distribution of consultant and other medical services throughout the country. Would it not be better to distribute the extra consultants and resources to those districts that have a disproportionately low level of consultant provision and yet can be seen to use their consultants efficiently? Kenneth Clarke stated on television that the "size of the GP list is probably the best indication of workload," and the same could be said for consultants: catchment population per whole time equivalent consultant surgeon is probably the best indication of surgical workload.

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1 Secretaries of State for Health, Wales, Northern Ireland, and Scotland. *Working for patients*. London: HMSO, 1989. (Cmd 555.)

Slicing up authors

SIR,—We found the news item "Publication: duplicate, salami, meat extender—all redundant" distasteful.¹ Redundant publication is a legitimate point of debate but criticisms that potentially impugn authors' professional integrity, even by implication, should be made with great caution. Authors' redress is limited to the right of reply, and in this instance this courtesy was not extended to Saad *et al.* Instead, readers were directed to their reply published in the *New England Journal of Medicine*,² presumably on the basis that to publish their letter in the *BMJ* would have clogged "the literature and databases with the same material," wasted time of the editors and secretaries, and excluded "more deserving authors who play by the rules."

Medical journals should adhere to the journalistic standards expected of the serious press. Sensational headlines are usually inappropriate. It should be recognised that, however unjustified, "mud usually sticks." Impugning by implication the reputation of these authors was unwarranted. Your reviewers acknowledged that the two articles on the natural history of impaired glucose tolerance in the Pima Indians were "not identical and dealt with different aspects of the same problem with largely different patient groups." The paragraphs in com-

mon were largely from the methods sections. We, unlike the editor, were reassured to find in both papers the same methods described for a long term continuing study of a defined population.

Whether a single publication would have been "more cohesive and informative than two" is a matter of judgment. In our opinion, a single publication would have been excessively long and more difficult for readers to follow. The editorial entitled "Redundant publication" in the *New England Journal of Medicine* recognised the pressures placed on investigators to publish or perish but failed to recognise the additional pressures placed on authors by editors and reviewers to present complex topics from large studies in short papers. Our experience suggests that if both topics had been well explored in the same article it would have received the common editorial response: "shorten and simplify."

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- 1 Lock S. Publication: duplicate, salami, meat extender—all redundant. *Br Med J* 1989;298:1203-4. (6 May.)
- 2 Saad MF, Knowler WC, Pettitt DJ, Nelson RG, Mott DM, Bennett PH. Publication of closely related reports. *N Engl J Med* 1989;320:1221-2.
- 3 Angell M, Relman AS. Redundant publication. *N Engl J Med* 1989;320:1212-3.

*Editors are not asking that authors should never submit more than one paper from a single study, but simply that they should inform editors at the outset. We have been making this point for years—for example, in instructions to authors—but the number of cases of redundant publication, and the fact that Dr Neil and colleagues seem also to have missed the point, suggests that the message needs hammering home. —Ed, *BMJ*.

Drug Points

Are β blockers and nifedipine safe?

Dr IVAN H STOCKLEY (Department of Physiology and Pharmacology, Queen's Medical Centre, Nottingham NG7 2UH) writes: Drs A B Hassell and J E Creamer suggest, after describing two patients of theirs taking β blockers who showed profound bradycardia when given diltiazem, that "diltiazem should be avoided and nifedipine or nicardipine preferred,"¹ the implication being that concurrent treatment with one of these calcium channel blockers is safer.

While it may be true that concurrent use can be valuable and usually uneventful, the combination of β blockers and nifedipine also sometimes causes problems. There is a report of two patients with angina taking alprenolol or propranolol who developed cardiac failure when additionally given 10 mg nifedipine three times daily.² The signs of failure disappeared when the nifedipine was withdrawn.² Cardiac failure has also been described in two other patients taking atenolol who were given nifedipine^{3,4} and in another patient taking propranolol whose hypotension became so severe and prolonged that it may have been a factor which led to fatal myocardial infarction.⁵ These reports are few compared with those describing the advantages of concurrent use, but they clearly illustrate

that the use of nifedipine is also sometimes not uneventful.

Clearly reports of adverse reactions like those described by Drs Hassell and Creamer should be published and become known, but it is equally important that a well rounded picture of the alternatives should be presented so that "safer" substitutes which also carry a risk, however small, are not suggested without some qualification being added.

- 1 Hassell AB, Creamer JE. Profound bradycardia after the addition of diltiazem to a β blocker. *Br Med J* 1989;298:675. (11 March.)
- 2 Anastassiades CJ. Nifedipine and beta-blocker drugs. *Br Med J* 1980;281:1251.
- 3 Opie LH, White DA. Adverse interaction between nifedipine and beta-blockade. *Br Med J* 1981;281:1462.
- 4 Staffurth JS, Emery P. Adverse interaction between nifedipine and beta-blockade. *Br Med J* 1981;282:225.
- 5 Robson RH, Vishwanath MC. Nifedipine and beta-blockade as a cause of cardiac failure. *Br Med J* 1982;284:104.

Acute epiglottitis and Chloraseptic

Drs SHU-LEONG HO and K HOLLINRAKE (Department of Medicine, Walsgrave Hospital, Coventry CV2 2DX) write: We describe the development of acute epiglottitis, resulting in a life threatening

airway obstruction, after the use of Chloraseptic (phenol) throat spray. It is advertised as a remedy for instant relief for sore throat.

A 49 year old Afro-Caribbean woman was admitted in cardiorespiratory arrest. Resuscitation was started. She regained a full volume pulse less than half a minute after external cardiac massage was started. The electrocardiographic monitor confirmed a sinus rhythm of 160 beats/min. When she was intubated pronounced erythema and oedema of her epiglottis and larynx, causing airway obstruction, were noted. There was no skin lesion. Blood cultures and laryngeal swabs were taken. Arterial blood gases showed gross respiratory acidosis with pH 6.86, carbon dioxide pressure 17.4 kPa, and oxygen pressure 5.1 kPa. She was ventilated for 48 hours. Her temperature was 37.5°C and a chest radiograph showed consolidation in her right lung, thought to be right sided aspiration pneumonia. Antibiotics and dexamethasone were started. On extubation examination of her epiglottis and larynx showed that the inflammation had settled. Recovery was uneventful.

She had complained of a sore throat and runny nose two weeks before admission. She bought a Vicks Chloraseptic throat spray and sprayed it five times as prescribed on the bottle. She then felt a burning sensation in her throat and shortly afterwards became acutely dyspnoeic. She lost consciousness in the ambulance. In 1983 she had undergone microlaryngoscopy for laryngeal nodules, which had not troubled her since. She had a six year history of a symptomless non-toxic goitre and had been receiving nifedipine for the past year for hypertension. There was no previous exposure to Chloraseptic spray or any history of atopic reactions.

On admission her haemoglobin was 179 g/l, white cell count $15.8 \times 10^9/l$, neutrophils $10.9 \times 10^9/l$, lymphocytes $3.45 \times 10^9/l$, and eosinophils $0.04 \times 10^9/l$; a sickle cell test was negative. Blood cultures and laryngeal swabs taken before antibiotics were started were sterile. Sputum specimens taken after she was extubated showed no growth. Viral studies, including chlamydia, coxiella, and mycoplasma, showed no rise in titres. Her IgE value was 300 IU/l (normal <200 IU/l). Complement studies were normal: C1 inhibitor concentration was 0.27 g/l (normal range 0.18-0.30), C3 1.19 g/l (0.75-1.75), and C4 0.32 g/l (0.14-0.54). Five days after she was extubated a repeat chest radiograph showed clear lung fields.

The Chloraseptic liquid used in the throat spray contains as its active ingredient phenol and sodium phenolate equivalent to total phenol 1.4%, with sodium borate, menthol, thymol, and glycerol. It has also been used as a gargle. Phenol is bacteriostatic in concentrations of 0.02-1%, bactericidal to some micro-organisms in concentrations as low as 0.04% and to all above 1.6%, and fungicidal in concentrations above 1.3%.¹ It is not sporicidal. Concentrations above 0.5% cause a depolarising local anaesthesia. Its activity is greatly reduced or abolished in alcohol, glycerol, or alkaline solution. When moderate concentrations of phenol are applied to the skin or mucous membranes the surface becomes white and opaque owing to the denaturation of tissue proteins. Oral ingestion can result in mucocutaneous and gastrointestinal corrosion.

The Committee on Safety of Medicines has received only nine reports of adverse reactions to single constituent preparations containing phenol. (Chloraseptic is the only one available in the United Kingdom.) These include angio-oedema (one), erythematous rash (two), glottitis (one), and contact dermatitis (two). We think that this woman suffered a life threatening adverse effect from the Chloraseptic throat spray, although we are uncertain whether it was due to an anaphylatic type reaction or a direct toxic effect.

1 Reynolds JEF, ed. *Martindale: the extra pharmacopoeia*. 28th ed. London: Pharmaceutical Press, 1982.