The effects of biofeedback for the treatment of essential hypertension: a systematic review

J Greenhalgh, R Dickson and Y Dundar
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The effects of biofeedback for the treatment of essential hypertension: a systematic review

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NIHR Health Technology Assessment programme

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Abstract

The effects of biofeedback for the treatment of essential hypertension: a systematic review

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*Corresponding author

Objectives: To assess the evidence for the long-term effectiveness of biofeedback for the treatment of essential hypertension in adults and to model any clinical benefits.

Data sources: Bibliographic databases including the Cochrane Library, EMBASE, MEDLINE, ISI Web of Knowledge/Web of Science, ISI Web of Knowledge/ISI Proceedings, the Cochrane Library 2007, CINAHL, AMED and PsycINFO were searched up to May 2007.

Review methods: A systematic review following accepted guidelines was conducted. Randomised controlled trials (RCTs) that compared biofeedback procedures with antihypertensive medication, placebo (sham biofeedback treatment), no intervention or other behavioural treatments were included. The outcome measure was change in blood pressure.

Results: A total of 927 non-duplicate references were identified by the search strategy and subsequently screened for inclusion in the review. From these, 41 publications (including three abstracts) reporting 36 RCTs with a total population of 1660 treated patients met the inclusion criteria of the review. Twenty-one trials employed biofeedback treatment with no adjunctive therapy and 15 trials used biofeedback treatment alongside another treatment. The majority of trials were small with no post-treatment follow-up or follow-up of less than 6 months. The poor quality of the trials, differences in interventions and inconsistencies in the measurement of outcomes meant that it was inappropriate to pool data across studies. A narrative summary of the data based on trial author conclusions is presented. No studies reported long-term (> 12 months) follow-up of patients. Data were grouped first by treatment type and then by comparator. Trial results were variable and conflicting, demonstrating no consistent benefits of biofeedback in relation to moderation of hypertension. The lack of evidence of clinical effectiveness negated the need to conduct an economic analysis.

Conclusions: No evidence was found that consistently demonstrated the effectiveness of any particular biofeedback treatment in the control of essential hypertension when compared with pharmacotherapy, placebo (sham biofeedback treatment), no intervention or other behavioural treatments. Given the current standards for the treatment of hypertension, further research is likely to be considered only as an adjunct to pharmacological interventions.
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List of abbreviations

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<th>AAPB</th>
<th>American Association for Applied Physiology and Biofeedback</th>
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<tr>
<td>ABPM</td>
<td>ambulatory blood pressure monitor</td>
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<tr>
<td>ACE</td>
<td>angiotensin-converting enzyme</td>
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<td>ASH</td>
<td>American Society for Hypertension</td>
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<tr>
<td>BFE</td>
<td>Biofeedback Foundation of Europe</td>
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<td>BHS</td>
<td>British Hypertension Society</td>
</tr>
<tr>
<td>CCB(s)</td>
<td>calcium channel blocker(s)</td>
</tr>
<tr>
<td>CRD</td>
<td>Centre for Reviews and Dissemination</td>
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<tr>
<td>CVD</td>
<td>cardiovascular disease</td>
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<tr>
<td>DBP</td>
<td>diastolic blood pressure</td>
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<tr>
<td>EHS</td>
<td>European Society for Hypertension</td>
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<td>EMG</td>
<td>electromyographic</td>
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<td>GSR</td>
<td>galvanic skin response</td>
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<tr>
<td>HR</td>
<td>heart rate</td>
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<tr>
<td>HSE</td>
<td>Health Survey for England</td>
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<tr>
<td>ICHSC</td>
<td>Information Centre for Health and Social Care</td>
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<tr>
<td>IQM</td>
<td>inner quality management</td>
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<tr>
<td>ITT</td>
<td>intention to treat</td>
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<tr>
<td>NCCAM</td>
<td>National Centre for Complementary and Alternative Medicine</td>
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<tr>
<td>NICE</td>
<td>National Institute for Health and Clinical Excellence</td>
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<tr>
<td>QUOROM</td>
<td>quality of reporting of meta-analyses</td>
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<tr>
<td>RCT(s)</td>
<td>randomised controlled trial(s)</td>
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<tr>
<td>SBP</td>
<td>systolic blood pressure</td>
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<td>TBF</td>
<td>thermal biofeedback</td>
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<td>WHO</td>
<td>World Health Organization</td>
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All abbreviations that have been used in this report are listed here unless the abbreviation is well known (e.g. NHS), or it has been used only once, or it is a non-standard abbreviation used only in figures/tables/appendices, in which case the abbreviation is defined in the figure legend or in the notes at the end of the table.
Executive summary

Background

Hypertension is defined as persistently high blood pressure, with currently accepted thresholds in the UK at 140/90 mmHg. It is one of the most prevalent and powerful risk factors contributing to the development of cardiovascular disease (CVD) and one of the most important preventable causes of premature morbidity and mortality in developed and developing countries. The estimated lifetime risk of middle-aged men and women developing hypertension is 80–90%. The most common type of hypertension is essential hypertension, which has no known cause. Its estimated prevalence is 30.6%. Current treatment options include lifestyle changes and pharmacological agents.

Biofeedback is defined as a group of non-pharmacological therapeutic procedures that use electronic instruments to measure, process and provide information (feedback) to patients regarding their neuromuscular and autonomic nervous system activity. Patients have been taught these procedures in an attempt to control their blood pressure. If shown to be effective they could be used in the treatment of essential hypertension.

Objectives

The primary objective of this report was to assess the evidence for the long-term effectiveness of biofeedback procedures in treating adults with essential hypertension. Other objectives were to model any clinical benefits of biofeedback for the treatment of essential hypertension, provide an overview of currently used biofeedback equipment and offer recommendations for future research.

Methods

Two recent systematic reviews with meta-analyses were critically appraised and used as a basis for this updated systematic review, which compares biofeedback procedures with placebo (sham biofeedback treatment), no intervention or other behavioural treatments, as well as with antihypertensive medication.

Results

The two existing systematic reviews were judged to be of high quality although there is a question regarding the appropriateness of the pooling of data. Neither review considered any evidence for biofeedback treatment versus antihypertensive medication. The authors of the first review concluded that biofeedback was more effective than no intervention, but was only superior to sham or non-specific interventions when combined with a relaxation technique. The second systematic review indicated that both biofeedback and active control treatments (relaxation training, cognitive therapy and home monitoring) reduced systolic blood pressure (SBP) and diastolic blood pressure (DBP), but only biofeedback significantly reduced SBP and DBP when compared with inactive control treatments (waiting list, blood pressure measured in a clinic, placebo biofeedback controls).
The systematic review presented here compared biofeedback treatment with antihypertensive medication, placebo (sham biofeedback treatment), no intervention or another behavioural therapy (including biofeedback) and the primary outcome was effect on blood pressure. The patient population was limited to adults with essential hypertension (taking or not taking antihypertensive medication) as defined above.

A total of 927 non-duplicate references were identified by the search strategy and subsequently screened for inclusion in the review. From these, 41 publications (including three abstracts) reporting 36 RCTs with a total population of 1660 treated patients met the inclusion criteria of the review. In total, 21 trials employed biofeedback treatment with no adjunctive therapy and 15 used biofeedback treatment alongside another treatment. The majority of trials were small and had either no post-treatment follow-up or follow-up of less than 6 months.

No statistical meta-analysis was carried out as the general quality of reporting of trials was poor and there was a large degree of heterogeneity in terms of treatments and comparators. Outcome measures were inconsistently reported. A narrative summary of the data is presented. Data were grouped first by treatment type and then by comparator. In addition, the type of biofeedback was used to further delineate trials. Author conclusions regarding the efficacy or otherwise of biofeedback treatment versus the comparator were summarised and used as the basis of the analysis.

Trial results were variable and conflicting, demonstrating no evidence of short- or long-term benefits of biofeedback in relation to moderation of hypertension. The trials comparing biofeedback with antihypertensive treatment were small and dated and showed no clear evidence for the efficacy of biofeedback treatment. The evidence was equivocal for the effectiveness of biofeedback treatment compared with either no intervention or placebo (sham biofeedback treatment). There was also no clear evidence for the superiority of biofeedback over other behavioural treatments. When benefits were shown they were within the standard error of reproducibility of blood pressure measurement and may therefore have arisen by chance. No trials reporting long-term outcomes were identified for inclusion in the review.

The information obtained concerning biofeedback equipment is summarised. Front-runner technologies could not be identified within this review as the treatment protocols were diverse. There was no consistent evidence of a treatment effect and therefore we were unable to model any benefits.

Conclusions

The quality of research in this area is poor. There is currently no evidence that consistently demonstrates the effectiveness of the use of any particular biofeedback treatment in the control of essential hypertension when compared with pharmacotherapy, placebo (sham biofeedback treatment), no intervention or other behavioural therapies. The lack of evidence of clinical effectiveness negated the need to conduct an economic analysis. Given the current standards for the treatment of hypertension, further research is likely to be considered only as an adjunct to pharmacological interventions.
Chapter 1

Background

Introduction

Description of the health problem

Hypertension (also known as high blood pressure) is defined as persistently high blood pressure, with currently accepted thresholds in the UK at 140/90 mmHg. Hypertension is one of the most prevalent and powerful risk factors contributing to the development of cardiovascular disease (CVD). Systolic blood pressure (SBP) is the major determinant of risk for CVD, particularly for adults over the age of 32 years.

The World Health Organization (WHO) has identified hypertension as one of the most important preventable causes of premature morbidity and mortality in developed and developing countries. People with hypertension have an increased incidence of stroke, transient ischaemic attack, left ventricular hypertrophy, heart failure, myocardial infarction, angina, peripheral vascular disease, fundal haemorrhages or exudates, papilloedema, and proteinuria and renal impairment.

The UK government predicts that the treatment of hypertension would produce large benefits at the population level in terms of avoided CVD. A White Paper published in 1999 identified action to improve the detection of hypertension and increase the number of persons receiving adequate treatment for high blood pressure as a priority. Currently, within the new National Service Frameworks for general practitioners, there are five quality indicators for hypertension and 158 out of 550 clinical points relate directly to hypertension, demonstrating the commitment of the Department of Health to action on this condition.

Causes of hypertension

Biological

Although several factors contribute to the pathogenesis of hypertension, renal mechanisms probably play a primary role while other mechanisms amplify (e.g. sympathetic nervous system activity and vascular remodelling) or buffer (e.g. increased natriuretic peptide or kallikrein–kinin expression) the pressor effects of renal salt and water retention.

Baroreceptors located in several organs detect changes in blood pressure and adjust mean arterial pressure by altering the force and speed of the heart’s contractions as well as the total peripheral resistance (resistance to blood flow). The renin–angiotensin system allows the kidney to activate angiotensin II (a natural vasoconstrictor). Aldosterone (a steroid hormone) is released from the adrenal cortex in response to angiotensin II or high serum potassium levels. It stimulates sodium retention and potassium excretion by the kidneys. As sodium is the main ion that determines the amount of fluid in the blood vessels by the process of osmosis, aldosterone increases fluid retention and, indirectly, blood pressure. The three systems are not necessarily independent of each other.

Drugs and diseases

Some medications such as non-steroidal anti-inflammatories, oral contraceptives, steroids and various cold cures may bring about an increase in blood pressure. Other diseases and syndromes may also cause hypertension: renal disease, renovascular disease, phaeochromocytoma, Conn syndrome, coarctation and Cushing syndrome. Hypertension is twice as common in those with diabetes.

Genetics

Family history may contribute to the risk of developing hypertension with the risk dependent on the age of the family member and number of close relatives with hypertension. Within families of both natural and adopted children, the association for blood pressure levels is higher between biological siblings and biological parent–child pairs than between an adopted child and non-adopted siblings or parents. The exact nature of this genetic predisposition is not yet clear.

Lifestyle

Lifestyle factors documented as significantly impacting on blood pressure include being overweight and obese, lack of physical activity, high alcohol consumption, underconsumption of fruit and vegetables, high dietary intake of saturated fat, high intake of dietary sodium and low intake
Background

of dietary potassium. Changes in lifestyle may lower blood pressure by as much as a single blood pressure-lowering drug, and combinations of two or more lifestyle modifications can achieve even better results.

Epidemiology

The lifetime risk of hypertension is high, with longitudinal data from the Framingham study indicating a lifetime risk of 80–90% in middle-aged men and women. The Health Survey for England (HSE) 2003 gives estimates of the overall prevalence of hypertension of 30.6%. This survey also reports a steep increase in prevalence with age for both men and women.

Prevalence is higher among men than women up to age 64 years, but women show a steeper increase with age compared with men so that men and women show the same prevalence of hypertension between the ages of 65 and 74 years. Beyond 75 years there are a greater proportion of women than men with hypertension.

There are limitations associated with the findings reported in the HSE, primarily related to the definition of hypertension. In the survey, three blood pressure measurements were taken per respondent, each at 1-minute intervals, and the mean of the second and third measurements was calculated. All participants with blood pressure greater than or equal to 140/90 mmHg, whether treated or untreated, were classified as hypertensive.

In clinical practice, hypertension is diagnosed after two measures are taken at two different time points and it has been argued that the HSE statistics may be an overestimate of true prevalence because they were based on recordings taken on the same day. The NHS Information Centre for Health and Social Care (ICHSC) makes available data from GP practices in England; the reported level of hypertension for 2005/6 was 12%. Although the ICHSC figures do not include the number of people with undiagnosed hypertension and definitions of hypertension vary, these data do highlight hypertension as a condition that affects a high proportion of patients in GP practices.

Diagnosis and assessment of hypertension

The majority of people are unaware that they have hypertension because it frequently does not present with specific symptoms. Current National Institute for Health and Clinical Excellence (NICE) British Hypertension Society (BHS) guidance recommends that hypertension be identified by taking at least two measures of blood pressure on two separate occasions ‘under the best conditions available’. Table 1 presents the blood pressure classifications as published by the BHS.

According to these classifications, hypertension is diagnosed when systolic or diastolic pressure or both is above 140/90 mmHg. It may then be classified as either essential (most common) or secondary. Essential hypertension has no specific medical cause whereas in secondary hypertension the elevated blood pressure is a result of another condition, such as kidney disease or particular tumours.

Other relatively uncommon types of hypertension include malignant, isolated systolic, white coat, resistant and pulmonary artery. In addition, other forms of hypertension exist in pregnancy: chronic, pre-eclampsia and transient.

Current treatment options

In the UK, current BHS and NICE guidance recommends that drug therapy should be offered to patients with:

- persistent high blood pressure of 160/100 mmHg or more
- persistent blood pressure of more than 140/90 mmHg when there is raised cardiovascular risk (10-year risk of CVD of 20% or more or existing CVD or target organ damage)
- isolated systolic hypertension of more than 160 mmHg.

The BHS recommends a blood pressure target of < 150/90 mmHg as an audit standard, with lower targets (≤ 130/90 mmHg) for higher risk patients, whereas NICE guidance states that the aim of antihypertensive treatment is for blood pressure to be maintained at 140/90 mmHg or below, the optimal for reducing major cardiovascular events.
There are varying levels of treatment. For those with high-normal blood pressure, lifestyle changes and regular checks are emphasised to reduce the likelihood of the development of hypertension and the need for drug therapy. With regard to drug treatment, three types of antihypertensive medication are recommended in the guidance produced by NICE\(^1\) in agreement with the BHS: angiotensin-converting enzyme (ACE) inhibitors (or angiotensin II receptor antagonist if ACE inhibitors are not tolerated), calcium channel blockers (CCBs) and thiazide-type diuretics. These are prescribed according to age and ethnicity as outlined in Table 2.

Beta-blockers are no longer preferred as a routine initial therapy for hypertension as it has been shown that they are less effective at reducing major cardiovascular events and are associated with an increased incidence of diabetes, particularly when combined with diuretics.\(^1\) However, beta-blockers may be considered as an option for younger people such as women of childbearing potential, patients with evidence of increased sympathetic drive or those who have an intolerance of, or contraindications to, ACE inhibitors and angiotensin II receptor antagonists. If a single drug does not sufficiently control hypertension, combinations of drugs may be prescribed. In almost 50\% of cases, more than one drug is required.\(^10\)

The majority of adults in England with hypertension have blood pressure levels above recommended targets.\(^10\) Reasons for this inability to maintain the recommended blood pressure levels are multifactorial and could include factors such as patient adherence, inadequate/ineffective treatment and lack of patient monitoring.\(^7\)

**Biofeedback**

Biofeedback can be defined as a group of non-pharmacological therapeutic procedures that use electronic instruments to measure, process and provide information (feedback) to patients regarding their neuromuscular and autonomic nervous system activity. This feedback may be in the form of analogue (or binary) and/or visual (or auditory) signals.\(^11\)

The notion of gaining control over biological processes that are ordinarily involuntary has
been linked to ancient yogis who were able to demonstrate amazing skills such as temporarily stopping the heart from pumping blood, making the heart skip a beat at a given signal, and controlling pain and blood flow. These abilities may be thought of as mystic, but psychologists have been able to demonstrate that it is possible for ‘ordinary’ people to learn to manage their own bodily functions through techniques such as biofeedback.

In relation to blood pressure, early work on biofeedback with rats demonstrated that the animals could learn to increase or decrease their systolic blood pressure when reinforced for doing so. Further work with human adult males showed that they were also able to increase, but to a much greater extent decrease, systolic blood pressure when given feedback (light and tone) and rewards.

In simple biofeedback training for hypertension, a patient is connected to an instrument that provides continuous information about their blood pressure. Whenever blood pressure falls to a specified level, a signal (aural or visual) is given. The patient then reflects on what they were thinking or doing when the blood pressure was low and tries to repeat the activity in order to keep it low. In this way, the patient learns to identify sensations that accompany reductions in blood pressure and, after several training sessions, the patient may be able to develop skills to maintain control of blood pressure. The type of information given to patients may differ; as well as direct blood pressure biofeedback measures, other indirect indicators may be used including thermal (TBF), galvanic skin response (GSR), heart rate (HR) and electromyographic (EMG) activity.

In TBF the patient is given information regarding the temperature of their finger or toe and instructed to warm their hands or feet in relation to this feedback. The physiological rationale is that increased sympathetic activity commonly observed during stress constricts the blood vessels in the skin and the decreased blood flow results in a cooler temperature. In contrast, decreased sympathetic activity results in less vasoconstriction, thereby increasing blood flow. As individuals warm their hands, they are actually learning to decrease neurally-mediated vasoconstriction and subsequently to decrease total peripheral resistance.

In EMG feedback the patient is given information regarding muscle tension. EMG is thought to mediate relaxation, and changes in muscle contraction affect blood flow; the muscle receives more blood flow during a weak contraction than during a strong contraction. GSR gives a measure of sweat gland activity by measuring skin conductance. Sweating is a sympathetically mediated response to stressful conditions; the less active the sweat glands are, the less aroused the patient is. Biofeedback training may include other techniques in addition to the biofeedback, for example relaxation, meditation or yoga.

The website of the Association for Applied Psychophysiology and Biofeedback affirms (based on the evidence of two systematic reviews and meta-analyses reviewed below) that numerous high-quality studies have demonstrated that people having high blood pressure – especially if stress related – can benefit extensively from biofeedback as long as they learn and practice the skills needed to control their blood pressure, and that many hypertensives no longer need any medication after successful biofeedback training. The Association rate biofeedback therapy for hypertension as efficacious (level 4 on a scale of 1–5, with 5 being the best).

Outcome measures

The majority of published trials of biofeedback report data taken in the laboratory or clinic. However, it has been suggested that office- or clinic-based measures used in the biofeedback trials may be somewhat unreliable as they cannot detect ‘white coat’ hypertension, wherein the patient exhibits elevated blood pressure but only in the clinical setting. This phenomenon may affect between 20% and 30% of patients diagnosed with hypertension. In trials, habituation to the setting can also occur, resulting in declines in blood pressure that may be mistaken for treatment effects. Short baselines can exacerbate this problem.

Both ambulatory blood pressure monitoring (ABPM) and home monitoring offer the opportunity to screen out white coat hypertension, and drug treatment research is increasingly using ambulatory measures as clinical end points. With regard to clinical practice, current NICE guidance recommends the use of measures taken in a GP clinic to diagnose hypertension and does not recommend the routine use of ABPM or home measurement devices as their value has not been adequately established. However, the BHS acknowledges that ABPM provides more
information than home or GP clinic measurements (mean day- and night-time measurements and blood pressure variability) and may be a better predictor than office measures of CVD risk and target organ damage as well as a better method of assessing treatment effects. With regard to home blood pressure monitoring (given advances in equipment design) such measures can also provide more information than those taken in a GP clinic and have the advantage of involving the patient more closely in their own care and treatment. It should be noted, however, that home monitoring (rather than ABPM) is not thought to predict cardiovascular risk or outcomes more effectively than clinic readings.4

A further issue with regard to outcomes is the effect of initial baseline measures of blood pressure. It is now well documented that high pretreatment values can result in greater treatment effects than lower values.11,25,29 Lower values may be subject to the so-called ‘floor effect’,28,30 whereby only small reductions are possible. Most biofeedback trials only include patients considered to be ‘mildly hypertensive’, at the lower end of the hypertensive threshold; thus, the effects of any treatment are likely to be small. It has also been argued that other critical outcomes such as the ability of a treatment to prevent the development or worsening of heart disease and the ultimate reduction in cardiovascular mortality be assessed in addition to the usual immediate changes in blood pressure.29

Systematic reviews of biofeedback

Two systematic reviews have previously reported on the efficacy of biofeedback treatment for hypertension.11,25 We quality assessed these reviews51 and the results are summarised in Table 3. Neither review considered any evidence for biofeedback treatment versus antihypertensive medication.

The reviews both used internationally accepted standards and were judged to be of good quality. Both reviews pooled data and reported small effect sizes with the use of biofeedback. The appropriateness of such an analysis is questioned given the variation in the methods of biofeedback, differences in comparators and variations in the timing of outcome measures. The reviews provided limited information regarding the data used in the meta-analyses (e.g. which studies were included, actual data input, time point of outcome measure, etc.).

In addition, both reviews reported a need for significant manipulation of data to allow for the pooling. Nakao et al.11 pointed out in their analysis that ‘...standard errors of pre- and post-treatment blood pressure changes...’ were not reported in a number of studies and these had to be calculated from available data. Yucha et al.25 also pointed out a need to calculate standard deviations within studies and to make assumptions regarding appropriate measures of correlation. In a later paper, when referring to her previous biofeedback review Yucha22 reported:

While doing this meta-analysis, I noticed that these studies were plagued with inconsistency in their methodology and reporting, making statistical combination difficult if not impossible.

Therefore the meta-analyses from these two reviews should be considered with extreme caution.

The aim of the review by Nakao et al.11 was to examine the blood pressure-lowering effects of biofeedback treatment in patients with essential hypertension. A total of 22 randomised controlled trials (RCTs) with a patient population of 905 essential hypertensive patients were included in a meta-analysis. The analysis took account of biofeedback types (alone or combined with another therapy) and control types (no intervention and a non-specific intervention). The authors concluded that biofeedback intervention decreased SBP and diastolic blood pressure (DBP) more than non-intervention controls but not more than sham or non-specific intervention controls. Only relaxation-assisted biofeedback significantly decreased both SBP and DBP compared with sham or non-specific behavioural interventions. The authors concluded that biofeedback was more effective than no intervention, but was only superior to sham or non-specific interventions when combined with a relaxation technique.

The second review, by Yucha et al.,25 aimed to determine the effectiveness of biofeedback in the treatment of essential hypertension. A total of 23 RCTs were included and interventions were categorised as biofeedback, active treatment control and inactive treatment control. Active treatments were relaxation training, cognitive therapy and home monitoring, and inactive treatments were waiting list, blood pressure measured in a clinic and sham biofeedback treatment controls. The biofeedback and active control treatments were found to reduce SBP and DBP, but only
biofeedback significantly reduced SBP when compared with inactive control treatments.

The results of the two reviews generally support one another in that they conclude that biofeedback can lower blood pressure by small amounts. It is worth noting that, for ethical reasons, most biofeedback trials are populated with patients who have mild or borderline blood pressure or who are taking antihypertensive medication. Therefore, effects of biofeedback may be masked.

The current project

The purpose of the current project was to assess the evidence (short and long term) regarding the clinical effectiveness of biofeedback treatment for the treatment of essential hypertension. Long term was considered to be at least 6 months and preferably 12, although evidence from trials that were of a shorter duration was considered. If evidence of effectiveness had been demonstrated then these effects would have been incorporated into an economic analysis. Limited information on currently available biofeedback equipment is provided.

<table>
<thead>
<tr>
<th>Quality assessment checklist item</th>
<th>Nakao 2003</th>
<th>Yucha 2001</th>
</tr>
</thead>
<tbody>
<tr>
<td>Did the review address a clearly focused research question?</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Was the search strategy adequate (i.e. did the reviewers identify all relevant studies)?</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Are the inclusion/exclusion criteria specified?</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Did the review include the right type of studies?</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Did the reviewers assess the quality of the included studies?</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Was the method of data extraction reported?</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Were appropriate measures of outcomes used?</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>If the results of the studies have been combined, was it reasonable to do so?</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Are appropriate subgroup analyses presented?</td>
<td>NA</td>
<td>_</td>
</tr>
<tr>
<td>Are the main results of the review reported (e.g. numerical results included with the confidence intervals)?</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>Are issues of generalisability addressed?</td>
<td>✓</td>
<td>✓</td>
</tr>
</tbody>
</table>

✓, yes; ×, no; ✓/×, partially; NA, not applicable.

a Poor quality of trials, inconsistency in reporting of trials and lack of information on dropouts may mitigate against a meta-analysis.
Chapter 2
Methods

Review of clinical effectiveness

Search strategy
A comprehensive search strategy was developed and used (YD) to examine the electronic databases listed in Table 4. Details of the electronic search strategies used and the number of references retrieved for each search are provided in Appendix 1. All references were exported to the EndNote® reference database version X.0.2 (ISI ResearchSoft, Berkeley, CA).

The search did not include methodological filters that would limit results to a specific research study design. The search was restricted to reports that included abstracts written in English. Searches for the first seven databases had no date restriction and were carried out from database commencement to May 2007. The search of PsycINFO was carried out at a later date and the search was extended to October 2007. To ensure comprehensiveness, an updated search of all databases was carried out in the final month before the completion of this report.

Reference lists of retrieved articles were searched to identify further studies. An advisory panel was established to guide the review process: the role of the panel was to answer specific questions as the review progressed and to comment on an early draft of the report, including identifying missed or ongoing trials, and to advise on types of biofeedback instrumentation and current usage.

Inclusion and exclusion criteria
The identified articles were assessed for inclusion through two stages and disagreements were resolved by discussion. In stage one, two reviewers (JG, RD) independently scanned all of the titles and abstracts and identified the potentially relevant articles to be retrieved. To ensure that the screening was comprehensive, inclusion at stage one incorporated any behavioural or complementary therapy that might be relevant to biofeedback. In stage two, full text copies of the selected papers were obtained and each was assessed independently by two reviewers (JG, RD) for inclusion. Details of the inclusion and exclusion criteria are presented in Table 5. A quality of reporting of meta-analyses (QUOROM) flow diagram summarising the selection and inclusion of studies is provided in Appendix 3.

Data extraction
Data extraction was carried out by two reviewers (JG, NR). Individual trial data relating to trial design and findings were extracted and checked using a pretested data extraction form. Data were cross-checked by one reviewer (YD).

Quality assessment
The methodological quality of each trial was independently evaluated by at least two reviewers (JG, NR, YD) using criteria based on guidance issued by the Centre for Reviews and Dissemination (CRD). Any differences in quality grading were resolved through discussion. Inter-rater reliability was not assessed.

TABLE 4 Databases searched

| MEDLINE                                    |
| EMBASE                                     |
| ISI Web of Knowledge/Web of Science       |
| ISI Web of Knowledge/ISI Proceedings       |
| Cochrane Library 2007                     |
| CINAHL (Cumulative Index to Nursing and Allied Health Literature) |
| AMED (Allied and Complementary Medicine)   |
| PsycINFO was searched after the above were completed |
TABLE 5  Inclusion and exclusion criteria

<table>
<thead>
<tr>
<th>Trial design</th>
<th>Randomised controlled trials (RCTs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient population</td>
<td>Adults with essential hypertension (i.e. ≥ 140/90 mmHg), medicated or unmedicated with antihypertensive drugs</td>
</tr>
<tr>
<td>Interventions</td>
<td>Biofeedback treatment alone or in combination</td>
</tr>
<tr>
<td>Comparators</td>
<td>Antihypertensive medication, placebo (sham biofeedback), no treatment, other types of biofeedback treatment, other behavioural treatments</td>
</tr>
<tr>
<td>Outcomes</td>
<td>Blood pressure measures</td>
</tr>
<tr>
<td>Exclusion criteria</td>
<td>Patients with other types of hypertension, non-RCT, narrative reviews, editorials or opinions</td>
</tr>
</tbody>
</table>

Analysis of results

A narrative summary of the data is presented. The qualitative heterogeneity across the trials, including the poor quality of the trial reports, the diversity of biofeedback protocols and the inconsistency in reporting of outcomes, precluded a statistical synthesis of the included trial results. Biofeedback treatments were divided into those that were used alone and those that were used in combination with another therapy. These were categorised further into antihypertensive medication, placebo (sham biofeedback treatment), non-intervention control and other behavioural treatments. The type of feedback (direct or indirect) was also noted. Author conclusions regarding the efficacy or otherwise of biofeedback treatment versus the comparator were summarised and used as the basis of the analysis.

Methods for reviewing currently available biofeedback equipment

We identified biofeedback equipment by contacting organisations involved in the treatment of hypertension. These included the BHS, the American Society for Hypertension (ASH), the American Association for Applied Physiology and Biofeedback (AAPB), the National Centre for Complementary and Alternative Medicine (NCCAM), the Biofeedback Foundation of Europe (BFE) and the European Society for Hypertension (ESH). Equipment used in RCTs was also noted. Additionally, a panel of clinical advisers was also asked to provide opinions. The findings are presented in Appendix 2.
Chapter 3

Results

Clinical effectiveness

Selection of included trials

A total of 927 non-duplicate references were identified by the search strategy and subsequently screened for inclusion in the review. From these, 100 papers were obtained in full text to facilitate the application of inclusion/exclusion criteria. A total of 41 relevant publications (including three abstracts) reporting 36 RCTs met the inclusion criteria (Table 6). A further recent RCT was identified during a subsequent update of searches.

Of the included trials, 34 (including three abstracts) were published in peer-reviewed journals. The remaining two were abstracts from PhD theses. One report presented data from two studies, and another trial, reported in two papers, compared two different types of biofeedback.

The included trials reported comparisons between biofeedback treatments [either biofeedback alone (n = 21) or combined (n = 6) with an adjunctive therapy], antihypertensive medications, placebo (sham biofeedback treatment), non-intervention controls or other behavioural treatments.

Reports of trials that did not fulfil the inclusion criteria (along with reasons for exclusion) appear in Appendix 4.

Quality assessment of included trials

The methodological quality of the included trials was assessed using the checklist described in the CRD Report No. 4, a summary of the assessment is provided in Table 7.

Overall, the methodological quality of the included trials was poor. All stated that patients were randomly allocated to treatment groups; however, only four described the method of randomisation and only two noted whether or how allocation was concealed. Only eight provided information regarding the blinding of assessors and, with the exception of the four trials in which blinding was inherent in the trial design, blinding of either administrators or participants was not mentioned. None of the trials reported any assessment of blinding procedures. It is worth noting that, without the use of a sham placebo treatment, blinding of treatment providers and patients is difficult to achieve; however, blinding of assessors can and should always be managed. Intention-to-treat (ITT) analyses were not specifically reported in any trial; thus, ITT was assumed in cases in which it appeared that all patients randomly assigned to one of the treatment groups were included in the final analysis whether or not they completed or received that treatment. Co-interventions (antihypertensive medication) were well reported. Baseline comparability was achieved or partially achieved in 25 trials. With the exception of three trials, details of eligibility criteria were recorded. It is worth noting that the included trials were relatively old: seven from the 1970s, 16 from the 1980s, 10 from the 1990s and just three from 2000 onwards. The quality of reporting did not appear to improve over time.

Trial characteristics

Trial characteristics are presented in Tables 20 and 21 in Appendix 5.

The 36 included trials incorporated a total population of approximately 1660 treated patients, with cohorts ranging in size from 1256 to 158. The trial populations were generally small (less than 50); only four included more than 100 patients. All were single centred and the majority were conducted in the USA. Of the non-US trials, three were UK based; others were conducted in Canada, Australia, Italy, the USSR, Japan, Venezuela, Taiwan and Israel. Four trials employed a placebo treatment, whereas the remainder were all comparative with two or more arms. The number of biofeedback sessions ranged across trials from 4 to 20.

The majority of trials included either no post-treatment follow-up or less than 6 months’ follow-up. Fifteen included post-treatment follow-up periods of 6 months up to a maximum of 12 months. When funding
Results

TABLE 6  Summary of included trials

<table>
<thead>
<tr>
<th>Biofeedback alone</th>
<th>Biofeedback combinations</th>
<th>Combination therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Achmon 1989^18</td>
<td>Berglund 199^15a,b</td>
<td>Menninger protocol</td>
</tr>
<tr>
<td>Billion 1980^3a,b</td>
<td>Canino 1994^66</td>
<td>+ relaxation + anger management</td>
</tr>
<tr>
<td>Blanchard 1979^54</td>
<td>Chesney 1987^49</td>
<td>+ relaxation</td>
</tr>
<tr>
<td>Blanchard 1987^68</td>
<td>Frankel 1978^61</td>
<td>+ relaxation</td>
</tr>
<tr>
<td>Blanchard 1988^36 (USA)</td>
<td>Friedman 1978^7,36c</td>
<td>+ hypnosis</td>
</tr>
<tr>
<td>Blanchard 1988^56 (USSR)</td>
<td>Hafner 1982^22</td>
<td>+ relaxation + meditation</td>
</tr>
<tr>
<td>Blanchard 1993^62</td>
<td>Irvine 1991^42</td>
<td>+ relaxation + meditation + imagery</td>
</tr>
<tr>
<td>Blanchard 1996^16</td>
<td>Jacob 1992^43</td>
<td>+ relaxation</td>
</tr>
<tr>
<td>Bonso 2005^66</td>
<td>Jurek 1992^25</td>
<td>+ relaxation</td>
</tr>
<tr>
<td>Friedman 1978^37,38c</td>
<td>Khramelashvili 1986^59a</td>
<td>+ relaxation</td>
</tr>
<tr>
<td>Goldstein 1982^41</td>
<td>McCraty 2003^46</td>
<td>+ inner quality management</td>
</tr>
<tr>
<td>Hager 1978^55</td>
<td>McGrady 1981^19</td>
<td>+ relaxation</td>
</tr>
<tr>
<td>Hatch 1985^29</td>
<td>McGrady 1994^43</td>
<td>+ relaxation</td>
</tr>
<tr>
<td>Hunyor 1997^47</td>
<td>Patel 1975^45</td>
<td>+ relaxation</td>
</tr>
<tr>
<td>Luborsky 1982^60</td>
<td>Patel 1988^40</td>
<td>+ relaxation</td>
</tr>
<tr>
<td>Nakao 1997^15</td>
<td>Thananopavarn 1979^56a</td>
<td></td>
</tr>
<tr>
<td>Tsai 2007^35</td>
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<td></td>
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<tr>
<td>Walsh 1977^37</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zurawski 1987^61</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a  Abstract only.

b Indicates abstract from PhD.

c Trial included two different types of biofeedback as comparators.

was reported, trials were frequently supported by grants from independent sources; only two trials^40,42 reported some funding support from a pharmaceutical company. The commonly cited primary outcome of the trials was the effect of the interventions on direct measures of blood pressure, although the primary outcome in two trials^58,62 was the reduction in medication from two antihypertensive drugs to one. Three trials^32,54,55 described patients as having ‘borderline hypertension’, 11 ‘mild hypertension’^16,21,22,41,42,47,49,51,56,60,64 and the remainder described patients as ‘hypertensive’. A number of biofeedback modalities were employed: blood pressure;^15,21,35,38,39,41,47,54,55,60 HR;^18, EMG;^19,21,22,36,45,49,51,55 TBF;^16,43,45,46,48–52,58,62,65,66 pulse wave velocity;^57 GSR^32,40,42,45,61 and heart rate variability.^44 In some cases more than one modality was employed within the same trial.

Biofeedback alone

Of the biofeedback alone trials, three^41,36,60 were included in the category of biofeedback alone versus antihypertensive medication, three^35,47,53 were included in the category of biofeedback alone versus placebo (sham biofeedback treatment) and eight^15,16,18,38,39,41,62,64 were included in the category of biofeedback alone versus non-intervention treatment. In the last category, patients in the control arm had blood pressure checks at clinics, self-monitored their own blood pressure or had no treatment beyond baseline and end of intervention blood pressure measures taken. Fifteen trials^18,36,38,39,41,53,58,60–62,68 were included in the biofeedback alone versus
### TABLE 7 Quality assessment of included trials

<table>
<thead>
<tr>
<th>Checklist items</th>
<th>Randomisation</th>
<th>Baseline comparability</th>
<th>Blinding</th>
<th>Withdrawals</th>
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<td>Allocation concealment</td>
<td>Number stated</td>
<td>Present</td>
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<td>✓ ✓ ✓</td>
<td>✓/x f</td>
</tr>
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<td>✓/x h</td>
</tr>
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<td>✓/x i</td>
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<td>NS</td>
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<td>✓/x j</td>
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<td>✓/x k</td>
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<td>✓/x l</td>
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<td>✓ ✓ ✓</td>
<td>✓/x p</td>
</tr>
<tr>
<td>Hunyor 1997</td>
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<td>NS</td>
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</tr>
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<td>Nakao 1997</td>
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<td>✓ ✓ ✓</td>
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</tr>
<tr>
<td>Thananopavarn</td>
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<td>NS</td>
<td>✓ ✓ ✓</td>
<td>✓/x s</td>
</tr>
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</table>

continued
### TABLE 7 Quality assessment of included trials (continued)

<table>
<thead>
<tr>
<th>Biofeedback combinations</th>
<th>Truly Random</th>
<th>Allocation concealment</th>
<th>Number stated</th>
<th>Baseline comparability</th>
<th>Eligibility criteria specified</th>
<th>Co-interventions identified</th>
<th>Blinding</th>
<th>Administration</th>
<th>Participants</th>
<th>Procedure assessed</th>
<th>&gt; 80% in final analysis</th>
<th>Reasons stated</th>
<th>Intention to treat</th>
</tr>
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<td>Checklist items</td>
<td>Randomisation</td>
<td>Baseline comparability</td>
<td>Blinding</td>
<td>Withdrawals</td>
<td>Intention to treat</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Truly Random</td>
<td>Allocation concealment</td>
<td>Number stated</td>
<td>Presented</td>
<td>Eligibility criteria specified</td>
<td>Co-interventions identified</td>
<td>Assessors</td>
<td>Administration</td>
<td>Participants</td>
<td>Procedure assessed</td>
<td>&gt; 80% in final analysis</td>
<td>Reasons stated</td>
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<tr>
<td>McCratty 200354</td>
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<td>✓</td>
<td>✓</td>
<td>✓/✗</td>
<td>✓</td>
<td>✓</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>✓</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>McGrady 198119</td>
<td>NS</td>
<td>NS</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
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<td>NS</td>
<td>NS</td>
<td>✓</td>
<td>x</td>
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<tr>
<td>McGrady 199463</td>
<td>NS</td>
<td>NS</td>
<td>✓</td>
<td>✓</td>
<td>✓/✗</td>
<td>✓</td>
<td>✓</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>x</td>
<td>✓</td>
<td></td>
</tr>
<tr>
<td>Patel 197545</td>
<td>NS</td>
<td>NS</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
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<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>✓</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>Patel 198840</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓/✗</td>
<td>NS</td>
<td>✓</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>✓</td>
<td>x</td>
<td></td>
</tr>
</tbody>
</table>

✓, yes; ✓/✗, item partially addressed; x, no; NS, not specified; NA, not applicable.

a No trials explicitly state intention-to-treat analysis. We have inferred this to be the case when all patients appear to be included in the final analysis.

b Greater % males in control group.

c Abstract only.

d Stated by authors.

e Authors state that first two sessions not distinguishable to patients.

f Blood pressures lower in group 1.

g At end of treatment phase.

h Greater proportion of males.

i Differences between groups with regard to numbers of previously medicated patients.

j Patient blood pressures not presented.

k At 12-month follow-up.

l Only blood pressure measurements presented; these appeared to be different between groups.

m Age, gender and baseline blood pressure differences – noted in paper.

n Blood pressure data not presented.

o Allocation concealment addressed, but not clear; likely to be yes as randomisation by central computer.
other behavioural treatments category. These treatments included cognitive group therapy for anger,18 relaxation,39,41,55-58,60 relaxation plus EMG biofeedback,54 TBF at home,69 autogenic training,70 EMG biofeedback,54 hypnosis,38 meditation55 and stress management.61

Biofeedback combinations

None of these trials compared biofeedback combination treatment with antihypertensive medication. One trial61 compared biofeedback combinations with a placebo (sham biofeedback treatment), and 13 trials19,21,22,28,40,44,46,49,52,59,63 were included in the biofeedback combinations versus non-intervention control category. Eight trials22,38,42,44,49,50,59 compared biofeedback combinations with other behavioural treatments. These included hypnosis,38 meditation,29 non-specific support therapy,42 stress education43 and relaxation.50,49 One49 of these trials employed three behavioural comparators.

The majority of biofeedback treatment was combined with relaxation.19,21,40,45,46,49,50,59,63 Others combinations included the Menninger protocol,52 relaxation plus meditation,22 relaxation plus anxiety management,46 relaxation plus imagery plus meditation,42 relaxation plus diuretics,51 yoga,45 hypnosis38 and inner quality management (IQM).44

Patient characteristics

Patient characteristics tables are presented in Appendix 6.

Sixteen16,35,36,38,41-44,46,49,51,52,57,62,68 of the included trials had a population of more than 60% males; moreover, three36,52 of these trials included only males. Seven trials19,21,36,59,49,51,63 reported the ethnic origin of patients, all predominantly white. Of the included trials, eight16,35,36,42,44,47,61 included only patients not taking antihypertensive medication, three45,56,60 compared patients not taking antihypertensive medication in the biofeedback treatment arm with those in an arm treated with drugs only, two15,51 included only patients taking antihypertensive drugs, three18,56,62 included patients on a specific two-drug regimen (with the primary outcome as a reduction in these drugs) and 1611,18,19,21,22,38-40,44,45,49,50,54,57,61,63 included a mix of patients taking or not taking antihypertensive medication. In one of these last trials59 the number of patients prescribed antihypertensive drugs changed across the course of the trial. Four trials32,55,55,59 did not state the medication status of the patients.

When mean ages of patients were given, these ranged from 30.910 to 59.945 years. When stated, patients had been diagnosed with hypertension for between 4 months22 and 14 years.41

Clinical results and analysis

The preceding section indicates that the included trials were of poor quality and the treatments and comparators were heterogeneous. These factors mitigated against any statistical analysis of the data (in these circumstances a meta-analysis is likely to provide misleading results); thus, a narrative summary of the findings is presented. Results have been grouped first by biofeedback type (i.e. biofeedback alone or in combination with another therapy) and then by comparator [antihypertensive medication, placebo (sham biofeedback treatment), non-intervention control, other behavioural treatments]. In addition, the type of biofeedback has been used to further delineate trials. In this way blood pressure biofeedback (direct biofeedback) is marked out from other (indirect) modes of biofeedback. All measures are mean changes in mmHg with standard deviations shown whenever reported. When mean changes were not specifically reported, these were calculated by subtracting the post-treatment from the pre-treatment blood pressures (standard deviations were not calculated in these cases). When patient numbers are quoted, these represent numbers reported in results rather than numbers randomised. Table 20 in Appendix 5 documents both the number of patients randomised in each trial and the number of patients included in the final analysis.

With reference to the two meta-analyses referred to earlier in this report11,25 there were differences and similarities between the included trials. The present review included 12 trials that were not featured in the previous reviews and excluded three trials that were featured in these reviews. Table 8 documents the additions and exclusions.

Biofeedback alone versus antihypertensive medication

Three trials compared biofeedback with antihypertensive medication (Table 9). These trials were small (total n = 51) and dated, with no long-term follow-up data. With regard to data collected in the laboratory, two trials41,60 reported medication to be significantly more effective than biofeedback treatment for SBP, but not for DBP. The third trial36 did not present statistical comparisons, but stated that biofeedback may be as effective as drug treatment. The ‘home’ data from the Goldstein41
TABLE 8 Trial differences between reviews

<table>
<thead>
<tr>
<th>Trials extra to previous reviews</th>
<th>Trials included in previous reviews, but excluded in the present review</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blanchard 1986</td>
<td>Paran 1996</td>
</tr>
<tr>
<td>Blanchard 1987</td>
<td>Patel 1973</td>
</tr>
<tr>
<td>Bonso 2005</td>
<td>Patel 1981</td>
</tr>
<tr>
<td>Chesney 1987</td>
<td></td>
</tr>
<tr>
<td>Cohen 1983</td>
<td></td>
</tr>
<tr>
<td>Friedman 1978</td>
<td></td>
</tr>
<tr>
<td>Hager 1978</td>
<td></td>
</tr>
<tr>
<td>Khramelashvili 1986</td>
<td></td>
</tr>
<tr>
<td>McCratty 2003</td>
<td></td>
</tr>
<tr>
<td>Thananopavarn 1979</td>
<td></td>
</tr>
<tr>
<td>Tsai 2007</td>
<td></td>
</tr>
<tr>
<td>Walsh 1977</td>
<td></td>
</tr>
</tbody>
</table>

trial reported medication to be significantly better than biofeedback for both SBP and DBP ($p < 0.01$). Only the Goldstein trial presented data beyond the treatment period, but this was limited to the biofeedback arm. These data (presented in a graph) showed that, at 6 months, SBP in the biofeedback group (note reduced numbers) returned to levels above those recorded at baseline whereas DBP remained at post-treatment levels.

**Biofeedback alone versus placebo (sham biofeedback treatment)**

Three trials compared biofeedback with placebo (sham biofeedback) treatment (Table 10). They were small, populated by a total of no more than 123 patients. Overall findings are contradictory and there are no long-term data.

The two main trials report conflicting results. Hunyor et al. reported no significant difference between active biofeedback and placebo treatment, whereas Tsai et al. reported a significant difference ($p < 0.001$) between treatments. Both reported outcomes on SBP only and at similar time points. Neither present long-term data. There are no data presented in the Billion abstract, but the author notes no significant differences between groups.

**Biofeedback alone versus non-intervention control**

The majority of the eight small trials ($n = 235$ approximately) showed no significant effects of biofeedback treatment compared with non-intervention controls post treatment (Table 11). There is scant evidence regarding long-term efficacy. Only three trials reported significant differences between the biofeedback treatment and non-intervention control groups for SBP and DBP. One of these, Achmon et al., reported a significance level of $p < 0.0005$. A fourth trial found biofeedback to be significantly better than control for DBP only. None of the trials reporting positive effects of biofeedback provided any long-term data in comparison to the control.

**Biofeedback alone versus other behavioural treatments**

Of the 16 trials ($n \geq 465$ approximately) three found biofeedback to be superior to other behavioural interventions, two for both SBP and DBP, and one for DBP only (Table 12). Two trials found other treatments superior to biofeedback. Seven other trials reported no differences between biofeedback treatment and other interventions. One trial did not report an outcome. Comparative data were not available for four trials. Change data from three trials were not relevant as the purpose of these trials was to reduce antihypertensive medication while maintaining optimum blood pressure. Longer-term data from Achmon et al. reported that biofeedback treatment continued to be superior to cognitive therapy at 6 months, but only for SBP.

**Biofeedback combinations versus placebo (sham treatment)**

One small and dated trial compared a biofeedback combination with placebo (sham biofeedback) treatment (Table 13). No differences were reported between treatment and control groups.
### TABLE 9 Biofeedback alone versus antihypertensive medication: changes in blood pressure

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre-post treatment (mmHg), mean</th>
<th>Change in DBP pre-post treatment (mmHg), mean</th>
<th>Change in SBP at follow-up (mmHg), mean</th>
<th>Change in DBP at follow-up (mmHg), mean</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood pressure biofeedback</strong></td>
<td><strong>Goldstein 1982</strong></td>
<td>Treatment</td>
<td>Laboratory</td>
<td>149.1</td>
<td>97.3</td>
<td>-4.1</td>
<td>-4.4</td>
<td>Graph shows return to baseline at 6 months (n = 5)</td>
<td>Graph shows maintenance of effect at 6 months (n = 5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Home</td>
<td>147.2</td>
<td>94.6</td>
<td>-4.5</td>
<td>-3.9</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(n = 9)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Control (meds)</td>
<td>Laboratory</td>
<td>144.2</td>
<td>98.2</td>
<td>-14.8</td>
<td>-5.6</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Home</td>
<td>144</td>
<td>96</td>
<td>-17.6</td>
<td>-10.4</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(n = 9)</td>
<td></td>
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</tr>
<tr>
<td></td>
<td><strong>Luborsky 1982</strong></td>
<td>Treatment</td>
<td>Laboratory (n = 14):</td>
<td>Standing</td>
<td>138.3</td>
<td>93.2</td>
<td>-6.5</td>
<td>-5.5</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lying</td>
<td>136.7</td>
<td>86.2</td>
<td>-2.6</td>
<td>-4.3</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Control (meds)</td>
<td>Laboratory (n = 10):</td>
<td>Standing</td>
<td>144.7</td>
<td>101.3</td>
<td>-18.8</td>
<td>-10.3</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lying</td>
<td>143.7</td>
<td>91.8</td>
<td>-13.5</td>
<td>-7.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>Type of biofeedback not specified</strong></td>
<td><strong>Thananopavarn 1979</strong></td>
<td>Treatment</td>
<td>Laboratory</td>
<td>155 (6)</td>
<td>96 (4)</td>
<td>-12.0</td>
<td>-7</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Home</td>
<td>159 (7)</td>
<td>94 (3)</td>
<td>-13.0</td>
<td>-6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(n = 5)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Control (meds)</td>
<td>Laboratory</td>
<td>142 (4)</td>
<td>95 (2)</td>
<td>-22</td>
<td>-5</td>
<td>NR</td>
<td>NR</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Home</td>
<td>146 (4)</td>
<td>100 (3)</td>
<td>-14</td>
<td>-9</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(n = 4)</td>
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</tr>
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</table>

DBP, diastolic blood pressure; meds, antihypertensive medication; NR, not reported; SBP, systolic blood pressure; SD, standard deviation. Italics indicate review group calculations.

a. Abstract only.
**TABLE 10  Biofeedback alone versus placebo: change in blood pressure**

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in DBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in SBP at follow-up (mmHg), mean (SD)</th>
<th>Change in DBP at follow-up (mmHg), mean (SD)</th>
<th>Author conclusions</th>
</tr>
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<tbody>
<tr>
<td><strong>Blood pressure biofeedback</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hunyor 1997&lt;sup&gt;17&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory (n = 28)</td>
<td>153 (9)</td>
<td>97 (4)</td>
<td>5 (7.2)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>No difference between treatment and control</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>Laboratory (n = 28)</td>
<td>154 (8)</td>
<td>98 (4)</td>
<td>6 (7.6)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>Tsai 2007&lt;sup&gt;25&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory (n = 20)</td>
<td>148.4 (8.6)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>12.6 (8.8)</td>
<td>NR</td>
<td>Biofeedback treatment superior to placebo (p &lt; 0.001); 3.6–13.5 (CI)</td>
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<tr>
<td></td>
<td>Control</td>
<td>Laboratory (n = 18)</td>
<td>142.1 (5.9)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>4.1 (5.7)</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td><strong>Indirect biofeedback</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Billion 1980&lt;sup&gt;2,36&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory (n = ns)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>No significant difference between groups</td>
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<td>Control</td>
<td>Laboratory (n = ns)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
</tbody>
</table>

CI, confidence interval; DBP, diastolic blood pressure; NR, not reported; ns, not specified; SBP, systolic blood pressure; SD, standard deviation.

<sup>a</sup> Abstract only.
### TABLE 11  Biofeedback versus non-intervention control: blood pressure changes

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre-post treatment (mmHg), mean (SD)</th>
<th>Change in DBP pre-post treatment (mmHg), mean (SD)</th>
<th>Change in SBP at follow-up (mmHg), mean</th>
<th>Change in DBP at follow-up (mmHg), mean</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Friedman 1977, 1978</td>
<td>Treatment Laboratory</td>
<td>13 (n=13)</td>
<td>146.5 (range 130–175) (mean of median)</td>
<td>95.8 (range 85–105) (mean of median)</td>
<td>NC</td>
<td>−4.3 (mean of median)</td>
<td>−7 (1 month) (mean of median)</td>
<td>−4 (1 month), −7.4 (6 months) (mean of median)</td>
<td>Post-treatment 1 month and 6 months: no significant differences for SBP or DBP</td>
</tr>
<tr>
<td></td>
<td>Control (clinic monitor)</td>
<td>Laboratory</td>
<td>139.9 (range 120–170)</td>
<td>94.7 (range 85–105)</td>
<td>NC</td>
<td>−2.9 (mean of median)</td>
<td>−1 (1 month) (n=11)</td>
<td>−2.8 (1 month), −2.9 (6 months) (n=11)</td>
<td></td>
</tr>
<tr>
<td>Goldstein 1982</td>
<td>Treatment Laboratory</td>
<td>12 (n=9)</td>
<td>149.1 (range 130–170)</td>
<td>97.3 (mean of median)</td>
<td>−4.1</td>
<td>−4.4</td>
<td>Graph shows return to baseline at 6 months (n=5)</td>
<td>Graph shows maintenance of effect at 6 months (n=5)</td>
<td>Post-treatment laboratory measures showed no significant difference between biofeedback and self-monitoring for SBP Biofeedback superior to self-monitoring for DBP (p &lt; 0.05)</td>
</tr>
<tr>
<td></td>
<td>Home (n=9)</td>
<td>Home</td>
<td>147.2 (range 120–170)</td>
<td>94.6 (mean of median)</td>
<td>−4.6</td>
<td>−3.2</td>
<td>Graph shows slight increase above baseline at 6 months (n=9)</td>
<td>Graph shows slight increase above baseline at 6 months (n=9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (self-monitor)</td>
<td>Laboratory</td>
<td>141.2 (range 130–170)</td>
<td>94.9 (mean of median)</td>
<td>+3.5</td>
<td>+2.6</td>
<td>Graph shows slight increase above baseline at 6 months (n=11)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Home (n=9)</td>
<td>Home</td>
<td>137 (range 120–170)</td>
<td>93.9 (mean of median)</td>
<td>0</td>
<td>+0.6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nakao 1997</td>
<td>Treatment Clinic</td>
<td>15 (15)</td>
<td>158 (16)</td>
<td>95 (9)</td>
<td>NR</td>
<td>NR</td>
<td>−17 (18) (at 2 weeks)</td>
<td>−8 (7)</td>
<td>Significant differences between biofeedback and control on clinic measures of SBP and DBP (p &lt; 0.05 and p &lt; 0.01 respectively)</td>
</tr>
<tr>
<td></td>
<td>Home (n=15)</td>
<td>Home</td>
<td>133 (11)</td>
<td>85 (9)</td>
<td>NR</td>
<td>NR</td>
<td>−1 (10) (at 2 weeks)</td>
<td>−2 (7)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (self-monitor)</td>
<td>Clinic</td>
<td>161 (21)</td>
<td>94 (6)</td>
<td>NR</td>
<td>NR</td>
<td>+3 (9)</td>
<td>−1 (4)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Home (n=15)</td>
<td>Home</td>
<td>141 (16)</td>
<td>87 (11)</td>
<td>0</td>
<td>−2 (10) (at 2 weeks)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial</td>
<td>Group</td>
<td>Setting/measure</td>
<td>Baseline SBP (mmHg), mean (SD)</td>
<td>Baseline DBP (mmHg), mean (SD)</td>
<td>Change in SBP pre–post treatment (mmHg), mean (SD)</td>
<td>Change in DBP pre–post treatment (mmHg), mean (SD)</td>
<td>Change in SBP at follow-up (mmHg), mean (SD)</td>
<td>Change in DBP at follow-up (mmHg), mean (SD)</td>
<td>Author conclusions</td>
</tr>
<tr>
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<td>--------------------------------</td>
<td>--------------------------------</td>
<td>--------------------------------</td>
<td>--------------------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>Hatch 1985</td>
<td>Treatment</td>
<td>Laboratory (n = 13)</td>
<td>134.5 (12.7)</td>
<td>79.5 (8.5)</td>
<td>-8.9 (at 1 month)</td>
<td>-7.2 (at 1 month)</td>
<td>-6.3 (3 months) (n = 13), +0.1 (12 months) (n = 5)</td>
<td>-6.1 (3 months) (n = 13), -1.7 (12 months) (n = 5)</td>
<td>No significant differences found between groups on any measure or at any time</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Home (n = 11)</td>
<td>132.5 (11.5)</td>
<td>85.7 (10.5)</td>
<td>-0.5 (3 months) (n = 11), +7.2 (12 months) (n = 3)</td>
<td>-0.8 (3 months) (n = 11), +2.3 (12 months) (n = 3)</td>
<td>No significant differences found between groups on any measure or at any time</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (no treatment)</td>
<td>Laboratory (n = 13)</td>
<td>136 (13)</td>
<td>87.7 (4.8)</td>
<td>-6.6 (at 1 month)</td>
<td>-4.7 (at 1 month)</td>
<td>-5.1 (3 months) (n = 13), -10.8 (12 months) (n = 5)</td>
<td>-6.2 (3 months) (n = 13), -5.5 (12 months) (n = 5)</td>
<td>No significant differences found between groups on any measure or at any time</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Home (n = 11)</td>
<td>135 (11)</td>
<td>87 (2.9)</td>
<td>-0.8 (3 months) (n = 11), +7.4 (12 months) (n = 3)</td>
<td>+1.4 (3 months) (n = 11), +4.2 (12 months) (n = 3)</td>
<td>No significant differences found between groups on any measure or at any time</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Indirect biofeedback**

Achmon 1989

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in DBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in SBP at follow-up (mmHg), mean (SD)</th>
<th>Change in DBP at follow-up (mmHg), mean (SD)</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Treatment</td>
<td>Laboratory (n = 27)</td>
<td>155 (13.52)</td>
<td>99.75 (7.14)</td>
<td>-26.55</td>
<td>-15.44</td>
<td>-19.77 (6 months)</td>
<td>-11.68 (6 months)</td>
<td>Post treatment biofeedback significantly different to control for SBP and DBP (p &lt; 0.0005)</td>
</tr>
<tr>
<td></td>
<td>Control (lectures)</td>
<td>Laboratory (n = 20)</td>
<td>155.42 (19.95)</td>
<td>96.12 (6.26)</td>
<td>-3.05</td>
<td>+0.8</td>
<td>NR</td>
<td>NR</td>
<td>Post treatment biofeedback significantly different to control for SBP and DBP (p &lt; 0.0005)</td>
</tr>
</tbody>
</table>

Blanchard 1993

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in DBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in SBP at follow-up (mmHg), mean (SD)</th>
<th>Change in DBP at follow-up (mmHg), mean (SD)</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Treatment</td>
<td>Laboratory (n = 11)</td>
<td>NA</td>
<td>NA</td>
<td>See text</td>
<td>See text</td>
<td>See text</td>
<td>See text</td>
<td>See text</td>
</tr>
<tr>
<td></td>
<td>Control (self-monitor)</td>
<td>Home (n = 12)</td>
<td>NA</td>
<td>NA</td>
<td>See text</td>
<td>See text</td>
<td>NA</td>
<td>NA</td>
<td>No differences between groups</td>
</tr>
</tbody>
</table>

*continued*
### TABLE II Biofeedback versus non-intervention control: blood pressure changes (continued)

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in DBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in SBP at follow-up (mmHg), mean</th>
<th>Change in DBP at follow-up (mmHg), mean</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blanchard 1996&lt;sup&gt;16&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory</td>
<td>142.1 (9.1) 93.2 (54)</td>
<td>−1.2</td>
<td>−1.9</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>No differences between groups</td>
</tr>
<tr>
<td></td>
<td>Control (self-monitor)</td>
<td>Home</td>
<td>140 (14.6) 90.1 (6)</td>
<td>+1.9</td>
<td>+1.1</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Bonso 2005&lt;sup&gt;46&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory</td>
<td>NR</td>
<td>NR</td>
<td>−11</td>
<td>−10</td>
<td>NA</td>
<td>NA</td>
<td>'Clinic blood pressure for treatment group reduced but remained unchanged in control...' (SBP, ( p &lt; 0.018 ); DBP, ( p &lt; 0.001 ))</td>
</tr>
<tr>
<td></td>
<td>Control (self-monitor)</td>
<td>Home</td>
<td>NR</td>
<td>NR</td>
<td>0</td>
<td>0</td>
<td>NA</td>
<td>NA</td>
<td>'Home measures decreased in biofeedback group but not in control...' (( p &lt; 0.001 ))</td>
</tr>
</tbody>
</table>

DBP, diastolic blood pressure; meds, antihypertensive medication; NA, not applicable; NC, not calculated; NR, not reported; ns, not specified; SBP, systolic blood pressure; SD, standard deviation. Italics indicate review group calculations.

<sup>a</sup> Abstract only.
<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean</th>
<th>Change in DBP pre–post treatment (mmHg), mean</th>
<th>Change in SBP at follow-up (mmHg), mean</th>
<th>Change in DBP at follow-up (mmHg), mean</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blanchard 1979&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory (n = 10)</td>
<td>Graph only</td>
<td>Graph only</td>
<td>Graph only</td>
<td>−8.1 (4 months)</td>
<td>−1.9 (4 months)</td>
<td></td>
<td>No significant differences post treatment</td>
</tr>
<tr>
<td></td>
<td>Control (relaxation)</td>
<td>Laboratory (n = 9)</td>
<td>Graph only</td>
<td>Graph only</td>
<td>Graph only</td>
<td>−9.5</td>
<td>−2.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (EMG)</td>
<td>Laboratory (n = 9)</td>
<td>Graph only</td>
<td>Graph only</td>
<td>Graph only</td>
<td></td>
<td></td>
<td>+1.4 (6 months)</td>
<td>+1.2</td>
</tr>
<tr>
<td>Friedman 1978&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory (n = 13)</td>
<td>146.5 (range 130–175) (mean of median)</td>
<td>95.8 (range 85–105) (mean of median)</td>
<td>NC</td>
<td>−4.3 (mean of median)</td>
<td>−4 (1 month), −7.4 (6 months) (n = 12) (mean of median)</td>
<td>−8.2 (1 month), −13.3 (6 months) (mean of median)</td>
<td>Post treatment showed hypnosis to be significantly better than other treatments for DBP and SBP (p &lt; 0.05)</td>
</tr>
<tr>
<td></td>
<td>Control (hypnosis)</td>
<td>Laboratory (n = 13)</td>
<td>142.5 (range 120–195) (mean of median)</td>
<td>93.1 (range 85–105) (mean of median)</td>
<td>NC</td>
<td>−10.1 (1 month), −8.5 (6 months) (mean of median)</td>
<td>−8.0 (1 month), −8.5 (6 months) (mean of median)</td>
<td>−8.2 (mean of median)</td>
<td>At follow-up hypnosis significantly better than biofeedback for DBP; SBP not reported</td>
</tr>
<tr>
<td>Goldstein 1982&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory</td>
<td>149.1</td>
<td>97.3</td>
<td>−4.1</td>
<td>−4.4</td>
<td></td>
<td>Graph only (6 months) (n = 5)</td>
<td>Laboratory measures: no significant post-treatment differences for SBP; biofeedback significantly different to relaxation for DBP (p &lt; 0.05)</td>
</tr>
<tr>
<td></td>
<td>Home</td>
<td>(n = 9)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Graph only (6 months) (n = 5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (relaxation)</td>
<td>Laboratory (n = 9)</td>
<td>147.2</td>
<td>94.6</td>
<td>−4.6</td>
<td>−3.2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Home</td>
<td>(n = 8)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>143</td>
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</table>

Continued.
## TABLE 12  Biofeedback alone versus other behavioural treatments: change in blood pressure (continued)

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean</th>
<th>Change in DBP pre–post treatment (mmHg), mean</th>
<th>Change in SBP at follow-up (mmHg), mean</th>
<th>Change in DBP at follow-up (mmHg), mean</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hager 1978²⁵</td>
<td>Treatment</td>
<td>Home (self-measure) (n = 7)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>No significant differences between groups</td>
</tr>
<tr>
<td>Control</td>
<td>meditation/relaxation (n = 10)</td>
<td>Home (self-measure)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Hatch 1985²⁹</td>
<td>Treatment</td>
<td>Clinic (n = 13)</td>
<td>134.5 (12.7)</td>
<td>79.5 (8.5)</td>
<td>−8.9</td>
<td>−7.2</td>
<td>+0.1 (1 year)</td>
<td>+5.5 (1 year)</td>
<td>No significant differences among the three groups for clinic or home</td>
</tr>
<tr>
<td>Control</td>
<td>relaxation</td>
<td>Home (n = 13)</td>
<td>132.5 (11.5)</td>
<td>85.7 (10.5)</td>
<td>−0.5</td>
<td>−0.8</td>
<td>+7.2 (1 year)</td>
<td>+2.3 (1 year)</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>self-relaxation (n = 13)</td>
<td>Clinic</td>
<td>147.6 (10.6)</td>
<td>83.4 (5.8)</td>
<td>−17.7</td>
<td>−5.8</td>
<td>−18.3 (1 year)</td>
<td>−4.4 (1 year)</td>
<td></td>
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<tr>
<td>Luborsky 1982⁶⁰</td>
<td>Treatment</td>
<td>Laboratory (n = 14): Standing</td>
<td>138.3</td>
<td>93.2</td>
<td>−6.5</td>
<td>−5.5</td>
<td>NR</td>
<td>NR</td>
<td>No significant differences between groups</td>
</tr>
<tr>
<td>Control</td>
<td>relaxation</td>
<td>Laboratory (n = 14): Standing</td>
<td>142.1</td>
<td>98.8</td>
<td>−6.3</td>
<td>−5.4</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>exercise</td>
<td>Laboratory (n = 14): Standing</td>
<td>137.6</td>
<td>101.1</td>
<td>−4.7</td>
<td>−3.0</td>
<td>NR</td>
<td>NR</td>
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</table>
### Table: Blood Pressure Management

<table>
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<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean</th>
<th>Change in DBP pre–post treatment (mmHg), mean</th>
<th>Change in SBP at follow-up (mmHg), mean</th>
<th>Change in DBP at follow-up (mmHg), mean</th>
<th>Author conclusions</th>
</tr>
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<tr>
<td><strong>Indirect biofeedback</strong></td>
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<td></td>
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<td></td>
</tr>
<tr>
<td>Achmon 1989&lt;sup&gt;10&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory (n = 27)</td>
<td>155 (13.52)</td>
<td>99.75 (7.14)</td>
<td>–26.55</td>
<td>–15.44</td>
<td>–19.7 (6 months)</td>
<td>–11.64 (6 months)</td>
<td>At post treatment biofeedback was significantly better than cognitive therapy for SBP and DBP (p &lt; 0.05)</td>
</tr>
<tr>
<td>Control (cognitive therapy)</td>
<td></td>
<td>Laboratory (n = 30)</td>
<td>153.98 (15.27)</td>
<td>98.71 (9.23)</td>
<td>–17.05</td>
<td>–11.40</td>
<td>–11.48</td>
<td>–8.71</td>
<td></td>
</tr>
<tr>
<td>Blanchard 1986&lt;sup&gt;38,40–49&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Clinic (n = 41)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA (1 year)</td>
<td>NA (1 year)</td>
<td>Results...significantly favoured thermal biofeedback both in the short term and long term</td>
</tr>
<tr>
<td>Control (relaxation)</td>
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<td>Home (n = 37)</td>
<td>See text</td>
<td>See text</td>
<td>See text</td>
<td>See text</td>
<td>See text</td>
<td>See text</td>
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</tr>
<tr>
<td>Blanchard 1987&lt;sup&gt;40&lt;/sup&gt;</td>
<td>Treatment (clinic based)</td>
<td>Office (n = 9):</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Home-based treatment unsuccessful compared with clinic-based treatment</td>
</tr>
<tr>
<td>Control (home based)</td>
<td></td>
<td>Standing (n = 9):</td>
<td>134</td>
<td>84</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Supine (n = 9):</td>
<td>135</td>
<td>91</td>
<td>See text</td>
<td>See text</td>
<td>See text</td>
<td>See text</td>
<td></td>
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<tr>
<td>Blanchard 1988&lt;sup&gt;36&lt;/sup&gt; (USA)</td>
<td>Treatment</td>
<td>Laboratory (n = 10)</td>
<td>134.7 (11.2)</td>
<td>94 (5.6)</td>
<td>–4.4</td>
<td>–8.2</td>
<td>–12.7 (6 months)</td>
<td>–10.2 (6 months)</td>
<td>No comparative analysis</td>
</tr>
<tr>
<td>Control (relaxation)</td>
<td></td>
<td>Laboratory (n = 8)</td>
<td>137.4 (6)</td>
<td>95.7 (3.7)</td>
<td>+1.1</td>
<td>–7.8</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>Control (autogenic training)</td>
<td></td>
<td>Laboratory (n = 11)</td>
<td>138.4 (10.1)</td>
<td>96 (3.4)</td>
<td>–4.8</td>
<td>–2.8</td>
<td>–3.8 (6 months)</td>
<td>–6.9 (6 months)</td>
<td></td>
</tr>
</tbody>
</table>

**Continued**
### TABLE 12 Biofeedback alone versus other behavioural treatments: change in blood pressure (continued)

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean</th>
<th>Change in DBP pre–post treatment (mmHg), mean</th>
<th>Change in SBP at follow-up (mmHg), mean</th>
<th>Change in DBP at follow-up (mmHg), mean</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blanchard 1988&lt;sup&gt;36&lt;/sup&gt; (USSR)</td>
<td>Treatment</td>
<td>Laboratory (n = 10)</td>
<td>153.8 (8.6)</td>
<td>100.2 (6.7)</td>
<td>−10.8</td>
<td>−10.7</td>
<td>−13.2 (6 months) (n = 9)</td>
<td>−12.3 (6 months)</td>
<td>No comparative analysis</td>
</tr>
<tr>
<td></td>
<td>Control (relaxation)</td>
<td>Laboratory (n = 10)</td>
<td>149.5 (8.8)</td>
<td>96.7 (5.6)</td>
<td>−6.9</td>
<td>−4.5</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (autogenic training)</td>
<td>Laboratory (n = 10)</td>
<td>154.3 (7.4)</td>
<td>97 (5.1)</td>
<td>−14.7</td>
<td>−7.3</td>
<td>−17 (6 months) (n = 9)</td>
<td>−9.5 (6 months)</td>
<td></td>
</tr>
<tr>
<td>Blanchard 1993&lt;sup&gt;62&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Clinic (n = 11)</td>
<td>120.5 (12.5)</td>
<td>79.7 (8.7)</td>
<td>NA, see text</td>
<td>NA, see text</td>
<td>NA, see text</td>
<td>NA, see text</td>
<td>No significant advantage of biofeedback over other treatment</td>
</tr>
<tr>
<td></td>
<td>Control (EMG)</td>
<td>Clinic (n = 13)</td>
<td>125.3 (11.7)</td>
<td>81.2 (6.8)</td>
<td>NA, see text</td>
<td>NA, see text</td>
<td>NA, see text</td>
<td>NA, see text</td>
<td></td>
</tr>
<tr>
<td>Walsh 1977&lt;sup&gt;37&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory: Medicated (n = 5)</td>
<td>NR</td>
<td>NR</td>
<td>Graph only</td>
<td>−2.4</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (relaxation)</td>
<td>Laboratory: Medicated (n = 7)</td>
<td>NR</td>
<td>NR</td>
<td>Graph only</td>
<td>−1.71</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (relaxation)</td>
<td>Laboratory: Not medicated (n = 6)</td>
<td>NR</td>
<td>NR</td>
<td>+0.83</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Billion 1980&lt;sup&gt;53a&lt;/sup&gt;</td>
<td>Treatment</td>
<td>Laboratory (n = ns)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>Treatment protocols were equally efficacious</td>
</tr>
<tr>
<td></td>
<td>Control (relaxation)</td>
<td>Laboratory (n = ns)</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td>Trial</td>
<td>Group</td>
<td>Setting/measure</td>
<td>Baseline SBP (mmHg), mean (SD)</td>
<td>Baseline DBP (mmHg), mean (SD)</td>
<td>Change in SBP pre–post treatment (mmHg), mean</td>
<td>Change in DBP pre–post treatment (mmHg), mean</td>
<td>Change in SBP at follow-up (mmHg), mean</td>
<td>Change in DBP at follow-up (mmHg), mean</td>
<td>Author conclusions</td>
</tr>
<tr>
<td>---------------</td>
<td>--------------------------------</td>
<td>----------------</td>
<td>-------------------------------</td>
<td>-----------------------------</td>
<td>-----------------------------------</td>
<td>-----------------------------------</td>
<td>-----------------------------------</td>
<td>-----------------------------------</td>
<td>----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Zurawski 1987</td>
<td>Treatment Laboratory (n = 11)</td>
<td></td>
<td>137.89 (19.22)</td>
<td>85.25 (17.10)</td>
<td>–1.62</td>
<td>+4.11</td>
<td>–11.08 (6 months) (n = 8)</td>
<td>–6.06 (6 months) (n = 8)</td>
<td>At post treatment stress management training significantly better than biofeedback for DBP (p &lt; 0.01); stress management training superiority for SBP approached significance</td>
</tr>
<tr>
<td></td>
<td>Control Laboratory (n = 14)</td>
<td></td>
<td>137.07 (16.22)</td>
<td>87.14 (16.71)</td>
<td>–9.19</td>
<td>–7.8</td>
<td>–6.06 (6 months) (n = 8)</td>
<td>–7.8 (6 months)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(stress management training)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thananopavarn 1979</td>
<td>Treatment Laboratory (n = 5)</td>
<td></td>
<td>155 (6)</td>
<td>96 (4)</td>
<td>–12.0</td>
<td>–7</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Home (n = 5)</td>
<td></td>
<td>159 (7)</td>
<td>94 (3)</td>
<td>–13.0</td>
<td>–6</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control Laboratory (n = 3)</td>
<td></td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Home (n = 3)</td>
<td></td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td>NR</td>
<td></td>
</tr>
</tbody>
</table>

**Type of biofeedback not stated**

DBP diastolic blood pressure; EMG, electromyographic; NA, not applicable; NC, not calculated; NR, not reported; ns, not specified; SBP, systolic blood pressure; SD, standard deviation. Italics indicate review group calculations. a Abstract only.
### TABLE 13 Biofeedback combination versus placebo: change in blood pressure

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in DBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in SBP at follow-up (mmHg), mean (SD)</th>
<th>Change in DBP at follow-up (mmHg), mean (SD)</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood pressure and indirect biofeedback</strong></td>
<td>Frankel 1978	extsuperscript{II}</td>
<td>Treatment (+ relaxation) Laboratory (n = 7): Supine</td>
<td>148 (4.9)</td>
<td>95 (1.9)</td>
<td>+3</td>
<td>+1</td>
<td>NA</td>
<td>NA</td>
<td>No differences between groups</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Standing</td>
<td>147 (6.0)</td>
<td>102 (2.6)</td>
<td>+2</td>
<td>–1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (placebo)</td>
<td>Laboratory (n = 7): Supine</td>
<td>150 (7.6)</td>
<td>95 (1.9)</td>
<td>–1</td>
<td>–2</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Standing</td>
<td>150 (9.8)</td>
<td>102 (1.9)</td>
<td>–1</td>
<td>+1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

DBP, diastolic blood pressure; NA, not applicable; SBP, systolic blood pressure; SD, standard deviation.
Biofeedback combinations versus non-intervention control

The evidence for the effectiveness of biofeedback compared with a non-intervention control is equivocal (Table 14). Of the 13 trials (n ≥ 558), five19,40,44,46,59 reported a significant benefit for biofeedback treatment over control. The McCraty et al.44 trial reported on SBP only. Five other trials21,22,38,49,51 reported no significant differences between groups. Two trials22,53 did not present comparisons between group outcomes. No data were reported for the Berglund52 trial although significant support for the effectiveness of the biofeedback combination was noted. Long-term efficacy was reported only by Patel and Marmot40 at 1 year for both SBP and DBP.

Biofeedback combinations versus other behavioural treatments

Eight trials (n ≥ 408 approximately) compared biofeedback combinations with another behavioural treatment (Table 15). Of these, Patel and North45 reported a significant difference between biofeedback treatment and relaxation for both SBP and DBP. No data were reported for Khramelashvili et al.,59 although the abstract stated that blood pressure decline was significantly more marked in the treatment groups than in the control groups. Five other trials22,38,42,43,49 found no significant effects of biofeedback treatment. One trial50 did not report comparative data. Results at 12 months from the Patel and North45 trial showed that biofeedback treatment combined with yoga continued to be more effective than relaxation.

Summary of results

Table 16 summarises the foregoing results.

Summary of data beyond 6 months

Of the 15 trials reporting outcomes beyond 6 months, only eight had any usable data. These trials are summarised in Table 17.
<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in DBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in SBP at follow-up (mmHg), mean (SD)</th>
<th>Change in DBP at follow-up (mmHg), mean (SD)</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Biofeedback</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Friedman 1978</td>
<td>Treatment (+ hypnosis)</td>
<td>Laboratory</td>
<td>139.8 (range 117–180) (mean of median)</td>
<td>91.8 (range 85–105) (mean of median)</td>
<td>NC</td>
<td>-2.8 (mean of median)</td>
<td>NC</td>
<td>-2.5 (1 month), -4.0 (6 months) (mean of median)</td>
<td>No significant differences between groups at any time point</td>
</tr>
<tr>
<td></td>
<td>Control (clinic monitor)</td>
<td></td>
<td>139.9 (range 120–170) (mean of median)</td>
<td>94.7 (range 85–105) (mean of median)</td>
<td>NC</td>
<td>-2.9</td>
<td>-1 (1 month)</td>
<td>-2.8 (1 month), -2.9 (6 months) (mean of median)</td>
<td></td>
</tr>
<tr>
<td><strong>Blood pressure and indirect biofeedback</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Average blood pressure did not change significantly for any group</td>
</tr>
<tr>
<td>Frankel 1978</td>
<td>Treatment (+ relaxation)</td>
<td>Clinic (n = 7):</td>
<td>Supine 148 (4.9)</td>
<td>95 (1.9)</td>
<td>+3</td>
<td>+1</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Standing 147 (6.0)</td>
<td>102 (2.6)</td>
<td>+2</td>
<td>-1</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Control (clinic monitor)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Berglund 1991</td>
<td>Treatment (Menninger)</td>
<td>Laboratory (n = ns)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>Significant support for the effectiveness of the Menninger treatment</td>
</tr>
<tr>
<td></td>
<td>Control (self-monitor)</td>
<td>Home (n = ns)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Trial</td>
<td>Group</td>
<td>Setting/measure</td>
<td>Baseline SBP (mmHg), mean (SD)</td>
<td>Baseline DBP (mmHg), mean (SD)</td>
<td>Change in SBP pre–post treatment (mmHg), mean (SD)</td>
<td>Change in DBP pre–post treatment (mmHg), mean (SD)</td>
<td>Change in SBP at follow-up (mmHg), mean (SD)</td>
<td>Change in DBP at follow-up (mmHg), mean (SD)</td>
<td>Author conclusions</td>
</tr>
<tr>
<td>---------</td>
<td>-----------------------------------------------------------------------</td>
<td>-----------------</td>
<td>---------------------------------</td>
<td>---------------------------------</td>
<td>--------------------------------------------------</td>
<td>--------------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Canino 1994&lt;sup&gt;44&lt;/sup&gt;</td>
<td>Treatment (+ relaxation and anxiety management)</td>
<td>Laboratory</td>
<td>147</td>
<td>96</td>
<td>-13</td>
<td>-12</td>
<td>-10 (6 months)</td>
<td>-8 (6 months)</td>
<td>Post-treatment differences between treatment and no intervention for SBP and DBP (p &lt; 0.05 and p &lt; 0.001 respectively)</td>
</tr>
<tr>
<td></td>
<td>Control (no treatment)</td>
<td>Laboratory</td>
<td>145</td>
<td>97</td>
<td>0</td>
<td>-0.1</td>
<td>NA</td>
<td>NA</td>
<td>No follow-up data for controls</td>
</tr>
<tr>
<td></td>
<td>Control (behavioural placebo treatment)</td>
<td>Laboratory</td>
<td>156</td>
<td>97</td>
<td>-7</td>
<td>-1</td>
<td>-6 (2.5 months)</td>
<td>-1 (2.5 months)</td>
<td>No follow-up data for controls</td>
</tr>
<tr>
<td>Chesney 1987&lt;sup&gt;45&lt;/sup&gt;</td>
<td>Treatment (+ relaxation)</td>
<td>Clinic</td>
<td>137.6</td>
<td>94.4</td>
<td>NR</td>
<td>NR</td>
<td>-5.5 (54 weeks)</td>
<td>-4.2 (54 weeks)</td>
<td>No difference between the behavioural groups (as a whole) and the monitoring group</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Worksite</td>
<td>138.8</td>
<td>98.4</td>
<td>NR</td>
<td>NR</td>
<td>-1.4 (54 weeks)</td>
<td>-5.2 (54 weeks)</td>
<td>No difference between the behavioural groups (as a whole) and the monitoring group</td>
</tr>
<tr>
<td></td>
<td>Treatment (+ relaxation and cognitive restructuring)</td>
<td>Clinic</td>
<td>138.9</td>
<td>94.4</td>
<td>NR</td>
<td>NR</td>
<td>-8.5 (54 weeks)</td>
<td>-1.7 (54 weeks)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Worksite</td>
<td>143</td>
<td>98.1</td>
<td>NR</td>
<td>NR</td>
<td>-10.5 (54 weeks)</td>
<td>-6.9 (54 weeks)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (clinic monitor)</td>
<td>Clinic</td>
<td>139.1</td>
<td>94.3</td>
<td>NR</td>
<td>NR</td>
<td>-11.5 (54 weeks)</td>
<td>-6.1 (54 weeks)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Worksite</td>
<td>136.9</td>
<td>95.1</td>
<td>NR</td>
<td>NR</td>
<td>-2.4 (54 weeks)</td>
<td>-3.5 (54 weeks)</td>
<td></td>
</tr>
<tr>
<td>Cohen 1983&lt;sup&gt;46&lt;/sup&gt;</td>
<td>Treatment (+ relaxation)</td>
<td>Clinic</td>
<td>144</td>
<td>95</td>
<td>-13</td>
<td>-12</td>
<td>Graph only</td>
<td>Graph only</td>
<td>Blood pressure not primary outcome. Unable to determine whether there are differences between groups on blood pressure measures</td>
</tr>
<tr>
<td></td>
<td>Control (blood pressure monitor)</td>
<td>Home</td>
<td>143</td>
<td>97</td>
<td>Graph only</td>
<td>Graph only</td>
<td>Graph only</td>
<td>Graph only</td>
<td></td>
</tr>
</tbody>
</table>

*continued*
### TABLE 14 Biofeedback combinations versus non-intervention control: blood pressure changes (continued)

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in DBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in SBP at follow-up (mmHg), mean (SD)</th>
<th>Change in DBP at follow-up (mmHg), mean (SD)</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hafner 1982</td>
<td>Treatment (+ relaxation and meditation)</td>
<td>Laboratory (n = 7)</td>
<td>160 106.6</td>
<td></td>
<td>-21.6</td>
<td>-15.1</td>
<td>-20.8 (3 months) (n = 7)</td>
<td>-14.7 (3 months) (n = 7)</td>
<td>No significant effects of biofeedback at post treatment or at follow-up</td>
</tr>
<tr>
<td></td>
<td>Control (no treatment)</td>
<td>Laboratory (n = 7)</td>
<td>159.1 98.3</td>
<td></td>
<td>NS</td>
<td>NS</td>
<td>-8.6 (3 months) (n = 7)</td>
<td>-2 (3 months)</td>
<td></td>
</tr>
<tr>
<td>Jurek 1992</td>
<td>Treatment (+ relaxation)</td>
<td>Laboratory (n = 20)</td>
<td>132.2 (14.6) 89.4 (5.7)</td>
<td>-1.3</td>
<td>-3.9</td>
<td>-1.5 (10 months) (n = 16)</td>
<td>-4 (10 months) (n = 16)</td>
<td>No differences between two groups</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (diuretic only)</td>
<td>Laboratory (n = 10)</td>
<td>134.2 (8.3) 92 (5.2)</td>
<td>+4.0</td>
<td>-1.4</td>
<td>NA</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Khramelashvili 1986</td>
<td>Treatment (+ relaxation)</td>
<td>NS (n = 30)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>'Blood pressure decline significantly more marked in the treatment groups as compared to controls'</td>
</tr>
<tr>
<td></td>
<td>Control (no treatment)</td>
<td>NS (n = 20)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>McCratty 2003</td>
<td>Treatment (+ IQM)</td>
<td>Worksite (n = 18)</td>
<td>130.4 (11.1) 82.9 (10.2)</td>
<td>NA</td>
<td>NA</td>
<td>-9 (3) (SEM); adjusted change -10.6 (2.1) (SEM) (3 months)</td>
<td>-5.5 (2.3) (SEM); adjusted change -6.3 (1.2) (SEM) (3 months)</td>
<td>A significant reduction in SBP in the treatment group compared with control (p &lt; 0.05)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (no treatment)</td>
<td>Worksite (n = 14)</td>
<td>128.1 (8) 84.1 (7.6)</td>
<td>NA</td>
<td>NA</td>
<td>-5.7 (3.1) (SEM); adjusted change -3.7 (2.4) (SEM) (3 months)</td>
<td>-4.9 (2.3) (SEM); adjusted change -3.9 (1.4) (SEM) (3 months)</td>
<td>No significant difference in DBP</td>
<td></td>
</tr>
<tr>
<td>Trial</td>
<td>Group</td>
<td>Setting/measure</td>
<td>Baseline SBP (mmHg), mean (SD)</td>
<td>Baseline DBP (mmHg), mean (SD)</td>
<td>Change in SBP pre–post treatment (mmHg), mean (SD)</td>
<td>Change in DBP pre–post treatment (mmHg), mean (SD)</td>
<td>Change in SBP at follow-up (mmHg), mean (SD)</td>
<td>Change in DBP at follow-up (mmHg), mean (SD)</td>
<td>Author conclusions</td>
</tr>
<tr>
<td>-----------</td>
<td>---------------------------------</td>
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<td>-----------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>McGrady 1981&lt;sup&gt;19&lt;/sup&gt;</td>
<td>Treatment (+ relaxation)</td>
<td>Laboratory (n = 22)</td>
<td>144.41 (19.83)</td>
<td>90.59 (10.47)</td>
<td>-11.23</td>
<td>-5.68</td>
<td>NA</td>
<td>NA</td>
<td>Significant difference between biofeedback and control for SBP and DBP (&lt; 0.02 and &lt; 0.004 respectively)</td>
</tr>
<tr>
<td></td>
<td>Control (clinic monitor)</td>
<td>Laboratory (n = 16)</td>
<td>140.67 (19.36)</td>
<td>90.94 (11.74)</td>
<td>-1.42</td>
<td>-6.3</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>McGrady 1994&lt;sup&gt;3&lt;/sup&gt;</td>
<td>Treatment (+ relaxation)</td>
<td>Laboratory (n = 70)</td>
<td>132.4 (12.6)</td>
<td>85.8 (8.6)</td>
<td>-5.9</td>
<td>-3.2</td>
<td>-2.6 (10 months) (n = 36)</td>
<td>0.7 (10 months) (n = 36)</td>
<td>No comparison between groups</td>
</tr>
<tr>
<td></td>
<td>Control (no treatment)</td>
<td>Laboratory (n = 70)</td>
<td>130.9 (11.2)</td>
<td>85.6 (9.8)</td>
<td>-0.9</td>
<td>+1</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Patel 1988&lt;sup&gt;4,6&lt;/sup&gt;</td>
<td>Treatment (+ relaxation)</td>
<td>Laboratory (n = 49)</td>
<td>144.9 (14.68)</td>
<td>88.6 (7.50)</td>
<td>NA</td>
<td>NA</td>
<td>-4.9 (1 year) (n = 49)</td>
<td>-1.5 (1 year) (n = 49)</td>
<td>Significant differences between biofeedback and control for SBP and DBP (&lt; 0.0001 and p &lt; 0.015 respectively)</td>
</tr>
<tr>
<td></td>
<td>Control (no treatment)</td>
<td>Laboratory (n = 54)</td>
<td>135.7 (16.44)</td>
<td>85.1 (9.67)</td>
<td>NA</td>
<td>NA</td>
<td>+7.1 (n = 54)</td>
<td>+2.6 (n = 54)</td>
<td>After adjusting for blood pressure at entry there was a significant decrease in SBP, but not DBP</td>
</tr>
</tbody>
</table>

DBP, diastolic blood pressure; IQR, inner quality management NA, not applicable; NC, not calculated; NR, not reported; ns, not stated; SBP, systolic blood pressure; SD, standard deviation; SEM, standard error of the mean.

Italics indicate review group calculations.

a Abstract only.
### TABLE 15  
**Biofeedback combinations versus other behavioural treatments: change in blood pressure**

<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in DBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in SBP at follow-up (mmHg), mean (SD)</th>
<th>Change in DBP at follow-up (mmHg), mean (SD)</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood pressure biofeedback</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Friedman 1978a</td>
<td>Treatment (+ hypnosis)</td>
<td>Laboratory</td>
<td>139.8 (range 117–180) (mean of median)</td>
<td>91.8 (range 85–105) (mean of median)</td>
<td>-2.8 (mean of median)</td>
<td>-2.5 (1 month) (mean of median)</td>
<td>-3.3 (1 month), -4.0 (6 months) (n = 10) (mean of median)</td>
<td>-3.3 (1 month), -4.0 (6 months) (mean of median)</td>
<td>No significant effects of biofeedback treatment</td>
</tr>
<tr>
<td></td>
<td>Control (hypnosis)</td>
<td>Laboratory</td>
<td>142.5 (range 120–195) (mean of median)</td>
<td>93.1 (range 85–105) (mean of median)</td>
<td>NC</td>
<td>-8.2 (mean of median)</td>
<td>-10.1 (1 month) (mean of median)</td>
<td>-8.0 (1 month), -8.5 (6 months) (mean of median)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (biofeedback alone)</td>
<td>Laboratory</td>
<td>146.5 (range 130–175) (mean of median)</td>
<td>95.8 (range 85–105) (mean of median)</td>
<td>NC</td>
<td>-4.3 (mean of median)</td>
<td>-7.0 (1 month) (mean of median)</td>
<td>-4.0 (1 month), -7.4 (6 months) (mean of median)</td>
<td></td>
</tr>
<tr>
<td><strong>Indirect biofeedback</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chesney 1987m</td>
<td>Treatment (+ relaxation)</td>
<td>Clinic</td>
<td>137.6 94.4 NA NA</td>
<td>138.8 98.4</td>
<td>-5.5 (54 weeks)</td>
<td>-4.2 (54 weeks)</td>
<td>Blood pressure does not appear to show any differences</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Worksite</td>
<td>(n = 24)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Treatment (+ relaxation and cognitive restructuring)</td>
<td>Clinic</td>
<td>138.9 94.2 NA NA</td>
<td>143.0 98.1</td>
<td>-8.5 (54 weeks)</td>
<td>-1.7 (54 weeks)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Worksite</td>
<td>(n = 25)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (relaxation)</td>
<td>Clinic</td>
<td>139.2 95.3 NA NA</td>
<td>141.3 95.6</td>
<td>-9.8 (54 weeks)</td>
<td>-6.9 (54 weeks)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Worksite</td>
<td>(n = 24)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (relaxation + cognitive restructuring)</td>
<td>Clinic</td>
<td>136.8 95.6 NA NA</td>
<td>139.2 98</td>
<td>-12.2 (54 weeks)</td>
<td>-6.9 (54 weeks)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Worksite</td>
<td>(n = 24)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (health behaviour)</td>
<td>Clinic</td>
<td>136.1 94.7 NA NA</td>
<td>138.4 96.1</td>
<td>-7.5 (54 weeks)</td>
<td>-6.8 (54 weeks)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial</td>
<td>Group</td>
<td>Setting/measure</td>
<td>Baseline SBP (mmHg), mean (SD)</td>
<td>Baseline DBP (mmHg), mean (SD)</td>
<td>Change in SBP pre–post treatment (mmHg), mean (SD)</td>
<td>Change in DBP pre–post treatment (mmHg), mean (SD)</td>
<td>Change in SBP at follow–up (mmHg), mean (SD)</td>
<td>Change in DBP at follow–up (mmHg), mean (SD)</td>
<td>Author conclusions</td>
</tr>
<tr>
<td>---------------</td>
<td>------------------------------------</td>
<td>-----------------------------</td>
<td>---------------------------------</td>
<td>--------------------------------</td>
<td>---------------------------------------------------</td>
<td>---------------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Cohen 1983</td>
<td>Treatment (+ relaxation)</td>
<td>Clinic (n = 10)</td>
<td>144 (95)</td>
<td>95 (0)</td>
<td>-13 (-12)</td>
<td>Graph only</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>Blood pressure not primary outcome. Unable to determine differences between groups on blood pressure measures</td>
</tr>
<tr>
<td></td>
<td>Control (relaxation)</td>
<td>Clinic (n = 10)</td>
<td>143 (94)</td>
<td>94 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td></td>
</tr>
<tr>
<td>Hafner 1982</td>
<td>Treatment (+ relaxation and meditation)</td>
<td>Laboratory (n = 7)</td>
<td>160 (106.6)</td>
<td>106.6 (21.6)</td>
<td>-21.6 (-15.1)</td>
<td>-20.8 (14.7)</td>
<td>-8.6 (2)</td>
<td>-2 (2)</td>
<td>No significant effects at either post treatment or follow–up</td>
</tr>
<tr>
<td></td>
<td>Control (meditation)</td>
<td>Laboratory (n = unclear)</td>
<td>145.5 (102.5)</td>
<td>NR (NR)</td>
<td>NR (NR)</td>
<td>-8.6 (3 months)</td>
<td>-2 (3 months)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Khramelashvili 1986</td>
<td>Treatment (+ relaxation)</td>
<td>NS (n = 30)</td>
<td>NS (NS)</td>
<td>NS (NS)</td>
<td>NS (NS)</td>
<td>NS (NS)</td>
<td>NS (NS)</td>
<td>Blood pressure decline significantly more marked in the treatment groups as compared to controls’</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (no treatment)</td>
<td>NS (n = 20)</td>
<td>NS (NS)</td>
<td>NS (NS)</td>
<td>NS (NS)</td>
<td>NS (NS)</td>
<td>NS (NS)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patel 1975</td>
<td>Treatment (+ yoga)</td>
<td>Laboratory (n = 17)</td>
<td>167 (23.6)</td>
<td>99.6 (9.3)</td>
<td>NA (NA)</td>
<td>-26.1 (16.5)</td>
<td>-15.2 (8.1)</td>
<td></td>
<td>Significant differences in the biofeedback group for SBP and DBP (p &lt; 0.005 and p &lt; 0.001 respectively)</td>
</tr>
<tr>
<td></td>
<td>Control (relaxation)</td>
<td>Laboratory (n = 17)</td>
<td>168.9 (20)</td>
<td>100.6 (11.4)</td>
<td>NA (NA)</td>
<td>-8.9 (14.5)</td>
<td>-4.2 (5.9)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Irvine 1991</td>
<td>Treatment (+ relaxation)</td>
<td>Worksite (n = 50)</td>
<td>137.3 (8.4)</td>
<td>94.1 (2.8)</td>
<td>-5.6 (-5.1); 3.7–7.5 (CI)</td>
<td>-5.1 (4.9); 3.7–6.5 (CI)</td>
<td>-7. (6.6); 5.5–9.3 (CI) (6 months) (n = 47)</td>
<td>-6.5 (3.8); 5.4–7.6 (CI) (6 months) (n = 47)</td>
<td>No significant differences between groups at post treatment or at follow–up</td>
</tr>
<tr>
<td></td>
<td>Control (NSST)</td>
<td>Worksite (n = 51)</td>
<td>136.4 (7.4)</td>
<td>93.6 (3)</td>
<td>-5.8 (4.2); 3.8–7.8 (CI)</td>
<td>-4.2 (4.8); 2.8–5.6 (CI)</td>
<td>-5.3 (7.6); 3.1–7.5 (CI) (6 months) (n = 51)</td>
<td>-4.9 (4.8); 3.5–6.3 (CI) (6 months) (n = 51)</td>
<td></td>
</tr>
</tbody>
</table>

continued
<table>
<thead>
<tr>
<th>Trial</th>
<th>Group</th>
<th>Setting/measure</th>
<th>Baseline SBP (mmHg), mean (SD)</th>
<th>Baseline DBP (mmHg), mean (SD)</th>
<th>Change in SBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in DBP pre–post treatment (mmHg), mean (SD)</th>
<th>Change in SBP at follow-up (mmHg), mean (SD)</th>
<th>Change in DBP at follow-up (mmHg), mean (SD)</th>
<th>Author conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jacob</td>
<td>Treatment (+ relaxation)</td>
<td>Therapist</td>
<td>133.1</td>
<td>89.7</td>
<td>−2.2 (2.0 SE)</td>
<td>−3.1 (1.4 SE)</td>
<td>NA</td>
<td>NA</td>
<td>No significant differences between groups on any measure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clinic (sup)</td>
<td>141.7</td>
<td>88.1</td>
<td>+2.2 (3.1 SE)</td>
<td>+5.1 (1.8 SE)</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clinic (st)</td>
<td>132.0</td>
<td>85.2</td>
<td>+1.2 (3.6 SE)</td>
<td>+3.8 (2.9 SE)</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>ABPM</td>
<td>122.2</td>
<td>84.7</td>
<td>+3.8 (3.8 SE)</td>
<td>+4.9 (2.4 SE)</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(n = 10)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Control (stress education)</td>
<td>Therapist</td>
<td>125.9</td>
<td>89.0</td>
<td>−4.6 (2.1 SE)</td>
<td>−3.2 (1.5 SE)</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clinic (sup)</td>
<td>130.6</td>
<td>83.1</td>
<td>−0.3 (3.3 SE)</td>
<td>+0.6 (1.9 SE)</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clinic (st)</td>
<td>126.0</td>
<td>84.7</td>
<td>−2.7 (3.8 SE)</td>
<td>−1.4 (3.0 SE)</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>ABPM</td>
<td>119.2</td>
<td>80.7</td>
<td>−3.7 (4.3 SE)</td>
<td>−3.1 (2.7 SE)</td>
<td>NA</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(n = 9)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ABPM, ambulatory blood pressure monitor; CI, confidence interval; DBP, diastolic blood pressure; IQM, inner quality management; NA, not applicable; NC, not calculated; NR, not reported; NS, not stated; NSST, non-specific support therapy; SBP, systolic blood pressure; SE, standard error; st, standing; sup, supine.

* Italics indicate review group calculations.

a Abstract only.
## TABLE 16 Summary of results

<table>
<thead>
<tr>
<th>Trial type</th>
<th>Number of RCTs</th>
<th>Dates</th>
<th>Combined sample size</th>
<th>Direct/indirect biofeedback</th>
<th>Summary of results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biofeedback alone vs antihypertensive medication</td>
<td>3</td>
<td>1979–82</td>
<td>51</td>
<td>2 direct, 1 indirect</td>
<td>1 trial favored biofeedback, 2 trials favored medication</td>
</tr>
<tr>
<td>Biofeedback alone vs placebo</td>
<td>3</td>
<td>1980–2007</td>
<td>123 (estimate)</td>
<td>2 direct, 1 indirect</td>
<td>1 trial favored biofeedback, 2 trials found no difference</td>
</tr>
<tr>
<td>Biofeedback alone vs no intervention</td>
<td>8</td>
<td>1977–2005</td>
<td>235 (estimate)</td>
<td>4 direct, 1 ns</td>
<td>3 trials favored biofeedback, 1 trial favored biofeedback only for DBP, 4 trials found no difference</td>
</tr>
<tr>
<td>Biofeedback alone vs other behavioural treatments</td>
<td>16</td>
<td>1977–93</td>
<td>465 (estimate)</td>
<td>6 direct, 9 indirect</td>
<td>3 trials favored biofeedback, 7 trials found no difference, 2 trials found other interventions superior, 4 trials did not report comparative data</td>
</tr>
<tr>
<td>Biofeedback combination vs placebo</td>
<td>1</td>
<td>1978</td>
<td>22</td>
<td>1 direct</td>
<td>1 trial found no difference</td>
</tr>
<tr>
<td>Biofeedback combination vs no treatment</td>
<td>13</td>
<td>1978–2003</td>
<td>558 (estimate)</td>
<td>1 direct, 1 direct and indirect</td>
<td>5 trials favored biofeedback, 5 trials reported no difference, 2 trials did not compare groups, 1 trial did not report data</td>
</tr>
<tr>
<td>Biofeedback combination vs other behavioural treatments</td>
<td>8</td>
<td>1978–2003</td>
<td>378 (estimate)</td>
<td>1 direct, 7 indirect</td>
<td>2 trials favored biofeedback, 5 trials reported no difference, 1 trial did not report comparative data</td>
</tr>
</tbody>
</table>

ns, not stated; RCTs, randomised controlled trials

Note that the number of RCTs and patients in this table is greater than the quoted overall total as some trials include more than one comparator.
<table>
<thead>
<tr>
<th>Trial</th>
<th>Biofeedback type</th>
<th>Comparator</th>
<th>Outcome at follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Achmon 1989</td>
<td>Alone</td>
<td>Cognitive behavioural therapy</td>
<td>Biofeedback superior to cognitive behavioural therapy for SBP only</td>
</tr>
<tr>
<td>Friedman 1978</td>
<td>Alone</td>
<td>BPM</td>
<td>No significant differences</td>
</tr>
<tr>
<td>Friedman 1978</td>
<td>Alone</td>
<td>Hypnosis</td>
<td>Hypnosis significantly better</td>
</tr>
<tr>
<td>Friedman 1978</td>
<td>Combined</td>
<td>Hypnosis</td>
<td>No significant effects of intervention</td>
</tr>
<tr>
<td>Friedman 1978</td>
<td>Combined</td>
<td>Combined</td>
<td>No significant effects of intervention</td>
</tr>
<tr>
<td>Hatch 1985</td>
<td>Alone</td>
<td>Relaxation</td>
<td>No significant differences</td>
</tr>
<tr>
<td>Zurawski 1987</td>
<td>Alone</td>
<td>Stress management training</td>
<td>No significant differences</td>
</tr>
<tr>
<td>Chesney 1987</td>
<td>Combined</td>
<td>Range of therapies</td>
<td>No significant differences</td>
</tr>
<tr>
<td>Chesney 1987</td>
<td>Combined</td>
<td>BPM</td>
<td>No significant differences between behavioural group as a whole and control</td>
</tr>
<tr>
<td>Irvine 1991</td>
<td>Combined</td>
<td>Non-specific support treatment</td>
<td>No significant differences</td>
</tr>
<tr>
<td>Jurek 1992</td>
<td>Combined</td>
<td>No treatment</td>
<td>No significant differences</td>
</tr>
<tr>
<td>Patel 1988</td>
<td>Combined</td>
<td>No treatment</td>
<td>After adjusting for blood pressure at entry there was a significantly greater lowering of SBP than DBP</td>
</tr>
</tbody>
</table>

BPM, blood pressure monitoring; DBP, diastolic blood pressure; SBP, systolic blood pressure.
Chapter 4
Discussion

The objective of this report was to assess the evidence for the long-term effectiveness of biofeedback procedures in treating adults with essential hypertension. Other objectives were to model the cost-effectiveness of the use of biofeedback for the treatment of essential hypertension, summarise information on currently used biofeedback equipment and identify any leading technologies that could be used in a future clinical trial.

The review included 36 small RCTs of ≥ 1660 patients. These included two treatment designs, those that exclusively employed biofeedback and those that used biofeedback with an adjunctive therapy. A number of biofeedback modalities were used and the number of training sessions varied across trials. Patients were described as mildly hypertensive, borderline hypertensive or just hypertensive. There were trials that included patients taking antihypertensive drugs, others with patients not taking antihypertensive drugs and others with a mixture of patients taking these medications. Thus, a range of interventions, biofeedback protocols and outcome measures were reported. This heterogeneity, combined with the poor quality of reporting, indicated that statistical analysis of the results would be inappropriate. No trials reporting long-term (> 12 months) outcomes were identified for inclusion in the review. Of the 15 trials reporting outcomes beyond 6 months, only eight had any usable data.

We assessed the level of evidence in relation to the effectiveness of biofeedback compared with antihypertensive drug therapy, placebo, no intervention and other behavioural therapies using trial author conclusions. Trial results were variable and conflicting and the small numbers involved makes generalisation of results questionable. No short- or long-term benefits of biofeedback in relation to moderation of hypertension were demonstrated. The measurement of blood pressure is not an exact science, with variations noted in relation to the person taking the reading and the equipment. When benefits were shown they were within the standard error of reproducibility of blood pressure measurement and may therefore have arisen by chance. This lack of demonstrated benefit precluded a need to assess the cost-effectiveness of the intervention.

Although we were unable to identify any particular treatment as promising this report does provide a partial list of currently available biofeedback equipment.

Our findings differ somewhat to those of the two previous reviews, which reported more positive findings. We have discussed the problems inherent in the meta-analysis from these two reviews and that they should therefore be considered with extreme caution. Treatment interventions differed across studies, as did the comparators and the time of measurement of outcomes. Both authors reported a need to estimate standard deviations and standard errors from data presented in the included trials to allow meta-analysis to be conducted. One of the authors later reported the problems inherent in the meta-analysis process that was used.

The meta-analysis of Nakao et al. reported biofeedback to be more effective than non-intervention controls, but only superior to sham or non-specific behavioural interventions when combined with relaxation. The second review excluded from quantitative analysis trials that reported no measure of variability. This review also reported that both biofeedback and active treatments could produce small reductions in blood pressure, but that only biofeedback combined with adjunctive therapy was superior to no intervention. Of interest is that even though these meta-analyses reported statistical significance in a few instances they do not consistently achieve the clinically significant levels of 5–6 mmHg that has been shown to reduce the incidence of CVD events (e.g. acute myocardial infarction and stroke).

A factor brought out in the review by Nakao et al. and also mentioned by one of our advisory panel is the impact of pretreatment blood pressures of the patients involved in biofeedback trials. Patients entering a trial with pretreatment grade 2 or grade 3 hypertension (> 150 mm Hg) were shown in the Nakao et al. review to have demonstrated greater overall decreases in systolic blood pressure.
However, the number of patients in these trials is small (approximately 130) and mean blood pressure readings for all trial participants were used in the analysis. Therefore, it is difficult to differentiate the actual effect in this subgroup of patients.

It is likely that many of the trials included in the review reported here were insufficiently powered to detect differences between treatment groups. Overall, the trial sizes were small and only four of the 36 trials included provided a sample size calculation. Although combining data from several small trials would increase our ability to assess the effectiveness of the intervention, as stated earlier, given the lack of trial quality and the variation in interventions and outcome reporting, we were unable to justify carrying out such an analysis. These difficulties have also been noted by other reviewers. We did not go beyond the data presented in the published papers and relied upon authors’ conclusions related to the effectiveness of the biofeedback interventions. In some cases, when statistical comparisons between groups were not presented in the published report, no results were reported for these trials.

Other issues emerged during the compilation of this review, many of which have been reported previously. To demonstrate effectiveness there is a need for trials of longer duration. Such trials would need to address the issue of the white coat effect by including blood pressure measures taken outside of the laboratory/clinic environment. There is also a need to provide a more rounded picture of blood pressure readings in different circumstances. This might be achieved through the use of ABPM or patient self-monitoring at home. It has also been suggested that endpoints beyond blood pressure changes should be assessed, and these might include effects of treatment on end-organ damage. In addition, changes in technology could be integrated in any future research. For example, advice from the AAPB (Robert Crago, 2007, personal communication) indicates that ‘...heart rate variability training – the heart math product – is currently being investigated.’
Chapter 5

Research recommendations

Of major concern is the poor quality of existing trials. Any proposed future trials need to address the major design weaknesses highlighted in this and previous reviews. That is, they need to be suitably powered to detect meaningful (clinically significant not just statistically significant) differences between treatment groups, randomise patients to groups using robust techniques, employ credible placebo treatments and ensure that adequate blinding procedures are in place. Patient attrition must be adequately reported and dealt with in any final analyses. In addition, researchers need to adequately report the details of the intervention and ensure that participants are appropriately trained in the biofeedback technique. Issues of patient subgroups also need to be addressed, for example patients at the upper end of the hypertension scale, older patients and patients from varied ethnic backgrounds.

Although researchers in the area will be disappointed in the results of this review, the poor quality of the currently available research, the diversity of interventions and the inconsistent and incomplete reporting of study outcomes mean that there is currently no evidence that demonstrates the clinical effectiveness of the use of biofeedback in the treatment of hypertension. Given the current standards for the treatment of hypertension, further research is likely to be considered only as an adjunct to pharmacological interventions.
Chapter 6

Conclusion

There is currently no evidence that consistently demonstrates the effectiveness of the use of any particular biofeedback treatment in the control of essential hypertension when compared with pharmacotherapy, placebo (sham biofeedback treatment), no intervention or other behavioural therapies. The lack of evidence of clinical effectiveness negated the need to conduct an economic analysis. Further research might be considered into the potential role of biofeedback as an adjunct to drug therapy.
Acknowledgements

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We are also pleased to acknowledge Naveen Rao’s assistance with the data extraction for this report.

Contributions of authors (alphabetically)

Ms Rumona Dickson was involved in project management and provided input into all aspects of the review. Dr Yenal Dundar developed the search strategies and participated in study quality assessment and data extraction and checking. Dr Janette Greenhalgh was the principal review co-ordinator. All contributors took part in the editing and production of this report.

About the assessment group

The Liverpool Reviews and Implementation Group (LRiG) was established within the University of Liverpool in April 2001. It is a multidisciplinary research group whose purpose, in the first instance, is to conduct systematic reviews commissioned by the National Institute for Health Research Health Technology Assessment programme.
References


64. Bonso E, Palomba D, Perkovic D, Palatini P. Effect of a biofeedback system using an auto-shaping method on blood pressure at rest and during stress in mild hypertension. *Am J Hypertens* 2005; **18**:211A.


71. Patel C, Marmot MG, Terry DJ. Controlled trial of biofeedback-aided behavioural methods in...


**Appendix 1**

**Search strategy**

<table>
<thead>
<tr>
<th>Database</th>
<th>Years</th>
<th>Search strategy</th>
<th>References identified</th>
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<tbody>
<tr>
<td>MEDLINE</td>
<td>1950 to May 2007 (week 2)</td>
<td>See below</td>
<td>570</td>
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<tr>
<td>EMBASE</td>
<td>1980 to 2007 (week 20)</td>
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<tr>
<td>ISI Web of Knowledge/Web of Science</td>
<td>1945 to 2007</td>
<td>Biofeedback and hypertension</td>
<td>105</td>
</tr>
<tr>
<td>ISI Web of Knowledge/ISI Proceedings</td>
<td>1990 to 2007</td>
<td>As above</td>
<td>16</td>
</tr>
<tr>
<td>Cochrane Library 2007 (2)*</td>
<td>2007 (2)</td>
<td>As above</td>
<td>57 (CENTRAL: 54, other reviews: 2, HTA: 1)</td>
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<tr>
<td>CINAHL</td>
<td>1982 to May 2007 (week 3)</td>
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<td>86</td>
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<tr>
<td>AMED</td>
<td>1985 to May 2007 (week 3)</td>
<td>See below</td>
<td>96</td>
</tr>
<tr>
<td>PsycINFO</td>
<td>1967 to October 2007</td>
<td>See below</td>
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<td><strong>Total</strong></td>
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<td><strong>927</strong></td>
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</tbody>
</table>

*a* Includes the Cochrane Register of Controlled Trials (CENTRAL), Cochrane Database of Systematic Reviews, Database of Abstracts of Reviews of Effects (DARE), Health Technology Assessment (HTA) database and the NHS Economic Evaluation Database (NHS EED).

---

**Search strategy: MEDLINE (Ovid)**

1. hypertens$.tw.
2. (blood adj pressure).tw.
3. exp Hypertension/
4. exp “Biofeedback (Psychology)/
5. (bio-feedback$or biofeedback$).tw.
6. **”Mind-Body and Relaxation Techniques”/"
7. *Cognitive Therapy/or *Behavior Therapy/
8. ((relax$or cognitive) adj3 (therap$or technique$)).tw.
9. or/1–3
10. or/4–8
11. 9 and 10
12. animals/
13. humans/
14. 12 not 13
15. 11 not 14

**Search strategy: EMBASE (Ovid)**

1. hypertens$.tw.
2. (blood adj pressure).tw.
3. exp Hypertension/
4. (bio-feedback$or biofeedback$).tw.
5. ((relax$or cognitive) adj3 (therap$or technique$)).tw.
6. *Feedback System/
7. or/1–3
8. or/4–6
9. 7 and 8
10. limit 9 to human

**Search strategy: AMED (Ovid)**

1. hypertens$.tw.
2. (blood adj pressure).tw.
3. exp hypertension/
4. exp Biofeedback/or Relaxation/or Cognitive therapy/
5. (bio-feedback$or biofeedback$).tw.
6. ((relax$or cognitive) adj3 (therap$or technique$)).tw.
7. or/1–3
8. or/4–6
9. 7 and 8
Search strategy: CINAHL (Ovid)

1. hypertens$.tw
2. (blood adj pressure).tw.
3. exp hypertension/
4. exp “BIOFEEDBACK (IOWA NIC)”/or exp BIOFEEDBACK/
5. (bio-feedback$or biofeedback$).tw.
6. *SIMPLE RELAXATION THERAPY (IOWA NIC)”/or *RELAXATION TECHNIQUES/
7. ((relax$or cognitive) adj3 (therap$or technique$)).tw.
8. or/1–3
9. or/4–7
10. 8 and 9

Search strategy: PsycINFO 1967 to October 2007

1. hypertens$.tw.
2. (blood adj pressure).tw.
3. exp HYPERTENSION/
4. exp BIOFEEDBACK/
5. (bio-feedback$or biofeedback$).tw.
6. (Mind-Body and Relaxation Techniques).mp. [mp=title, abstract, heading word, table of contents, key concepts]
7. *relaxation therapy/
8. *Cognitive Therapy/
9. ((relax$or cognitive) adj3 (therap$or technique$)).tw.
10. or/1–3
11. or/4–8
12. and/10–11
13. limit 12 to human
Appendix 2

Biofeedback equipment

Table 18 presents the responses from various organisations regarding biofeedback equipment. The BHS and the ASH were unable to recommend any equipment. We had no response from the BFE or the EHS. The AAPB provides a spreadsheet that lists equipment and suppliers and a separate web page that presents advice on selecting and purchasing biofeedback equipment. One of our clinical advisers (CY) recommended that we just list websites of sellers or biofeedback equipment to ‘...allow the reader to explore and come to their own conclusions, or refer the reader to the AAPB website for their spreadsheet, which I assume is objective.’

Table 19 shows the equipment described in some of the biofeedback trials included in this review. They are grouped by modality type. It should be noted that some trials are very old and the instruments are likely to have been updated or superseded. The three most recent trials are those by Tsai et al., McCraty et al., and Yucha et al.
## TABLE 18 Equipment list

<table>
<thead>
<tr>
<th>Organisation</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td>British Hypertension Society</td>
<td>Unable to recommend any biofeedback equipment; however, there is a list of recommended blood pressure monitors for home use</td>
</tr>
<tr>
<td>National Centre for Complementary and Alternative Medicine</td>
<td>Unable to recommend any equipment but suggested looking at trials that they had funded and contacting authors</td>
</tr>
<tr>
<td>American Hypertension Society</td>
<td>Unable to recommend any equipment</td>
</tr>
<tr>
<td>American Association for Applied Physiology and Biofeedback</td>
<td>The AAPB website has a PDF spreadsheet providing a survey of instrumentation and a guide to buying equipment as well as details of US Food and Drugs Administration certification requirements: <a href="http://www.aapb.org/">www.aapb.org/</a>, <a href="http://www.aapb.org/">www.aapb.org/</a>, <a href="http://www.aapb.org/">www.aapb.org/</a></td>
</tr>
<tr>
<td>Biomedical Central (a supplier)</td>
<td>Our most popular instrument is the ProComp 8 with INFINITI software, which interfaces with your personal computer. This is an eight-channel system that can be tailored to your practice. Most impressive is the ability to create your own personal design screens with the latest developer tools</td>
</tr>
<tr>
<td>Biofeedback Foundation of Europe</td>
<td>No response</td>
</tr>
<tr>
<td>European Society for Hypertension</td>
<td>No response</td>
</tr>
<tr>
<td>A recent Hayes review(^1) included a section on equipment and lists the following as popular devices</td>
<td>Autogenic Systems: Autogen AT 42 Portable Single Channel Temperature Instrument, Autogen AT 53 Portable Dual Channel EMG, Autogen AT 62 Portable Single Alpha-Theta EEG, Autogen AT 64 Portable Single Channel SCR Instrument Biofeedback Instrument Company: ProComp Infiniti+ System Therapeutic Alliances Inc: NeuroEDUCATOR(^3) 3 EMG Biofeedback System NeuroDyne Medical Corp: MEDAC System/3R</td>
</tr>
<tr>
<td><a href="http://www.meditations-uk.com/products/wilddivine.html">www.meditations-uk.com/products/wilddivine.html</a></td>
<td>The Wild Divine computer game</td>
</tr>
</tbody>
</table>

AAPB, American Association for Applied Psychology and Biofeedback; EEG, electroencephalogram; EMG, electromyographic.
<table>
<thead>
<tr>
<th>Trial</th>
<th>Biofeedback modality</th>
<th>Biofeedback equipment used</th>
</tr>
</thead>
<tbody>
<tr>
<td>Friedman H, Taub HA. 6-month follow-up of use of hypnosis and biofeedback procedures in essential hypertension. <em>Am J Clin Hypn</em> 1978;20:184–8; also used data from Friedman and Taub 197737</td>
<td>BP</td>
<td>London Pressureometer, model 1905</td>
</tr>
<tr>
<td>Yucha CB, Tsai P, Calderon KS, Tian L. Biofeedback-assisted relaxation training for essential hypertension: who is most likely to benefit? <em>J Cardiovasc Nurs</em> 2005;20:198–205</td>
<td>Heart rate</td>
<td>Biofeedback-assisted relaxation included eight sessions of thermal, EMG and RSA biofeedback using Procomp/Multitrace biofeedback system (Thought Technology, West Chazy, NY)</td>
</tr>
</tbody>
</table>

*continued*
TABLE 19 Biofeedback equipment used in trials (continued)

<table>
<thead>
<tr>
<th>Trial</th>
<th>Biofeedback modality</th>
<th>Biofeedback equipment used</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walsh P, Dale A, Anderson DE. Comparison of biofeedback pulse wave velocity and progressive relaxation on essential hypertensives. Percept Mot Skills 1977;44:839–43</td>
<td>Pulse wave velocity</td>
<td>PWR monitored and recorded on Grass Polygraph model 7WC8PA. Timing of trials and assessment carried out with Coulbourn solid state logic system</td>
</tr>
</tbody>
</table>

BF, biofeedback; BP, blood pressure; DBP, diastolic blood pressure; EMG, electromyographic; GSR, galvanic skin response; HRV, heart rate variability; PW, pulse wave; RSA, respiratory sinus arrhythmia; SBP, systolic blood pressure; TBF, thermal biofeedback.
Appendix 3

QUOROM flow diagram of trial selection

Potentially relevant papers identified and screened for retrieval $n = 927$

Papers retrieved for more detailed evaluation $n = 100$

Total publications $n = 41$
- RCTs included in analysis $n = 36$
  - Two trials reported in 1 publication
  - One trial reported in 6 publications
  - One trial reported in 2 publications

Papers excluded $n = 827$
- non-RCT, not biofeedback, not population

Papers excluded $n = 59$
- non-RCT, not biofeedback, not population, patients from previous trial, paper unavailable, unable to distinguish results between treatments
### Appendix 4

#### Excluded trials

<table>
<thead>
<tr>
<th>Trial</th>
<th>Reason for exclusion</th>
</tr>
</thead>
</table>

*continued*
<table>
<thead>
<tr>
<th>Trial</th>
<th>Reason for exclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Engel BT, Glasgow MS, Gaarder KR. Behavioral treatment of high blood pressure. III. Follow-up results and treatment recommendations. <em>Psychosom Med</em> 1983;45:23–9</td>
<td>Non-RCT</td>
</tr>
<tr>
<td>Hahn YB, Ro YJ, Song HH, Kim NC, Kim HS, Yoo YS. The effect of thermal biofeedback and progressive muscle relaxation training in reducing blood pressure of patients with essential hypertension. <em>Image J Nurs Sch</em> 1993;25:204–7</td>
<td>Non-RCT</td>
</tr>
<tr>
<td>Jacob RG, Shapiro AP, Reeves RA, Johnsen AM, McDonald RH, Coburn PC. Relaxation therapy for hypertension. Comparison of effects with concomitant placebo, diuretic, and beta-blocker. <em>Arch Intern Med</em> 1986;146:2335–40</td>
<td>Not biofeedback treatment</td>
</tr>
<tr>
<td>Trial</td>
<td>Reason for exclusion</td>
</tr>
<tr>
<td>---------------------------------------------------------------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>Southam MA, Agras WS, Taylor CB, Kraemer HC. Relaxation training. Blood pressure lowering during the working day. Arch Gen Psychiatry 1982;39:715–17</td>
<td>Not biofeedback treatment</td>
</tr>
<tr>
<td>Taylor CB, Farquhar JW, Nelson E, Agras S. Relaxation therapy and high blood pressure. Arch Gen Psychiatry 1977;34:339–42</td>
<td>Not biofeedback treatment</td>
</tr>
</tbody>
</table>

continued
<table>
<thead>
<tr>
<th>Trial</th>
<th>Reason for exclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wadden TA. Relaxation therapy for essential hypertension: specific or nonspecific effects? <em>J Psychosom Res</em> 1984; <strong>28</strong>:53–61</td>
<td>Not biofeedback treatment</td>
</tr>
</tbody>
</table>

RCT, randomised controlled trial.
Appendix 5

Trial characteristics
<table>
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<tr>
<th>Trial</th>
<th>Report type</th>
<th>Intervention: type of biofeedback and number of training sessions</th>
<th>Comparator(s) and number of sessions</th>
<th>Number of patients, total and by arm</th>
<th>Timing of post-treatment follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Achmon 1989&lt;sup&gt;18&lt;/sup&gt;</td>
<td>Full</td>
<td>Heart rate: 17 sessions, one per week</td>
<td>CGTA: 17 sessions, 1.5 hours per week No treatment: two lectures + monthly checks</td>
<td>Randomised: 97 Treatment: 37; CGTA: 40; no treatment: 20 Reported: 77 Treatment: 27; CGTA: 30; no treatment: 20</td>
<td>6 months</td>
</tr>
<tr>
<td>Billion 1980&lt;sup&gt;23a&lt;/sup&gt;</td>
<td>Abstract</td>
<td>EMG: 16 sessions, two sessions per week</td>
<td>Relaxation Placebo: non-contingent EMG posed as EEG alpha biofeedback (sham biofeedback) Two sessions per week for 8 weeks</td>
<td>Randomised: NS Reported: 29</td>
<td>NA</td>
</tr>
<tr>
<td>Blanchard 1979&lt;sup&gt;34&lt;/sup&gt;</td>
<td>Full</td>
<td>SBP: 12 sessions</td>
<td>Relaxation EMG biofeedback 12 sessions</td>
<td>Randomised: 33 Reported: 28 Treatment: 10; EMG: 9; relaxation: 9</td>
<td>4 months</td>
</tr>
<tr>
<td>Blanchard 1986&lt;sup&gt;30&lt;/sup&gt;</td>
<td>Full</td>
<td>TBF: 16 sessions, two per week, + home practice with glass thermometer</td>
<td>Relaxation: eight sessions, one per week, + home practice using tape</td>
<td>Randomised: 87 Reported: 71 Treatment: 44 (withdraw then treat: 22; treat then withdraw: 22); relaxation: 43 (withdraw then treat: 20; treat then withdraw: 23)</td>
<td>Up to 1 year</td>
</tr>
<tr>
<td>Blanchard 1987&lt;sup&gt;30&lt;/sup&gt;</td>
<td>Full</td>
<td>TBF (laboratory): 16 sessions, two per week</td>
<td>TBF (home): 8 weeks, five sessions</td>
<td>Randomised: 18 Reported: 18 Laboratory: 9; home: 9</td>
<td>4–9 weeks</td>
</tr>
<tr>
<td>Blanchard 1988&lt;sup&gt;30&lt;/sup&gt; (USA)</td>
<td>Full</td>
<td>TBF: 20 sessions, two per week, + home practice</td>
<td>AT: 20 sessions, two per week Relaxation: 20 sessions, two per week</td>
<td>Randomised: unclear Reported: 29 Treatment: 10; AT: 11; relaxation: 8</td>
<td>1, 3, 6, 9 and 12 months (including booster treatment session)</td>
</tr>
<tr>
<td>Blanchard 1988&lt;sup&gt;30&lt;/sup&gt; (USSR)</td>
<td>Full</td>
<td>TBF: 20 sessions, two per week, + home practice</td>
<td>AT: 20 sessions, two per week Relaxation: 20 sessions, two per week</td>
<td>Randomised: unclear Reported: 30 Treatment: 10; AT: 10; relaxation: 10</td>
<td>1, 3, 6, 9 and 12 months (including booster treatment session)</td>
</tr>
<tr>
<td>Blanchard 1993&lt;sup&gt;32&lt;/sup&gt;</td>
<td>Full</td>
<td>TBF: 16 sessions, two per week, + regular home practice</td>
<td>EMG: 16 sessions, two per week, + regular home practice Home BP monitor: 8 weeks</td>
<td>Randomised: 41 Reported: 33 Treatment: 14 (3 w/d); EMG: 16 (3 w/d); self-monitor: 14 (2 w/d)</td>
<td>62</td>
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<td>Primary outcome</td>
<td>Location</td>
<td>Inclusion criteria</td>
<td>Exclusion criteria</td>
<td>Funding source</td>
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<td>-----------------</td>
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<td>--------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td></td>
</tr>
<tr>
<td>To compare the efficacy of methods in the treatment of hypertension</td>
<td>Israel</td>
<td>GP referred 25–60 years BP ≥ 140/90 for at least 6 months ≥ 8 years education Patient interested in participating and gave informed consent</td>
<td>No heart or renal disease No beta-blockers (diuretics OK) No psychiatric disease or organic brain syndrome</td>
<td>NS</td>
<td></td>
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<tr>
<td>Reduction in blood pressure</td>
<td>USA</td>
<td></td>
<td>NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Effects of intervention on SBP and DBP</td>
<td>USA</td>
<td>Essential hypertension: SBP &gt; 140 mmHg; DBP &gt; 90 mmHg</td>
<td>End-organ damage</td>
<td>NHLBI</td>
<td></td>
</tr>
<tr>
<td>To control BP using single drug (diuretic)</td>
<td>USA</td>
<td>Essential hypertension diagnosed by physician and study physician Controlled to 140/90 mmHg on two drugs</td>
<td>End-organ damage Serious medical or psychiatric conditions</td>
<td>NHLBI</td>
<td></td>
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<tr>
<td>To compare clinic-based and home-based regimen of biofeedback</td>
<td>USA</td>
<td>Essential hypertension</td>
<td>NS</td>
<td>NHLBI</td>
<td></td>
</tr>
<tr>
<td>Reduction in DBP</td>
<td>USA</td>
<td>DBP 90–110 mmHg on repeat screening not taking antihypertensive medication</td>
<td>End-organ damage Secondary hypertension Life-threatening illness Severe psychiatric disorder</td>
<td>NHLBI</td>
<td></td>
</tr>
<tr>
<td>Reduction in DBP</td>
<td>USSR</td>
<td>DBP 90–110 mmHg on repeat screening not taking antihypertensive medication</td>
<td>End-organ damage Secondary hypertension Life-threatening illness Severe psychiatric disorder</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Discontinuation of sympatholytic medication from two-drug regimen with diuretic as second drug</td>
<td>USA</td>
<td>Adults with moderate hypertension well controlled on metoprolol plus diuretic</td>
<td>Cardiac disease Diabetes Asthma Could not stabilise on metoprolol BP not controlled</td>
<td>NHLBI</td>
<td></td>
</tr>
</tbody>
</table>

continued
<table>
<thead>
<tr>
<th>Trial</th>
<th>Report type</th>
<th>Intervention: type of biofeedback and number of training sessions</th>
<th>Comparator(s) and number of sessions</th>
<th>Number of patients, total and by arm</th>
<th>Timing of post-treatment follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blanchard 1996</td>
<td>Full</td>
<td>TBF: 16 sessions, two per week</td>
<td>Home BP monitor: two per day for 4 weeks</td>
<td>Randomised: 46 Reported: 42 Treatment: 21; self-monitor: 21</td>
<td>12 months of follow-up (0, 3, 6 and 12 months’ follow-up)</td>
</tr>
<tr>
<td>Bonso 2005</td>
<td>Abstract</td>
<td>NS: four sessions, one per week, 2 weeks follow-up</td>
<td>Self-monitor: 6 weeks</td>
<td>Randomised: NS Reported: 29 Group allocation: NS</td>
<td>2 weeks</td>
</tr>
<tr>
<td>Friedman 1978</td>
<td>Full</td>
<td>BP: seven sessions, daily home practice</td>
<td>Hypnosis + BF: seven Hypnosis only: seven Clinic monitor: seven</td>
<td>Randomised: 48 Reported: 48 Treatment: 13; BF + hypnosis: 10; hypnosis: 13; clinic monitor: 12</td>
<td>1 month and 6 months</td>
</tr>
<tr>
<td>Goldstein 1982</td>
<td>Full</td>
<td>SBP and DBP: 16 sessions, two per week</td>
<td>Antihypertensive medication Relaxation Self-monitor</td>
<td>Randomised: 36 Reported: 36 Treatment: 9; relaxation: 9; medication: 9; self-monitor: 9</td>
<td>6 months</td>
</tr>
<tr>
<td>Hager 1978</td>
<td>Full</td>
<td>BP: 40 sessions, 4 weeks</td>
<td>Meditation: 40 sessions, 4 weeks</td>
<td>Randomised: 30 Reported: 17 Treatment: 7; meditation: 10</td>
<td>NA</td>
</tr>
<tr>
<td>Hatch 1985</td>
<td>Full</td>
<td>DBP: 12 sessions</td>
<td>Progressive deep muscle relaxation training Self-directed relaxation training No treatment</td>
<td>Randomised: 52 Reported: 52 Treatment: 13; relaxation:13; self-relaxation:13; no treatment:13</td>
<td>12 months</td>
</tr>
<tr>
<td>Hunyor 1997</td>
<td>Full</td>
<td>SBP: eight sessions</td>
<td>Placebo (sham biofeedback treatment): eight sessions</td>
<td>Randomised: 58 Reported: 56 Treatment: 28; placebo: 28</td>
<td>NA</td>
</tr>
<tr>
<td>Primary outcome</td>
<td>Location</td>
<td>Inclusion criteria</td>
<td>Exclusion criteria</td>
<td>Funding source</td>
<td></td>
</tr>
<tr>
<td>-----------------</td>
<td>-----------</td>
<td>--------------------</td>
<td>--------------------</td>
<td>----------------</td>
<td></td>
</tr>
<tr>
<td>DBP &lt; 90mmHg</td>
<td>USA</td>
<td>DBP ≥ 90mmHg at second/third screening visit Unmedicated</td>
<td>DBP &gt; 105mmHg or SBP &gt; 180mmHg, DBP &lt; 90mmHg</td>
<td>NHLBI</td>
<td></td>
</tr>
<tr>
<td>Reduction in BP</td>
<td>Italy</td>
<td>Stage 1 hypertension</td>
<td>NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Effects on diastolic blood pressure</td>
<td>USA</td>
<td>Diagnosis of hypertension Minimum DBP 85 mmHg during baseline Able to complete all sessions and 1-week follow up</td>
<td>NS</td>
<td>Medical Research Service of the Veterans Administration</td>
<td></td>
</tr>
<tr>
<td>To evaluate BF and Benson relaxation, and to compare their effectiveness with drug therapy</td>
<td>USA</td>
<td>DBP: 90–105 mmHg SBP: 150–165 mmHg</td>
<td>Secondary hypertension Obesity Drug abuse Alcoholism Heart disease Psychotherapy and organicsy</td>
<td>NHLBI</td>
<td></td>
</tr>
<tr>
<td>To compare biofeedback and meditation–relaxation in reducing SBP and DBP</td>
<td>USA</td>
<td>History SBP 145 mmHg or DBP &gt; 95 mmHg, Essential hypertension</td>
<td>NS</td>
<td>NIMH</td>
<td></td>
</tr>
<tr>
<td>To compare the effectiveness of direct DBP-BF and progressive deep muscle relaxation in patients whose BP is already effectively controlled pharmacologically</td>
<td>USA</td>
<td>Essential hypertension Active pharmacological treatment Age range 21–70 years</td>
<td>Evidence of psychiatric disorder or other serious medical disorder Concomitant medications (HRTs, cardio, psychotropic) NIH research</td>
<td></td>
<td></td>
</tr>
<tr>
<td>The capability of SBP lowering of ≥ 5 mmHg using continuous pressure feedback</td>
<td>Australia</td>
<td>Mildly hypertensive: SBP &lt; 200 mmHg, DBP &lt; 115 mmHg</td>
<td>SBP ≥ 200mmHg DBP ≥ 115 mmHg Inability to make time commitment Evidence of target organ damage LVH Retinal haemorrhages</td>
<td>National Health and Medical Research Council, National Heart Foundation (Australia), the Government Health Employees Research Fund (NSW), North Shore Heart Research Foundation</td>
<td></td>
</tr>
</tbody>
</table>
### TABLE 20  Trial characteristics: biofeedback alone (continued)

<table>
<thead>
<tr>
<th>Trial</th>
<th>Report type</th>
<th>Intervention: type of biofeedback and number of training sessions</th>
<th>Comparator(s) and number of sessions</th>
<th>Number of patients, total and by arm</th>
<th>Timing of post-treatment follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Luborsky 1982&lt;sup&gt;60&lt;/sup&gt;</td>
<td>Full</td>
<td>BP: five sessions, one per week</td>
<td>Antihypertensive medication</td>
<td>Randomised: 51</td>
<td>3 months</td>
</tr>
<tr>
<td></td>
<td></td>
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<td>Metronome-conditioned relaxation</td>
<td>Reported: 51</td>
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<td>Mild exercise</td>
<td>Treatment: 14;</td>
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<td>medication: 10;</td>
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<td>relaxation: 16;</td>
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<td></td>
<td></td>
<td></td>
<td>exercise: 11</td>
<td></td>
</tr>
<tr>
<td>Nakao 1997&lt;sup&gt;15&lt;/sup&gt;</td>
<td>Full</td>
<td>SBP: four sessions, one per week</td>
<td>No treatment</td>
<td>Randomised: 31</td>
<td>3 months</td>
</tr>
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<td></td>
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<td>Reported: 30</td>
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<td>Treatment: 15;</td>
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<td>self-monitor: 15</td>
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<tr>
<td>Thananopavarn 1979&lt;sup&gt;34a&lt;/sup&gt;</td>
<td>Abstract</td>
<td>NS: 2 hours, 3 days per week</td>
<td>Relaxation: 2 hours, 3 days per week</td>
<td>Randomised: NS</td>
<td>NA</td>
</tr>
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<td></td>
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<td>Reported: 12</td>
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<td>Treatment: 5;</td>
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<td>relaxation: 3;</td>
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<td>medication: 4</td>
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<tr>
<td>Tsai 2007&lt;sup&gt;26&lt;/sup&gt;</td>
<td>Full</td>
<td>BP: four sessions, one per week</td>
<td>Placebo (sham biofeedback treatment)</td>
<td>Randomised: 42</td>
<td>12 weeks (8 weeks after treatment)</td>
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<td>Reported: 38</td>
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<td>Treatment: 20;</td>
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<td></td>
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<td></td>
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<td>placebo: 18</td>
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</tr>
<tr>
<td>Walsh 1977&lt;sup&gt;27&lt;/sup&gt;</td>
<td>Full</td>
<td>Pulse wave velocity: five sessions, one per week</td>
<td>Relaxation: five sessions, one per week</td>
<td>Randomised: 24</td>
<td>NA&lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
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<td>Reported: 24</td>
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<td>Treatment: 11;</td>
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<td></td>
<td>relaxation: 13</td>
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<tr>
<td>Zurawski 1987&lt;sup&gt;41&lt;/sup&gt;</td>
<td>Full</td>
<td>GSR: eight sessions, one per week, 60–90 minutes, + home practice</td>
<td>SMT: eight sessions, one per week, 60–90 minutes, + home practice</td>
<td>Randomised: 29</td>
<td>6 months</td>
</tr>
<tr>
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<td>Reported: 25</td>
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<td>Treatment: 14;</td>
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<td></td>
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<td>SMT: 11</td>
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</tr>
</tbody>
</table>

<sup>a</sup> Data derived from abstract.

AT, autogenic training; BF, biofeedback; BP, blood pressure; CGTA, cognitive group therapy for anxiety; DBP, diastolic blood pressure; EEG, electroencephalograph; EMG, electromyographic; GSR, galvanic skin response; HRT, hormone replacement therapy; LVH, left ventricular hypertrophy; NA, not applicable; NHLBI, National Heart, Lung and Blood Institute; NHRI, National Human Rights Institution; NIH, National Institutes for Health; NSF, National Science Foundation; NS, not stated; SBP, systolic blood pressure; SMT, stress management training; TBF, thermal biofeedback; w/d, withdrawn.
<table>
<thead>
<tr>
<th>Primary outcome</th>
<th>Location</th>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
<th>Funding source</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Comparison of pharmacotherapy and behavioural therapy</strong></td>
<td>USA</td>
<td>BP &gt; 140/90 mmHg and &lt; 165/103 mmHg and 20–55 years</td>
<td>Evidence of target organ damage</td>
<td>Research grant</td>
</tr>
<tr>
<td><strong>To study the efficacy of this system for the treatment of essential hypertension, compare simple blood pressure self-monitoring and self-monitoring + blood pressure biofeedback and investigate the physiological changes that occur during blood pressure biofeedback</strong></td>
<td>Japan</td>
<td>Diagnosis of essential hypertension according to WHO and 35–65 years and Antihypertensive medication unchanged for 3 weeks</td>
<td>History of beta-blocker use and History of cerebral vascular accident and NS</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Change in BP</strong></td>
<td>USA</td>
<td>Mild essential hypertension and No medication for at least 4 weeks and DBP &gt; 90 mmHg</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Change in SBP</strong></td>
<td>Taiwan</td>
<td>Stage 1 hypertension (SBP 140–159 mmHg or DBP 90–99 mmHg) and 19–56 years and Able to read and write</td>
<td>Receiving/received cardiovascular medication for hypertension within previous 2 months and Kidney or liver disease and Neurological disorder and Psychiatric disorder and Diabetes</td>
<td>NHRI and National Science Council Taiwan</td>
</tr>
<tr>
<td><strong>To evaluate the clinical effectiveness of two behavioural treatments for essential hypertension</strong></td>
<td>USA</td>
<td>NS</td>
<td>NS</td>
<td>Supported by NSF</td>
</tr>
<tr>
<td><strong>The effectiveness of SMT relative to GSR BF in the treatment of essential hypertensive blood pressure at rest and in response to simulated stressful situations</strong></td>
<td>USA</td>
<td>Consecutive casual BP ≥ 140/90 mmHg and Under care of physician and Diagnosis of essential hypertension and Age 18–60 years and Not excessively overweight and Willing to monitor type and dosage of medications taken throughout project</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Trial</td>
<td>Report type</td>
<td>Intervention: type of biofeedback and number of sessions</td>
<td>Combination therapy</td>
<td>Comparator(s) and number of sessions</td>
</tr>
<tr>
<td>---------------</td>
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</tr>
<tr>
<td>Berglund 1991</td>
<td>Abstract</td>
<td>TBF: 12 sessions</td>
<td>Menninger protocol</td>
<td>Self-monitor</td>
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<tr>
<td>Canino 1994</td>
<td>Full</td>
<td>TBF: 15 sessions</td>
<td>Relaxation + anxiety management</td>
<td>Placebo behavioural therapy; No treatment</td>
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<tr>
<td>Chesney 1987</td>
<td>Full</td>
<td>TBF and EMG (modality alternated across sessions): 13 over 17 weeks then five sessions follow-up over 36 weeks</td>
<td>Relaxation</td>
<td>Combined behavioural group consisting of relaxation, RCR, BFCR, HBC, clinic BPM</td>
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<tr>
<td>Cohen 1983</td>
<td>Full</td>
<td>EMG and TBF: 20 sessions, two per week</td>
<td>Relaxation</td>
<td>Relaxation: five sessions, one per week, and again at week 15; Waiting list</td>
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<tr>
<td>Frankel 1978</td>
<td>Full</td>
<td>DBP and EMG: 20 sessions over 16 weeks + home practice</td>
<td>Relaxation</td>
<td>Placebo (sham biofeedback treatment): 20 sessions over 16 weeks; Clinic blood pressure monitor</td>
</tr>
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<tr>
<td>Friedman 1978</td>
<td>Full</td>
<td>BP: seven sessions</td>
<td>Hypnosis</td>
<td>Biofeedback only; Hypnosis only; Clinic blood pressure monitor; Seven sessions</td>
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<tr>
<td>Hafner 1982</td>
<td>Full</td>
<td>GSR or EMG: eight sessions, one per week</td>
<td>Relaxation + meditation</td>
<td>Meditation, one session per week for 8 weeks; No treatment</td>
</tr>
<tr>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Timing of post-treatment follow-up</td>
<td>Primary outcome</td>
<td>Location</td>
<td>Inclusion criteria</td>
<td>Exclusion criteria</td>
</tr>
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</tr>
<tr>
<td>NS</td>
<td>Change in blood pressure</td>
<td>USA</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>6 months</td>
<td>Reduction in DBP and SBP, effects of behavioural therapy on control + reduction of blood pressure</td>
<td>Venezuela</td>
<td>Established essential hypertension 25–48 years Mean blood pressure 140/90 mmHg No antihypertensive medication</td>
<td>NS</td>
</tr>
<tr>
<td>54 weeks</td>
<td>Change in blood pressure between behavioural therapy and BPM groups</td>
<td>USA</td>
<td>DBP between 90 and 104 mmHg Not taking antihypertensive medication</td>
<td>DBP &gt; 90 mmHg but medicated Secondary hypertension DBP &gt; 105 mmHg SBP &gt; 170 mmHg</td>
</tr>
<tr>
<td>4 months</td>
<td>Effects of interventions on attentional dimensions</td>
<td>USA</td>
<td>Diagnosis of hypertension for 2 years</td>
<td>Not essential hypertension Major disease-related complications Serious medical or psychological illness</td>
</tr>
<tr>
<td>NA</td>
<td>Effects of interventions on blood pressure</td>
<td>NS</td>
<td>Uncomplicated hypertension</td>
<td>NS</td>
</tr>
<tr>
<td>1 month and 6 months</td>
<td>Effects on DBP</td>
<td>USA</td>
<td>Hypertension Minimum DBP 85 mmHg during baseline Able to complete all training sessions and 1-week follow-up</td>
<td>NS</td>
</tr>
<tr>
<td>3 months</td>
<td>Is a combination of meditation and biofeedback-aided relaxation superior to meditation alone?</td>
<td>UK</td>
<td>Essential hypertension No relevant lesions or disorders</td>
<td>NS</td>
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</tbody>
</table>

continued
### TABLE 21  Trial characteristics: biofeedback combinations (continued)

<table>
<thead>
<tr>
<th>Trial</th>
<th>Report type</th>
<th>Intervention: type of biofeedback and number of sessions</th>
<th>Combination therapy</th>
<th>Comparator(s) and number of sessions</th>
<th>Number of patients, total and by arm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Irvine 1991</td>
<td>Full</td>
<td>GSR: 6–12 sessions</td>
<td>Relaxation + imagery + meditation</td>
<td>NSST</td>
<td>All: 110  Reported: 101  Treatment: 50; NSST: 51</td>
</tr>
<tr>
<td>Jacob 1992</td>
<td>Full</td>
<td>TBF: 12 sessions</td>
<td>Relaxation</td>
<td>Stress education: 12 sessions</td>
<td>Randomised: 20  Reported: 19  Treatment: 10; stress education: 9</td>
</tr>
<tr>
<td>Jurek 1992</td>
<td>Full</td>
<td>EMG and TBF: 16 sessions, two per week</td>
<td>Relaxation + diuretic</td>
<td>Diuretic only</td>
<td>Randomised: 47  Reported: 30  Treatment: 20; diuretic only: 10</td>
</tr>
<tr>
<td>Khramelashvili 1986</td>
<td>Abstract</td>
<td>NS</td>
<td>Relaxation</td>
<td>NS</td>
<td>Randomised: NS  Reported: 80  Treatment: 30; autotraining: 30; no intervention: 20</td>
</tr>
<tr>
<td>McCraty 2003</td>
<td>Full</td>
<td>HR variability: 12 hours in 2 weeks</td>
<td>IQM</td>
<td>Waiting list</td>
<td>Randomised: 38  Reported: 32  Treatment: 18; waiting list: 14</td>
</tr>
<tr>
<td>McGrady 1981</td>
<td>Full</td>
<td>EMG: 16 sessions, two per week</td>
<td>Relaxation</td>
<td>Blood pressure monitoring</td>
<td>Randomised: 43  Reported: 38  Treatment: 22; blood pressure monitor: 16</td>
</tr>
<tr>
<td>Timing of post-treatment follow-up</td>
<td>Primary outcome</td>
<td>Location</td>
<td>Inclusion criteria</td>
<td>Exclusion criteria</td>
<td>Funding source</td>
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</tr>
<tr>
<td>6 months</td>
<td>To evaluate relaxation behaviour therapy as sole treatment for uncomplicated and previously untreated mild hypertension</td>
<td>Canada</td>
<td>Untreated hypertensives with mean DBP &lt; 105 mmHg</td>
<td>SBP ≥ 200 mmHg at first screening DBP ≥ 120 mmHg at any screening DBP averaged &gt; 114 mmHg after third screening DBP averaged &gt; 104 mmHg after fifth screening Myocardial infarction Congestive heart failure Stroke Angina pectoris Currently taking antihypertensive medication</td>
<td>Ontario Ministry of Health, National Health and Research Development, Ciba Geigy</td>
</tr>
<tr>
<td>NS</td>
<td>Comparison of biofeedback and stress education in reduction of blood pressure in hypertensive patients whose antihypertensive medications were experimentally controlled</td>
<td>USA</td>
<td>DBP &gt; 90 mmHg</td>
<td>NS</td>
<td>NHLBI</td>
</tr>
<tr>
<td>10–12 months</td>
<td>Effect and comparison of two arms in lowering of SBP and DBP</td>
<td>USA</td>
<td>21–60 years Diagnosis of hypertension 1 year</td>
<td>NS</td>
<td>Northwestern Ohio Heart Association</td>
</tr>
<tr>
<td>NS</td>
<td>Changes in blood pressure, stress tolerance and psychological status</td>
<td>NS</td>
<td>Essential hypertension (stages IIA–IIB)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>3 months</td>
<td>Impact of a workplace-based stress management programme on blood pressure, emotional health and workplace-related measures in hypertensive employees</td>
<td>USA</td>
<td>Regular schedule of hypertensive medications At least 1/4 baseline BP readings in a range of 90–105 mmHg DBP or 140–179 mmHg SBP</td>
<td>Changes in hypertensive medications Schedule conflicts and/or personal reasons</td>
<td>NS</td>
</tr>
<tr>
<td>None</td>
<td>Effect of BF + relaxation on treatment of essential hypertension</td>
<td>USA</td>
<td>Essential hypertension</td>
<td>NS</td>
<td>North Western Ohio Heart Association</td>
</tr>
</tbody>
</table>
### TABLE 21  Trial characteristics: biofeedback combinations (continued)

<table>
<thead>
<tr>
<th>Trial</th>
<th>Report type</th>
<th>Intervention: type of biofeedback and number of sessions</th>
<th>Combination therapy</th>
<th>Comparator(s) and number of sessions</th>
<th>Number of patients, total and by arm</th>
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</thead>
<tbody>
<tr>
<td>McGrady 1994&lt;sup&gt;65&lt;/sup&gt;</td>
<td>Full</td>
<td>TBF: eight sessions, one per week</td>
<td>Relaxation</td>
<td>Waiting list</td>
<td>Randomised: 138</td>
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<td>Reported: 101</td>
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<td></td>
<td>Treatment: 70; waiting list 31</td>
</tr>
<tr>
<td>Patel 1975&lt;sup&gt;45&lt;/sup&gt;</td>
<td>Full</td>
<td>GSR EMG: 12 sessions, two per week, + home practice</td>
<td>Yoga</td>
<td>Relaxation: 12 sessions, two per week, 30 minutes each</td>
<td>Randomised: 37</td>
</tr>
<tr>
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<td></td>
<td></td>
<td>Reported: 34</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>Treatment: 17; yoga: 17</td>
</tr>
<tr>
<td>Patel 1988&lt;sup&gt;40&lt;/sup&gt;</td>
<td>Full</td>
<td>GSR: eight sessions, one per week, + home practice</td>
<td>Relaxation</td>
<td>No treatment</td>
<td>Randomised: 116</td>
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<tr>
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<td></td>
<td>Reported: 103</td>
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<td>Treatment: 49; no treatment: 54</td>
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</tbody>
</table>

BF, biofeedback; BFCR, biofeedback + cognitive restructuring; BP, blood pressure; BPM, blood pressure monitoring; DBP, diastolic blood pressure; EMG, electromyographic; GSR, galvanic skin response; HBC, health behaviour change; HR, heart rate; IQM, inner quality management; MRC, Medical Research Council; NA, not available; NHLBI, National Heart, Lung and Blood Institute; NS, not stated; NSST, non-specific support therapy; RCR, relaxation + cognitive restructuring; RHA, Regional Health Authority; SBP, systolic blood pressure; TBF, thermal biofeedback.

<sup>a</sup> Data derived from abstract.
<table>
<thead>
<tr>
<th>Timing of post-treatment follow-up</th>
<th>Primary outcome</th>
<th>Location</th>
<th>Inclusion criteria</th>
<th>Exclusion criteria</th>
<th>Funding source</th>
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</thead>
<tbody>
<tr>
<td>10 months</td>
<td>Effects of relaxation and TBF on BP and related psychological and physiological parameters</td>
<td>USA</td>
<td>Essential hypertension, Medicated or unmedicated diagnosed by physician</td>
<td>Not clear</td>
<td>City of Toledo Health Department</td>
</tr>
<tr>
<td>3 months</td>
<td>Effects of therapy on blood pressure</td>
<td>UK</td>
<td>Medicated for at least 6 months with initial DBP levels of at least 110 mmHg on two separate days</td>
<td>NS</td>
<td>Support from South West Thames RHA</td>
</tr>
<tr>
<td>1 year</td>
<td>Changes in SBP and DBP</td>
<td>UK</td>
<td>The last 134 recruits to the second phase of a 6-year MRC trial who consented to take part</td>
<td>NS</td>
<td>Support from British Heart Foundation; Wyeth Laboratories sponsored workshops for doctors and nurses</td>
</tr>
</tbody>
</table>
Appendix 6

Patient characteristics
<table>
<thead>
<tr>
<th>Trial</th>
<th>Gender (% male)</th>
<th>Ethnic origin</th>
<th>Age (years), mean (SD)</th>
<th>% antihypertensive medication</th>
<th>Years hypertensive, mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Achmon 1989</td>
<td>Treatment: 63%; CGTG: 57%; no treatment: 75%</td>
<td>NS</td>
<td>Treatment: 40.1 (8.3); CGTG: 41.6 (9.0); no treatment: 40.0 (8.6)</td>
<td>Treatment: 48%; CGTG: 40%; no treatment: 35%</td>
<td>Treatment: 5.5 (4.7); CGTG: 4.2 (3.3); no treatment: 5.1 (4.8)</td>
</tr>
<tr>
<td>Billion 1980</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Blanchard 1979</td>
<td>48.40%</td>
<td>NS</td>
<td>39.5 (range 23–56)</td>
<td>45%</td>
<td>NS</td>
</tr>
<tr>
<td>Blanchard 1986</td>
<td>Treatment: 64%; Relaxation: withdraw then treat: 55%; treat then withdraw: 44%</td>
<td>NS</td>
<td>Treatment: withdraw then treat: 50.7; treat then withdraw: 50.1</td>
<td>100%</td>
<td>Treatment: withdraw then treat: 6.5; treat then withdraw: 9.2</td>
</tr>
<tr>
<td>Blanchard 1987</td>
<td>All: 72%; laboratory: 63%; home: 88.8%</td>
<td>NS</td>
<td>All: 48.05; laboratory: 45.8; home: 50.3</td>
<td>100%</td>
<td>Laboratory: 5.9; home: 6.4</td>
</tr>
<tr>
<td>Blanchard 1988 (USA)</td>
<td>All: 100%</td>
<td>White</td>
<td>Treatment: 45 (8.26) (range 34–61); AT: 44 (6.1) (range 36–52); relaxation: 40.75 (10.12) (range 27–57)</td>
<td>None</td>
<td>Treatment: 5.6 (6) (range 0.5–18); AT: 6.2 (6.2) (range 0.5–20); relaxation: 2.9 (2.0) (range 1–7)</td>
</tr>
<tr>
<td>Blanchard 1988 (USSR)</td>
<td>All: 100%</td>
<td>White</td>
<td>Treatment: 30.9 (5.3) (range 24–42); AT: 33.6 (11.2) (range 23–48); relaxation: 35.2 (9.2) (range 21–50)</td>
<td>None</td>
<td>Treatment: 6.9 (5.7) (range 1–17); AT: 10.7 (7.3) (range 2–28); relaxation: 7.6 (3.6) (range 3–13)</td>
</tr>
<tr>
<td>Blanchard 1993</td>
<td>All: 61%; treatment: 64%; EMG: 60%; self-monitor: 58%; withdrawn: 75%</td>
<td>NS</td>
<td>Treatment: 48.4; EMG: 53.5; self-monitor: 52.8; withdrawn: 51.4</td>
<td>100%</td>
<td>Treatment: 8.2; EMG: 10.0; self-monitor: 10.0; withdrawn: 7.0</td>
</tr>
<tr>
<td>Blanchard 1996</td>
<td>All: 67%; treatment: 71%; self-monitor: 59%</td>
<td>NS</td>
<td>Treatment: 50.0 (range 32–61); self-monitor: 51.0 (range 40–62)</td>
<td>None</td>
<td>Treatment: 8.3; self-monitor: 8.4</td>
</tr>
<tr>
<td>Bonso 2005</td>
<td>NS</td>
<td>NS</td>
<td>22–55 (range)</td>
<td>None</td>
<td>NS</td>
</tr>
<tr>
<td>Friedman 1978</td>
<td>Biofeedback + hypnosis: 80%; biofeedback: 77%; hypnosis: 83%; clinic blood pressure monitor: 83%</td>
<td>NS</td>
<td>Treatment: 47.2 (range 29–54); biofeedback + hypnosis: 48.2 (range 32–53); hypnosis: 47.1 (range 23–60); clinic blood pressure monitor: 48.3 (range 31–59)</td>
<td>Treatment: 62%; biofeedback + hypnosis: 60%; hypnosis: 69%; clinic blood pressure monitor: 75%</td>
<td>NS</td>
</tr>
<tr>
<td>Trial</td>
<td>Gender (% male)</td>
<td>Ethnic origin</td>
<td>Age (years), mean (SD)</td>
<td>% antihypertensive medication</td>
<td>Years hypertensive, mean (SD)</td>
</tr>
<tr>
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</tr>
<tr>
<td>Goldstein 1982</td>
<td>Treatment: 70%; relaxation: 80%; antihypertensive medication: 80%; self-monitor: 80%</td>
<td>NS</td>
<td>Treatment: 51.1; relaxation: 51.2; antihypertensive medication: 54.6; self-monitor: 49.1 (range 35–60)</td>
<td>Medication arm only</td>
<td>Treatment: 11.2; relaxation: 6.7; antihypertensive medication: 14.1; self-monitor: 8.5</td>
</tr>
<tr>
<td>Hager 1978</td>
<td>50%</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Hatch 1985</td>
<td>Treatment: 30.7%; relaxation: 53.8%; self-relaxation: 30.7%; no treatment: 46.1%</td>
<td>NS</td>
<td>Treatment: 51.6; relaxation: 50.2; self-relaxation: 50.4; no treatment: 52.2 (range 21–70)</td>
<td>NS</td>
<td>Treatment: 8.0; relaxation: 5.2; self-relaxation: 7.7; no treatment: 5.8</td>
</tr>
<tr>
<td>Hunyor 1997</td>
<td>NS</td>
<td>NS</td>
<td>18–69 (range)</td>
<td>None</td>
<td>All: 9.5 (9.2) (range 0–45)</td>
</tr>
<tr>
<td>Luborsky 1982</td>
<td>NS</td>
<td>NS</td>
<td>38 (range 20–55)</td>
<td>Antihypertensive medication arm only</td>
<td>NS</td>
</tr>
<tr>
<td>Nakao 1997</td>
<td>All: 33%; treatment: 33%; self-monitor: 33%</td>
<td>NS</td>
<td>All: 56; treatment: 55 (8); self-monitor: 56 (8)</td>
<td>Treatment: 33%; self-monitor: 47%</td>
<td>Treatment (months) 49 (72); self-monitor (months) 42 (57)</td>
</tr>
<tr>
<td>Thananopavarn 1979</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>Antihypertensive medication arm only</td>
<td>NA</td>
</tr>
<tr>
<td>Tsai 2007</td>
<td>Treatment: 50%; placebo: 78%</td>
<td>NS</td>
<td>Treatment: 46.5 (10.3); placebo: 39.9 (10.8)</td>
<td>None</td>
<td>NS</td>
</tr>
<tr>
<td>Walsh 1977</td>
<td>All: 63%</td>
<td>NS</td>
<td>All: 24–69 (range)</td>
<td>Treatment: 45%; relaxation: 54%</td>
<td>NS</td>
</tr>
<tr>
<td>Zurawski 1987</td>
<td>Treatment: 40%; SMT: 57%</td>
<td>NS</td>
<td>Treatment: 45.99; SMT: 47.5</td>
<td>Treatment: 73%; SMT: 53%</td>
<td>NS</td>
</tr>
</tbody>
</table>

AT, autogenic training; CGTA, cognitive group therapy for anxiety; EMG, electromyographic; NA, not available; NS, not stated; SD, standard deviation; SMT, stress management training.

a Information derived from abstract.
<table>
<thead>
<tr>
<th>Trial</th>
<th>Gender (% male)</th>
<th>Ethnic origin</th>
<th>Age (years), mean (SD)</th>
<th>% antihypertensive medication</th>
<th>Years hypertensive, mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Berglund 1991</td>
<td>All: 100%</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Canino 1994</td>
<td>All: 66%</td>
<td>NS</td>
<td>NS</td>
<td>None</td>
<td>NS</td>
</tr>
<tr>
<td>Chesney 1987</td>
<td>All: 89%</td>
<td>All: 87% white</td>
<td>All: 47</td>
<td>Increased weekly; treatment from 1.7% to 16.9%; clinic blood pressure monitoring from 0% to 12.5%</td>
<td>NS</td>
</tr>
<tr>
<td>Cohen 1983</td>
<td>Treatment: 40%; relaxation: 50%; waiting list: 40%</td>
<td>NS</td>
<td>Treatment: 47.4 (range 26–72); relaxation: 48.2 (range 31–68); waiting list: 37.8 (range 28–54)</td>
<td>90%</td>
<td>2</td>
</tr>
<tr>
<td>Frankel 1978</td>
<td>Active treatment: 57.1%; placebo treatment: 57.1%; clinic blood pressure monitor: 50%</td>
<td>Active treatment: white: 71%; black: 29%; Placebo treatment: white: 86%; black: 14%; Clinic blood pressure monitor: white: 38%; black: 63%</td>
<td>Active treatment: 43.8; placebo treatment: 50.4; clinic blood pressure monitor: 43.5</td>
<td>32%</td>
<td>Active treatment: Placebo treatment: Clinic blood pressure monitor: 11.3</td>
</tr>
<tr>
<td>Friedman 1978</td>
<td>Treatment: 80%; biofeedback only: 77%; hypnosis: 85%; clinic blood pressure monitor: 83%</td>
<td>NS</td>
<td>Treatment: 48.2 (range 32–53); biofeedback only: 47.2 (range 29–54); hypnosis: 47.1 (range 23–60); clinic blood pressure monitor: 48.3 (range 31–59)</td>
<td>Treatment: 60%; biofeedback only: 62%; hypnosis: 69%; clinic blood pressure monitor: 75%</td>
<td>NS</td>
</tr>
<tr>
<td>Hafner 1982</td>
<td>All: 57%</td>
<td>NS</td>
<td>All: 48.9 (range 25–68)</td>
<td>90%</td>
<td>All: 4.1 (range 4 months to 10 years)</td>
</tr>
<tr>
<td>Irvine 1991</td>
<td>Treatment: 82%; NSST: 82%</td>
<td>NS</td>
<td>Treatment: 46.7 (8.1); NSST: 45.8 (8.5)</td>
<td>None</td>
<td>NA</td>
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<tr>
<td>Jacob 1992</td>
<td>Treatment: 60%; stress education: 78%</td>
<td>NS</td>
<td>Treatment: 46.5 (11.4); stress education: 51.4 (8.3)</td>
<td>100%</td>
<td>Treatment: 13 (range 3–37); stress education: 10 (range 2.5–30)</td>
</tr>
<tr>
<td>Jurek 1992</td>
<td>All: 63.3%; treatment: 60%; diuretic only: 70%</td>
<td>White: 80%; black: 20%</td>
<td>Treatment: 49; diuretic only: 48</td>
<td>100%</td>
<td>NS</td>
</tr>
<tr>
<td>Khramelashvili</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Trial</td>
<td>Gender (% male)</td>
<td>Ethnic origin</td>
<td>Age (years), mean (SD)</td>
<td>% antihypertensive medication</td>
<td>Years hypertensive, mean (SD)</td>
</tr>
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</tr>
<tr>
<td>McCratty 2003&lt;sup&gt;44&lt;/sup&gt;</td>
<td>Treatment: 72%; waiting list: 71%</td>
<td>NS</td>
<td>Treatment: 48.2 (6.5); waiting list: 43.1 (5.6)</td>
<td>Treatment: 78%; waiting list: 79%</td>
<td>NS</td>
</tr>
<tr>
<td>McGrady 1981&lt;sup&gt;17&lt;/sup&gt;</td>
<td>Treatment: 32%; clinic blood pressure monitor: 31%</td>
<td>Treatment: black: 5%; white: 95% Clinic blood pressure monitor: white: 100%</td>
<td>Treatment: 55; clinic blood pressure monitor: 42</td>
<td>Treatment: 86%; clinic blood pressure monitor: 75%</td>
<td>NS</td>
</tr>
<tr>
<td>McGrady 1994&lt;sup&gt;63&lt;/sup&gt;</td>
<td>Treatment: 34%; waiting list: 48%</td>
<td>Treatment: black: 27%; white: 73% Waiting list: black: 19%; white: 81%</td>
<td>Treatment: 48; waiting list: 49</td>
<td>Treatment: 78%; waiting list: 74%</td>
<td>Treatment: 8.2; waiting list: 8.6</td>
</tr>
<tr>
<td>Patel 1975&lt;sup&gt;35&lt;/sup&gt;</td>
<td>Treatment: 35%; relaxation: 41%</td>
<td>NS</td>
<td>Treatment: 59.5 (range 37–95); relaxation: 58.6 (range 34–75)</td>
<td>Treatment: 86%; relaxation: 100%</td>
<td>NS</td>
</tr>
<tr>
<td>Patel 1988&lt;sup&gt;30&lt;/sup&gt;</td>
<td>Treatment: 51%; no treatment: 50%</td>
<td>NS</td>
<td>Treatment: 35–44 years: 10; 45–54 years: 15; &gt; 55 years: 24 No treatment: 35–44 years: 10; 45–54 years: 17; &gt; 55 years: 24</td>
<td>Treatment: 30%; no treatment: 30%</td>
<td>At least 6</td>
</tr>
</tbody>
</table>

NA, not available; NS, not stated; NSST, non-specific support therapy; SD, standard deviation.

a Data derived from abstract only.
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# Health Technology Assessment programme

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<td>Dr John C Pounsford, Consultant Physician, North Bristol NHS Trust</td>
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<td>Chair</td>
<td>Mrs Val Carlill, Service User Representative</td>
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<td>Mrs Anthea De Barton-Watson, Service User Representative</td>
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<td></td>
<td>Mr Mark Emberton, Senior Lecturer in Oncological Urology, Institute of Urology, University College Hospital, London</td>
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<td>Professor Steve Goodacre, Professor of Emergency Medicine, University of Sheffield</td>
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<td>Professor Christopher Griffiths, Professor of Primary Care, Barts and The London School of Medicine and Dentistry</td>
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<tr>
<td>Chair</td>
<td>Mr Paul Hilton, Consultant Gynaecologist and Urogynaecologist, Royal Victoria Infirmary, Newcastle upon Tyne</td>
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<tr>
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<td>Professor Nicholas James, Professor of Clinical Oncology, University of Birmingham, and Consultant in Clinical Oncology, Queen Elizabeth Hospital</td>
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<td>Dr Peter Martin, Consultant Neurologist, Addenbrooke’s Hospital, Cambridge</td>
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<tr>
<td>Chair</td>
<td>Dr Kate Radford, Senior Lecturer (Research), Clinical Practice Research Unit, University of Central Lancashire, Preston</td>
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<td>Mr Jim Reece, Service User Representative</td>
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<td>Dr Karen Roberts, Nurse Consultant, Dunston Hill Hospital Cottages</td>
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<td>Observers</td>
<td>Dr Philip Leech, Principal Medical Officer for Primary Care, Department of Health</td>
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<td>Ms Kay Pattison, Section Head, NHS R&amp;D Programme, Department of Health</td>
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<td>Dr Morven Roberts, Clinical Trials Manager, Medical Research Council</td>
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<td>Professor Tom Valley, Director, NIHR HTA programme, Professor of Clinical Pharmacology, University of Liverpool</td>
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<td>Dr Ursula Wells, Principal Research Officer, Department of Health</td>
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### Disease Prevention Panel

**Members**

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<thead>
<tr>
<th>Role</th>
<th>Name</th>
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<tr>
<td>Chair</td>
<td>Dr Edmund Jessop, Medical Adviser, National Specialist, National Commissioning Group (NCG), London</td>
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<tr>
<td>Deputy Chair</td>
<td>Dr David Pencheon, Director, NHS Sustainable Development Unit, Cambridge</td>
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<td>Dr Elizabeth Fellow-Smith, Medical Director, West London Mental Health Trust, Middlesex</td>
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<tr>
<td>Chair</td>
<td>Dr John Jackson, General Practitioner, Parkway Medical Centre, Newcastle upon Tyne</td>
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<tr>
<td>Deputy Chair</td>
<td>Dr Chris McCall, General Practitioner, The Hadleigh Practice, Corfe Mullen, Dorset</td>
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<td>Ms Jeannett Martin, Director of Nursing, BarnDoc Limited, Lewisham Primary Care Trust</td>
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<tr>
<td>Chair</td>
<td>Dr Julie Mytton, Locum Consultant in Public Health Medicine, Bristol Primary Care Trust</td>
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<tr>
<td>Deputy Chair</td>
<td>Professor Ian Roberts, Professor of Epidemiology and Public Health, London School of Hygiene &amp; Tropical Medicine</td>
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<td>Professor Ken Stein, Senior Clinical Lecturer in Public Health, University of Exeter</td>
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<tr>
<td>Chair</td>
<td>Dr Kieran Sweeney, Honorary Clinical Senior Lecturer, Peninsula College of Medicine and Dentistry, Universities of Exeter and Plymouth</td>
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<tr>
<td>Deputy Chair</td>
<td>Professor Carol Tannahill, Glasgow Centre for Population Health</td>
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<td>Professor Margaret Thorogood, Professor of Epidemiology, University of Warwick Medical School, Coventry</td>
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<td>Observers</td>
<td>Ms Christine McGuire, Research &amp; Development, Department of Health</td>
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<td>Dr Caroline Stone, Programme Manager, Medical Research Council</td>
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</table>
Expert Advisory Network

Members

Professor Douglas Altman, Professor of Statistics in Medicine, Centre for Statistics in Medicine, University of Oxford

Professor John Bond, Professor of Social Gerontology & Health Services Research, University of Newcastle upon Tyne

Professor Andrew Bradbury, Professor of Vascular Surgery, Solihull Hospital, Birmingham

Mr Shaun Brogan, Chief Executive, Ridgeway Primary Care Group, Aylesbury

Mrs Stella Burnside OBE, Chief Executive, Regulation and Improvement Authority, Belfast

Ms Tracy Bury, Project Manager, World Expert Advisory Network

Professor T A Cameron, Professor of Obstetrics and Gynaecology and Head of the School of Medicine, University of Southampton

Dr Christine Clark, Medical Writer and Consultant Pharmacist, Rosendale

Professor Collette Clifford, Professor of Nursing and Head of Research, The Medical School, University of Birmingham

Professor Barry Cookson, Director, Laboratory of Hospital Infection, Public Health Laboratory Service, London

Dr Carl Counsell, Clinical Senior Lecturer in Neurology, University of Aberdeen

Professor Howard Cuckle, Professor of Reproductive Epidemiology, Department of Paediatrics, Obstetrics & Gynaecology, University of Leeds

Dr Katherine Barton, Information Unit, MIND – The Mental Health Charity, London

Professor Carol Dezateux, Professor of Paediatric Epidemiology, Institute of Child Health, London

Mr John Dunning, Consultant Cardiothoracic Surgeon, Papworth Hospital NHS Trust, Cambridge

Mr Jonathan Earnshaw, Consultant Vascular Surgeon, Gloucestershire Royal Hospital, Gloucester

Professor Martin Eccles, Professor of Clinical Effectiveness, Centre for Health Services Research, University of Newcastle upon Tyne

Professor Pam Enderby, Dean of Faculty of Medicine, Institute of General Practice and Primary Care, University of Sheffield

Professor Gene Feder, Professor of Primary Care Research & Development, Centre for Health Sciences, Barts and The London School of Medicine and Dentistry

Mr Leonard R Fenwick, Chief Executive, Freeman Hospital, Newcastle upon Tyne

Mrs Gillian Fletcher, Antenatal Teacher and Tutor and President, National Childbirth Trust, Henfield

Professor Jayne Franklyn, Professor of Medicine, University of Birmingham

Mr Tam Fry, Honorary Chairman, Child Growth Foundation, London

Professor Fiona Gilbert, Consultant Radiologist and NCRN Member, University of Aberdeen

Professor Paul Gregg, Professor of Orthopaedic Surgical Science, South Tees Hospital NHS Trust

Bec Hanley, Co-director, TwoCan Associates, West Sussex

Dr Maryann L Hardy, Senior Lecturer, University of Bradford

Mrs Sharon Hart, Healthcare Management Consultant, Reading

Professor Robert E Hawkins, CRC Professor and Director of Medical Oncology, Christie CRC Research Centre, Christie Hospital NHS Trust, Manchester

Professor Richard Hobbs, Head of Department of Primary Care & General Practice, University of Birmingham

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Professor Allen Hutchinson, Director of Public Health and Deputy Dean of SchARR, University of Sheffield

Professor Peter Jones, Professor of Psychiatry, University of Cambridge, Cambridge

Professor Stan Kaye, Cancer Research UK Professor of Medical Oncology, Royal Marsden Hospital and Institute of Cancer Research, Surrey

Dr Duncan Kesley, General Practitioner (Dr Burch & Pinns), The Health Centre, Thame

Dr Donna Lamping, Research Degrees Programme Director and Reader in Psychology, Health Services Research Unit, London School of Hygiene and Tropical Medicine, London

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Professor James Lindesay, Professor of Psychiatry for the Elderly, University of Leicester

Professor Julian Little, Professor of Human Genome Epidemiology, University of Ottawa

Professor Alister McGuire, Professor of Health Economics, London School of Economics

Professor Rajan Madhok, Medical Director and Director of Public Health, Directorate of Clinical Strategy & Public Health, North & East Yorkshire & Northern Lincolnshire Health Authority, York

Professor Alexander Markham, Director, Molecular Medicine Unit, St James’s University Hospital, Leeds

Dr Peter Moore, Freelance Science Writer, Ashhead

Dr Andrew Mortimore, Public Health Director, Southampton City Primary Care Trust

Dr Sue Moss, Associate Director, Cancer Screening Evaluation Unit, Institute of Cancer Research, Sutton

Professor Miranda Mugford, Professor of Health Economics and Group Co-ordinator, University of East Anglia

Professor Jim Neilson, Head of School of Reproductive & Developmental Medicine and Professor of Obstetrics and Gynaecology, University of Liverpool

Mrs Julietta Patnick, National Co-ordinator, NHS Cancer Screening Programmes, Sheffield

Professor Robert Peveler, Professor of Liaison Psychiatry, Royal South Hants Hospital, Southampton

Professor Chris Price, Director of Clinical Research, Bayer Diagnostics Europe, Stoke Poges

Professor William Rosenberg, Professor of Hepatology and Consultant Physician, University of Southampton

Professor Peter Sandercock, Professor of Medical Neurology, Department of Clinical Neurosciences, University of Edinburgh

Dr Susan Schonfield, Consultant in Public Health, Hillingdon Primary Care Trust, Middlesex

Dr Eamonn Sheridan, Consultant in Clinical Genetics, St James’s University Hospital, Leeds

Dr Margaret Somerville, Director of Public Health Learning, Peninsula Medical School, University of Plymouth

Professor Sarah Stewart-Brown, Professor of Public Health, Division of Health in the Community, University of Warwick, Coventry

Professor Ala Szczepura, Professor of Health Service Research, Centre for Health Services Studies, University of Warwick, Coventry

Mrs Joan Webster, Consumer Member, Southern Derbsyire Community Health Council

Professor Martin Whittle, Clinical Co-director, National Co-ordinating Centre for Women’s and Children’s Health, Lymington

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The Correspondence Page on the HTA website (www.hta.ac.uk) is a convenient way to publish your comments. If you prefer, you can send your comments to the address below, telling us whether you would like us to transfer them to the website.

*We look forward to hearing from you.*