Bedside dialysis for patient with severe wound infection

Lessons learnt

Several lessons may be learnt from this experience. Firstly, the appropriate aid must arrive rapidly at the right place to lessen natural triage. Obviously after a major disaster in which the local medical services have either been destroyed or overwhelmed replacing or augmenting them rapidly will help reduce both mortality and morbidity. The incidence of acute renal failure after crush injuries may be dramatically reduced by urgent replacement of intravenous fluid. Patients who do develop acute renal failure will die of hyperkalaemia within one to two days if dialysis is not

started. Such a rapid response can be achieved only by a permanent relief team available to depart at a few hours' notice. This is amply shown by the fact that the West German team arrived three days after the disaster compared with our arrival time four days later (a week after the event).

Secondly, any aid of this type must be fully self supporting. All equipment required to perform a task, both disposables and machinery, must be taken with technical support to ensure that the machinery continues to work. The team should have several forms of transport at their disposal, depending on the geography of the disaster area. This ensures that the staff and the equipment arrive together without affecting local transport facilities, which may already be overwhelmed. Back up and good communications are vital both to resupply the team and to maintain morale. Previous knowledge of local conditions is invaluable, particularly for dialysis as the availability of a power supply and a source of water will determine what method of treatment is appropriate.

Finally, but most importantly, you must remember at all times that you are guests in someone else's country.

- 1 Bywaters EGL, Beall D. Crush injuries with impairment of renal function. *Br Med J* 1941;i:427-32.
- 2 Beall D, Bywaters EGL, Belsey RHR, Miles JAR. A case of crush injury with renal failure. *Br Med J* 1941;i:432-4.
- 3 Michaelson M, Taitelman U, Bshouty Z, Bar-Joseph G, Bursztein S. Crush syndrome: experience from the Lebanon War. *Isr J Med Sci* 1984;20:305-7.
- 4 Santangelo ML, Usberti M, Di Salvo E. A study of the pathology of crush syndrome. *Surg Gynecol Obstet* 1982;154:372-4.
- 5 Glasscock R. Haematuria and pigmenturia. In: Massry SG, Glasscock R, eds. *Textbook of nephrology* Baltimore: Williams and Wilkins, 1984;4:20-3.
- 6 Wing AJ, Parsons FM, Drukker W. Dialysate regeneration. In: Drukker W, Parsons FM, Maher JF, eds. *Replacement of renal function by dialysis*. Boston: Nijhoff, 1983:323-40.

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80 000 Pills: a personal history of hypertension

B D Mills

My hypertension was discovered when I was 21, in 1950, just before the era of antihypertensive drugs. Thus my adult life has coincided with the evolution of the treatment of hypertension, and both the disease and its treatment have had a major impact on my professional and family life. Having retired at 57 after an exacting career in engineering and industrial management, I firmly believe that success in the treatment of chronic diseases can be achieved with active self management based on some understanding of both the disease and the treatment.

Early life

I spent an outdoor childhood in North Queensland playing tennis, cricket, and rugby as often as possible. School ensured that one contracted the full gamut of childhood diseases, my only distinction being the inclusion of jaundice in my portfolio. From a secondary school with a strong tradition of sporting achievement I went to Queensland University, where I graduated in electrical engineering in 1949 and in science in 1950.

The Australian Department of Supply announced in mid-1950 its intention to award several scholarships allowing successful candidates an opportunity to study for a higher degree in the United Kingdom. At the end of the selection process I was awarded one "subject to a satisfactory medical examination." I had not the slightest doubt that I would sail through any medical

with flying colours. In the event, excessive albumin was detected in a urine sample and my systolic blood pressure was said to be highly labile and above 150 mm Hg (at age 21). The department decided that I was a poor risk (as the quid pro quo of the three year scholarship was a requirement to work for the department for five years afterwards), and my award was withdrawn.

Disappointed at my failure to achieve the scholarship, I consulted a Brisbane specialist in the hope that treatment might correct the condition. The outcome was shattering: a more relaxed lifestyle was the only recommendation. "Slow down—give up sport—don't run for the tram, there is always another one—take life at a slower pace." To someone used to leading a full academic and athletic life this advice was distinctly unpalatable. Second opinions from medical friends supported these recommendations, so I gave up all sport and attempted to slow down.

Despite this unwelcome development I decided to go to Britain to gain technical experience and to see as much of Europe as possible; 1951 passed quickly with an interesting job and touristic excursions at weekends. By spring 1952 life seemed a little dull without sporting challenge, and as I felt well I decided to take up social tennis. I enjoyed a marvellous summer of club tennis until palpitations developed on court—mild at first, but by autumn too fierce to be disregarded. An evening visit to a local doctor led to an

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insistent recommendation of immediate admission to hospital: a reading of 240/140 mm Hg on the sphygmomanometer in a 24 year old was not to be ignored.

The Vegolysen watershed: 1952-4

If having hypertension in the prime of life rates as a misfortune then the scales were balanced in Orpington Hospital, where I became a patient of a fine physician. A full range of tests confirmed the earlier diagnosis of malignant hypertension and kidney damage; the prognosis was not good. It was decided to try intramuscular treatment with a new drug, Vegolysen (hexamethonium bromide). Never will I forget its traumatic side effects, many not then identified. But it was dramatically effective in lowering blood pressure, which fell like a stone within 15 minutes of the injection. Visual accommodation deteriorated, and I became weak, nauseous, and lightheaded. The only prudent course was to do nothing. Any food or drink taken within one and a half hours of an injection was quickly lost. After an hour or so normal activities could be resumed as blood pressure slowly recovered. A second slug of Vegolysen was given eight to 10 hours later.

In December 1952 I was released from the warm embrace of the hospital ward into a cold and very foggy world (the last peasouper) armed with one syringe, one size 12 needle, and a supply of Vegolysen. The outlook from my chilly room in local "digs" seemed a little discouraging. But my understanding employer accepted two lost periods of one hour each in the working day, and visits to hospital gradually became more infrequent. Holidays and weekends lost a little gloss with bruised thighs, occasional bleeding, blunt needles, and clandestine sterilisation of syringes on a methylated spirit stove in some hotel bedroom. Saturday visits to the west end of London meant finding a doorway in a side street in which to administer an injection and then a nearby park seat on which to hibernate for an hour.

Looking back, I am beholden to Vegolysen in that it bought me time, but it wrought substantial change in my apparent health and wellbeing. It was now obvious to all that I was unwell and that hypertension would have an impact on my way of life in several ways. One was the twice daily chore of giving myself injections; another was the question of marriage. Was my prognosis under the umbrella of Vegolysen sufficiently improved to make this a reasonable proposition? As I was in the vanguard of experience with this drug there was no evidence on which to base an answer. We took the decision and married in November 1953. Bless my physician—and my wife; we have celebrated 35 anniversaries to date. It was an unusual wedding day and honeymoon: lone breakfast in a bachelor flat, injection, one hour's non-existence, wedding and reception, second injection and lost hour, then foggy flight to Paris. Our days were inevitably organised around the needle: an injection at the hotel, then a hurried walk to the Pont des Arts over the Seine, where I would pass out incognito on a bench seat while my wife enjoyed the Parisian scene.

Stability: 1955-75

For family reasons we moved to Australia in July 1954. A close medical friend introduced me to a young physician at Brisbane General Hospital who telephoned me a few months later to say that they had obtained some encouraging results by treatment with two oral drugs. After over 1600 intramuscular injections I immediately volunteered for conversion to Ansolysen (pentolinium) and Serpasil (reserpine). They served me well for over 20 years. Of course they

were not without unpleasant side effects. Ansolysen produced regular diarrhoea, a dry mouth, postural hypotension, and some nausea and Serpasil induced nasal congestion, an inability to sleep with the mouth closed, depression, and occasional incidents of irrational behaviour.

In February 1957 we returned to England, and I moved up an engineering career ladder and into industrial management in 1964—all in an American owned company, well known as a high stress environment. I spent the rest of my career with the same company and progressed to the upper levels of management. I record these facts solely to make the point that from unlikely beginnings medical help and progress in the pharmaceutical industry allowed me to hold down challenging jobs in a stressful environment. Our children were born in 1957 and 1961, and the 1960s was a decade of great happiness and reasonable health. Within this period there were, however, many ups and downs. Two major setbacks were consequences of hypertension and its treatment, and both were unpleasant surprises: gout in 1958 and ischaemic heart disease in 1964.

Attacks of gout

So improbable was gout at the age of 29 (I had never had port or pheasant in my life) that my wife and I failed to realise what had inflamed my big toe. But not for long. As all sufferers of gout will confirm, the pain can be so severe that even the proximity of third parties to an affected joint can cause an anxiety based increase in pain. At the zenith of an acute attack the victim can think of little else. A limping visit to the doctor produced, unbelievably, a prescription for spirits of colchicum, the same remedy that physicians had prescribed for 200 years. The large bottle of watery fluid produced drastic purgative effects before it gave limited relief from the gout—and took many days to do so.

A characteristic history of the development of gout is a doubling of the frequency of acute attacks each year until the periods in between became so short that the patient is said to have chronic gouty arthritis. By 1964 I was either having an acute attack or on the very edge of it, sometimes with gout in both feet. Oversize slippers were often the only footwear I could tolerate, even on my return to work or in a hotel dining room on business travel. Gout became a major influence on my lifestyle; it imposed physical constraints from time to time, added to the daily intake of powerful drugs, and influenced the choice of food and drink.

In the 1950s the major dietary emphasis in the management of hypertension was on the intake of salt. For 15 years from 1957 my wife cooked all our food without salt, adding it afterwards for herself and the children. With the onset of gout further dietary constraint was inevitable. I can confirm beyond doubt a direct correlation between alcohol intake and an acute attack. Quantity and type of alcoholic beverage also correlate: red wine and fortified drinks like port and sherry are more damaging than white wine and lager, but regrettably all forms of alcohol may cause attacks. Thus by 1960 I was obliged to forgo alcoholic drinks in any form and did so for over four years.

High purine foods had to be avoided. Top of the league was fish roe: this was not a problem as we could not afford caviar. Nor was the elimination of liver, kidneys, sweetbreads, heart, and anchovies from our menus a problem; I had never liked them anyway. But the reduction in high protein food was a disappointment for an Australian raised on red meat. In the 1970s these constraints were compounded by a low cholesterol diet after high serum cholesterol concentrations were discovered.

Treating gout

But even gout, which had withstood progress in the pharmaceutical industry for over 200 years, succumbed in the early 1960s. The first breakthrough was the production of colchicine tablets: a few tablets had the same impact as a whole bottle of the spirits. Acute attacks were relieved in about two days, but there was a price to pay—diarrhoea. In my case this was particularly severe as it was already a daily side effect of the Ansolysen/Serpasil regimen. The essence of effectively using colchicine was to assess the possible severity of the attack and limit the amount taken in the hope of containing the inevitable diarrhoea. Probenecid (Benemid), first tested in the early 1950s, improves the efficiency with which the body excretes uric acid. It was first prescribed for me in 1964, and I am still taking it every day. Although it has not eliminated acute attacks, it has dramatically reduced their frequency and allowed me to relax marginally my abstemious diet to the extent of one or two drinks a day.

In the mid-1960s another drug, Indocid (indomethacin), became available for the treatment of acute attacks. This was welcome as it did not induce diarrhoea, although it did cause a thumping headache and some mental confusion. About 1970 allopurinol (Zyloric) was introduced; this inhibited the generation of uric acid and virtually eliminated acute attacks for many people. I made two attempts in 10 years to convert to it but each time suffered distressingly from diarrhoea. At about the same time another treatment for acute attacks became available. This was phenylbutazone; it was marvellously effective and would almost stop an attack in its tracks; attacks were never as painful as previously. I took it mostly as Butacote (to protect the stomach) and thereafter never travelled without it. In my business career much travelling was necessary to Europe, America, and, less frequently, to South Africa and South America. Coping with various ills on such trips took discipline, and acute attacks overseas were a misery: one such in Rio de Janeiro in pre-phenylbutazone days remains a nightmare. The gout and its treatment with the Brazilian version of colchicine left me so weak and exhausted that I needed the help of friends to board the plane home. Unfortunately, phenylbutazone was withdrawn from general use in 1984 owing to serious side effects.

In early 1964 chest pains that persisted for some weeks marked the onset of heart disease. One peculiarity in my case was an inability to lift items that would present no problem even to children. My weight limit, established empirically, declined to about 5 kg, which meant suitcases were always small and packed on the bathroom scales. But it was not all bad news. In 1966 I felt well enough to resume social tennis after a total break of 14 years and was able to teach my children to play this marvellous game. About 1970 my chest pains became more frequent and playing tennis was no longer practical, so I translated my interest and weekend activity to golf.

The difficult years: 1975-81

The beginning of 1975 brought an end to over 20 years of stability in my hypertension. On presenting the usual prescription to a pharmacist I was told that Ansolysen was no longer available. My doctor wrote to the manufacturers, asking them to make a further batch of Ansolysen, but was told that production had ceased many years previously. Great progress had, however, been made in the treatment of hypertension since my regimen had been initiated, and no problems were anticipated. The favoured approach at that time was to use drugs that worked centrally on the brain rather than ganglion blocking agents, so Aldomet

(methyldopa) was prescribed. I did not enjoy the change. Side effects were unpleasant: drowsiness, depression, headaches, and the ubiquitous diarrhoea. In addition, it upset a lifetime's ability to fall asleep easily. Valium (diazepam) was prescribed, but I tried to survive without it because of concern about its addictive characteristics.

After an unhappy year persevering with Aldomet I changed to another central adrenergic inhibitor, Catapres (clonidine hydrochloride). This was extremely unpleasant, causing drowsiness, confusion, headaches, and loss of weight (a feature of Aldomet too). In the 18 months I took these two drugs my weight fell by almost 14 kg, and my poor general appearance attracted comment from friends. So a third approach, the use of diuretics, was tried. Aldactone (spironolactone) seemed to be quite effective and fairly free of side effects other than diarrhoea and the inevitable increase in the frequency of micturition, which meant at least two visits to the toilet during the night, a sleep pattern that has persisted ever since. Aldactone alone did not provide complete control and left me somewhat sensitive to stress so, reluctantly, Serpasil was reintroduced.

This regimen started in 1977 and seemed to offer the prospect of controlling the hypertension into the future. But throughout the 1970s angina had become periodic, culminating in a cardiac attack in September 1978, which forced me to celebrate my 50th birthday in hospital. Anturan (sulphinpyrazone) was prescribed, but two months of this drug plus Serpasil did not control the blood pressure. β Blockers were suggested as an alternative, and Tenormin (atenolol) was chosen as only one tablet a day is necessary.

Initially it seemed a great success, but progressively it slowed the pulse rate to the low 40s and the resultant shortage of oxygen made normal exertion such as climbing stairs an exhausting experience. More insidious, however, was the effect on kidney function: blood tests showed high creatinine and urea concentrations. Three months of Apresoline (hydralazine hydrochloride) and Serpasil did not achieve adequate control, so it was decided to revert to Aldactone. Two days before we were due to leave for a summer holiday in Spain my left breast became sore and then enlarged. It was not sufficiently uncomfortable to seek medical advice or cancel the holiday: a boil perhaps, or some form of cyst? But in Spain my right breast alarmingly followed suit, and both continued to enlarge. Aldactone was summarily stopped. Thiazides were off limits in view of the gout, so the daily regimen became Dytac (triamterene), Serpasil, and probenecid. A second attack of gynaecomastia a month later, however, brought my lifetime interaction with diuretics to a conclusive end.

By this time it was becoming clear that none of the current approaches was as effective as the vasodilator regimen that had served me so well for 20 years. Hypovase (prazosin hydrochloride) seemed to work but required careful management as its side effects are numerous. Postural hypotension and nausea are frequent occurrences an hour or so after ingestion and have caused minor dramas on the golf course; ambient temperature changes, as in holidays in Singapore or Australia, have led to embarrassing collapses in public places. Four years of taking Hypovase, Serpasil, and probenecid produced reasonable though far from ideal control. Two attempts were made to eliminate Serpasil and to ameliorate the problems of the Hypovase programme. Another β blocker, Corgard (nadolol), was tried but abandoned after two months owing to low pulse rate. Yet a fourth mechanism of blood pressure control was prescribed, consisting of Frumil (frusemide plus amiloride chloride) in parallel with Hypovase, but this was not successful.

Retirement: 1985

The stresses of an exacting career, an adult lifetime of hypertension and heart disease, and over 30 years of side effects of many drugs led me to request early retirement at 57, and I left the company in October 1985, after 32 years' service.

I believe that this is a success story. Few doctors in 1952 would have taken bets on my survival to age 60: my blood pressure at age 24 was 240/140 mm Hg and kidney damage was already apparent. The contributions to this survival have been many, but some are less obvious than others. Firstly, I am fortunate to have been treated by several doctors, both general practitioners and company medical officers, who have taken a close interest in my wellbeing. Secondly, my adult life has coincided almost exactly with the era of antihypertensive drugs: my disease was discovered in 1950, and the first widespread use of drugs to control blood pressure was in 1951. Also important have been the effort and time my wife and I committed to the management of the disease. We shared the chore of injections and the reading necessary to gain an understanding of the disease; she took most of the domestic load while I earned the family income. We have been a good team.

Part of my good fortune has been to have several medical friends who have talked at length about the disease and have brought to my attention relevant books and journal articles. To them I am most grateful; an essential element of successful self management of a chronic disease is an understanding of the disease and the mode of operation of its treatment. Such knowledge leads to more efficient use of precious time during consultations with doctors; it allows day to day decisions on diets, lifestyle, and responses to side effects to be made with confidence. *Davidson's Principles and Practice of Medicine*,¹ *Black's Medical Dictionary*,² *Medicines* by Professor Parish,³ and various "Home Doctor" books have been on the bookshelves for years, and *Martindale's Pharmacopoeia* has been referred to at the local library on many occasions.⁴

A knowledge of potential side effects is, I believe,

helpful in managing treatment and in ensuring successful use of drugs. Antihypertensive drugs, almost irrespective of their mode of operation, show a clear pattern of side effects. In reducing blood pressure these drugs induce diarrhoea, nausea, dry mouth, and drowsiness. Some can also produce impotence, gynaecomastia, depression, nightmares, and postural hypotension—all unpleasant surprises if they arrive unannounced. After one or two such experiences I made it my practice whenever a new drug was prescribed to research possible side effects before proceeding with the treatment.

A key accessory to the successful self management of hypertension is a personal sphygmomanometer. Several simple electronic measuring systems are now available that avoid the possible hazards of spilt mercury and require little skill to determine both systolic and diastolic pressures. We bought a mercury sphygmomanometer and a stethoscope many years ago. The stethoscope has proved unnecessary: threshold levels of blood flow can be detected as air pressure is released in the cuff, and even third parties can see the associated hiccups in the mercury column. This approach may give marginally different figures from those taken by a doctor, but the difference is small, and my doctors have accepted my figures for 17 years. A portable (aneroid) instrument has also proved useful and accompanies me on all travels; it simplifies decisions on appropriate drug doses when temperatures or planned activity programmes are abnormal.

I hope this is not the end of my story, as further progress in medical science and the pharmaceutical industry must surely lead to improved treatments for hypertension. In the mean time I persist with daily cocktails of Hypovase, Serpasil, and probenecid.

1 Macleod J, Edwards C, Bouchier I, eds. *Davidson's principles and practice of medicine*. 15th ed. Edinburgh: Churchill Livingstone, 1987.

2 Thomson WA. *Black's medical dictionary*. 34th ed. London: A and C Black, 1984.

3 Parish P. *Medicines: a guide for everybody*. 5th ed. Harmondsworth: Penguin, 1985.

4 Reynolds JEF, Prasad AB, eds. *Martindale: the extra pharmacopoeia*. 28th ed. London: Pharmaceutical Press, 1982.

ANY QUESTIONS

What precautions should be taken for a mentally handicapped adult with epilepsy who has family holidays abroad?

The steps that anyone would take before going abroad on holiday should be taken. This includes completing the Department of Social Security's form E111, which enables the individual to benefit from reciprocal health care arrangements where these exist. Details are given in DSS leaflets SA30, SA35, and SA36. In addition, I recommend that private health insurance is obtained which would cover the epileptic patient if he or she needed medical treatment.

It is advisable to obtain a doctor's note confirming that medication is being taken abroad to treat epilepsy, obviating problems with customs officers who may be searching for illegal drugs. Extra supplies of necessary antiepileptic medication should be taken to allow for the possibility of travel delays. This should not be confined to one item of baggage in case this is lost or stolen. Most antiepileptic medication is obtainable abroad but the foreign equivalent may be known under a different name and the British Epilepsy Association can provide advice on this.

Some people with epilepsy are more prone to seizures if their sleep patterns are disturbed and allowances should be made for this factor. Flying in a pressurised aircraft does not precipitate seizures, but it is advisable to notify the airline that a member of the party has a history of epilepsy. This will lessen panic if a seizure occurs and it is helpful if the individual is in an aisle seat towards the rear of the compartment. This will allow the person to be placed on the floor if this becomes necessary.

People who have seizures triggered by photosensitive stimuli—for example, bright light reflected off the water ("flicker effect")—may find polarised sunglasses helpful. Advice about swimming and epilepsy may be obtained from the British Epilepsy Association.

Some people require antisuffocation pillows because they have seizures

while asleep. If it is impractical to take a pillow people should sleep without one. If the individual suffers from incontinence a rubber sheet could be taken to use on hotel beds, etc.

Travel sickness tablets may cause drowsiness and should be used only on medical advice.

The British Epilepsy Association is willing to provide information about similar organisations in other countries, which are able to give advice about treatment facilities.—RICHARD PUGH, *director of social work, British Epilepsy Association, Anstey House, 40 Hanover Square, Leeds LS3 1BE*

A woman treated with tamoxifen for breast cancer noticed that when she took the proprietary form Nolvadex her periods were suppressed but when she received a generic product her periods were normal. Does this difference reflect a variation in the effectiveness of the preparation?

I cannot give a definite answer to this question. Amenorrhoea is a direct result of the antioestrogen effect of tamoxifen. Menstrual disturbance, however, occurs in fewer than half the women taking the drug so it is not a necessary indicator of efficacy. Presumably it is an idiosyncratic response depending on the hormonal state of the individual patient. In this patient the return of her periods when she changed from Nolvadex to a generic preparation suggests a reduction in the antioestrogen effect of the drug. It is, however, not possible to say whether this is likely to be associated with loss of the therapeutic effect. There are many generic preparations of tamoxifen, and doubts about the quality of some of these have been expressed before.¹—LINDA BEELEY, *consultant clinical pharmacologist, Birmingham*

1 Hayward JL, Fentiman IS. Generic prescribing. *Br Med J* 1986;292:762.