

## THE CLINICAL MANAGEMENT OF HYPERTENSION\*\*

DR. WILLIAM THOMSON WALKER, M.B.E., M.A., M.D., F.F.HOM.

Some twenty-two years ago I presented a paper<sup>1</sup> here on the clinical management of hypertension which took the form of an analysis of the results of one hundred cases treated personally. That is not my intention today, though I do believe that to be valid a series is required (rather than anecdotal cases). Orthodox treatment then consisted of phenobarbitone and potassium thiocyanate, the first futile, the second frankly dangerous and soon abandoned. As I hope to indicate, the general profession is still in disarray on this subject, the history of the intervening years being of rejection of one drug after another because of the unacceptable penalties which attach to them if they are persisted with.

Although it is generally conceded that we are far from knowing all the underlying causes of hypertension, many are known and must be looked for in each new hospital case.

The accepted criterion of hypertension seems to have changed considerably, moving upwards recently. It is usually given as a systolic pressure above 160 and a diastolic above 90 mmHg.<sup>2</sup> but personally I would only give prophylactic advice under systolic 170 and diastolic 105.

Table I lists the main causes of hypertension<sup>3</sup> which I shall not consider

TABLE I  
CAUSES OF HYPERTENSION

Vascular	Renal	Endocrine	Iatrogenic
Atherosclerosis	Nephritis	Phaeochromocytoma	Irradiation of kidneys
Polyarteritis	Pyelonephritis	Cushing's Disease	Steroids
Renal artery stenosis*	Hydronephrosis*	Aldosteronism	Analgesic nephropathy <sup>4</sup> (largely irreversible)
Essential	Polycystic kidneys*	Acromegaly	Diuretics, prolonged use <sup>1</sup>
Coarctation of aorta	Amyloid	Menopause	Oral contraceptives
	Gout	Toxaemia of pregnancy	Monoamine oxidase inhibitors c. cheese, or combined with pressor drugs
	Wilm's tumour (nephroblastoma)	Diabetes	adrenalin, noradrenalin <sup>5</sup> +tricyclic drugs 4-8 times
		Ovarian dysgenesis	

Conditions marked \* are amenable to surgery.

\*\*A paper read to the Post-Graduate Course in Homoeopathy on 21 February 1973.

in detail. I merely draw your attention to column 4—an ever-increasing cause resulting directly from misguided attempts at cure. The Hippocratic Oath enjoins the physician "When he can do no good let him do no harm". This can hardly be said today of the general treatment of hypertension.

In order to sort out the diseases listed in Table I it is necessary to perform the routine tests listed in Table II. We will find that we have positive results in only 10 per cent of cases. All others in our present ignorance we call essential hypertension. Only 1 per cent of all cases go on to malignant hypertension.<sup>10</sup>

TABLE II  
ROUTINE INVESTIGATIONS FOR CAUSE OF HYPERTENSION

Urine	Microscopy Culture Urinary keto-steroids
Blood	Full count ESR Coomb's test Blood urea Creatinine Cholesterol Serum electrolytes (Serum renin, immuno-globulin G (IgG index of vascular change))
Electrocardiography	T wave inversions S-T elevations Evidence of left ventricular hypertrophy or strain
Ophthalmoscopy	Retinopathy grades I—IV
Radiological	Heart Lungs Intravenous pyelogram (Rarely, renal arteriogram, pre-sacral oxygen insufflation)
Routine palpation of femoral pulses to eliminate coarctation.	

A great majority of cases of essential hypertension have no symptoms whatever and are usually picked up casually on insurance examination. A few have congestive headaches, may complain of transient giddiness or undue weariness and we find ourselves treating them mainly in the hope of avoiding the complications of cerebral or cardiac infarction, or left heart failure. Objective evidence of arteriolar change is seen best in the retina and the severity readily graded: (1) Minimal change; (2) Moderate changes, silver wiring and arterio-venous nipping (hyaline changes); (3) Severe changes with haemorrhages and/or exudates; (4) Papilloedema (vasculitis).

The greater the impedance to left ventricular ejection the greater is the heart load, oxygen requirement and hypertrophy. In turn this state accelerates atherogenesis, giving impetus to the vicious circle. Fortunately, in

spite of the pessimism of the insurance companies, the condition is not necessarily progressive, as one lives and learns by experience.

Some of the leaders of our profession have recently made statements indicating their pessimism and dissatisfaction with present-day treatment of hypertension.

Professor A. G. W. Whitfield (Birmingham) said<sup>11</sup> "Hypotensors are not often helpful in patients over 60 years". Professor Fergusson Anderson (Professor of Geriatrics, Glasgow), goes further and says "The only benefit to be obtained from hypotensors is when they are stopped". With these opinions in mind I ask you to glance at Table III which indicates the reasons for their pessimism.

TABLE III  
COMMONLY USED HYPOTENSORS

Drug	Harmful effects
A. Ganglion Blockers	
1. Pentolinium (Ansolysen)	These sympathetic and para-sympathetic blockers are now considered too dangerous —tremor, weakness, collapse, impotence, paralysis of accommodation
2. Mecamylamine (Inversine)	
3. Pempidine (Perolysen)	
B. Adrenergic Blockers	
1. Guanethidine (Ismelin)	Diarrhoea, hypotension with exertion. Fatigue, drowsiness <sup>12</sup>
2. Alpha Methyl dopa (Aldomet)	20 per cent. Coomb's test positive, if persists, goes on to haemolytic anaemia, leucopenia Liver damage
3. Bethanidolol (Esbatal) (currently approved)	Impotence, hypotensive episodes Dangerous with amphetamines Diarrhoea, depression
C. Beta Blockers	
1. Propranolol (Inderal)	While all these may be useful, they depress the myocardium and may produce left heart failure. <sup>13</sup> All harmful in asthma <sup>13</sup> Practolol: macular rash with scaling <sup>14</sup>
2. Practolol (Eraldin)	
3. Oxprenolol (Trasicor)	
D. Reserpine (Serpasil) Prevents synthesis of noradrenaline.	Powerful mental depressant Nasal congestion May precipitate heart failure
E. Clonidine (Catapres) In favour at present 200 micrograms daily	Contraindicated in manic-depressives Mental changes reported in 4 out of 28 recent cases <sup>14</sup> Cold extremities

TABLE III—(Contd.)

Drug	Harmful effects
F. Diuretics	
1. Thiazides (12)	Effective initially Potassium loss and magnesium loss <sup>17</sup>
2. Frusemide (Lasix)	Oedema ultimately resistant, due to intracellular potassium depletion I.V. gives transient cardiac arrhythmias
3. Ethacrynic acid	Electrolyte depletion <sup>18</sup> Hyperuricaemia Thrombocytopenia Skin rashes Deafness (in renal failure)
4. Spironolactone (Aldactone-A)	Used in combination with thiazides. Gynaecomastia, hirsutism, mental confusion, impotence (in the male), irregular menses, mammary discomfort (in the female) <sup>19</sup>
5. Diazoxide I.V. (currently approved)	Not effective orally For emergency, 5 mg/kg undiluted Skin eruptions and thrombocytopenia Raises blood sugar level

All diuretics eventually raise plasma renin activity<sup>20</sup>.

Lest you should think I am over-painting the picture, just listen to some extracts from this recent discharge letter from a leading London teaching hospital:

"Dear Doctors,

*Re: Mr. X.Y.—aet 66*

"This man has just moved to your area.

"He has been attending this hospital since June 1967 for treatment of hypertension... (clinical details follow)..."

"Treatment for moderate uncomplicated essential hypertension was started using Aldomet. Control remained good until March 1969 when he was found to have haemolytic anaemia as a result of Aldomet. He was therefore changed to bethanidine. This caused quite marked postural hypotension even without good control of the rising blood pressure. (It in fact caused such a serious collapse that the patient had to be revived, though the letter does not say this.) In November 1970 he was therefore changed to propranolol. This put him into left ventricular failure so had to be abandoned and in August 1971 he was put on clonidine (Catapres) on which he remains..."

"He has had an unfortunate history of therapeutic misadventure during

the course of treatment for this hypertension. However, he is remaining stable on his present treatment.

Yours sincerely . . ."

(It may well be that the unfortunate man may not be finished with his troubles and yet develop the manic-depressive symptoms reported in a recent series in 15 per cent of the cases where clonidine has been used.)

Before turning to homoeopathic treatment one should remember that mere reduction of blood pressure is not necessarily beneficial and may actually be disastrous. In the presence of carotid or vertebro-basilar disease cerebral infarction may result and co-existence of coronary insufficiency may lead to myocardial infarction.

Also, reduction of blood pressure may reduce renal artery flow and increase blood urea.<sup>21</sup> Nor does reduction prevent development of coronary occlusion.

In treating patients homoeopathically one must also consider and as far as possible deal with the factors which predispose to hypertension. For brevity these are listed in Table IV.

TABLE IV  
FACTORS WHICH PREDISPOSE TO HYPERTENSION

1. Heredity	Family History: Race: Sex Genetic differences affect Fibrinolytic Activity
2. Cortical drive	"Get on at any cost types" General degree of stress to which exposed necessarily
3. Emotional	Reaction to prolonged anxiety Hyper-reactive pressor responses which derange the baroreceptor reflexes with repetitive stimulation
4. Over-weight	From excessive calorie intake Excessive Sodium intake or retention
5. Smoking tobacco	Raises the free fatty acid level <sup>22</sup>
6. Lack of exercise	
7. Soft water and low residue diet <sup>23</sup>	
8. Common accompaniments	Baldness Early Arcus Senilis Xanthelasmata

It has to be admitted that for hypertension Homoeopathy has no specific remedy any more than it has for any other condition, but even more does it apply that a constitutional remedy must be sought in the usual way by re-per-torization. However, as one would expect, the polychrests come up the most frequently, in my experience Lycopodium heads the list (42%). Sulphur

comes a good second (22%); followed by Nat. mur. (14%), Lachesis (12%), Phosphorus (4%), Sepia (4%), and Pulsatilla (2%).

Others have suggested helpful remedies:

Dr. Hamish Boyd<sup>22</sup> } Carbo sulph.  
                              } Ars. arsenite (to Sulphur types with giddiness);

Dr. Campbell, Conium, especially in the elderly;

Dr. Fergus Stewart and the Glasgow school generally used "C.G.P.", Crataegus, Glonoin and Passiflora in a mixture;

H. MacNeill suggests Lachesis.

At one time my own favourite remedy was Crataegus  $\phi$  which appeared to serve me well combined with the constitutional remedy, but for the last ten years my allegiance has moved to Lycopus lx m vii t.d.s. Probably milder cases do better with Crataegus, and more severe cases with Lycopus. Of the polychrests, I find that I need Lycopodium twice as frequently as any other, in potencies from 200 to 10M. I am in good company in this, for Hughes, Dunham, Clarke, Harold Fergie-Woods and Frank Bodman have made similar comments.

Irritability, morning giddiness, acid dyspepsias are the marks of the harassed business or professional man or woman who needs Lycopodium. Such a one was R.M. aet. 56, a lawyer, who complained of tiredness, occipital headache, rush of blood to the head with confusion, dyspepsia and insomnia. Blood pressure was initially 240/115 mmHg. There was slight left heart hypertrophy and a few ventricular extra-systoles showing on ECG. I.V.P. revealed bilateral hydronephrotic kidneys, though the urine showed no RBCs or casts and merely a trace of albumin. Blood urea was 35 mg/100 ml. The retinae showed silver wiring, but no haemorrhage or exudates. 4-8 pm <. Heat <. Warm food >. Uncovering >. Although he continued in a responsible post, his blood pressure fell to 180/100 over a period of three months. When he asked "What was that remarkable medicine you gave me?" the answer was Lycopodium, in ascending potencies from 200-1M, with, of course, a strict low sodium diet to which he stuck rigidly.

The best set of indications for Lycopus I know is that of Dr. Borland:<sup>23</sup> "Patients tend to be pale rather than cyanotic, and they are always restless. The outstanding symptom... is a horrible tumultuous sensation in the cardiac region... accompanied by intense throbbing extending up into the neck and into the head... There is a marked tendency to cough... Distress is increased by turning on the right side—(in contrast to the snake poisons). Lycopus patients have a dislike of food, even the smell of food.

"The heart is just beginning to dilate in early failure and the pulse is tending to become a little irregular."

In my view failures with the use of Lycopus stem from not continuing the remedy for long enough... several months at least... it is very slow-acting. Here is the outline of such a case: Mrs. W. M., aet 63, a housewife, who came with the story that 6 months ago she had been given 6 months to

live. She felt dreadfully ill and "What could I do to save her?". Blood pressure was 260/140, cardiomegaly, cardiac asthma, anginal attacks, orthopnoea, rough aortic systolic bruit. (Previously upset by Aldomet, Hygroton and Inderal.) Heart was tumultuous with violent palpitation. Claudication and cramp. Pipe-stem arteries and Grade II retinopathy. Occasional syncopal attacks. Sense of strangling in the chest. ECG showed severe I.V.P. Blood urea 50 mg/100 ml. Urine: Trace of albumin. No RBCs or casts. I.V.P. negative.

I gave her *Lycopus* 1x m vii t.d.s. with *Apocynum* 1x m vii t.d.s. and *Aurum mur.* 30. On her next visit she appeared the same to me and I gave a grave prognosis to her husband, but she insisted that she felt much better, and that her anginal attacks were much less. I gave her *Secale* 6 instead of the *Apocynum* and at the next monthly visit the claudication was improving and the blood pressure down to 200/120. She had troublesome giddiness which *Glonoin* 30 helped and epistaxis with sour eructations for which *Ferrum phos.* 3x was given, *Lycopus* 1x being continued throughout. Now, 8 months from her first visit, although her blood pressure is still 195/115 (180/110 yesterday and I believe she would feel worse if it were to fall lower), she is still gradually improving and full of confidence that her life will be further prolonged. She had, of course, a salt-free diet. I saw her yesterday, improvement continues. She had shovelled some snow! I believe her case to be, not a malignant hypertension but essential hypertension over many years, where atherosclerosis seriously involved heart and arteries. She feels a little resentful that this should have happened to her in spite of being a vegetarian for many years!

Obviously one must use intercurrent remedies for acute episodes and related complaints.

Of these, *headache* of a congestive type is the most common, although some very severe cases have no headache and there may be associated *giddiness* related to changes of position, especially in the portly, choleric types. For these symptoms *Glonoin* is most likely to be helpful, but *China* or *Coca* will help the less florid types.

*Indigestion* is a very common accompaniment of all heart disorders, and whether mere flatulent dyspepsia or hiatus hernia, each affects the other, even by direct mechanical pressure, and so become important. Our old friends *Carbo veg.* 3x-6, and *Lycopodium* 6-1M, the former to the person who is sluggish, fat and lazy with aversion to milk, meat and fat; the latter to the thin, withered sensitive intellectual who gets burning eructations and likes everything warm.

Persistent peripheral resistance is likely to produce left ventricular failure, hypertrophy being eventually replaced by dilatation and a failing myocardium. For this situation the best remedies I find are *Crataegus*  $\phi$  m v-xx, especially where there is *high diastolic* and *low systolic*. This is excellent in support of the failing myocardium (but only lowest potencies), or *Adonis vernalis* 1x if there is superadded congestive cardiac failure (which accounts

for more than 50 per cent. of deaths), or *Iberis* 1x where there is palpitation, vertigo and choking or cardiac asthma, or *Veratrum alb.* 30 if there is cold clammy collapse.

*Cardiac asthma* may necessitate the short-term use of sedation, diuretics and aminophyllin. But, of course, in addition to these remedies one is likely to be using in this situation *Arsen. alb.* 1M-10M, taken every 15-30 minutes where there is mental distress, extreme fear, burning in chest, gets out of bed around 2-3 a.m., or *Arnica* especially if the patient, in spite of his parlous state, insists that he is 'feeling fine'. (*Ars. alb.*, *Apis* and *Apocynum* are too slow to dehydrate in acute congestive cardiac failure). *Spongia* is often a help here, or, when peripheral cyanosis is an accompaniment, try *Antimon. tart.*

This state is often accompanied by tumultuous palpitation for which one would consider *Belladonna*, *Spigelia*, *Kalmia*, or *Naja*, or *Lycopus*, according to the detailed symptoms.

If there is cold, sweaty collapse and pallor, use *Carbo veg.* 10M.

Involvement of the renal arteries carries a bad prognosis and treatment is very difficult. For one thing reduction of blood pressure actually then reduces renal blood flow and puts up the blood urea. The remedy one would choose here is *Eel Serum* which is said to be more effective if there is hypertension and oliguria without oedema; alternatives would be *Arsenicum alb.*, or *Apis*.

*Threatened apoplexy* gives most nearly the picture of *Veratrum viride* but *Gelsemium*, *Baptisia*, *Belladonna* and *Aconite* may also prove helpful, and *Arnica*, *Opium* or *Causticum* used immediately after the event.

*Malignant hypertension* fortunately occurs only in 1 per cent. of all cases progressing rapidly from another, simpler form, mainly in younger people (not over 60). They develop dyspnoea, papilloedema, headache and albuminuria early. If there is no renal failure survival may be as long as six years with vigorous treatment. Apart from renal dialysis units, where they tend to end up, one physician sees few and can quote only individual instances, but they make sorry reading, progressing usually to renal failure, though a minority succumb first to cerebral haemorrhage or heart failure. In such cases the plasma renin activity is increased ten-fold. Even after bilateral nephrectomy there is still some renin activity and this is thought to be splanchnic in origin.<sup>26</sup>

Prolonged effective diuretics are now known to increase plasma renin activity and increase blood pressure.

In the past five years I have seen only one case of malignant hypertension and that for a brief fourteen days. This was a headmaster, aged 40, who told me that he had lost control of his staff and of his school, and this had caused his illness. Whether this was the true cause I don't know. His blood urea was soaring to 200 mg. He asserted that the drugs he had been given were killing him, hence his demand to come into the homoeopathic hospital, alas too late, though whether anyone could have helped him by any means is very doubtful. I gave him *Lycopodium* 1M 6 hourly—*Eel Serum* 200 iv—



Crataegus  $\phi$  m x t.d.s., and Veratrum viride IM, all to no avail.

Most cases of essential hypertension do very well and some of the most severe survive to a ripe old age, even with 'pipe-stem' arteries, perhaps because the blood pressure falls as the ischaemic process involves the myocardium and that danger is naturally compensated.

One old lady patient of mine, aged 80, was actually told by a Consultant Physician that her hypertension would kill her within two years. She lived to be 85 and it was the physician who died prematurely.

In *primary aldosteronism* (Conn's Syndrome; 1 per cent. of all cases) where the aldosterone level is high (renin activity is low), there is some hypertension, and Conn thought that it accounted for up to 25 per cent. of cases of so-called essential hypertension.<sup>27</sup> Spironolactone is used for this condition but is not always effective, and as I have mentioned, has its considerable side effects (gynacomastia and impotence in men, irregular menses and mammary discomfort in women).

Some quite seriously high hypertensives, especially women, go on remarkably well for many years, making both prognosis very difficult and also an estimate as to the success of treatment nearly impossible. The natural variables of personality and heredity, combined with the variables of remedies and potencies and dietetic restriction used, make it well-nigh impossible to prove the value of any one remedy.

The main thing is that over-all we believe that with our method we can produce much better results and certainly do no harm.

#### REFERENCES

1. Walker, W. T.: 'Hyperpiesia', *The British Homoeopathic Journal* (1951) 41, 50.
2. Davies, I. J. T.: 'Criteria of Hypertension', *Post-Graduate Medicine*. London: Lloyd-Luke (1969).
3. MacNeill, A. D.: 'Hypertension and Its Treatment', *The British Homoeopathic Journal* (1964) 53, 278 (After McMichael).
4. Kingsley, D. P. E., Goldberg, B., Abrahams, C. et al: 'Analgesic Nephropathy', *The British Medical Journal* (1972) 4, 656-659.
5. Leading Article: 'New Thoughts on Essential Hypertension', *The British Medical Journal* (1972) 2, 121.
6. Banks, A. J.: 'Interactions Between Sympathomimetic Amines and Antidepressant Agents in Man', *The British Medical Journal* (1973) 1, 311-315.
7. Brown, J. J., Fraser, R., Lever, A. F. et al: (of M.R.C. Blood Pressure Unit, Western Infirmary, Glasgow) 'Hypertension with Aldosterone Excess', *The British Medical Journal* (1972) 2, 391-396.
8. Ebinger, A., Doyle, A. E.: 'Raised IgG Levels in Hypertension', *The British Medical Journal* (1970) 2, 146-148.
9. Hamer, J., Shonebourne, E., Fleming, J.: 'Electrocardiographic Evidence of Hypertension', *The British Medical Journal* (1969) 1, 81.
10. Owen, S. G., Stretton, T. B., Vallence-Owen, J.: *Essentials of Cardiology*, 2nd ed., Ch. 21. London: Lloyd-Luke (1968).
11. Whitfield, A. G. W.: 'Iatrogenic Misadventure', *The British Medical Journal* (1972) 1, 733.
12. Davies, I. J. T.: 'Criteria of Hypertension', *Postgraduate Medicine*, 45-50. London: Lloyd-luke (1969).

13. Zacharias, F. J., Cowen, K. J.: 'Controlled Trial of Propranolol in Hypertension', *The British Medical Journal* (1970) 1, 471-474.
14. Leading Article: 'Beta-Adrenergic Blocking Drugs', *The British Medical Journal* (1971) 1, 243.
15. Gaddie, J., Skinner, C. (Department of Medicine, The University of Aberdeen) in letter to *The British Medical Journal* (1972) 1, 749.
16. Macdougall, A. I. *et al*: 'Treatment of Hypertension with Clonidine', *The British Medical Journal* (1970) 3, 440-442.
17. Lim, P., Jacobs, E.: 'Deficiency of Magnesium in Patients on Long Term Diuretics', *The British Medical Journal* (1972) 3, 620.
18. Horrobin, D., Gunn, A.: 'Ethacrynic Acid', *The International Handbook of Medical Science*, Section B 190, Oxford, M.T.P.
19. Horrobin, D., Gunn, A.: 'Spironolactone', *The International Handbook of Medical Science* (1972) 2nd ed., Section B—192, Oxford, M.T.P.
20. Leading Article: 'New Thoughts on Essential Hypertension', *The British Medical Journal* (1973) 2, 121.
21. Leading Article: 'Predicting Coronary Artery Disease', *The British Medical Journal* (1972) 1, 3.
22. Burkitt, R.: 'Cholesterol Metabolism and Coronary Heart Disease', *The British Medical Journal* (1973) 1, 277.
23. Whitfield, A. G. W.: 'Iatrogenic Misadventure', *The British Medical Journal* (1970) 1, 733.
24. Boyd, H.: 'Use of Carbo. Sulph.', *The British Homoeopathic Journal* (1964) 53, 284.
25. Borland, D. M.: 'Lycopus in Heart Disease', *The British Homoeopathic Journal* (1948) 38, 3.
26. Barnardo, D. E.: Source of Extra-renal Renin in letter to *The British Medical Journal* (1973) 1, 297.
27. Leading Article: 'Conn's Syndrome', *The British Medical Journal* (1970) 1, 769.

—*The British Homoeopathic Journal*, January 1974

---