Hypertension—II

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Dr. Wood: Our third patient* is a man aged 63; he complained of headache, rapidly failing eyesight, and increasing breathlessness at night. His blood pressure was recorded as 160/110 mm Hg when he had an inguinal herniorrhaphy 15 years ago. On this admission it was found to be 280/160 mm Hg. His heart was enlarged and he had bilateral papilloedema with many haemorrhages and exudates, and a prominent macular star. His E.C.G. showed signs of gross left ventricular hypertrophy and strain, while the blood urea was 50 mg/100 ml, and the urine contained some albumin and red cells.

We gave him oral bendrofluazide, which relieved his left ventricular failure, and the hypertension has been controlled with bendithanidine, increasing over six weeks to a dose of 20 mg four times daily. He now has a supine pressure of 220/115 mm Hg, falling to 160/90 mm Hg on standing and 130/70 mm Hg on exercise. The papilloedema has regressed and no new haemorrhages can be seen. The urine is now free of blood and albumin, the urea is stable at 60–70 mg/100 ml, and the creatinine clearance is 45 ml/min.

Professor MacGregor: This is a case of "accelerated" essential hypertension. I would not criticize the doctors who did not treat his hypertension 15 years ago. That was quite early in the ganglion blocker era, and at that time symptomless patients were seldom treated, and, more relevantly, were rarely reviewed. That one can reverse this accelerated phase comes across well in this instance. The prognosis is poor in patients like this, but he can certainly expect to survive longer than those with grossly impaired renal function. Why did you use bendithanidine in this case?

Dr. Wood: Bethanidine and guanethidine are both potent adrenergic neurone blocking drugs. They probably act by interfering with the release of noradrenaline at the postganglionic sympathetic nerve endings. When they are given in higher doses (particularly guanethidine) the actual concentration of amines may be reduced. Both drugs cause considerable postural hypotension, especially bethanidine with which further falls in pressure may occur on exercise. Men on these drugs frequently become impotent.

Guanethidine causes more troublesome side effects than bethanidine, notably stiffness of the nose, explosive diarrhoea, and bradycardia. The doses needed of these drugs tend to rise in the first few weeks of treatment, so that it is a good thing if dosage can be stabilized in hospital. It is easier to adjust the dosage with bethanidine, which is a short-acting drug. Guanethidine is a cumulative drug and a change in dosage may produce effects lasting for up to a week.

The lower incidence of side effects, and the more rapid response to change in dosage makes bethanidine a more satisfactory drug. It is usually given three to four times daily at a dose not normally exceeding a total of 200 mg per day. Debrisoquine is a very similar drug to bethanidine. All of these drugs reduce glomerular filtration and so fluid retention may occur.

Professor MacGregor: Drug interaction may occur with sympathomimetics such as ephedrine, amphetamine, or adrenaline. When these are given to a patient on an adrenergic neurone blocker, a definite hypertensive reaction occurs. Similarly, when guanethidine or bethanidine is given to a patient whose hypertension is due to release of catecholamines from a phaeochromocytoma, a pressor response can occur and may lead to that diagnosis being suspected.

Dr. Petrie: We must also emphasize that the initial dosage of any adrenergic neurone blocker must be very small. For example, bethanidine should not be introduced at larger dosage than 5 mg three times a day.

Professor MacGregor: You used bethanidine, but did you consider ganglion blockers on admission?

House Physician: I had intended to use pentolinium intramuscularly thinking that papilloedema and a pressure of 280/160 mm Hg required urgent treatment.

Dr. Wood: The reason I advised against using this was that the left heart failure, which is common in very severe hypertension, was the first priority. Of course, you are quite right about the urgent need to reduce the pressure, but, as you saw, bethanidine was very effective. If this man had had hypertensive encephalopathy bethanidine could have been used intravenously. Even so, I must admit that some doctors still use ganglion blockers, especially in hypertensive encephalopathy. You will find that pentolinium for parenteral use and mepyramine for oral use are kept in the ward pharmacy here.

Dr. Petrie: I can see no indication now for an oral ganglion blocker. Why did you use a thiazide and bethanidine when the patient had renal insufficiency?

Dr. Wood: Methyldopa is less effective in very severe hypertension. But your point is valid—if the blood urea rises further one can add methyldopa and then reduce the dose of bethanidine, as there is evidence of synergism between the two drugs. We might also be able to stop the diuretic, especially now that the heart failure has been relieved.

Professor MacGregor: So far we have discussed patients for whom there has been no debate about the actual need for treatment. But we do see cases where no treatment is really needed at all.

Dr. Wood: We had a good example recently. This was a woman of 75 who had fallen and sustained a Colles fracture. She came to a medical ward because she was too dizzy to be allowed home. Two days before her admission she had...
complained of slight dizziness and had been started on betanidine 50 mg thrice daily after her blood pressure had been found to be 200/80 mm Hg. She should never have had betanidine, and in any case the dose was quite excessive.

**House Physician:** The postural effect was really most striking. Her supine blood pressure was 150/80 mm Hg but it fell to under 100 mm Hg systolic when she sat up.

Prof. MacGregor: This is a typical case of systolic hypertension in the elderly, and it requires no treatment. It is possible to criticize the pharmaceutical industry for their promotion of hypotensives, but I must blame the doctor here. He could have understood hypertension very incompletely, and the drug not at all. This sort of misadventure is by no means uncommon.

**Case 4—Asymptomatic Hypertension**

Dr. Petrie: This man works as an executive and is 35 years old. At an insurance medical examination he was found to have a blood pressure of 140/90 mm Hg. In the ward serial diastolic pressures were in a range of 95 to 105 mm Hg. There were no other abnormal findings.

**Prof. MacGregor:** Were any of these readings obtained in the basal state?

**House Physician:** We took the blood pressure three or four times early one morning before he had breakfasted or got out of bed, and the diastolic pressure was found to be between 95 and 100 mm Hg.

Prof. MacGregor: I must repeat my doubts about whether treatment has any effect on the ultimate prognosis in this kind of patient. Dr. Wood, I know you would like to treat him.

Dr. Wood: Yes, I would, though I accept that only a controlled trial of therapy will ever settle the issue. But one factor which makes it easier to decide whether to treat such patients is the relative lack of side effects of drugs we now use in mild hypertension. A thiazide is possible here, but I would choose a beta adrenergic blocking drug.

Prof. MacGregor: They are being increasingly used, and not just in mild hypertension. What place do you see for them?

Dr. Wood: Their effect on moderate and severe hypertension may be a matter of dosage. With propranolol in high dosage one can certainly obtain good supine and standing control in quite severe hypertension. In lower dosage—that is, up to 150 mg a day—the drug has a range of activity comparable with the thiazides.

Propranolol seems quite satisfactory for treating both mild and moderate hypertension. A patient like this man would start on 20 mg three or four times daily and might need no increase in dosage.

Prof. MacGregor: Some authors have reported the use of propranolol in doses of 1,500 mg per day.

Dr. Petrie: We have no experience of this kind of dose. For the most part we use propranolol in mild hypertension but sometimes, when renal function is poor, we use it to reduce the doses needed of the adrenergic neurone blockers.

Prof. MacGregor: This term "beta blocking drug" is just a name to many doctors, I think you ought to explain what it means.

**Beta Effects**

Dr. Petrie: Though noradrenaline is the transmitter substance at all adrenergic nerve endings, sometimes adrenaline has a greater effect. These effects, which are inhibitory except on the heart, are called beta effects (Fig. 1). They lead to relaxation of the bronchi, decrease in the vascular tone of the muscle and liver, and reduced tone and motility in the gut. The stimulatory effects on the heart cause an increase in rate, improved conduction, increased force of ventricular contraction, and an increased tendency for ectopic pacemakers to discharge. Beta effects, which can be obtained clinically by the use of synthetic drugs such as isoprenaline—a beta acting agent with minimal alpha effect—are antagonized by beta blocking drugs like propranolol.

**House Physician:** Since isoprenaline, which is a beta stimulator, causes bronchial relaxation, presumably propranolol causes bronchoconstriction?

Dr. Petrie: Yes, propranolol must not be used in bronchitics or asthmatics, and it may even cause some normal people to start wheezing. Practolol, a newer drug, has less pronounced effects on the bronchi but remains quite a potent beta blocker on the heart.

Prof. MacGregor: By definition beta blocking drugs depress the heart, so that they are contraindicated in patients with latent or overt heart failure.

Dr. Wood: The hypotensive action of the beta blockers arises because of their action on the heart, not on peripheral resistance. This is why there are no postural effects and why the hypotensive response can summate with that caused by adrenergic neurone blockers.

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Dr. Wood: The hypotensive action of the beta blockers arises because of their action on the heart, not on peripheral resistance. This is why there are no postural effects and why the hypotensive response can summate with that caused by adrenergic neurone blockers.
Physician: But does not phentolamine, an alpha blocking drug, also reduce blood pressure?

Dr. Wood: Yes, but alpha blockade is very hard to control, and is almost never used therapeutically. Alpha effects are mostly stimulatory and noradrenaline produces these more readily than adrenaline (Fig. 2). They include vasoconstriction in the gut and skin vessels, and pupillary dilatation due to contraction of the radial muscle of the iris. Synthetic amines such as metaraminol, which have mostly alpha effects, are, and may be used as, pressors. Alpha blocking drugs like phentolamine antagonize metaraminol and the alpha effects of noradrenaline. Their hypotensive action is most definite when the blood pressure is being kept up by noradrenaline release.

This was the basis for using phentolamine in a diagnostic test for phaeochromocytoma.

Professor Macgregor: To sum up, an understanding of the pharmacology of the sympathetic nervous system is essential if the drugs used in hypertension are to be used safely and effectively. The four patients illustrate the spectrum of essential hypertension. The more severe cases need treatment and the outlook is improved by treatment. The problem area remains mild hypertension, where the effect of treatment on prognosis is uncertain and where each doctor must still make up his own mind about the lower level of a raised blood pressure which requires treatment.

Hospital Topics

Obstetric Anaesthesia Services in the United Kingdom

Gordon Taylor

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Summary

In a survey of obstetric anaesthetic services in the United Kingdom questionnaires were sent to 398 hospital maternity units and 347 general-practitioner maternity units, of which 344 and 272 respectively were returned. Many hospitals were unable to provide an anaesthetist for obstetric surgery only, and few consultant anaesthetist sessions were allocated to obstetric surgery, particularly in regional hospitals in England and Wales. Constant supervision of junior anaesthetic staff with under 12 months' experience was lacking in several hospitals. Endotracheal intubation is widely used throughout the United Kingdom. Though regional analgesic techniques are used by most anaesthetists it is impossible to provide a 24-hour regional analgesic service in all but a few hospitals.

Introduction

The Report on Confidential Enquiries into Maternal Deaths in England and Wales, 1964-66 (Arthure et al., 1969) has given rise to anxiety in anaesthesia and obstetric circles (Crawford, 1970; British Medical Journal, 1970). While every other cause of maternal death has decreased in the past 15 years, those due to anaesthesia have increased. The confidential inquiry does not set out to give the reasons for these deaths. As a result the possible deficiencies which occur during obstetric anaesthesia are left to conjecture.

The present survey was designed to determine as near as possible the anaesthetic cover for maternity units over the 24-hour period, including the number of consultant obstetric

anaesthetic sessions. Other aspects of the survey dealt with types of general anaesthesia used and whether regional analgesic techniques were available. In addition, the anaesthetist's traditional role in neonatal resuscitation was examined.

Method

As a result of a pilot survey conducted in about 90 hospitals a more comprehensive inquiry was carried out. The present survey was divided into two phases. Firstly, secretaries of hospital management committees concerned in maternity services were asked to give the names of hospitals with maternity beds, the number of deliveries there per annum, and the name of the consultant anaesthetist responsible for the obstetric anaesthesia service; 95% of the circulars were returned. Secondly, two types of questionnaire were sent to the consultant anaesthetist in charge. The first, which comprised three pages and was designed for maternity units in hospitals, inquired into anaesthetic cover for obstetric surgery, including the number of consultant obstetric anaesthetic sessions, and also the use of general anaesthetic and regional analgesic techniques. A second, short questionnaire was for general-practitioner units where essentially only normal deliveries occur. In all instances first-class postage was used and stamped addressed envelopes were enclosed. The dispatch and return of questionnaires was completed between October 1969 and February 1970. The data were then put on to Visiscan punched feature cards and the results checked.

Results

For this survey the United Kingdom was divided into four parts—namely, teaching hospitals in England and Wales (those hospitals as defined by the Hospital Year Book, 1970), regional hospitals in England and Wales, and all hospitals in Scotland and in Northern Ireland. The coverage of the survey and the number of questionnaires returned from hospital maternity units are shown in Table I. The survey had a bet-