ABC of AIDS
Development of the epidemic

Michael W Adler

This article has been adapted from the forthcoming 5th edition of ABC of AIDS. *The book will be available from the BMJ bookshop and at www.bmjbooks.com*

The first recognised cases of the acquired immune deficiency syndrome (AIDS) occurred in the summer of 1981 in America. Reports began to appear of *Pneumocystis carinii* pneumonia and Kaposi's sarcoma in young men, who it was subsequently realised were both homosexual and immunocompromised. Even though the condition became known early on as AIDS, its cause and modes of transmission were not immediately obvious. The virus now known to cause AIDS in a proportion of those infected was discovered in 1983 and given various names. The internationally accepted term is now the human immunodeficiency virus (HIV). Subsequently a new variant has been isolated in patients with West African connections—HIV-2.

The definition of AIDS has changed over the years as a result of an increasing appreciation of the wide spectrum of clinical manifestations of infection with HIV. Currently, AIDS is defined as an illness characterised by one or more indicator diseases. In the absence of another cause of immune deficiency and without laboratory evidence of HIV infection (if the patient has not been tested or the results are inconclusive), certain diseases when definitively diagnosed are indicative of AIDS. Also, regardless of the presence of other causes of immune deficiency, if there is laboratory evidence of HIV infection, other indicator diseases that require a definitive, or in some cases only a presumptive, diagnosis also constitute a diagnosis of AIDS.

In 1993 the Centers for Disease Control (CDC) in the USA extended the definition of AIDS to include all persons who are severely immunosuppressed (a CD4 count $< 200 \times 10^6$ cells/l) irrespective of the presence or absence of an indicator disease. For surveillance purposes this definition has not been accepted within the UK and Europe. In these countries AIDS continues to be a clinical diagnosis defined by one or more of the indicator diseases mentioned. The World Health Organization (WHO) also uses this clinically based definition for surveillance within developed countries. WHO, however, has developed an alternative case definition for use in sub-Saharan Africa. This is based on clinical signs and does not require laboratory confirmation of infection. Subsequently this definition has been modified to include a positive test for HIV antibody.

These case definitions are complex and any clinician who is unfamiliar with diagnosing AIDS should study the documents describing them in detail.

AIDS defining conditions without laboratory evidence of HIV

- Diseases diagnosed definitely
- Candidiasis: oesophagus, trachea, bronchi, or lungs
- Cryptococcosis: extrapulmonary
- Cryptosporidiosis with diarrhoea persisting > 1 month
- Cytomegalovirus disease other than in liver, spleen, nodes
- Herpes simplex virus (HSV) infection
  - Mucocutaneous ulceration lasting > 1 month
  - Pulmonary, oesophageal involvement
- Kaposi's sarcoma in patient < 60 years of age
- Primary cerebral lymphoma in patient < 60 years of age
- Lymphoid interstitial pneumonia in child < 13 years of age
- *Mycobacterium avium*: disseminated
- *Mycobacterium kansasii*: disseminated
- *Pneumocystis carinii* pneumonia
- Progressive multifocal leucoencephalopathy
- Cerebral toxoplasmosis

AIDS defining conditions with laboratory evidence of HIV

Diseases diagnosed definitely

- Recurrent/multiple bacterial infections in child < 13 years of age
- Coccidiomycosis—disseminated
- HIV encephalopathy
- Histoplasmosis—disseminated
- Isosporiasis with diarrhoea persisting > 1 month
- Kaposi's sarcoma at any age
- Primary cerebral lymphoma: at any age
- Non-Hodgkin's lymphoma: diffuse, undifferentiated B cell type, or unknown phenotype
- Any disseminated myobacterial disease other than *M tuberculosis*
- Mycobacterial tuberculosis
- Salmonella septicaemia: recurrent
- HIV wasting syndrome
- Recurrent pneumonia within 1 year
- Invasive cervical cancer

Diseases diagnosed presumptively

- Candidiasis: oesophagus
- Cytomegalovirus retinitis with visual loss
- Kaposi's sarcoma
- Mycobacterial disease (acid-fast bacilli; species not identified by culture): disseminated
- *Pneumocystis carinii* pneumonia
- Cerebral toxoplasmosis

Transmission of the virus

HIV has been isolated from semen, cervical secretions, lymphocytes, cell-free plasma, cerebrospinal fluid, tears, saliva, urine, and breast milk. This does not mean, however, that these fluids all transmit infection since the concentration of virus in them varies considerably. Particularly infectious are semen, blood, and possibly cervical secretions. The commonest mode of transmission of the virus throughout the world is by sexual intercourse. Whether this is anal or vaginal is unimportant. Other methods of transmission are through the receipt of infected blood or blood products, donated organs, and semen. Transmission also occurs through the sharing or reuse of contaminated needles by injecting drug users or for therapeutic procedures, and from mother to child. Transmission from

mother to child occurs in utero and also possibly at birth. Finally, the virus is transmitted through breast milk.

The virus is not spread by casual or social contact. Health care workers can, however, be infected through needlestick injuries, and skin and mucosal exposure to infected blood or body fluids. Prospective studies in health care workers suffering percutaneous exposure to a known HIV seropositive patient indicate a transmission rate of 0.32%. As of December 1999 there have been 96 reported cases of documented seroconversion after occupational exposure in such workers.

The precautions and risks for such groups are covered in detail in chapter 15. Finally, there is no evidence that the virus is spread by mosquitoes, lice, bed bugs, in swimming pools, or by sharing cups, eating and cooking utensils, toilets, and air space with an infected individual. Hence, HIV infection and AIDS are not contagious.

Transmission of the virus

- Sexual intercourse
  Anal and vaginal
- Contaminated needles
  Injecting drug users
  Needlestick injuries
  Injections
- Mother → child
  In utero
  At birth
  Breast milk
- Organ/tissue donation
  Semen
  Kidneys
  Skin, bone marrow, corneas, heart valves, tendons, etc

Growth and size of the epidemic

Even though North America and Europe experienced the first impact of the epidemic, infections with HIV are now seen throughout the world, and the major focus of the epidemic is in developing/resource-poor countries.

Worldwide

The joint United Nations programme on AIDS (UNAIDS) has estimated that by the end of 1999 there were 34.3 million people living with HIV/AIDS (33.0 million adults and 1.3 million children < 15 years). The new infections during that year were 5.4 million, approximately 15 000 new infections per day.

Currently, 95% of all infections occur in developing countries and continents, the major brunt of the epidemic being seen in sub-Saharan Africa and South East Asia. It is now recognised that cases of AIDS were first seen in Central Africa in the 1970s even though at that time it was not recognised as such. Current surveys from some African countries show that the prevalence of infection is high amongst certain groups—50-90% of prostitutes, up to 60-70% of those attending departments for sexually transmitted diseases and antenatal

HIV Transmission: global summary

Type of exposure	Percentage of global total
Blood transfusion	3-5
Perinatal	5-10
Sexual intercourse	70-80
(vaginal)	(60-70)
(anal)	(5-10)
Injecting drug use (sharing needles, etc)	5-10
Health care (needlestick injury, etc)	< 0.01

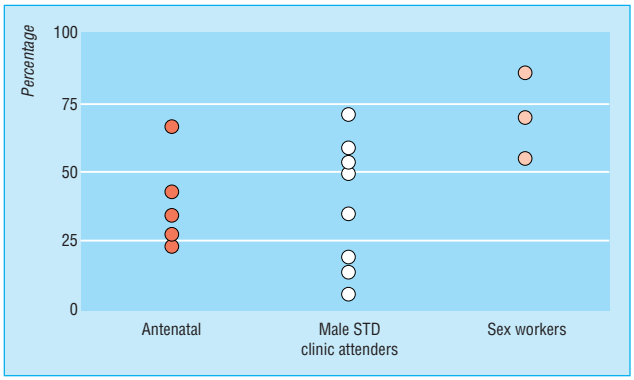
Regional HIV/AIDS statistics and features, end of 2000

Region	Epidemic started	Adults and children living with HIV/AIDS	Adults and children newly infected with HIV	Adult prevalence rate*	% of HIV positive adults who are women	Main mode(s) of transmission† for adults living with HIV/AIDS
Sub-Saharan Africa	late 1970s to early 1980s	25.3 million	3.8 million	8.8%	55%	Hetero
North Africa and Middle East	late 1980s	400 000	80 000	0.2%	40%	Hetero, IDU
South and South East Asia	late 1980s	5.8 million	780 000	0.56%	35%	Hetero, IDU
East Asia and Pacific	late 1980s	640 000	130 000	0.07%	13%	IDU, hetero, MSM
Latin America	late 1970s to early 1980s	1.4 million	150 000	0.5%	25%	MSM, IDU, hetero
Caribbean	late 1970s to early 1980s	390 000	60 000	2.3%	35%	Hetero, MSM
Eastern Europe and Central Asia	early 1990s	700 000	250 000	0.35%	25%	IDU
Western Europe	late 1970s to early 1980s	540 000	30 000	0.24%	25%	MSM, IDU
North America	late 1970s to early 1980s	920 000	45 000	0.6%	20%	MSM, IDU, hetero
Australia and New Zealand	late 1970s to early 1980s	15 000	500	0.13%	10%	MSM
Total		**36.1 million**	**5.3 million**	**1.1%**	**47%**	

*The proportion of adults (15-49 years of age) living with HIV/AIDS in 2000, using 2000 population numbers.
†Hetero = heterosexual transmission; IDU = transmission through injecting drug use; MSM = sexual transmission among men who have sex with men.

clinics. In the developing world, HIV is spread mainly by heterosexual intercourse.

At a family level, UNAIDS estimates that by the end of 1999 the epidemic has left behind a cumulative total of 13.2 million AIDS orphans (defined as those having lost their mother or both parents to AIDS before reaching the age of 15 years). Many of these maternal orphans have also lost their father. Orphans in Zimbabwe are expected to total 1 million by 2005 and 2 million in South Africa by 2010. Traditional family structures and extended families are breaking down under the strain of HIV. Population growth and death rates are increasingly affected. Life expectancy in countries with adult prevalences of over 10% (for example, Botswana, Kenya, Zimbabwe, South Africa, Zambia, Rwanda) are expected to see an average reduction in life expectancy of 17 years by 2010-2015. Young, highly productive adults die at the peak of their output, which has a considerable impact on a country's economy.



Prevalence of HIV—different groups

USA, UK and Europe

By June 1999, 702 748 adult cases of AIDS had been reported in the USA. In addition there were 8596 paediatric cases (< 13 years old). Most of the cases in children (91%) occur because a patient suffered from HIV or belonged to a group at increased risk of HIV; 4% occurred through blood transfusion; 3% in children with haemophilia. Information on risk factors for the remaining 2% of the parents of these children is not complete.

Adult cases in Europe totalled 224 359 by June 1999, and those in the UK 12 780. There are five times more people infected with HIV at any one time than have AIDS. The rate for AIDS cases varies throughout Europe, with particularly high rates in Italy, Portugal, Spain, France and Switzerland, where the commonest mode of infection is through injecting drug use and the sharing of needles and equipment.

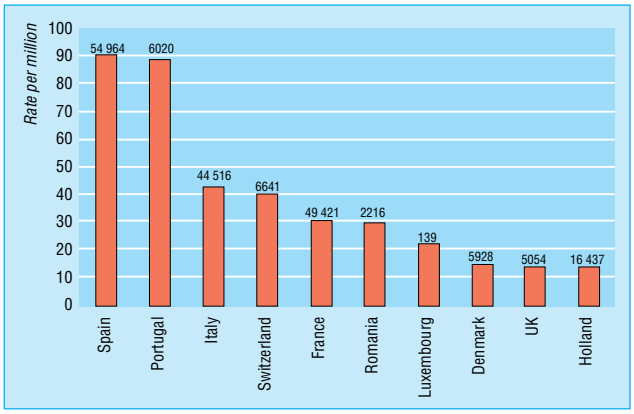
In North America and the UK the first wave of the epidemic occurred in homosexual men. In the UK, proportionally more homosexual men have been notified than in America: 66% of cases compared with 48% respectively. Even though infections amongst men who have sex with men still arise, an increasing proportion of new infections in the USA is occurring amongst injecting drug users sharing needles and equipment. There is also an increase amongst heterosexuals in both the USA and the UK. Currently in the USA, 16% of cases of AIDS have occurred amongst women, and although the commonest risk factor amongst such women is injecting drug use (42%), the next most common mode of transmission is heterosexual contact (40%).

The nature of the epidemic within the UK is changing with more heterosexual transmission. In the UK 12% of adult cases of AIDS have occurred in women, 70% of which have resulted from heterosexual intercourse. In 1999 there were more new annual infections of HIV than ever before and for the first time more occurring as a result of heterosexual sex than men having sex with men. Most heterosexually acquired infections are seen in men and women who have come from or have spent time in sub-Saharan Africa.

The advent of an antibody test in 1984 has allowed for a clearer understanding of the changing prevalence and natural history of HIV infection. Surveys show that the proportion of individuals infected needs to be high before cases of AIDS start to become apparent. It also underlines the importance of health education campaigns early in the epidemic, when the seroprevalence of HIV is low. Once cases of AIDS start to appear the epidemic drives itself and a much greater effort is required in terms of control and medical care.

AIDS: adult patient groups in the USA and UK

	USA (June 99)		UK (Dec 99)	
Patient groups	**n**	**%**	**n**	**%**
Men who have sex with men	334 073	48	11 063	66
Intravenous drug user	179 228	26	1 065	6
Men who have sex with man and IV drug user	45 266	6	293	2
Received blood/haemophilia	13 440	2	813	5
Heterosexual contact	70 582	10	3 049	18
Mother to infant			349	2
Other/undetermined	60 159	8	174	1
Total	**702 748**	**100**	**16 806**	**100**

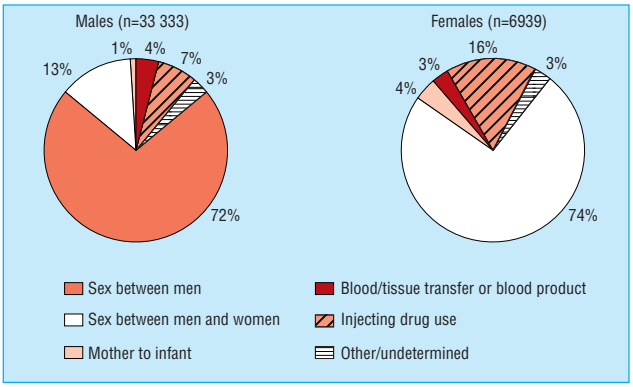


AIDS in Europe—top ten countries 1999

Within countries one finds considerable variation in seroprevalence levels for HIV. Over 70% of cases of AIDS and HIV infection within the UK occur and are seen in the Thames regions (London and the surrounding area). Among different groups one also finds geographic differences. For example, the rates among drug users is higher in Edinburgh than London, and for gay men higher in London than anywhere else in the UK. This is also found in the developing world—for example, in Tanzania and Uganda, the urban level of HIV infection in men and women can be five times higher than rural rates.

The use of highly active antiretroviral therapy in resource-rich countries has resulted in an increase in life expectancy. This, in combination with the increase in new HIV infections, means that the prevalent pool of those infected, and potentially infectious, is increasing. This presents a continuing challenge for health promotion and a re-statement of the importance of safe sex techniques, particularly condom use.

AIDS results in a considerable cost not only in human suffering, but also to health services. Other costs include time off work and the effect of the deaths of young people on national productivity. AIDS represents a major public health problem in the world. A clear understanding of the epidemiology forms the basis of developing a strategy or control ranging from health education to research.



HIV infected individuals diagnosed in the UK by exposure category: to end of 1999

The data on AIDS/HIV in the UK is reproduced with permission from the Communicable Disease Surveillance Centre (CDSC) and the United Nations AIDS Programme.

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Evidence based management of hypertension

What to do when blood pressure is difficult to control

Jane E O'Rorke, W Scott Richardson

There is no universally accepted definition of blood pressure that is difficult to control, uncontrolled, resistant, or refractory. One consensus based US national guideline defines resistant hypertension as blood pressure that cannot be reduced below 140/90 mm Hg (below 160 mm Hg for isolated systolic hypertension) in patients who are complying with adequate triple drug regimens in appropriate dosage.[1] This definition covers many hypertensive patients but not those for whom the target is an even lower pressure.

Both clinical experience and research surveys suggest that resistant blood pressure is common in everyday practice. For example, population and clinic surveys in North America, Europe, and Australia show that in as many as 50% to 75% of people being treated for hypertension target blood pressure levels are not achieved.[2] Fifty per cent of the 19 196 participants in one recent trial had uncontrolled blood pressure levels when enrolled despite already being treated for hypertension; 59% of these uncontrolled patients were being treated with a single drug, and 41% with two drugs. During the trial, 72% of patients needed more than one drug to reduce their diastolic blood pressures to 80 mm Hg.[3] In another large trial in hypertensive diabetic patients, 60% of the participants needed two or more drugs to achieve blood pressure levels of less than 150/85 mm Hg, and 33% needed three or more drugs.[4] These findings confirm that, even when patients are closely followed, blood pressure can be difficult to control.

Summary points

Consider the following causes for apparently resistant blood pressure: inaccurate measurement, antagonising substances such as non-steroidal anti-inflammatory drugs, aggravating conditions such as obesity or sleep apnoea, suboptimal treatment regimens, non-compliance

When apparent resistance remains unexplained or when there are clues suggesting white coat hypertension, consider this and arrange for multiple measurements by self monitoring, visits to or by nurses or health visitors, or ambulatory monitoring

If hypertension is still unexplained, or for patients who fit specific patterns of higher risk, consider a selective, sequential evaluation for secondary causes of hypertension, starting with relatively common conditions, such as renovascular causes and renal parenchymal disease

Work closely with patients to identify preferred and feasible solutions for correcting any cause that is found

Consider referral of patients with severe or persistently resistant hypertension to a centre specialising in its diagnosis and treatment

This is the last in a series of five articles

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AP PHOTO/JIM COLE

Measurements of blood pressure taken in the patient's home can be combined with those obtained in the doctor's office to create a fuller picture of blood pressure control

Assessing cause and incidence of resistant blood pressure

The categories of causes of resistant hypertension are

- Inaccurate blood pressure measurement
- White coat hypertension
- Disease progression
- Suboptimal treatment
- Non-compliance with prescribed treatments
- Antagonising substances
- Coexisting conditions
- Secondary hypertension.

Two or more of these categories may be relevant in one patient.

There is little evidence as to how often these eight categories are found to be responsible for difficulty in controlling blood pressure, but a small descriptive survey from a referral clinic gave the following relative proportions: suboptimal treatment, 40%; non-adherence to prescribed treatment, 10% to 50%; white coat hypertension, 2% to 4%; and secondary causes, 10%.[5] The figures could be quite different in primary care, with lower rates of secondary causes, but there is little direct evidence on the point.

Inaccurate blood pressure measurement

The accuracy of blood pressure readings depends on the use of proper technique and on the conditions under which the measurements are made. Before you conclude that a patient has resistant hypertension, blood pressure measurements should be repeated under good conditions and with as near to ideal technique as possible.

White coat hypertension

Some patients have acceptably controlled blood pressure while they are at home but have higher readings when examined by the clinician, who may be misled to think that the patient's blood pressure is poorly controlled. To exclude this possibility, arrange for multiple measurements of the patient's blood pressure by, for example, visits to or from nurses or other healthcare workers, self monitoring with a home sphygmomanometer, or ambulatory blood pressure monitoring.

If good technique is used with well calibrated equipment, these measurements can be combined with those in the doctor's office to create a fuller picture of blood pressure control. If these external readings are also persistently raised, one can conclude that the blood pressure is not yet satisfactorily controlled.

Disease progression

With time, the blood pressure in adults with hypertension will gradually increase.[6] There is no recent, rigorous evidence as to how often disease progression is the sole cause of resistant hypertension, but without firm evidence to the contrary it seems unwise to accept it as such until other causes have been excluded.

Suboptimal treatment

The regimens prescribed for patients may not be optimally individualised for many reasons. Many patients require aggressive treatment with several drugs to achieve target blood pressure levels. To detect suboptimal treatment, review all the drugs a patient is taking, as well as the patient's dietary habits and exercise pattern. Consider whether the dosage of each drug prescribed conforms in all respects with recommendations for its rational use and with what is known about the patient's unique health status and preferences.

Hypervolaemia resulting from a high intake of dietary sodium frequently plays an important part in resistant hypertension, and better use of diuretics is often the answer when a patient's blood pressure is difficult to control.[7–9] Clinicians should review regimens regularly to see if the patients' treatment plans are optimal.

Non-compliance

No matter what treatments are prescribed, they will have no effect if drugs are not taken. For any number of reasons, patients may not take their drugs as prescribed or may not take them at all. Direct and non-judgmental questioning at routine clinic visits is the best way of detecting non-compliance. A systematic review of the discriminatory power of such simple questioning estimated its sensitivity to be 55% and its specificity to be 87%.[10]

Antagonists that can increase blood pressure

- Adrenal steroids (especially mineralocorticoids)
- Alcohol
- Amphetamines—for example, appetite suppressants
- Anaesthetics, local and general
- Antidiuretic hormone and angiotensin
- Caffeine
- Cocaine
- Cyclosporin
- Disulfiram
- Erythropoietin
- Licorice and carbenoxolone
- Monoamine oxidase inhibitors, combined with foods containing tyramine or with amphetamine
- Medications containing sodium—for example, antacids or parenteral antibiotics
- Non-steroidal anti-inflammatory drugs
- Oral contraceptives
- Sympathomimetic agents—for example, nasal decongestants or bronchodilators
- Withdrawal of antihypertensive agents—for example, β blockers or clonidine

Antagonising substances

Patients may be taking many prescription and non-prescription drugs and dietary and other substances that can increase blood pressure or oppose the actions of antihypertensive drugs (see box). No rigorous studies have evaluated the frequency or magnitude of effects of such substances on blood pressure. Non-steroidal anti-inflammatory drugs (NSAIDs) account for 5% to 10% of all prescriptions in developed countries.[11 12] If they do raise blood pressure or oppose the effects of antihypertensive drugs, then they could, because of the extent to which they are taken, have a considerable impact on blood pressure control.

Two systematic reviews have examined the effect of non-steroidal anti-inflammatory drugs on blood pressure. One summary of findings from 54 trials found that treatment with non-steroidal anti-inflammatory drugs increased mean arterial blood pressure by 1.1 mm Hg in normotensive patients and by 3.3 mm Hg in patients with hypertension.[13] Most of these trials were short (less than six weeks), and none included elderly patients. Among the non-steroidal anti-inflammatory drugs, indometacin had the largest effect on blood pressure and aspirin the least. Another systematic review summarised 50 trials and estimated that non-steroidal anti-inflammatory drugs increased mean supine blood pressure by 5 mm Hg.[14]

Coexisting conditions

While usually not considered as causes of secondary hypertension, several disorders may coexist in patients with hypertension and either increase blood pressure or interfere with its treatment. These include

- Alcohol use, more than 12-14 g of absolute alcohol per day
- Anxiety disorders, including hyperventilation or panic attacks
- Delirium, with agitation and autonomic excess
- Hyperinsulinism with insulin resistance
- Obesity
- Pain, acute or chronic
- Pregnancy
- Sleep apnoea
- Smoking

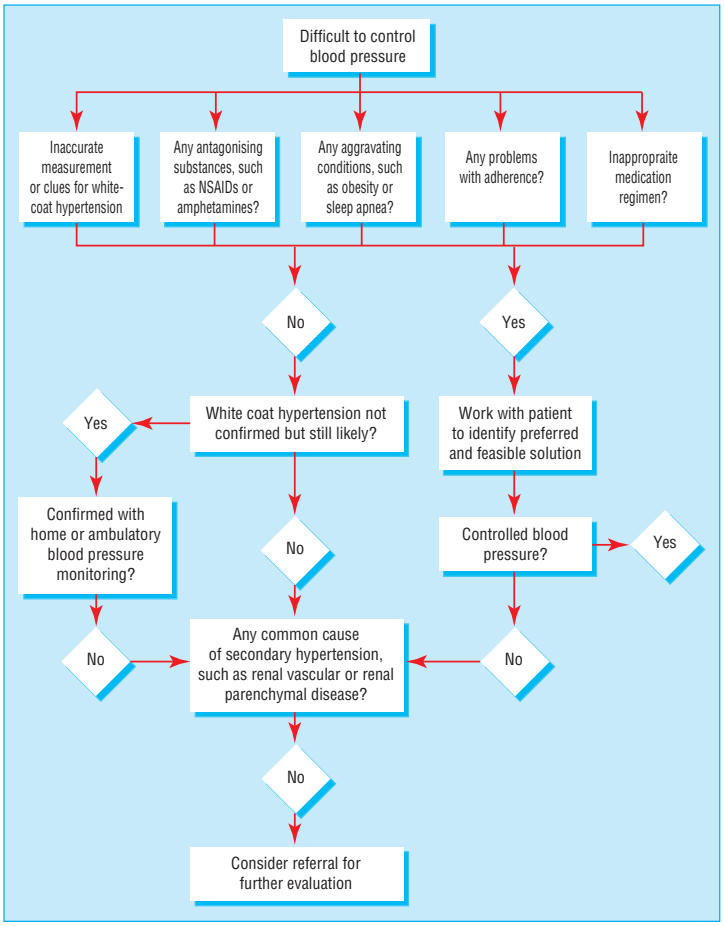
Both clinical experience and research suggest that some of these conditions commonly coexist with hypertension. For example, population surveys have repeatedly shown that up to 40% of people with hypertension are obese.[15] Obesity can interfere with accurate blood pressure measurement, can truly increase blood pressure, and can interfere with the effectiveness of antihypertensive drugs. Recognising the coexistence of such disorders makes it possible to treat them and so, potentially, improve control of blood pressure, although to what extent is not clear.

Secondary hypertension

By definition, patients with secondary hypertension have an underlying disorder believed to be a direct cause of their hypertension. Detecting such a disorder offers the prospect of giving specific treatment that will lower the blood pressure to normal without the need for specific antihypertensive treatment.

The prevalence of secondary hypertension in primary care is uncertain but it is probably an uncommon form of hypertension. Studies of secondary hypertension and the relative proportions of its causes have had several methodological limitations, including selection of patients from referral settings, differing diagnostic criteria, and differing evaluations.[16–20] For example, a study from a referral clinic in the 1960s reported the prevalence of renovascular disease as 4.4%, of phaeochromocytoma as 0.2%, and of hyperaldosteronism as 0.4%.[16] At another referral clinic in the early 1970s, the prevalence of renovascular disease was 0.18%, while that of phaeochromocytoma was 0.04% and that of hyperaldosteronism was 0.01%.[16] Such rates observed at referral centres are probably much higher than those seen in primary care today. Given this evidence, renovascular and renal parenchymal disorders are the most likely causes of such secondary hypertension seen in primary care practice.

The overall rarity of disorders causing secondary hypertension presents a diagnostic challenge. Experts recommend investigation of patients who seem to be at above average risk on such epidemiological grounds as age and sex or on the presence of symptoms or signs of specific disorders. With the exception of renal artery stenosis,[21–23] we could not find evidence of how often presenting patterns thought to be associated with specific secondary causes occur among all patients with high blood pressure in primary care settings. Furthermore, we found relatively little evidence about how powerfully these features discriminate between those who do and those who do not have secondary hypertension.



A commonsense approach to evaluating resistant hypertension

Determining the cause(s) of hypertension and adjusting treatment plans

Ideally we would like to recommend a coherent strategy for evaluating and managing patients whose blood pressure is difficult to control, made up of well studied elements aggregated into a systematic approach itself shown to do more good than harm. Unfortunately, there is little or no evidence beyond expert opinion to guide clinicians, so, as a result, our recommendations, shown in the figure, are tentative. Although the recommendations are presented sequentially, we recommend that clinicians consider many of the options simultaneously and use their own judgment as to the appropriate order of queries.

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The book *Evidence-Based Hypertension*, edited by Cynthia D Mulrow, can be purchased through the BMJ Bookshop (www.bmjbookshop.com).

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A memorable patient

My namesake

While reading in Mikhail Bulgakov's *A Country Doctor's Notebook* about the author's experiences of clinical situations that, as a newly qualified doctor in 1916, he was really quite unqualified to deal with, both in terms of education and experience, my mind turned to my own exposure to such problems when I was newly qualified in the 1940s. I think things are better now.

My baptism came one January night. I had done one surgical house job, and while awaiting my call up to military service, which came to us all in those days, I was filling in time as a locum for an elderly, singlehanded practitioner. Part of the practice remit was the care of a small midwifery unit.

The telephone rang in the small hours. "Night sister speaking, doctor. I have a primip who has been in the second stage for more than two hours. I think she needs a forceps delivery."

Time spent on reconnaissance is never wasted, so I had a quick look at the book. "Assess the lie of the head. Which way are the ears pointing? Which blade goes in first? Why does this have to happen to me?"

I arrived at the unit. "Who gives the anaesthetic, sister?" I asked. I had not heard of pudendal block at that stage, and they would not have been equipped.

"You start her off, doctor, and I'll take over."

Induction by chloroform with rag and bottle is perhaps as easy a ride as one can give a novice. To my surprise, it seemed to go reasonably well, and the low forceps delivery was easier than I had any right to expect.

Sister guided me through tying off the cord and delivering the placenta and then gave me a welcome cup of tea, which I drank while chatting to the new mother and making what I hoped were appropriate comments about her new son.

"What is your first name doctor?" asked the mother.

I was a little hesitant in answering. First names in those days, in contrast to today, were the preserve of family and close friends. "Richard," I answered.

"Then I'll call him Richard."

Driving back to my digs with that warm glow when the gods have been kind, I pondered. This business of names—it must be a frequent compliment.

After the army, and a couple of obstetric house jobs, I found myself in a country practice with its own cottage hospital and nine obstetric beds and a turnover of some 240 deliveries a year. Many years and scores of babies later, I retired, but it never happened again.

I wonder where young Richard is now? He must be 54.

R C Humphreys *former general practitioner, Crickhowell, Powys*

We welcome articles of up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake*, or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk. Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.