

Research article

Breathing Patterns in Acute Illness

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Abstract

Background: Clinical observations of breathing are rudimentary, compared with, for example, the use of ultrasound to display circulatory pathophysiology. However measurements of respiratory flow can be easily made with nasal cannula pressure, and could be displayed on a monitor screen or smartphone. Respiratory flow measurements were recorded in patients admitted to hospital with acute illness, and often showed abnormalities which might prove useful in diagnosis or prognosis. Methods: A cohort of patients had been recruited to allow assessment of a new device intended for continuous measurement of respiratory rate. The comparison was with a signal from a nasal cannula, which is minimally invasive, and gives a signal analogous to flow. Nasal flow recordings were obtained from 63 patients (35 were female), age 61 (17) (Mean, SD). On inspection, this set of records showed features of interest, so a subsequent formal analysis of abnormal patterns was done, and is reported. Results: The median respiratory rate the patients was 25 (22, 29) (median, quartiles) breath.min⁻¹. Abnormal breathing patterns were very frequent. These included early inspiration in 82%, and also expiratory flow limitation, passive expiration, and delayed or absent active inspiration (63%). Cyclical variation in breath size was noted in 8% of the patients, indicating a minor degree of Cheynes-Stokes pattern breathing. Conclusions: In acutely ill medical patients, unexpected abnormal breathing is frequent. This can be detected by nasal pressure measurement. Some abnormalities could be useful for diagnosis and prognosis, such as incipient ventilatory failure: formal prospective study could be valuable.

Keywords: breathing pattern, incomplete expiration, acute illness, nasal flow, breath timing

Introduction

Although respiratory rate is a “vital sign”, and often emphasised in practice guidelines, I could find no systematic study of the characteristics of breathing in acute illness. In the course of clinical evaluation of a new device, we had recorded breathing in acutely ill patients in a tertiary care hospital, using a nasal cannula and pressure transducer. These adult patients, studied shortly after admission to hospital, had a variety of acute illnesses. The recordings allowed their breathing patterns to be described. These suggest that closer examination of breathing, using a visual display of nasal pressure, could allow better recognition of respiratory dysfunction, and might be a simple useful diagnostic tool.

The method of using nasal cannulae to monitor breathing came from adapting oxygen cannulae [1] for physiological measurements. It has been widely adopted to assess breathing in patients with sleep disturbances, but is rarely if ever used in patients with other illnesses. In the course of developing a new device to measure respiratory rate in hospital patients, we compared rate measurements made using the new device with measurements obtained from nasal cannula pressure recordings. Review of these nasal cannula recordings allowed other features of breathing to be

identified that might not be apparent during routine clinical examination.

A large proportion of the patients had abnormal features that in many instances would not have been recognised clinically. Some of these are well known phenomena such as Cheyne – Stokes breathing, which can be noted clinically when very careful examination is conducted, or when the changes in rate and depth of breathing are extreme. Some features, such as incomplete expiration, have been described in laboratory studies. However other abnormalities were noted that have not been previously formally described. All of these features were recognised from a visual display of the nasal pressure recording, which is easily and simply obtained.

Methods

To measure nasal pressure, adult nasal pressure cannulae (Sleep Sense 15802-2, Medes Ltd., Shenley Hill, Radlett, Herts, UK) were used, with a separate Millipore bacterial filter. The cannulae were positioned on the patient in the same way as oxygen nasal cannulae, placed at the nasal openings and then looped over the top of the ears. The tubes then ran down to the

front of the neck where the assembly was tightened with a sliding fastening. The two tubes joined to a single tube which connected through a Millipore bacterial filter to a pressure sensor, which was usually taped to the patient's pillow. After measurement, the signal was sampled at 12.5 Hz and transmitted digitally by Bluetooth radio connection to an iPod device at the bedside, where it was stored in memory. The signals were then archived on a secure server.

The pressure tracing closely resembles flow at the nose, the pressure decreases during inspiration. The signals were replayed using proprietary laboratory processing software (Spike2 version 5.21). Measurements were made of respiratory frequency by detecting the decrease in cannula pressure at the onset of each inspiration. The entire recording of each patient was inspected, first generally and then in detail, and the presence of abnormal features noted. After repeated reviews, features that were frequent were noted and tabulated. The records were then formally analysed for the presence of these specific features. Representative examples have been taken from the recordings to illustrate these features. To aid interpretation and illustration of some of the events, in some cases the nasal pressure signal was processed to provide a volume signal, by integration of the square root of the pressure signal [2].

The primary aim of the recordings had been to evaluate the new monitoring device. For this reason, we wished to study a population that was truly representative of acutely ill patients. We sought, and were granted, ethical permission for the primary study on the understanding that some patients might not have full capacity to give permission. Because fully informed consent could not be assured from all the subjects, our national Ethical Committee restricted the data that we were permitted to collect, and no follow up was allowed. Consequently information about the patients is limited. In particular, the clinical progress of the patients could not be followed. The only clinical details available were those recorded by the nursing staff caring for the patient, which were the reasons for admission to hospital and known comorbidities. If required, significance testing for categorical data was done with the Chi squared test with a two tailed P value set to 0.05.

Results

Nasal pressure records could be analysed from 63 patients. The median respiratory rate was 25 (22, 29) (median, quartiles) breath.min⁻¹.

Patient Characteristics

The age of the patients was 61 (17) years (Mean, SD), and 35 out of the 63 were female. Many of the patients had one or more noted co-morbid conditions: 32 had cardiovascular disease (commonly hypertension, ischaemic heart disease, or cardiac failure): 19 had lung disease, predominantly chronic obstructive lung disease or asthma: 13 had clinically important renal impairment, and 11 were diabetic. Full details of height and weight were not available for all the patients. For the 51 patients with complete data, height was 1.66 (0.11) m and weight was 78 (17) Kg (Mean, SD). In these 51 patients, 17 had a body mass index greater than 30.

Features noted during expiration

In many records, the early part of expiration appeared to be influenced by muscle activity. This is a normal pattern, where the muscle action can be inferred from a progressive reduction of the rate of increase in flow after the onset of expiration. Expiratory flow thus reaches a rounded maximum, and is followed by a decrease that resembled an exponential decline (Figure 1, breaths 1 and 2).

However we noted that in some breaths in many patients, this pattern could vary. This variation could be explained by either absence of action, or poor co-ordination, of inspiratory and expiratory muscles. For example, the third breath in figure 1 shows an expiration which is very different from the breath cycle before or after. Expiration is longer, the flow rate is less and declines more gradually, and ends with a burst of expiratory flow, suggesting that only at this time in expiration were expiratory muscles active. At the start of the third expiration, flow appears to be passive, caused by offset of inspiratory muscle action, and the elastic recoil of the respiratory system. Examples of three breaths seen in another patient record are shown in figure 2.

In each of these samples, there appears to be a period without active expiration. I conclude (see discussion for further explanation) that these abnormal patterns result from a disturbance of the stereotypical pattern, normally generated by combination of declining inspiratory muscle action, and active expiratory muscle action. Obvious changes in inspiratory and expiratory flow patterns were frequent in some patients, suggesting variable contributions from inspiratory, post inspiratory, and expiratory muscle activity, as shown in figure 3.

In many patients, an abrupt transition from expiration to inspiration was noted before expiratory flow ceased. I classified this feature as "early inspiration". It was found in recordings from 52 patients (82%). Figure 4 shows a typical example. In this patient, the median respiratory rate was 26 breath.min⁻¹. However in all patients with evidence of early inspiration, the respiratory rate was 20 (17, 25) (median, quartiles) breath.min⁻¹, which was not significantly greater than the rate in those patients in whom early inspiration was not noted.

In patients with slower respiratory rates, a plausible explanation for "early inspiration" could be flow limitation. In figure 5, a patient with a low respiratory rate (18 breath.min⁻¹) has a long expiratory phase in breaths 1 and 2, followed by a deep inspiration.

Lower record

The pressure signal has been linearised to approximate flow (see methods) and then integrated to indicate lung volume changes associated with breaths numbered 2 to 5. After a pause, breath 3 is substantially greater than breath 2. Expiration slows progressively and lung volume does not return to FRC before the onset of breath 4. After breath 4, which is smaller than the preceding breath, lung volume returns to FRC.

I estimated lung volume (relative to functional residual capacity (FRC)) by integrating the square root of the nasal flow signal, taking the pressure at the end of the first breath as zero flow, and assuming that lung volume at this point was FRC. Although the durations of expiration for breaths 1 and 3 are

very similar, the inspired flow and tidal volume of breath 3 are more than twice as large. The first part of expiration of breath 3 exhales about 50% of the tidal volume, and in the later part of expiration, the flow decrease is exponential, suggesting a passive elastic process. Expiratory flow continues to decline but can be detected until the onset of the next inspiration (breath 4). After breath 4 the lung volume returns to FRC. The pattern of passive expiration (an exponential decay) ended by an active inspiration can only be clearly recognised in patients with longer expiratory periods. Thus it was not possible, in many cases, to attribute “early inspiration” specifically to incomplete expiration, as in this example, or to early onset of inspiration illustrated in figure 4.

Less commonly (in 39 (62%) of the patients) I found some occasions where expiration continued after the preceding tidal volume had been exhaled. After a large expiration of this sort, the respiratory system was noted to subsequently fill passively (i.e. passive inspiration) before active inspiration started. In patients that showed this feature, such events were generally less common, and smaller, than passive expiration. An example of this feature is shown in figure 6.

Lower record: The pressure signal has been linearised to approximate flow (see methods) and integrated to indicate lung volume changes. This example shows two similar breaths (1, 2) followed by a large inspiration (3I). The expiration that follows is passive, and prolonged (labelled “out”). It ends with two episodes of active expiration (E’), and then a further sudden inspiration. After the two active expiratory efforts, lung volume is less than the passive elastic balance position, so that passive inspiratory flow occurs after these expiratory events (labelled “in”)

Abnormal breath cycles

In some patients, the regular coordination of inspiration and expiration was disturbed. This was seen in several forms. In some patients, active expiration was either substantially delayed (Figures 1 and 7, lower panel) or absent (Figure 7, upper panel).

More frequently, as already described, expiration was interrupted (Figure 2). Usually, but not always, interruption of the regular breathing patterns was preceded by a deeper breath. Delayed or absent active expiration was noted in 40 patients (63%). In 43 patients I observed sequences of several acts of inspiration or expiration alone, without a full respiratory cycle taking place. Examples are shown in figure 8.

Cyclical changes in breathing amplitude

Waxing and waning of the flow rates were seen in 5 (8%) of the patients, indicating variations in ventilation. The variations in maximum amplitude in different patients varied from subtle to very prominent. Only when the changes were prominent was there an associated change in respiratory frequency (Figure 9).

Breath timing

The duration of the breathing cycles was measured in parts of the recordings when this was feasible for substantial periods, i.e. when breathing was steady and there were no artefacts

caused by moving, coughing, swallowing, or cannula displacement. The number of breaths analysed for each patient were 630 (367,1286) (median, quartiles). There was a considerable range in the median duration of the respiratory cycle between the patients. However within each patient the variation of breath duration was limited, with the interquartile range of breath durations generally less than 1 second. In those patients with longer breath cycles (smaller breathing frequencies), there was greater variation in breath cycle duration (Figure 10).

Discussion

There was no prior intention to make these specific observations, and these findings can only be considered descriptive and exploratory. The recordings were made for another specific purpose, to measure respiratory rate. To provide a valid assessment, we had chosen a convenience sample of acute medical admissions, which would be the conditions in which the device would be useful. Respiratory rate assessment appears to accurately identify severe disease in acutely ill patients [3].

The extent of ethical permission allowed for our study was restricted substantially, since fuller medical details and follow up of the patients was not considered necessary for the limited purpose of the study. Clearly, the observations we report here would have greater meaning if they could have been combined with additional information from clinical examination, the results of other investigations, and with outcome, or if we had the opportunity to repeat the recordings when the patient had improved.

Nevertheless, the findings are remarkable for the incidence, range and degree of abnormalities that could be detected. The contrast in information gain, compared with visual inspection of the chest for a few breath cycles, is analogous to looking at an ECG to assess a cardiac rhythm, in contrast to mere palpation of the arterial pulse.

Features of expiratory flow

Tidal airflow patterns have been used for clinical evaluation of patients with respiratory disease, and specific features of the pattern can be related to standard measures such as forced expiratory volume in 1 second. Morris et al. [4] investigated patients referred for assessment with airway obstruction. During breathing via a mouthpiece, they observed that in patients with airway obstruction, expiration ended with a sudden decrease to zero flow. They suggested that the feature was a useful indication of airway obstruction in patients who could not cooperate in more complex tasks, and devised some methods to quantify how expiration was curtailed. I found that similar observations are possible without the need for mouthpiece breathing, and can be made in the acute setting.

Commonly, normal expiration is considered a passive process, with flow generated by the elastic recoil of the respiratory system when the inspiratory muscles relax. However, studies showed that in normal subjects, breathing via a mouthpiece, inspiratory muscle activity in both the diaphragm [5] and the intercostal muscles [6], declines gradually during expiration. The extent of persistent inspiratory activity (PIA), in both magnitude and duration, varies substantially from subject to subject, and also within subjects [7]. The effect of this activity

is to “brake” the expiratory flow, and it is reduced when factors such as mechanical loads to inspiration are present [8]. In addition the larynx can exert a “braking” effect, described both in normal quiet breathing [9] and during bronchoconstriction [10].

Early studies of expiration considered that the force generating expiration was passive elastic recoil of the respiratory system, after it had been displaced from its relaxation position by the inspiratory muscles. More recent studies of patients with COPD found that the transversus abdominis (not an easy muscle to study) was active in many subjects with airway obstruction even during quiet breathing [11]. These studies are supported by other studies of respiratory mechanics, using measurements of gastric pressure and abdominal dimensions [12]. Even in normal subjects during exercise, expiratory activity of the abdominal muscles increases [13].

My observations of some expiratory patterns (Figures 1, 2, 7 and 8) show that expiratory flow is very different from the usual. These abnormal patterns of expiration have a simple exponential decrease, as would be expected from a passive elastic process, where a semi logarithmic plot of nasal pressure against time would be linear. However the time constant of the decline is long, about 3 seconds, which is much greater than predicted from the elastance and resistance of the respiratory system. In normal subjects this would be about 0.5 seconds (assuming a compliance of 0.5 litre.kPa⁻¹, and a resistance of 1 kPa. l-1. sec-1) In patients with COPD, this time constant would be greater, because both resistance and compliance are greater. These observations support studies in patients with complete neuromuscular block, where passive expiration could be separated into two processes, the first, rapid process generated by passive elastic recoil, and the second slow process with a time constant of about 3 seconds, probably representing the viscoelastic relaxation of the lung and chest wall [14]. One puzzle remains: how is it possible for the first process of passive emptying to be almost absent from some of the observed abnormal expiratory periods? Figure 1 and figure 3, top trace, show almost only “slow flow”. The only explanation I can suggest for the absence of substantial initial expiratory flow is that after the preceding inspirations (which appear relatively normal) that persistent inspiratory activity has also been present as normal. Although expiratory activity has been suppressed in at least part of the expiratory cycle, the preceding inspiration, with an element of persistent inspiratory activity, is still present, which could prevent a rapid onset of entirely passive expiration.

In obstructive airway disease, dynamic airway collapse during forced expiration is a principal mechanism of the flow limitation and a hallmark of this disease. In severe disease, flow limitation can occur during tidal breathing. Flow limitation can be detected by applying a negative pressure to the mouthpiece during expiration: if flow is not limited, it increases when the additional pressure gradient is applied [15]. In patients with severe COPD, flow limitation is frequent during quiet breathing, especially when the patient is supine [16]. Flow limitation is frequent in the elderly, it is often associated with dyspnoea, [17] and has also been detected in obese patients [18].

A frequent consequence of flow limitation is incomplete expiration. If expiration has been incomplete, greater inspirato-

Our observations support those of Morris [4] and Bake,[19] who both reported that in many patients expiratory flow ceases abruptly (Figure 4) and suggest that flow limitation is responsible for the residual flow. In contrast, expiration in normal subjects is frequently, but unpredictably, close to complete, before the onset of the next inspiration [19]. In the patients studied, this pattern was infrequent, and when it was present, it was noted in association with a greater and more variable respiratory period, such as the pattern previously associated with opioid medication [20].

Some of these disturbances in breathing control, for example a sequence of several inspiratory or expiratory attempts, indicate an unusual breakdown of the normal coordination of the phases of breathing, which is usually considered to be set by ponto-medullary control processes. We can only speculate, but suggest that observing such features could prove clinically useful, for example in predicting incipient respiratory failure.

Conclusions

Nasal pressure measurement is simple to implement and observe, particularly when combined with a display method such as a smartphone. It illustrates abnormal respiratory mechanics and disturbances in the control of breathing, which could be of potential clinical value. Systematic observation and correlation with other clinical and pathological features would allow us to understand and appreciate information from this additional measurement, which could ultimately become a valuable clinical tool. The patterns described are easily recognised: not dissimilar those present in the ECG, which is part of routine clinical practice.

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References

1. Guyatt AR, Parker SP, McBride MJ. Measurement of human nasal ventilation using an oxygen cannula as a pitot-tube. *Am Rev Respir Dis.* 1982; 126: 434-8.
2. Montserrat JM, Farre R, Ballester E, et al. Navajas, Evaluation of nasal prongs for estimating nasal flow. *Am J Respir Crit Care Med.* 1997; 155: 211-5.
3. Cuthbertson BH, Boroujerdi M, Prescott G. The use of combined physiological parameters in the early recognition of the deteriorating acute medical patient, *J R Coll Physicians Edinb.* 2010; 40:19-25.
4. Morris MJ, Lane DJ. Tidal expiratory flow patterns in airflow obstruction. *Thorax.* 1981; 36; 135-42.
5. Agostoni E, Sant’Ambrogio G, del PH Carrasco. Electromyography of the diaphragm in man and transdiaphragmatic pressure. *J Appl Physiol.* 1960;

- 15: 1093-7.
6. Green JH, Howell JBL. The correlation of intercostal muscle activity with respiratory air flow in conscious human subjects. *J Physiol.* 1959; 149: 471-6.
 7. Shee CD, Ploy-Song-Sang Y, Milic-Emili J. Decay of inspiratory muscle pressure during expiration in conscious humans. *J Appl Physiol.* 1985; 58:1859-65.
 8. Martin J, Aubier M, Engel LA. Effects of inspiratory loading on respiratory muscle activity during expiration. *Am Rev Respir Dis.* 1982; 125: 352-8.
 9. England SJ, Bartlett DJ, Daubenspeck JA. Influence of human vocal cord movements on airflow and resistance during eupnea. *J Appl Physiol.* 1982; 52:773-9.
 10. Collett PW, Brancatisano T, Engel LA. Changes in the glottic aperture during bronchial asthma. *Am Rev Respir Dis.* 1983;128: 719-23.
 11. De Troyer A, Estenne M, Ninane V, et al. Transversus abdominis muscle function in humans. *J Appl Physiol.* 1990; 68: 1010-6.
 12. Martinez FJ, Couser JI, Celli BR. Factors influencing ventilatory muscle recruitment in patients with chronic airflow obstruction. *Am Rev Respir Dis.* 1990; 142: 276-82.
 13. Dodd DS, Brancatisano T, Engel LA. Chest wall mechanics during exercise in patients with severe chronic air-flow obstruction, *Am Rev Respir Dis.* 1984; 129: 33-8.
 14. Chelucci GL, Brunet F, Dall'Ava-Santucci J, et al. A single-compartment model cannot describe passive expiration in intubated, paralysed humans. *Eur Respir J.* 1991; 4: 458-64.
 15. Koulouris NG, Valta P, Lavoie A, et al. A simple method to detect expiratory flow limitation during spontaneous breathing. *Eur Respir J.* 1995; 8: 306-13.
 16. Eltayara L, Becklake MR, Volta CA, et al. Relationship between chronic dyspnea and expiratory flow limitation in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med.* 1996; 154: 1726-34.
 17. de Bisschop C, Marty ML, Tessier JF, et al. Expiratory flow limitation and obstruction in the elderly. *Eur Respir J.* 2005; 26: 594-601.
 18. Pankow W, Podszus T, Gutheil T, et al. von Wichert, Expiratory flow limitation and intrinsic positive end-expiratory pressure in obesity. *J Appl Physiol.* 1998; 5: 1236-43
 19. Bake B, Houlzt B, Sjolund P, et al. High tidal end expiratory flow- an index of dynamic hyperinflation?, *Clin Physiol Funct Imaging.* 2007; 27: 116-21.
 20. Drummond GB, Bates A, Mann J, et al. Characterization of breathing patterns during patient-controlled opioid analgesia. *Br J Anaesth.* 2013; 111: 971-8.

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