

blood pressure

all you need to know

Hugh Coni
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About the Authors

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Foreword

Hypertension has been one of the success stories of modern medicine, up to a point – it is easy to diagnose (in most patients) and relatively easy to treat (in most patients). When writing a prescription for hypertension, we have more classes of drugs to choose from than when prescribing for any other common condition. Most of these drug classes have been shown as effective not only in reducing blood pressure, but also in preventing the long-term complications of hypertension, namely heart attacks and strokes. Yet most patients either do not receive the drugs that they need or have not been convinced that daily consumption of medicines for an asymptomatic condition is, as CS Lewis might have said, ‘the pain now that pays for the pleasure of avoiding strokes later’. Although hypertension is listed by the World Health Organization as the third most important cause of morbidity in the western world (after smoking and alcohol), there are only a handful of specialists in the UK. We therefore need help to spread information about the nature and importance of hypertension, and so the target audience of this book is particularly appropriate. Until a few years ago, the specialists, lacking evidence to the contrary, took the somewhat condescending view that high blood pressure in older people was a good thing, helping to push blood down the hardening arteries. Now we know, as the Conis illustrate well, that the contrary is true. Older people stand to gain the most from having their blood pressure lowered – simply because they are the most at risk if it is not.

Our confidence in our understanding of what hypertension is and what causes it is on shakier ground than our conviction about the merits of treatment. We understand well enough how blood pressure is controlled in healthy subjects; and at a superficial level we can divide hypertension into two types depending on the age of presentation (as in diabetes). Type 1, in younger patients, is associated with raised levels of the kidney hormone renin and responds well to drugs that suppress the renin system, eg ACE inhibitors and beta-blockers. Type 2, in older patients, is associated with suppressed levels of renin and responds best to drugs which eliminate salt and elevate renin, eg calcium channel blockers and diuretics. The serendipitous sequence of these drugs’ initials has let us enunciate an ABCD of hypertension – a rule which at once explains the condition and recommends appropriate treatment.

But peer below the surface, and our understanding of hypertension becomes vague – appropriate, perhaps, for a condition which we diagnose with a stethoscope applied to a large artery in the arm, but whose origins lie in the almost invisible arterial branches at the end of the arterial tree. We live in an era when true understanding of a disease demands not only detailed *dramatis personae*, goodies and baddies, in the form of multiple molecular sequences, but also an entire script for how these interact scene by scene. Not so long ago, professors argued for their favourite candidate gene as the single cause of hypertension – as likely to be resolved as the number of angels dancing on a pin. It took genetic studies of the well-recognized inherited contribution to hypertension to show that no single gene is responsible and that the number of genes involved is probably as large as that of the angels. Progress is being made in this research, but as the rate of progress slows, our confidence that genetics will provide the main answers to hypertension is ebbing away. This book explores some of the alternatives, including the controversial idea that we can blame our cardiovascular risk on our mothers' habits during pregnancy.

Whether the causes of hypertension will be found in our parents or our stars, books like this help to ensure that the consequences of hypertension will be strongly influenced by ourselves. Further knowledge of the causes of hypertension might satisfy intellectual curiosity and may eventually lead to yet more choice of treatments; but all patients can now have knowledge of the consequences and how to prevent them. The Conis draw on their rich and varied experience of hypertension in everyday practice to impart this knowledge in an intelligent and intelligible fashion. After all, 'the patient with knowledge is the patient in control'.

Morris J Brown

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Preface

Diseases are important for many reasons: because they are common, because they can be fatal, because they cause a great deal of disability, because they cause considerable suffering or because of their great cost to society. Hypertension fulfils all these criteria.

More and more people develop hypertension as they grow older and it is unbelievably prevalent among middle-aged and elderly people in the developed world. It probably affects over half the population aged over 65 years of industrialized countries and is mankind's most common chronic disease. Hypertension is associated with a very high mortality, especially from related coronary artery disease and stroke. Survivors of stroke comprise approximately 25% of severely disabled people living within the community. Few disorders can therefore have such a legitimate claim to be one of the greatest afflictions of our time.

Hypertension is almost always without symptoms. When we say 'I suffer from hypertension', we do not mean we are really 'suffering' in the accepted sense of the word. What we actually mean is that a doctor, or perhaps a life insurance company, has strongly hinted that they do not think we are going to live as long as we had hoped. Paradoxically, hypertension causes headache – but only in those who have been told they have hypertension!

This is what has made hypertension such an unfashionable disease. Not only is it very unglamorous, conjuring up cartoon images of portly, middle-aged, puce-faced business men, but even those afflicted do not really want to know about it. Who wishes to be constantly reminded of their own mortality? And as for doctors – a great deal of their job satisfaction comes from making people feel better, but you cannot make a symptom-free individual feel better with pills, although you can certainly make them feel worse.

Hypertension is therefore not always treated or monitored in the most appropriate ways, either by hospital specialists, GPs or those who have the condition. A visit to the out-patient clinic at the hospital is likely to be associated with:

- ❖ the stress of getting time off work
- ❖ a difficult journey to the hospital
- ❖ problems parking the car

- ❖ problems finding the right clinic
- ❖ being kept waiting before a brief consultation with a stranger.

Consequently, hypertension is mainly dealt with by GPs – the surgery may be closer, the parking easier and the nurse less intimidating, but visits are still likely to be very infrequent. No wonder that in the surgery, doctors are inclined to be satisfied with blood pressure levels that may be higher than are desirable, and simply hope that the rest of the time, the readings would be lower.

The best hope for all the hundreds of thousands, probably millions, of people with hypertension is to cultivate an informed interest in the subject, just like many diabetics take a keen interest in diabetes. They need to take charge of their own blood pressure control, loosely under the supervision of the GP or the specialist, to take their own readings which they share with their medical adviser, to adopt sensible lifestyle changes and to discuss their medication. They need to become what the Department of Health has called ‘expert patients’ – an idea borrowed from the University of Stanford, California. This book will try to educate those affected and their families to enable them to do so. The authors are a GP who undertakes the supervision of most of the hypertensive patients in his practice and a recently retired hospital physician who takes regular medication for high blood pressure.

During the 1990s, a new buzz-word (or buzz-phrase) infiltrated medi-speak: it was ‘evidence-based medicine’ (EBM). Medicine has, in fact, been evidence-based ever since Sir Austin Bradford Hill devised the trials which were to provide statistical proof of the effectiveness of certain drugs against tuberculosis in the late 1940s and early 1950s – EBM was not exactly a new idea! Most of the information in this book is evidence-based, and so is the general concept that the ‘expert patient’ enjoys improved healthcare outcomes. However, we have to acknowledge that the core themes of this book (dissemination of information to the public and self-measurement of blood pressure) are only now beginning to enjoy an evidence base. Nevertheless one can die of a heart attack or a stroke while waiting for the evidence to become rock-solid, and there are a few ideas which are so intuitively right, so devoid of adverse effects, and so inexpensive, that it does seem reasonable to try them while waiting for the evidence to accumulate.

We have tried to set out as much information on the subject as possible, in what we hope is a reasonably ‘jargon-free’ manner. Some of the medical terminology is unavoidable and we have tried to provide explanations where necessary. We have devoted the majority of the book to high blood pressure but have included information on normal blood pressure and also low blood pressure. If anyone picks up this book and finds the subject becomes more interesting than they anticipated, writing it will have been worth while.

Nurses, who are undertaking an increasing proportion of the detection and supervision of hypertensive subjects in primary care, will also find the book informative, and we believe that medical students and pharmacists will find it a useful handbook.

Abbreviations

ACE	angiotensin-converting enzyme
ADH	antidiuretic hormone
AE	adverse effects
ANS	autonomic nervous system
BP	blood pressure
CHD	coronary heart disease
CNS	central nervous system
CO	cardiac output
CVS	cardiovascular system
DBP	diastolic blood pressure
ECG	electrocardiogram
GP	general practitioner
HDL	high-density lipoprotein (cholesterol)
HRT	hormone replacement therapy
ISH	isolated systolic hypertension
JNC	Joint National Committee. Sixth report on hypertension (USA) 1997
LA	left atrium
LDL	low-density lipoprotein (cholesterol)
LV	left ventricle
LVH	left ventricular hypertrophy
MABP	mean arterial pressure
MI	myocardial infarction
NHS	National Health Service
PR	peripheral resistance
RA	right atrium
RAS	renin–angiotensin system
RV	right ventricle
SBP	systolic blood pressure
SBPM	self blood pressure measurement
SV	stroke volume

Chapter One

The circulation and the blood pressure

In order to understand the blood pressure, its disorders and their treatment, it is necessary to have some knowledge of the blood circulation. Here we describe something of the **anatomy** (structure) and **physiology** (function) of the heart and blood vessels and their contribution to the blood pressure. We also discuss what the blood pressure actually is and what sort of level can be described as 'normal'.

How does the heart pump blood around the body?

Before describing the various disorders of the blood pressure, it seems logical to start with a brief account of the blood circulation (Figure 1.1).

In the average adult, the circulatory system contains approximately five litres of blood (eight imperial pints), and this is the **circulating blood volume**. At rest, the heart will pump almost this amount of blood around the body each minute. At this rate, the heart will pump out 7200 litres of blood per day – weighing 100 times more than the average human body. The blood pumped by the heart takes 250 ml of oxygen each minute from the lungs to the body tissues, all of which are completely dependent on this supply of oxygen.

Assuming that a person is resting, the heart will beat approximately 70 times each minute (the **heart rate**), although the rate is variable and depends on many factors other than the level of

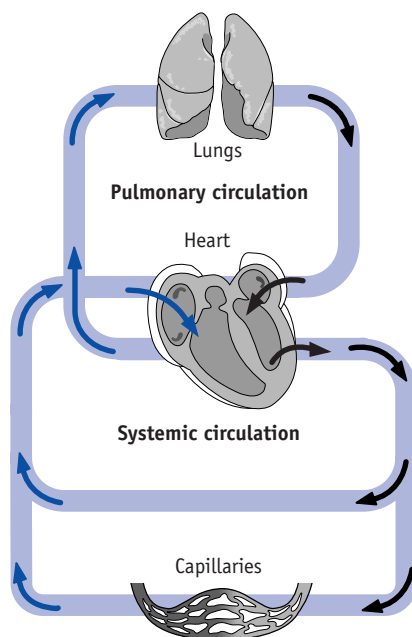



Figure 1.1 The circulation of the blood. Diagrammatic representation of the heart, lungs and circulation.

→ Oxygenated
→ Deoxygenated



The average human adult body contains about five litres of blood

physical activity. Therefore, each heartbeat propels about 70 ml of blood from the left side of the heart into the **aorta**, the great arterial motorway where the journey around the circulation begins. The amount of blood pumped into the aorta during each heartbeat is known as the **stroke volume**.


Where CO is cardiac output, SV is stroke volume, and HR is heart rate, it is easy to see that:

$$\text{CO} = \text{SV} \times \text{HR}$$

In the example above, the **cardiac output** would be 4900 mL per minute.

As Figure 1.1 indicates, there are effectively two separate circulations each fed by their own pump. Each pump corresponds to one side of the heart and has two chambers:

- ❖ the **atria** or receiving chambers
- ❖ the highly muscular **ventricles** or ejecting chambers.



The main artery that takes blood to the organs is called the aorta; the main veins that transport blood back to the heart are the superior and inferior vena cava

The right ventricle sends blood into the pulmonary circulation via the pulmonary arteries. These vessels take the blood to the lungs where it releases carbon dioxide and soaks up oxygen. The blood returns through the pulmonary veins to the left atrium, flows through a valve to the left ventricle and is then pumped through the aorta to the arteries. After delivering oxygen and other nutrients to the tissues, blood returns through the veins to the right atrium.

The direction of the bloodstream through the heart is secured by valves:

- ❖ One valve is situated between each atrium and ventricle to prevent the ventricle forcing blood backwards into the atrium.
- ❖ One valve is located at the outlet of each ventricle, so as the heart relaxes between beats, it does not refill with the blood it has just ejected.

The blood vessels

The aorta loops over in the chest in the shape of a question mark (Figure 1.2). Throughout its subsequent descent into the abdomen, numerous arteries branch off it. These arteries differ in size; if the aorta, measuring 2–3 cm in diameter and 50 cm in length, is the ‘motorway’, the large arteries are the ‘trunk roads’ which supply the major parts and organs of the body. On arrival at their destinations, the arteries divide repeatedly into smaller arteries and then into **arterioles**, which have a diameter of around 70 millionths of a metre (μm). Arterioles are therefore too small to be seen with the naked eye. The arterioles branch yet again to form

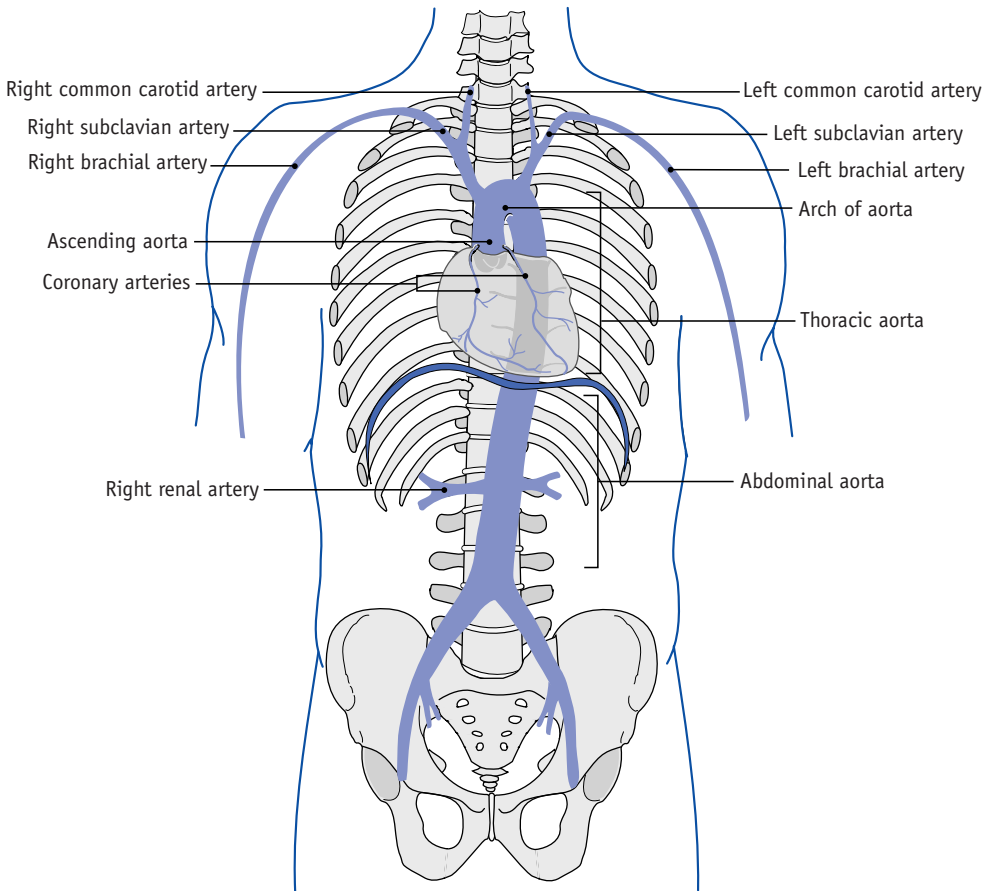


Figure 1.2 Arteries of the thorax and abdomen. The aorta and some of its branches in the thorax and abdomen.

the **capillaries** (10 μm or less in diameter). Capillaries are so narrow that red blood cells, which are 7 or 8 μm wide, are only just able to pass along them.

The capillaries are the vessels that are in the most intimate contact with body cells and the fluid which bathes them, and it is here that the vital functions of the microcirculation take place. To pursue the motorway analogy, these are the little pedestrian precincts in the centre of the city, where the miniscule day to day transactions involving the exchange of life's necessities are carried out.

The smallest blood vessels, the capillaries, are so narrow that red blood cells can only just flow through them

Collectively, all the capillaries are often referred to as the **capillary bed**. After the exchange of oxygen and nutrients for carbon dioxide and waste products has been completed, the blood needs to be returned to the heart so that it can:

- ❖ be sent to the lungs to dispose of the carbon dioxide which had accumulated in the tissues
- ❖ pick up new oxygen supplies.

Table 1.1: Vessels of the circulation

Type of vessel	Diameter (mm)	Number in parallel	Total area (cm ²)
Aorta	25	1	2.5
Arteries	2	600	5
Arterioles	0.07	50 million	40
Capillaries	0.005–0.01	1000 million	1,700
Venules	0.03	100 million	375
Great veins	13	2	10

Table 1.2: Location of blood at any instant in a resting individual

Structure	% blood
Pulmonary circulation	12
Heart	6
Arteries + arterioles	11
Capillaries	5
Veins & venules	66

Therefore, capillaries reunite with each other to form **venules**, and these gradually join up to form the great veins which take blood to the right atrium.

It has been estimated that the body of an adult may contain 60,000 miles of blood vessels. An approximate idea of the relative scale of the vessels

of the circulation is given in Table 1.1, and the amount of blood present in the various parts of the circulation at any given moment is detailed in Table 1.2.

Where does all the blood go to?

Table 1.3: Distribution of blood flow at rest

Organ	Blood flow (litres per min)
Brain	0.75
Heart muscle	0.25
Other muscles	1.2
Skin	0.5
Kidneys	1.1
Gut	1.4

The supply of blood to a region or organ is called the **perfusion** of that region or organ. Table 1.3 shows how much of the output from the heart is allocated to the perfusion of different organs in the resting state. The distribution of the blood can be adjusted to meet special requirements, such as during exercise or a cold environment (see Chapter 2, p18).

What makes the blood return to the heart?

The heart muscle pumps the blood out through the ‘arterial tree’ to the tissues, but the force of its contractions is progressively weakened as the blood moves down the arterioles and into the capillary bed. By the time the blood has reached the venules, the pumping force from the heart is hardly felt. There are three main forces that help the venous circulation:

- ❖ gravity – in the case of the head and neck, and the arms when elevated
- ❖ the negative pressure generated within the thorax every time we inhale, which sucks the blood into the heart
- ❖ the pumping action of the leg muscles – the muscles squeeze the leg veins and the blood within the veins can only proceed in an upwards direction due to a system of internal valves.

Guardsmen and soldiers are liable to faint when they stand motionless because blood tends to pool in the lower extremities, starving the heart and therefore the brain of blood and oxygen. Attempting to make imperceptible leg movements may prevent the soldier from actually passing out.

Structure of blood vessels

The different types of blood vessel have several striking differences in their structure (Figure 1.3). This is because they have very different functions. Both arteries and veins consist of:

- ❖ two layers of muscle lined on the inside by a single layer of smooth **endothelial cells**
- ❖ variable quantities of collagen, a relatively rigid protein
- ❖ variable quantities of elastin, a highly stretchable protein.

There is more collagen in the walls of the arteries and arterioles and more elastin in the walls of veins and venules. Capillaries lack the layers of muscle and are constructed only of endothelium. Fluid, rich in essential nutrients and the waste products of metabolism, can pass to-and-fro through the capillary walls.

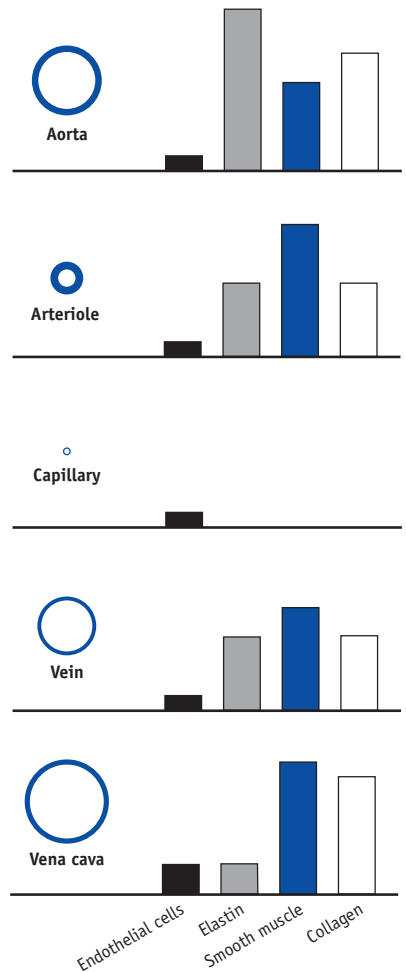


Figure 1.3 Blood vessel walls. The structure of the different blood vessel walls varies in thickness and composition.

The walls of the arteries contain more muscle than the veins – after trauma, contraction of this muscle helps to staunch bleeding

The arteries are more muscular and much thicker-walled than the veins. This is important for the body to achieve damage limitation following trauma. Being directly downstream of the heart, arterial

bleeding is often torrential. Muscular contraction leading to constriction of the artery (**vasoconstriction**) is the first line of defence and reduces the flow before the blood-clotting mechanism has time to act. Vasoconstriction is more effective in younger subjects as older people have more collagen in their arterial walls, which renders them stiffer and less able to constrict.

What happens between heartbeats?

The contraction of the ventricles is called **systole** and the interval between contractions when the ventricles are refilling with blood is called **diastole**. During

Systole is the contraction of the heart and diastole is the interval between heartbeats

systole, the aorta and the large arteries do stretch, but only to a limited extent. This requires a considerable amount of energy, which is stored in the vessel walls. During

diastole, this energy is released in the form of elastic recoil. This squeezing effect helps to maintain the blood pressure and the flow of blood through the capillaries.

What is the peripheral resistance?

Arterioles are muscular and many times more numerous than the arteries. However, their number is low compared to that of the capillaries and venules (Table 1.1). When the muscle contracts, the arteriole constricts; when this process is occurring simultaneously in a large proportion of the arterioles, their total cross

section becomes greatly reduced and the resistance to the forward flow of blood is increased. For this reason, the arterioles are sometimes classified as **resistance vessels**.

When the muscle in the arteriole wall relaxes, the vessel dilates (**vasodilatation**). Constriction or dilatation of the arterioles supplying a particular

Table 1.4: How different vessels contribute to the peripheral resistance

Blood vessels	% of peripheral resistance
Arterioles	40
Arteries	25
Capillaries	20
Venous system	15

tissue will determine what proportion of the cardiac output reaches that tissue's capillaries. The total of all the resistances of the arterioles supplying the **systemic circulation** (as opposed to pulmonary circulation) is the major constituent of the total **peripheral resistance** (Table 1.4). Peripheral resistance is the resistance to the passage of blood through the systemic circulation.

What about the resistance in the veins and venules?

The veins and venules contain a great deal of elastin in their comparatively thin walls, which makes them very stretchy, or **compliant**. At any given moment, they contain a very high proportion of the total circulating blood volume. The veins and venules are capable of holding large, variable quantities of blood without the pressure within them fluctuating dramatically. They are therefore often referred to as **capacitance vessels**.

The veins and venules are stretchy due to the protein elastin in their walls – these vessels can hold very variable quantities of blood



What has all this to do with the blood pressure?

When we talk about the blood pressure (BP), we are speaking of:

- ❖ the pressure in the systemic rather than the pulmonary circulation
- ❖ the measurable pressure within the large arteries and not in the veins
- ❖ the pressure in a large, readily accessible artery in the arm – although this may seem quite far from the heart, the pressure has not yet decreased significantly. By the time blood reaches the capillaries, the pressure is very much lower.

When giving a BP reading, two figures are always given – the systolic (SBP) and the diastolic (DBP). The reading is therefore the pressures during these two phases of the heart's action. The difference between the SBP and the DBP is the **pulse pressure** and the **mean arterial blood pressure** (MABP) is taken to be the DBP plus one-third of the pulse pressure.

Normally, blood pressure is measured in the brachial artery in the arm



The BP depends upon three factors:

- ❖ the blood volume (see Chapter 2, p15)
- ❖ the cardiac output (CO)
- ❖ the peripheral resistance (PR).

For any given blood volume, the latter two are expressed by the mathematical formula:

$$\text{MABP} = \text{CO} \times \text{PR}$$

Put very simply:

- ❖ the higher the cardiac output (hence heart rate and stroke volume) and peripheral resistance, the higher the BP
- ❖ the lower the cardiac output and peripheral resistance, the lower the BP.



Increasing the peripheral resistance by constriction of the arterioles leads to a rise in blood pressure; dilatation of the arterioles lowers the blood pressure

The way the BP is usually expressed looks like a fraction, but is really just the SBP above the line and the DBP below the line.

What is the ‘normal’ BP?

There is really no such thing as a ‘normal’ BP – it varies enormously, not just between individuals, but in the same individual from time to time. The figure that is generally quoted to medical students as being ‘normal’ is 120/80 mmHg (measured in mm of mercury).

For those concerned for the welfare of their domestic pets, it may be added that measuring the BP in other animals is, for obvious reasons, a much less

Table 1.5: ‘Normal’ blood pressure levels in animals

Animal	‘Normal’ BP (mmHg)	Additional information
Horse	112/77	High BP may cause nosebleeds
Cattle	100–140/50–85	
Dog	112–148/56–87	Hypertension causes heart failure, stroke or kidney failure
Cat	104–171/73–123	
Mouse	81	(MABP)
Rat	130/91	Hypertension causes stroke
Frog	30/20	
Salmon	30/22	
Octopus	44/22	
Lobster	13/1	At rest
	27/13	When active
Giraffe	280/80	

routine part of a medical examination than it is in humans. ‘Normal’ levels in other species are less well documented. Table 1.5 shows some figures for other animals.

Investigations have found that continuous emotional arousal of a solitary animal, ie lone family pet, will eventually lead to a measure of chronic hypertension.

Age and the blood pressure

Older people have more collagen in their arterial walls than younger people. This makes the arteries stiffer and less elastic. The arteries of a 70-year-old are said to be only 50% as compliant ('stretchy') as those of a young adult. A decrease in arterial distensibility ('elasticity') causes the SBP to rise. In the developed world it is exceedingly common to find that the BP, especially the SBP, rises with age (Table 1.6).

The figures in Table 1.6 are often quoted, but they are also hotly disputed. It all depends on what you mean by 'normal' – the word is used in two quite different senses:

- ❖ It can be taken to mean what is common in the UK (or France or the USA or wherever the speaker happens to reside).
- ❖ It can be used to denote someone who is healthy and free from disease.

Table 1.6 is only valid in the former sense. But, using the latter definition of normality, the figures from age 35 onwards are distinctly worrying.

Other factors affecting the BP

The cardiac output influences the BP and the stroke volume influences the cardiac output. An increase in the heart rate is the main factor in the rise in cardiac output that occurs during exercise (see Chapter 2, p17). Many day-to-day events influence the BP on a transient basis (see Chapter 7, p61).

There are some long-term situations in which the stroke volume is elevated, resulting in a fall in the DBP and possibly a slight rise in the SBP so that the pulse pressure is larger. These include:

- ❖ pregnancy
- ❖ very slow pulse (highly trained athletes or heart block, which is when conduction of the heartbeat from its natural pacemaker has been interrupted)
- ❖ severe anaemia
- ❖ high fever
- ❖ disease of the aortic valve (between the left ventricle and the aorta).

In the developed world, blood pressure tends to rise with age



Table 1.6: 'Normal' BPs at different ages

Age (years)	Blood pressure (mmHg)
5–10	90/60
13–18	105/65
22–25	120/80
35	130/85
45	140/90
55	145/95
65	150/95
75	150/100

Conclusion

Much of the information in this chapter will initially seem to have little relevance to most of the readers of this book, many of whom will have hypertension. But one of the core themes of the book is that hypertension is like diabetes: if you have the condition, you are well advised to study it, to become absorbed in it, to become an authority on it. An understanding of the physiology of the BP is a prerequisite to understanding the control of the BP and the mechanisms by which the various drugs exert their beneficial (and adverse) effects.

Summary points

- ❖ We have described the circulation of the blood: the blood pressure measured is the pressure prevailing in the arteries and is usually measured in the main artery in the arm.
- ❖ The pressure during cardiac contractions (SBP) is higher than that during the phase of relaxation (DBP); these two figures are quoted in a blood pressure reading.
- ❖ With any given blood volume, the mean arterial blood pressure is the product of the stroke volume and the peripheral resistance.
- ❖ The muscular arterioles (the 'resistance vessels') are responsible for the peripheral resistance.
- ❖ The frequently quoted level for a 'normal' blood pressure is 120/80 mmHg, but higher levels are extremely prevalent, especially in older people.
- ❖ People who have hypertension are advised to learn about the condition and understand the physiology and management of this serious disorder.