Supplies of the Cardiff symphysis-fundus chart may be obtained from Titan Press Ltd, Wroughton Place, Fairwater, Cardiff (tel: 0222-569321).

Requests for reprints should be sent to Dr J P Calvert, lecturer in obstetrics and gynaecology, Welsh National School of Medicine, Heath Park, Cardiff CF4 4XN.

References

- Rumboltz WL, McGoogan LS. Placental insufficiency and the small undernourished full-term infant. *Obstet Gynecol* 1953;**1**:294-301.
- Beazley JM, Underhill RA. Fallacy of the fundal height. *Br Med J* 1970; iv:404-6.
- Campbell S. Fetal growth. In: Beard RW, Nathanielsz PW, eds. *Fetal physiology and medicine*. London: W B Saunders, 1976:278.
- Tejani N, Mann LI, Weiss RR. Antenatal diagnosis and management of the small-for-gestational-age fetus. *Obstet Gynecol* 1976;**47**:31-6.
- Rosenberg K, Grant JM, Hepburn M. Antenatal detection of growth retardation: actual practice in a large maternity hospital. *Br J Obstet Gynaecol* 1982;**89**:12-5.
- Hall MH, Chng PK, MacGillivray I. Is routine antenatal care worth while? *Lancet* 1980;ii:78-80.
- Effer SB. Perinatal intensive care: obstetrical considerations. In: Goodwin JW, Godden JO, Chance GW, eds. *Perinatal medicine*. Baltimore: Williams and Wilkins, 1976:578-92.
- Westin B. Gravidoqram and fetal growth. *Acta Obstet Gynecol Scand* 1977;**56**:273-82.
- Belizan JM, Villar J, Nardin JC, Malamud J, de Vicuna LS. Diagnosis of intra-uterine growth retardation by a simple clinical method: measurement of uterine height. *Am J Obstet Gynecol* 1978;**131**:643-6.
- Quaranta P, Currell R, Redman CJ, Robinson JS. Prediction of small-for-dates infants by measurement of the symphysial-fundal-height. *Br J Obstet Gynaecol* 1981;**88**:115-9.
- Thomson AM, Billewicz WZ, Hytten FE. The assessment of fetal growth. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1968; **75**:903-16.
- Nie NH, Hull CH, Jenkins JG, Steinbrenner K, Bent DH. *Statistical package for the social sciences*. 2nd ed. New York: McGraw Hill, 1975.

(Accepted 22 July 1982)

SHORT REPORTS

Terbutaline aerosol given through pear spacer in acute severe asthma

Pressurised aerosols are an ineffective way of giving bronchodilators in acute severe asthma. Less than 10% of a correctly administered dose from a standard pressurised aerosol reaches the bronchial tree,¹ and this may be reduced in acute severe asthma. Even under normal circumstances not more than 75% of patients are able to use pressurised aerosols efficiently.² Those who are unable to co-ordinate aerosol actuation with inhalation obtain benefit from the addition of an extension tube or spacer to the mouthpiece of the aerosol.³ The pear spacer (Nebuhaler) is a plastic tube (750 ml volume) with a one-way valve that obviates the need for co-ordination. It is designed to contain the shape of the aerosol cloud when it leaves the actuator and increase the availability of the drug for inhalation. Newman *et al*⁴ showed that the deposition of radio aerosol in the whole lung is improved from 7.8% to 11.5% with a tube spacer and to 13% with a pear spacer.⁴

We used a pear spacer to deliver an aerosol bronchodilator (terbutaline) to patients with acute severe asthma and compared it with an electric nebuliser.

Patients, methods, and results

We studied 18 patients (11 women, seven men; age 15-67 years). All gave informed consent and satisfied the following: pulse rate 110/min or greater; forced expiratory volume in one second 25% predicted or less; arterial oxygen pressure 9.3 kPa (70 mm Hg) or less; and forced expiratory volume in one second and forced vital capacity (best of three) measured on admission and 10 minutes later not varying by more than 10%. Patients who had received treatment with a bronchodilator within the previous two hours were excluded. Intravenous hydrocortisone 200 mg was given at the start of the study and oxygen administered throughout.

Patients were randomly allocated into two groups: one group received terbutaline 4 mg via an electric nebuliser (RTU 4; Medic Aid Ltd) followed after 30 and 60 minutes by two doses of terbutaline 2 mg (eight puffs) via a Nebuhaler; the second group received terbutaline 4 mg (16 puffs) via a Nebuhaler followed by two doses of 2 mg via a nebuliser 30 and 60 minutes later.

Forced expiratory volume in one second, forced vital capacity (Vitalograph dry spirometer), pulse, and blood pressure were measured on admission and 10 minutes later immediately before the first administration. Forced expiratory volume in one second, forced vital capacity, and pulse were recorded immediately after each administration and at 10-minute intervals until a plateau was reached. The blood pressure was recorded after 30 minutes and at the end of the study. The figure shows the mean baseline values after each administration of terbutaline.

The data were subjected to analysis of variance. Each treatment produced a significant rise in forced expiratory volume in one second and forced vital capacity ($p < 0.01$), but there was no significant difference between the two

modes of administration. Pulse rate and blood pressure tended to fall throughout the study, and no adverse side effects were noted despite a total of 8 mg inhaled terbutaline being given.

Mean baseline values and mean maximum values of heart rate, forced expiratory volume in one second (FEV₁), and forced vital capacity (FVC) after each administration of terbutaline.

Comment

In severe asthma a bronchodilator given by any route may produce a fall in arterial oxygen partial pressure. Oxygen should be given before, during, and after its administration, as it was in this study.

Nebulised terbutaline produces just as effective bronchodilation as the intravenous drug in acute severe asthma.⁵ In our patients terbutaline administered via a conventional pressurised aerosol with a Nebuhaler attached was as effective as wet nebulisation of the drug. The Nebuhaler requires no power supply and is smaller, less complicated, easier to assemble, and vastly less expensive than a nebuliser. We recommend its use in the treatment of acute severe asthma both in the home and in hospital.

¹ Davies DS. Pharmacokinetics of inhaled substances. *Postgrad Med J* 1975; **51**, suppl 7:69-75.

² Earis JE, Bernstein A. Misuse of pressurised nebulisers. *Br Med J* 1978; **i**:1554.

- ³ Godden DJ, Crompton GK. An objective assessment of the tube spacer in patients unable to use a conventional pressurised aerosol efficiently. *Br J Dis Chest* 1981;**75**:165-8.
- ⁴ Newman SP, Moren F, Pavia D, Little F, Clzrke SW. Deposition of pressurised suspension aerosols inhaled through extension devices. *Am Rev Res Dis* 1981;**124**:317-20.
- ⁵ Williams SJ, Winner SJ, Clark TJH. Comparison of inhaled and intravenous terbutaline in acute severe asthma. *Thorax* 1981;**36**:629-31.

(Accepted 28 May 1982)

Stoke Mandeville Hospital, Aylesbury, Bucks HP21 8AL

M D L MORGAN, MB, MRCP, registrar
B V SINGH, MB, BS, senior house officer
M H FRAME, PHD, clinical scientist, Astra clinical research unit
S J WILLIAMS, MB, MRCP, consultant physician

Antihypertensive treatment with beta-blockers in patients aged over 65

The use of beta-blockers in elderly patients with hypertension has been questioned owing to the increased incidence of heart failure in this age group. As our clinical impression at the outpatient hypertension clinic was that beta-blockers were well tolerated by elderly patients,¹ we decided to analyse the use and tolerability of beta-blockers in all patients over 65 who were being treated with these drugs, with special reference to the occurrence of heart failure.

Patients, methods, and results

A total of 301 patients aged over 65 years were being treated at the clinic. Two hundred and seventeen of these were aged from 65 to 69, 58 from 70 to 74, and 26 over 75.

Each year the doctors complete a computer record form for each patient with standardised questions about the case history, current antihypertensive treatment, side effects, and reasons for changes in treatment. Dyspnoea on exertion was diagnosed as heart failure if treatment with diuretics or digitalis was indicated.

Analysis of the prescription of beta-blockers in the different groups showed that three-quarters of the patients from 65 to 74 years and half of those aged over 75 were receiving beta-blockade. Analysis of the total treatment showed that 42 (14%) were receiving beta-blockers alone, 51 (17%) were receiving diuretics alone, and 193 (64%) were receiving combined treatment with beta-blockers and diuretics or other drug combinations; 15 (5%) were receiving no treatment. The mean dose for the two most commonly used beta-blockers was 189 mg/day for the beta₁-selective metoprolol and 252 mg/day for the non-selective propranolol.

Analysis of the blood pressures showed good pressure control of 156/91 mm Hg in those aged 65-69, 166/94 mm Hg in those aged 70-74, and 174/93 mm Hg in those aged over 75.

Changes of the beta-blocker medication fell into three groups: discontinuation (21 patients (10%)), reduction of the dose (12 patients (5.5%)), and change to another type (49 patients (22.5%))—for example, from a non-selective to a beta₁-selective blocker (30 patients (14%)). The reasons for changing from a non-selective to a beta₁-selective blocker were similar to those leading to withdrawal of beta-blockade (see table).

Heart failure was diagnosed in 23 patients, of whom 16 (70%) were on beta-blockade in combination with diuretics, digitalis, or both.

Reason for withdrawal of beta-blockers

	No	%
Sleep disturbances	1	0.5
Gastrointestinal symptoms	2	1.0
Obstructive lung disease and/or dyspnoea	3	1.4
Tiredness	2	1.0
Exanthema	2	1.0
Cold hands or feet	3	0.5
Bradycardia	1	0.5
Heart failure (without digitalis)*	1	0.5
Heart failure (on digitalis)	1	0.5
Diabetes mellitus	4	1.8
Reason not stated	4	1.8
Total	21	9.7

* Treatment with a beta₁-selective blocker was resumed after giving the patient digitalis.

Comments

This analysis showed that the beta-blockers were generally well tolerated in hypertensive patients over 65. The side effects and patterns of dosage resembled those seen in younger age groups. Heart failure was the reason for discontinuing the treatment in only one patient. Most of the side effects could be overcome by changing from a non-selective to a beta₁-selective blocker or by reducing the dose.

Heart failure was diagnosed in 23 patients (11%) and of these patients 16 (70%) were receiving beta-blockade in combination with diuretics or digitalis. In many of these patients symptoms of heart failure might have been due not to bad systolic contraction but to poor filling in stiff hearts. Even at heart rates as low as 120 beats/min the filling time becomes critical to the elderly heart.² If the heart rate falls from 100 to 70 beats/min the diastolic filling time increases by 100% without the ejection time being affected.³ This favourable effect of beta-blockers on the diastolic filling is probably particularly important in the aged, stiff, hypertensive heart.

Beta-blockers have been claimed to reduce the incidence of coronary heart disease.⁴ If good efficacy and tolerance is achieved with beta-blockers alone or in combination with other drugs in elderly patients with hypertension this could be important since the incidence of coronary heart disease is increasing with age in both men and women and since coronary heart disease is the most common end-point in hypertension.⁵

¹ Andersson O, Berglund G, Hansson L, *et al.* Organisation and efficacy of an outpatient hypertension clinic. *Acta Med Scand* 1978;**203**:391-8.

² Harrison TR, Dixon K, Russel RO Jr, Bidwai PS, Coleman HN. The relation of age to the duration of contraction, ejection, and relaxation of the normal human heart. *Am Heart J* 1964;**67**:189-99.

³ Rothe CF. Cardiodynamics. In: Selkurt EE, ed. *Physiology*. 4th ed. Boston: Little, Brown and Co, 1976:338.

⁴ Dollery CT. Does it matter how blood pressure is reduced? *Clin Sci* 1981;**61**:413-20.

⁵ Berglund G, Sannerstedt R, Andersson P, *et al.* Coronary heart-disease after treatment of hypertension. *Lancet* 1978;**i**:1-5.

(Accepted 11 June 1982)

Outpatient Hypertension Clinic, Department of Medicine I, Sahlgrenska Hospital, University of Göteborg, Sweden

JOHN WIKSTRAND, MD, associate professor of clinical physiology
GÖRAN BERGLUND, MD, associate professor of medicine

High plasma calcitonin concentrations in chronic bronchitis

Studies of extrathyroidal and extralung production of calcitonin showed calcitonin-like immunoreactivity in brain and lung of lizards and pigeons.¹ Immunofluorescence studies and electron microscopy of lizard lung showed secretory granules of calcitonin-like material in what were apparently Kulchitsky cells.² Other workers had found Kulchitsky cells in human bronchial and bronchiolar epithelium and had also isolated calcitonin-like material from human lung.³

In chronic bronchitis the acini of mucous glands contain increased numbers of mucus-secreting cells. If Kulchitsky cells were similarly increased patients with chronic bronchitis might be expected to have raised circulating calcitonin concentrations. To test this hypothesis we measured plasma calcitonin concentrations in patients with mucopurulent disease and in a group of healthy, non-smoking controls. We also looked to see if there was some correlation between the plasma calcitonin concentrations and the cumulative numbers of cigarettes smoked during life.

Subjects, methods, and results

The study group comprised 45 men aged 36-78 years (mean 58.9) with chronic mucopurulent bronchitis.⁴ All were smokers or ex-smokers of cigarettes. Measurements were made during a quiescent phase of their illness when they were clear of acute infection and had stable blood gas values. None was taking any medication. Twelve healthy men aged 29-76 years (mean 55.0 years) who had never smoked served as controls.