Framework for patient-ventilator asynchrony during long-term non-invasive ventilation

Jesus Gonzalez-Bermejo,[1,2] Jean-Paul Janssens,[3] Claudio Rabec,[4] Christophe Perrin,[5] Frédéric Lofaso,[6] Bruno Langevin,[7] Annalisa Carlucci,[8] Manel Lujan,[9] On behalf of the SomnoNIV group

ABSTRACT
Episodes of patient-ventilator asynchrony (PVA) occur during acute and chronic non-invasive positive pressure ventilation (NIV). In long-term NIV, description and quantification of PVA is not standardised, thus limiting assessment of its clinical impact. The present report provides a framework for a systematic analysis of polygraphic recordings of patients under NIV for the detection and classification of PVA validated by bench testing. The algorithm described uses two different time windows: rate asynchrony and intracycle asynchrony. This approach should facilitate further studies on prevalence and clinical impact of PVA in long-term NIV.

INTRODUCTION
Nocturnal non-invasive ventilation (NIV) is an effective treatment for chronic hypercapnic respiratory failure (CHRF), provided mainly overnight. Previous papers from our SomnoNIV group[1] suggested a systematic approach for monitoring nocturnal NIV and summarised the semiology of undesired events detected by polygraphy (PG) performed under NIV.[1] Patient-ventilator asynchrony (PVA) under NIV was not emphasised in these publications, although it has been extensively described in intensive care unit (ICU) patients, with its clinical consequences.[2] Over the past 10 years, PVA has also been reported in a small number of studies performed on chronic NIV patients, using polygraphic assessment.[3–5] PVA is often paucisymptomatic or asymptomatic. In a recent report, adding parasternal electroneuromyography (EMG) increased the prevalence of PVA to a remarkable 79% in 28 patients with obstructive and restrictive disorders during initiation of long-term NIV, but without any evidence of major physiological consequences.[6] There is therefore a lack of studies allowing an appropriate assessment of the prevalence and relevance of PVA in chronic NIV based on a consensual definition of events.

This report proposes a standardised systematic analysis and description of PVA in long-term NIV using pressure and flow tracings from an external pneumotachograph combined with abdominal and thoracic belts to facilitate its identification, management and associated clinical research.[3,4]

METHODS
Over a 2-year period, nocturnal PG tracings of patients during NIV were reviewed and thoroughly

discussed by a multinational expert group in work sessions focusing on PVA. Definitions, description, pathophysiological mechanisms and classification of PVA presented here are (1) the result of a consensus between all participants and (2) were reproduced on a bench test for reliability of description (see online supplementary file 1 for details). The reporting of PVA as described requires that leaks and residual upper airway obstruction have been dealt with and corrected (online supplementary files 1; 2).[1]

RESULTS
We found nine different PVA all confirmed by bench reproduction.

PVA classification was based on a visual analysis of PG tracings with two different time windows, leading to identification of (1) rate asynchronies and (2) intracycle asynchronies (figure 1).

Rate asynchronies were defined as a mismatch between ventilator and patient rates (figure 2). When ventilator rate was above patient's rate, the following events were identified and described: 'double triggering' (figure 2 and online supplementary figure S3); 'auto-triggering'(figure 2 and online supplementary figure S4), and 'uncoupling' (figure 2 and online supplementary figure S5) with two particular variants: (1) 'isolated uncoupling' (online supplementary figure S5) and (2) 'reverse triggering' (online supplementary figure S6) but without being able to reproduce it on the bench.

When the patient's rate was above the ventilator rate, we identified this as 'ineffective efforts' (online supplementary figure S7).

When patient and ventilator rates were completely dissociated, we described this as 'prolonged uncoupling' (online supplementary figure S8).

Intracycle asynchronies (figure 2) were defined as distortions of the flow and pressure curves, during inspiration and/or expiration. When a distortion of the inspiratory curves occurred during any portion of the inspiratory cycle or lasts for the entire cycle, we reported this as 'intracycle underassistance' (online supplementary figure S9). This event results from an insufficient unloading of inspiratory effort. The degree of distortion of the flow curve will depend on the intensity and the timing of the patient's effort.

A pressure-overshoot (online supplementary figure S10) can occur at the beginning of the pressurisation, in devices using fasts turbines.

When the end of the pressure and flow curves are out of phase with the thoracic and/or abdominal belts, we described this as delayed cycling (or long
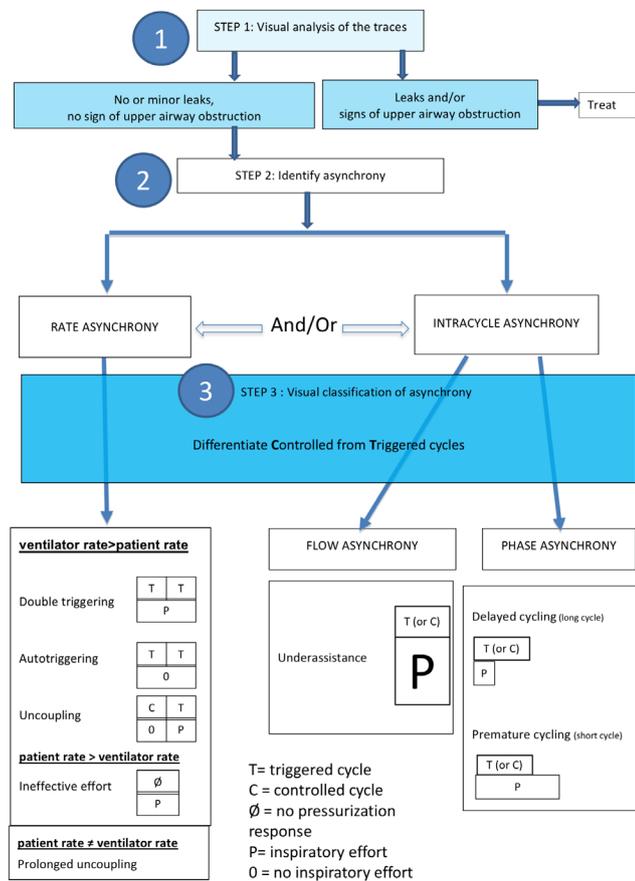
Figure 1 Algorithm for analysis of PVA by type III polygraphy performed under NIV. NIV, non-invasive positive pressure ventilation; PVA, patient-ventilator asynchrony.

Figure 2 Patient/ventilator interaction, aspect of curves and EMG for each PVA. 0, no inspiratory effort; ø, no pressurisation response; C, controlled cycle; EMG, electromyography; P, inspiratory effort; PVA, patient-ventilator asynchrony; T, triggered cycle.

cycle) (online supplementary figure S11) or premature cycling (or short cycle) (online supplementary figure S12).

Suggestions for making the distinction between triggered and controlled cycles are detailed in the online supplementary file 2.

DISCUSSION

The present report proposes a pragmatic non-invasive and systematic approach of PVA in the setting of CHRF treated by non-invasive ventilation. The detailed description and classification of PVA events provided in this report are based on a visual analysis of polygraphic tracings. The patient's respiratory efforts were analysed using abdominal and thoracic tracings. Bench testing was used to confirm hypothesised mechanisms involved. It has been possible for all the events except one (only due to technical limits of the bench). Because leaks and residual upper airway obstructive events may generate per se PVA, our approach deals with PVA only after having corrected leaks and upper airway obstruction.[1]

One obvious limitation of our suggested algorithm for analysis of PVA is the absence of gold-standard signals of inspiratory effort (such as oesophageal pressure, Pdi, EAdi, diaphragmatic or parasternal EMG[6]). As shown in the ICU setting,[2] it is possible that detection and quantification of PVA according to this algorithm underestimates the number of events.

Alternative non-invasive signals of inspiratory muscle effort such as parasternal EMG,[6] although theoretically simpler and used in clinical studies, require expertise and have limitations, s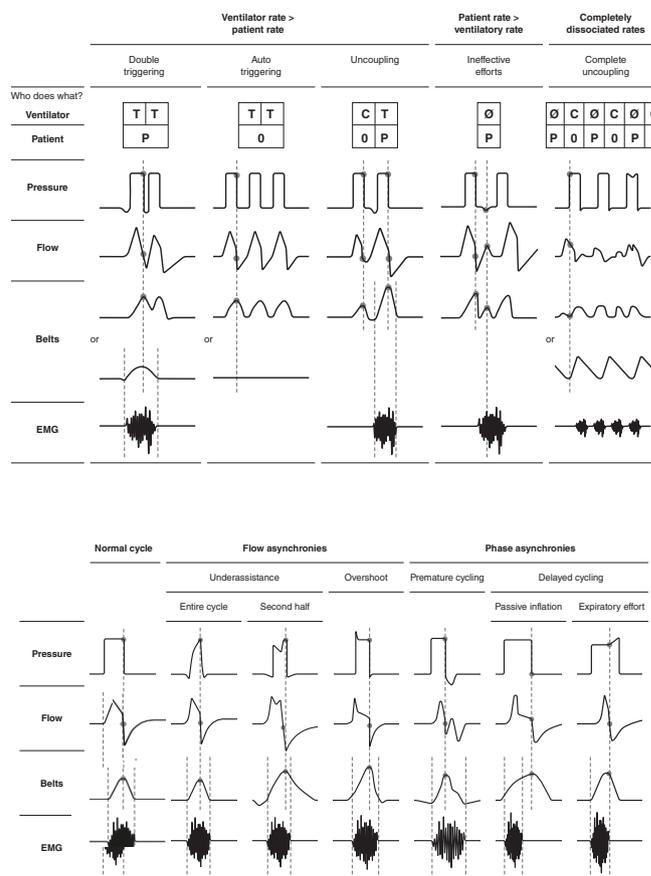uch as postural artefacts and potential contamination of signals by other chest muscles.[5] Other signals of inspiratory effort, such as analysis of mandibular movements,[7] have not been validated under NIV and require further testing. Pulse transit time, also an indicator of inspiratory effort,[8] although helpful to distinguish obstructive from central events under NIV, does not have the resolution and the precise time coupling for detecting unrewarded efforts or intracycle asynchronies.

We found that no PVA is specific/pathognomonic of a given pathophysiological situation. Management of PVA will have to integrate the type(s) of PVA identified according to the presented algorithm and also the patient's pulmonary mechanics and settings.

Clinical relevance and usefulness of searching for PVA in chronic NIV is also a critical issue and requires further studies.

In summary, the present report provides a pragmatic approach for a systematic analysis of PVA in patients under long-term NIV. Standardising the analysis of PVA, which occurs frequently in long-term NIV, will be helpful for future clinical studies exploring the impact of PVA on comfort, efficacy of NIV, morbidity and mortality.

Author affiliations
[1]UMRS1158 Neurophysiologie respiratoire expérimentale et clinique, Sorbonne Universités, UPMC Univ Paris 06, INSERM, Paris, France
[2]Groupe Hospitalier Pitié-Salpêtrière, Service de Pneumologie et Réanimation Médicale, Assistance Publique – Hopitaux de Paris, Paris, France
[3]Division of Pulmonary Diseases, Geneva University Hospital, Geneva, Switzerland
[4]Pulmonary Division, Centre Hospitalier Universitaire de Dijon, Dijon, France
[5]Service de pneumologie, Centre Hospitalier Princesse Grace, Monaco, Monaco

[6]Service de Physiologie-Explorations Fonctionnelles, AP-HP, Hôpital Raymond Poincaré, Garches, France
[7]Réanimation, Pôle soins aigus, centre hospitalier Ales, Ales, France
[8]Pulmonary Rehabilitation, Istituti Clinici Scientifici Maugeri, Pavia, Italy
[9]Pneumology Department, Hospital de Sabadell, Corporació Sanitaria Parc Taulí, Universitat Autònoma de Barcelona, Sabadell, Spain

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