August 2018 VOLUME 19, ISSUE 2

ARTP Association for Respiratory Technology & Physiology

Inspire

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INSPIRE is published three times per year: 1st April, August, December. Deadline for articles two weeks prior to publication.

FIRST WORD

VOLUME 19, ISSUE 2. AUGUST 2018

A sizzling summer special for you in this record-breaking weather. Well OK, maybe it isn't as exciting as that but there are still a few thought-provoking reads while you sit by the pool realising it isn't coming home (yet). Two high quality ARTP Education bursary winners, which I recall from the conference in January, look at an objective measure for early determination of occupational asthma and an alerting system to improve communication for MND patients using NIV. The bursaries enabled the authors to present their work at conference and are one of the benefits of being a member of ARTP.

Emphysema v Idiopathic Pulmonary Fibrosis; how do they differ in terms of lung function testing? Why? Thoughts on this and more are here. I wonder if anyone out there would consider an article in response?

For some time I have been considering including an article on Research Methods in Inspire. One has an interesting research idea but how best to proceed? This discussion was touched on in the last issue and Dr Chinn has responded with a generous offer to provide a superb series of research skills guides he developed for NHS Fife. Researchers in respiratory function now have no excuse! It is heartening to me that an Inspire article prompted such a response and also testimony to the nature of the ARTP 'community'.

Speaking of 'community', what do the COPD community think about COPD? See here.

The regulars; ARTP Manufacturers take holidays, too, meaning On the Blower is not quite so packed this issue but Matt has still managed to rustle up a few items of interest. Karl has information about what ARTP as an organisation is up to and Suhilla has compiled some news bites, tweets and articles for holiday reading. I have added 'From the Museum' (thanks again to Dr. Chinn) and I hope it will be a new regular feature.

Finally, it is back for the summer; another lung function related cryptic crossword with absolutely no prizes other than your satisfaction at having completed it before your colleagues. Answers in December.

I hope you enjoy this issue and manage to have a relaxing summer holiday. Please consider submitting your articles for Inspire—you can contact me at: Inspire@artp.org.uk. Aidan Laverty

Dr. Karl Sylvester

ARTP Honorary Chair

A WORD FROM THE CHAIR

Phew what a scorcher….and that's just this edition of Inspire! I hope you've all been dealing with the heat as best you can. I'm sure we're not complaining though given the wish for warmer summers in years gone by. However, this really isn't the climate for those with a lung disease.

But don't worry, the winter months will soon be upon us and the warming thought of another ARTP conference. This year in Glasgow. Hmm, Glasgow in January, just remember these current heady heights of 30 degrees and more. Although as always, ARTP will provide and if you're not one for wrapping up and facing the bracing chill our Events committee have plenty to keep you warm and entertained so you need never leave the comfort of the DoubleTree by Hilton. There will be the glow of neurons firing during the thought-provoking educational programme and muscles generating plenty of heat with the evening's entertainment. As you will I'm sure have seen, conference registration is very much open and this year with an early bird registration deadline, so get in to guarantee 2018 prices in 2019.

There are many benefits to being an ARTP member, as outlined in our Editor's section. Your ARTP Executive Board are always looking for ways to give you more or try and improve on existing benefits. With the latter in mind, we had a very constructive and creative meeting with our new website hosts, Senior Internet. The current website has certainly seen better days and we know you need a resource that can give you so much more than the existing platform currently does. It will take some months to construct but we are hopeful that by conference 2019 we will have a fully functioning new website to attract you in and keep you coming back for more.

Another benefit of ARTP membership is the ARTP forum and I'm glad to see a recent resurgence in posts. Most posts are usually requests for assistance or guidance from other forum members and there is always great discussion and debate. There is no such thing as a stupid question. If you're thinking of asking the question I can guarantee you there will be plenty of others who want to ask the same question, so take the leap. If you really would rather remain anonymous then

send your questions through to either myself or the webmaster, Chris Jones, who will be happy to post these for you. Take advantage of others with many years experience behind them. They like having their craniums massaged with testing conundrums and it keeps us all on our toes. It also means a wealth of knowledge is shared, increasing the knowledge base of all of the membership.

Speaking of cranium massaging, good to see the crossword back, ably constructed by our very own quiz master, Aidan Laverty, Inspire Editor. See how well you've done when he reveals the answers at a later date.

Hope you all enjoy the rest of your summer hols and see you on the other side.

Karl

A STUDY COMPARING SUBJECTIVE AND OBJECTIVE MEASURES OF NASAL RESPONSE TO ALLERGEN IN ADULTS WITH CHALLENGE-PROVEN OCCUPATIONAL ASTHMA

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Introduction

Asthma and rhinitis are common allergic conditions, with symptoms often triggered by environmental aeroallergens such as house dust mite and grass[1]. By contrast, occupational asthma (OA) and occupational rhinitis (OR) are caused by agents encountered in the workplace; they are currently the most frequently reported occupational respiratory diseases in the United Kingdom.

Many agents have been identified as workplace respiratory allergens, causing OA and OR[2]. Allergens can be categorised by their weight into high molecular weight (HMW) agents (greater than 2kDa), generally proteins such as flour, and low molecular weight (LMW) agents (less than 2kDa) including platinum salts and isocyanates[2].

One of the most commonly documented forms of occupational lung disease reported in industrialised countries is OA, which is estimated to have a role in 9 to 15% of all cases of new or recurrent adult asthma[3]. OA and OR develop as a result of exposure to inhaled allergens, irritants or corrosive gases and both can have significant impact on quality of life (QoL)[4]. One key question is whether OA and OR are part of the same disease or exist as separate entities. There has been some understanding of a nasal-bronchial link stating that OA and OR coexist, the 'united airway disease' (UAD) hypothesis. This postulates that rhinitis and asthma illustrate manifestations of one syndrome in two parts, the upper and lower airways respectively[5,6].

OA and OR frequently coexist, OR appears to be more common than OA, with a greater frequency and intensity of nasal symptoms following sensitisation and subsequent exposure to HMW than LMW agents[7]. OR is known to be associated with an increased risk of developing OA; this risk is highest in the year after onset of rhinitis, a disease characteristic that may provide weight to the UAD hypothesis[8]. Cullinan et al. have reported a higher prevalence of occupational rhinitis in bakeries ranging from 15% to 40%[9]. OA has a well described diagnostic pathway where the gold standard test for diagnosis is a specific inhalation challenge test (SIC). However, this requires experienced personnel and involves a long hospital attendance. In contrast to OA, varied methods are used to diagnose OR in populations or clinical patient groups, including questionnaire, physiological and laboratory tests and no such gold standard exists[3]. Therefore, there is a need for objective tests for this disease allowing for accurate diagnosis at an early stage, to reduce symptom burden and hopefully avoid progression to OA, with notable benefits to clinicians, employers and, above all, the workers.

Hypothesis

Adults with challenge-proven OA due to HMW agents have a significantly greater nasal response to SIC than those with OA due to LMW agents, as measured by:

a) Nasal symptoms in both the early phase and late phase, using total nasal symptom score (TNSS) - primary outcome measure

b) Early phase nasal tryptase – secondary outcome measure

Method

We recruited adult workers exposed to low molecular weight (LMW) or high molecular weight (HMW) agents, who underwent SIC (shown in Figures 1 and 2) and then objective and subjective nasal measurements were compared: Nasal tryptase and Total Nasal Symptom Score (TNSS) respectively. The primary endpoints were early and late phase TNSS and the secondary outcome measure was early and late phase nasal tryptase measurements. Prospective patients exclusively performed peak nasal inspiratory flow (PNIF) throughout the challenge and RQLQ at baseline. Nasal tryptase was measured at baseline and post 15 minutes and compared following control and active challenges.

Figure 1: Specific Inhalation Challenge Test.

In the chamber using the 'dust tipping technique'.

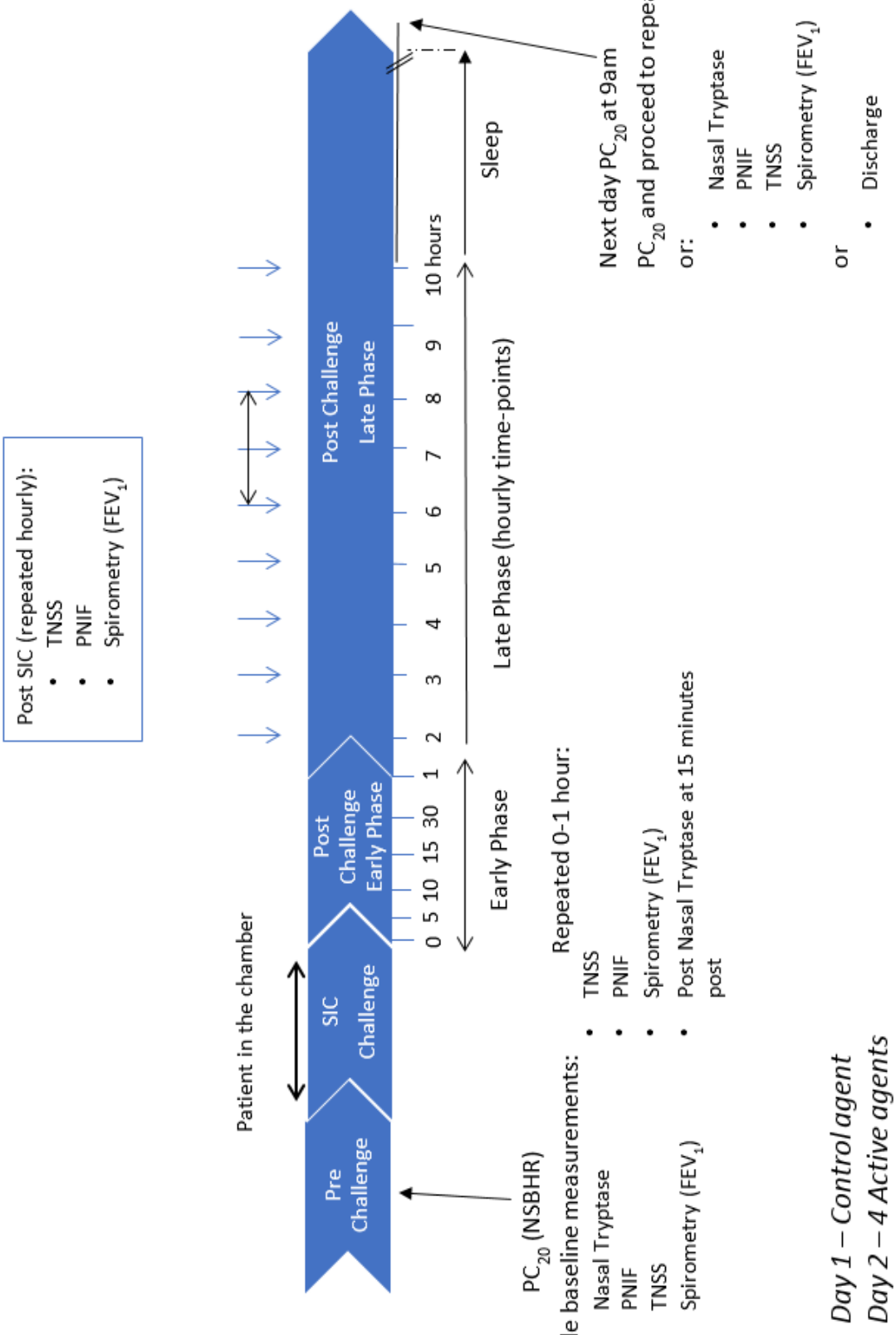

Figure 2: This illustrates the study procedure from Day 1 to Day 4

Results

Figure 3 is a summary of patient recruitment and data acquisition. A total of seven participants with OA confirmed by SIC were included in the study, four with challenge-proven OA to LMW agents and three with challenge-proven OA to HMW agents.

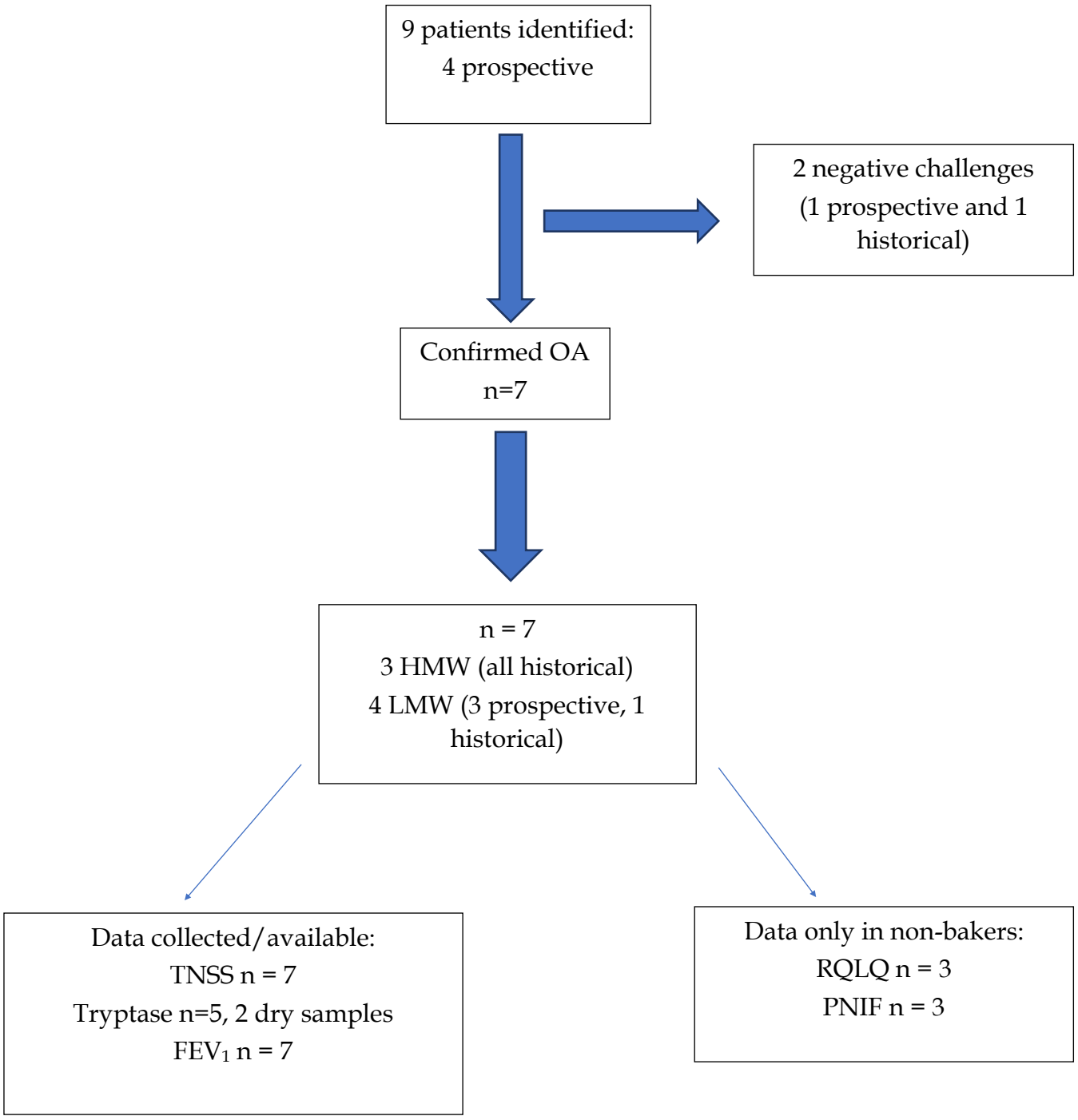

Figure 3: Study flow chart showing data acquisition

Table 1 summarises the clinical and baseline characteristics of the LMW and HMW groups. There was a higher prevalence of atopy (67% versus 25%), cigarette smoking (67% versus 0%), and lower mean age (28 years versus 43.5 years); in the HMW than the LMW group. The proportion with early (1/4 versus 1/3), late (2/4 versus 1/3) or dual phase (1/4 versus 1/3) responses and of NSBHR (PC20≤16mg/ml histamine) was similar between the two groups (2/3 in both).

Table 1: Characteristics of participants with challenge-proven occupational asthma triggered by low (LMW) or high (HMW) molecular weight agents. Data shown are number (%) unless described otherwise.

	LMW (n=4) ∞	HMW (n=3)
Age in years (median (range))	44 (41, 61)	28 (24, 54)
Male gender	4 (100%)	3 (100%)
Atopy*	1 (25%)	2 (67%)
Current smoker	0 (0%)	2 (67%)
History suggestive of occupational rhinitis	3 (75%)	3 (100%)
Years of exposure, median (range)	4 (1, 8)	5 (2, 32)
Years of symptoms, median (range)	1 (0.5, 2)	1 (0.5, 2)
Isolated early response	1 (25%)	1 (33%)
Isolated late response only	2 (50%)	1 (33%)
Dual response	1 (25%)	1 (33%)
Bronchial histamine response	2/3 (66%)†	2/3 (66%)

∞ Each individual was sensitised to a different LMW agent. The agents were: an acrylate-based solution; metal-working fluid; rhodium & iridium salt; and epoxy resin. The HMW cohort were all sensitised to wheat flour.

** Proportion positive to common aeroallergens on SPT*

† One participant (LMW-4) did not have a reproducible bronchial histamine response

TNSS Early phase nasal response was higher in the HMW group but this was not statistically significant (p=0.40). Similarly, early phase nasal tryptase was higher in the HMW (median 11.6 µg/L range 4.5, 200) than LMW group (median 1.15 range 0, 7.0): p=0.11 (shown in figure 4). Late phase nasal symptoms responses and early and late phase bronchial responses to allergen were similar between the two groups.

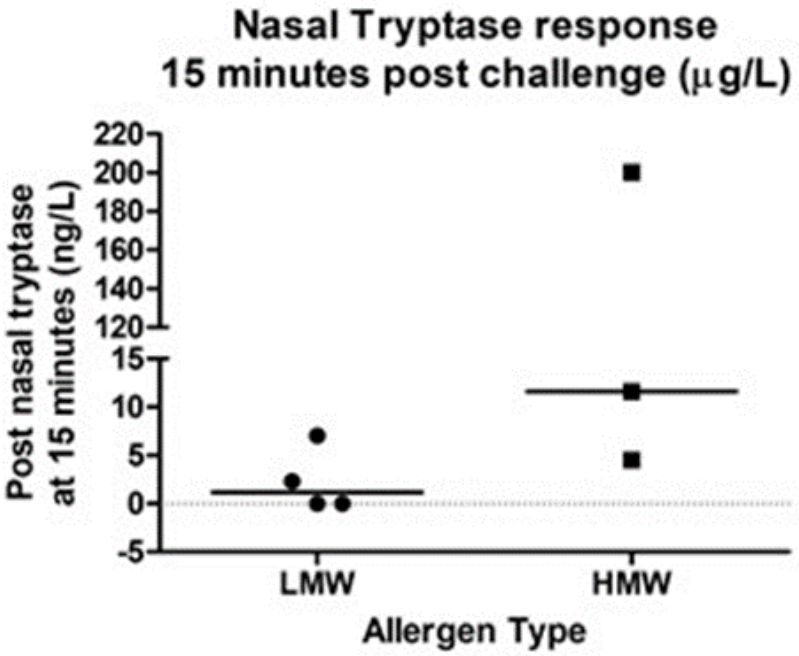

Figure 4 shows nasal tryptase responses µg/L taken 15 minutes (both Low Molecular Weight (LMW) and High Molecular Weight (HMW)) after specific inhalation challenges (4 vs. 3, p=0.11)

Discussion

In this study of specific inhalational challenges, in patients with challenge-proven OA, subjective (TNSS) and objective (nasal tryptase) measurements of nasal early phase responses increased more following challenge with HMW than LMW agents. However, the differences between the groups were not statistically significant in this small study and limited conclusions can be drawn. The data do provide support and feasibility for future larger pieces of work designed to better understand differences in pathophysiology between HMW OA and LMW OA.

Other authors have also found differing responses to HMW and LMW agents during SIC that support this suggestion. Malo et al., reported in a SIC study that OR appears before OA in the case of HMW agents but the main area of interest in that study was rhinoconjunctivitis[10]. The study reported rhinitis was more intense for HMW subjects (19 out of 24), where three or more subjects had symptoms of runny nose, itchy nose, nasal blockage and sneezing than LMW (5 out of 14 subjects, p<0.01). In a different case series, Castano et al., identified the frequency of nasal symptoms was higher in the group exposed to HMW agents compared to those exposed to LMW agents[11]. In addition, a positive nasal and bronchial challenge was observed in 13 instances. The association was reported significant (p=0.04) and more prevalent in patients challenged with HMW agents (n=11/22) than with LMW agents (n=2/21). This suggests, in comparing the signals seen in this study and the reported case series by Castono et al., there does appear to be a link between OA and OR in line with the UAD model. Castano et al., observed a significant increase in eosinophils in the nasal lavage thirty minutes post exposure in subjects with a positive nasal challenge to an occupational allergen, which provides insight for future work as well as tryptase as a biomarker[11].

HMW and LMW occupational agents are thought to have differing allergic mechanisms. HMW agents being proteins that are typically thought to cause sensitisation through an IgE-mediated mechanism via a Th2 response (a 'classical' type 1 hypersensitivity)[12]. HMW OA is associated with a background of atopy[13] and this is seen to be more prevalent in the HMW group as opposed to LMW group in this study. The lack of association between atopy or raised total IgE and occupational asthma to low molecular weight (LMW) allergens suggests a different mechanism of OA caused by non-protein chemicals[13]. The physical state of occupational agents differs: HMW agents are typically dry or liquid aerosols and LMW agents are more typically gases or vapours. The "dusty" nature of flour and other HMW agents may increase deposition in the upper airways and the subsequent development of rhinitis symptoms; the findings of this study would support this. Nasal obstruction if present is likely to cause mouth-breathing and therefore increase the risk of inhaling sensitising occupational agents directly into the lower airways without passing the nasal filter[14].This effect may be present in both HMW and LMW groups. Meca et al., carried out a study in 78 patients concluding OA caused by LMW agents may be more severe than that caused by HMW agents[14].

Furthermore, it has been documented that inhaled particles with aerodynamic diameters equal or above 10μm can cause eye or nose irritations[15]. Literature suggests the majority of particles larger than 10μm and up to 80% of particles between 5 and 10μm can become stuck in the nasopharyngeal region because of inertial impaction and centrifugal condensation as the airstream has the highest velocity here[15]. Most particles are removed by the mucociliary system within a few hours, yet a large exposure can overwhelm macrophages in their elimination of these particles and hence allow them to penetrate into the interstitium[15]. Alternatively, particles with diameters above 0.5μm are deposited into the bronchi, bronchioles and alveoli via sedimentation and impaction. Consequently, these particles are removed much more slowly where the ciliated epithelium is not present, residing there for up to several hundred days[15]. An interesting finding by Houba et al., was that α-amylase is generally present in particles with an aerodynamic diameter above 9 μm[16].

The mechanism of LMW allergy is less well characterised. A number of LMW allergens have been shown to cause an IgE-mediated hypersensitivity (type 1 hypersensitivity) reaction, measurable in at least some individuals (e.g. isocyanates). Other agents such as precious metal salts can be used in skin prick testing. It is worth remembering that some agents may cause allergic responses through more than one mechanism, with case reports of agents such as epoxy resins provoking both type 1 and IV allergic reactions in the same patient[17]. A late response is commonly seen in positive epoxy resin SIC, such as in subject LMW-3. Some authors propose that the epoxy resin agent acts as a hapten molecule, which combines with a serum protein to form a conjugate which induces an immune response. The characteristic late phase response may therefore be explained by the time taken for the hapten molecule to enter the lower airways and combine with the serum protein before creating a conjugate molecule. Hox et al developed a mouse model showing that OA caused by LMW agents may depend on neuro-immune interaction with both mast cell activation and transient receptor potential ankyrin (TRPA)1-dependent stimulation of sensory neurons[14,18].

To our knowledge this is the first analysis of nasal tryptase data from occupational SIC testing. The timing of tryptase sampling in this study was based on the dose-response curves from nasal provocation challenges using intranasal doses of allergen[19]. Occupational challenge patients had higher levels of nasal tryptase responses in this study in comparison to measurements made in nasal allergen provocation challenges for allergic rhinitis (AR) using intranasal doses[19]. One reason for this difference is likely to be the method of allergen administration; intranasal administration is likely to have produced more direct absorbance with a more acute peak of exposure as a consequence, whereas occupational SIC may have allowed slower exposure but greater persistence of allergen in the nasal passages. Studies in AR have looked at tryptase at a number of time points ranging from 15 minutes to 24 hours post challenge and showed significant increases in tryptase (45%) where immediate responders showed a fast release with a peak at 15 min post nasal challenge to house dust mite[20]. Further examination of nasal tryptase levels measured at different time points after SIC would allow a similar dose-response curve to be established in order to find the optimal sampling time. One should consider that different agents may have different responses. Alternative mediators such as IL-5 or eosinophilic cationic protein and evaluation of the timing should also be considered for measurement[21,22].

Conclusion

A larger study would allow further investigation into the differences in nasal inflammation in OR triggered by these two diseases, adjusting analyses as appropriate for any differences in cigarette smoking, age or atopy between groups. A survey of workers collecting “cross-shift” (pre- and post) samples for nasal tryptase would provide cross-sectional as well as prospective data to assess nasal tryptase as a predictive factor of OR in workplace surveillance.

Further refinement of diagnostic tests could improve diagnostic accuracy and prognostication in the clinical setting; non-invasive tests could be used in the workplace setting to risk stratify exposed workers with rhinitis, this could be evaluated as part of a prospective study.

The project has provided helpful information to the clinical team to potentially improve diagnostic accuracy of OR in patients undergoing SIC.

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COMPARISON OF IDIOPATHIC PULMONARY FIBROSIS (IPF) WITH EMPHYSEMA (FIXED/ IRREVERSIBLE TYPE COPD) USING RESPIRATORY FUNCTION TESTING.

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Background

I am writing this article with the assumption that most readers are either qualified or well experienced student respiratory physiologists. Alternatively, this article may be of interest for any clinical health professional who has experience of dealing with patients who have clinical pulmonary respiratory diseases and some knowledge of lung function testing.

The pathway and disease process of COPD is very well known as is the pattern of respiratory lung function test results in quantifying and measuring the severity of COPD present in an individual patient who suffers from it[1,2].

It is important to realise that COPD is an umbrella term; Chronic Obstructive Pulmonary Disease, and has subset member disease types namely: Emphysema, Bronchitis and possibly Asthmatic components. The subset disease components may well overlap, or not, to differing degrees in actual individual patients.

When I first started working in Respiratory Physiology, over 35 years ago, these pulmonary disease types were defined at being looked at individually i.e. Emphysema type, Asthma type etc. rather than the umbrella COPD term. Only pure asthma disease is now given this status.[3,4,5].

Similarly, there is another umbrella term for different pulmonary disease; called ILD or Interstitial Lung Disease, which includes very many different individual pulmonary diseases. For example, many industrial lung diseases such as Asbestosis, Pneumoconiosis and alternatively many Fibrotic Lung disease types. What all these lung diseases have in common under the umbrella term ILD is that they consist of direct disease and damage to the actual alveolar membrane which is by far the main part of the disease process in these diseases.

Some COPD disease, especially those with a degree of asthmatic component, can be improved by decreasing airways obstruction using bronchodilator medication. This means some patients can have symptoms improved, or reversed, to a degree and this is especially true of asthma. Asthma is also an obstructive lung disease but is variable in condition and there is no lung membrane damage present in pure asthma which distinguishes it from COPD/ Emphysema in its pure form. There are some ILD diseases which can be improved and treated to a degree e.g. pneumonia with antibiotics, bird fanciers' lung disease and farmers' lung disease (which can be life threatening) can be improved greatly by use of steroids (if diagnosed and caught early enough).[6,7,8,9]

In terms of patient numbers there are many more COPD and pure Asthma sufferers than of specific ILD sufferers. However, in general terms ILD can be much more severe in outcome and prognosis. Both COPD and Asthma do have treatment plans that can work well with large numbers of patients who adhere to their treatment plans provided to them. ILD patients (unless of a specific reversible type e.g. responding to steroid treatment, such as Bird Fanciers Disease) are not usually so fortunate. This is especially true of Idiopathic Pulmonary Fibrosis (IPF) which is progressive in prognosis and until recently has had no real treatment. IPF is non-responsive to steroid treatment but drugs like Pirifendidone and Nintedanib have been introduced which show some evidence of at least improving symptoms in mild or moderate IPF[40,41].

This paper is mainly a direct comparison of lung function test results of "pure" Emphysema against "pure" IPF result patterns however empyhsema/COPD and IPF sometimes co-exist[38] which would give different lung function results and patterns again.

Table 1. Results of Lung Function testing patterns comparing Emphysema against Idiopathic Pulmonary Fibrosis.[11,12,13,14,15,16,42]

	Emphysema or Fixed type of COPD		Idiopathic Pulmonary Fibrosis
1	Obstructive Spirometry with significantly decreased FEV_1. VC and FVC reduces with disease progression but reduction is not usually as significant as FEV_1 reduction especially in mild to moderate COPD. *Please note obstructive spirometry is first marker or indicator of early COPD.*	1	Non-Obstructive Spirometry which becomes increasingly restrictive as disease progresses. Both FEV_1 and FVC decreases as disease progression increases. *Please note this restrictive spirometry marker of IPF is only clearly indicated around middle to later stages of IPF disease.*
2	FEV_1/FVC or FEV_1/VC ratio decreases as severity of airways obstruction and Emphysema increases. This is also defined by guidelines given such as NICE or GOLD.	2	FEV_1/FVC ratio is normal or the ratio increases with increasing restriction linked with increasing IPF progression.
3	Mid Expired and end stage expired flows very small and flattens completely as Obstruction and Emphysema worsens.	3	Most notable flow marker is that PEF/FVC ratio increases as disease worsens in IPF.
4	The Forced Expired Time FET needed to reach plateau of FVC increases significantly on Emphysema and subsequent increasing airways obstruction.	4	The Forced Expired Time FET needed to reach plateau of FVC decreases significantly with IPF progression.
5	Static Lung volumes RV and FRC increase as Emphysema worsens with VC decreasing as emphysema worsens. AS RV and FRC increase significantly though with severity overall TLC increases giving hyperinflated lungs and much bigger overall lung volumes with increased severity.	5	All lung volumes including FRC and VC decrease significantly with IPF progression. Hence overall TLC decreases with increased IPF severity leading to significant smaller lung volumes during mid and end stages of disease.
6	T_LCO is reduced with increasing severity of Emphysema as capillary/alveolar membrane becomes damaged due to increasing loss of elastase in these membranes with increasing severity of Emphysema. Please note though this is not the first marker which occurs with early COPD the obstructive spirometry pattern is.	6	T_LCO is reduced with reduction continuing to get more significant with IPF progression. Please note this is the first marker which occurs in early IPF and occurs before notable restrictive changes in spirometry.
7	Airways resistance increases with increasing severity of emphysema	7	Airways resistance decreases with increasing severity of IPF.

The Main respiratory function markers for COPD/ Emphysema are:

1. Described in Table 1, above.
2. Oxygen desaturation on exercise does not usually occur for mild COPD/Emphysema but some desaturation occurs with moderate COPD/Emphysema and the desaturation increases steadily with increasing severity of Emphysema[43, 44].

The Main respiratory function markers for IPF are:

1. Reduced T_LCO with non-obstructive spirometry.
2. Usually significant Oxygen desaturation seen on exercise e.g. can be shown by recording pulse oximetry graph on a 6-minute walk[42].

The significant factors of Emphysema are that it can usually be stabilised by the patient stopping smoking (this stops continued disease progression) together with symptom improvement via pulmonary rehabilitation including tailored exercise. The late Dr Tim Griffiths proved this by his research work on COPD patients.[17,18,19].

However, IPF patients unfortunately do not have this option as the disease continues to progress and the benefit of pulmonary rehabilitation is limited (due mainly to oxygen desaturation on limited exercise and very importantly no disease stabilisation). Pulmonary rehabilitation may give short-time benefits to ILD patients with mild to moderate disease, however, probably through improving ventilation technique.[20]

Respiratory failure and the late suffering in IPF patients may not usually be witnessed by respiratory physiologists working in respiratory function laboratories except for those of a tertiary nature (e.g. centres which perform lung transplantation).[21, 39].

In conclusion regarding direct comparison between Emphysema and IPF they are two completely different lung diseases, each of which changes permanently the lung anatomy/physiology in the individual patient.

In Emphysema there is a loss of elastase and inflammation of airways causing obstructive airways disease with damage to alveolar-capillary membrane and large lung volumes.

In IPF there is an increase and build-up of elastase with lung membrane scarring and lung membrane thickening; this then leads to reduced alveolar air volume thus reducing all lung volumes giving increasingly smaller lung volumes with increasing severity of disease.

Discussion and opinion hypothesis/ postulation on reasons for IPF.

We know that generally Emphysema is caused directly by smoking. This causes inflammation of airways, loss of cilia, and increased mucus all causing direct airways obstruction and making pulmonary infection more likely. Elastin degeneration occurs and there is direct damage to the alveolar membrane and the alveolar capillary bed.[22,23].

A less common reason for Emphysema is Alpha-1 antitrypsin deficiency which causes Elastin degeneration within the lung membrane to be more probable. Hence very early severe onset Emphysema can be seen in young adults who are both smokers and have Alpha-1 antitrypsin deficiency.[24]

Now the causes of Idiopathic Pulmonary Fibrosis are unknown and 'Idiopathic' means exactly that.

What we do know, however, is that in IPF there is a continual build-up of Elastin and scarring of lung membrane. The lung membrane becomes increasingly thicker with consequential decrease in alveolar air volume.[29]

Some researchers have suggested that smoking causes IPF and it seems to me that most lung disease gets linked to smoking. Obviously, there are lung diseases which do have a direct causal link to smoking, such as emphysema and primary lung cancer.

However. there are pulmonary diseases which are not caused by smoking e.g. pure Asthma and Cystic Fibrosis[28] (obviously people who have these diseases will suffer worse symptoms and outcomes if they smoke but my point here is that smoking did not directly cause their original lung disease. IPF is in my opinion another lung disease type which has this same characteristic i.e. not directly caused by smoking although I fully accept that smoking increases morbidity in IPF patients and smoking cessation would increase life expectancy in this group.

I started my career in NHS as a qualified clinical physiologist in cardio-respiratory work. In my twenties there was an epidemic in UK of coronary artery disease with high morbidity and mortality due to Myocardial Infarction (Heart Attacks). Most of these could also be directly linked to smoking.[25,26,27].

However, because of better funding and resources, cardiac disease research and hence treatment has hugely increased over the recent decades. This has given massive improvements for UK's population in cardiac health. Cardiac disease research has been fortunate as unlike respiratory disease there has not been as direct a link in the public and political perception between cardiac disease and smoking. There always has been such a link and perception in respiratory disease of all types which I personally believe has indirectly led to underfunding and under-resourcing on respiratory research.

I feel there is no direct link between IPF and smoking because as I hope I have already demonstrated Emphysema and IPF are two directly opposing lung diseases. My question is what are the mechanisms by which smoking acts to present as two different and opposing pulmonary diseases? Where is the evidence to show that smoking can cause emphysema in one person (which has been proved) but can cause IPF in a different person (which has not been proved).

More likely in my opinion is that the IPF process occurs due to a "faulty template" occurring at birth or very early childhood. Now we know the lungs are the last major organ systems to form and function

(they are not directly needed pre-birth in the mother's womb). In fact, the lungs are the last organ system needed to function to give individual viable life to newly born babies.[30,31,32,33].

A damaged alveolar template could be linked to low birth weight. Also, it is known that several babies can have respiratory problems caused by "mini-aspirations" by stomach acid reflux, resulting in acid managing to enter the newly functioning lung structure which causes lung membrane damage. This usually dissipates and seems to clear with passing time and growth but, in this hypothesis, (shared by others[45, 46]) the lung membrane template is damaged leading to IPF in much later adult life.

There is a disease type which possibly has some similarity as described in Cardiology. Rheumatic Fever in Children causes illness which there can be some recovery from but again later in adult life Rheumatic Heart Disease occurs which is linked to original Rheumatic Fever. Rheumatic Heart Disease causes poorly functioning heart valves. This problem can be solved by cardiac surgery and replacing heart valves with artificial valves.[34].

I have written this article as a qualified respiratory physiologist but I feel there may be another clinical area where IPF can be investigated from;. We need Qualified Biomedical Scientists and Haematology clinicians to look at capillary alveolar blood in IPF and Emphysema during disease progression.

Specific Coagulation studies, Biochemistry and ESR blood tests may well prove valuable research data on this side of pulmonary research information as indeed it already has done so.[35,36,37]

Conclusions

I hope I have shown by the first two parts of this article; the description of lung function testing and respiratory physiology, the reasoning behind my assertion that Emphysema and IPF are opposite types of pulmonary disease and I hope these will be of value particularly to respiratory physiologists.

The third part of this article is my opinion. However, its purpose though- even if my assumptions and hypothesis are ultimately proved wrong- is to stimulate respiratory research thinking via respiratory physiology.

Hopefully it does show that more research work into IPF, with greater detail and increased scientific complexity, is needed to understand this disease pathway. This is very much needed for the respiratory patients we serve who have this disease and the long term aim must be to find evidence of possible treatment pathways to improve the very severe symptoms and prognosis of this disease.

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Do you need help to develop your generic skills particularly for research?

Dr David Chinn davidchinn@nhs.net

The April issue of INSPIRE included an account by Samantha Irving of a 3-day training course to develop research skills for Healthcare Scientists. We were reminded that research activity is recommended as part of the ARTP generic job descriptions of respiratory clinical physiologists in Band 3 and above. Despite this, relatively few staff actually engage in research (and development) activity. One of the recognised barriers to research activity is lack of appropriate skills. Fortunately, help is available in understanding aspects of the research process as at NHS Fife we have produced a series of introductory study guides for Health Service Staff. The topics covered are listed in Table 1.

n	Guide
1	How to devise a research question and choose a study design
2	How to write a protocol
3	How to critically appraise a paper
4	How to apply for a 'favourable' opinion from an ethics committee and for R&D management approval (at NHS Fife) *
5	How to write a research grant application
6	How to avoid pitfalls when designing a questionnaire
7	How to plan your data collection and analysis
8	How to manage a research project
9	An introduction to qualitative research
10	An introduction to medical statistics
11	How to calculate sample size and statistical power
12	How to choose a statistical test
13	How to make sense of numbers
14	An introduction to SPSS (for version 22) **
15	How to analyse data with Excel
16	How to achieve success with your dissertation
17	How to write an abstract
18	How to write up and get your work published

Table 1. NHS Fife Study guides

** Study guide 4 is specific to NHS Fife but the processes should be similar in other Health Boards and Trusts. Please note, this guide was written in 2016 and it may be best to first check with your local ethics committee concerning the current process for applying for a favourable opinion.*

*** Comes with 3 SPSS datafiles*

In her article Samantha Irving had listed topics identified as important learning outcomes by those attending the research training course. Many of these subjects are included in the list of NHS Fife guides some of which, incidentally, are also relevant for those engaged in audit or service evaluation.

Most of the guides have been written by Dr David Chinn (a respiratory physiologist and epidemiologist) based on his teaching practice, his long experience of research work, writing for publication, his time sitting on an NHS ethics committee and as a referee for a number of journals. The draft guides were reviewed and amended in light of feedback. They are designed as stand-alone documents intended to be both informative and simple to follow. For example, many people find statistics a difficult subject to grasp (let alone teach) and we have written guides 10 – 14 with this in mind. All the guides are introductory only and are not a substitute for a good quality book on medical statistics or research methods. However, they have been well received by staff who have been complimentary in their evaluations.

The guides are copyrighted to NHS Fife and are freely available for personal use by NHS Staff, including those undertaking postgraduate courses. They may be obtained from roy.halliday@nhs.net at the Research & Development Department, Queen Margaret Hospital, Dunfermline, NHS Fife.

The guides will be subject to revision over the next year. Accordingly, we would welcome any feedback (good or bad) that readers may wish to give. For example, did the guide cover what you would have expected in an introductory text? Are there any aspects you think should be expanded on or additional areas included to help the reader understand the overall subject? Any comments should be sent to Dr David Chinn davidchinn@nhs.net or djc20@st-andrews.ac.uk , formerly the Senior Research Adviser at NHS Fife, and now semi-retired.

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DOMICILIARY NON-INVASIVE VENTILATION (NIV) HOSPITAL ALERT SYSTEM FOR PATIENTS WITH MOTOR NEURONE DISEASE (MND).

Parkes, E., Greer, E., Gilsenan, C & Shakespeare, J.
Respiratory and Sleep Sciences, University Hospital, Coventry and Warwickshire, UK.

Motor Neurone Disease (MND) is a terminal, neurodegenerative condition which affects the transmission of nerve impulses along motor neurones which are located in the brain and spinal cord[1,2]. The prevalence of MND has been reported to be up to 5,000 in the UK at any one given point in time with up to 6 people dying every day from the disease[3]. The risk of developing MND has been reported to be 1 in 300 with a third of people dying within a year and 50% within 2 years[3]. Symptoms of MND include muscle stiffness, atrophy, progressive weakness, excessive saliva production which will eventually lead to difficulty in speaking, swallowing and breathing[3]. MND symptoms can be subcategorised into either 'limb-onset' or 'bulbar-onset' with 70% of diagnoses predominately being 'limb-onset' in nature where the upper and lower motor neurones that reside in the brain and spinal cord become damaged. Around 25% of diagnoses are 'bulbar-onset' where the lower motor neurones and the motor neurones located in the medulla oblongata start to dysfunction[4]. Both onsets of MND have different characteristics and vary in their clinical presentation. Forms of MND include Amyotrophic lateral sclerosis (ALS), Progressive muscular atrophy (PMA), Progressive bulbar palsy (PBP) and Primary lateral sclerosis (PLS)[5].

Type II respiratory failure secondary to respiratory muscle weakness in the form of aspirational pneumonia is the most common cause of death in MND. Respiratory muscle weakness causes insufficient ventilation which leads to the development of hypercapnia (raised PCO_2), hypoxia (reduced PO_2), breathlessness and poor sleep quality[6]. Non-invasive ventilation (NIV) is a treatment modality that is recommended by the National Institute for Health and Care Excellence (NICE) MND guidelines to provide both ventilatory support and symptomatic relief[7]. This support is provided by both an inspiratory positive airway pressure (IPAP) and an expiatory positive airway pressure (EPAP) in combination with a breathing backup rate (BR).

The ventilatory support is delivered via a facial or nasal mask interface. Domiciliary NIV has been previously described to significantly improve survival in MND patients with moderate-quality evidence from a Cochrane review reporting an estimated 205 day survival advantage compared to 'standard care'[8].

The domiciliary NIV service at University Hospital, Coventry and Warwickshire was commissioned in October 2016 for Coventry and Rugby central commissioning group (CCG). Currently care is provided to 112 patients from Coventry and Rugby, North and South Warwickshire, Solihull and Worcestershire CCG's. In total 60 patients have been diagnosed with MND and are currently receiving respiratory follow-up appointments every 3 months. The current service provision includes a lead Respiratory Consultant, two Clinical Scientists, one Ventilation Nurse, one Respiratory Physiotherapist and one Associate Physiologist.

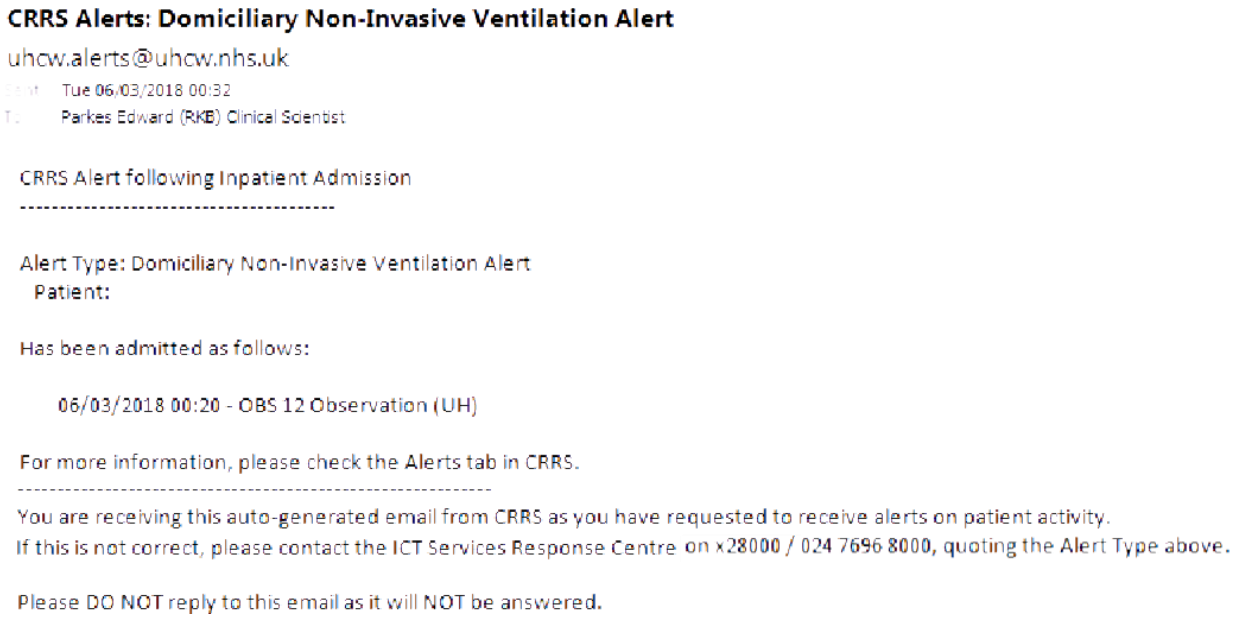
This compliment of staff allows one Physiology-led NIV initiation and follow-up clinic and one Joint Consultant/Physiology clinic per week in combination with options for urgent outpatient clinic appointments, home/hospice visits and daily remote monitoring of therapy compliance.

MND patients are both acutely and electively admitted to hospital for a variety of clinical reasons including chest infections, falls, acute worsening of the underlying condition, disease progression and endoscopic percutaneous gastronomy (PEG) insertion. The service started to observe that when MND patients utilising NIV therapy were admitted to hospital, staff were not notified until the patient was in the final stages of discharge or in some cases several days after they had returned home or to their designated hospice. As part of the recommended multidisciplinary team (MDT) delivery of care our service provides support for the facilitated discharge of patients on domiciliary NIV and provides a home visit between 5 and 7 days after the patient has been discharged.

The breakdown in communication between the acute hospital services and the domiciliary NIV service in some patient cases may lead to a detrimental effect on the level of high quality care that is delivered. In order to ensure clear and concise communication was achieved at all times, the service aimed to setup two types of alerting systems for MND patients admitted to hospital. For those patients residing in the Coventry and Rugby CCG and who were subsequently admitted to University Hospital a domiciliary NIV alert was integrated into the Trust's Clinical Results and Reporting System (CRRS). A list of patients diagnosed with MND and who are receiving domiciliary NIV treatment were inputted into the alert database which was then linked to CRRS. When a patient listed in the database is admitted to the Accident and Emergency Department (A&E) an email alert is sent to the domiciliary NIV staff (**Figure 1**). Subsequent alert emails are then sent when the patient is transferred to the appropriate ward for medical care. For the ward based medical and nursing staff the electronic patient record (EPR) also shows an alert which provides the team in charge of the patients care with details of the domiciliary NIV therapy, including IPAP, EPAP and BR settings (**Figure 2**). For those patients residing in neighbouring CCG's and admitted to other hospitals a relatively simplistic alert keyring containing the appropriate contact details of the service will be used to provide awareness to the clinical staff that the patient is receiving domiciliary NIV (**Figure 3**).

The domiciliary NIV team have introduced a new clinical alert system in response to an identified problem oriented around communication between healthcare providers. This required significant partnership working practices with other hospital departments including Information Communication Technology (ICT), respiratory ward staff and community palliative and physiotherapy services. This breakdown in communication has been identified to contribute towards delayed discharges, a disruption in the continuity of patient care and delays in home/ hospice follow up appointments by our dedicated ventilation nurse specialist and/or respiratory physiotherapist.

By alerting healthcare professionals that patients are receiving an important and indeed complex therapy, patient safety and quality of patient care can be improved. Over the forthcoming months the effectiveness of this strategy will need to be monitored, reviewed and amended in order to not only further develop the alerting system but also to ensure that it is in keeping with the Trust's key values which include partnership, collaborative working and improving the care that is delivered to patients. Overall this will aim to contribute towards the Trusts mission statement of becoming a world class healthcare provider.



CRRS Alerts: Domiciliary Non-Invasive Ventilation Alert

uhcw.alerts@uhcw.nhs.uk

Tue 06/03/2018 00:32

Parkes Edward (RKB) Clinical Scientist

CRRS Alert following Inpatient Admission

Alert Type: Domiciliary Non-Invasive Ventilation Alert

Patient:

Has been admitted as follows:

06/03/2018 00:20 - OBS 12 Observation (UH)

For more information, please check the Alerts tab in CRRS.

You are receiving this auto-generated email from CRRS as you have requested to receive alerts on patient activity.

If this is not correct, please contact the ICT Services Response Centre on x28000 / 024 7696 8000, quoting the Alert Type above.

Please DO NOT reply to this email as it will NOT be answered.

Figure 1. An example of an email alert sent from Accident and Emergency to domiciliary NIV staff

Domiciliary Non-Invasive Ventilation Alert (Neuromuscular Disease)

NIV Details

This patient is being treated for: Neuromuscular Disease

more than one treatment? ☐

NIV Settings

Date NIV commenced: 16/05/2017

Mode: Please Select -->

IPAP: cmH2O 20 ☐ Not Applicable

EPAP: cmH2O 10 ☐ Not Applicable

BF: Breaths per min 14 ☐ Not Applicable

General Details

Created on: 19/01/2018

Created by:

This patient is having treatment for NIV.
For any advice or further information please contact the **Non-Invasive Ventilation Team** on **Ext. 26734 / Bleep. 4930.**

Submit Cancel

Figure 2 EPR alert of NIV settings

Figure 3. Alert keyring for neighbouring CCGs

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https://www.carenity.co.uk/

What Carenity members think about COPD

For the occasion of **World COPD awareness day 2017 (November 15th)**, we have decided to ask the members of our **COPD community*** for their opinion about the disease and its impact on **their life.** ***128 respondents from the United Kingdom**

COPD is not a very well-known health condition. Among our COPD members, 66% **did not know anything about it** before being diagnosed. It is said that the disease is strongly linked to smoking, and indeed, 74% of respondents smoked in the past. However, the remaining 36% have never touched a cigarette. Those who have succeeded in quitting smoking are very contented with the result: *"since I gave up smoking last October and lost 3 stone since April this year I've been a lot better"*.

It is also an invisible illness, which often tends to be mistaken for asthma or stress, due to its respiratory symptoms.

A serious condition, but can be controlled with a healthy lifestyle

COPD is a serious debilitating condition (according to half of the respondents), a condition that can provoke isolation and make a drastic change in the patient's lifestyle: "*It has turned my life around I have to get people to do things for me"; "I find I can no longer do a lot of the things I use to enjoy"; "It limits my ability to play with grandchildren for long"; "I feel very alone".* Some also talk about having no more sex life, or having to retire too early. In fact, the **impact of COPD** on **professional life** is what seems to bother the Carenity members quite a lot: *"COPD has stopped my professional career", "I have had to medically retire from work", "feel I'm going to have to give up working because of my unreliability".*

However, a lot of them try to **lead a normal and healthy life** despite their diagnosis, go out with family and friends who understand and support them ("*the difficulties I have are recognised by family"*), and even travel, even though it means carrying inhalers and other devices with them all the time. **70% of the respondents do physical activities**, either regular ("*I try to do some exercises each day"*) or occasional, and only 27% cannot do sport because they do not feel well. Physical activity is a part of COPD treatment, as it improves the overall state of health of the patient, strengthens muscles, and helps lose weight, which together with medications has a positive impact on the patient's life.

Treatment effective for 20% of the respondents

The most popular treatment for COPD is, of course, **oxygen therapy**, as well as oral medications (corticosteroids, etc.). Even though our respondents are aware that the treatment does not cure COPD, and even though for 20% of them it is an obstacle to a normal social life, a quarter of them still find it effective: *"At the moment the meds are helping, but also cause problems of their own"; "the treatment I get is very helpful".* Only 6% of the respondents think that they are not very well taken care of by healthcare professionals: "*I'm fed up of going to drs with problems and feel like I'm wasting their time because they do say blood tests and nothing else",* while others *"have great care, so just get on with it"*. However, 81% consider that there is not enough **preventive care for COPD**.

Preventive care means helping people to quit smoking, adopt a healthy lifestyle, do sports, but also making local doctors and nurses more available, doing more frequent tests, and in a more global way, establishing a healthier environment by fighting against pollution. This is one of the goal of World Awareness days. But preventive care also means **prevention of COPD complications,** which is equally important and concerns the same issues: healthy lifestyle, no smoking, diet, sports, etc., but is also focused on vaccinations (flu, pneumonia, etc.).

Turning to alternative medicine for better management of COPD

Apart from conventional treatment, there also exist alternative medicines, to which a lot of our members turn, in order to improve their overall health, and **ease the COPD symptoms**. Thus, 39% and 38% of the respondents go in for **pulmonary rehabilitation programmes** (which assist patients in keeping a healthy lifestyle by elaborating a programme of exercise and a healthy diet, and helping manage the disease on a daily basis, but also providing psychological support which is not negligible) and **coughing techniques** (which help clear the bronchial tubes) respectively, to improve their breathing. **Diet** seems to be as important in controlling the symptoms, as the first two non-medication techniques, with 37% of the respondents who find it effective (there are certain foods to avoid, others to favour, etc.). Then comes **meditation** with 15% of members who practice it, and **yoga**, not very popular among our respondents (only 9% of them have become yogi and yogini). Yet, yoga is said to be of great use for COPD patients, as it teaches you to breathe correctly, by learning special diaphragmatic breathing techniques.

These alternative medicines, together with prescribed medications and family support helps COPD patients get a better control of their condition and reduce its impact on their daily life by accepting the disease: *"I'm on my own and I cope well with my C.O.P.D. as I accepted my condition"*.

Article with approval from: https://www.carenity.co.uk/condition-information/news-from-the-media/what-carenity-members-think-about-copd-373

Matt Rutter

Alan Moore

Brendan Cooper

ON THE BLOWER

This edition of 'On the Blower' has the regular news and product updates. A more digestible version after the bumper conference edition. Mark Wilson from Numed Healthcare has provided an article on Turbine sensors and the problem of low flow.

Manufacturers Survey

The manufacturer survey is opening again this August and will close on the 31st October. After last years success of responses, we want to try and beat it. Please try to take the time to complete it, as the results are really appreciated by the manufacturers. There will again be an industry grant for conference for one lucky respondent which consists of free registration and accommodation to next year's conference, as well as a contribution toward travel to get there. Just keep in mind that if you have an issue with your equipment and have not reported it to the manufacturer or to the watchdog@artp.org.uk, it is not fair to judge them for that.

MR

COMPANY NEWS:

Baywater Healthcare has completed the acquisition of Intus Healthcare, a leading online provider of therapeutic medical products and diagnostic studies for the treatment of sleep disordered breathing.

Intus, founded by the current Managing Director Martin Heller over ten years ago, has established a niche position in the UK healthcare market, supplying both private patients and NHS sleep clinics with a wide variety of equipment to treat Obstructive Sleep Apnoea. This transaction confirms Baywater Healthcare's desire to expand their expertise into the online private healthcare and sleep markets.

Adam Sullivan, Baywater Healthcare's CEO commented; "I've *known Martin for several years and admired how he has created a very successful business in this growing sector using his entrepreneurial flair. We look forward to working closely with the Intus team to continue the fantastic work they have done in delivering such impressive growth.*"

Martin Heller stated; "*Baywater Healthcare was a natural fit for my business, the company possess very similar values to ours and are committed to developing innovative solutions for patients. People are now really starting to understand how*

important sleep is to maintaining good health and the treatment model we have developed over many years is perfectly positioned to support the increasing need for these services in the future."

Acquired by French healthcare specialist Bastide Groupe in January 2018, Baywater Healthcare supports over 26,000 patients across the UK, supplying a range of respiratory therapies and associated services.

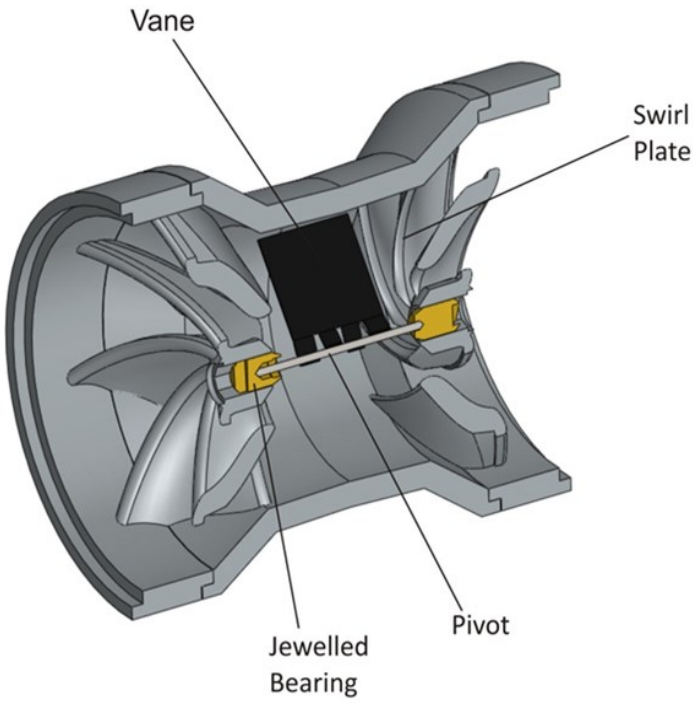
The next article was provided by Mark Wilson from Numed Healthcare, distributor for the Medchip Spiroconnect. Turbine transducers have had a historical problem measuring low flow, Mark has described how their new device overcomes this. At the conference in Brighton their stand had an excellent demonstration of these principles at work.

MR

New Spirometry Turbine Transducer – Low Flow Problem Solved.

The use of turbine technology for spirometers was first described in 1982[1] and has become popular in general practice.

The turbine transducer consists of two fixed swirl plates at either end of a short tube. Between the swirl plates a low mass vane on a pivot is supported by two jewelled bearings:



As air flows through the turbine a vortex is created by the swirl plates and the vane rotates within the vortex. The rotation of the vane is detected by the interruption of an infra-red beam passing through the turbine. The number and rate of interruptions is proportional to the volume and flow through the turbine respectively.

The number of interruptions per litre depends on the geometry of the swirl plates and therefore the calibration is very stable[2] and is also largely independent of air density and altitude[3].

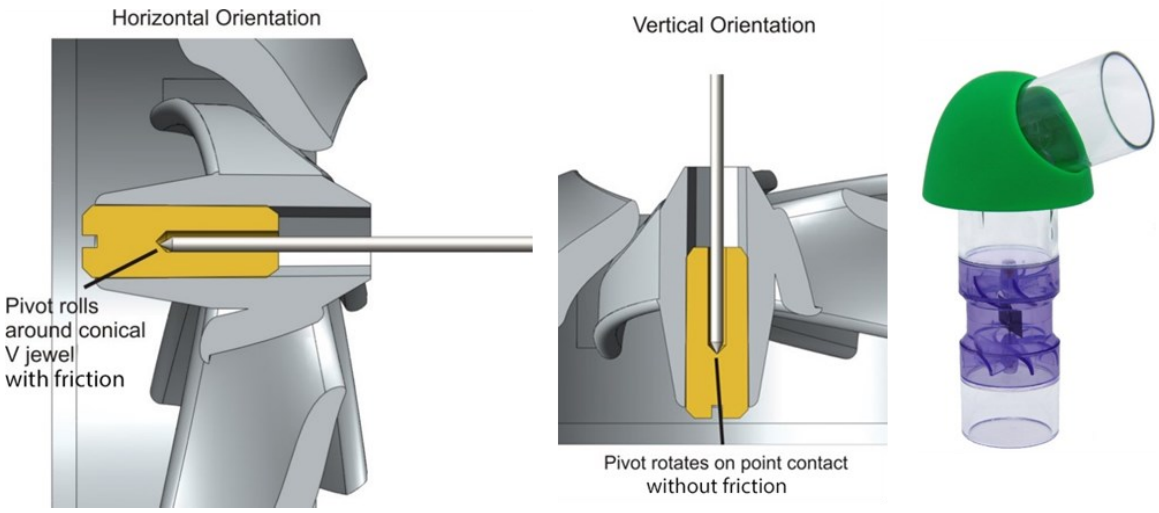
A turbine vane will stop rotating when the torque on the vane due to the vortex is balanced by opposing forces of friction in the bearings, torque required to rotate the vane against gravity, and static attraction between the vane and swirl plates. These forces are insignificant at high flows but dominate at very low flows.

It is known that many turbines only respond to flows well in excess of 0.025l/s and therefore fail to comply with international standards[4].

ON THE BLOWER

The patented[5] design of the new SpiroConnect turbine (MedChip Solutions Ltd, Kent, UK) reduces all of the opposing forces that limit the low flow sensitivity of other turbine designs:

1. Friction in the bearings is reduced by orientating the turbine vertically.
2. Torque required to lift the vane against gravity is also eliminated as the vane now rotates in a horizontal plane.
3. Static attraction between the vane and the segments of the swirl plates has been eliminated by using transparent anti-static plastic for the construction.



All of these innovations have resulted in a turbine performance that now exceeds the low flow sensitivity requirements of all international standards.

If you are interested in a demonstration of this improved low flow response, contact Mark Wilson at Numed Healthcare - mark.wilson@numed.co.uk

SpiroConnect is a PC based spirometer that can be used with a desktop or laptop computer. There is also an App for use on an Android tablet or phone, which is ideal for community or hospital ward spirometry testing. Full integration with the SystmOne, EMIS and Vision clinical systems is also available through Numed's Intelligent Integration Interface (i3) for safer, faster and easier spirometry testing. Visit www.numed.co.uk or call 0114 2433896 for more details.

1. Pocket-sized device for measuring forced expiratory volume in one second and forced vital capacity. Chowienczyk PJ, Lawson CP. Br Med J (Clin Res Ed).1982 Jul 3;285(6334):15-7.
2. Long term performance of a handheld spirometer. Asger Dirksen, Flemming Madsen, Ole Find Pedersen, Anne Mette Vedel, Axel Kok-Jensen Thorax 1996;51:973-976
3. Portable peak flow meters: physical characteristics, influence of temperature, altitude, and humidity. O.F. Pedersen, M.R. Miller, T. Sigsgaard, M. Tidley, R.M. Harding Eur Respir J, 1994, 7, 991–997 DOI: 10.1183/09031936.94.07050991
4. Standardisation of Spirometry. Eur Respir J 2005; 26: 319–338
5. The following patents rights apply to the vertical turbine technology: European Patent Application Number 13724330.9 | GB Patent Number 2500893

ON THE BLOWER

PRODUCT UPDATES:

Konica Minolta Pulsox Announcement

Drive DeVilbiss Health regret to announcement that the Konica Minolta Pulsox range is being made obsolete with immediate effect (July 2018).

Konica Minolta have made the decision to cease manufacture of the popular Pulse Oximetry range. The Oximetry range is a small, niche product group for Konica Minolta and they are focussing on their core businesses.

Drive DeVilbiss Healthcare will still continue to support and maintain all devices purchased from them. They have very limited stocks left of the oximetry units and probes, however supplies may still be available from other resellers.

At present a replacement has not been found that matches the quality and value of the Konica Minolta Pulsox range but a notification will be sent out when a suitable product has been sourced.

Drive DeVilbiss would like to thank customers for purchasing the range over the years and for their understanding with this matter.

If you have any questions or would like to know current stock availability please contact 0845 0600 333 or info@drivedevilbiss.co.uk

NEW 10 Litre Oxygen Concentrator

Weighing only 19 kg, the 10 Litre Oxygen Concentrator is one of the smallest, most compact high-volume concentrators on the market, perfect for patients who require more than 5 lpm of oxygen. Equipped with the exclusive DeVilbiss Oxygen Sensing Device (OSD®) for monitoring oxygen produced, this concentrator helps to ensure patient safety and product reliability.

Other key features are;

- Utilises the same footprint as the popular 5 litre 525 oxygen concentrator, ensuring that the 10 litre concentrator is one of the most compact and lightweight models available today.
- Oxygen outlet incorporating a fire protection adapter for patient safety.
- Assembled in the USA and utilises approximately 80% of the same reliable components used for the 5 litre 525 oxygen concentrator.
- Can easily incorporate a compatible transfill system, via the auxiliary oxygen port and compatible Transfill Caddy
- Visual and audible alarms built into the device indicate – power failure, high

gas temperature, high pressure, low/high flow, low oxygen and service required.

For more information on the 10 Litre Oxygen Concentrator, please contact Ian Swapp on 07734 720449 or ian.swapp@drivedevilbiss.co.uk.

ON THE BLOWER

RemServe Medical are excited to bring a new product line to the UK market with the iBreeze series from ResVent. As well as the humidifier, the iBreeze series also features a 5.0 inch touch screen panel on the BiLevel model which makes navigating the basic menu system a breeze. Whilst including a menu dial (both CPAP and BPAP) for traditional access, there is also Bluetooth & WIFI functionality which allows access and control of the device via smart devices and computers.

Quoted as “Smart. Comfortable. Quiet” it’s easy to see that they have spent a lot of time perfecting their product line. The integrated humidifier being an excellent feature especially when considering the size and general aesthetics of the devices. A great feature of the humidifier is its ability to actively monitor the water level and once emptied it will deactivate without any interaction from the patient/user.

ResVent have also developed a great piece of software (“iMatrix”) which can be accessed by patients and clinicians. This software will allow easy access to data collected from the device which can be held on a cloud data server, as well as the option to store it on an SD Card. This information can be accessed via an app available for tablets/smart devices as well as on desktop PCs/laptops.

FROM THE MUSEUM

A strip of paper the width of the drum is held above a smoking gas flame, the smoke from which settles on the paper to leave a surface of carbon soot. The smoked strip is wrapped around the drum and secured in place. The drum is set to run at a suitable speed (speeds available of 1, 10, 25, or 640 $mm.s^{-1}$) and a stylus from a water spirometer (or other physiological recording device) is held against the strip. The recording is 'scored' into the carbon surface. Afterwards a calibration mark is applied and the strip is removed and passed through a 'fixing fluid' to create a permanent record that, after drying can be measured. It was a convenient way to record physiological signals but could be a very mucky job!

Image by Dr. David Chinn

Sherrington Stirling Kymograph Recording Drum (C F Palmer (London) Ltd), c. 1955

Present YOUR old LFT equipment in the ARTP virtual museum? Inspire@artp.org.uk

ASSORTED READING

European Lung Foundation—Hypoxic Challenge Test: http://www.europeanlung.org/en/lung-disease-and-information/air-travel/fit-to-fly

Smokers forced to quit on their own after funding cuts https://www.theguardian.com/society/2018/jul/15/smokers-prescriptions-nhs-funding-cuts-england

'"Disneyland for Big Tobacco': how Indonesia's lax smoking laws are helping next generation to get hooked" — http://theconversation.com/disneyland-for-big-tobacco-how-indonesias-lax-smoking-laws-are-helping-next-generation-to-get-hooked-97489

TOP TWEETS

NHS at 70 - The Story of our Lives @NHSat70 Jul 5

Thanks to **#NationalLottery** players, we've received £785,000 to create the first shared social history of the NHS. We are absolutely thrilled to share this funding news with you on the 70th anniversary of the NHS.

The Lancet **@TheLancet** Jul 5

"NHS history affirms that things can be made better. It can inspire us to address longstanding problems & inequities. And it testifies that the future is always to play for". Stephanie Snow, Director @NHSat70

BTS **@BTSrespiratory** Jul 4
Lung disease is still one of the top 3 killer diseases in the UK **@lunguk @asthmauk @NACAPaudit @RespTrainees** https://tinyurl.com/ybdfr9zm **#NHS70 #RespisBest** https://twitter.com/BTSrespiratory

BTS **@BTSrespiratory** Jul 4
Lung disease is still one of the top 3 killer diseases in the UK.

ARTP Retweeted Wessex CLAHRC RESP **@WesxCLAHRCResp** Jul 2

Well done NHS Wales...forward thinking...every GP practice in Wales has or will be offered funding for 1 member of staff to attend accredited spirometry training and are offered new spirometry equipment.

ARTP Retweeted ERS publications **@ERSpublications** Jun 22

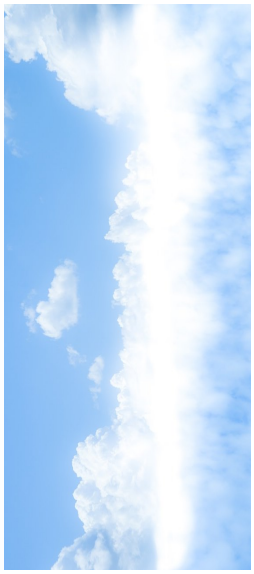
Nasal NO is feasible in infants, increases from 0–2 years and is suppressed to PCD levels by respiratory infections http://ow.ly/7AsR30jPMSf

http://erj.ersjournals.com/content/51/6/1702503?ctkey=shareline

@ThoraxBMJ Jul 2
Inspiratory muscle training in COPD: can data finally beat emotion?

By Suhilla Hashimi

https://thorax.bmj.com/content/early/2018/06/26/thoraxjnl-2018-212070?papetoc=

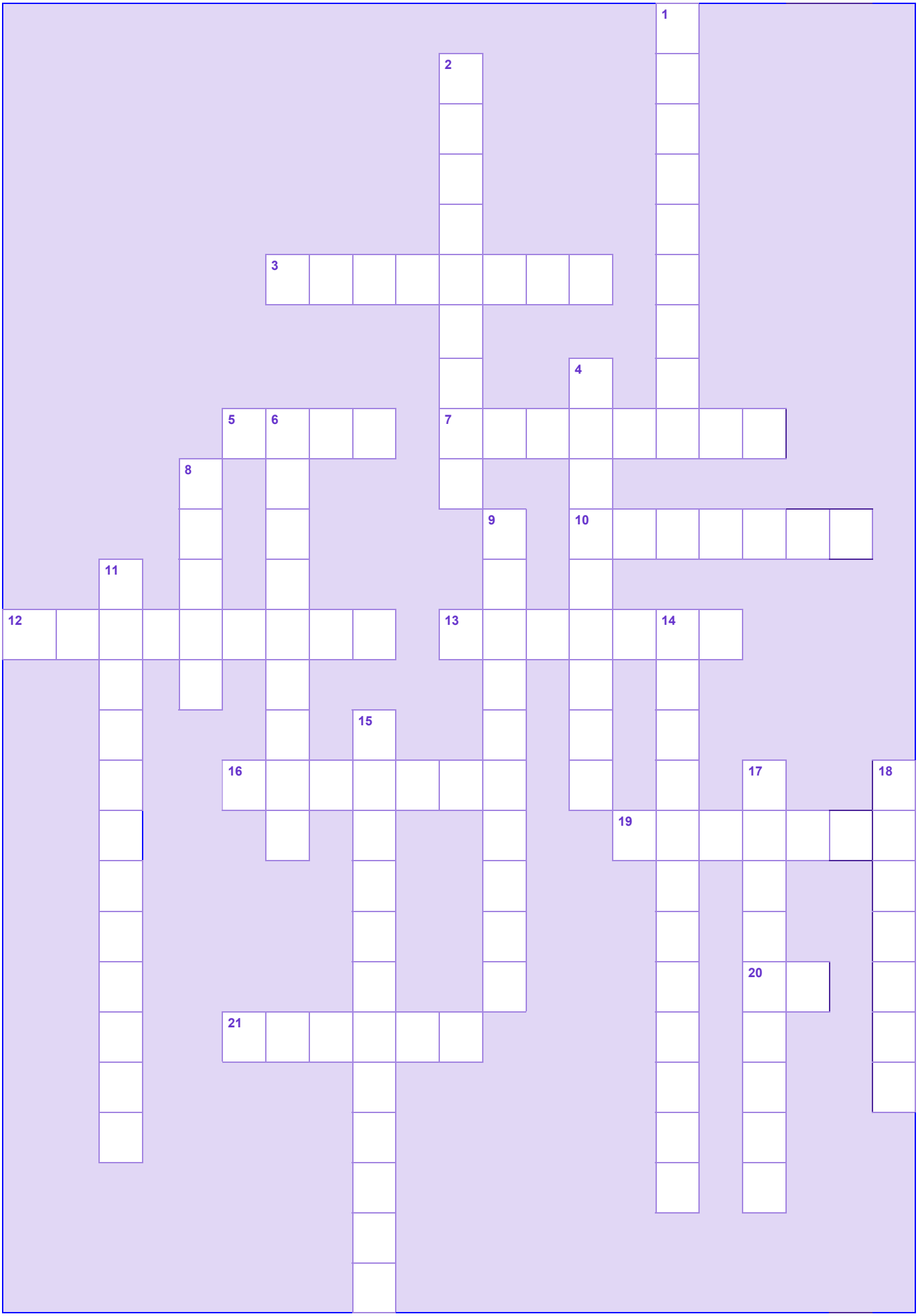


FROM THE JOURNALS

- Making sense of telemedicine in the management of COPD. Jean Bourbeau and Raquel Farias Eur Respir J 2018 51.http://erj.ersjournals.com/content/51/5/1800851?etoc
- Putting the brake on accelerated lung function decline in asthma. Dominic Shaw. Eur Respir J 2018 51. http://erj.ersjournals.com/content/51/2/1702630?etoc
- Does physical fitness enhance lung function in children and young adults?. Robert J. Hancox and Finn Rasmussen. Eur Respir J 2018 51: http://erj.ersjournals.com/content/51/2/1701374?etoc

ARTP CONFERENCE 2019. REGISTRATION NOW OPEN

INSPIRE HOLIDAY SPECIAL CRYPTIC CROSSWORD



CLUES

Across

3. About Look for investigation? (8)

5. Initially Clever People Ate Plums as an airway splint (4)

7. Absence of South Eastern attachment gives minor lung function equipment (8)

10. Airway is a mixed-up Royal Academy cheat (7)

12. In accordance with synthesis of the passage of blood? (9)

13. Order of the Empire for what could be London and overweight (7)

16. Is Nula able to create a thin tube? (7)

19. They inserted spoken lyrics for treatment (7)

20. Short for telebision? (2)

21. Sounds like sister's spasmodic twitch commonly mentioned with fibrosis (6)

Down

1. Sounds like Larry and Jill are related to the voice box (9)

2. There's a jumbled atheism in allergy (9)

4. Well-shaped American sister has a cough? (9)

6. An icy ship transported medic (9)

8. Contagion contained in Henry VI Russia (5)

9. Toil a Conservative for a lung function workplace (10)

11. Big Ron decided to copy his airway examination (12)

14. Chatty romeos can help you breathe in adversity (12)

15. Elitist train muddles between things (12)

17. British Rail consuming Hospital within for respiration (9)

18. Without oxygen below the 6th-century (7)

All answers related to lung function.

ARTP Association for Respiratory Technology & Physiology

Answers will be published in the December issue