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Non-invasive ventilation: Essential requirements and clinical skills for successful practice

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ABSTRACT

Audits and case reviews of the acute delivery of non-invasive ventilation (NIV) have shown that the results achieved in real life often fall short of those achieved in research trials. Factors include inappropriate selection of patients for NIV and failure to apply NIV correctly. This highlights the need for proper training of all involved individuals. This article addresses the different skills needed in a team to provide an effective NIV service. Some detail is given in each of the key areas but it is not comprehensive and should stimulate further learning (reading, attendance on courses, e-learning, etc.), determined by the needs of the individual.

Key words: communication skills, education, non-invasive ventilation, physiology, training.

INTRODUCTION: WHY IS THIS ISSUE IMPORTANT?

Non-invasive ventilation (NIV) has become part of routine care for patients with acute respiratory failure.[1,2] It should usually be the first-choice modality of ventilatory support for patients with acute-on-chronic hypercapnic respiratory failure, particularly due to an acute exacerbation of chronic obstructive pulmonary disease (AECOPD). Although lacking the same evidence base, in terms of randomized controlled trials (RCT), domiciliary NIV is well established in the management of patients with chronic respiratory failure due to chest wall deformity, neuromuscular diseases, AECOPD and obesity. It can also be used in hypoxic respiratory failure, but usually only in a high-intensity, intensive care environment, with careful monitoring and rapid recourse to invasive ventilation if the patient is deteriorating.

The excellent results obtained in clinical trials are often not seen in everyday clinical practice. Audit is uncommon, but when it is done the results raise significant concerns. However, concerns have also been raised about the way that NIV is delivered in routine practice. In a 2008 national audit of the acute care, including NIV, of patients admitted to hospital with AECOPD,[3] a total of 232 hospital units collected data on 9716 patients. Disappointingly, acidotic patients receiving NIV had a worse prognosis than acidotic patients who did not receive NIV. Contrary to guidelines, 42% of patients with progressive or acute respiratory acidosis did not receive NIV. Use of oxygen with an unrestricted fractional inspired oxygen concentration (FIo_2) was common and associated with the need for ventilation (22% of the patients who received >35% oxygen vs 9% of those who received <35% oxygen) and mortality (11.1% of the patients who received >35% oxygen vs 7.2% of those who received <35% oxygen). National audits were repeated by the British Thoracic Society (BTS) in consecutive years from 2010 to 2013.[4] These showed high mortality rates. There are a number of potential explanations for this including exclusion of high-risk patients in most RCT and NIV being offered to patients with end-stage disease for whom there is little chance of success. However, the results suggest that poor patient selection rather than inappropriate technical application of NIV is the main problem explaining worse outcomes in these audits compared to the RCT.

The National Confidential Enquiry into Patient Outcome and Death (NCEPOD) in the UK undertook a review of the delivery of acute NIV in AECOPD.[5] This report raised several major concerns about the way in which NIV is delivered and lessons learnt from this are the subject of a recent review.[6] Particular concerns raised were:

1. Patient selection: ward-based NIV is not recommended for patients with pneumonia but consolidation was present in 40% and when present mortality was higher.
2. Possible treatment delay: delay in starting NIV was identified in 27.4% of patients
3. Location of NIV delivery: despite the fact that median pre-NIV pH was 7.24, 91% of patients were not treated in a high dependency unit and mortality was highest for patients who started NIV in general medical wards (59%)
4. High mortality rates and low rates of intubation if NIV failed.

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In most patients the decision to start NIV in AECOPD was made by a non-specialist (65% of patients). In 18.8% of patients the use of NIV was considered inappropriate. The quality of NIV was considered good in 27.5%, adequate in 48.5%, poor in 19.6% and unacceptable in 4.4%. In 33%, monitoring was considered not to be frequent enough. These data confirm that training of *all* clinical staff working in the acute sector in how to select the *right patient* for NIV, to apply it at the *right time* and in the *right way* is crucial. The same principles apply with regard to the provision of long-term home ventilation.

WHAT SKILLS ARE NEEDED TO BECOME AN NIV EXPERT?

As with any practical intervention, the skill of the operator and knowledge of when it is appropriate to use the intervention, and when it is not, is key to a successful outcome. Although training and competency assessment are frequently recommended, there is little published and no internationally agreed competency framework. Indeed, the NCEPOD review[5] found that in 45.4% of hospitals staff without a defined competency directly supervised patients on NIV. In this paper, we explore the skills needed for a clinician to be a successful NIV operator, both in the acute environment and in chronic home care. These are summarized in Figure 1.

AN UNDERSTANDING OF PHYSIOLOGY IS IMPORTANT

Respiratory failure results when there is an imbalance between the load against which the respiratory muscle pump must act and/or the drive to the system. It is important to understand how the load/drive/capacity inter-relationships of the respiratory system might be affected by different conditions and the implications of this both for standard medical therapy and also for how a ventilator should be used in the management of respiratory failure. A schema for how respiratory failure can develop in a patient with COPD is shown in Figure 2. It can be seen that there are a number of potentially interrelating vicious cycles and explains how respiratory failure can evolve rapidly in the at-risk patient, and also how interrupting one downward spiral and replacing it with a virtuous cycle can also lead to a rapid improvement. For example, a reduction in respiratory rate, by offloading the inspiratory muscles means more time for expiration, which leads to a reduction in hyperinflation, which in turn improves the mechanical advantage of the respiratory muscles of the inspiratory muscles, leading to an improvement in the capacity of the respiratory muscle pump and a further reduction in respiratory rate. Alveolar ventilation improves, bringing the pH back to normal, thereby removing the deleterious effect of acidosis upon muscle function, with a further improvement in capacity. There are however factors which will not respond directly to ventilation (e.g. secretions and bronchoconstriction), emphasizing the need for maximal medical therapy, in addition to ventilatory support (it is however pertinent to note that effective NIV may help patients e.g. with cystic fibrosis, to cooperate with physiotherapy[7–9] and therefore clear secretions more effectively; also positive pressure may have a direct effect upon the airways themselves[10]).

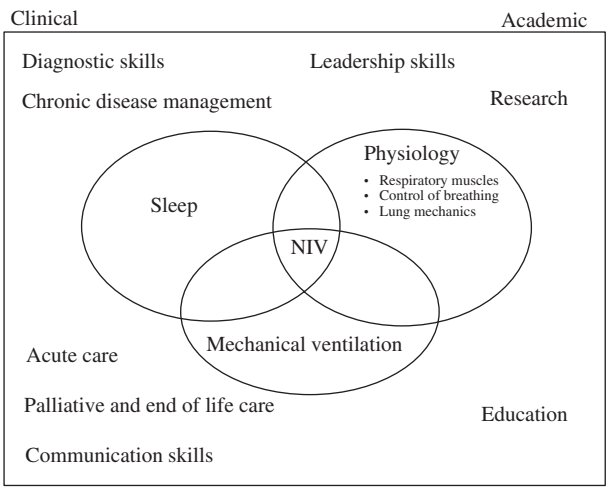

Figure 1 A Venn diagram of the integrated skills and knowledge required for non-invasive ventilation (NIV).

Contrast this with a patient with motor neurone disease (MND), both medical management and the way the ventilator is set will be very different. There is no role for bronchodilators, but secretion management is much more important. Because lung compliance is not increased low inflation pressures generally suffice, but external positive end expiratory pressure (PEEP) is required to recruit atelectatic lung at the lung bases, which has occurred as a result of extrapulmonary restriction due to the weak inspiratory muscles.

It is a useful exercise to work out how load, drive and capacity can be impacted by any condition requiring NIV and then work out the implications of this for both the way that the ventilator should be set and what other aspects of therapy are important. Table 1 shows the relative contribution of each component in two conditions in which NIV is commonly used.

Together with an understanding of respiratory physiology, knowledge of how a ventilator works and interacts with abnormal patient physiology is vital. First, it is important to understand the nomenclature for different types of ventilator as unfortunately there is no agreed standardization of ventilator modes and refinements with different terms and acronyms being used to describe the same thing. A detailed discussion of all the different types of ventilators and their nomenclature is beyond the scope of this article but it is key that the operator should understand how the ventilator(s) they are using works.

An understanding of the way that the ventilator transitions between inspiration and expiration is important as the respiratory cycle of the machine should mirror that of the patient, to ensure both comfort and optimal ventilatory support. The machine blowing air in when the patient is trying to breathe out is uncomfortable and inefficient.

Timing of ventilation can be set by the operator ('controlled' or 'timed') or determined by the patient,

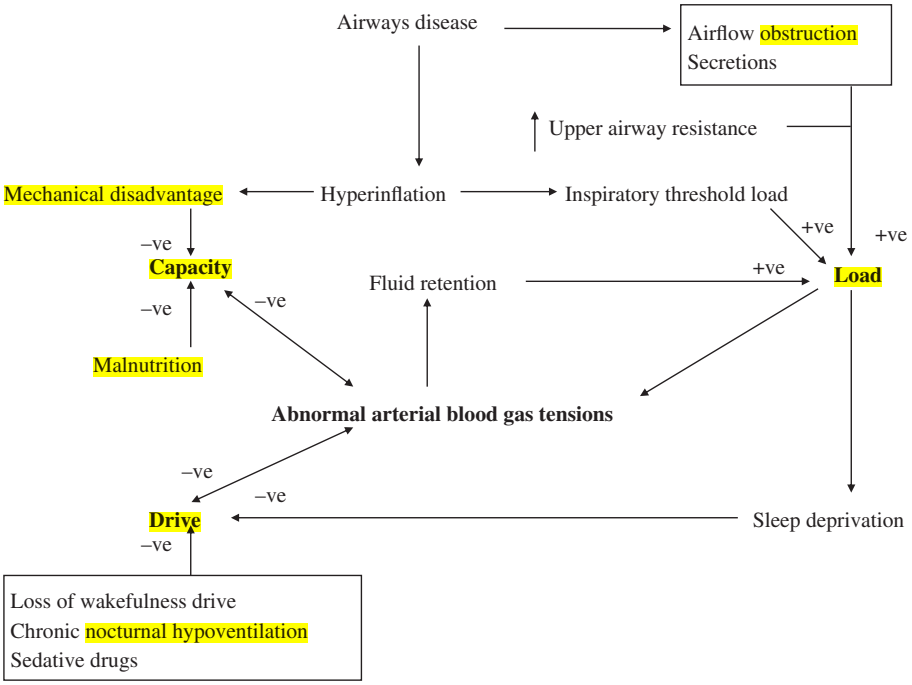

Figure 2 The inter-relationships between load, drive and capacity in COPD and how vicious, and virtuous, cycles may develop.

'spontaneous' or 'assisted' ventilation. The ventilator 'triggers' into inspiration and 'cycles' into expiration based upon changes in airflow. A failure to trigger into inspiration may be either because the patient cannot generate sufficient inspiratory airflow, that is a patient with severe muscle weakness, or because of intrinsic PEEP in COPD; the initial part of the inspiratory effort is spent overcoming positive pressure in the alveoli with inspiratory airflow only occurring when alveolar pressure drops below the pressure at the mouth/nose. The answer to ineffective triggering is different. In a patient with neuromuscular disease, it is particularly important to have the trigger set as sensitive as possible. In a patient with COPD, appropriate setting of extrinsic PEEP (or expiratory positive airway pressure (EPAP)) improves effective trigger sensitivity, patient ventilator synchrony and also leads to a greater reduction in the work of breathing than with pressure support alone.[11] The ventilator cycles into expiration when, usually, inspiratory airflow falls to a percentage of peak; if there is excessive leak airflow is maintained and inspiration, as determined by the ventilator, continues even though the patient has started to breathe out.[12] Again, this is uncomfortable and inefficient.

DIAGNOSTIC SKILLS

NIV is indicated when the patient develops respiratory failure/sleep-related hypoventilation. Diagnostic skills and an understanding of the natural history of a condition are important both in the chronic situation to identify patients at risk for hypoventilation proactively and to institute NIV before there is an acute crisis, and also when patients present acutely to identify correctly why they have respiratory failure. Although it is often obvious, misdiagnosis occurs in patients with a heavy smoking history misdiagnosed with COPD, when the respiratory failure is actually due to obesity or undiagnosed neuromuscular disease (e.g. MND),[13] or the patient does have COPD, but it is not sufficiently severe to explain the respiratory failure which is caused by something else.

It is important that patients receive appropriate medical therapy; this may improve symptoms, but occasionally may obviate the need for NIV (for instance, appropriate diagnosis and treatment of myasthenia gravis). When attempting to transition a patient from invasive to NIV after an acute episode, a clear understanding of what is causing the respiratory failure and then correcting that which is reversible leads to the best chance of getting a patient on to the least ventilatory support possible. This includes no support; the less the better in terms of patient, and very importantly family, quality of life, independence and cost. Figure 3 indicates some of the factors to consider. A multidisciplinary approach involving doctors, nurses, physiotherapists, speech and language therapists, dietician, psychologist, etc. is key. Involvement of other specialists (e.g. neurologist and cardiologist) is also important. If in doubt involve others.

WHY SOME KNOWLEDGE OF SLEEP MEDICINE IS IMPORTANT

Domiciliary NIV is usually applied during sleep and therefore some knowledge of the physiology of sleep is important. For the patient at risk of developing chronic respiratory failure, abnormalities first become apparent during rapid eye movement (REM) sleep, followed by non-REM sleep and ultimately with the development of daytime respiratory failure. Transient hypoventilation leads to a rise in arterial carbon dioxide tension ($PaCO_2$) which in turn causes acidosis. The kidneys respond by retaining bicarbonate to return pH to

Table 1 An analysis of primary and secondary effects on load, drive and capacity in two common conditions and the implications for NIV

	Consequences	Load	Drive	Capacity
Motor neurone disease	Primary	Unchanged	Unchanged	Reduced because of muscle weakness
	Secondary	Increased due to excessive secretions Increased during sleep due to upper airway obstruction Increased due to atelectasis	Reduced due to injudicious use of respiratory depressant drugs Reduced as a consequence of bicarbonate retention due to NH	Reduced due to poor nutrition
	Implications for NIV	EPAP to recruit atelectatic lung NIV prior to physiotherapy EPAP during sleep to prevent UAO	Control of NH (reduced CO_2) restores central drive	NIV provides the ventilation which the weakened respiratory muscles cannot achieve—low levels of IPAP usually suffice Trigger set to as sensitive as possible to ensure that triggering occurs. If inspiratory muscles too weak, ensure back up rate set appropriately
COPD	Primary	Increased due to airways obstruction and the intrinsic threshold load due to PEEPi	Unchanged	Reduced due to mechanical disadvantage of inspiratory muscles
	Secondary	Increased due to excessive secretions Increased during sleep due to upper airway obstruction	Reduced due to injudicious use of respiratory depressant drugs Reduced as a consequence of bicarbonate retention due to NH	Reduced due to poor nutrition and steroids
	Implications for NIV	Adequate EPAP to counterbalance PEEPi Higher IPAP reduces airways resistance EPAP during sleep to prevent UAO	Control of NH (reduced CO_2) restores central drive	NIV provides the ventilation which is reduced due to the combination of increased load and reduced capacity—higher levels of IPAP usually required

EPAP, expiratory positive airway pressure; IPAP, inspiratory positive airway pressure; NH, nocturnal hypoventilation; NIV, non-invasive ventilation; PEEPi, intrinsic positive end expiratory pressure; UAO, upper airway obstruction.

normal, which leads to a blunting of the central chemosensitivity to CO_2, but also manifests in a rise in the standard bicarbonate and base excess. An elevated bicarbonate and base excess, in the absence of hypokalaemia, suggests nocturnal hypoventilation (NH) and should prompt appropriate monitoring during sleep. The symptoms of NH are very non-specific and it is important to have a high index of suspicion in at-risk individuals; in particular, these should include all patients with neuromuscular disease and chest wall deformity.

Full polysomnography is not required to diagnose NH. Pulse oximetry, provided that the patient is not receiving supplemental oxygen, provides very useful information and has the great advantage of simplicity and low cost.[14] It can easily be performed in the patient's home, over several nights, with equipment sent out and returned by post. End-tidal CO_2 ($EtCO_2$) monitoring is cheaper and easy but should *never* be used as a measure of $PaCO_2$ in a spontaneously breathing patient with lung disease. It is even less reliable in patients receiving NIV, because of mixing of gases within the mask and the nasopharynx.[15] Transcutaneous CO_2 ($TcCO_2$) monitoring using modern monitors does accurately reflect $PaCO_2$, with the sensor preferentially applied to the ear lobe[16] and can be used for monitoring the effectiveness of NIV, but the equipment is significantly more costly and will usually require an overnight stay in hospital. It can also be used in the acute situation and for weaning.[17,18]

In some patients, more detailed respiratory variable sleep studies are required to disentangle the relative contribution of upper airway obstruction and sleep-related hypoventilation to the development of respiratory failure. It is important to understand how each may lead to hypercapnia (Fig. 4). During an obstructive apnoea, $PaCO_2$ rises but is usually returned to normal by the brief hyperventilation that accompanies arousal. However, if the apnoea is prolonged, or the increase in ventilation with arousal is attenuated for instance due to coexistent respiratory muscle weakness or severe obesity, more CO_2 is retained during the apnoea than can be excreted

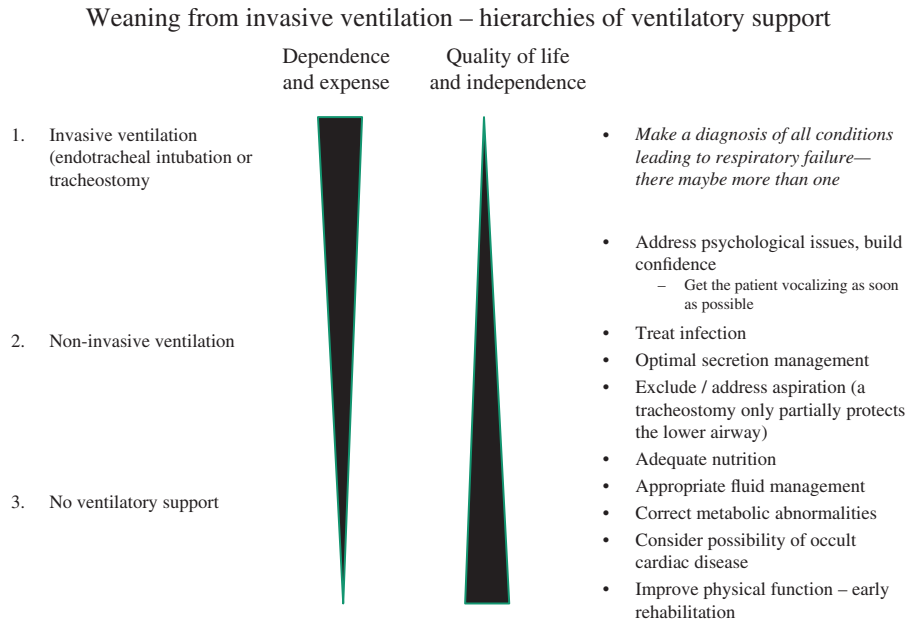

Figure 3 Factors to consider when transitioning a patient from invasive ventilation to non-invasive ventilation or no ventilatory support.

with arousal, $PaCO_2$ rises and acidosis develops which stimulates renal retention of bicarbonate (see above). This is best described as hypercapnic obstructive sleep apnoea (OSA); if the upper airway is controlled, using CPAP, there will be no carbon dioxide retention— a reduction in apnoea–hypopnoea index (AHI), easily obtainable from machine's built-in software is the appropriate therapeutic target. This probably explains why in

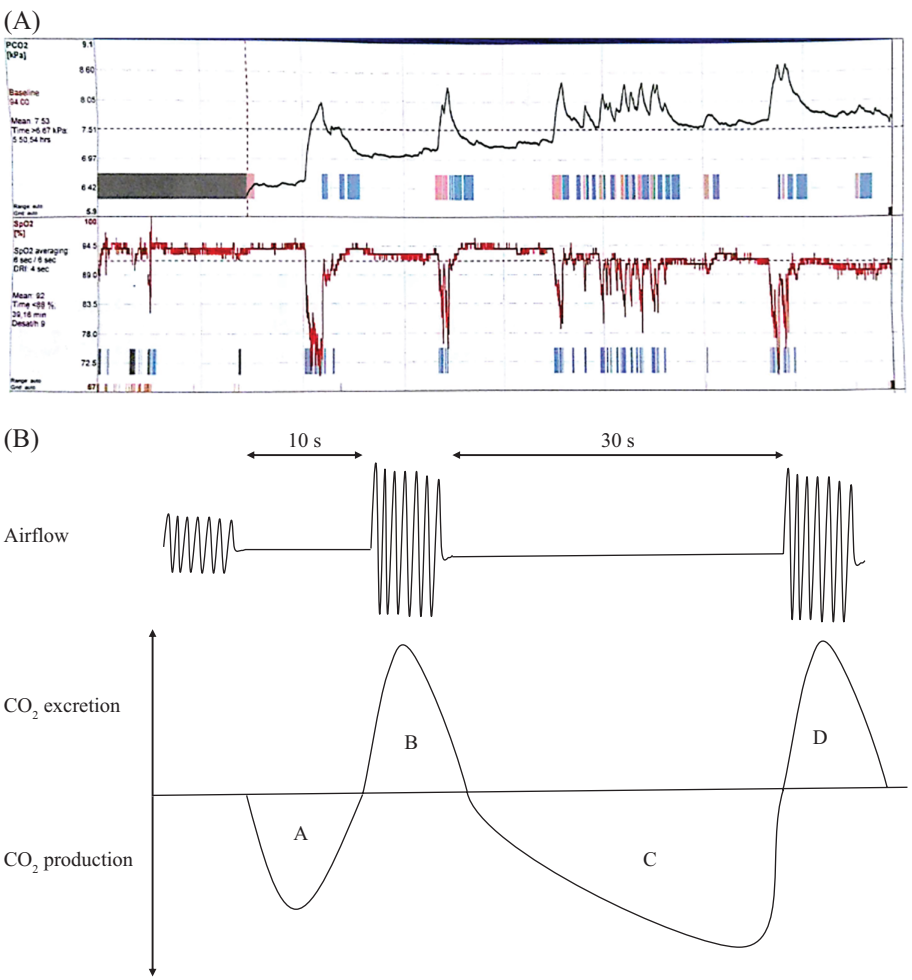

Figure 4 (A) Transcutaneous CO_2 ($TcCO_2$) and oxygen saturation recorded over night. The $TcCO_2$ rises progressively through the night with transient peaks, associated with more pronounced oxygen desaturation occurring during episodes of rapid eye movement (REM) sleep. (B) During normal breathing, CO_2 excretion by the lungs equals metabolic CO_2 production. The CO_2 retained during a 10-s apnoea can be excreted during the arousal-associated hyperpnoea (A + B). However, during a more prolonged apnoea, the period of hyperventilation is not sufficient to excrete the CO_2 produced during the apnoea (C > D). $PaCO_2$ will rise over the night with repeated prolonged apnoeas.

Table 2 The key importance of NH: Know in which patients to look for it, how to record it and to ensure that NIV is being delivered effectively

	Skills needed
I. Identify that patient at risk for NH	1. An understanding of natural history of conditions which may lead to NH 2. Recognition of symptoms of NH 3. Ability to measure, and interpret, spirometry, mouth and sniff pressures 4. Interpretation of blood gas measurements, in particular appreciate the importance of a raised HCO_3 or BE
II. Identify NH in the patient	1. Understand the role and limitations of monitoring techniques during sleep 2. Learn to recognize different patterns of over night oximetry
III. Ensure that ventilation being delivered effectively during sleep and if not what adjustments are required	1. Understand how to interpret data provided by (most) ventilators' software 2. Learn to recognize different patterns of overnight oximetry on NIV and when more detailed respiratory variable sleep studies are needed

BE, base excess; NH, nocturnal hypoventilation; NIV, non-invasive ventilation.

patients with obesity hypoventilation syndrome, even with significant hypercapnia (mean 60 mm Hg) at baseline CPAP can improve the CO_2 to the same degree as with NIV.[19] In contrast, with hypoventilation that occurs throughout sleep, but is most marked in REM sleep, such as is seen in patients with neuromuscular disease, the CO_2 rises progressively over the night with the most marked rises seen during REM sleep. For these patients, an increase in ventilation breath-by-breath is required and NIV is needed to correct respiratory failure. The target parameter now is the CO_2 overnight; the AHI is easily obtained from the ventilator but a low AHI does not mean that the therapy is being delivered appropriately. The difference between hypercapnic OSA and NH can be usually determined from the pattern of oximetry. For OSA alone to cause hypercapnia, it will always be severe and easily diagnosed by oximetry alone.

To ensure that NIV is being applied effectively, it is very important to monitor the effect of ventilation during sleep and sometimes the way that the patient is interacting with the ventilator. A reduction in carbon dioxide is the most important end point and this is best monitored continuously using a modern transcutaneous monitor. The aim of NIV is ideally to normalize $TcCO_2$ or at the very least to bring about an improvement. This will almost always bring about a substantial improvement in oxygen saturation. Provided that the FiO_2 is normal (i.e. NIV with no added oxygen), continuous monitoring of oxygen saturation is a useful surrogate marker for improved ventilation. If *any* oxygen is added to the circuit, its use as a surrogate for improved ventilation will be lost.

The SOMNO NIV group[20–23] has suggested how the delivery of NIV should be monitored. This starts with an understanding of how ventilators work and in particular how different ventilator modes impact upon monitoring systems. Information available from the ventilator is very helpful. A failure to use NIV at all may explain no change in symptoms, daytime blood gas tensions etc., and problems such as excessive leak, a high AHI etc., are easily obtained. This can then be supplemented with simple tools, such as oximetry etc., with respiratory polygraphy, and very occasionally full polysomnography, needed to troubleshoot problems during NIV. An algorithm is provided suggesting how the effectiveness of overnight NIV should be monitored.[20] This series is essential reading for any practitioner looking after patients on domiciliary ventilation. Table 2 highlights the steps in the recognition of NH and its successful control with NIV.

COMMUNICATION SKILLS

Clinicians delivering NIV will often be involved in difficult discussions with patients who have life-limiting conditions. This will be true both in domiciliary ventilation (e.g. discussions about tracheostomy) and in the acute situation (e.g. escalation to invasive ventilation or palliation). It is always easier to do something and as a clinician you are less likely to be criticized for doing something than not doing it. However, because you can does not mean you should; inappropriate use of a treatment which is futile can cause unnecessary suffering. For the patient to make a properly informed decision, they must be presented with accurate information in a way that they can understand. This requires an understanding of the natural history of a condition and the way this can be impacted by treatment. Sometimes, patients want everything done without realizing that there is a downside and that the intervention is not going to return them to a normal life, but may rather, at best, return them to their baseline in the recent past. Conversely, patients may have declined an intervention because of inappropriate pessimism or overemphasis of the negatives. In these situations, it is important to listen. In one study, doctors and patients rated how well a difficult discussion had gone; if the doctor spoke more their satisfaction was higher but that of the patients less. If the doctor spoke less, they thought the consultation had not gone so well whereas patient satisfaction was higher.[24] The clinician should not impose their values on their patients. Very disabled patients can still enjoy a good quality of life. Indeed in one study,[25] patients rated their quality of life better than that of the healthcare workers looking after them in some domains. The healthcare workers also underestimated the quality of life of the patients and overestimated the burden of ventilation.

LEADERSHIP SKILLS

NIV is a multidisciplinary endeavour involving a team. This requires leadership and while this will usually be a doctor, this is not always the case. It is important that someone takes final responsibility for decisions and actions and ensures that the whole team is functioning together appropriately. They should also ensure that staff members are adequately trained, competencies assessed and that the quality of delivery of the service is good. Some individuals are natural leaders, others will require specific training.

EDUCATION

Training is key to the successful implementation of any clinical skill. This is particularly true for NIV. It is relatively easy to access training as short courses are commonly provided. However, having been on a course does not mean that an individual is competent. There should be a formal assessment of competency (e.g. http://nipecportfolio.hscni.net/compro/ReadOnly/rCAT/view.asp?compID=72 or https://www.e-lfh.org.uk/programmes/acute-niv/). It is very important that any formal learning is supplemented by on-the-job training and refresher sessions. Simulators are also a useful resource (e.g. https://www.ers-education.org/e-learning/simulators.aspx).

Some clinicians will only be involved in NIV when covering on call etc., whereas others will be very actively involved on a day-to-day basis. It is very important to make a distinction between these two groups in terms of the skills that are needed. Training curricula are constantly expanding, as medical knowledge increases, and there is a tendency for each subspecialty to insist that all trainees have extensive knowledge within their niche area. As a result, trainees can end up as a 'jack of all trades and master of none'. It becomes impossible for any trainee to satisfy all these criteria with the result that competency assessment is diluted and to some extent becomes meaningless. The NIV community needs to establish the core competencies necessary to select patients for NIV and how it is applied acutely. The acute physician should know when NIV is indicated in longer term and whom to refer for more specialist assessment but a detailed understanding of long-term domiciliary NIV is not required. The practitioner only involved in chronic care must have some understanding of acute illness and its implications for NIV, as some of their patients will at times decompensate.

ACADEMIA AND CLINICAL RESEARCH

It will not be for everybody and opportunities are limited, but it is vital that some individuals working in the field are actively involved in academia and research. Although there is a lot that we now know and understand there is so much more that we do not. New techniques are evolving and it is very important that we understand the physiological basis and implications to ensure that they are implemented optimally. All clinical applications should, when possible, be underpinned by RCTs, which not only prove efficacy, but can also highlight, unexpected, important adverse effects, such as was seen in the SERVHF trial.[26] Even though most practitioners will not be actively involved in initiating clinical research opportunities for participation in clinical trials abound. Time spent during training in a research environment is important both for understanding the strengths and limitations of the research of others, and also giving a more detailed understanding of the field.

AUDIT AND QUALITY ASSURANCE

The assumption is generally made that any medical intervention is applied appropriately. However, audit frequently demonstrates that this is not the case and this is certainly true for NIV delivered acutely (see above). Any service should be subject to regular audit and clinicians need to understand the principles of audit. Unfortunately, comprehensive audit can be time-consuming and is often unfunded. Purchasers of healthcare services should insist on being provided with evidence that what they are paying for is being delivered appropriately and they should build funding for this into reimbursement. Although in many countries standards are provided for what is needed to deliver an acute NIV service, this is not always the case. That an organization is adhering to these standards is only confirmed by self-certification, often based on little hard evidence, with no peer review or quality assurance from an outside body.

With regard to domiciliary ventilation and weaning services, things are a little better with some countries having nationally agreed standards and certification of units, usually by the national respiratory society. Ideally, all services should be externally quality assured, however it is probably unrealistic to expect this as it is time-consuming and expensive. It is therefore incumbent on providers of NIV services to have an understanding of audit and quality assurance and to be able to document to their own satisfaction and to that of others that they are providing a quality service. Useful resources include the NCEPOD self-assessment checklist,[27] the BTS quality standards for acute NIV[28] and the NCEPOD audit toolkit (https://www.ncepod.org.uk/2017report2/toolkit/NIVauditTool.xlsx).

CONCLUSION

The successful NIV team needs to have individuals with many different skills. One person does not need to be able to do everything, but the team requires a named individual responsible for leadership and ensuring that all the issues are covered. Every clinician looking after patients with respiratory failure must be aware of which patients should receive NIV, when it should be started and ensure this happens without delay. Frontline staff delivering NIV must understand how to use the ventilator in different conditions and to trouble shoot when the patient is not improving. Training should be tailored to the individual. Competency must

Table 3 Key points and misconceptions

Always make a diagnosis—respiratory failure always has a cause(s)
End-tidal CO_2, while easy to measure, is unreliable and should not be used to monitor respiratory failure/assess adequacy of ventilation
Understand ventilator nomenclature and how the machines that are being used work. When moving a patient from one device to another, consider differences in how pressures are set, the machine triggers into inspiration and cycles into expiration
Complex, and expensive, monitoring during sleep is seldom necessary: a great deal can be done with an oximeter, a good history and some common sense
A tracheostomy only provides partial protection for the lower respiratory tract
Communication with patient and family—in difficult and/or sensitive situations, try to listen as much as you speak
Multidisciplinary working is very important—no professional group has all the necessary skills
Never stop learning and share what you know with others

be assessed and assured. Table 3 summaries key points and misconceptions.

Disclosure statement

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Abbreviations: AECOPD, acute exacerbation of COPD; AHI, apnoea–hypopnoea index; BTS, British Thoracic Society; EPAP, Expiratory positive airway pressure; FiO_2, Fractional inspired oxygen concentration; IPAP, Inspiratory positive airway pressure; MND, motor neurone disease; NCEPOD, National Confidential Enquiry into Patient Outcome and Death; NH, nocturnal hypoventilation; NIV, non-invasive ventilation; OSA, obstructive sleep apnoea; $PaCO_2$, Arterial carbon dioxide tension; PEEP, positive end expiratory pressure; PEEPi, Intrinsic positive end expiratory pressure; RCT, randomized controlled trial; REM, rapid eye movement; $TcCO_2$, transcuta-neous CO_2; UAO, Upper airway obstruction.

REFERENCES

1 Davidson AC, Banham S, Elliott M, Kennedy D, Gelder C, Glossop A, Church AC, Creagh-Brown B, Dodd JW, Felton T *et al.* BTS/ICS guideline for the ventilatory management of acute hypercapnic respiratory failure in adults. *Thorax* 2016; **71**(Suppl. 2): ii1–35.
2 Rochwerg B, Brochard L, Elliott MW, Hess D, Hill NS, Nava S, Navalesi P Members Of The Steering Committee, Antonelli M, Brozek J, Conti G *et al.*; Raoof S Members Of The Task Force. Official ERS/ATS clinical practice guidelines: noninvasive ventilation for acute respiratory failure *Eur. Respir. J.* 2017; **50:** pii: 1602426.
3 Roberts CM, Stone RA, Buckingham RJ, Pursey NA, Lowe D, National Chronic Obstructive Pulmonary Disease Resources and Outcomes Project Implementation Group. Acidosis, non-invasive ventilation and mortality in hospitalised COPD exacerbations. *Thorax* 2011; **66**: 43–8.
4 BTS audit reports 2010 to 2013. Available from URL: https://wwwbrit-thoracicorguk/publication-library/bts-reports/
5 The National Confidential Enquiry into Patient Outcome and Death (NCEPOD). 2017 Inspiring change. A review of the quality of care provided to patients receiving acute non-invasive ventilation. Available from URL: http://www.ncepod.org.uk/2017report2/downloads/InspiringChange_FullReport.pdf
6 Davies MG, Juniper MC. Lessons learnt from the National Confidential Enquiry into Patient Outcome and Death: acute non-invasive ventilation. *Thorax* 2018; **73**: 904–7.
7 Holland AE, Denehy L, Ntoumenopoulos G, Naughton MT, Wilson JW. Non-invasive ventilation assists chest physiotherapy in adults with acute exacerbations of cystic fibrosis. *Thorax* 2003; **58**: 880–4.
8 Rodriguez Hortal MC, Nygren-Bonnier M, Hjelte L. Non-invasive ventilation as airway clearance technique in cystic fibrosis. *Physiother. Res. Int.* 2017; **22**; https://doi.org/10.1002/pri.1667.
9 Rodriguez Hortal MC, Hedborg A, Biguet G, Nygren-Bonnier M. Experience of using non-invasive ventilation as an adjunct to airway clearance techniques in adults with cystic fibrosis-a qualitative study. *Physiother. Theory Pract.* 2018; **34**: 264–75.
10 De Backer L, Vos W, Dieriks B, Daems D, Verhulst S, Vinchurkar S, Ides K, De Backer J, Germonpre P, De Backer W. The effects of long-term noninvasive ventilation in hypercapnic COPD patients: a randomized controlled pilot study. *Int. J. Chron. Obstruct. Pulmon. Dis.* 2011; **6**: 615–24.
11 Appendini L, Purro A, Gudjonsdottir M, Baderna P, Patessio A, Zanaboni S, Donner CF, Rossi A. Physiological response of ventilator-dependent patients with chronic obstructive pulmonary disease to proportional assist ventilation and continuous positive airway pressure. *Am. J. Respir. Crit. Care Med.* 1999; **159**: 1510–7.
12 Calderini E, Confalonieri M, Puccio PG, Francavilla N, Stella L, Gregoretti C. Patient-ventilator asynchrony during noninvasive ventilation: the role of expiratory trigger. *Intensive Care Med.* 1999; **25**: 662–7.
13 Lad TS. An 'acute' presentation of motor neuron disease. *Acute Med.* 2011; **10**: 140–1.
14 Fu ES, Downs JB, Schweiger JW, Miguel RV, Smith RA. Supplemental oxygen impairs detection of hypoventilation by pulse oximetry. *Chest* 2004; **126**: 1552–8.
15 Sanders MH, Kern NB, Costantino JP, Stiller RA, Strollo PJ, Studnicki KA, Coates JA, Richards TJ. Accuracy of end-tidal and transcutaneous Pco2 monitoring during sleep. *Chest* 1994; **106**: 472–83.
16 Conway A, Tipton E, Liu WH, Conway Z, Soalheira K, Sutherland J, Fingleton J. Accuracy and precision of transcutaneous carbon dioxide monitoring: a systematic review and meta-analysis. *Thorax* 2018; https://doi: 10.1136/thoraxjnl-2017-211466.
17 van Oppen JD, Daniel PS, Sovani MP. What is the potential role of transcutaneous carbon dioxide in guiding acute noninvasive ventilation? *Respir. Care* 2015; **60**: 484–91.
18 Lermuzeaux M, Meric H, Sauneuf B, Girard S, Normand H, Lofaso F, Terzi N. Superiority of transcutaneous CO2 over end-tidal CO2 measurement for monitoring respiratory failure in non-intubated patients: a pilot study. *J. Crit. Care* 2016; **31**: 150–6.
19 Howard ME, Piper AJ, Stevens B, Holland AE, Yee BJ, Dabscheck E, Mortimer D, Burge AT, Flunt D, Buchan C *et al.* A randomised controlled trial of CPAP versus non-invasive ventilation for initial treatment of obesity hypoventilation syndrome. *Thorax* 2017; **72**: 437–44.
20 Janssens JP, Borel JC, Pepin JL; SomnoNIV Group. Nocturnal monitoring of home non-invasive ventilation: the contribution of

simple tools such as pulse oximetry, capnography, built-in ventilator software and autonomic markers of sleep fragmentation. *Thorax* 2011; **66**: 438–45.
21 Elliott MW. Non-invasive ventilation during sleep: time to define new tools in the systematic evaluation of the technique. *Thorax* 2011; **66**: 82–4.
22 Rabec C, Rodenstein D, Leger P, Rouault S, Perrin C, Gonzalez-Bermejo J; SomnoNIV Group. Ventilator modes and settings during non-invasive ventilation: effects on respiratory events and implications for their identification [Review]. *Thorax* 2011; **66**: 170–8.
23 Gonzalez-Bermejo J, Perrin C, Janssens JP, Pepin JL, Mroue G, Leger P, Langevin B, Rouault S, Rabec C, Rodenstein D. Proposal for a systematic analysis of polygraphy or polysomnography for identifying and scoring abnormal events occurring during non-invasive ventilation. *Thorax* 2012; **67**: 546–52.
24 Tulsky JA, Chesney MA, Lo B. How do medical residents discuss resuscitation with patients? *J. Gen. Intern. Med.* 1995; **10**: 436–42.
25 Bach JR, Campagnolo DI, Hoeman S. Life satisfaction of individuals with Duchenne muscular dystrophy using long-term mechanical ventilatory support. *Am. J. Phys. Med. Rehabil.* 1991; **70**: 129–35.
26 Cowie MR, Woehrle H, Wegscheider K, Angermann C, d'Ortho MP, Erdmann E, Levy P, Simonds AK, Somers VK, Zannad F *et al.* Adaptive servo-ventilation for central sleep apnea in systolic heart failure. *N. Engl. J. Med.* 2015; **373**: 1095–105.
27 NCEPOD self assessment checklist. Available from URL: http://www.ncepod.org.uk/2017niv.html
28 Davies M, Allen M, Bentley A, Bourke SC, Creagh-Brown B, D'Oliveiro R, Glossop A, Gray A, Jacobs P, Mahadeva R *et al.* British Thoracic Society quality standards for acute non-invasive ventilation in adults. *BMJ Open Respir. Res.* 2018; **5**: e000283.

