.²

The breathing pattern was again independently described by William Stokes (1804-1878) in 1854. Propitiously, William Stokes had trained at the Meath Hospital in Dublin where Cheyne had been an attending physician; this placed him in a unique situation to further study the peculiar dyspnea described by Cheyne in 1818. Dr. Stokes again recognized this sort of breathing pattern from a mere observation and tried to connect it, physiologically, to “degeneration of the heart” which he thought was quite specific to the disease state resulting in the dyspneic symptoms. In 1854, he wrote:

It consists in the occurrence of a series of inspirations, increasing to a maximum, and then declining in force and length, until a state of apparent apnea is established. In this condition the patient may remain for such a length of time as to [appear] dead, when a low inspiration...marks the commencement of a new ascending and then descending series of inspirations...The decline in the length and force of respirations is as regular and remarkable as their progressive increase. (4)

Cheyne-Stokes respiration was well-known by the later part of the 19th century, and curiously, it was Camille Biot, known for the eponymous Biot Respiration, who conducted much of the research.

We now know that Dr. Stokes was at least partially right in his assumption of this breathing pattern’s etiology; however, because Cheyne-Stokes respiration occurs as a result of damage to the respiratory control centers whereby the normal feedback for increasing or decreasing pCO2 levels is diminished, it is not entirely specific for congestive heart failure as he suggested. Less commonly, it can be a sign of stroke (apoplexy), toxic encephalopathies, traumatic encephalopathies, high-altitude pulmonary edema, and sleep apnea.

Today, during emergency situations, its presence can be a valuable clue for the bedside physician; its prompt recognition during the physical assessment can lead to earlier initiation of therapy while waiting on more advanced diagnostics such as radiographs.

Biot Respiration

As an intern in Lyon, France, in 1876, Camille Biot (1850-1918) wrote of a 16-year-old-patient with tuberculous meningitis:

Peculiarly, this breathing pattern lacks the crescendo-decrescendo cycles attributed to Cheyne-Stokes breathing and is completely irregular with varying periods of apnea. The breathing pattern is irregular and rapid, with rhythmical pauses lasting 10-30 seconds, but sometimes with alternating periods of apnea and tachypnea.³ (5, 6)

³ Tachypnea is defined as a respiratory rate greater than twenty-two times per minute

² Apnea refers to the absence of respiration
Originally, this breathing pattern was called “rhythme meningitique” as it was routinely used by physicians as a diagnostic tool for meningitis; however, today we know it as Biot respiration. (5) Although others before him, such as Armand Trousseau, had remarked on this “peculiar” breathing pattern, Biot was the first to categorically set it as a distinct entity.

It is characterized by a highly irregular or ataxic breathing pattern with variable tidal volumes and random periods of apnea (Figure 4). Unfortunately, there is much historical confusion regarding the definition of Biot respiration. Biot, who was an expert in the study of Cheyne-Stokes respiration, first described his eponymous respiratory pattern in a paper examining Cheyne-Stokes respiration, and this may have initially led to the confusion. However, most conspicuous is the misunderstanding between Biot respiration and cluster breathing, both of which are types of agonal breathing patterns. Definitions of Biot respiration vary from “rapid, shallow breaths” to “clustered, periodic breathing” but Biot respiration should properly refer only to breathing patterns with markedly variable tidal volume, random apneas, and no regularity:

This irregularity of the respiratory movements is not periodic, sometimes slow, sometimes rapid, sometimes superficial, sometimes deep, but without any constant relation of succession between the two types, with pauses following irregular intervals, preceded and often followed by a sigh more or less prolonged. (5)

This description lucidly illustrates that Biot respiration or ataxic respiration is easily distinguished from cluster breathing by its irregularity. As suggested by the name, cluster breathing implies a regular pattern with variable tidal volumes and periods of apnea (Figure 5).

The clinical significance of Biot respiration today can be appreciated by again examining the setting in which he initially described it. The index case was a sixteen year old man with severe central nervous system pathology, i.e. tuberculous meningitis; thus, it is not unexpected that it is still considered a sign of brainstem infarction or uncal herniation. (7) Additionally, some have suggested that Biot respiration can also occur in prolonged, chronic opiate use (8) as well as being erroneously attributed to high-altitude pulmonary edema, where it was confused with the correctly associated Cheyne-Stokes respirations. (9)

As with any type of agonal breathing, recognition of Biot respiration should prompt urgent action by the physician especially if it occurs in conjunction with altered mental status, decreased level of consciousness, or other neurologic deficits. In these settings, where seconds and minutes are critical to patient outcomes, the alert physician who observes Biot respiration will be able to act immediately rather than waiting for additional diagnostics.

**Conclusion**

Respiratory patterns can provide valuable and rapid insight into the clinical scenario of patients, and vigilance in observation should be encouraged. By presenting and clarifying these eponymous respiratory patterns, it is hoped that a rejuvenation of interest will be sparked and lead to enhanced patient care.

**References.**


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