Two for the Price of One

SIR,—You published an article (13 April, p. 113) on new hospital development under the title "Two for the Price of One." Some daily papers also derived this inference from a vague statement made by the Minister of Health at a press conference (1 April 1968) and from a booklet¹ concerning two new hospital projects, in which it was stated that "the overall saving may amount to 40% or more." It can be affirmed categorically that, using the ordinary meaning of the term "hospital capital cost," it is impossible by any architectural method to cut the cost to that extent. The Ministry of Health has not achieved the impossible; but, starting with both the laudable aim of reasonable economy and by the exploitation of some more advanced thinking current in our profession on the future role of the district hospital, the Ministry has succeeded in altering the distribution of capital costs, some of which are transferred to other sectors to the benefit of the hospital's costing. A careful scrutiny of this wordy booklet is revealing.

In the assessment of needs of acute beds for a population of 170,000 on the traditional figure of three beds per thousand people some 540 beds would be required. The project team reckoned that by using a policy of high efficiency of administration and of admissions and discharges, by the integration of community care and aftercare within the hospital, by the extended use of "day surgery," and by the fuller use of outpatient investigation (often a questionable policy) it would be feasible to reduce the acute beds to 340—that is, at the rate of two beds per 1,000 population. Thus with one blow a 33% cut is made in the major inpatient provision. The increased cost of the community services so involved is of course on another account, and the extra cost of staff (doctors and nurses) required when the degree of "nursing dependency" is heightened goes to annual expenditure. The suggestion, too, that supplementary peripheral outpatient clinics will be required postpones allocation of capital to a later date. Provision is made for only 50 geriatric beds (acute and assessment), and the rest are to be housed in upgraded existing accommodation, and the cost of upgrading will appear in another balance-sheet, while modern views of geriatric care are ignored. Other similar operations reduce the capital costs: firstly by limiting residential building to trainee nurses and essential medical and other staff, relying on the local housing authority's allocation of available accommodation, and secondly by providing some of the industrial zone functions such as laundry, workshops, and the central sterilizing supply department off the site.

The description of the architectural features designed to cut costs also tends to mislead. Such flat sites with adjacent well-serviced roads do not represent average conditions. There is no scale on the sketch plans, and the reader must estimate the dimensions; the *Architects' Journal*² filled in some helpful details. It is clear that standards have been lowered. The wards have only 11% of single beds, which are unfunctionally distributed; the other patients are in six and eight open bays (with small day spaces) of approximately 550 sq. ft. (51 sq. m.). Clerestory lighting is used, which can

produce cold draughts in the wards beneath, treatment rooms are very distant from the wards, and the traffic in lengthy communication corridors is mixed with motorized floats. The claim of flexibility in ward use reveals the planners' ignorance of the significance of the Falkirk ward.³

The hospitals occupy over five acres each, and are built on two floors with only two lifts and a service ramp. The departments most likely to grow, pathology and radiology, are closely hemmed in and have only a derisory possibility of expansion—the hallmark of poor planning. It is even suggested that if the pathology department gets overloaded some of the work could be moved elsewhere, and the idea of changing the user of this mostly expensively serviced department is viewed with equanimity. The entrance concourse, with its snack bar for everybody, shop, bank, and registration office, carries the outpatient traffic and all the two-way traffic of the dispensary, x-ray, and pathology departments and breaks every traffic rule. The records office is separated from the outpatients department by the emergency and accident department, and the approach from the hospital to the physiotherapy area looks unsatisfactory in the sketch.

These low-rise buildings absolutely squander land—the Ministry ignores this. The majority of urban sites are not so extensible, and the Ministry's policy on on-costs is as shortsighted as it is wasteful in its inevitably excessive site-coverage. A notable omission is any mention of the increase of porters required for such hospitals.

Whatever may be one's personal views of the relative importance of the priorities of public expenditure, it is regrettable that the Ministry should describe so much false economy in such misleading terms.—I am, etc.,

London N.W.8. HUGH GAINSBOROUGH.

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- ¹ *Rationalization of Planning and Design*, 1968. Ministry of Health.
- ² *Architects' Journal*, 1968, 10 April, p. 721.
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Assessing "Clinical Competence"

SIR,—“Trained examiners or professional actors have been used to play the part of patients to test the history-taking ability . . .” I am all for a leavening of wit in medical writing, and, coming across these words in a recent leading article of yours (27 April, p. 188), I was all set to join in the chuckle until I continued, “. . . but many examiners feel that such devices are far removed from the real clinical situation,” and received the uncomfortable impression that whether the *B.M.J.* approved or disapproved they were, incredibly, taking this nonsense seriously. My faith in the medical profession as one of the few scientific bodies who had not been seduced by the “technological revolution” but had retained some respect for the human being was further shaken by another statement in the article: “The student types in his request for data and the computer provides the appropriate response. Such dialogues between students and computer

may well be found to be increasingly valuable in the future.”

Whatever next? A new generation of students prodding plastic tummies and analysing artificial urine? Are we going to let the profession's technomaniacs get away with the pretence that medicine is an intellectual game to be played with fake patients in fake situations, or that we can learn clinical medicine by carrying on typewritten conversations with a sickly computer?

A few years ago that leading article could have found itself in print only as a satirical endpiece. Perhaps it was a belated “April fool.” For everyone's sake, I sincerely hope so.—I am, etc.,

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Deaths from Asthma

SIR,—It is with interest and a good deal of disappointment that I have followed your leading article (10 February, p. 329), the excellent articles referred to therein (pp. 335, 339), and the subsequent correspondence. Here in this country we can only agree with your statistics about the disturbing increase in the death rate from bronchial asthma. It is from careful studies such as the two referred to in your leading article that we hope to find out the cause of this alarming increase and then to try to eradicate it by good preventive medicine.

It is, however, with astonishment that I note a complete absence of mention of the role of allergic factors in the causation of asthma, be it ingestants or inhalants, and what we can do to overcome this through avoidance and appropriate hyposensitization. With my great affection and respect for British medicine I fail to understand the complete lack of interest in this important field and the potential benefit that asthmatic patients are liable to receive from a carefully controlled anti-allergy management consisting of avoidance and possible hyposensitization. Any effective tool can be misused and mishandled, but the occasional abuse of hyposensitization in untrained hands makes the result we obtain in clinical practice no less spectacular.

In the article “Investigation Into Use of Drugs Preceding Deaths from Asthma” (p. 339) I am astonished by the brief and cursory reference to theophylline and aminophylline intake prior to the death of these patients. In this country we consider aminophylline or its derivatives as one of the major iatrogenic causes of death in the asthmatic patient. Aminophylline is capable of causing either sudden death or severe and frequently fatal toxic involvement of the cardiovascular system, the central nervous system, or the gastrointestinal tract. Investigation into the intake of this drug, either in combination with other bronchodilators or on its own, is worthy of a great deal of consideration. So far as corticosteroids and pressurized aerosols are concerned, we certainly agree with our British colleagues, except that we would like to be more conservative in the use of corticosteroids. We most emphatically endorse your leading article that “sedatives are absolutely contraindicated in acute asthma.” The

asthmatic patient's restlessness and irrational behaviour is due to his anoxaemia and hypercarbia and can be safely relieved only by adequate ventilation. So far as the actual management of status asthmaticus is concerned, I am again highly surprised to see so many experts putting such great accent on the use of corticosteroids without mention of the fundamental approach of hydration of the patient in status asthmaticus. This is imperative if we are to encourage the thinning out and eventual expectoration of the dried-up secretions and mucous plugs. Undetected respiratory infections have been reported as one of the causes of death in bronchial asthma, and a high index of suspicion with prompt and adequate institution of an appropriate antibiotic therapy could save many lives.

Finally, close follow-up of the clinical picture as well as frequent evaluation of the level of blood gases and the availability of a team of trained personnel for possible assisted respiration is invaluable if we are to do all in our power to save these patients.—I am, etc.,

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N. SABOKAR.

SIR,—I was very much intrigued by the articles by Dr. F. E. Speizer and others (10 February, pp. 335 and 339) on the subject of mortality from asthma and your leading article (p. 329) on the same subject. However, it was singularly unfortunate that Speizer and his co-workers chose the year 1952 as the baseline for their studies. I believe that had they gone back a little more than a score years before the issue would have come into better focus, in particular the effect of the steroids.

Writing on the same subject in an article entitled "A Historical Account of Death from Asthma," Professor H. L. A. Alexander¹ states that we may recognize three periods during which the occurrence of death from asthma showed a characteristic trend. The first is measured by a century, the second by a few decades, and the third by a few years.

Quoting directly from Dr. Alexander's paper: "The first ended about the year 1930. Prior to this date death during an asthmatic attack was almost unknown. To be sure, in that era there was less clinical material in terms of hospital population, fewer autopsies, fewer medical periodicals, and fewer opportunities to present cases at medical meetings, compared to facilities of the present day. Nevertheless, the evidence that death during an asthmatic paroxysm was exceedingly uncommon is overwhelming.

"During the succeeding 25 years there was, at first, a gradual increase in the number of reported deaths from asthma (187 instances had been assembled in 1937), and after that there was an acceleration in the number of fatalities. According to the National Vital Statistics Division of the United States Public Health Service, under the designation 'Deaths from Asthma,' the peak occurred between the years 1951 and 1953. During the latter year there were 6,737 such deaths. More dramatic still was the New Zealand experience, where the death rate from asthma rose from 1.7 in 1939 to 10.4 [per 100,000] in 1953.

"Approximately after the year 1953 there appeared to be a decided diminution in the

number of asthmatic deaths. The apparent abrupt decline in fatalities appeared at a time when cortisone and related hormones were coming to be used extensively in the treatment of asthma."—I am, etc.,

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J. J. ROBBINS.

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- 1 Alexander, H. L. A., *J. Allergy*, 1963, 34, 305.

SIR,—In answer to Dr. B. H. Gandeia (6 April, p. 50), we wished in our letter (2 March, p. 575) to emphasize the importance of a rising arterial carbon-dioxide tension (P_{aCO_2}) in a severe attack of asthma as a sign of serious deterioration and of having a significance quite different from this finding in a bronchitic patient.

He appears to have misunderstood the point of our paper¹ to which he refers, which was a comparison between spirometry and blood-gas tensions in asthmatics and bronchitics, where we found that, although they may have comparable airway obstruction and hypoxaemia, bronchitics have significantly higher P_{aCO_2} levels, but at this stage in the asthmatic the P_{aCO_2} is normal or even reduced owing to hyperventilation. Hypercapnia in the bronchitic is usually of some standing, and compensatory mechanisms have been brought into play, but in the asthmatic hypercapnia usually develops over a short period and often increases rapidly, so that there is no time for adjustment of homeostatic mechanisms. This leads to falls in cerebrospinal fluid pH, and in addition, as the P_{aCO_2} level rises acutely, they are unable to maintain adequate oxygen saturation because the arterial oxygen tension falls markedly. These changes, we think, contribute to failure of respiratory and vasomotor centres, which seems likely to be the cause of the sudden and unexpected deterioration which occurs in severe asthma and which not infrequently leads to death. For these reasons we are in full agreement with Dr. P. Meisner and Dr. P. Hugh-Jones (24 February, p. 470) and others,² who regard a rising P_{aCO_2} level as heralding a severe emergency, which is often followed by death unless ventilation is artificially achieved.—We are, etc.,

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- 1 Palmer, K. N. V., and Diamant, M. L., *Lancet*, 1967, 2, 383.
- 2 Marchand, P., and van Hasselt, H., *Lancet*, 1966, 1, 227.

Atrial Septostomy

SIR,—Further to the memorandum by Dr. S. P. Singh and his colleagues on the haemodynamic effects of balloon septostomy in tricuspid atresia (27 January, p. 225), which suggests that this procedure should decrease central venous pressure and relieve cardiac failure, we should like to report an additional case with further details.

The patient was noted to be breathless from birth when feeding, at 5 weeks cyanosis was obvious, and she developed cardiac failure following a respiratory tract infection. Examination showed central cyanosis, sinus rhythm 140/min., the apex beat was not displaced, there were no thrills, the second sound in the pulmonary area was normal in intensity and single, there

was a grade 2/6 pansystolic murmur at the lower right sternal border, but no diastolic murmurs. The liver was enlarged two fingerbreadths, the chest was clear, and the respiratory rate 40/min. Chest radiography showed situs solitus, and cardiomegaly with reduced pulmonary vascularity. The electrocardiogram showed sinus rhythm 120 (axis -30°), gross right atrial (4 mm.) and left ventricular hypertrophy.

Cardiac catheterization with right atrial and left ventricular angiocardiography confirmed the diagnosis of tricuspid atresia with a ventricular septal defect and normally related great vessels. In view of the P waves of 4 mm. and a right atrial pressure of 25 mm., it was decided to perform an atrial septostomy. This was done initially with a No. 4 Rashkind catheter with 2.5 ml. of dye in the balloon. This burst during the procedure and a No. 5 Fogarty embolectomy balloon catheter was then used with 2.0 followed by 2.5 ml. of dye in the balloon. This produced a reduction in right atrial pressure from 25 to 12 mm. Hg. Initially difficulty was experienced in visualizing the non-opaque embolectomy catheter, but this was overcome by partially filling it with radio-opaque dye. The Fogarty catheter and balloon, as pointed out by Dr. Singh and his colleagues, are more robust, cheaper, and more easily available in a greater variety of sizes than the Rashkind instrument. Although the latter has the advantage of giving a direct pressure measurement in the larger sizes, this is not found in the No. 4, which may be more frequently used in small children.

Our patient showed an initial improvement over the first 48 hours following the procedure, but subsequently developed progressive cardiac failure and died on the seventh post-operative day. Necropsy showed the foramen ovale to have been split open completely (see Figure).

Right postero-lateral view of partly dissected heart with lateral surface of right atrium reflected. Right atrium is above, and the central dark area represents the deficiency in the atrial septum as a result of the Rashkind procedure.

We would suggest that atrial septostomy should be regarded with some circumspection following the death of both the infants reported, although their condition at the time of operation was poor; certainly this small experience contrasts badly with the largely good results we have obtained with the Rashkind procedure used in infants with transposition of the great vessels.—We are, etc.,

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