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Oxygen therapy in the breathless patient

Beasley and colleagues argue persuasively that oxygen delivery to hypoxaemic patients should be optimised to provide adequate oxygen delivery while reducing the adverse effects of hyperoxaemia and preventing delay in identifying a patient with deteriorating gas exchange.¹ However, their consistent use of the term high-flow oxygen instead of high-concentration oxygen perpetuates another widespread misunderstanding regarding oxygen therapy—that oxygen flow to the patient has a consistent and predictable relationship to the fractional inspired oxygen concentration (FiO_2) delivered to the alveoli. This incorrect assumption threatens to undermine the wisdom and potential benefits of their insightful editorial.

When oxygen is delivered by nasal cannula, Hudson mask or reservoir bag mask, the inspiratory flow generated by the patient will generally exceed the oxygen flow delivered.² Room air is entrained by the patient and thus the inspired oxygen is diluted. During the expiratory phase, oxygen flow continues and has a variable and unpredictable effect of flushing exhaled gases from the device and filling the upper airways with high concentration oxygen.

Therefore, as respiratory rate, inspiratory flow and tidal volume change, so does the FiO_2 arriving in the patient's alveoli. These devices are referred to as "variable performance devices". Venturi systems blend oxygen and gas at a fixed ratio and the total gas flow delivered to the patient usually exceeds inspiratory flow when FiO_2 is <40% (fig 1). These devices, along with gas blenders and mechanical ventilators, are "fixed performance".

In the case example shown on page 841 of the editorial by Beasley *et al*,¹ the theoretical patient is provided in part (a) with an FiO_2 of 0.3 (2–3 litres via nasal cannula) and subsequently (b) with an FiO_2 of 0.6 (8–10 litres via a Hudson mask). These numbers are erroneous since both employ a variable performance device.

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Author's reply

We thank Dr Fox for the important point he makes regarding the use of fixed performance devices to deliver a predetermined concentration of oxygen. However, we consider that his concerns are not central

to the theme of the commentary. In our case example, oxygen was administered by nasal prongs and a Hudson mask to describe two different approaches to oxygen therapy commonly used in the management of pneumonia. As discussed in our commentary, we contend that, in this clinical situation, the crucial issue is the titration of oxygen therapy to relieve hypoxaemia without causing hyperoxia, with continuous monitoring of the response by measurement of the oxygen saturation.

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High effectiveness of short treatment with bupropion for smoking cessation in general care

Since the appearance of the seminal publication on sustained release bupropion versus placebo for smoking cessation exactly 10 years ago,¹ bupropion has become a mainstay of nicotine addiction therapy.² Some concerns relating to increased risk of seizures remain, in particular as exclusion of predisposed patients may be suboptimal in general practice.³ Furthermore, the cost of about €135 or US\$180 for a pack of 100 pills required for a standard course of treatment of at least 7 weeks represents a substantial barrier to wider use.⁴ We present data suggesting that much shorter treatments with bupropion may be as effective as standard regimens.

In a cluster randomised trial in German general care investigating the effects of practitioner education and financial incentives for the physician or cessation drug costs reimbursement for his/her patients on smoking cessation, 577 heavy smokers (10+ cigarettes/day) aged 36–75 years and willing to participate were recruited regardless of their intentions to quit.⁴ At the 12 month follow-up, 76 participants (13%) reported having used bupropion (Zyban) during the 1 year study period. Consistent with expectations from clinical trials,² cotinine confirmed point prevalence of abstinence after 1 year in subjects who had taken bupropion was 26% (20/76). However, we observed that the majority of treated patients reported intake durations clearly below the recommendations. In particular, 25 (33%) and 34 (45%) reported having taken bupropion for only 1–2 and 3–4 weeks, respectively. Intriguingly, in adjusted analyses, strong and significant associations of bupropion with cessation were evident only in the two categories representing treatment clearly shorter than current standards (table 1). This was preserved when using a stricter outcome (ie, 6 months of continuous abstinence).

Figure 1 Composition of total gas flows through a typical Venturi valve.

Table 1 Effects of self-reported use of bupropion on abstinence at the 1 year follow-up (n = 577)

Bupropion use	n (abstinent; %)	OR (95% CI)*	OR _{adj} (95% CI)†
Point abstinence after 1 year			
No bupropion	501 (36; 7%)	1 —	1 —
1–2 weeks	25 (7; 28%)	5.00 (1.91–13.14)	4.46 (1.54–12.90)
3–4 weeks	34 (10; 29%)	5.27 (2.03–13.69)	4.49 (1.66–12.17)
5+ weeks	17 (3; 18%)	2.74 (0.72–10.47)	1.30 (0.25–6.75)
6 months continuous abstinence			
No bupropion	501 (19; 4%)	1 —	1 —
1–2 weeks	25 (4; 16%)	4.56 (1.31–15.91)	4.01 (1.10–14.67)
3–4 weeks	34 (7; 21%)	5.57 (1.64–18.88)	5.89 (1.79–19.37)
5+ weeks	17 (3; 18%)	5.08 (1.23–21.06)	2.67 (0.49–14.61)

From logistic regression models predicting abstinence (SAS 9.1, SAS Institute, Cary, North Carolina, USA).

*Unadjusted models included a random effect accounting for the correlation between observations originating from some smokers being treated in the same practitioner office, which was negligible (converged to 0) in adjusted analyses.

†Adjusted for intervention “physician education and medication costs reimbursement” and established cessation predictors: Fagerström nicotine dependence score, smoking stage of change, nicotine replacement therapy (self-reported), intensity of smoking, education, marital status, having a smoking partner, history of cancer, myocardial infarction or stroke. n = 497 because of missing values in covariates.

While the overall high odds ratios for bupropion must be seen in relation to the low abstinence rate in the reference group, we consider it a highly interesting observation that very short treatment courses appeared as effective, or more effective, as standard bupropion regimens. The established standard duration appears to be primarily based on experience from nicotine replacement therapy,^{1,2} and we are aware of only a single, somewhat inconclusive, attempt to systematically investigate the performance of shorter regimens, which suggested equivalence of 1 and 2 months of treatment without reporting quantitative results.⁵ Our own study cannot

rule out some distortion of effect estimates caused by residual confounding or recall bias, although it seems unlikely that such phenomena would be the main cause for our results. Possible explanations, such as bupropion refractory patients frustratingly continuing treatment, remain speculative. Given the potential reduction in seizure risk and substantial economic benefits associated with shorter treatment at the individual and community level, we believe that evaluation of short and more affordable regimens of bupropion for smoking cessation is warranted and should be adequately addressed in randomised clinical trials.

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