

better of the two devices in identifying minutes of moderate intense physical activity.

D Langer,^{1,2} R Gosselink,^{1,2} R Sena,² C Burtin,^{1,2} M Decramer,^{1,2} T Troosters^{1,2}

¹ Respiratory Rehabilitation and Respiratory Division, UZ Gasthuisberg, Leuven, Belgium; ² Faculty of Kinesiology and Rehabilitation Sciences, Katholieke Universiteit Leuven, Leuven, Belgium

Correspondence to: Dr T Troosters, UZ Gasthuisberg, Respiratory Division, Herestraat 49, B-3000 Leuven, Belgium; thierry.troosters@med.kuleuven.be

Funding: This research was funded by Research Foundation Flanders grants G0523.06 and G05989.09

Competing interests: None.

DL and CB are doctoral fellows of the Research Foundation Flanders.

► Additional data are published online only at <http://thorax.bmj.com/content/vol64/issue7>

Accepted 17 March 2009

Thorax 2009;64:641–642. doi:10.1136/thx.2008.112102

REFERENCES

1. **Morgan M.** Life in slow motion: quantifying physical activity in COPD. *Thorax* 2008;63:663–4.
2. **Pitta F, Troosters T, Probst VS, et al.** Quantifying physical activity in daily life with questionnaires and motion sensors in COPD. *Eur Respir J* 2006;27:1040–55.
3. **Pitta F, Troosters T, Spruit MA, et al.** Characteristics of physical activities in daily life in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2005;171:972–7.
4. **Walker PP, Burnett A, Flavahan PW, et al.** Lower limb activity and its determinants in COPD. *Thorax* 2008;63:683–9.
5. **Haskell WL, Lee IM, Pate RR, et al.** Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. *Circulation* 2007;116:1081–93.
6. **Nelson ME, Rejeski WJ, Blair SN, et al.** Physical activity and public health in older adults: recommendation from the American College of Sports Medicine and the American Heart Association. *Circulation* 2007;116:1094–105.

Epoprostenol use in a National Pulmonary Hypertension Centre from 1997 to 2007

The cost-effectiveness of prostanoids in pulmonary arterial hypertension (PAH) has recently been called into question by the National Institute for Health and Clinical Excellence (NICE),¹ and the possibility exists that this treatment would not be recommended by this body. This would be the first time that a treatment already in routine clinical practice would be withdrawn as a result of NICE recommendations. Guidelines published by the UK, European and US authorities still advocate prostanoid use in certain patient groups.^{2–4} Of the disease-targeted therapy available for PAH, only epoprostenol has been shown to improve patient survival in the context of a randomised controlled trial.⁵

To assess the impact that withdrawal of intravenous epoprostenol in 1997 would have had, its use was retrospectively reviewed in a well-defined population of

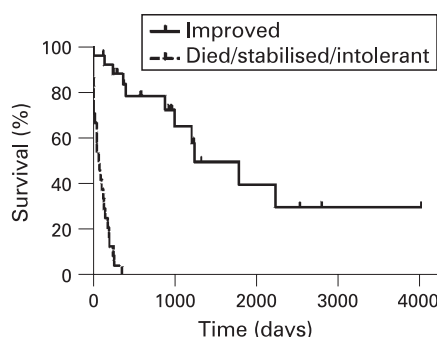


Figure 1 Survival from time of initiation of epoprostenol in the entire cohort.

patients with PAH. This analysis includes eras before and after the licensing of oral agents in 2002.

All patients treated with intravenous epoprostenol by the Scottish Pulmonary Vascular Unit from 1997 to 2007 were identified. Case notes of this cohort were reviewed to determine (1) the reason for initiation of epoprostenol, (2) the outcome after 12 months of treatment as assessed by changes in conventional outcome measures and survival, and (3) the outcome in those patients whose treatment with epoprostenol commenced after licensing of oral disease-targeted therapy in 2002. Patient improvement was defined as either an improvement in functional class or an improvement in the 6-minute walk distance (6MWD) 3 months after initiation of treatment. Patients were then divided into the following groups: (1) improvement and survived >12 months; (2) stabilised (ie, no improvement but survived >12 months); (3) died within 12 months; or (4) intolerant of treatment. Where available, N-terminal proB type natriuretic peptide (NT-proBNP) and quality of life (QOL) measurements were also analysed.

Fifty (20%) of 252 patients with PAH were identified as having received epoprostenol during this period. Of this group, 30 (60%) either improved or stabilised for >12 months. Data on the improvements in 6MWD, QOL, functional class and NT-proBNP are included in the online supplement. Figure 1 shows the survival advantage for patients whose functional class or 6MWD improved with intravenous epoprostenol.

For the subset of patients following the introduction of oral disease-targeted therapy agents in 2002, 30 (15%) out of 199 required epoprostenol; 17 (57%) improved, 9 (30%) died before 12 months, 3 (10%) stabilised and 1 was intolerant of the agent. Included in this group were 12 patients who required epoprostenol after deterioration on oral agents; 7 improved, 3 died, 1 stabilised and 1 was intolerant. Data on the improvements in 6MWD, QOL, functional class and NT-proBNP in this group of patients are also shown in the online supplement.

Point prevalence data (see online supplement) still demonstrated an ongoing need for epoprostenol in the current era of oral agents.

This study suggests that, had intravenous epoprostenol not been available over the last decade as a treatment for PAH, a significant number of patients in Scotland would have been denied an effective treatment. Where data are available in the subjects, there were improvements in all outcome measures (functional class, 6MWD, NT-proBNP, QOL). More compelling, however, is the survival analysis of the entire cohort which demonstrated a large survival advantage in those patients in whom functional class or 6MWD improved. This situation remains true in the era of oral treatments. This should have implications for future NICE review of the role of epoprostenol in PAH.

A C Church, D Sanabria, A Peacock, M Johnson

Scottish Pulmonary Vascular Unit, Glasgow, UK

Correspondence to: Dr A C Church, Golden Jubilee National Hospital, Beardsmore Street, Glasgow G61 2DZ, UK; colinchurch@doctors.org.uk

Funding: National Services Division.

Competing interests: None.

► Additional data are published online only at <http://thorax.bmj.com/content/vol64/issue7>

Accepted 26 March 2009

Thorax 2009;64:642. doi:10.1136/thx.2008.110643

REFERENCES

1. **National Institute for Health and Clinical Excellence (NICE).** Appraisal document. London: NICE, 2008.
2. **National Pulmonary Hypertension Centres of the UK and Ireland.** Consensus statement on the management of pulmonary hypertension in clinical practice in the UK and Ireland. *Thorax* 2008;63(Suppl 2):ii1–41.
3. **Badesch DB, Abman SH, Ahearn GS, et al.** Medical therapy for pulmonary arterial hypertension: ACCP evidence-based clinical practice guidelines. *Chest* 2004;126(1 Suppl):35–62S.
4. **Galie N, Torbicki A, Barst R, et al.** Guidelines on diagnosis and treatment of pulmonary arterial hypertension. The Task Force on Diagnosis and Treatment of Pulmonary Arterial Hypertension of the European Society of Cardiology. *Eur Heart J* 2004;25:2243–78.
5. **Barst RJ, Rubin LJ, Long WA, et al.** A comparison of continuous intravenous epoprostenol (prostacyclin) with conventional therapy for primary pulmonary hypertension. The Primary Pulmonary Hypertension Study Group. *N Engl J Med* 1996;334:296–302.

Do all occupational respiratory sensitisers follow the united airways disease model?

Castano *et al*¹ applied specific inhalational challenge testing to examine the important relationship between occupational rhinitis (OR) and occupational asthma (OA) in 43 subjects. Using this approach they found that OR occurred in 76.4% of confirmed cases of OA, and this provides strong evidence that the united airways disease