

In acute attacks of asthma administration of bronchodilators is an important part of treatment. Nevertheless, the use of metered dose inhalers will probably be ineffective if the patient has severe airway narrowing. A wet aerosol of one of the  $\beta_2$ -selective stimulants produced by a nebuliser or in association with an intermittent positive pressure ventilator is probably the best way of producing the greatest effect with the lowest dose and the least risk of side effects.<sup>11</sup> If parenteral treatment is indicated then subcutaneous terbutaline<sup>12</sup> and intravenous salbutamol<sup>13</sup> are effective. In theory, all of these agents may adversely affect ventilation-perfusion relationships and make hypoxia worse, but severely ill patients in whom this would be clinically important should be receiving controlled oxygen treatment.<sup>14</sup> The longer-acting selective  $\beta_2$ -adrenoceptor stimulants are also useful for maintenance treatment, though it is advisable to confirm that they do produce a worthwhile improvement in lung function tests before starting such treatment. Again, aerosols are preferable, and metered-dose inhalers are convenient and safe provided the patient is warned to seek medical advice if his inhaler does not bring relief when used according to instructions. Some patients cannot synchronise inhalation with aerosol activation, or may develop inspiratory flow rates that are too low for adequate delivery of the drug; in these cases oral preparations should be used.

Stimulation of the  $\beta$ -adrenoceptors increases the activity of adenylyl cyclase and this promotes the formation of cyclic AMP within the cell and results in bronchodilatation.<sup>15</sup> Theophylline and its derivatives prevent the breakdown of cyclic AMP by inhibiting phosphodiesterase, and in adequate doses they can be effective bronchodilators,<sup>16</sup> though some preparations irritate the stomach. Theophylline and sympathomimetics in combination have additive effects,<sup>17</sup> and tablets containing theophylline, ephedrine, and a barbiturate are often popular with patients. When prescribing these preparations, however, the doctor must remember that ephedrine may precipitate urinary retention in elderly men and that barbiturates may depress the respiratory centre or increase corticosteroid requirements by inducing hepatic enzymes. Rectal aminophylline is widely used, but the hazards of rapid intravenous injection of aminophylline or uncontrolled infusion for over 24 hrs are now well recognised.<sup>18</sup>

Some patients may become resistant to sympathomimetics,<sup>19</sup> and this might be one reason for using the least popular group of bronchodilators, the parasympatholytics. Atropine and its derivatives have been widely used for centuries to relieve asthma, and a recent symposium has reviewed the place of these drugs in current treatment.<sup>20</sup> The role of the parasympathetic nervous system in the regulation of airway calibre has been shown in animals,<sup>21</sup> and atropine can prevent the responses of asthmatic patients to inhaled irritants<sup>22</sup> or even allergens.<sup>23</sup> Atropine is generally as effective a bronchodilator as isoprenaline, though its effects take longer to develop and last longer.<sup>3</sup> This made the combination of atropine and isoprenaline in aerosols rational before the development of the longer acting sympathomimetics. As with other bronchodilators, potential side effects (such as urinary retention and failure of accommodation) may be avoided by the use of aerosols. The theoretical risk of drying bronchial secretions and blocking airways with viscid sputum does not seem to be a problem in practice.<sup>24</sup> In fact, studies of both atropine<sup>25</sup> and an analogue of atropine, ipratropium bromide,<sup>26</sup> have suggested that parasympatholytics may be more effective in patients with chronic bronchitis than in patients with asthma. Further long-term studies are needed, but this type of drug may be of particular use for patients with chronic airway obstruction

who are already receiving maximal doses of sympathomimetics, or in those who find their side effects troublesome.

- <sup>1</sup> Paterson, J W, and Sheffield, G M, *BTTA Review (Tubercle)*, 1974, **4**, 25 and 61.
- <sup>2</sup> Paterson, J W, *et al*, *Lancet*, 1968, **2**, 426.
- <sup>3</sup> Chamberlain, D A, Muir, D C F, and Kennedy, K P, *Lancet*, 1962, **2**, 1019.
- <sup>4</sup> Marlin, G E, and Turner, P, *British Journal of Clinical Pharmacology*, 1975, **2**, 41.
- <sup>5</sup> Engelhardt, A von, *et al*, *Arzneimittel-Forschung*, 1961, **11**, 521.
- <sup>6</sup> Lands, A M, *et al*, *Nature*, 1967, **214**, 597.
- <sup>7</sup> Cullum, V A, *et al*, *British Journal of Pharmacology*, 1969, **35**, 141.
- <sup>8</sup> Persson, H, and Olsson, T, *Acta Medica Scandinavica*, 1970, Suppl **512**, 11.
- <sup>9</sup> *Drug and Therapeutics Bulletin*, 1972, **10**, 7.
- <sup>10</sup> *Drug and Therapeutics Bulletin*, 1973, **11**, 39.
- <sup>11</sup> Choo-Kang, Y F J, Parker, S S, and Grant, I W B, *British Medical Journal*, 1970, **4**, 465.
- <sup>12</sup> Arner, B, *et al*, *Acta Medica Scandinavica*, 1970, suppl **512**, 41.
- <sup>13</sup> Fitchett, D H, McNichol, M W, and Riordan, J F, *British Medical Journal*, 1975, **1**, 53.
- <sup>14</sup> *British Medical Journal*, 1976, **1**, 609.
- <sup>15</sup> Robinson, G A, Butcher, R W, and Sutherland, E W, *Annals of the New York Academy of Sciences*, 1967, **139**, 703.
- <sup>16</sup> Piafsky, K M, and Ogilvie, R I, *New England Journal of Medicine*, 1975, **292**, 1218.
- <sup>17</sup> Cander, L, and Comroe, J H, *Journal of Allergy*, 1955, **26**, 210.
- <sup>18</sup> *Lancet*, 1973, **2**, 950.
- <sup>19</sup> Paterson, J W, Evans, R J C, and Prime, F J, *British Journal of Diseases of the Chest*, 1971, **65**, 21.
- <sup>20</sup> *Postgraduate Medical Journal*, 1975, suppl **51**, 7.
- <sup>21</sup> Cabezas, G A, Graf, P D, and Nadel, J A, *Journal of Applied Physiology*, 1971, **31**, 651.
- <sup>22</sup> Simonsson, B G, Jacobs, F M, and Nadel, J A, *Journal of Clinical Investigation*, 1967, **46**, 1812.
- <sup>23</sup> Yu, D Y C, Galant, S P, and Gold, W M, *Journal of Applied Physiology*, 1972, **32**, 823.
- <sup>24</sup> Lopez-Vidriero, M T, *et al*, *Thorax*, 1975, **30**, 543.
- <sup>25</sup> Alliott, R J, *et al*, *British Medical Journal*, 1972, **1**, 539.
- <sup>26</sup> Petrie, G R, and Palmer, K N V, *British Medical Journal*, 1975, **1**, 430.

## Student loans

With the announcement of yet another round of public expenditure cuts, yesterday's inconceivable solution for saving money becomes tomorrow's candidate for discussion. There seems a strong argument, therefore, for taking out of the drawer—and re-examining—the case for introducing some system of student loans<sup>1</sup> to relieve the financial pressure on the higher education system. The Government's new and much higher scale of university fees appears to be designed primarily to make foreign students pay a higher proportion of the costs of their education, but it would be optimistic to assume that this will be the last attempt to economise on higher education. Moreover, since medical education is inescapably and unavoidably one of the most expensive forms of training, it may well attract the attention of the Treasury hatchet-men.

The higher education sector is already under considerable pressure. The spending plans announced by the Government in February were based on the assumption that the student numbers would increase faster than expenditure levels. As the Expenditure White Paper<sup>2</sup> put it, somewhat euphemistically, "The planned level of current expenditure will require a progressively more effective use of resources." In other words, spending per head is intended to fall.<sup>3</sup> So any further cuts in the higher education budget would almost certainly mean lower standards.

In this new context, many of the old doubts about student loans are not as persuasive as they used to be. If a system of loans were to permit students to pay more towards the cost of their own education—and if, consequently, the higher education sector were to be at least partly emancipated

from its dependence on the Treasury—then the balance of the arguments may have tipped in favour of introducing such an experiment.

The case against student loans was firmly put by the Robbins Committee on Higher Education<sup>4</sup> in 1963, which pointed out that their effect might be to discourage children from poorer homes from going to a university. Furthermore, the committee argued “either British parents would be strengthened in their age-long disinclination to consider their daughters to be as deserving of higher education as their sons, or the eligibility for marriage of the more educated would be diminished by the addition to their charms of what would in effect be a negative dowry.” However, the Robbins report also conceded that as time went on there might be a case for some experiment.

Arguments for such an experiment in student loans have been strengthened since the Robbins report appeared by evidence that many of the disadvantages can in fact be overcome. In particular, schemes have been designed in the United States<sup>5</sup> which make the repayment of loans contingent on earnings: that is, the repayments are calculated as a fixed percentage of income. Thus, for example, a woman doctor would repay the loan only while she was in employment; if she dropped out of the labour force temporarily, the payments would stop. So the problem of a negative dowry (or of a millstone of debt hanging round the neck of every new-fledged graduate) seems to be avoidable.

There are special problems in the medical profession. One result of introducing a loan system, it may be argued, might be to increase still further the attractions of emigration, since it would be almost impossible, administratively and legally, to enforce debt repayment from abroad. As against this, it could be maintained that a loan system would permit new incentives to be introduced to persuade doctors to move into unpopular specialties or underdoctored parts of the country. Every year's service might count for double so far as debt repayments were concerned. Whether such a radical solution would be acceptable to the medical profession is another matter. Still, given the pressures on all public services, it seems worth investigating the possibilities inherent in what until now have been regarded as unthinkable policy options. The alternative—of making still further economies—may be more unthinkable still.

<sup>1</sup> *British Medical Journal*, 1974, 2, 458.

<sup>2</sup> *Public Expenditure to 1970-80*, Cmnd 6393. London, HMSO, 1976.

<sup>3</sup> Klein, R, Buxton, M, and Outram, Q, *Social Policy and Public Expenditure, 1976: Constraints and Choices*. London, Centre for Studies in Social Policy, 1976.

<sup>4</sup> *Committee on Higher Education Report*, Cmnd 2154, London, HMSO, 1963.

<sup>5</sup> Challoner, D R, *New England Journal of Medicine*, 1974, 290, 160.

## Hard water story: no recommendations

In 1960 Schroeder showed that male deaths from cardiovascular diseases in 163 cities in the United States were less common where the concentrations of calcium and magnesium in drinking-water were high.<sup>1 2</sup> Within a few days of receiving these results, Morris and his co-workers at the Medical Research Council Social Medicine Unit in London used data already in their hands to confirm that this negative correlation

also existed in England and Wales, at least with respect to total water hardness and calcium concentration.<sup>3</sup> In 1962 they said:<sup>4</sup> “It is intriguing that further work confirms the existence of a water factor in cardiovascular disease of middle age and early old age, though no hypothesis regarding its nature or action is yet forthcoming.” In general terms that statement remains as true today as 14 years ago.

Siegfried Heyden has recently reviewed<sup>5</sup> the water story, assessed published work, and raised valid questions about whether it is wise to offer the public advice about their tap-water. The evidence that hard water is protective or soft water harmful is not compelling. True, mortality studies in the USA,<sup>1 2</sup> England and Wales,<sup>3 6</sup> and Canada<sup>7</sup> have shown a negative association between hardness and cardiovascular mortality, but the features associated with mortality have differed among studies, and other investigations have failed to show any association. For example, in North Carolina (where mortality from cardiovascular and particularly cerebrovascular causes is high even for the United States) the lowest rates were found in the areas with the softest water.<sup>8</sup> Neither were significant correlations found between hardness and cardiovascular mortality in rural counties of Oklahoma, in which geographic, environmental, and social variables would have been more uniform than in the larger national studies.<sup>9</sup> Nor was a relation found in three communities in Los Angeles which were similar for age, sex, race, income, socioeconomic status, and stability but different in water hardness.<sup>10</sup> In a careful study in Maryland no difference was found between the hardness of the home tap-water of men aged 45-64 who had died from arteriosclerotic or degenerative heart disease and that of controls.<sup>11</sup> In studies in Ireland<sup>12</sup> and England and Wales,<sup>13 14</sup> too, negative or inconsistent results have been obtained.

Since there is no attractive hypothesis which could unite all these diverse findings, some epidemiologists have thought that other factors might be related both to water hardness and to cardiovascular mortality and that water hardness might be merely an index. Gardner, Crawford, and Morris<sup>15</sup> investigated the independent associations between cardiovascular mortality and a social factor score, a measure of air pollution, latitude, long period rainfall, and water calcium. Latitude and rainfall as well as water calcium were all independently associated with cardiovascular mortality. A climatic factor was also found in an analysis of data from 116 metropolitan areas in the USA, for which a “comfort index” was derived from temperature and relative humidity.<sup>16</sup> This factor was a more important correlate of mortality than the water variables. The authors suggested that thermal stress occurring in both hot and humid or cold and damp periods of the year might have a direct effect on the cardiovascular system. Because of the strong inverse correlation between rainfall and water hardness,<sup>15</sup> data from South Wales<sup>14</sup> and England and Wales<sup>14 17</sup> were analysed to determine how important long- and short-term climatic factors were as correlates of mortality from ischaemic heart disease and whether they would account for the apparent association of mortality with water hardness. The results indicated that water calcium owed its association with heart disease almost entirely to its own association with temperature and rainfall. The possibility that the relation between temperature and mortality from ischaemic heart disease is causal has been discussed recently.<sup>18</sup>

This fascinating story is not tied up by any means. Reports of the association continue to appear,<sup>19 20</sup> and to be explained away.<sup>21</sup> Nevertheless, despite all these inconsistencies in the evidence two official publications have made recommenda-