

# Relaxation therapies for the management of primary hypertension in adults (Review)

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[Intervention Review]

# Relaxation therapies for the management of primary hypertension in adults

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## ABSTRACT

### Background

Lifestyle interventions are often recommended as initial treatment for mild hypertension, but the efficacy of relaxation therapies is unclear.

### Objectives

To evaluate the effects of relaxation therapies on cardiovascular outcomes and blood pressure in people with elevated blood pressure.

### Search methods

We searched the Cochrane Library, MEDLINE, EMBASE, Science Citation Index, ISI Proceedings, ClinicalTrials.gov, Current Controlled Trials and reference lists of systematic reviews, meta-analyses and randomised controlled trials (RCTs) included in the review.

### Selection criteria

Inclusion criteria: RCTs of a parallel design comparing relaxation therapies with no active treatment, or sham therapy; follow-up  $\geq 8$  weeks; participants over 18 years, with raised systolic blood pressure (SBP)  $\geq 140$  mmHg or diastolic blood pressure (DBP)  $\geq 85$  mmHg; SBP and DBP reported at end of follow-up. Exclusion criteria: participants were pregnant; participants received antihypertensive medication which changed during the trial.

### Data collection and analysis

Two reviewers independently extracted data and assessed trial quality. Disagreements were resolved by discussion or a third reviewer. Random effects meta-analyses and sensitivity analyses were conducted.

## Main results

29 RCTs, with eight weeks to five years follow-up, met our inclusion criteria; four were excluded from the primary meta-analysis because of inadequate outcome data. The remaining 25 trials assessed 1,198 participants, but adequate randomisation was confirmed in only seven trials and concealment of allocation in only one. Only one trial reported deaths, heart attacks and strokes (one of each). Meta-analysis indicated that relaxation resulted in small, statistically significant reductions in SBP (mean difference: -5.5 mmHg, 95% CI: -8.2 to -2.8,  $I^2 = 72\%$ ) and DBP (mean difference: -3.5 mmHg, 95% CI: -5.3 to -1.6,  $I^2 = 75\%$ ) compared to control. The substantial heterogeneity between trials was not explained by duration of follow-up, type of control, type of relaxation therapy or baseline blood pressure.

The nine trials that reported blinding of outcome assessors found a non-significant net reduction in blood pressure (SBP mean difference: -3.2 mmHg, 95% CI: -7.7 to 1.4,  $I^2 = 69\%$ ) associated with relaxation. The 15 trials comparing relaxation with sham therapy likewise found a non-significant reduction in blood pressure (SBP mean difference: -3.5 mmHg, 95% CI: -7.1 to 0.2,  $I^2 = 63\%$ ).

## Authors' conclusions

In view of the poor quality of included trials and unexplained variation between trials, the evidence in favour of causal association between relaxation and blood pressure reduction is weak. Some of the apparent benefit of relaxation was probably due to aspects of treatment unrelated to relaxation.

## PLAIN LANGUAGE SUMMARY

### Relaxation for high blood pressure in adults which has no clearly identified cause

The World Health Organisation estimates that high blood pressure leads to over 7 million deaths each year, about 13% of the total deaths worldwide. If people lower their blood pressure, they are less likely to die or to have heart attacks and strokes. If someone's blood pressure is only slightly too high, they may prefer trying to lower it by changing their lifestyle rather than starting on drugs. Although we know that relaxing can counteract the short-term increases in blood pressure that are caused by stress, we don't know if a sustained programme of relaxation can produce long-term reductions in blood pressure or decrease the risk of death, heart attack and stroke.

Our review pooled findings from 1,198 people with blood pressure over 140/85 mmHg who were enrolled in 25 randomised controlled trials. These trials compared the effect of relaxation either with no treatment or with a dummy treatment which wasn't expected to reduce blood pressure. Overall, relaxation reduced blood pressure by a small amount: the average reduction was 5/3 mmHg, but might be anywhere between 8/5 mmHg and 3/2 mmHg. Different trials gave different – sometimes inconsistent – results. Many of the trials were not well designed or conducted. In the good quality trials, relaxation resulted in smaller average reductions in blood pressure and the results could even be consistent with an average increase in blood pressure. Even when all the trials were put together, the combined group of all the people in all the trials wasn't large enough and the trials didn't run for long enough to tell us whether relaxation could reduce the risk of death, heart attack or stroke. Few people reported side-effects of relaxation and, on average, people were just as likely to report side-effects of the comparison treatment.

Different types of relaxation were taught in different trials. It was difficult to disentangle their effects, especially as many trials used a combination of methods. Overall, we found no evidence that autogenic training was effective. Progressive muscle relaxation, cognitive/behavioural therapies and biofeedback seemed to be more likely to reduce blood pressure. However, some of the reduction in blood pressure was almost certainly due to aspects of treatment that were not related to relaxation, such as frequent contact with professionals who were trying to help.

## BACKGROUND

High blood pressure (BP), or hypertension, is associated with a variety of structural changes in the blood vessels and heart which can lead to cardiovascular disease, stroke and renal diseases. It is

one of the ten leading risk factors influencing the global burden of disease and is estimated to lead to over 7 million deaths each year, about 13% of the total deaths worldwide (WHO 2002). Re-

ducing blood pressure levels is associated with significant reduction in cardiovascular and cerebrovascular morbidity and mortality (MacMahon 1990, PSC 2002). The most common form of hypertension, occurring in around 95% of all cases, is primary hypertension which is defined as high blood pressure with no identifiable cause (Brown 1997). Secondary hypertension is high blood pressure with an identifiable cause, e.g. renal disease or endocrine disturbances.

There is substantial evidence that stress can lead to short term elevation of blood pressure (Gibbons 1998). The physiological response to a stressor can be described by a three stage model: a short-term alarm reaction ('fight or flight') where the body responds to a stressor with sympathetic nervous system activity leading to increased blood pressure; resistance reaction where the body continues to fight the stressor after the initial alarm reaction passes; finally, exhaustion occurs if the body is unable to maintain this resistance (Schwartz 2003). Furthermore, hypertension is a physical symptom of the exhaustion stage when the body cannot maintain resistance to a stressor (von Onciul 1996).

While short term stress can lead to elevated blood pressure, the relevance of this to sustained hypertension remains unclear (Pickering 1991). Although some researchers have observed higher blood pressure while people were at work than when they were at home and others have reported an association between systolic blood pressure and the perception of having a stressful job, no strong epidemiological evidence exists for an association between stress and sustained hypertension. The physiological mechanisms that might link stress to the development of sustained hypertension are unclear. Long term hypertension may be caused by separate factors from those which cause short term elevation of blood pressure (Schwartz 2003). While short term blood pressure elevation can be attributed to sympathetic nervous system activity, long term changes may be perpetuated by vascular remodeling and endothelial dysfunction (Gibbons 1998).

A number of heterogeneous therapies that aim to reduce stress and encourage relaxation have been investigated for the treatment of hypertension. The relaxation response is the opposite of the fight or flight phenomenon; it reduces blood pressure and lessens the harmful effects of stress (Benson 1984). It is often elicited by repetition of a word or phrase, while adopting a passive attitude and decreased muscular tone (Eisenberg 1993). *Autogenic training* is a relaxation technique focusing on physical sensations of e.g. breathing or heartbeat, assisted by self-suggestion (Stetter 2002). It aims to elicit the relaxation response through repetitive mental focus and adoption of a passive attitude (Mandle 1996). *Cognitive therapy* teaches the individual to recognise and change irrational thought processes behind problematic emotions and so may modify the individual's response to stress (Astin 2003). *Behavioural therapy* uses reinforcements (e.g. rewarding or not rewarding specific behaviours) to change or elicit desired behavioural responses and so may likewise be useful in helping people deal with stress

(Astin 2003). *Meditation* includes various techniques for focusing the individual's attention and calming their thoughts (Astin 2003). *Guided imagery* requires the individual to focus on calming images with the goal of achieving relaxation (Astin 2003). *Biofeedback* is a therapeutic procedure where the individual is trained to alter a physiological response (e.g. blood pressure) through receiving visual or auditory feedback about the response (Astin 2003). *Progressive muscle relaxation* encourages relaxation through awareness of the sensation in the main muscle groups and is often accompanied by breathing exercises and guided imagery (Huntley 2002). *Breathing exercises* require the individual to maintain slow and regular breathing which may directly influence the cardiovascular system (Grossman 2001). *Yoga* techniques usually include stretching, postural and breathing exercises and meditation (Engbretson 2002).

We found five previous systematic reviews which included meta-analyses of a variety of relaxation therapies for treating hypertension (Eisenberg 1993; Linden 1994; Ebrahim 1998; Stetter 2002; Nakao 2003). These meta-analyses require updating, for several reasons. Firstly, although the earlier meta-analyses (Eisenberg 1993, Linden 1994 and Ebrahim 1998) considered a range of relaxation therapies, the recent meta-analyses (Stetter 2002; Nakao 2003) were restricted to autogenic training and biofeedback respectively. Secondly, these reviews provided conflicting evidence. Ebrahim 1998 aggregated studies of a variety of relaxation therapies and found that relaxation was associated with a very small overall reduction of 1/1 mmHg in blood pressure. Eisenberg 1993 found that relaxation was superior to no treatment but not to a credible sham therapy. Nakao 2003 found similar results for biofeedback. Linden 1994 found relaxation therapies were effective in reducing blood pressure. Stetter 2002 found autogenic training to be more effective than sham therapy for mild-to-moderate primary hypertension. Thirdly, only one of these meta-analyses (Ebrahim 1998) was restricted to studies with a minimum length of follow up (6 months); the meta-analyses of Stetter 2002 and Nakao 2003 included trials which lasted only two and three weeks respectively; those of Eisenberg 1993 and Linden 1994 did not report length of follow-up. Since treatment for hypertension is likely to be a life-long process, short-term studies may not be relevant to establishing the benefits of long-term treatment. Finally, some types of relaxation therapies may be effective in reducing blood pressure, while others may not (Linden 1994; Eisenberg 1993). Therefore there is a need for an up-to-date review of all relaxation treatments for lowering raised blood pressure, which excludes short-term studies and considers the possible differences between the effects of different therapies.

The aim of this review was to summarise the evidence about the benefits and harms of relaxation therapies for patients with primary hypertension, in order to inform decisions about recommendations for treatment.

## OBJECTIVES

To evaluate the effects of relaxation therapies on cardiovascular outcomes and blood pressure in adults with primary hypertension.

## METHODS

### Criteria for considering studies for this review

#### Types of studies

Randomised controlled trials of a parallel design that had an intervention period of at least 8 weeks and allowed an intention-to-treat analysis; “intention-to-treat” was interpreted as meaning that participants were analysed in the treatment groups to which they were assigned (see section 8.4.1 of [Deeks 2006](#))

#### Types of participants

Adults over 18 years of age, with elevated blood pressure (a minimum of 140 mmHg for SBP or 85 mmHg for DBP), without a known primary cause. The inclusion criterion for diastolic blood pressure is slightly lower than the standard (90 mmHg) for hypertension ([JNC VII 2003](#)), in order to include individuals at the lower end of the spectrum of raised blood pressure.

We excluded:

- studies of pregnant women, since hypertension during pregnancy is often due to syndromes such as preeclampsia, with a pathophysiology very different from that of other forms of hypertension ([NIH 2000](#));
- studies including participants on antihypertensive medication which was allowed to vary during the course of the study, since the results of such studies are difficult to interpret.

#### Types of interventions

- Intervention designed to promote relaxation.
- Control:
  - (i) no active treatment: this included usual treatment, or BP monitoring only; or
  - (ii) sham therapy designed to control for non-specific features of the treatment setting, in particular an equivalent level of treatment time and therapist contact, a highly credible treatment rationale, a high level of patient motivation and involvement with therapy. We excluded trials which evaluated a combination of relaxation therapies and other interventions such as diet or exercise. However, if participants in the treatment group received relaxation therapies and all participants received the same additional interventions, the trial was included.

#### Types of outcome measures

##### Primary outcome measures:

- death from all causes;
- coronary heart disease events (fatal or non-fatal myocardial infarction, excluding heart failure and if possible angina);
- cerebrovascular events (fatal or non-fatal strokes, excluding transient ischaemic attacks if possible);
- SBP at end of follow-up;
- DBP at end of follow-up.

If the mean or standard deviation of final BP was not reported, the difference in BP between baseline and the end of the study (change score) was used instead, if its standard deviation was available. If BP was measured both supine and standing, supine measurements were preferred; if BP was measured both sitting and supine, sitting measurements were preferred. If blood pressure was measured in clinic and at home or in the workplace, clinic measurements were preferred. If only ambulatory blood pressure was measured, 12/7 was added to these measurements, as the British Hypertension Society recommends an upward correction of 12/7 mmHg to ambulatory values before comparing them with clinic values ([Ramsay 1999](#)).

##### Secondary outcome measures:

- adverse events, categorised as uncontrolled hypertension and other;
- total withdrawals from treatment;
- withdrawals from treatment due to adverse events, categorised as uncontrolled hypertension and other.

#### Search methods for identification of studies

We searched the following for randomised controlled trials (RCTs):

- Cochrane Library (2007 issue 1)
- MEDLINE (2000 - Feb 2007)
- EMBASE (1999 - Feb 2007)
- Science Citation Index (1982 - Feb 2007)
- ISI Proceedings (1999 - Feb 2007)
- ClinicalTrials.gov
- Current Controlled Trials

Since the Cochrane Library incorporates RCTs from MEDLINE and EMBASE, searches of these databases were restricted to recent years.

We also identified systematic reviews and meta-analyses from these databases and checked their reference lists, as well as those of randomised controlled trials included in the review.

We based the search on the following strategy (where terms in capitals are MeSH headings). This strategy was constructed and developed further within MEDLINE, and was adjusted accordingly for the other databases:

#1 HYPERTENSION/

#2 hypertens\$.tw  
 #3 (blood ADJ pressure).tw  
 #4 #1 OR #2 OR #3  
 #5 Exp Mind-Body and Relaxation Techniques/  
 #6 (exercis\$ OR meditat\$ OR bio-feedback\$ OR biofeedback\$ OR yoga OR yogic OR breathing OR behaviour\$ OR behavior\$.tw  
 #7 (muscle ADJ3 (relax\$ OR stretch\$)).tw  
 #8 (therap\$ OR training OR education OR management OR technique\$) ADJ3 (relax\$ OR stress OR cognitive OR talk\$ OR assertiveness OR anger)  
 #9 #5 OR #6 OR #7 OR #8  
 #10 Cochrane highly sensitive search strategy (Dickersin 1994)  
 #11 #4 AND #9 AND #10

We also carried out a general web search using the search engines Google, Zapmeta and Dogpile, and searched the websites of the following organizations: Blood Pressure Association, British Hypertension Society, American Society of Hypertension, and Canadian Hypertension Society. We searched the reference lists of a recent review of meditation for healthcare which had searched databases specialising in complementary and alternative medicine (CAMPAIN, Cochrane Complementary Medicine Trials Register, PsychInfo, CINAHL and AMED) (AHRQ 2007); we contacted an expert who had reviewed studies on yoga published in Indian journals (Khalsa 2004).

There was no language restriction.

Following referees' comments, we recommend that the following databases should also be searched in any update of the review: CAMPAIN (Complementary and Alternative Medicine and Pain Database), Cochrane Complementary Medicine Trials Register, PsychInfo, CINAHL and AMED and the Indian Medlars Centre (<http://www.indmed.nic.in>).

## Data collection and analysis

### Identification of included studies

All titles and abstracts retrieved by electronic searching were screened independently by two reviewers and those studies which clearly did not meet the inclusion criteria were excluded. Copies of the full text of potentially relevant references were obtained and their eligibility was assessed by one of two methods: one reviewer was primary assessor and decisions were checked by a second reviewer or assessments were done independently by two reviewers. Differences between reviewers were resolved by discussion or by appeal to a third reviewer.

### Quality assessment

Methodological quality of included trials was assessed independently by two reviewers using the following criteria:

#### Blinding

We coded the blinding of participants, treatment providers and outcome assessors as:

- yes

- no
- unclear.

### Randomisation

We coded the randomisation of participants to intervention groups as:

- adequate e.g. a computer-generated random sequence or a table of random numbers;
- inadequate e.g. date of birth, clinic id-number or surname;
- unclear e.g. not reported.

### Allocation concealment

We coded the concealment of allocation sequence from treatment providers and participants as:

- adequate (A) i.e. where the allocation sequence could not be foretold;
- inadequate (B) e.g. a method of allocation which allowed treatment providers to predict which arm of the trial the next participant was assigned to;
- unclear (C) e.g. not reported.

### Loss to follow-up

We recorded the number of participants in each intervention arm whose blood pressure was not reported at the end of the study. We noted if loss to follow-up was not reported.

Two reviewers independently abstracted endpoint data and data describing the trial quality, study population (country in which the study was conducted, inclusion criteria, patient characteristics at baseline: age, gender, ethnicity, mean blood pressure, whether previously treated with antihypertensive drugs), and interventions (treatment provider, type of relaxation therapy; duration of intervention and follow-up) using a pre-specified form. Differences were reconciled by discussion or by consultation with a third reviewer. All corresponding authors were contacted for missing endpoint data: three replied supplying us with unpublished data (Canino 1994; Murugasan 2000; Schein 2001); two letters were returned as they did not reach the intended recipient; one author replied but was unable to supply the requested data.

We categorised the components of active interventions as: biofeedback; cognitive/behavioural therapy (including meditation, yoga and guided imagery); progressive muscle relaxation, or autogenic training.

### Statistical methods

The findings of included trials were aggregated in meta-analyses using Review Manager 4.2.8.

We planned to meta-analyse deaths, heart attacks and strokes, if more than one trial reported the outcome, by calculating a relative risk for each outcome for each trial and combining these using a random effects model (DerSimonian 1986).

For blood pressure, the mean difference (and standard deviation) between final blood pressure for relaxation and control interventions for both SBP and DBP was calculated. If standard deviations of final values were not available, change scores were used if their standard deviations were available. If trials had more than one



treatment arm (Achmon 1989; Bennett 1991; Blanchard 1979; Hafner 1982), we used a weighted mean of the outcome for all treatment arms; for trials with more than one control arm (Canino 1994; Frankel 1978; LaGrone 1988; Seer 1980) we likewise used a weighted mean of the outcome for all control arms. Mean differences were weighted according to the precision of each trial and combined in meta-analyses using a random effects model (DerSimonian 1986), to estimate an overall pooled mean difference and its 95% confidence interval (CI). Heterogeneity between trials was assessed using the  $I^2$  statistic (Higgins 2003).

Sub-group analyses were performed grouping the trials by:

- duration of follow-up: <6 months; 6 months and over;
- type of control: sham therapy, no active treatment;
- whether the active intervention included: biofeedback,

cognitive/behavioural therapy, progressive muscle relaxation, autogenic training.

For trials with two control arms (both sham therapy and no active treatment: Canino 1994; Frankel 1978; Seer 1980), these sub-group analyses compared half of the participants in the treatment group with the sham therapy group and half with the no active treatment control group. Likewise, for trials with two active intervention arms of different types (Achmon 1989), half the participants in the control group were compared with one active intervention arm and half with the other.

Although not specified in the original protocol, trials were also sub-grouped by whether the initial mean blood pressure among participants was above or below the median for all trials, as it seemed plausible that trials in which participants had a higher initial blood pressure would be more likely to show an effect of relaxation. Additionally, the comparison of biofeedback with control was sub-grouped by type of control (sham therapy/no active treatment) for comparison with the systematic review of Nakao 2003.

Sensitivity analyses were performed excluding trials which did not report (i) adequate concealment of allocation, (ii) blinding of the outcome assessor. Further sensitivity analyses were performed im-

puting standard deviations to those trials for which they were unavailable, using the highest and lowest SBP and DBP standard deviations for intervention and control in the primary meta-analysis. We assessed the tolerability of the intervention by calculating the difference in the rate of withdrawal in treatment and control arms, using a random effects model to calculate a pooled risk difference. We used the same methods to assess adverse events.

## RESULTS

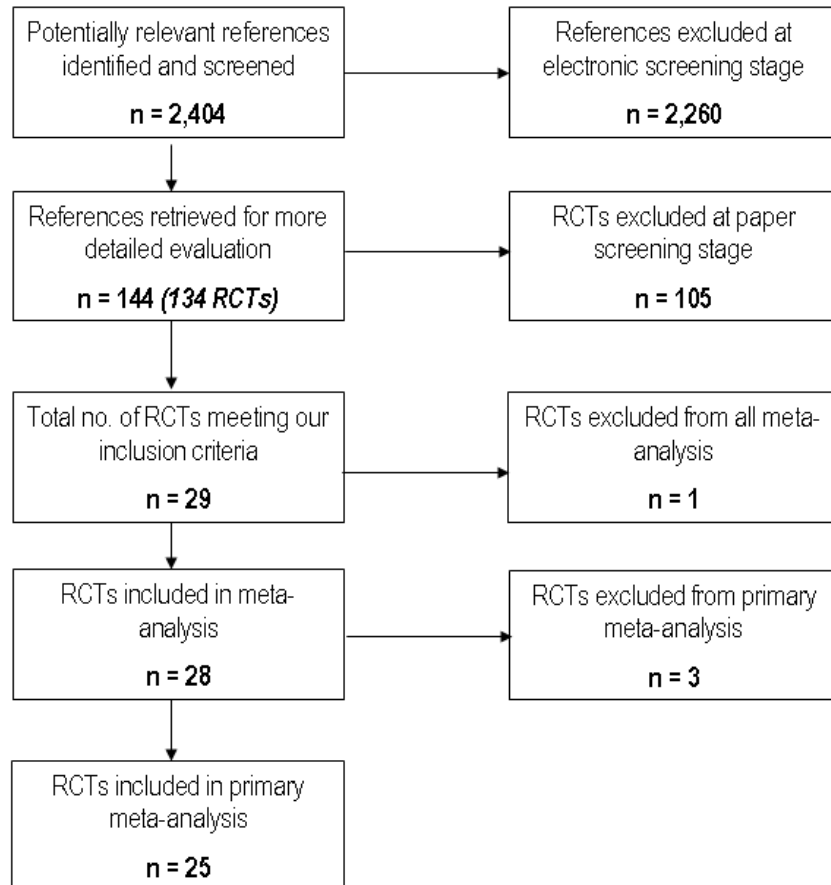
### Description of studies

See: [Characteristics of included studies](#); [Characteristics of excluded studies](#).

#### Identification of included studies

The search strategy found 2,404 potentially relevant references which we electronically screened (see Additional Figures: [Figure 1](#)). We excluded 2,260 references and retrieved 144 references, describing 134 studies, for detailed evaluation. We excluded 105 of these studies for the following reasons: not randomised (27 trials); antihypertensive medication varied during the trial (21 trials); normotensive participants (17 trials); unclear if participants were normotensive or hypertensive (1 trial), no control group (13 trials); less than 8 weeks follow-up (12 trials); no outcome BP reported (6 trials); data allowing an intention to treat analysis not reported (4 trials); participants were children (1 trial); comment/editorial (1 study); no control group outcome BP reported (1 trial); crossover design (1 trial) - see table *Characteristics of Excluded Studies*. The remaining 29 RCTs met our inclusion criteria and are described in the table *Characteristics of Included Studies*. We excluded four RCTs from the primary meta-analysis because of missing standard deviations (Bosley 1989; Hafner 1982; Khramelashvili 1986; LaGrone 1988).

**Figure 1. Fig 1. Flow chart: Identification of included trials**



## **Trials included in primary meta-analysis**

### **Design of trials**

The 25 RCTs included in the primary meta-analysis enrolled 1,419 participants in relaxation and control arms, of whom 1,198 were assessed; some trials (Amigo 1997; Yen 1996 - see *Characteristics of Included Studies*) included other arms which were not considered in this review. One trial was a 2 X 2 factorial trial (Adsett 1989), so we aggregated the relaxation intervention and control over the other interventions ( $\beta$ -blocker and placebo). One trial (Yen 1996) randomised communities rather than individuals, so the numbers of participants were adjusted to allow for this cluster randomisation (see section 8.11.2.2 of Deeks 2006).

### **Participants**

The number of participants in each trial ranged from 16 to 171 (median: 43). There was substantial heterogeneity between trials in the characteristics of the participants enrolled. Entry criteria varied between trials, and participants' SBP ranged from 130 to 164 mmHg (median: 144, inter-quartile range: 139-150 mmHg) and their DBP from 86 to 109 mmHg (median: 92, IQR: 89-97 mmHg) at baseline. Thirteen trials (Achmon 1989; Aivazyan 1988b; Amigo 1997; Blanchard 1979; Frankel 1978; Garcia-Vera 1997; Irvine 1986; McGrady 1981; McGrady 1994; Patel 1988; Schein 2001; Yen 1996; Zurawski 1987) enrolled a mixture of participants who were and were not being treated with antihypertensive medication; nine trials (Adsett 1989; Bennett 1991; Blanchard 1996; Canino 1994; Cottier 1984; Irvine 1991; Johnston 1993; Seer 1980; van Montfrans 1990) enrolled only participants who were not currently receiving antihypertensive medication, although two of these trials (Blanchard 1996; Cottier 1984) enrolled participants who had previously been on medication; one trial (Jacob 1992) enrolled only participants who were receiving antihypertensive medication; two trials (Carson 1988; Murugasan 2000) did not specify the medication status of participants. One trial (Bennett 1991) enrolled only participants with a "Type A" personality: a tendency to anger and hostility. Four trials were carried out in settings relevant to routine clinical care: Adsett 1989 conducted the trial at a worksite; in the trial of Patel 1988 the intervention was delivered by primary care physicians and nurses in their own practices; in the trial of Garcia-Vera 1997 treatment sessions were delivered at the participant's usual health centre; and Yen 1996 delivered the intervention to participants in their own home. Other trials delivered the interventions in settings which were more appropriate to research than to routine care. Participants were enrolled through: referrals from primary care physicians (Achmon 1989; Bennett 1991; Garcia-Vera 1997; Irvine 1986; Johnston 1993; Patel 1988; Seer 1980), their workplace (Adsett 1989; Irvine 1991), community screening (Yen 1996); a community public health centre (Zurawski 1987), referrals from secondary care facilities (Amigo 1997; Canino 1994; Carson 1988; Cottier 1984;

Frankel 1978; Murugasan 2000), a combination of sources (Jacob 1992) or advertisements for paid volunteers (Blanchard 1979); in other trials (Aivazyan 1988b; Blanchard 1996; McGrady 1981; McGrady 1994; Schein 2001; van Montfrans 1990) the source of participants was unclear. All trials reported gender and 63% of the participants were male. The overall mean age, reported in all trials except Patel 1988 and Murugasan 2000, was 47 years (range: 18 to 73 years).

Only six trials reported ethnicity (Blanchard 1996; Frankel 1978; Jacob 1992; McGrady 1981; McGrady 1994; Zurawski 1987) and in these 84% of the participants were white. Ten trials were conducted in the USA, three in the UK; two in Canada; two in Israel; two in Spain; one in the Netherlands; one in New Zealand; one in the USSR; one in Taiwan, one in India and one in Venezuela.

### **Treatments**

#### *Active interventions*

Three trials (Achmon 1989; Bennett 1991; Blanchard 1979) had multiple treatment arms. Several trials combined components from several types of relaxation therapies in one active treatment arm. Relaxation interventions included progressive muscle relaxation (16 trials); biofeedback (12 trials); autogenic training (3 trials) and cognitive or behavioural therapy or meditation (11 trials) - see Table 1. Within these categories, interventions were heterogeneous:

- Biofeedback included biofeedback of heart rate (Achmon 1989), blood pressure (Blanchard 1979), DBP and ECG (Frankel 1978), forehead muscle tension (Blanchard 1979; McGrady 1981), temperature of fingers, hands or feet (Blanchard 1996; Canino 1994; Jacob 1992; McGrady 1994), skin resistance (Irvine 1986; Irvine 1991; Patel 1988) and breathing (Schein 2001). It was generally used in combination with progressive muscle relaxation, sometimes also with cognitive and behavioural therapy, but in one trial (McGrady 1981) it was used with autogenic training alone and in one trial (Schein 2001) it was not combined with any other therapy.
- Cognitive and behavioural therapies included anger control (Achmon 1989; Bennett 1991; Patel 1988), stress management (Garcia-Vera 1997; Irvine 1986; Johnston 1993; Patel 1988; Zurawski 1987), coping strategies (Bosley 1989; Patel 1988; Zurawski 1987), anxiety management (Canino 1994), time management (Bennett 1991), assertiveness training (Achmon 1989; Bennett 1991), behavioural assignments (Bennett 1991; Zurawski 1987), meditation (Bennett 1991; Irvine 1991; Patel 1988; Seer 1980; van Montfrans 1990), yoga (Murugasan 2000), communication skills (Patel 1988).
- Most trials which taught progressive muscle relaxation encouraged participants to practise at home, often with the help of taped instructions (Adsett 1989; Canino 1994; Carson 1988; Cottier 1984; Frankel 1978; Garcia-Vera 1997; McGrady 1994;

Patel 1988; van Montfrans 1990; Yen 1996 ); some trials (Cottier 1984; Garcia-Vera 1997; Patel 1988) also encouraged participants to practise these relaxation techniques in stressful situations.

Relaxation therapies were delivered to participants in one of three ways. In 12 trials the intervention arm received the relaxation therapy in a group setting (Achmon 1989; Adsett 1989; Bennett 1991; Blanchard 1996; Canino 1994; Carson 1988; Frankel 1978; Jacob 1992; McGrady 1994; Patel 1988; van Montfrans 1990; Zurawski 1987), in eight trials therapies were delivered to participants individually (Amigo 1997; Cottier 1984; Garcia-Vera 1997; Irvine 1986; Irvine 1991; Johnston 1993; Schein 2001; Yen 1996). In five trials it was unclear whether participants received their intervention in a group or singly (Aivazyan 1988b; Blanchard 1979; McGrady 1981; Murugasan 2000; Seer 1980).

The median duration of treatment was 8 weeks (range: 5 to 26 weeks).

#### Controls

Three trials (Canino 1994; Frankel 1978; Seer 1980) had multiple control arms. Control groups received sham therapy (15 trials) or no active intervention (14 trials).

The sham therapies used varied considerably between trials: most were some form of group therapy; in some trials the sham therapy was designed to mimic all the components of the active intervention except that which the investigators believed to be effective; in other trials the sham therapy was less specific. Three trials (Amigo 1997; Irvine 1986; Johnston 1993) included mild physical exercise in the sham therapy; two trials (Irvine 1986; Zurawski 1987) included biofeedback of galvanic skin resistance; one trial (Frankel 1978) provided sham biofeedback of blood pressure; several trials encouraged relaxation (Blanchard 1979; Canino 1994; Carson 1988; van Montfrans 1990); several included counselling on stress (Bosley 1989; Jacob 1992; van Montfrans 1990); several trials (Adsett 1989; Bosley 1989; van Montfrans 1990) delivered education on hypertension; one trial used meditation without use of a mantra (Seer 1980); one (Schein 2001) used listening to synthesized music; one (Achmon 1989) delivered two lectures to encourage anticipation of reduction in blood pressure; one (Irvine 1991) delivered non-specific support therapy. Hence some trials used sham therapies which were similar to the active interventions in other trials.

Three trials (Blanchard 1996; Cottier 1984; McGrady 1981) which had no active control intervention monitored participants' blood pressure; two trials (Blanchard 1996; Garcia-Vera 1997) arranged for participants to measure their own blood pressure at home twice daily.

#### Outcomes

##### *Death, myocardial infarction and stroke*

No trials were designed to assess deaths or cardiovascular endpoints and only one trial (Patel 1988) reported deaths, heart attacks and strokes.

#### *Blood pressure*

Eighteen trials reported final values of blood pressure (Achmon 1989; Amigo 1997; Bennett 1991; Blanchard 1979; Blanchard 1996; Canino 1994; Frankel 1978; Garcia-Vera 1997; Irvine 1986; Irvine 1991; Johnston 1993; McGrady 1981; McGrady 1994; Murugasan 2000; Schein 2001; Seer 1980; Yen 1996; Zurawski 1987) and seven reported only change scores (Adsett 1989; Aivazyan 1988b; Carson 1988; Cottier 1984; Jacob 1992; Patel 1988; van Montfrans 1990).

Final blood pressure was measured in clinic in all trials except that of Yen 1996, in which it was measured at home; in most trials the participant was seated; in nearly all trials final blood pressure was averaged over two or more readings; about half the trials measured blood pressure using a mercury sphygmomanometer and about half used an automatic device.

#### *Adverse events*

Seven trials (Adsett 1989; Blanchard 1996; Cottier 1984; Irvine 1991; Patel 1988; Seer 1980; van Montfrans 1990) reported, by treatment arm, the numbers of participants who experienced adverse events. These adverse events were usually uncontrolled hypertension, but also included angina, heart failure, kidney damage, thrombosis of retinal vessels, a broken rib, cancer, chest pain, drug complications and an unspecified medical problem. The criteria used for uncontrolled hypertension were not consistent between trials: Adsett 1989 defined it as DBP>105 mmHg, Cottier 1984 and van Montfrans 1990 as DBP>115 mmHg and Irvine 1991 and Seer 1980 as starting on anti-hypertensive medication.

#### *Withdrawals*

Fourteen trials reported the number of withdrawals from treatment by treatment arm (Achmon 1989; Adsett 1989; Amigo 1997; Bennett 1991; Blanchard 1979; Blanchard 1996; Canino 1994; Irvine 1986; Jacob 1992; Johnston 1993; McGrady 1981; Murugasan 2000; Schein 2001; van Montfrans 1990). A further four trials reported overall withdrawal from treatment (Garcia-Vera 1997; Irvine 1986; Patel 1988; Zurawski 1987).

#### *Withdrawals due to adverse events*

Five trials (Adsett 1989; Blanchard 1996; Cottier 1984; Irvine 1991; van Montfrans 1990) reported numbers of withdrawals due to adverse events. Additionally, three trials (Canino 1994; Frankel 1978; Jacob 1992) reported no withdrawals in either arm and Johnston 1993 reported the total number of withdrawals due to adverse events.

#### **Follow-up**

The median duration of follow-up was 20 weeks (range: 8 weeks to 5 years); 13 trials had follow-up of less than 6 months and 12 trials had follow-up of 6 months or more.

#### **Trials not included in primary meta-analysis**

Three additional trials (Hafner 1982; Khramelashvili 1986; LaGrone 1988), enrolling 142 participants, were included in meta-analysis by imputing standard deviations. Hafner 1982 compared meditation, both with and without biofeedback, with non-intervention controls; LaGrone 1988 compared progressive muscle re-

laxation with both sham therapy and non-intervention controls; [Khramelashvili 1986](#) compared autogenic training and biofeedback with non-intervention controls. [Hafner 1982](#) and [LaGrone 1988](#) reported that 10/62 (16%) participants withdrew from treatment.

One further trial ([Bosley 1989](#)) could not be included in any meta-analysis as neither the numbers of participants enrolled nor the number assessed at the end of follow-up were reported by treatment group in the two control groups. This trial enrolled 41 participants in three arms - cognitive self-management training, sham therapy and non-intervention controls; adverse events and withdrawals from treatment were not reported and loss to follow-up was not reported by treatment group.

### Risk of bias in included studies

#### Trials included in primary meta-analysis

Although all 25 trials included in the primary meta-analysis claimed to be randomised, the method of randomisation was confirmed to be adequate in only seven trials ([Adsett 1989](#); [Cottier 1984](#); [Frankel 1978](#); [Johnston 1993](#); [Patel 1988](#); [Schein 2001](#); [van Montfrans 1990](#)). Concealment of allocation could be confirmed as adequate in only one trial ([Patel 1988](#)). Nine trials reported blinding of outcome assessors ([Achmon 1989](#); [Adsett 1989](#); [Amigo 1997](#); [Frankel 1978](#); [Jacob 1992](#); [Irvine 1991](#); [Johnston 1993](#); [Schein 2001](#); [Yen 1996](#)); one trial blinded both participants and treatment providers ([Schein 2001](#)). The remaining trials did not clearly report blinding.

Fourteen trials ([Achmon 1989](#); [Adsett 1989](#); [Amigo 1997](#); [Blanchard 1979](#); [Blanchard 1996](#); [Canino 1994](#); [Frankel 1978](#); [Irvine 1991](#); [Jacob 1992](#); [Johnston 1993](#); [McGrady 1981](#); [Murugasan 2000](#); [Schein 2001](#); [van Montfrans 1990](#)) reported loss to follow-up by treatment arm and, in these, 15% of participants were lost to follow-up. If loss to follow-up was not reported, we assumed that no participants were lost to follow-up.

Six trials ([Aivazyan 1988b](#); [Bosley 1989](#); [Carson 1988](#); [Patel 1988](#); [Yen 1996](#); [Zurawski 1987](#)) which included participants currently receiving antihypertensive medication did not clearly report whether the investigators attempted to keep this medication unchanged throughout the trial. Four trials ([Garcia-Vera 1997](#); [Irvine 1991](#); [McGrady 1994](#); [Seer 1980](#)) which attempted to keep antihypertensive medication constant throughout the trial excluded from analysis the few participants who altered their medication.

#### Trials not included in primary meta-analysis

Neither randomisation nor concealment of allocation was confirmed to be adequate in any of the three additional trials ([Hafner 1982](#); [LaGrone 1988](#); [Khramelashvili 1986](#)) which were included in meta-analysis by imputing standard deviations. In the trial of [LaGrone 1988](#), outcome assessors were blinded and antihyperten-

sive medication remained unchanged during the trial, but in the trials of [Hafner 1982](#) and [Khramelashvili 1986](#) these criteria were unclear. In the trials of [Hafner 1982](#) and [LaGrone 1988](#), 10/62 (10%) participants were lost to follow-up.

In the trial [Bosley 1989](#), which was excluded from meta-analysis, the adequacy of randomisation and concealment of allocation were unclear, but outcome assessors were blinded.

### Effects of interventions

#### **Relaxation vs. control: Death - see comparison 1, outcome 1.**

Only one trial reported deaths ([Patel 1988](#)) and in this only one death occurred, in the relaxation group, in 111 participants followed up. Relaxation was not associated with any significant difference in the risk of death (RR comparing relaxation with control = 3.2, 95%CI: 0.1 to 76).

#### **Relaxation vs. control: Myocardial infarction - see comparison 1, outcome 2.**

Only one trial reported myocardial infarctions ([Patel 1988](#)) and in this only one occurred, in the control group, in 103 participants assessed. Relaxation was not associated with any significant difference in the risk of myocardial infarction (RR comparing relaxation with control = 0.4, 95%CI: 0.02 to 8.8).

#### **Relaxation vs. control: Stroke - see comparison 1, outcome 3.**

Only one trial reported strokes ([Patel 1988](#)) and in this only one occurred, in the relaxation group, in 103 participants assessed. Relaxation was not associated with any significant difference in the risk of stroke (RR comparing relaxation with control = 3.3, 95%CI: 0.1 to 79).

#### **Relaxation vs. control: Primary meta-analysis of BP - see comparison 2.**

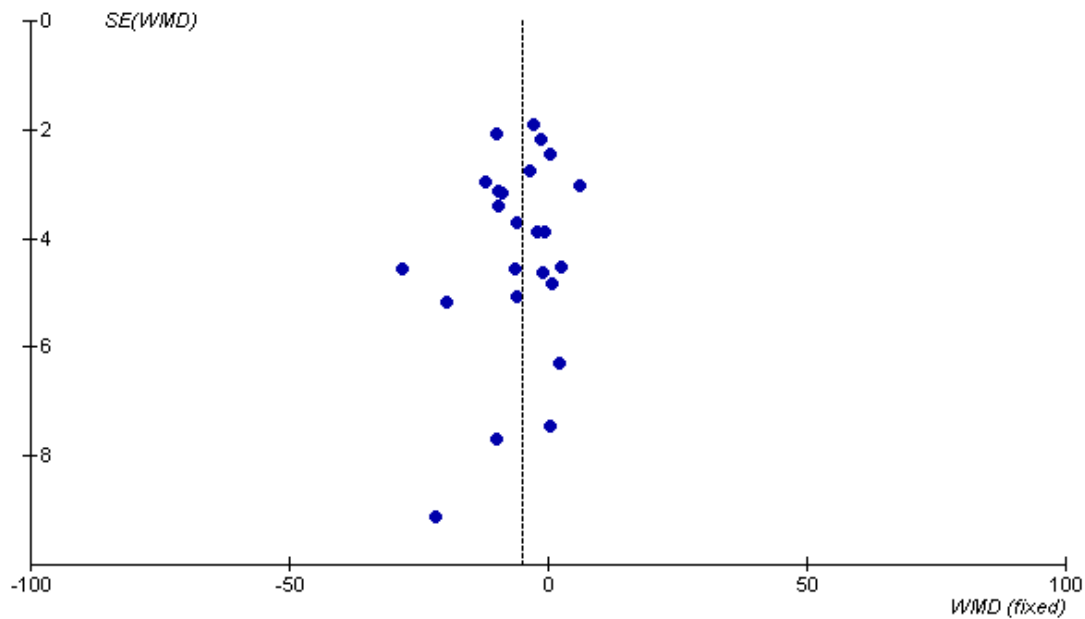
Six trials found a statistically significant reduction in both SBP and DBP favouring relaxation interventions ([Achmon 1989](#); [Aivazyan 1988b](#); [Canino 1994](#); [Garcia-Vera 1997](#); [Murugasan 2000](#); [Patel 1988](#)). One trial found a significant reduction in SBP alone ([Carson 1988](#)); and two trials reported a significant reduction in DBP alone ([Amigo 1997](#); [Cottier 1984](#)).

Meta-analysis of 25 trials, assessing 1,198 participants, found relaxation was associated with statistically significant reductions in both SBP (mean difference: -5.5 mmHg, 95% CI: -8.2 to -2.8) and DBP (mean difference: -3.5 mmHg, 95% CI: -5.3 to -1.6) compared to control. There was substantial heterogeneity for both SBP ( $I^2 = 72%$ ) and DBP ( $I^2 = 75%$ ).

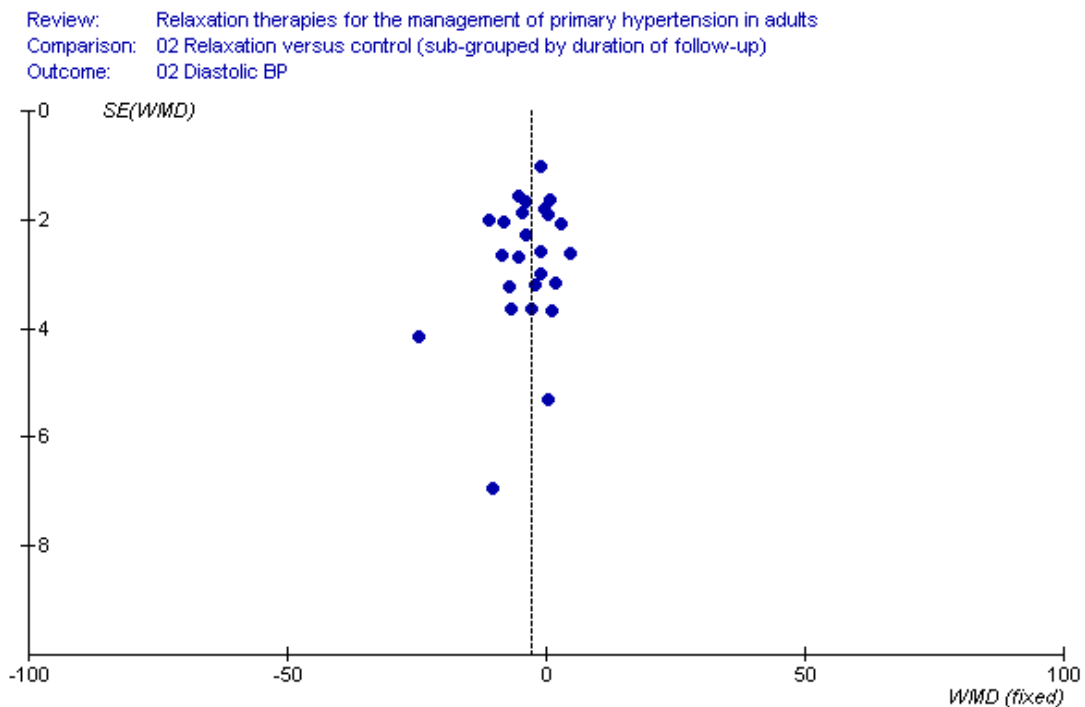
We considered the potential for small study effects by visually examining funnel plots of SBP and DBP outcome data; these provided little evidence of publication bias (see [Figure 2](#); [Figure 3](#)).

**Figure 2. Fig 2. SBP Funnel.** SBP funnel plot: standard error of estimated treatment effect vs. estimated treatment effect

Review: Relaxation therapies for the management of primary hypertension in adults  
Comparison: 02 Relaxation versus control (sub-grouped by duration of follow-up)  
Outcome: 01 Systolic BP



**Figure 3. Fig 3. DBP funnel. DBP funnel plot: standard error of estimated treatment effect vs. estimated treatment effect**



One small trial (Murugasan 2000), which did not confirm adequate randomisation, concealment of allocation or blinding, reported a very large net decrease of 28/25 mmHg in BP in the relaxation group and had substantial influence on the results. Exclusion of this trial from the meta-analysis resulted in lower overall reductions in BP and less heterogeneity (SBP mean difference: -4.6 mmHg, 95% CI: -6.9 to -2.2,  $I^2 = 62%$ ; DBP mean difference: -2.9 mmHg, 95% CI: -4.5 to -1.3,  $I^2 = 66%$ ).

**Relaxation vs. control: BP, sub-grouped by duration of follow-up - see comparison 2.**

In the 13 short-term trials, assessing 590 participants, with follow-up of less than 6 months, relaxation was associated with statistically significant reductions in both SBP (mean difference: -7.1 mmHg, 95% CI: -11.4 to -2.8) and DBP (mean difference: -5.1 mmHg, 95% CI: -8.4 to -1.9). There was substantial heterogeneity for both SBP ( $I^2 = 75%$ ) and DBP ( $I^2 = 79%$ ).

In the 12 long-term trials, assessing 608 participants, with follow-up of 6 months or longer, relaxation was associated with slightly smaller but still statistically significant reductions in both SBP (mean difference: -4.0 mmHg, 95% CI: -7.6 to -0.5) and DBP (mean difference: -1.9 mmHg, 95% CI: -3.8 to -0.1). There was substantial heterogeneity for both SBP ( $I^2 = 71%$ ) and DBP ( $I^2 = 58%$ ).

The net blood pressure reductions obtained in short- and long-term trials were not significantly different.

**Relaxation vs. control: BP, sub-grouped by type of control - see comparison 3.**

In the 15 comparisons of relaxation with a control group receiving sham therapy, which assessed 564 participants, relaxation was associated with a non-significant reduction in both SBP (mean difference: -3.5 mmHg, 95% CI: -7.1 to 0.2) and DBP (mean difference: -1.8 mmHg, 95% CI: -4.4 to 0.8). There was substantial heterogeneity for both SBP ( $I^2 = 63%$ ) and DBP ( $I^2 = 72%$ ).

In the 13 comparisons of relaxation with a control group not receiving any active intervention, which assessed 634 participants, relaxation was associated with a larger, statistically significant reduction in both SBP (mean difference: -7.7 mmHg, 95% CI: -11.2 to -4.2) and DBP (mean difference: -5.3 mmHg, 95% CI: -7.7 to -2.8) compared to control. There was substantial heterogeneity for both SBP ( $I^2 = 69%$ ) and DBP ( $I^2 = 71%$ ).

The net blood pressure reductions obtained in comparisons with sham therapy controls were not significantly different from those in comparisons with non-intervention controls.

**Relaxation vs. control: BP, sub-grouped by initial blood pres-**

sure - see comparison 4.

In the 13 trials, assessing 607 participants, with initial SBP above or equal to the median (143.9 mmHg), relaxation was associated with a statistically significant reduction in SBP (mean difference: -7.3 mmHg, 95% CI: -11.3 to -3.3), with substantial heterogeneity ( $I^2 = 76\%$ ). In the 12 trials, assessing 591 participants, with initial SBP below the median, relaxation was associated with no significant reduction in SBP (mean difference: -3.3 mmHg, 95% CI: -6.6 to 0.1), with substantial heterogeneity ( $I^2 = 60\%$ ).

In the 13 trials, assessing 666 participants, with initial DBP above or equal to the median (92.4 mmHg), relaxation was associated with a statistically significant reduction in DBP (mean difference: -4.0 mmHg, 95% CI: -6.9 to -1.2), with substantial heterogeneity ( $I^2 = 85\%$ ). In the 12 trials, assessing 532 participants, with initial DBP below the median, relaxation was associated with a statistically significant reduction in DBP (mean difference: -2.9 mmHg, 95% CI: -4.9 to -0.8), with moderate heterogeneity ( $I^2 = 40\%$ ). The net blood pressure reductions obtained in trials with initial SBP - or initial DBP - above and below the median were not significantly different.

**Relaxation vs. control: BP, including trials with imputed data - see comparison 5.**

The missing standard deviations for three trials (Hafner 1982; LaGrone 1988; Khramelashvili 1986) were imputed, using the highest and lowest standard deviations for final values of SBP and DBP observed in the intervention and control groups in the primary meta-analysis. Imputing the highest standard deviations, this meta-analysis of 28 trials, assessing 1,330 participants, found statistically significant reductions in both SBP (mean difference: -5.7 mmHg, 95% CI: -8.4 to -3.1) and DBP (mean difference: -3.8 mmHg, 95% CI: -5.6 to -2.0), confirming the findings of the primary analysis. Imputing the lowest standard deviations, similar results were obtained for SBP (mean difference: -5.9 mmHg, 95% CI: -8.7 to -3.1) and DBP (mean difference: -4.0 mmHg, 95% CI: -6.0 to -2.1).

**Relaxation vs. control: BP - Sensitivity analysis including only trials reporting adequate concealment of allocation - see comparison 6.**

The one trial which reported adequate concealment of allocation (Patel 1988) compared an active intervention which combined progressive muscle relaxation and biofeedback with no intervention. It enrolled 134 participants of whom 31 (23%) were lost to follow-up. Based on the reported change scores, we estimated that relaxation was associated with statistically significant reductions in both SBP (mean difference: -12.0 mmHg, 95% CI: -17.8 to -6.2) and DBP (mean difference: -4.1 mmHg, 95% CI: -7.4 to -0.9) compared to control. However, participants in the relaxation group had a significantly higher baseline BP (145/89 mmHg) than those in the control group (136/86 mmHg). This baseline imbalance was thoroughly considered by the trial investigators and remained after including and excluding various sub-

groups of participants and also when comparing blood pressure readings taken prior to the baseline measurement. Because of the differences in initial blood pressure, the investigators carried out an analysis of covariance which adjusted for differences in blood pressure at entry. This analysis found relaxation was associated with net reductions in both SBP (mean difference: -7.3 mmHg, 95% CI: -12.6 to -2.0) and DBP (mean difference: -2.2 mmHg, 95% CI: -5.2 to 0.7) which were less marked than those which we found and statistically significant only for SBP.

**Relaxation vs. control: BP - Sensitivity analysis including only trials reporting blinding of outcome assessors - see comparison 7.**

In the nine trials, assessing 498 participants, which reported blinding of outcome assessors, relaxation was not associated with any statistically significant reduction in either SBP (mean difference: -3.2 mmHg, 95% CI: -7.7 to 1.4) or DBP (mean difference: -2.1 mmHg, 95% CI: -5.3 to 1.2) compared to control. Heterogeneity remained substantial for both SBP ( $I^2 = 69\%$ ) and DBP ( $I^2 = 79\%$ ).

**Relaxation vs. control: BP sub-grouped by with/without biofeedback - see comparison 8.**

In the 12 comparisons, assessing 592 participants, of a combination of relaxation and biofeedback with a control, relaxation with biofeedback was associated with statistically significant reductions in both SBP (mean difference: -5.4 mmHg, 95% CI: -8.7 to -2.1) and DBP (mean difference: -2.8 mmHg, 95% CI: -5.2 to -0.5). There was substantial heterogeneity for both SBP ( $I^2 = 52\%$ ) and DBP ( $I^2 = 67\%$ ).

In the 15 comparisons, assessing 608 participants, of relaxation (without biofeedback) with a control, relaxation was associated with smaller but still statistically significant reductions in both SBP (mean difference: -5.9 mmHg, 95% CI: -10.1 to -1.8) and DBP (mean difference: -4.6 mmHg, 95% CI: -7.4 to -1.8), with substantial heterogeneity for both SBP ( $I^2 = 80\%$ ) and DBP ( $I^2 = 79\%$ ).

The net blood pressure reductions obtained in comparisons with and without biofeedback were not significantly different.

**Relaxation with biofeedback vs. control: sub-grouped by type of control - see comparison 9.**

In the eight comparisons, assessing 283 participants, of relaxation and biofeedback with sham therapy, relaxation with biofeedback was associated with a statistically significant reduction in SBP (mean difference: -5.9 mmHg, 95% CI: -11.0 to -0.7) but a non-significant reduction in DBP (mean difference: -3.3 mmHg, 95% CI: -6.9 to 0.4). There was substantial heterogeneity for both SBP ( $I^2 = 60\%$ ) and DBP ( $I^2 = 76\%$ ).

In the six comparisons, assessing 309 participants, of relaxation and biofeedback with non-intervention controls, relaxation with biofeedback was associated with statistically significant reductions in both SBP (mean difference: -6.3 mmHg, 95% CI: -10.1 to -2.6) and DBP (mean difference: -3.2 mmHg, 95% CI: -5.7 to -0.8). There was low heterogeneity for SBP ( $I^2 = 24\%$ ) and moderate heterogeneity for DBP ( $I^2 = 30\%$ ).



The net blood pressure reductions obtained in comparisons with sham therapy controls were not significantly different from those in comparisons with non-intervention controls.

**Relaxation vs. control: sub-grouped by cognitive and behavioural therapies/other - see comparison 10.**

In the eleven comparisons, assessing 477 participants, of cognitive/behavioural therapy with a control, cognitive/behavioural therapy was associated with a statistically significant reduction in both SBP (mean difference: -6.3 mmHg, 95% CI: -11.7 to -0.8) and DBP (mean difference: -4.5 mmHg, 95% CI: -8.0 to -1.1). There was substantial heterogeneity for both SBP ( $I^2 = 83%$ ) and DBP ( $I^2 = 84%$ ).

In the 15 comparisons, assessing 721 participants, of other therapies with a control, other therapies were associated with a significant reduction in both SBP (mean difference: -5.1 mmHg, 95% CI: -7.8 to -2.4) and DBP (mean difference: -3.2 mmHg, 95% CI: -5.2 to -1.2). There was substantial heterogeneity for both SBP ( $I^2 = 50%$ ) and DBP ( $I^2 = 60%$ ).

The differences between net blood pressure reductions obtained in comparisons with and without cognitive/behavioural therapy were not significantly different.

**Relaxation vs. control: sub-grouped by with/without progressive muscle relaxation - see comparison 11.**

In the 16 comparisons, assessing 699 participants, of progressive muscle relaxation with control, progressive muscle relaxation was associated with a significant reduction in both SBP (mean difference: -4.8 mmHg, 95% CI: -7.2 to -2.4) and DBP (mean difference: -2.8 mmHg, 95% CI: -4.8 to -0.9). There was substantial heterogeneity for both SBP ( $I^2 = 47%$ ) and DBP ( $I^2 = 61%$ ).

In the ten comparisons, assessing 499 participants, of other therapies with a control, other therapies were associated with a statistically significant reduction in both SBP (mean difference: -6.8 mmHg, 95% CI: -13.2 to -0.5) and DBP (mean difference: -4.8 mmHg, 95% CI: -8.8 to -0.9). There was substantial heterogeneity for both SBP ( $I^2 = 84%$ ) and DBP ( $I^2 = 85%$ ).

The net blood pressure reductions obtained in comparisons with and without progressive muscle relaxation were not significantly different.

**Relaxation vs. control: sub-grouped by with/without autogenic training - see comparison 12.**

In the six trials, assessing 358 participants, comparing autogenic training with control, autogenic training was not associated with a significant reduction in either SBP (mean difference: -2.3 mmHg, 95% CI: -7.9 to 3.2) or DBP (mean difference: -1.6 mmHg, 95% CI: -4.8 to 1.6). There was substantial heterogeneity for both SBP ( $I^2 = 79%$ ) and DBP ( $I^2 = 69%$ ).

In the 19 trials, assessing 840 participants, comparing other therapies with a control, other therapies were associated with a significant reduction in both SBP (mean difference: -6.6 mmHg, 95% CI: -9.8 to -3.4) and DBP (mean difference: -4.2 mmHg, 95% CI: -6.4 to -1.9). There was substantial heterogeneity for both SBP ( $I^2 = 70%$ ) and DBP ( $I^2 = 77%$ ).

The net blood pressure reductions obtained in comparisons with and without autogenic training were not significantly different.

**Relaxation vs. control: adverse events - uncontrolled hypertension - see comparison 13, outcome 1.**

Only five trials, enrolling 287 participants, reported the numbers of participants experiencing uncontrolled hypertension in each treatment group. In these trials, rates of adverse events were 3% and 4% in the relaxation and control groups respectively. Meta-analysis showed no significant difference in the rate of uncontrolled hypertension in the relaxation and control groups (risk difference = 0.00, 95%CI: -0.05 to 0.04), with no heterogeneity between trials ( $I^2 = 0%$ ).

**Relaxation vs. control: other adverse events - see comparison 13, outcome 2.**

Only six trials, enrolling 389 participants, reported the numbers of participants experiencing other adverse events in each treatment group. In these trials, rates of other adverse events were 1% and 4% in the relaxation and control groups respectively. Meta-analysis showed no significant difference in the rate of these adverse events in the relaxation and control groups (risk difference = -0.02, 95%CI: -0.05 to 0.01), with no heterogeneity between trials ( $I^2 = 0%$ ).

**Relaxation vs. control: withdrawal from treatment - see comparison 13, outcome 3.**

Only 14 trials, enrolling 695 participants, reported the numbers of participants withdrawing from treatment in each treatment group. In these trials, rates of withdrawal were 13% and 9% in the relaxation and control groups respectively. Meta-analysis showed no significant difference in the rate of withdrawal in relaxation and control groups (risk difference = 0.03, 95%CI: -0.03 to 0.09). There was moderate heterogeneity ( $I^2 = 41%$ ), largely due to the high rate of withdrawal (25%) in the relaxation groups of the trial of [Achmon 1989](#).

In four further trials ([Garcia-Vera 1997](#); [Irvine 1986](#); [Patel 1988](#); [Zurawski 1987](#)) which reported overall withdrawal from treatment, but not withdrawal by treatment arm, the rate of withdrawal was 19%.

**Relaxation vs. control: withdrawal from treatment due to adverse events - uncontrolled hypertension - see comparison 13, outcome 4.**

Only seven trials, enrolling 322 participants, reported the numbers of participants withdrawing from treatment due to uncontrolled hypertension in each treatment group. In these trials, rates of withdrawal due to uncontrolled hypertension were 2% in both the relaxation and control groups. Meta-analysis showed no significant difference in the rate of withdrawal due to adverse events in the relaxation and control groups (risk difference = 0.00, 95%CI: -0.04 to 0.04), with no heterogeneity between trials ( $I^2 = 0%$ ).

Additionally, [Johnston 1993](#) reported 2/96 (4%) of participants withdrawing because of uncontrolled high blood pressure, but did not report the numbers by treatment group.

**Relaxation vs. control: withdrawal from treatment due to other**

### **adverse events - see comparison 13, outcome 5.**

Only six trials, enrolling 261 participants, reported the numbers of participants withdrawing from treatment due to other adverse events in each treatment group. In these trials, rates of withdrawal due to adverse events were 2% in both relaxation and control groups. Meta-analysis showed no significant difference in the rate of withdrawal due to these adverse events in the relaxation and control groups (risk difference = -0.01, 95%CI: -0.05 to 0.03), with no heterogeneity between trials ( $I^2 = 0\%$ ).

### **Relaxation vs. control: loss to follow-up - see comparison 13, outcome 6.**

Only 13 trials, enrolling 675 participants, reported the numbers of participants lost to follow-up. In these trials, rates of loss to follow-up were 16% and 15% in the relaxation and control groups respectively. Meta-analysis showed no significant difference in the rate of loss to follow-up in the relaxation and control groups (risk difference = 0.01, 95%CI: -0.07 to 0.09), with substantial heterogeneity between trials ( $I^2 = 61\%$ ).

## **DISCUSSION**

### **Summary of findings**

The primary meta-analysis of 25 randomised controlled trials assessing 1,198 participants, with between eight weeks and 5 years follow-up, found that interventions to promote relaxation were associated with a small, statistically significant reduction in both SBP and DBP. The random effects model which we used assumes that the effect of treatment differs in different populations but that these effects cluster around a mean: this estimated mean was a reduction in SBP of 5.5 mmHg, (95% CI: 2.8 to 8.2) in participants receiving a relaxation intervention compared to those receiving a control intervention, with a concomitant reduction in DBP of 3.5 mmHg (95% CI: 1.6 to 5.3). However, this estimated reduction of 5/3 mmHg is probably an over-estimate of the effect of relaxation, as poor quality trials and comparisons with non-intervention controls generally over-estimate the effects of treatment, as discussed below. The combined sample was too small and the trials were too short-term to assess whether relaxation could reduce the risk of death, heart attack or stroke.

When relaxation was compared with a sham therapy – designed to mimic many of the components of the active treatment, but not the component thought to be effective – the mean reductions in blood pressure were smaller and not statistically significant. This is consistent with the evidence that sham therapy alone can reduce a continuous outcome by between 0.2 and 0.5 standard deviations (Hrobjartsson 2001), possibly due to therapeutic effects of the relationship between the participant and the treatment provider. Hence it seems likely that the actual effect of relaxation on blood pressure is less marked than estimated by our primary meta-analysis.

Most included trials were not of good quality. Inadequate concealment of allocation and lack of blinding are often associated with an exaggeration of the effects of treatment (Schulz 1995; Moher 1998). Restriction to the nine trials (assessing 498 participants) that reported blinding of the outcome assessor resulted in mean reductions in SBP and DBP which were smaller than those estimated by the primary meta-analysis and not statistically significant. The one included trial (assessing 103 participants) that reported adequate concealment of allocation yielded results which were similar to the primary meta-analysis, but this trial did not blind outcome assessors.

Progressive muscle relaxation, relaxation with biofeedback and cognitive/behavioural therapies (such as teaching strategies for stress management and anger control) were all associated with statistically significant net reductions in blood pressure. As biofeedback was most frequently used in combination with progressive muscle relaxation, it is unclear which strategy – or if only the combined strategies – might be effective. We found little evidence that autogenic training reduced blood pressure.

There was substantial heterogeneity between the estimated effects of relaxation in the various trials: between 70% and 80% of the variation between trials could not be explained by sampling variation. This heterogeneity does not appear to be due to bias in outcome assessment, as trials which blinded outcome assessors showed a comparable level of heterogeneity. Nor does it appear to be due to differential withdrawal from treatment or loss to follow-up, which was similar in treatment and control groups. Some possible factors – duration of follow-up, type of control, initial blood pressure and type of relaxation therapy – were evaluated by sub-group analyses but, for each factor considered, the treatment effects in the sub-groups were not significantly different and moderate or substantial heterogeneity remained within each sub-group. Therefore other, unidentified factors must largely explain the variation in findings of trials; this is not surprising, given the differences in the active and control interventions used and the differences between participants enrolled in the different trials – individuals may vary substantially in how they respond to relaxation therapy.

Funnel plots showed little evidence of publication bias. The rates of adverse events, withdrawal from treatment for any reason and withdrawal from treatment due to adverse events were similar in relaxation and control groups. The proportion of participants lost to follow-up was similar in relaxation and control groups and so loss to follow-up is unlikely to have introduced bias in the estimated effect of relaxation.

### **Strengths and weaknesses of review**

The review was limited by the design of the included trials. As many trials used a combination of strategies to encourage relaxation, it is difficult to ascribe the outcomes to specific components

of the therapy. We were not able to evaluate whether the effects of different components were additive.

Some trials that used several relaxation strategies in the active intervention also used the components of these strategies which were thought to be ineffective in lowering blood pressure as the control intervention (i.e. sham therapy); this is a valid design for evaluating the relaxation strategy hypothesised to be effective, despite the use of components of these sham therapies as active interventions in other trials.

#### *External validity*

Trials that are conducted in tightly controlled research environments may yield results which are not relevant to real life clinical situations. This aspect of trials may be assessed by whether the setting, the patients recruited, the people delivering the intervention, their training and support and the monitoring of the participants are representative of clinical practice or of a research setting. Although lifestyle interventions for mild-to-moderate hypertension are likely to be of most interest to patients in primary care, only four trials were performed in primary care settings. Furthermore, only 11 trials recruited participants from primary care settings, through primary care physicians, the workplace, or community screening. These participants usually had mild hypertension: many trials excluded patients with heart or renal disease, previous heart attacks or strokes, angina, diabetes and other serious medical disorders. Most of these trials used therapists, physicians or nurses to deliver the intervention, but a few (Irvine 1986; Irvine 1991) used researchers. The intervention was usually delivered in weekly group sessions lasting between 30 minutes and an hour, but some trials (Garcia-Vera 1997; Irvine 1991; Yen 1996) treated participants individually. Participants were usually monitored at baseline, the end of treatment and the end of follow-up, but in one trial (Yen 1996) ten physician sites volunteered to monitor patients' blood pressure free of charge. Hence fewer than half the included trials were typical of routine clinical practice.

None of the included trials reported the costs of implementing the intervention, although the excluded trial of Patel 1981 reported that providers of relaxation therapy spent a total time of less than an hour per participant during an eight week course.

We divided the trials into two sub-groups in eight different ways and compared two outcomes (SBP and DBP) in each of these sub-groups. Therefore many hypothesis tests were performed on the same set of trials and it is likely that one or two of these appeared to be statistically significant just by chance (see section 8.8.1 of Deeks 2006).

#### *Use of antihypertensive medication*

We excluded from our review 21 trials in which participants were taking antihypertensive medication which could vary during the course of the trial. If relaxation were effective in reducing blood pressure and changes in antihypertensive medication were allowed,

it is likely that a higher proportion of participants in the control group than the relaxation group would start on or increase their dose of antihypertensive medication. As antihypertensive drugs generally have a much more marked effect in lowering blood pressure - typically of the order of 9/5 mmHg for single drugs (Law 2003) - than the effect postulated for relaxation, the inclusion of trials allowing such medication could result in an under-estimate of the real effect of relaxation. Although exclusion of such trials is likely to yield a less biased estimate of the effect of relaxation, it will also result in a wider confidence interval for the estimated effect due to the smaller number of included trials. Hence the decision about whether to include or exclude trials that allow antihypertensive medication to vary is essentially a trade-off between bias and precision.

The efficacy of relaxation in the management of hypertension could best be assessed by trials which are long enough and large enough to detect a difference between relaxation and control arms in deaths and cardiovascular events. However, such long and large trials are more likely to allow participants to start on antihypertensive medication, or increase an existing dose; indeed it would not be ethical in such trials to withhold such medication. Hence the decision to exclude from review trials which allow antihypertensive medication to vary may have resulted in exclusion of some potentially informative studies.

In particular, the excluded trial of Patel 1981 enrolled 204 currently untreated participants, followed them up for 4 years and reported deaths and cardiovascular events. Although it allowed participants to start taking antihypertensive medication, about the same proportion did so in the relaxation and control groups (18% and 20% respectively), so this is unlikely to have resulted in bias. However, it was similar in design to the trial of Patel 1988 in that it did not have an active control group or blinding of outcome assessors; it reported a similar reduction in blood pressure in the relaxation group.

The difficulties of obtaining an unbiased estimate of the effect of relaxation treatment on blood pressure when some participants use varying doses of antihypertensive medication could be addressed by modification to the design of trials. Trial protocols could exclude participants if they start on (or change their dose of) antihypertensive medication, but include their last blood pressure measurements before exclusion. Alternatively, in large trials the efficacy of relaxation could be evaluated by survival analysis, treating starting on (or increasing) antihypertensive medication as a failure, with death and cardiovascular events as competing risks (Collett 2003).

#### **Comparison with other meta-analyses**

Five major meta-analyses of RCTs of relaxation therapies for hypertension have been conducted (Ebrahim 1998; Eisenberg 1993;

Linden 1994; Nakao 2003; Stetter 2002). These included different trials from our review because of the different time period for reporting of trials, different inclusion criteria, different judgements about whether individual trials were randomised, controlled and maintained any antihypertensive medication at a constant dose. They also used different methods of analysis: in particular, all analysed change scores whereas we preferred final values, since the difference between change scores in treatment and control groups is a biased estimator of the treatment effect (Matthews 1999).

Three meta-analyses (Ebrahim 1998; Eisenberg 1993; Nakao 2003) used weighted mean difference methods, as our review did. Although they used different methods in other aspects of their meta-analyses, they obtained similar results to our review:

#### Duration of follow-up

Ebrahim 1998 reported the findings of a fixed effects model used to aggregate the results of eight RCTs (six of which were included in our meta-analyses) that had at least 6 months follow-up and were conducted in hypertensive people aged 45 years or over; the threshold for hypertension was not defined. This meta-analysis found that relaxation was associated with a very small overall reduction of 1/1 mmHg in blood pressure. When we restricted our meta-analysis to the 12 studies with at least 6 months follow-up, we found similar small reductions in SBP and DBP (mean differences of -4.0 mmHg, 95%CI: -7.6 to -0.5 and -1.9 mmHg, 95%CI: -3.8 to -0.1 respectively).

#### Sham therapy/non-intervention controls

Eisenberg 1993 used a random effects model to aggregate the findings of 26 RCTs (12 of which were included in our meta-analyses) in people with DBP between 90 and 114 mmHg. This meta-analysis found that relaxation therapies were superior to no treatment but not to a credible sham therapy. This is consistent with our findings of significant overall reductions in blood pressure in 13 trials which compared relaxation with non-intervention controls, but smaller non-significant reductions in blood pressure in 15 trials which compared relaxation with sham therapy.

This meta-analysis also reported that trials in which baseline blood pressure assessments were made during a period of a day or less found, on average, much larger net reductions in blood pressure than those with longer baseline periods.

#### Biofeedback

Nakao 2003 used a random effects model to aggregate the findings of 22 RCTs (11 of which were included in our meta-analyses) of biofeedback in people with blood pressure over 140/90 mmHg. This meta-analysis found that biofeedback was superior to no treatment but not to a credible sham therapy. We found significant overall reductions in both SBP and DBP for six trials which compared biofeedback with non-intervention controls and similar reductions – significant for SBP but not for DBP – for

eight trials which compared biofeedback with sham therapy respectively. Our results differed from those of Nakao 2003 largely because of a different classification of the treatment and control groups in the trial of Achmon 1989, which reported very large reductions in blood pressure (24/13 mmHg) in the biofeedback group compared to the control group.

#### Autogenic training

Stetter 2002 used standardised mean difference methods which assume that all the variability between trials is due to differences in the measurement scale and that all trials have a similar amount of natural variation, which may not be true. As blood pressure was measured on the same scale in all trials, we would argue that weighted mean difference methods should be preferred. Stetter's review aggregated findings of four RCTs (one of which was included in our meta-analyses) of autogenic training for mild-to-moderate primary hypertension and found that it significantly reduced blood pressure immediately after treatment. However, based on three RCTs, Stetter's meta-analysis found – as our review did – that autogenic training had no significant effect on hypertension at the end of follow-up.

Linden 1994 meta-analysed the change in blood pressure between baseline and end of follow-up in participants who received a relaxation intervention, but did not compare this change score with that in a control group.

#### Biological plausibility

Blood pressure is determined by the rate of blood flow produced by the heart, blood volume and the resistance of the blood vessels, which is produced mainly in the small arteries and is known as peripheral vascular resistance. The physiological regulation of blood pressure is complex, resulting from actions of the kidneys, central and autonomic nervous systems, hypothalamic pituitary axis, vascular endothelium and other pathways. Acute rises in blood pressure due to stress are thought to result from the action of adrenaline on the sympathetic nervous system. Furthermore, it has been suggested that adrenaline produced in the acute phase may be stored and released over a more sustained period (Pickering 1991; Stone 1976). However, it is more plausible that chronic hypertension is mediated by factors which increase peripheral vascular resistance. Vascular remodelling, involving changes in blood vessel architecture, endothelial dysfunction and alterations in renal regulation of fluid balance, through the renin-angiotensin-aldosterone axis, are thought to be key components of this process (Schwartz 2003). In addition, modulation of glucocorticoid activity may also have a role (Pickering 1991). It is likely that these systems do not work in isolation but are interlinked by complex feedback mechanisms.

Behavioural methods of relaxation therapy aimed to reduce blood pressure through direct control of either the blood pressure or

the physiological processes involved in its regulation, whereas psychotherapeutic methods aimed to alter reactions to stress, thus indirectly lowering blood pressure. It is possible that stress reduction could work as a long-term strategy to decrease blood pressure through effects on known blood pressure regulatory mechanisms, for example through decreasing sympathetic nervous system activity or plasma concentrations of cortisol and aldosterone (McGrady 1981; Patel 1981).

## AUTHORS' CONCLUSIONS

### Implications for practice

In view of the poor methodological quality of studies included in the meta-analysis, it is difficult to draw any definitive conclusions about the efficacy or lack of efficacy of relaxation techniques for primary hypertension.

Some relaxation therapies may reduce blood pressure by a small amount in some patients. There was substantial variation between the effects of relaxation therapies in different populations and we were unable to identify the characteristics of patients in whom it was likely to be effective. Some of the reduction in blood pressure apparently associated with relaxation is probably due to the non-specific effects of treatment, such as frequent contact with treatment providers. Progressive muscle relaxation, biofeedback and cognitive/behavioural therapies were the relaxation therapies most likely to be effective; there was little evidence that autogenic training was effective.

Even if relaxation results in a reduction in blood pressure, the average reduction is probably less than 5/3 mmHg. We found no direct evidence that relaxation decreases the risk of morbidity and mortality. In contrast, drugs can singly reduce SBP by about 9.1 mmHg (95%CI: 8.8 to 9.3) and DBP by about 5.5 mmHg (95%CI: 5.4 to 5.7) (Law 2003), and are known to have sustained and consistent effects and to reduce morbidity and mortality (Psaty 2003). However, as hypertension is a common condition, even small changes in blood pressure in a large proportion of the population could prevent a large number of adverse cardiovascular outcomes (PSC 2002).

Since there is no good evidence that relaxation therapies result in meaningful reductions in blood pressure, patients with mild-to-moderate hypertension who prefer non-pharmacological interventions may wish to consider alternative strategies - such as diet, exercise, and restriction of intake of alcohol and salt - which result, on average, in small reductions in blood pressure (Dickinson 2006; Hooper 2004; Jürgens 2004; Mulrow 1998).

### Implications for research

Despite calls since 1978 for improvements in the methodological quality of trials of relaxation for hypertension (Blanchard 1979; Canino 1994; Carson 1988; Ebrahim 1998; Eisenberg 1993; Frankel 1978; Irvine 1986; Irvine 1991; Jacob 1992; Johnston 1993; Nakao 2003; Seer 1980; Canter 2004), in particular for larger sample sizes from representative populations, longer baseline periods for screening of potential participants, control "placebo" treatments that simulate the non-specific aspects of treatment, control of antihypertensive medication used by participants and blinding of outcome assessors, all the trials contributing to the evidence base were methodologically flawed. Furthermore, few studies have evaluated relaxation in settings such as primary care practices, community services or workplaces which are relevant to routine implementation.

Any future research should address the basic methodological issues, focus on primary care settings and use a design which deals with possible changes in levels of antihypertensive medication in an appropriate way.

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**Schulz 1995**

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**Schwartz 2003**

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Brosschot JF, Thayer JF, Christenfeld N, Linden W. Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosom Med* 2003;**65**:22–35.

**Stetter 2002**

Stetter F, Kupper S. Autogenic training: a meta-analysis of clinical outcome studies. *Applied Psychophysiology & Biofeedback* 2002;**27**:45–98.

**von Onciul 1996**

von Onciul, J. ABC of Work related Disorders: Stress at Work. *BMJ* 1996;**313**:745–748.

**WHO 2002**

World Health Organisation. *The world health report 2002: reducing risks, promoting healthy life*. Geneva: World Health Organisation, 2002.

\* Indicates the major publication for the study

## CHARACTERISTICS OF STUDIES

### Characteristics of included studies [ordered by study ID]

#### Achmon 1989

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - yes
Participants	97 adults (25-60) with currently treated or treatment naive essential hypertension, (BP >140/90). Israel. Mean age 41 years, 51% male, ?% white. Inclusion criteria: without heart or renal disease, not taking $\beta$ -blocker, diuretic use allowed but with no dose alteration. Baseline BP: 154.0/98.7 0% baseline CVD ?% baseline diabetes.
Interventions	I1: Cognitive group therapy for anger control. Weekly 1½ hour therapist-led group sessions: exercises, role-play, assertive behaviour, instructed to practise methods in real life and keep a daily diary I2: Heart rate biofeedback. Weekly 1 hour group sessions led by psychology student and cognitive therapist: participants instructed on how to lower heart rate, pulse rate recorded C: Sham therapy. Attended 2 lectures aimed at stimulating anticipation of BP change, told that monthly BP readings could lower BP, physician available to answer medical questions, free discussion between participants allowed Treatment duration: 17 weeks
Outcomes	BP at 17 weeks, measured in clinic, seated, averaged over 2 readings of digital sphygmomanometer. Deaths and cardiovascular events not reported. Adverse events not reported. Withdrawn from treatment: I1: 10/40 (25%) I2: 10/37 (27%) C: 0/20 (0%) Withdrawn due to Adverse events: Not reported.
Notes	Loss to follow-up: I1: 10/40 (25%) I2: 10/37 (27%) C: 0/20 (0%). Anti-hypertensive medication did not vary during the trial.

#### *Risk of bias*

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Adsett 1989**

Methods	Method of randomisation adequate. Factorial design. Blinding: Participant - no Treatment provider - no Outcome assessor - yes.
Participants	47 adults with currently untreated mild hypertensive, (mean DBP 90-105). Canada. Mean age 47 years, 100% male, ?% white. Inclusion criteria: without CVD, CVA, renal disease. Baseline BP: 145/96 0% baseline CVD 0% baseline diabetes.
Interventions	I: Relaxation therapy. Weekly one hour therapist-led group relaxation sessions: progressive muscle relaxation, information about hypertension, lifestyle & stress C: Sham therapy: Weekly one hour therapist-led group education sessions: information about hypertension, lifestyle & stress Treatment duration: 8 weeks This was a 2 X 2 factorial trial, in which participants were also randomised to $\beta$ -blocker or placebo; relaxation and sham therapy arms were aggregated over $\beta$ -blocker and placebo
Outcomes	BP at 3 months, measured in clinic, seated, averaged over 2 readings of random zero mercury sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects: I: 0/23 (0%) C: 1/24 (4%) due to high BP Withdrawn from treatment: I: 2/23 (9%) C: 1/24 (4%) Withdrawn due to adverse events: I: 0/23 (0%) C: 1/24 (4%) due to high BP
Notes	Loss to follow-up: I: 2/23 (9%) C: 1/24 (4%). Investigators attempted to keep anti-hypertensive medication constant during the trial. One participant who was started on anti-hypertensive medication at the start of the trial stopped using it within a month because of side-effects

***Risk of bias***

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear



**Aivazyan 1988b**

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - unclear.
Participants	90 adults (20-50) with currently treated and treatment naive mild to moderate essential hypertension. USSR. Men age 40 years, 78% male, ?% white. Inclusion criteria: not stated. Baseline BP: 164.5/100.1 ?% baseline CVD ?% baseline diabetes.
Interventions	I: Autogenic training C: No treatment Treatment duration: 6 months
Outcomes	BP at 5 years. Deaths and cardiovascular events not reported. Adverse effects: I: 17.1 sick days/year C: 27.3 sick days/year Withdrawn from treatment: not reported. Withdrawn due to adverse events: not reported.
Notes	Loss to follow-up: I: 0/44 (0%) C: 0/46 (0%) Unclear whether anti-hypertensive medication varied during trial

**Risk of bias**

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Amigo 1997**

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - yes.
Participants	45 adults (18-60) with treatment naïve (91%) and currently treated (9%) mild essential hypertension (DBP = 90-104 mm Hg). Spain. Mean age 43 years, 53% male, ?% white. Inclusion criteria: not treated for any other CVD or regularly exercising. Baseline BP: 143/88

**Amigo 1997** (Continued)

	0% baseline CVD ?% baseline diabetes.
Interventions	I1: Progressive muscle relaxation - 8 sessions of progressive muscle relaxation, 8 X 1 hr weekly sessions individually with therapist, inter-session homework assignments. C: Placebo exercise too mild to improve cardiovascular fitness: 24 30-minute sessions individually with therapist, 3 times weekly for 8 weeks An additional isotonic physical exercise arm was not considered in this review. Treatment duration: 8 weeks
Outcomes	BP at 6 months, measured in clinic, averaged over 2 readings of automatic sphygmomanometer (no further details reported). Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: I1: 1/16 (6%) C: 0/15 (0%) Withdrawn due to adverse events: Not reported.
Notes	Loss to follow-up: I1: 1/16 (6%) C: 2/15 (13%). Anti-hypertensive medication did not vary during the trial.

**Risk of bias**

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Bennett 1991**

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - unclear.
Participants	47 adults with currently untreated mild essential hypertension (DBP 90-104) and type A personality: (tendency to anger and hostility). UK. Mean age 46 years, 100% male, ?% white. Inclusion criteria: not stated. Baseline BP: 152/93 0% baseline CVD 0% baseline diabetes.

**Bennett 1991** (Continued)

Interventions	<p>I1: Type-A management. Weekly 2 hour therapist-led sessions: education, relaxation, cognitive restructuring, meditation, time management, anger control, assertiveness training            I2: Stress management. Weekly 2 hour therapist-led sessions; education, relaxation, cognitive restructuring, meditation; behavioural assignments &amp; diary completion            C: No intervention.            Treatment duration: 8 weeks            *All participants received handout based on British Heart Society booklet: guidance on BP, salt, exercise, stress before intervention</p>	
Outcomes	<p>BP at 8 weeks, measured in clinic, supine, averaged over 2 readings of automatic sphygmomanometer.            Deaths and cardiovascular events not reported.            Adverse effects not reported.            Withdrawn from treatment:            I1: Unclear            I2: 2/17 (12%)            C: Unclear            Withdrawn due to adverse events:            Not reported.            .</p>	
Notes	<p>Loss to follow-up:            I1: 1/16 (6.3%)            I2: 2/17 (11.8%)            C: 0/14 (0%).</p>	
<b>Risk of bias</b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Unclear	B - Unclear

**Blanchard 1979**

Methods	<p>Method of randomisation unclear.            Blinding:            Participant - unclear            Treatment provider - unclear            Outcome assessor - unclear.</p>	
Participants	<p>33 adults (23-61 years) with currently treated (45%) and untreated (55%) essential hypertension (BP &gt;= 140/90 mm Hg) with no end organ damage.            USA. Mean age 51 years, 49% male, ?% white.            Inclusion criteria: subjects on medication requested to keep dose stable.            Baseline BP: 141/87            ?% baseline CHD            ?% baseline diabetes.</p>	

**Blanchard 1979** (Continued)

Interventions	<p>I1: Direct biofeedback of BP - 12 X 40 minute group sessions, instructed to lower BP using feedback on a screen so as to discover a mental strategy that would work for them; instructed to practice relaxing and trying to lower their BP at home</p> <p>I2: Frontal EMG biofeedback - 12 X 40 minute group sessions, instructed to relax deeply using feedback signal and told relaxing would help lower BP; instructed to practice at home once daily</p> <p>C: Placebo control relaxation group - 12 X 40 minute sessions, instructed to try to relax as deeply as they could and told relaxing would help lower BP</p> <p>Treatment duration: 6-10 weeks</p> <p>All groups received 12 X 40 minute sessions over 6-12 weeks, followed by 8 follow-up sessions with no biofeedback</p> <p>Sessions were 12 x 40 mins over 6-10 weeks.</p>
Outcomes	<p>BP at 23-29 weeks, measured in clinic, standing, seated and supine (no further details reported).</p> <p>Deaths and cardiovascular events not reported.</p> <p>Adverse effects not reported.</p> <p>Withdrawn from treatment:</p> <p>I1: 1/11 (9%)</p> <p>I2: 2/11 (18%)</p> <p>C: 2/11 (18%)</p> <p>Withdrawn due to adverse events:</p> <p>Not reported.</p> <p>.</p>
Notes	<p>Loss to follow-up:</p> <p>I1: 2/11 (18%)</p> <p>I2: 4/11 (36%)</p> <p>C: 5/11 (45%)</p> <p>Anti-hypertensive medication did not vary during the trial.</p> <p>SDs of BP estimated from t-statistics in table 2.</p>

**Risk of bias**

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Blanchard 1996**

Methods	<p>Method of randomisation unclear.</p> <p>Blinding:</p> <p>Participant - no</p> <p>Treatment provider - no</p> <p>Outcome assessor - unclear.</p>
Participants	<p>46 adults (32-62) with previously treated (33%) and untreated (67%) hypertension (DBP <math>\geq</math> 90 mm Hg)</p> <p>.</p> <p>USA. Mean age 51 years, 67% male, 91% white.</p> <p>Inclusion criteria: excluded if could not be safely withdrawn from medication or previous life-threatening illness.</p>

**Blanchard 1996** (Continued)

	Baseline BP: 141.1/91.7 0% baseline CVD 0% baseline diabetes.	
Interventions	I: Thermal biofeedback - psychologist led 16 small group sessions (twice weekly) training hand then feet warming through relaxation, autogenic training or other strategies C: BP monitoring - BP self-measured at home twice daily Treatment duration: 8 weeks	
Outcomes	BP at 8 weeks, measured in clinic, seated, averaged over 3 readings of random zero sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects: I: 2/23 (9%) C: 2/23 (4%) 3 participants had high BP; 1 developed another medical problem. Withdrawn from treatment: I: 2/23 (9%) C: 2/23 (9%) Withdrawn due to adverse events: I: 2/23 (9%) C: 1/23 (4%)	
Notes	Loss to follow-up: I: 2/23 (9%) C: 2/23 (9%)	
<b><i>Risk of bias</i></b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Unclear	B - Unclear

**Bosley 1989**

Methods	Method of randomisation unclear. Blinding: Participant - unclear Treatment provider - unclear Outcome assessor - yes.
Participants	41 adults (42-68) with currently treated mild to moderate hypertension >6 months (DBP 77-104). USA. Mean age 57 years, 100% male, 0% white. Inclusion criteria: not reported. Baseline BP: 150/93 ?% baseline CVD ?% baseline diabetes.

**Bosley 1989** (Continued)

Interventions	I: Cognitive self-management training - counselling psychologist run weekly 45 minute group sessions; information presented about stress and hypertension; subjects trained to develop awareness about body's reaction and negative self-talk during stressful situations; homework assignments aimed to promote more functional analysis and labelling of situations and to increase frequency of supportive self-communications C1: Attention placebo control - counselling psychologist run weekly 45 minute group sessions; information presented about dynamics of stress and relationship of stress to hypertension; no suggestions made regarding coping strategies C2: Current conditions control - received only regular clinical care Treatment duration: 8 weeks
Outcomes	BP at 8 weeks, measured in clinic, seated, averaged over 3 readings of random zero sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: Not reported. Withdrawn due to adverse events: Not reported.
Notes	Loss to follow-up: 1/41 (2%) (Numbers not reported by group). Unclear whether anti-hypertensive medication varied during trial. Numbers enrolled and assessed in C1 and C2 not reported; hence trial excluded from meta-analysis

**Risk of bias**

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Canino 1994**

Methods	Method of randomisation unclear. Blinding: Participant - unclear Treatment provider - unclear Outcome assessor - unclear.
Participants	21 adults (25-46) with currently untreated hypertension (SBP > 140 and/or DBP >90). Venezuela Mean age 35 years, 67% male, ?% white. Inclusion criteria: without diabetes, heart, renal disease. Baseline BP: 148/97 0% baseline CVD 0% baseline diabetes.
Interventions	I1: Behavioural programme. Twice-weekly 1¼ hour therapist-led sessions: training in deep-muscle relaxation, biofeedback of temperature of middle finger, anxiety management C1: placebo treatment - 15 x 1¼ hour therapist-led sessions; no coping skills strategies training, instructed

**Canino 1994** (Continued)

	to record 'stressful life events' and relaxation encouraged C2: No intervention (waiting list). Treatment duration: 7½ weeks
Outcomes	BP at 18 weeks, measured in clinic, seated, averaged over 3 readings of automatic sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: I1: 0/8 (0%) C1: 0/4 (0%) C2: 0/9 (0%) Withdrawn due to adverse events: I1: 0/8 (0%) C1: 0/4 (0%) C2: 0/9 (0%) .
Notes	Loss to follow-up: I1: 1/8 (12%) C1: 0/4 (0%) C2: 0/9 (0%)

**Risk of bias**

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Carson 1988**

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - unclear.
Participants	16 adults (49-73) with history of high blood pressure and elevated cholesterol. USA. Mean age 64 years, 100% male, ?% white. Inclusion criteria: not stated. Baseline BP: 140/87 100% baseline CVD 50% baseline diabetes.
Interventions	I: Group class weekly, instructed participants in relaxation technique: listening to taped instructions on muscle relaxation, ½ hour, twice daily. C: Group class weekly, instructed participants in quiet reading of self-selected material, ½ hour, twice daily Treatment duration: 8 weeks *Both groups were nurse & dietician-led and received education on CHD & CHD risk management

**Carson 1988** (Continued)

Outcomes	BP at 8 weeks, (no further details reported). Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: Not reported. Withdrawn due to adverse events: Not reported.	
Notes	Loss to follow-up: I: 0/8 (0%) C: 0/8 (0%). Unclear whether anti-hypertensive medication varied during trial	
<b>Risk of bias</b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Unclear	B - Unclear

**Cottier 1984**

Methods	Method of randomisation adequate. Blinding: Participant - no Treatment provider - no Outcome assessor - no.	
Participants	30 adults with previously treated or untreated borderline-mild essential hypertension, (untreated clinic BP 140-170/90-115, home BP > 135/85). USA. Mean age 35 years, 70% male, ?% white. Inclusion criteria: treatment with no more than 2 drugs. Baseline BP: 130/90 ?% baseline CVD ?% baseline diabetes.	
Interventions	I: Progressive muscle relaxation. 8x45 minute physician-led individual sessions; taught to practise relaxation during particular situations - telephone calls, at traffic lights, watching television, asked to practise twice daily at home for 20 minutes with the aid of a tape and to keep a diary C: Control. Blood pressure measured only and attended clinic for physical examination Treatment duration: 22 weeks	
Outcomes	BP at 22 weeks, measured in clinic, seated, 3 readings of automated device and one reading of mercury sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects: I1: 2/30 (7%) High BP C: 1/30 (3%) Broken rib Withdrawn from treatment:	



**Cottier 1984** (Continued)

	I1: 2/30 (7%) High BP C: 1/30 (3%) Broken rib Withdrawn due to adverse events: I1: 2/30 (7%) High BP C: 0/30 (0%)	
Notes	Loss to follow-up: 4/30 (13%) (Numbers not reported by group).	
<b>Risk of bias</b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Unclear	B - Unclear

**Frankel 1978**

Methods	Method of randomisation adequate. Blinding: Participant - unclear Treatment provider - no Outcome assessor - yes.
Participants	22 adults (29-63) with currently treated (32%) and untreated (68%) uncomplicated essential hypertension (DBP 90-105). USA. Mean age 46 years, 55% male, 64% white. Inclusion criteria: not stated. Baseline BP: 148/95 0% baseline CVD 0% baseline diabetes.
Interventions	I: Biofeedback. 20 therapist-led laboratory sessions of combined DBP & ECG feedback; autogenic training & progressive relaxation exercises; requested to practice exercises at home using tapes C1: Sham treatment. 20 therapist-led laboratory sessions of sham BP feedback conveying a 'sense of success' C2: No intervention. Treatment duration: 16 weeks
Outcomes	BP at 16 weeks, measured in clinic, supine, averaged over 3 readings of automated device. Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: I: 0/7 (0%) C1: 0/7 (0%) C2: 0/8 (0%) Withdrawn due to adverse events: I: 0/7 (0%) C1: 0/7 (0%)

**Frankel 1978** (Continued)

	C2: 0/8 (0%)	
Notes	Loss to follow-up: I: 0/7 (0%) C1: 0/7 (0%) C2: 0/8 (0%) Anti-hypertensive medication did not vary during the trial.	
<b>Risk of bias</b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Unclear	B - Unclear

**Garcia-Vera 1997**

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - unclear.	
Participants	65 adults (29-63) with currently treated (375%) and untreated (25%) essential hypertension (SBP $\geq$ 140 or DBP $\geq$ 90). Spain. Mean age of those assessed 45 years, 100% male, ??% white. Inclusion criteria: uncontrolled hypertension. Baseline BP: 150/99	
Interventions	I: Stress management. 7 individual, usually weekly, 60-90 minute sessions of progressive relaxation, problem solving therapy, information about hypertension; participants received audio-cassettes to help practise relaxation at home C2: No intervention. Treatment duration: 8 weeks	
Outcomes	BP at 8 weeks, measured in clinic, averaged over 2-3 readings (no further details reported). Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: 5/65 (8%) 4 moved out of area, 1 unknown reasons (not reported by treatment arm) Withdrawn due to adverse events: Not reported	
Notes	Loss to follow-up: 22/65 (34%) (not reported by treatment arm) 4 moved out of area, 1 unknown reasons, 17 participants excluded as anti-hypertensive medication varied during the trial	
<b>Risk of bias</b>		

Garcia-Vera 1997 (Continued)

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Hafner 1982**

Methods	Method of randomisation unclear. Blinding: Participant - unclear Treatment provider - unclear Outcome assessor - unclear.
Participants	23 adults (25-68) with currently treated (90%) and currently untreated (10%) essential hypertension. UK. Mean age 49 years, 57% male, ?% white. Inclusion criteria: medication remained unchanged during the treatment phase. Baseline BP: 155.1/102.3 0% baseline CVD 0% baseline diabetes.
Interventions	I1: Meditation - 2 introductory and 8 x 1 hour weekly therapist-led sessions providing training to achieve relaxation by focusing on breathing or mental image I2: Meditation and biofeedback -2 introductory and 8 x 1 hour weekly therapist-led sessions as I1 with biofeedback of either skin resistance or electromyographic activity to decrease levels of physiological arousal C: No treatment Treatment duration: 8 weeks (BP measurement reported to subjects in I1 & I2 at end of each treatment session)
Outcomes	BP at 5 months, measured in clinic, seated, using automated device (no further details reported). Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: I1: 0/7 (0%) I2: 1/8 (12%) C: 0/8 (0%) Withdrawn due to adverse events: Not reported.
Notes	Loss to follow-up: I1: 0/7 (0%) I2: 1/8 (12%) C: 0/8 (0%) Unclear whether anti-hypertensive medication varied during trial. Not included in primary meta-analysis because of missing SDs of BP

***Risk of bias***

Item	Authors' judgement	Description
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**Hafner 1982** (Continued)

Allocation concealment?	Unclear	B - Unclear
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**Irvine 1986**

Methods	Method of randomisation unclear. Factorial design. Blinding: Participant - no Treatment provider - no Outcome assessor - yes.
Participants	38 adults (34-64) with currently treated (50%) and untreated (50%) hypertension (SBP < 200 mm Hg and DBP 90-109 mm Hg). Canada. Mean age 48, 53% male, ?% white. Inclusion criteria: without secondary complications arising from hypertension, previous MI/ CVA, angina, other concurrent serious medical disorder. Baseline BP: 139.1/91.2 ?% baseline CVD ?% baseline diabetes.
Interventions	I: Relaxation and stress management - training in relaxation with biofeedback of skin resistance, application of stress management techniques in everyday life, education about effects of stress on cardiovascular system; C: Mild physical exercise - mild physical exercise that would not improve cardiovascular fitness, with biofeedback of skin resistance - told increased arousal indicated improved blood circulation; education about effects of stress on cardiovascular system and told that exercise could lower peripheral resistance and reduce BP Treatment duration: 10 weeks Both interventions were 10 x one hour weekly therapist led sessions with individual subjects
Outcomes	BP at 6 months, measured by nurse in clinic, seated, averaged over 4 readings of random zero sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: 6/38 (16%) (Numbers not reported by group) Withdrawn due to adverse events: Not reported.
Notes	Loss to follow-up: 6/38 (16%) (Numbers not reported by group) Anti-hypertensive medication did not vary during the trial.

**Risk of bias**

Item	Authors' judgement	Description
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**Irvine 1986** (Continued)

Allocation concealment?	Unclear	B - Unclear
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**Irvine 1991**

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - yes.
Participants	110 adults (25-64) with currently untreated mild essential hypertension (BP DBP 85-104 in age 18-34, or DBP 90-104 in age 35-59). UK. Mean age 46 years, 82% male, ?% white. Inclusion criteria: not stated. Baseline BP: 137/94 0% baseline CVD 0% baseline diabetes.
Interventions	I: Biofeedback and relaxation therapy. Individual therapist-led weekly ½ hour sessions on hypertension, risks, muscle relaxation, meditation and mental imagery, 'mini-relaxation' training, biofeedback of galvanic skin response. C: Support therapy: behaviour therapist-led weekly sessions. Treatment duration: 12 weeks
Outcomes	BP at 6 months, measured in clinic, seated, averaged over 8 readings of random zero sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects: I: 2/55 (4%) high BP - started anti-hypertensive medication C: 3/55 (6%) 1 cancer, 2 high BP - started anti-hypertensive medication Withdrawn from treatment: I: 5/55 (9%) C: 4/55 (3%) Cancer Withdrawn due to adverse events: I: 2/55 (0%) High BP - started anti-hypertensive medication C: 3/55 (2%) 2 high BP - started anti-hypertensive medication, 1cancer Not reported.
Notes	Loss to follow-up: I: 8/55 (15%) C: 7/55 (13%).

**Risk of bias**

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Jacob 1992**

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - yes.
Participants	20 adults with currently treated hypertension (DBP >90 mmHg). USA. Mean age 49 years, 68% male, 90% white. Inclusion criteria: not stated. Baseline BP: 136.4/85.7 ?% baseline CVD ?% baseline diabetes.
Interventions	I: Relaxation: progressive muscle relaxation and biofeedback of finger temperature - 12 X weekly 40 mins sessions, led by psychiatric nurse. C: Stress education (attention placebo) led by psychiatric nurse Treatment duration: 12 weeks
Outcomes	BP at 12 weeks, measured in clinic, supine, (no further details reported). Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: I: 1/11 (9%) C: 0/9 (0%) Withdrawn due to adverse events: I: 0/11 (0%) C: 0/9 (0%)
Notes	Loss to follow-up: I: 1/11 (9%) C: 0/9 (0%). Anti-hypertensive medication did not vary during the trial.

***Risk of bias***

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Johnston 1993**

Methods	Method of randomisation adequate. Blinding: Participant - no Treatment provider - no Outcome assessor - yes.
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**Johnston 1993** (Continued)

Participants	96 adults (23-59) with currently untreated hypertension, (mean DBP 95-105, treated BP <110). USA. Mean 47 age years, 48% male, ?% white. Inclusion criteria: without CHD, diabetes, BMI >135. Baseline BP: 138/91 0% baseline CVD 0% baseline diabetes.
Interventions	I: Stress management: ten ½ hour psychologist-led sessions on passive relaxation & meditation C: Mild exercise: ten ½ hour psychologist-led sessions on simple stretching exercises Treatment duration: 6 months
Outcomes	BP at 12 months, measured in clinic, seated, averaged over 3 readings of random zero sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: I: 5/48(10%) C: 7/48(15%) Withdrawn due to adverse events: 2/96 (4%) high BP (Numbers not reported by group).
Notes	Loss to follow-up: I: 8/48(17%) C: 16/48(33%).

**Risk of bias**

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Khramelashvili 1986**

Methods	Method of randomisation unclear. Blinding: Participant - unclear Treatment provider - unclear Outcome assessor - unclear.
Participants	80 adults with essential hypertension. USSR mean age ?, ?% male, ?% white. Inclusion criteria: ?. Baseline BP: ? ?% baseline CVD ?% baseline diabetes.
Interventions	I1: Autogenic training I2: Biofeedback C: No treatment

**Khramelashvili 1986** (Continued)

	Treatment duration: Unclear	
Outcomes	BP at 12 months Adverse effects: unclear. Withdrawn from treatment: unclear.	
Notes	Loss to follow-up: Not reported. Not included in primary meta-analysis because of missing SDs of BP	
<b>Risk of bias</b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Unclear	B - Unclear

**LaGrone 1988**

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - yes.	
Participants	39 adults (33-66) with currently treated (97%) and untreated (3%) essential hypertension, BP > 140/90 mm Hg before drug treatment. USA. Mean age 51 years, 17% male, ?% white. Inclusion criteria: not stated. Baseline BP: 138.2/89.4 0% baseline CVD 0% baseline diabetes.	
Interventions	I1: 8 x 90 mins therapist-led education and relaxation sessions over 2 weeks; hypertension & circulatory system, sodium and potassium, smoking & exercise, personality factors, time management, stress and CVD, additional instruction and practice in progressive muscle relaxation after session I2: Education alone - as I1 but no relaxation training C: No treatment or instructions Treatment duration: 2 weeks Treatment duration:	
Outcomes	BP at 8 weeks, measured seated, averaged over 2 readings (no further details reported). Deaths and cardiovascular events not reported. Adverse effects: more than one third of participants associated adverse events (dry mouth, weight gain, thirst, drowsiness, loss of energy, muscle cramps, muscle pain, heart palpitations, sexual difficulty, fatigue, depression, insomnia) with their anti-hypertensive medications. Withdrawn from treatment: 9/39 (23%) (Numbers not reported by group). Withdrawn due to adverse events: Not reported.	



**LaGrone 1988** (Continued)

Notes	Loss to follow-up: 9/39 (23%) (Numbers not reported by group). Anti-hypertensive medication did not vary during the trial. Not included in primary meta-analysis because of missing SDs of BP
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**Risk of bias**

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**McGrady 1981**

Methods	Method of randomisation unclear. Blinding: Participant - unclear Treatment provider - unclear Outcome assessor - unclear.
Participants	43 adults with currently treated (88%) and untreated (12%) essential hypertension. USA. Mean age 50 years, 32% male, 97% male. Inclusion criteria: not stated. Baseline BP: 142.8/90.7 ?% baseline CVD ?% baseline diabetes.
Interventions	I: Biofeedback assisted relaxation - clinic staff led 30 minute sessions X twice weekly of EMG feedback with autogenic exercise training (to be practised twice daily for 15 mins) C: BP measuring only Treatment duration : 8 weeks
Outcomes	BP at 8 weeks, measured with automatic sphygmomanometer (no further details reported). Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: I: 3/25 (12%) C: 2/18 (11%) NB 3 withdrawals due to change in medication. Withdrawn due to adverse events: Not reported.
Notes	Loss to follow-up: I: 3/25 (12%) C: 2/18 (11%) Participants asked not to vary medication during the trial.

**Risk of bias**

McGrady 1981 (Continued)

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

McGrady 1994

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - unclear.
Participants	138 adults with currently treated (77%) and untreated (23%) essential hypertension. USA. Mean age 48 years, 39% male, 75% white. Inclusion criteria: not stated. Baseline BP: 132/86 0% baseline CVD 0% baseline diabetes.
Interventions	I: Group relaxation and feedback. Weekly 45 minute therapist-led sessions providing autogenic relaxation training, progressive muscle relaxation, biofeedback of finger temperature, encouraged to practise at home. C: No intervention (waiting list). Treatment duration: 8 weeks
Outcomes	BP at 11 weeks, measured in clinic, seated, averaged over 3 readings (no further details reported). Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: 37/138 (27%) (Numbers not reported by group). Withdrawn due to adverse events: Not reported.
Notes	Loss to follow-up: 37/138 (27%) (Numbers not reported by group). Numbers randomised to each group not reported. Participants excluded from analysis if medication varied during the trial

*Risk of bias*

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Murugasan 2000**

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - unclear.	
Participants	22 adults with essential hypertension. India. Mean age 48 years, gender, ethnicity not reported. Inclusion criteria: not stated. Baseline BP: 156/109 ?% baseline CVD ?% baseline diabetes.	
Interventions	I: Yoga: 2 half-hour sessions 6 days/week C: No intervention . Treatment duration: 11 weeks	
Outcomes	BP at 11 weeks, measured with standard sphygmomanometer (no further details reported). Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: I: 0/11 (0%) C: 3/11 (27%) Withdrawn due to adverse events: Not reported.	
Notes	Loss to follow-up: I: 1/11 (9%) C: 2/11 (18%) Participants excluded from analysis if emergency medical care needed	
<b><i>Risk of bias</i></b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Unclear	B - Unclear

**Patel 1988**

Methods	Method of randomisation adequate. Blinding: Participant - no Treatment provider - no Outcome assessor - no.	
Participants	134 adults (35-64) with currently treated (30%) and untreated (70%) mild essential hypertension (DBP 90-109). UK. Mean age 53 years, 51% male, ?% white. Inclusion criteria: not stated.	

Patel 1988 (Continued)

	Baseline BP: 140.1/86.8 0% baseline CVD 0% baseline diabetes.	
Interventions	I: Group relaxation and biofeedback. Weekly physician and nurse-led 1 hour sessions: stress management, anger control, coping strategies, communication skills, breathing exercises, deep muscle relaxation and simple meditation training provided; skin resistance biofeedback provided; home practice with taped instructions encouraged. C: No intervention. Treatment duration: 8 weeks	
Outcomes	BP at 1 year, measured in clinic, averaged over 2 readings (no further details reported). Deaths by 1 year: I: 1/49* (2%) carcinoma of colon C: 0/54* (0%) Angina/myocardial infarction by 1 year: I: 0/49* (0%) C: 2/54* (1%) 1 angina, 1 myocardial infarction; also 1 possible myocardial infarction and 1 possible myocardial ischaemia as determined by ECG Stroke by 1 year: I: 1/49* (2%) C: 0/54* (0%) * These are numbers of participants assessed at 1 year; numbers of participants randomised were not reported by treatment group. No other adverse events reported. Withdrawn from treatment: 23/134(17%) (Numbers not reported by group). Withdrawn due to adverse events: Not reported.	
Notes	Loss to follow-up: 31/134 (23%). (Numbers not reported by group). Unclear whether anti-hypertensive medication varied during the trial The trial was a sub-study of the MRC mild hypertension trial of active drug vs. placebo	
<b>Risk of bias</b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Yes	A - Adequate

**Schein 2001**

Methods	Method of randomisation adequate. Blinding: Participant - yes Treatment provider - yes Outcome assessor - yes.	
Participants	61 adults with currently treated or untreated essential hypertension (BP $\geq$ 140/90, home BP >135/85). Israel. Mean age 57 years, 47% male, ?% white. Inclusion criteria: without CHD, CVD, renal disease, diabetes, BMI >35 kg/m. Baseline BP: 156/95 0% baseline CVD 0% baseline diabetes.	
Interventions	I: BIM: 'breathe with interactive music' . Biofeedback using headphone and respiration sensor to allow participant to listen to sounds mimicking own breathing pattern but with prolonged expiration, which encourages participant to modify their breathing pattern. C: Passive treatment. Listening to quiet synthesised music with non-identifiable rhythm Treatment duration: 8 weeks Both groups: self-treatment at home, 10 minutes daily.	
Outcomes	BP at 8 weeks, measured in clinic, seated, averaged over 3-5 readings of mercury or aneroid sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: I: 0/32(0%) C: 0/33(0%) Withdrawn due to adverse events: I: 0/32(0%) C: 0/33(0%)	
Notes	Loss to follow-up: I: 0/32 (0%) C: 4/33 (12%). Anti-hypertensive medication did not vary during the trial.	
<b><i>Risk of bias</i></b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Unclear	B - Unclear

Seer 1980

Methods	Method of randomisation unclear. Blinding: Participant - no Treatment provider - no Outcome assessor - unclear.	
Participants	41 adults (22-62) with currently untreated essential hypertension. New Zealand. Mean age 43 years, 56% male, 7% white. Inclusion criteria: without CHD, diabetes, renal disease. Baseline BP: 150/102 0% baseline CVD 0% baseline diabetes.	
Interventions	I : Transcendental meditation. Psychiatrist-led sessions twice daily for 15-20 minutes with mantra recitation. C1: Sham control: psychiatrist-led training twice daily 15-20 minutes without mantra recitation. C2: No intervention (waiting list). Treatment duration: 5 weeks	
Outcomes	BP at 13 weeks, measured in clinic, seated, averaged over 5 readings of random zero sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects: I1: 1/14 (7%) started anti-hypertensive medication - excluded from analysis I2: 3/14 (21%) 2 started anti-hypertensive medication - excluded from analysis, 1 other drug complications C: 0/13 (0%). Withdrawn from treatment: Not reported. Withdrawn due to adverse events: Not reported.	
Notes	Loss to follow-up: I1: 2/14 (14%) 1 started anti-hypertensive medication C1: 3/14 (21%) 2 started anti-hypertensive medication C2: 0/13 (0%). 3 participants excluded from analysis if medication varied during the trial	
<b>Risk of bias</b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Unclear	B - Unclear

**van Montfrans 1990**

Methods	Method of randomisation adequate. Blinding: Participant - unclear Treatment provider - unclear Outcome assessor - unclear.
Participants	42 adults (24-60) with currently untreated hypertension. The Netherlands. Mean age 42 years, 52% male, 90% white. Inclusion criteria: without diabetes, CHD, target organ damage. Baseline BP: 154.7/99.8 0% baseline CVD 0% baseline diabetes.
Interventions	I: Relaxation therapy - therapist-led weekly one hour sessions on yoga, breathing, posture exercises, meditation, autogenic training and progressive muscle relaxation. C: Non-specific counselling - nurse led sessions encouraging passive relaxation and explaining role of stress in hypertension Treatment duration: 8 weeks
Outcomes	BP at 12 months, measured in clinic, averaged over 3 readings of random zero sphygmomanometer, (no further details reported). Deaths and cardiovascular events not reported. Adverse effects: I: 0/23 (0%) C: 2/19(11%) 1 chest pain, 1 high BP. Withdrawn from treatment: I: 3/23 (13%) C: 2/19(11%) Withdrawn due to adverse events: I: 0/23 (0%) C: 2/19(11%) 1 chest pain, 1 high BP.
Notes	Loss to follow-up: I: 5/23 (22%) C: 2/19(11%).

***Risk of bias***

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Yen 1996**

Methods	Method of randomisation unclear. Cluster randomised. Blinding: Participant - no Treatment provider - no Outcome assessor - adequate.	
Participants	284 adults with treatment naive or currently treated hypertension (SBP $\geq$ 140 or DBP $\geq$ 90 mm Hg). Taiwan. Mean age 54 years, 65% male, ?% white. Inclusion criteria: not reported. Baseline BP: 145.2/88.4 ?% baseline CVD ?% baseline diabetes.	
Interventions	I: Progressive relaxation technique training individually at home, once weekly by nurse for 8 weeks C: No intervention. Treatment duration: 2 months Additional arms which received (i) routine BP measurement and (ii) self-learning packages were excluded as the trial investigators considered these as active interventions	
Outcomes	BP at 4 months, measured at home, seated, averaged over 2 readings of mercury sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects not reported. Withdrawn from treatment: Most drop-outs refused to participate in therapy; numbers not reported. NB Numbers of participants randomised and withdrawn from treatment were adjusted to allow for inter-cluster correlation. Withdrawn due to adverse events: Not reported.	
Notes	Loss to follow-up: I1: 31/58 (53%) C: 60/113 (53%). Unclear whether anti-hypertensive medication varied during the trial. This is a cluster randomised trial. Numbers of participants randomised and lost to follow-up were adjusted to allow for inter-cluster correlation SDs of BP estimated from CI in Table 3.	
<b><i>Risk of bias</i></b>		
<b>Item</b>	<b>Authors' judgement</b>	<b>Description</b>
Allocation concealment?	Unclear	B - Unclear



**Zurawski 1987**

Methods	Method of randomisation unclear. Blinding: Participant - unclear Treatment provider - no Outcome assessor - unclear.
Participants	29 adults with currently treated or untreated essential hypertension. USA. Mean age 47 years, 28% male, 100% white. Inclusion criteria: not excessively overweight and those on medication had dosage stabilised for >= 3 months. Baseline BP: 137.5/86.3 ?% baseline CVD ?% baseline diabetes.
Interventions	I: Multi-modal stress management training. Weekly 1-1½ hour therapist-led group sessions: progressive muscular relaxation, role of cognitions in stressful situations and coping strategies, learned cue controlled breathing and relaxation imagery. C: Sham therapy. Weekly 1-1½ hour therapist-led group sessions: biofeedback of galvanic skin resistance Treatment duration: 8 weeks
Outcomes	BP at 6 months, measured in clinic, seated, averaged over 4 readings of digital sphygmomanometer. Deaths and cardiovascular events not reported. Adverse effects: some participants reported illness, but numbers not reported. Withdrawn from treatment: 4/29 (14%) (Numbers not reported by group). Withdrawn due to adverse events: Some participants withdrew due to illness, but numbers not reported
Notes	Loss to follow-up: 7/29 (24%) (Numbers not reported by group). Unclear whether anti-hypertensive medication varied during the trial

***Risk of bias***

Item	Authors' judgement	Description
Allocation concealment?	Unclear	B - Unclear

**Characteristics of excluded studies [ordered by study ID]**

Study	Reason for exclusion
Agras 1983	Anti-hypertensive medication varied during trial
Agras 1984	Anti-hypertensive medication varied during trial

(Continued)

Agras 1987	Anti-hypertensive medication varied during trial
Aivazyan 1988a	Anti-hypertensive medication varied during trial
Albright 1991	No control group
Alexander 1989	Unable to abstract data for intention-to-treat analysis
Alexander 1996	No control group
Allen 2001	No outcome BP data
Amigo 2001	Study design not randomised
Andersson 1985	Unclear if participants were hypertensive or normotensive
Bak 1991	Normotensive participants
Bali 1979	Study design not randomised
Benson 1974	No control group
Benson 1978	No outcome BP data
Bharshankar 2003	Study design not randomised
Blackwell 1976	No control group
Blanchard 1987	Anti-hypertensive medication varied during trial
Blanchard 1988	Anti-hypertensive medication varied during trial
Brauer 1979	Anti-hypertensive medication varied during trial
Bruning 1987	Normotensive participants
Campbell 1996	Normotensive participants
Cantor 1985	No outcome BP data
Carlson 1990	< 8 week follow-up
Carnahan 1975	Anti-hypertensive medication varied during trial
Charlesworth 1984	Unable to abstract data for intention-to-treat analysis
Chesney 1987	Anti-hypertensive medication varied during trial

(Continued)

Cheung 2005	No control group
Christoph 1978	Study design not randomised
Cohen 1983	Anti-hypertensive medication varied during trial
Crowther 1983	Anti-hypertensive medication varied during trial
Damodaran 2002	Study design not randomised
Datey 1969	Study design not randomised
DeFrank 1987	Study design not randomised
Delmonte 1985	< 8 week follow-up
Duivenvoorden 1991	No outcome BP data
Elliot 2004	Unable to abstract data for intention-to-treat analysis
Ewart 1984	Study design not randomised, no CV outcome reported
Friedman 1977	< 8 week follow-up
Glasgow 1982	Study design not randomised
Goebel 1980	Study design not randomised
Goebel 1993	Study design not randomised
Goldstein 1984	Study design not randomised
Goldstein 1987	< 8 week follow-up
Granath 2006	Normotensive participants
Greenspan 1980	No outcome BP data
Grossman 2001	Unable to abstract data for intention-to-treat analysis
Haber 1983	Anti-hypertensive medication varied during trial
Hager 1978	< 8 week follow-up
Hahn 1993	< 8 week follow-up

(Continued)

Harinath 2004	Normotensive participants
Harrison 1979	No control group
Hatch 1985	Anti-hypertensive medication varied during trial
Henderson 1998	< 8 week follow-up
Hoelscher 1986	Anti-hypertensive medication varied during trial
Hoelscher 1987	Anti-hypertensive medication varied during trial
Jacob 1986	Crossover design
Jin 1992	Normotensive participants
Jorgensen 1981	< 8 week follow-up
Kallinke 1982	No control group
Katzenstein 1974	Participants were children
Kondwani 2005	No control group
Larkin 1996	Study design not randomised
Lee 2004	Normotensive participants
Lehnert 1987	Study design not randomised, Anti-hypertensive-medication varied during trial
Linden 1997	Anti-hypertensive medication varied during trial
Linden 2003	Comment/editorial
Luborsky 1980	< 8 week follow-up
Mancini 1983	Normotensive participants
McCaffrey 2005	Study design not randomised
McGrady 1986	Study design not randomised
McGrady 1987	No control group
McGrady 1987b	No control group

(Continued)

Nakao 2000	< 8 week follow-up
Nath 1979	Study design not randomised
Nickel 2005	Normotensive participants
Olney 2005	Normotensive participants
Patel 1975a	Study design not randomised, anti-hypertensive medication varied during trial
Patel 1975b	No control group
Patel 1977	Study design not randomised
Patel 1981	Anti-hypertensive medication varied during trial
Paul-Labrador 2006	Normotensive participants
Pender 1985	Study design not randomised
Pollack 1977	No control group
Richter 1981	No control group outcome data
Richter 1982	Study design not randomised
Roberts 1979	Study design not randomised
Schneider 1995	No control group
Schneider 2005	Anti-hypertensive medication varied during trial
Shapiro 1997	Anti-hypertensive medication varied during trial
Shoemaker 1975	< 8 week follow-up
Stephoe 1976	Study design not randomised, < 8 week follow-up, normotensive participants
Stone 1976	Study design not randomised
Suls 1986	Normotensive participants
Surwit 1978	Study design not randomised
Tamez 1978	Normotensive participants

(Continued)

Taylor 1977	Anti-hypertensive medication varied during trial
TOHP 1992	Normotensive participants
Vinck 1978	Normotensive participants
Wadden 1984	Anti-hypertensive medication varied during trial
Walsh 1977	< 8 week follow-up
Wang 1989	No outcome BP data
Webb 2006	Normotensive participants
Wenneberg 1997	Normotensive participants
Wittrock 1988	Study design not randomised, normotensive participants,
Wood 1986	Study design not randomised

## DATA AND ANALYSES

### Comparison 1. Relaxation versus control

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Death	1		Risk Ratio (M-H, Fixed, 95% CI)	Subtotals only
2 Myocardial infarction	1		Risk Ratio (M-H, Random, 95% CI)	Subtotals only
3 Stroke	1		Risk Ratio (M-H, Random, 95% CI)	Subtotals only

### Comparison 2. Relaxation versus control (sub-grouped by duration of follow-up)

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-5.50 [-8.21, -2.78]
1.1 relaxation vs control < 6 months follow-up	13	590	Mean Difference (IV, Random, 95% CI)	-7.12 [-11.45, -2.79]
1.2 relaxation vs control >= 6 months follow-up	12	608	Mean Difference (IV, Random, 95% CI)	-4.03 [-7.59, -0.48]
2 Diastolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-3.49 [-5.34, -1.64]
2.1 relaxation vs control < 6 months follow-up	13	590	Mean Difference (IV, Random, 95% CI)	-5.15 [-8.44, -1.86]
2.2 relaxation vs control >= 6 months follow-up	12	608	Mean Difference (IV, Random, 95% CI)	-1.94 [-3.77, -0.12]

### Comparison 3. Relaxation versus control (sub-grouped by type of control)

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-5.61 [-8.25, -2.97]
1.1 relaxation vs sham therapy	15	564	Mean Difference (IV, Random, 95% CI)	-3.47 [-7.12, 0.18]
1.2 relaxation vs non-intervention control	13	634	Mean Difference (IV, Random, 95% CI)	-7.70 [-11.16, -4.25]
2 Diastolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-3.54 [-5.37, -1.71]
2.1 relaxation vs sham therapy	15	564	Mean Difference (IV, Random, 95% CI)	-1.81 [-4.40, 0.79]
2.2 relaxation vs non-intervention control	13	634	Mean Difference (IV, Random, 95% CI)	-5.27 [-7.72, -2.81]

#### Comparison 4. Relaxation versus control (subgrouped by initial BP)

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-5.50 [-8.21, -2.78]
1.1 Initial SBP above median	13	607	Mean Difference (IV, Random, 95% CI)	-7.30 [-11.33, -3.27]
1.2 Initial SBP equal to or below median	12	591	Mean Difference (IV, Random, 95% CI)	-3.26 [-6.60, 0.07]
2 Diastolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-3.49 [-5.34, -1.64]
2.1 Initial DBP above median	13	666	Mean Difference (IV, Random, 95% CI)	-4.04 [-6.92, -1.16]
2.2 Initial DBP equal to or below median	12	532	Mean Difference (IV, Random, 95% CI)	-2.86 [-4.89, -0.83]

#### Comparison 5. Relaxation versus control (including trials with imputed SDs)

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP (high imputed SDs)	28	1330	Mean Difference (IV, Random, 95% CI)	-5.72 [-8.35, -3.10]
1.1 Trials with reported SDs	25	1198	Mean Difference (IV, Random, 95% CI)	-5.50 [-8.21, -2.78]
1.2 Trials with imputed SDs	3	132	Mean Difference (IV, Random, 95% CI)	-8.72 [-20.41, 2.97]
2 Diastolic BP (high imputed SDs)	28	1330	Mean Difference (IV, Random, 95% CI)	-3.82 [-5.63, -2.02]
2.1 Trials with reported SDs	25	1198	Mean Difference (IV, Random, 95% CI)	-3.49 [-5.34, -1.64]
2.2 Trials with imputed SDs	3	132	Mean Difference (IV, Random, 95% CI)	-7.94 [-13.71, -2.17]
3 Systolic BP (low imputed SDs)	28	1330	Mean Difference (IV, Random, 95% CI)	-5.93 [-8.71, -3.14]
3.1 Trials with reported SDs	25	1198	Mean Difference (IV, Random, 95% CI)	-5.50 [-8.21, -2.78]
3.2 Trials with imputed SDs	3	132	Mean Difference (IV, Random, 95% CI)	-8.51 [-20.20, 3.18]
4 Diastolic BP (low imputed SDs)	28	1330	Mean Difference (IV, Random, 95% CI)	-4.05 [-6.01, -2.10]
4.1 Trials with reported SDs	25	1198	Mean Difference (IV, Random, 95% CI)	-3.49 [-5.34, -1.64]
4.2 Trials with imputed SDs	3	132	Mean Difference (IV, Random, 95% CI)	-7.48 [-12.82, -2.14]

#### Comparison 6. Relaxation versus control including only trials reporting adequate concealment of allocation

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP	1		Mean Difference (IV, Random, 95% CI)	Subtotals only
2 Diastolic BP	1		Mean Difference (IV, Random, 95% CI)	Subtotals only



**Comparison 7. Relaxation versus control including only trials reporting blinded outcome assessment**

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP	9	498	Mean Difference (IV, Random, 95% CI)	-3.15 [-7.66, 1.37]
2 Diastolic BP	9	498	Mean Difference (IV, Random, 95% CI)	-2.06 [-5.31, 1.18]

**Comparison 8. Relaxation versus control (sub-grouped by with/without biofeedback)**

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-5.68 [-8.38, -2.98]
1.1 Relaxation with biofeedback	12	592	Mean Difference (IV, Random, 95% CI)	-5.40 [-8.71, -2.08]
1.2 Relaxation without biofeedback	14	606	Mean Difference (IV, Random, 95% CI)	-5.95 [-10.11, -1.79]
2 Diastolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-3.72 [-5.54, -1.90]
2.1 Relaxation with biofeedback	12	592	Mean Difference (IV, Random, 95% CI)	-2.82 [-5.18, -0.46]
2.2 Relaxation without biofeedback	14	606	Mean Difference (IV, Random, 95% CI)	-4.60 [-7.42, -1.77]

**Comparison 9. Relaxation with biofeedback versus control (subgrouped by type of control)**

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP	12	592	Mean Difference (IV, Random, 95% CI)	-5.80 [-9.01, -2.59]
1.1 Biofeedback vs. sham therapy	8	283	Mean Difference (IV, Random, 95% CI)	-5.86 [-11.04, -0.67]
1.2 Biofeedback vs. non-intervention control	6	309	Mean Difference (IV, Random, 95% CI)	-6.34 [-10.08, -2.60]
2 Diastolic BP	12	592	Mean Difference (IV, Random, 95% CI)	-3.33 [-5.55, -1.10]
2.1 Biofeedback vs. sham therapy	8	283	Mean Difference (IV, Random, 95% CI)	-3.27 [-6.91, 0.37]
2.2 Biofeedback vs. non-intervention control	6	309	Mean Difference (IV, Random, 95% CI)	-3.24 [-5.69, -0.80]

**Comparison 10. Relaxation versus control (sub-grouped by with/without cognitive/behavioural therapy)**

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-5.68 [-8.38, -2.98]
1.1 Relaxation with cognitive/behavioural therapy	11	477	Mean Difference (IV, Random, 95% CI)	-6.29 [-11.73, -0.84]
1.2 Relaxation without cognitive/behavioural therapy	15	721	Mean Difference (IV, Random, 95% CI)	-5.06 [-7.77, -2.35]
2 Diastolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-3.72 [-5.54, -1.90]
2.1 Relaxation with cognitive/behavioural therapy	11	477	Mean Difference (IV, Random, 95% CI)	-4.55 [-8.04, -1.06]
2.2 Relaxation without cognitive/behavioural therapy	15	721	Mean Difference (IV, Random, 95% CI)	-3.16 [-5.15, -1.17]

**Comparison 11. Relaxation versus control (sub-grouped by progressive muscle relaxation/other)**

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-5.50 [-8.20, -2.81]
1.1 Progressive muscle relaxation	16	699	Mean Difference (IV, Random, 95% CI)	-4.82 [-7.25, -2.38]
1.2 Other	10	499	Mean Difference (IV, Random, 95% CI)	-6.84 [-13.21, -0.46]
2 Diastolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-3.46 [-5.33, -1.59]
2.1 Progressive muscle relaxation	16	699	Mean Difference (IV, Random, 95% CI)	-2.84 [-4.77, -0.90]
2.2 Other	10	499	Mean Difference (IV, Random, 95% CI)	-4.83 [-8.78, -0.88]

**Comparison 12. Relaxation versus control (sub-grouped by autogenic training/other)**

Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Systolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-5.50 [-8.21, -2.78]
1.1 Autogenic training	6	358	Mean Difference (IV, Random, 95% CI)	-2.33 [-7.86, 3.19]
1.2 Other	19	840	Mean Difference (IV, Random, 95% CI)	-6.61 [-9.79, -3.42]
2 Diastolic BP	25	1198	Mean Difference (IV, Random, 95% CI)	-3.49 [-5.34, -1.64]
2.1 Autogenic training	6	358	Mean Difference (IV, Random, 95% CI)	-1.60 [-4.80, 1.59]
2.2 Other	19	840	Mean Difference (IV, Random, 95% CI)	-4.17 [-6.44, -1.91]

### Comparison 13. Relaxation versus control

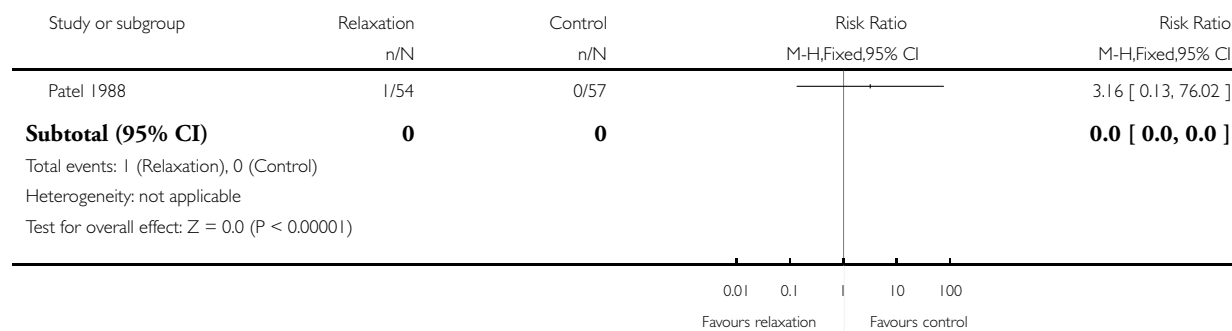
Outcome or subgroup title	No. of studies	No. of participants	Statistical method	Effect size
1 Adverse events - uncontrolled hypertension	5	287	Risk Difference (M-H, Random, 95% CI)	-0.00 [-0.05, 0.04]
2 Other adverse events	6	389	Risk Difference (M-H, Random, 95% CI)	-0.02 [-0.05, 0.01]
3 Withdrawal from treatment	14	695	Risk Difference (M-H, Random, 95% CI)	0.03 [-0.03, 0.09]
4 Withdrawals due to adverse events - uncontrolled hypertension	7	322	Risk Difference (M-H, Random, 95% CI)	-0.00 [-0.04, 0.04]
5 Withdrawals due to other adverse events	6	261	Risk Difference (M-H, Random, 95% CI)	-0.01 [-0.05, 0.03]
6 Loss to follow-up	13	675	Risk Difference (M-H, Random, 95% CI)	0.01 [-0.07, 0.09]

### Analysis 1.1. Comparison 1 Relaxation versus control, Outcome 1 Death.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 1 Relaxation versus control

Outcome: 1 Death

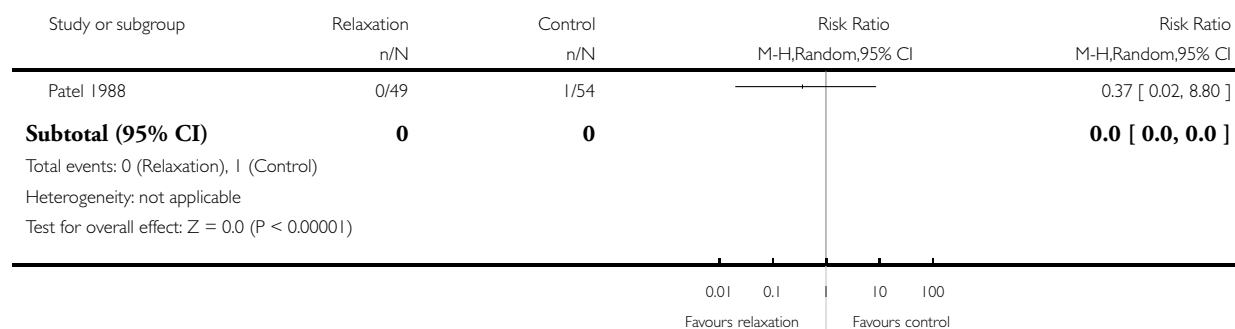


### Analysis 1.2. Comparison 1 Relaxation versus control, Outcome 2 Myocardial infarction.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 1 Relaxation versus control

Outcome: 2 Myocardial infarction

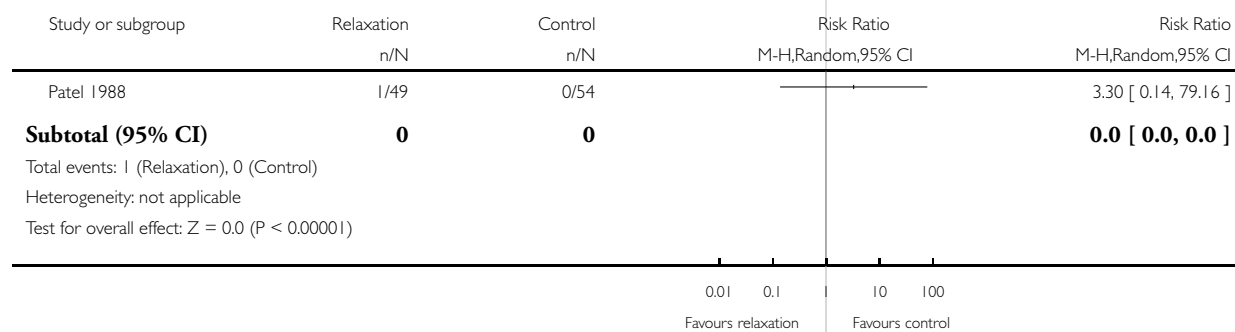


### Analysis 1.3. Comparison 1 Relaxation versus control, Outcome 3 Stroke.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 1 Relaxation versus control

Outcome: 3 Stroke

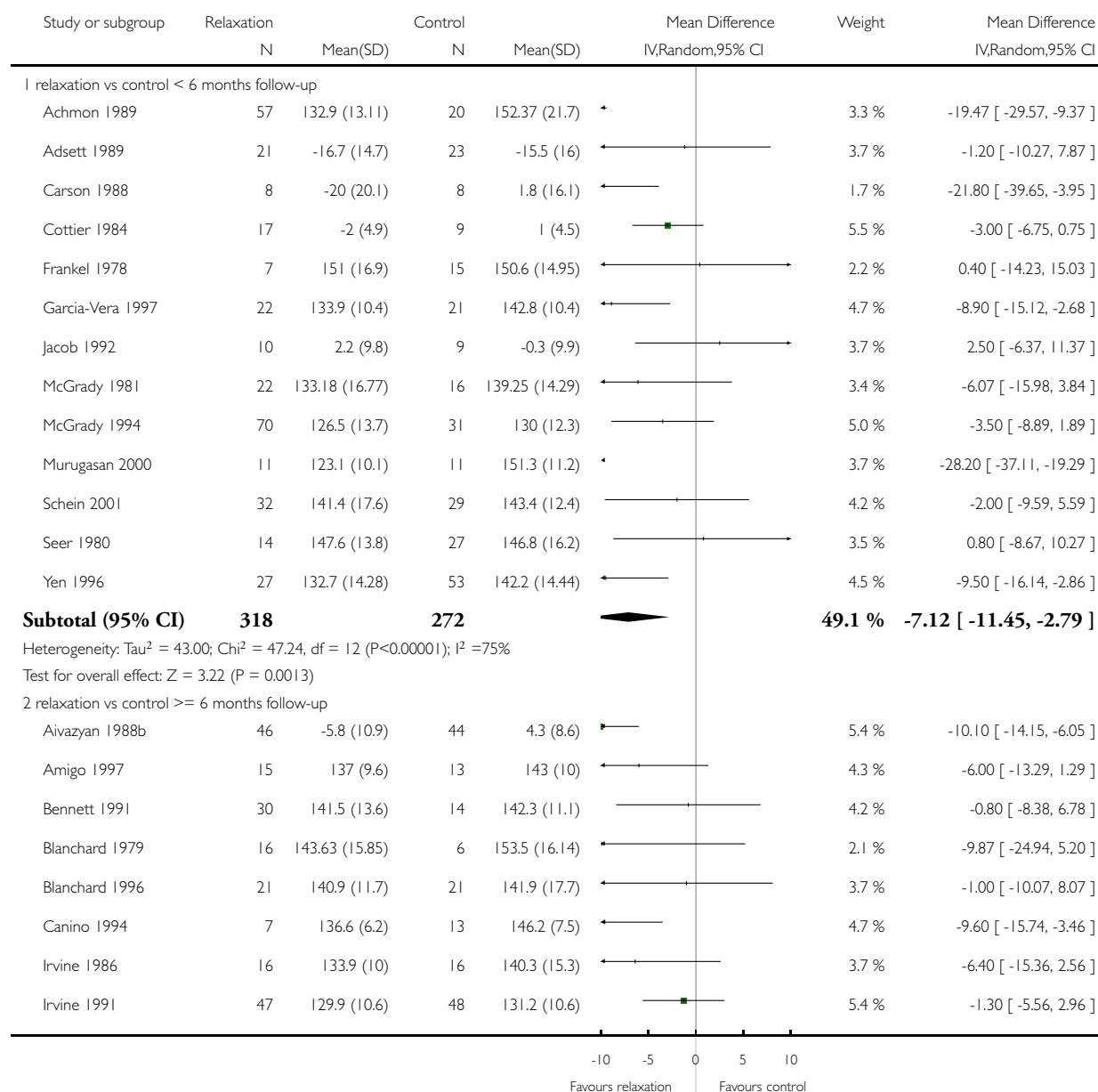


## Analysis 2.1. Comparison 2 Relaxation versus control (sub-grouped by duration of follow-up), Outcome 1 Systolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

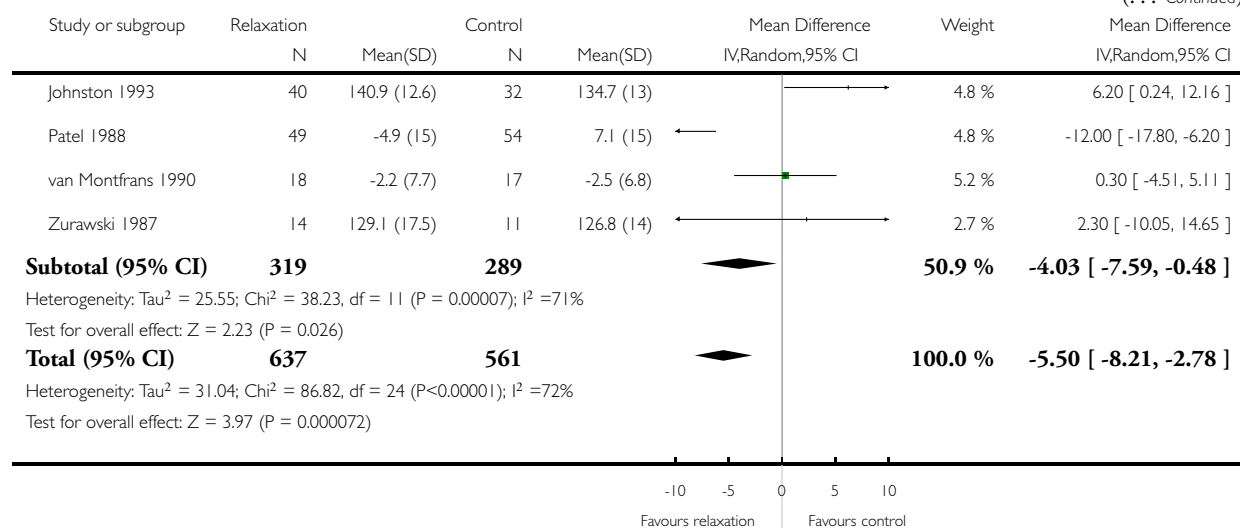
Comparison: 2 Relaxation versus control (sub-grouped by duration of follow-up)

Outcome: 1 Systolic BP



(Continued ...)

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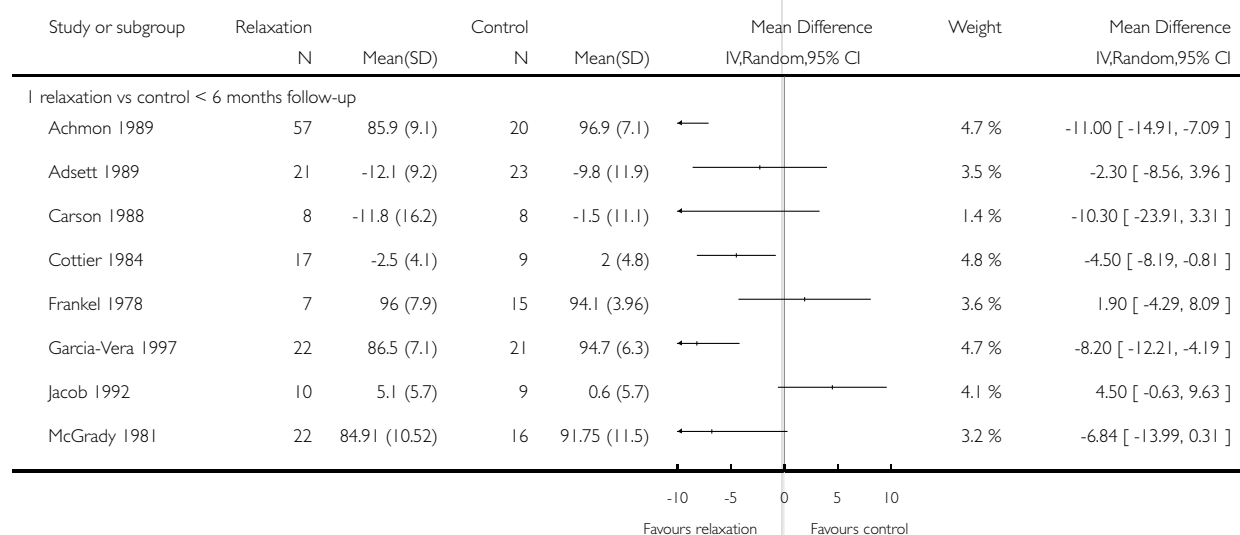


## Analysis 2.2. Comparison 2 Relaxation versus control (sub-grouped by duration of follow-up), Outcome 2 Diastolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

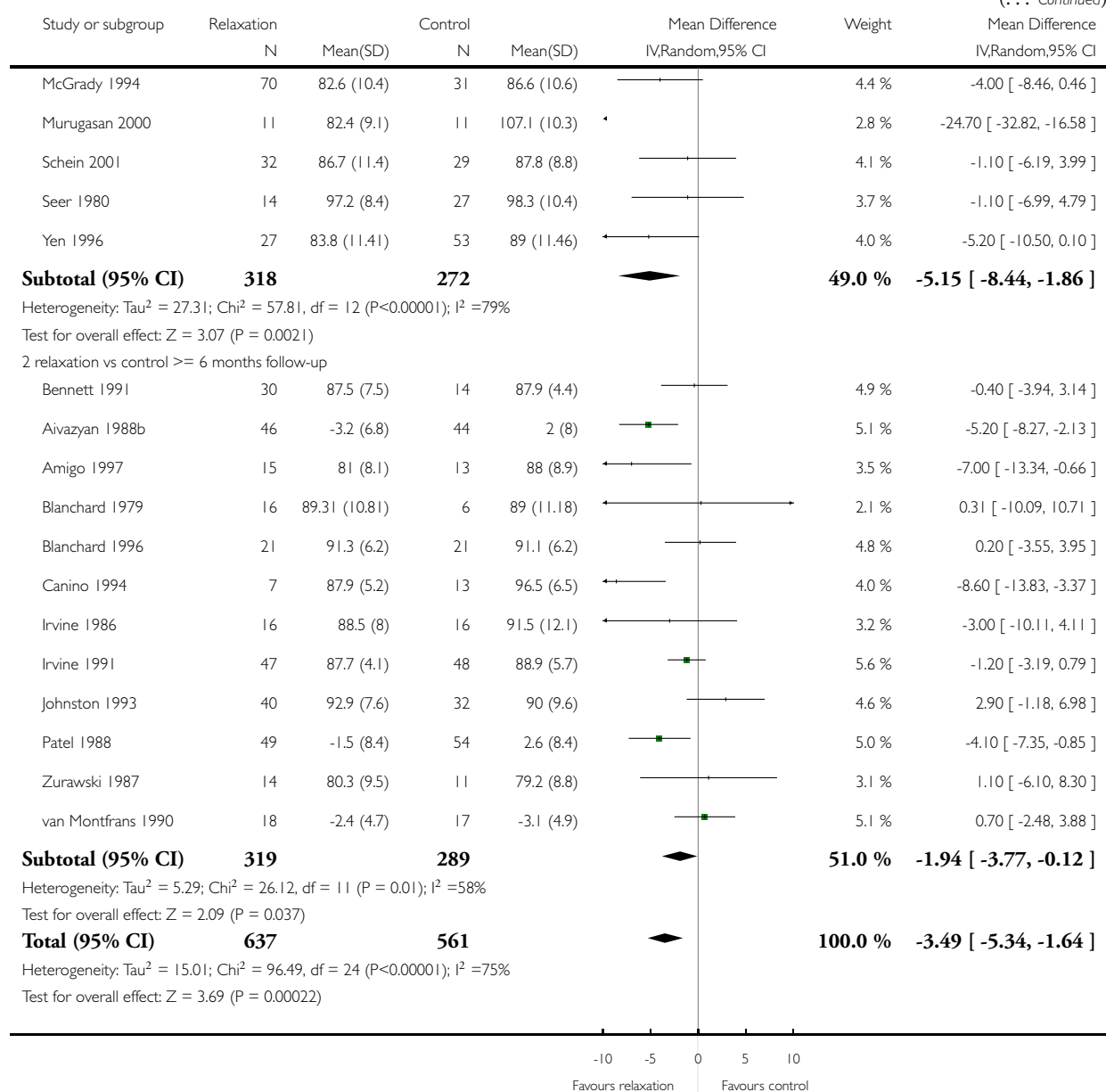
Comparison: 2 Relaxation versus control (sub-grouped by duration of follow-up)

Outcome: 2 Diastolic BP



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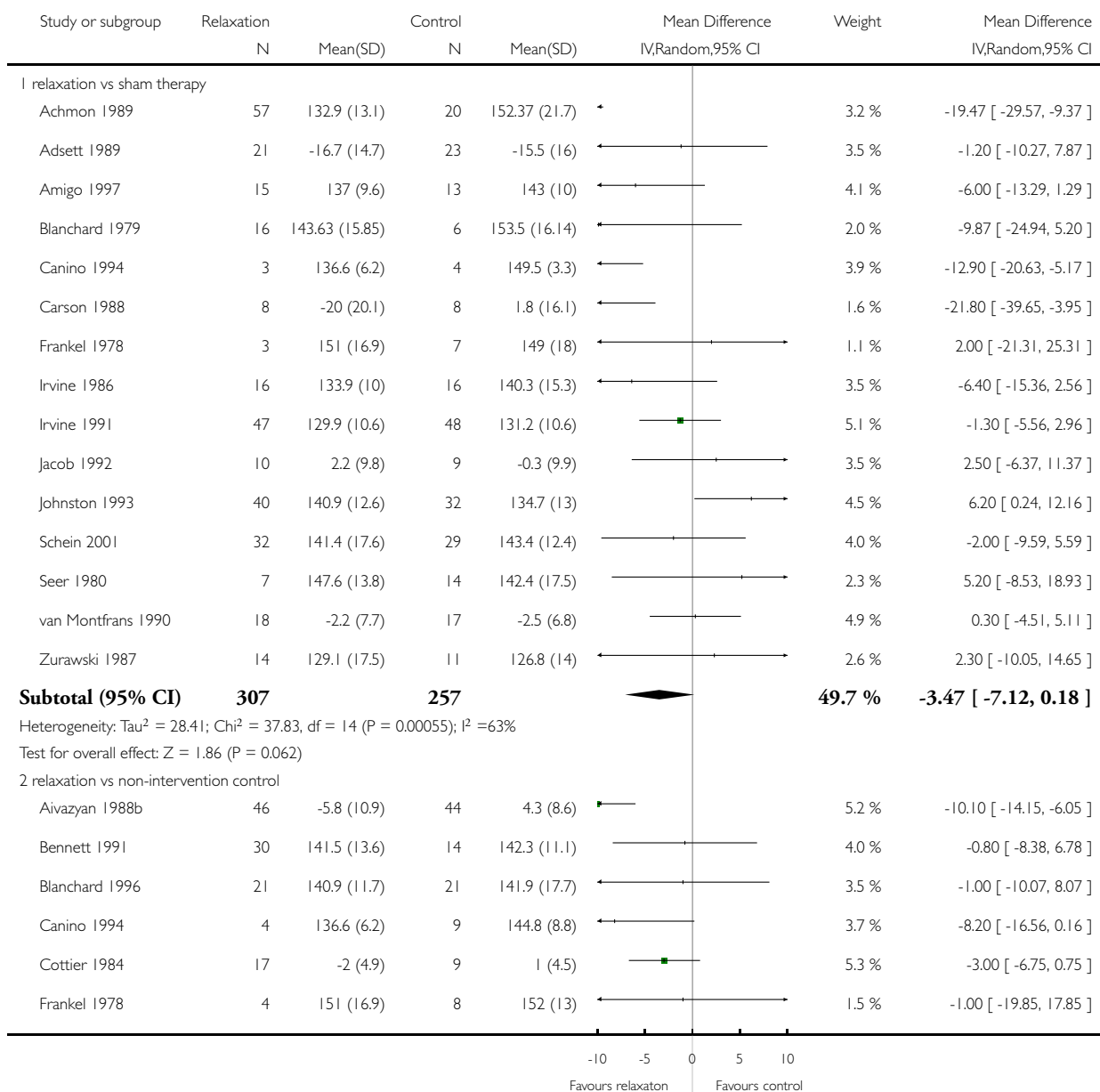


### Analysis 3.1. Comparison 3 Relaxation versus control (sub-grouped by type of control), Outcome 1 Systolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

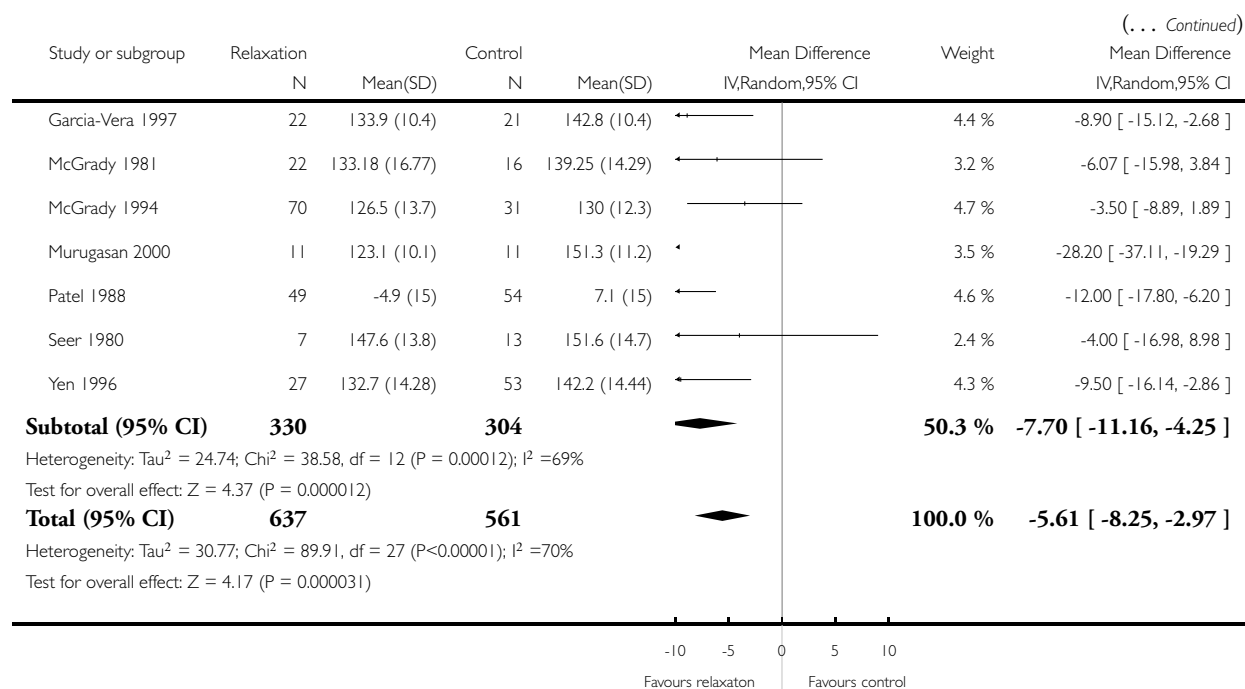
Comparison: 3 Relaxation versus control (sub-grouped by type of control)

Outcome: 1 Systolic BP



(Continued . . .)



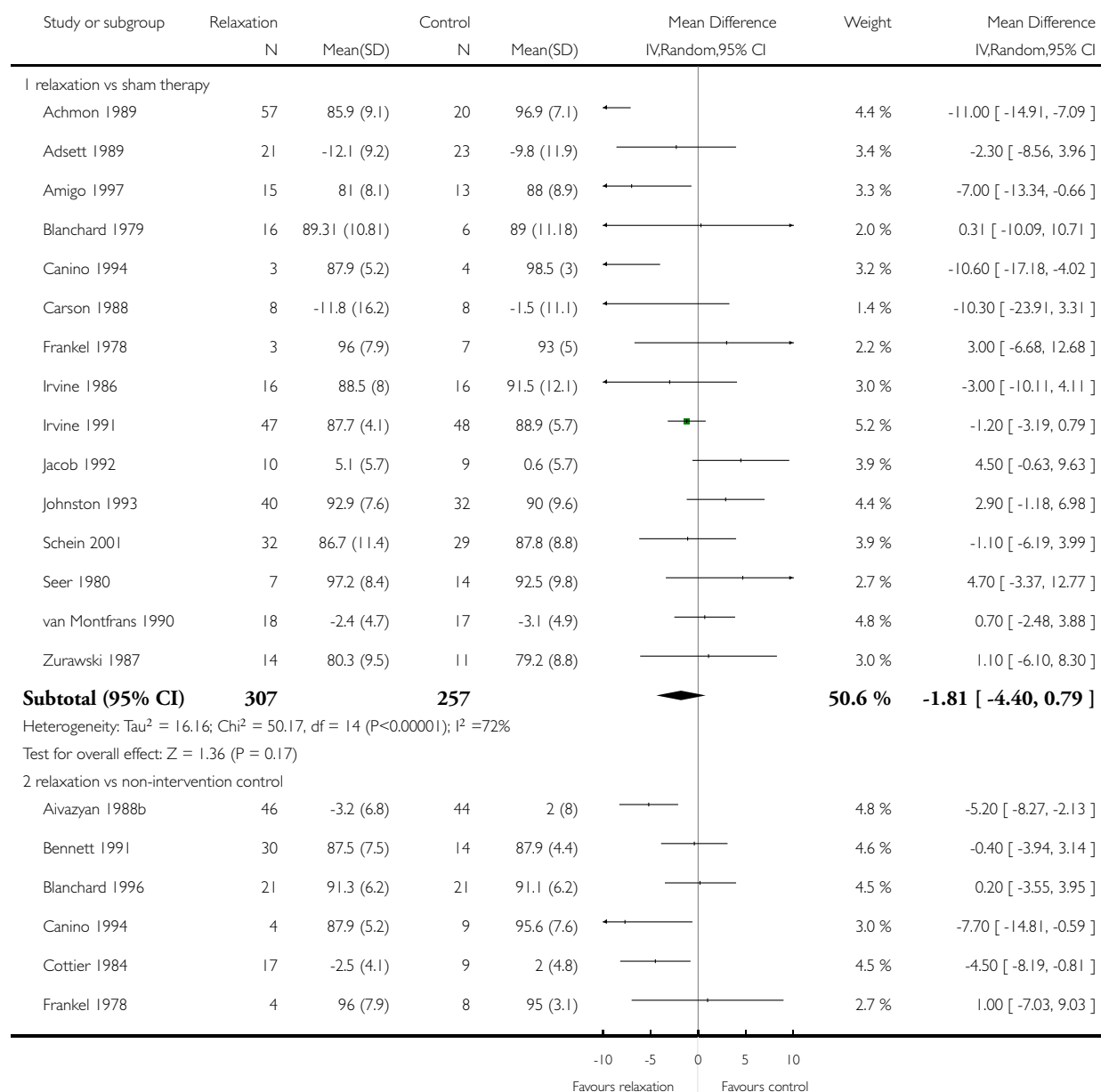


### Analysis 3.2. Comparison 3 Relaxation versus control (sub-grouped by type of control), Outcome 2 Diastolic BP.

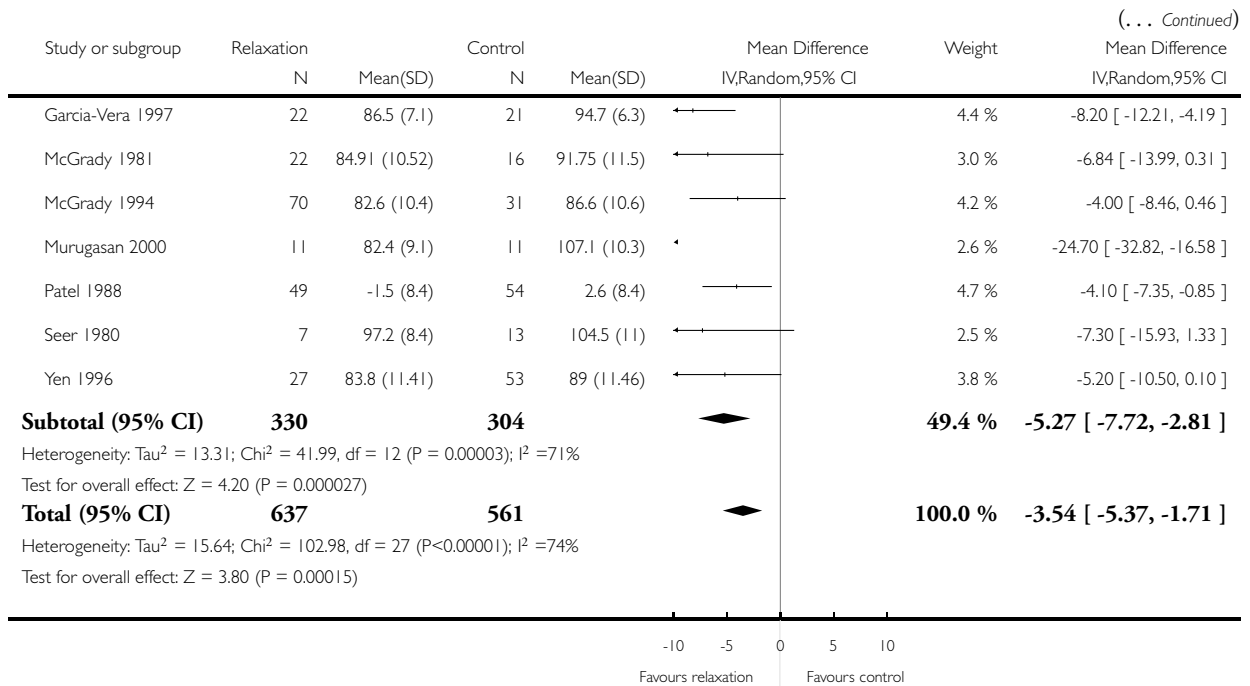
Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 3 Relaxation versus control (sub-grouped by type of control)

Outcome: 2 Diastolic BP



(Continued ...)

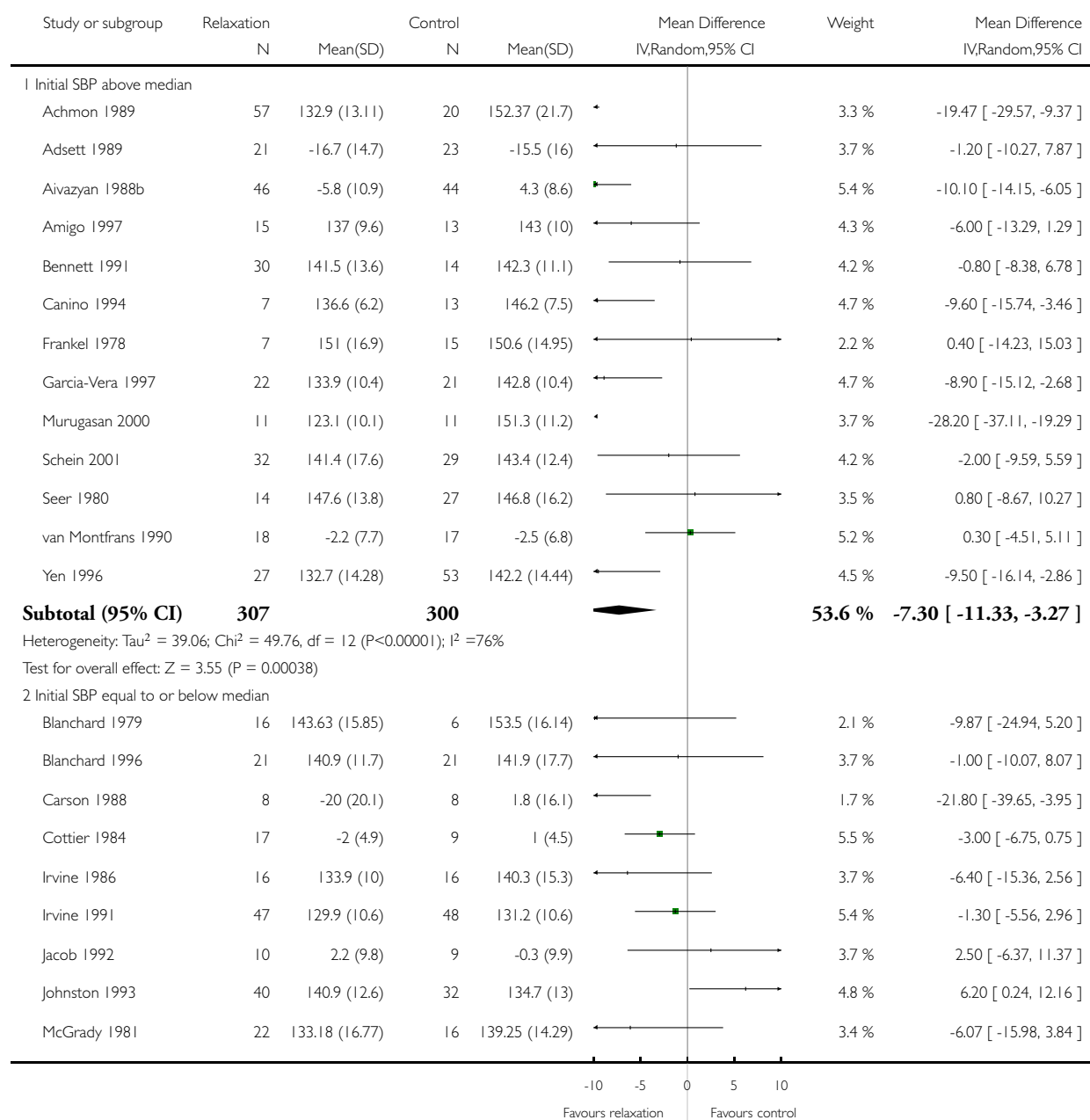


### Analysis 4.1. Comparison 4 Relaxation versus control (subgrouped by initial BP), Outcome 1 Systolic BP

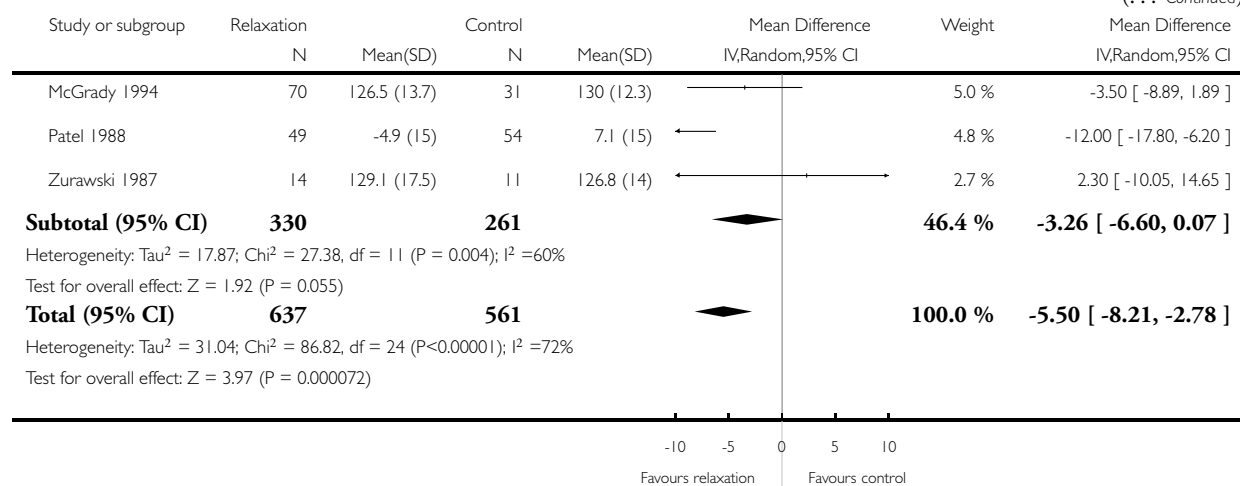
Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 4 Relaxation versus control (subgrouped by initial BP)

Outcome: 1 Systolic BP



(... Continued)

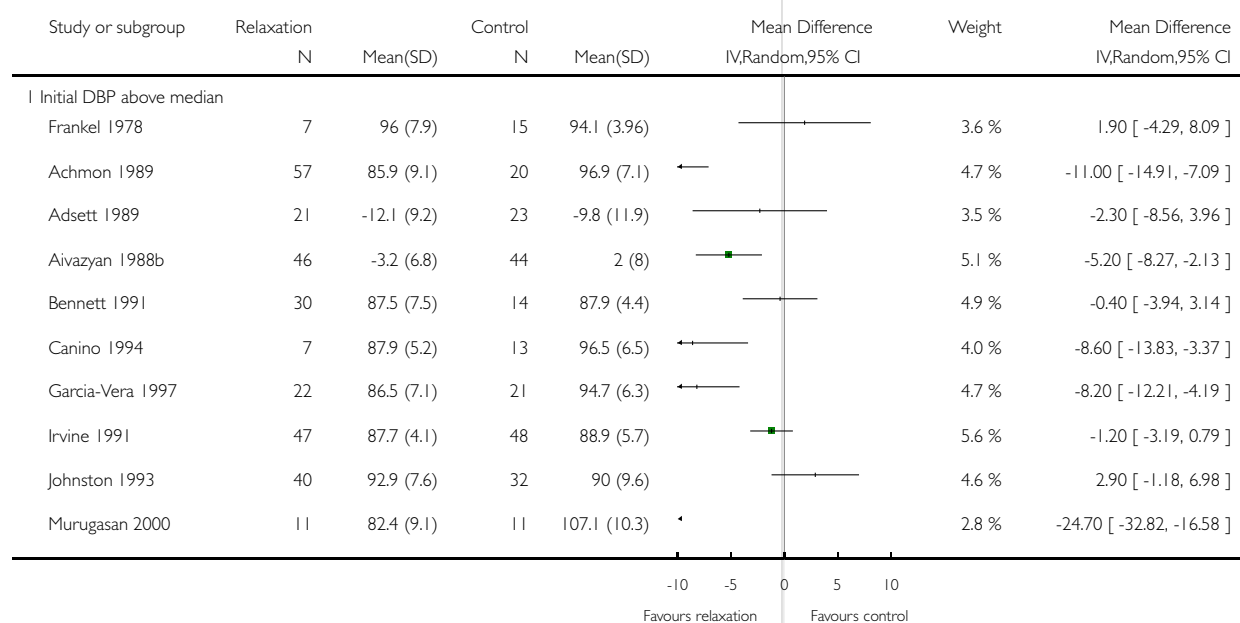


#### Analysis 4.2. Comparison 4 Relaxation versus control (subgrouped by initial BP), Outcome 2 Diastolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

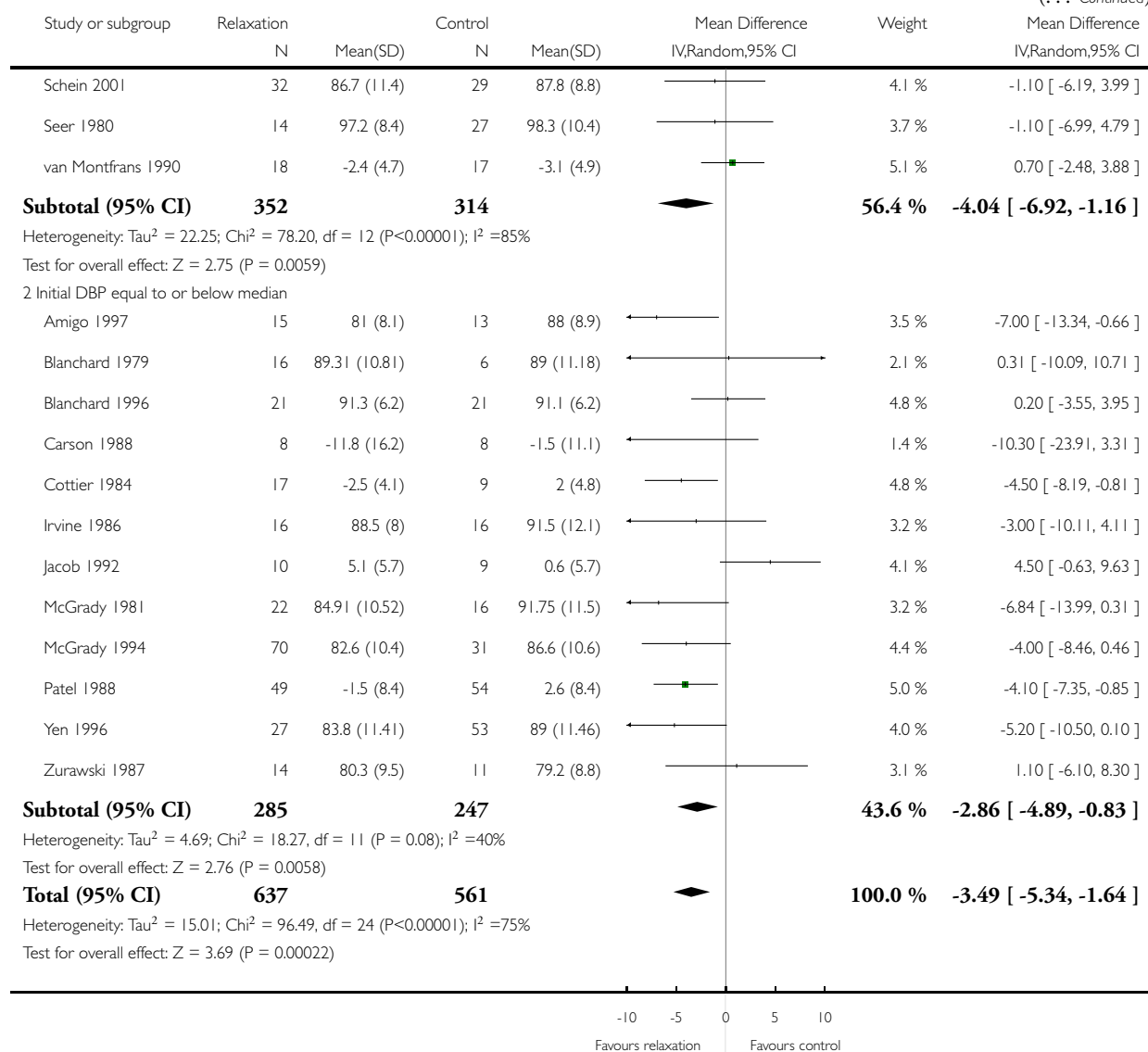
Comparison: 4 Relaxation versus control (subgrouped by initial BP)

Outcome: 2 Diastolic BP



(Continued ...)

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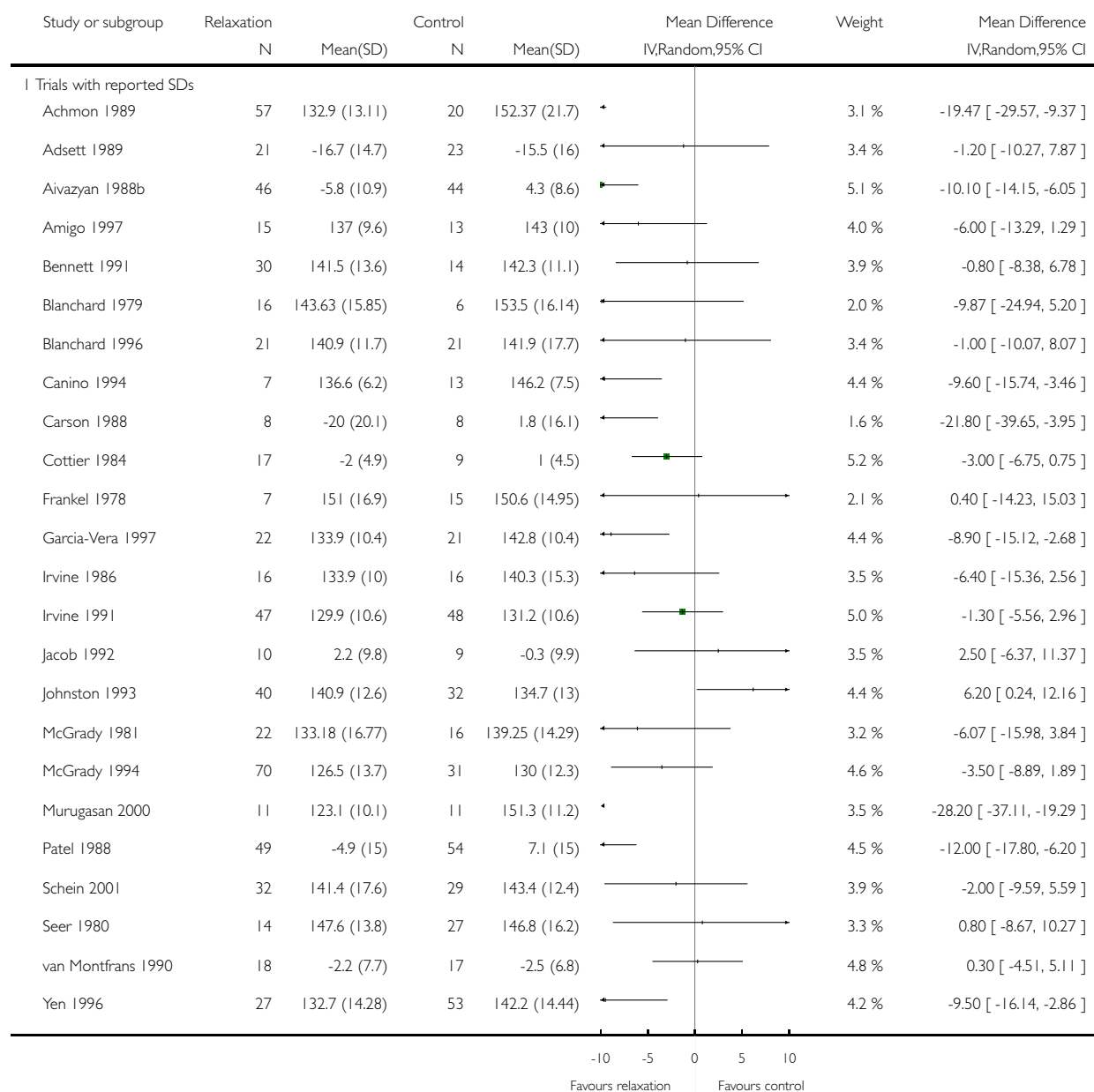


### Analysis 5.1. Comparison 5 Relaxation versus control (including trials with imputed SDs), Outcome 1 Systolic BP (high imputed SDs).

Review: Relaxation therapies for the management of primary hypertension in adults

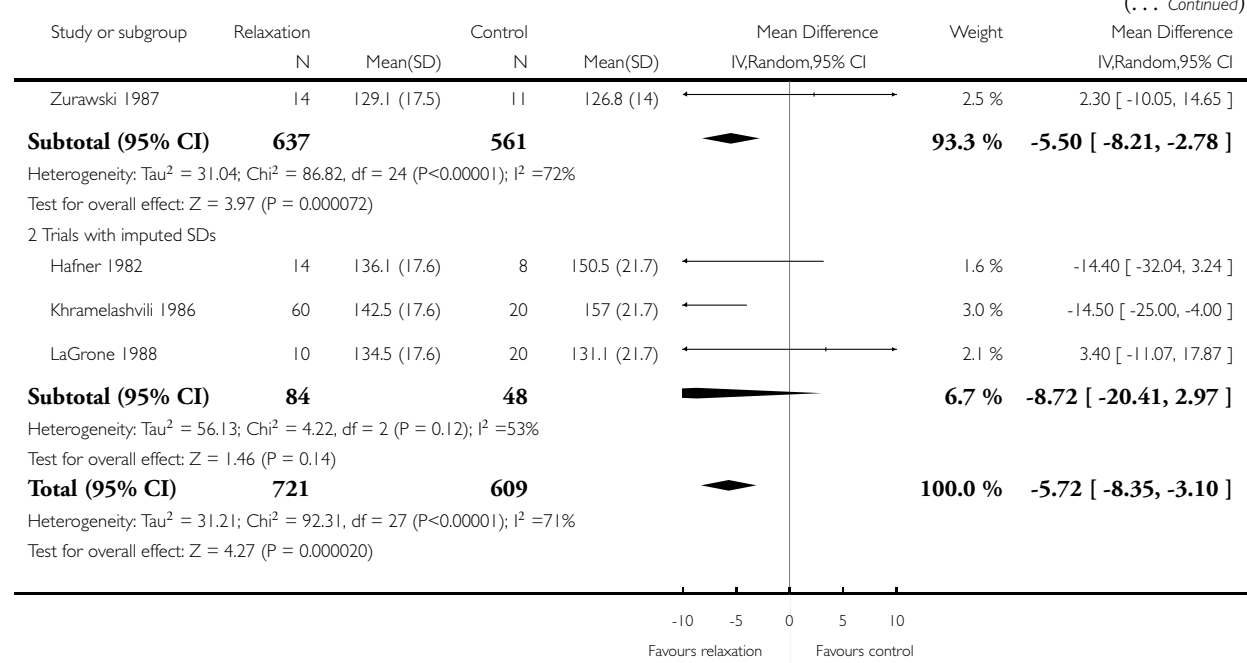
Comparison: 5 Relaxation versus control (including trials with imputed SDs)

Outcome: 1 Systolic BP (high imputed SDs)



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(... Continued)



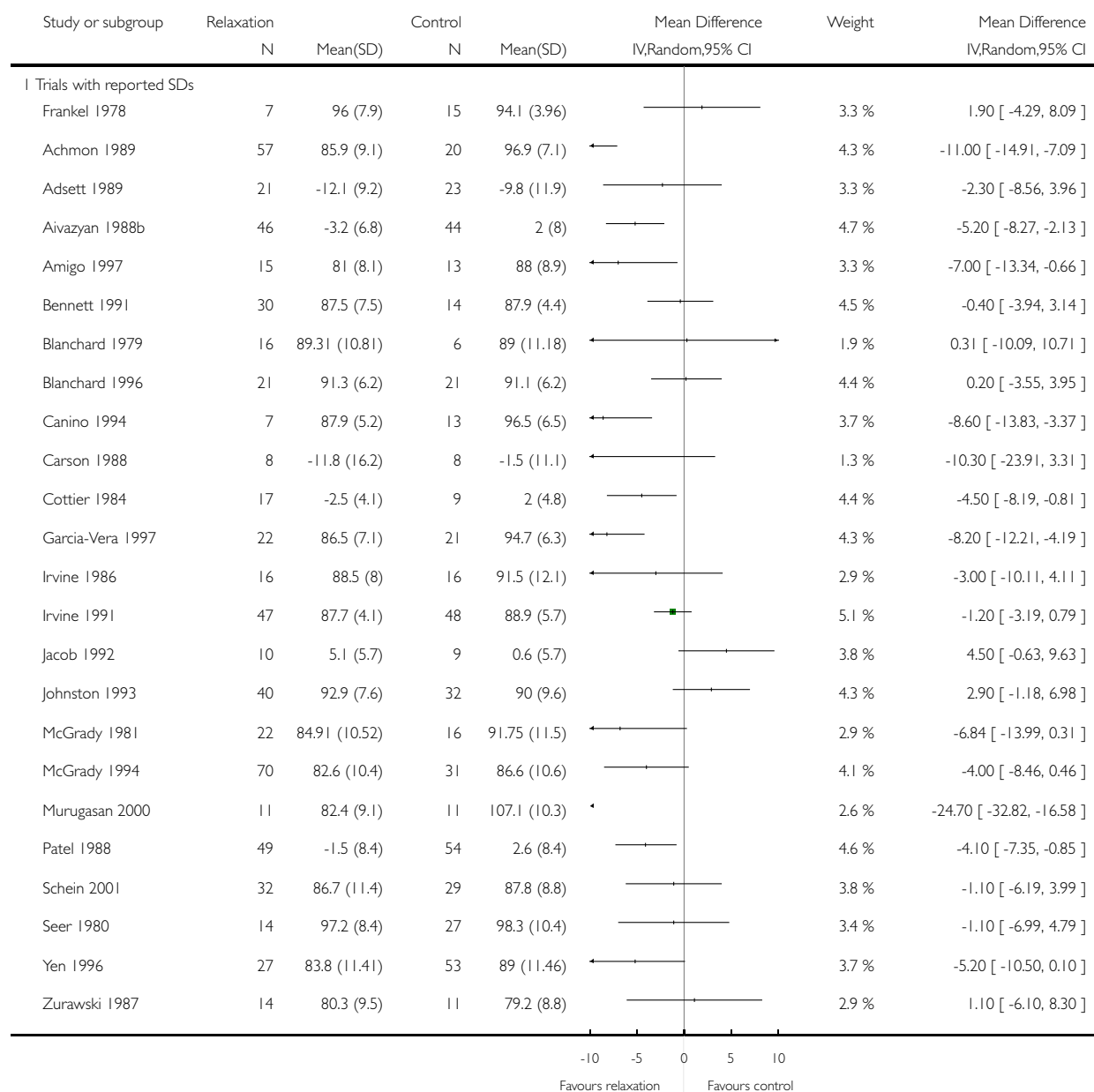


## Analysis 5.2. Comparison 5 Relaxation versus control (including trials with imputed SDs), Outcome 2 Diastolic BP (high imputed SDs).

Review: Relaxation therapies for the management of primary hypertension in adults

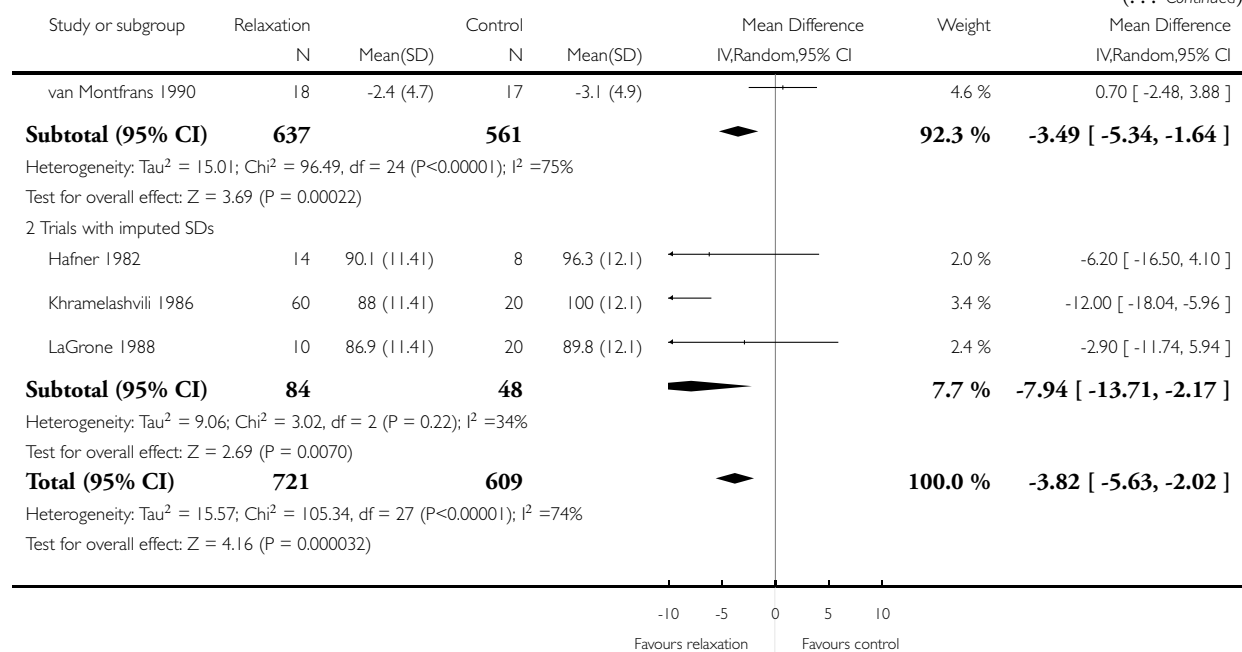
Comparison: 5 Relaxation versus control (including trials with imputed SDs)

Outcome: 2 Diastolic BP (high imputed SDs)



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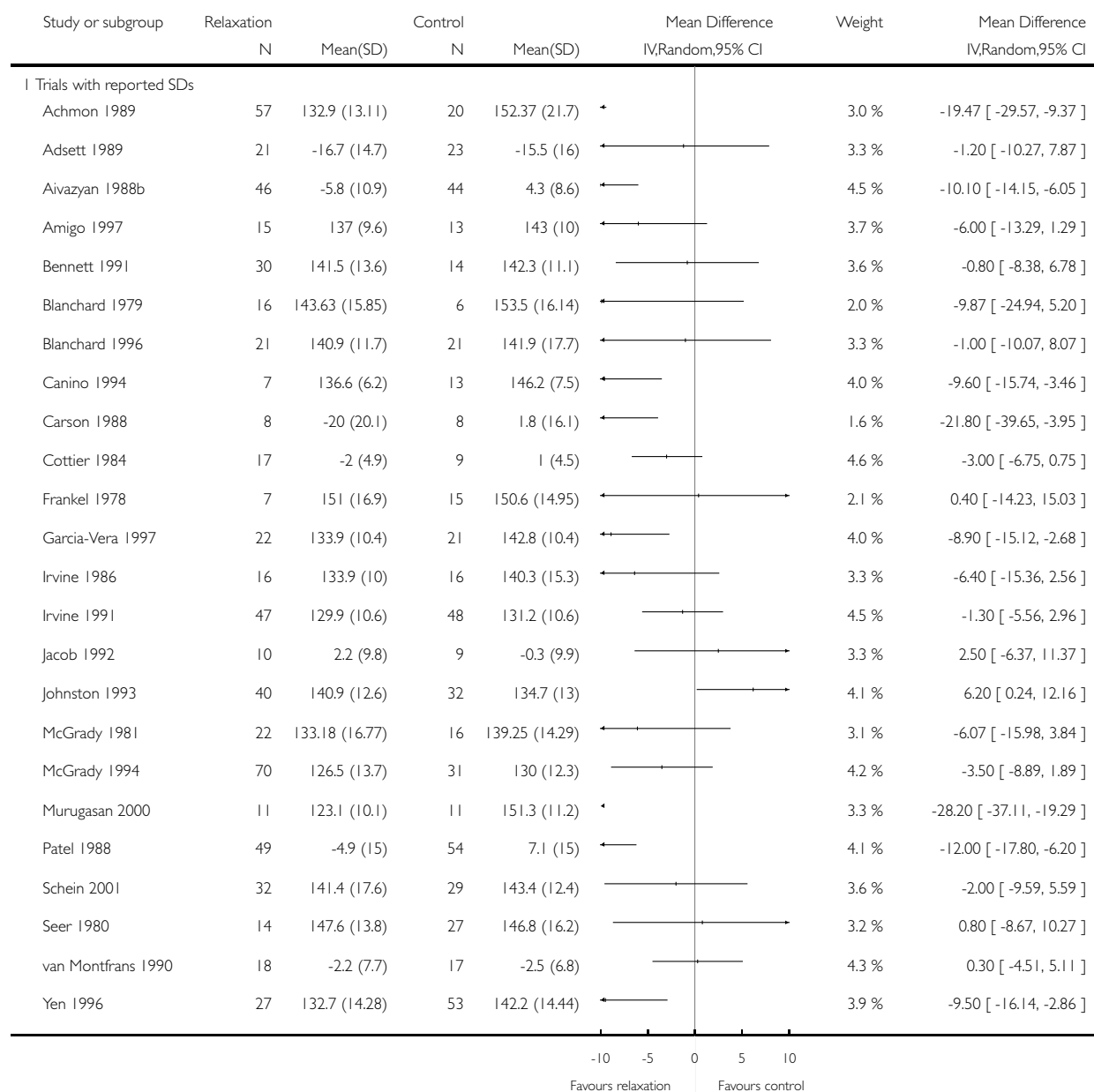


### Analysis 5.3. Comparison 5 Relaxation versus control (including trials with imputed SDs), Outcome 3 Systolic BP (low imputed SDs).

Review: Relaxation therapies for the management of primary hypertension in adults

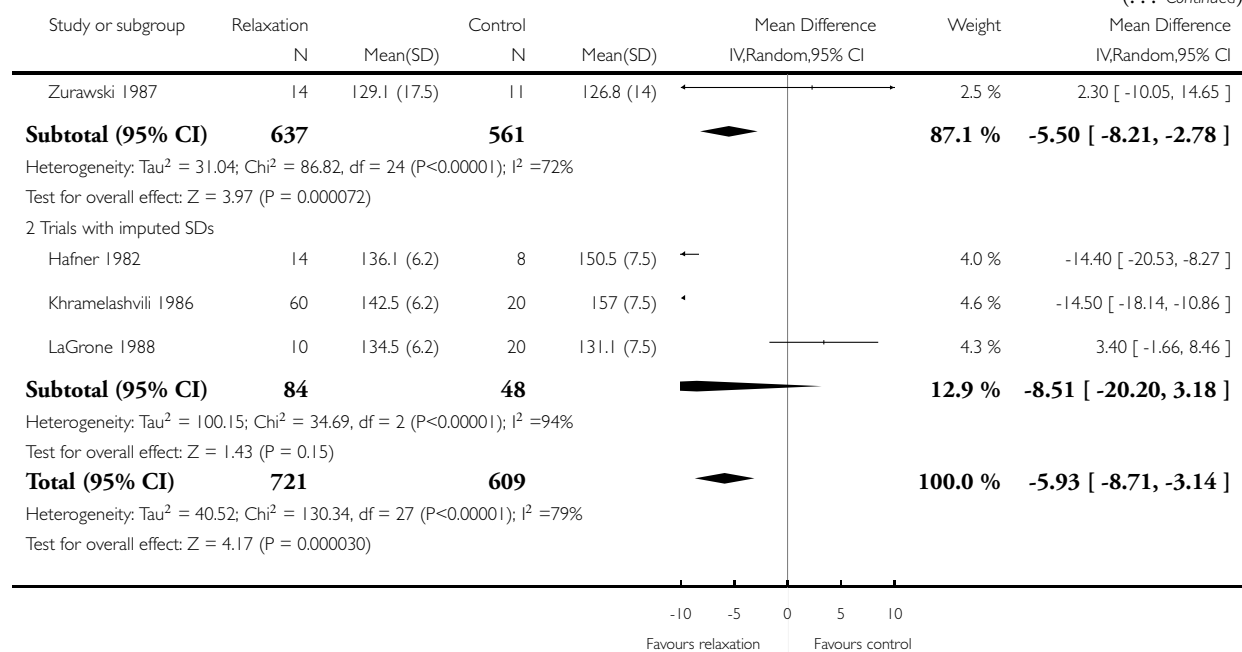
Comparison: 5 Relaxation versus control (including trials with imputed SDs)

Outcome: 3 Systolic BP (low imputed SDs)



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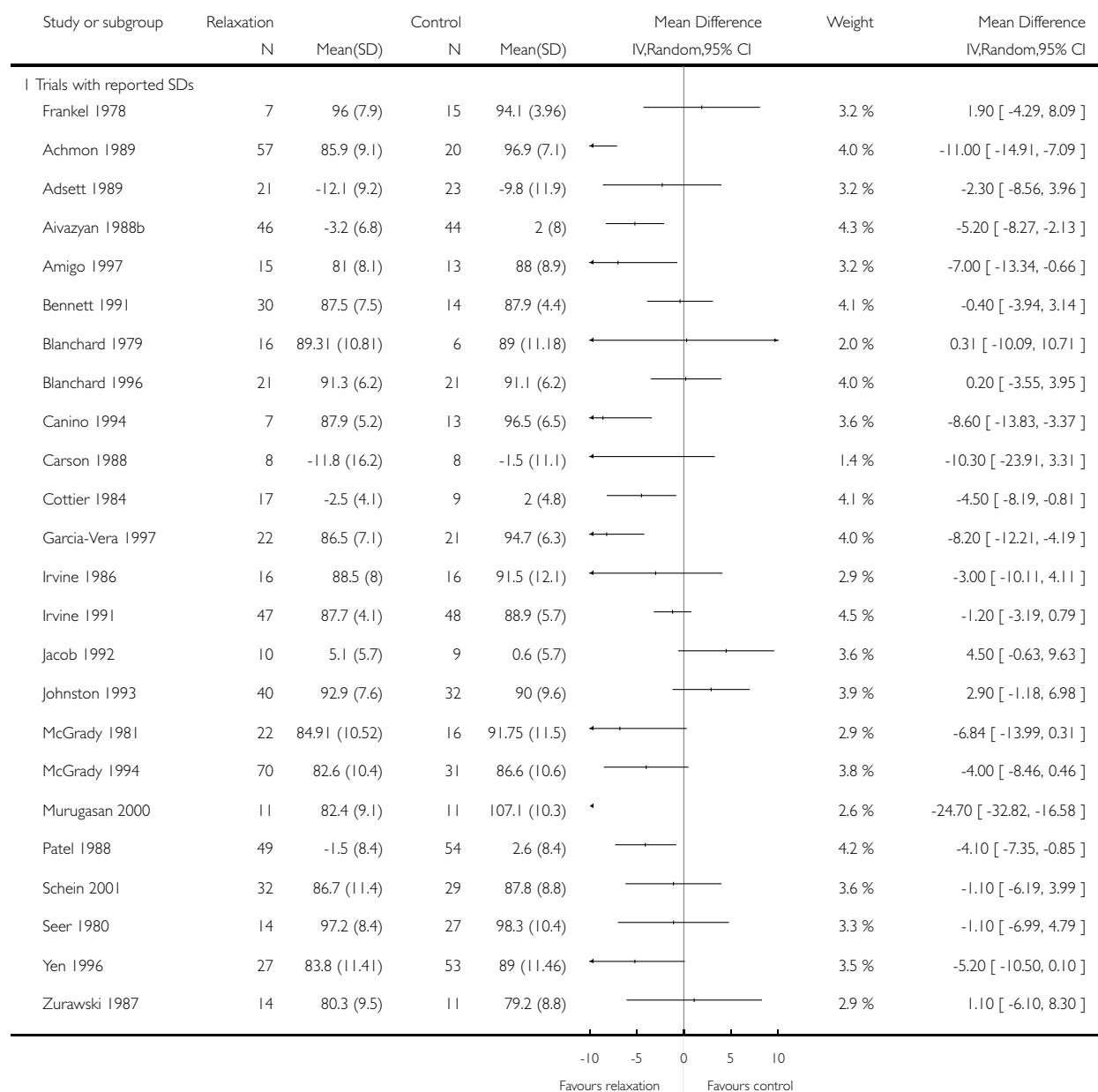


### Analysis 5.4. Comparison 5 Relaxation versus control (including trials with imputed SDs), Outcome 4 Diastolic BP (low imputed SDs).

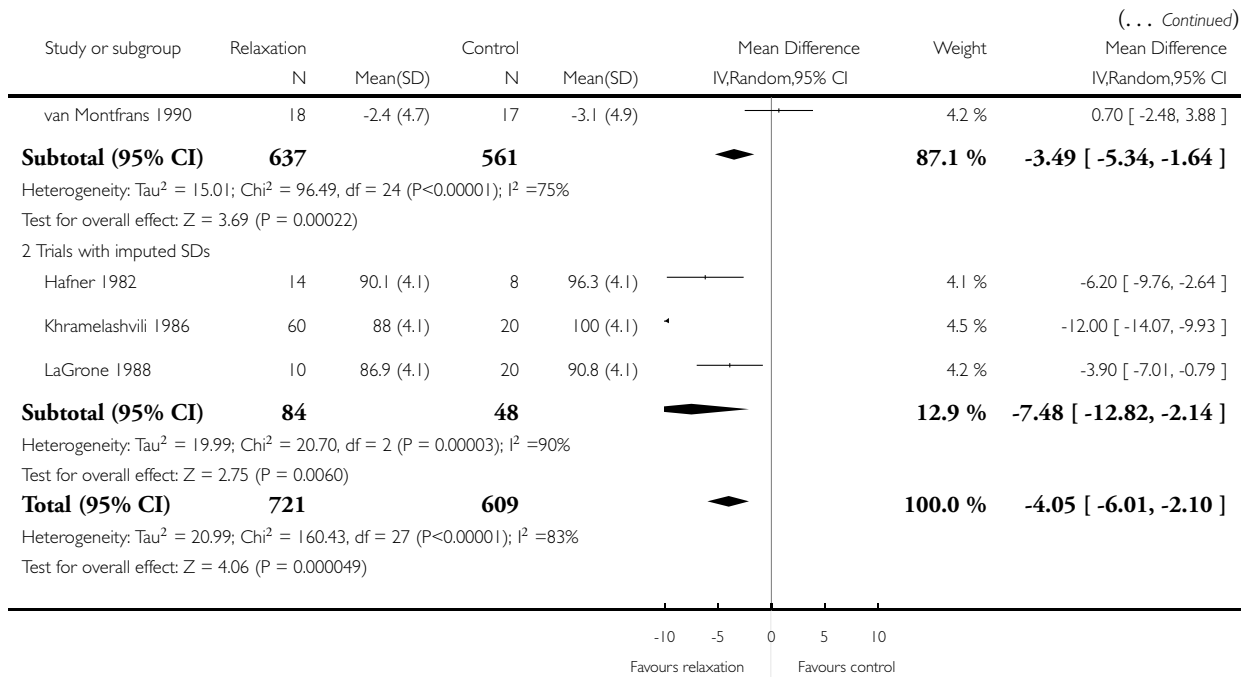
Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 5 Relaxation versus control (including trials with imputed SDs)

Outcome: 4 Diastolic BP (low imputed SDs)



(Continued ...)

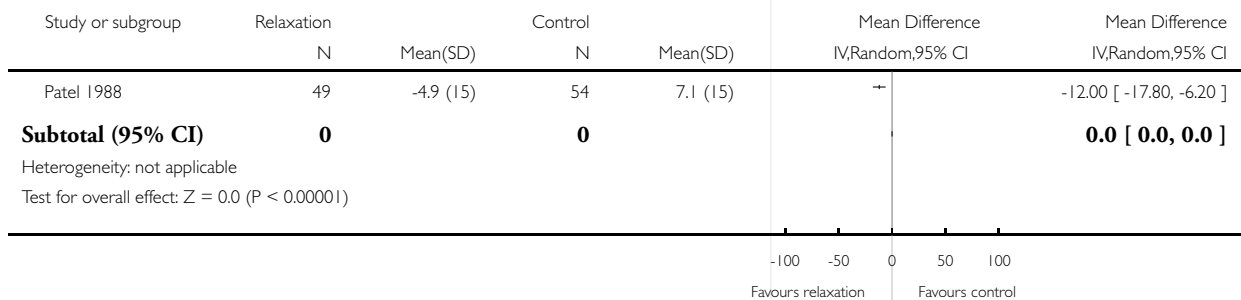


**Analysis 6.1. Comparison 6 Relaxation versus control including only trials reporting adequate concealment of allocation, Outcome 1 Systolic BP.**

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 6 Relaxation versus control including only trials reporting adequate concealment of allocation

Outcome: 1 Systolic BP

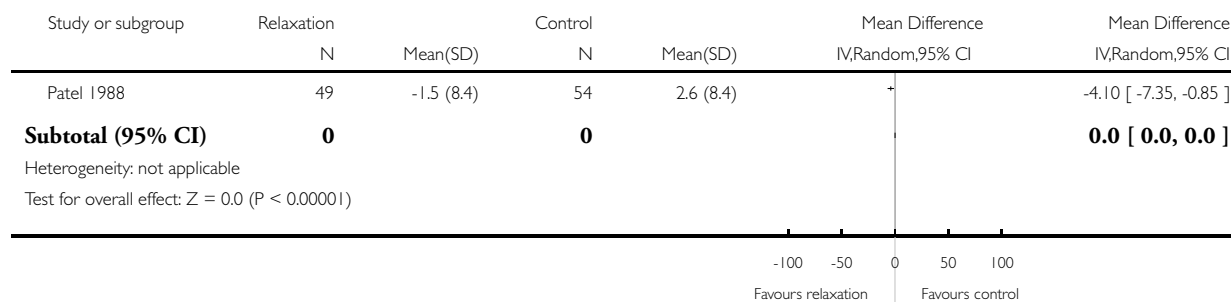


### Analysis 6.2. Comparison 6 Relaxation versus control including only trials reporting adequate concealment of allocation, Outcome 2 Diastolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 6 Relaxation versus control including only trials reporting adequate concealment of allocation

Outcome: 2 Diastolic BP

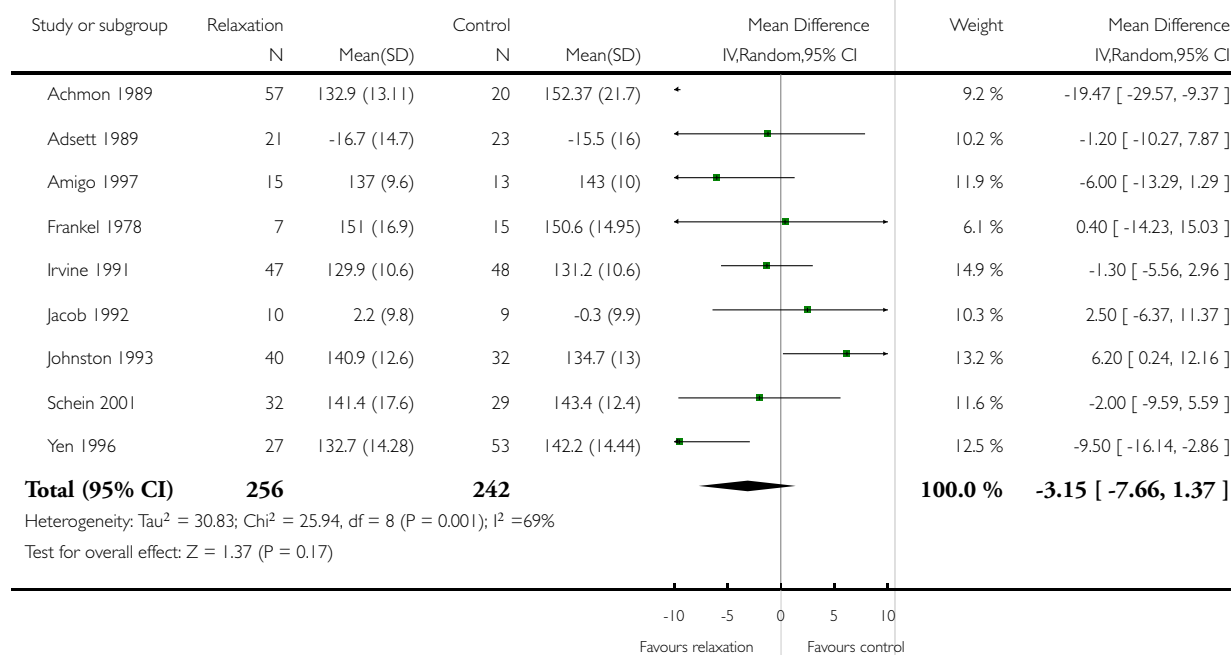


### Analysis 7.1. Comparison 7 Relaxation versus control including only trials reporting blinded outcome assessment, Outcome 1 Systolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 7 Relaxation versus control including only trials reporting blinded outcome assessment

Outcome: 1 Systolic BP

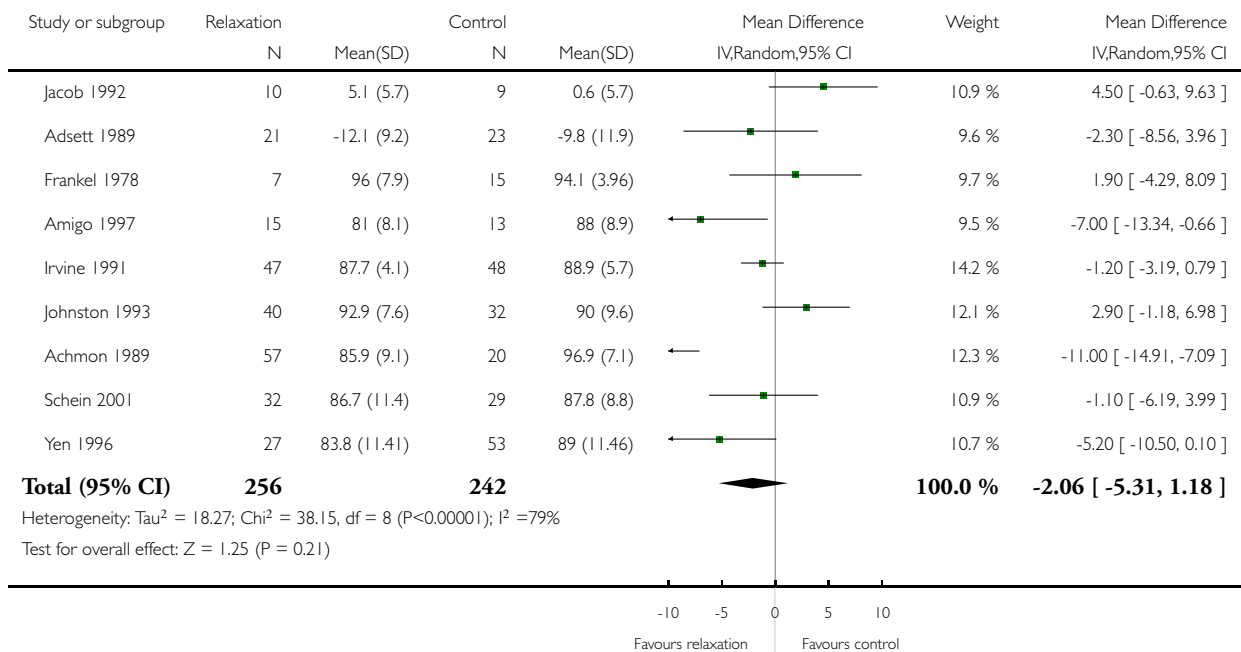


### Analysis 7.2. Comparison 7 Relaxation versus control including only trials reporting blinded outcome assessment, Outcome 2 Diastolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 7 Relaxation versus control including only trials reporting blinded outcome assessment

Outcome: 2 Diastolic BP



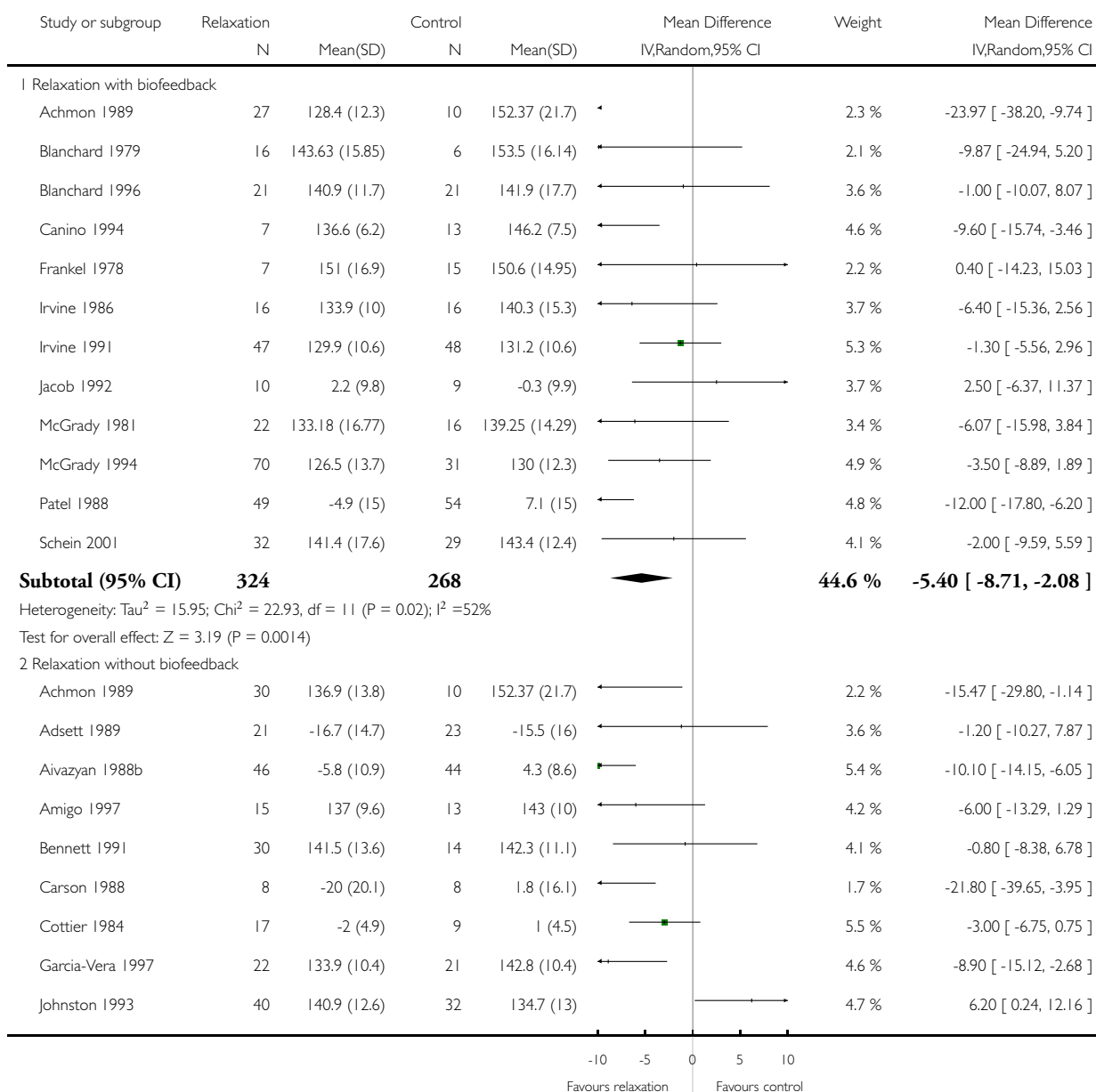


### Analysis 8.1. Comparison 8 Relaxation versus control (sub-grouped by with/without biofeedback), Outcome I Systolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

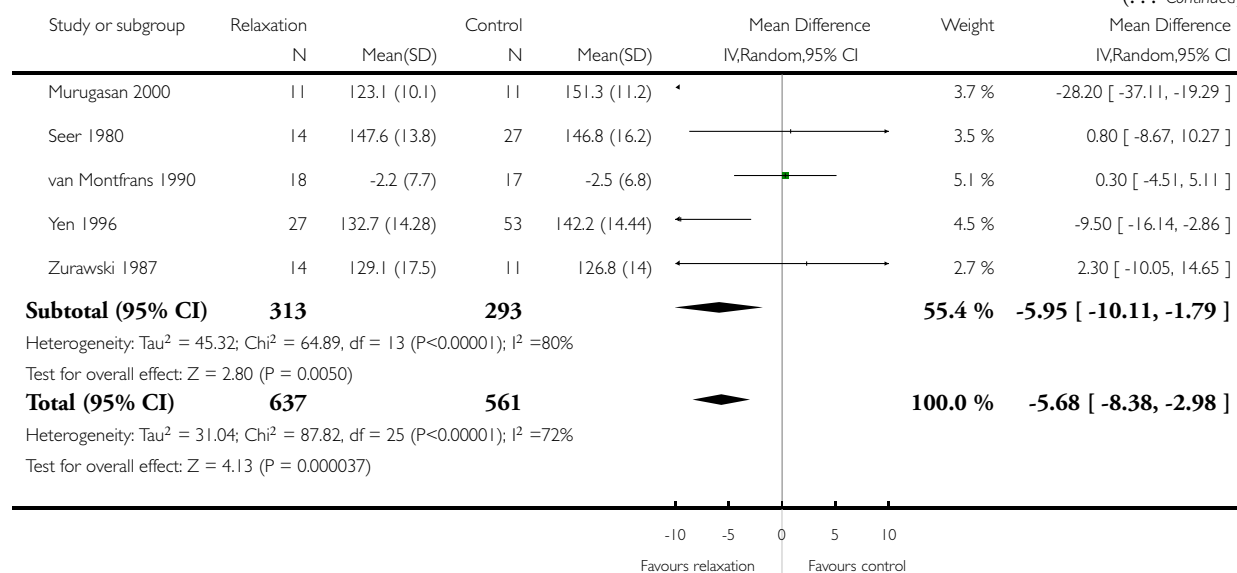
Comparison: 8 Relaxation versus control (sub-grouped by with/without biofeedback)

Outcome: I Systolic BP



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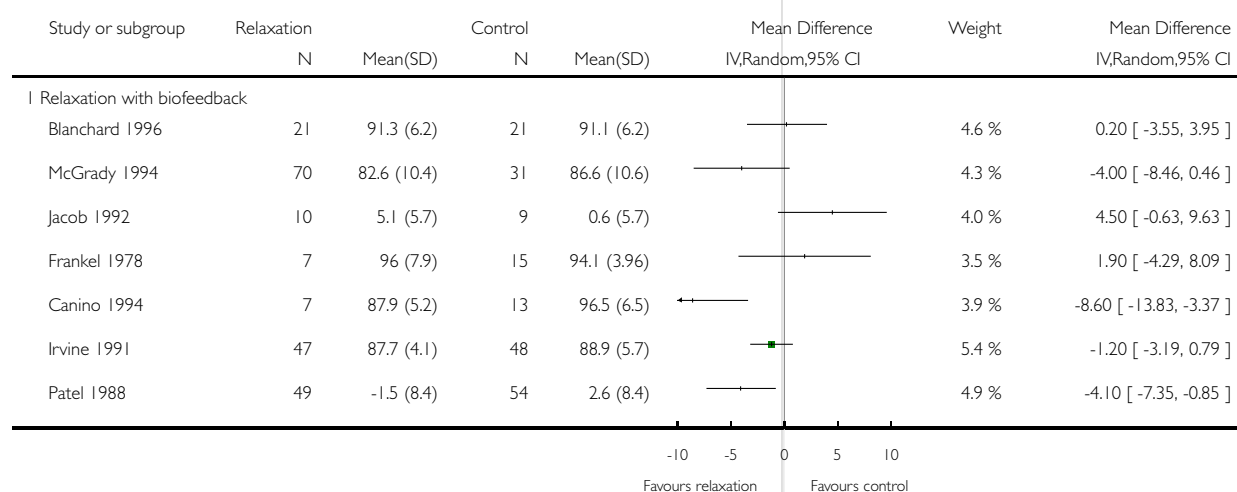


### Analysis 8.2. Comparison 8 Relaxation versus control (sub-grouped by with/without biofeedback), Outcome 2 Diastolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

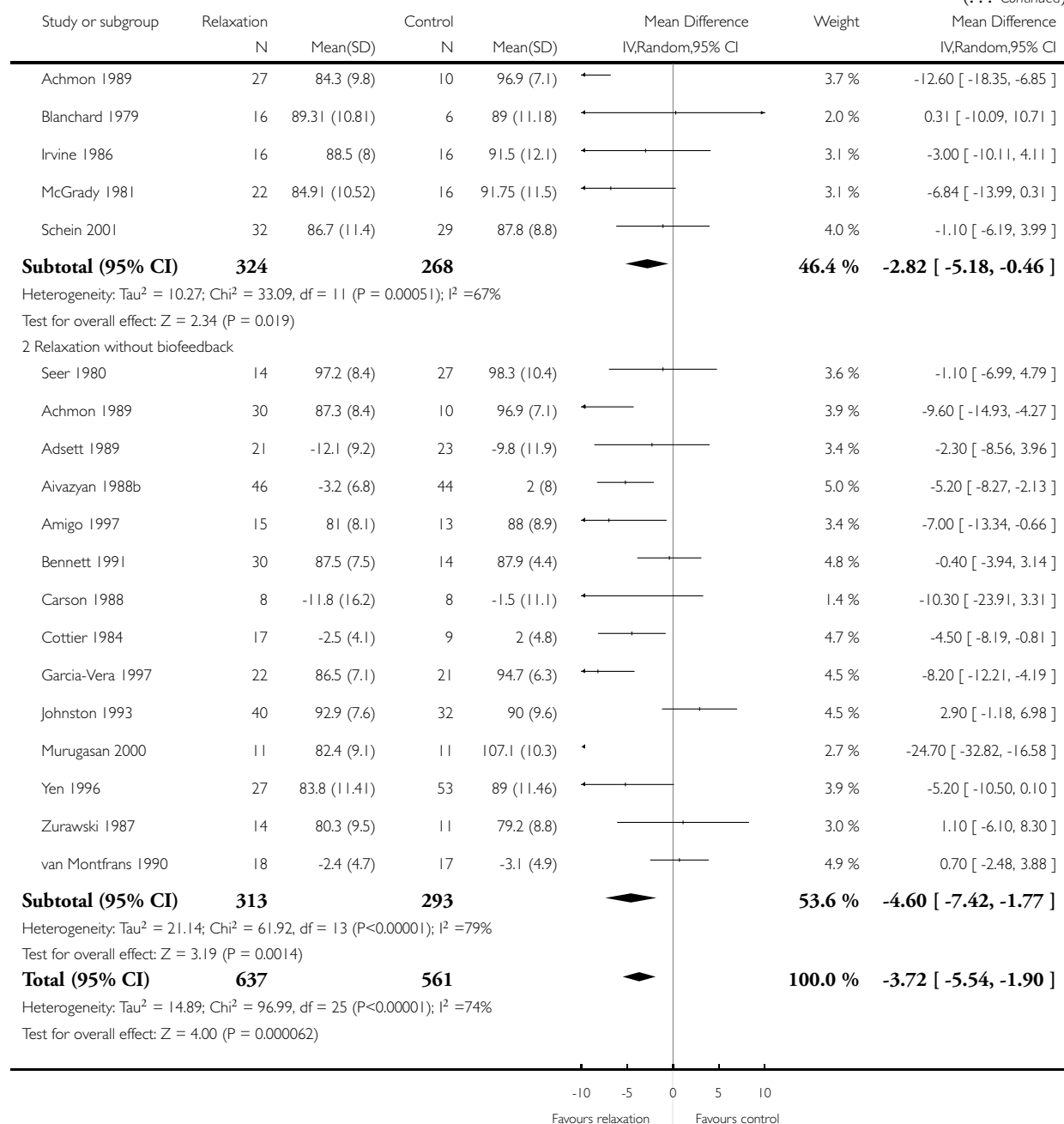
Comparison: 8 Relaxation versus control (sub-grouped by with/without biofeedback)

Outcome: 2 Diastolic BP



(Continued ...)

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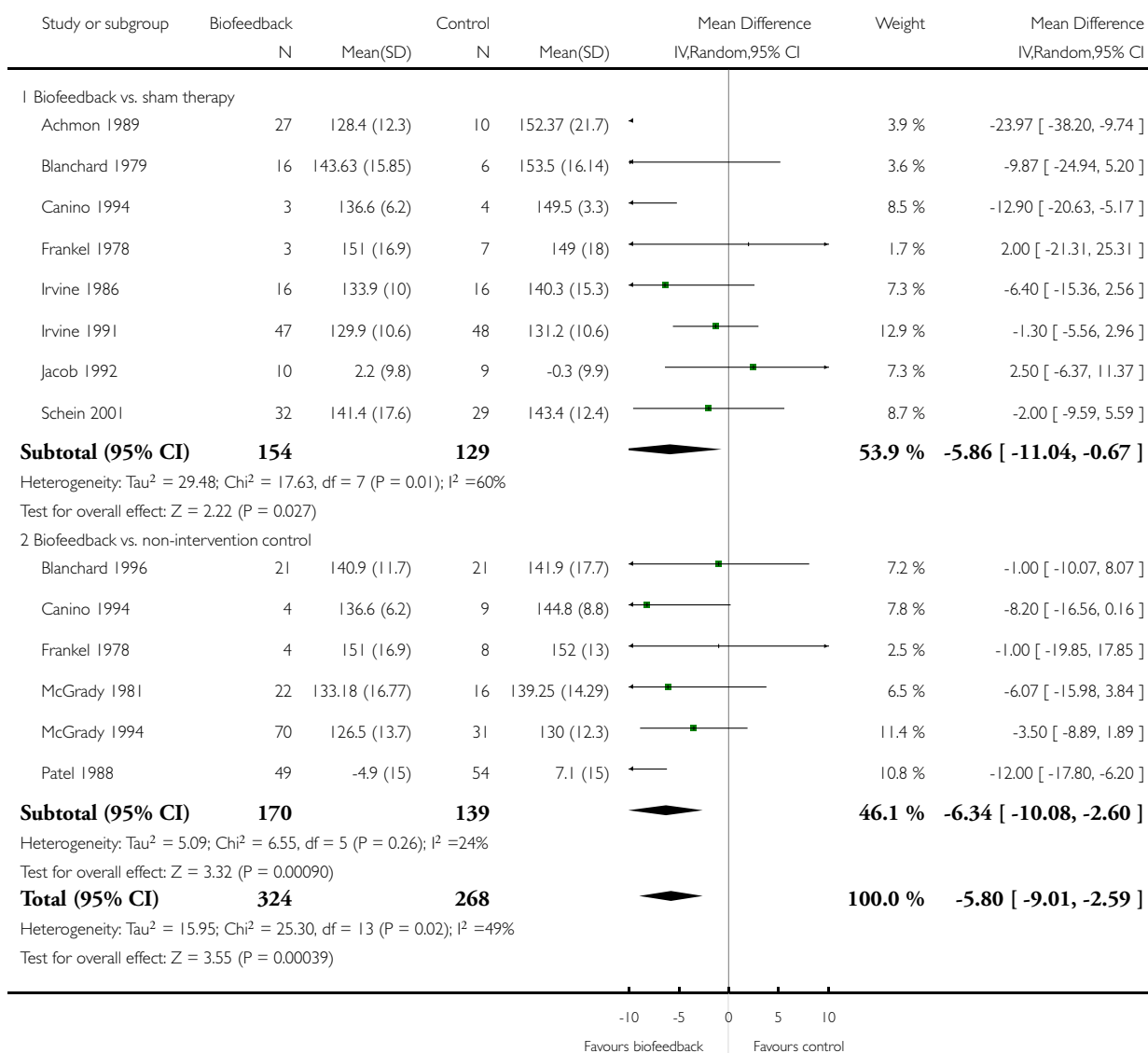


### Analysis 9.1. Comparison 9 Relaxation with biofeedback versus control (subgrouped by type of control), Outcome 1 Systolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 9 Relaxation with biofeedback versus control (subgrouped by type of control)

Outcome: 1 Systolic BP

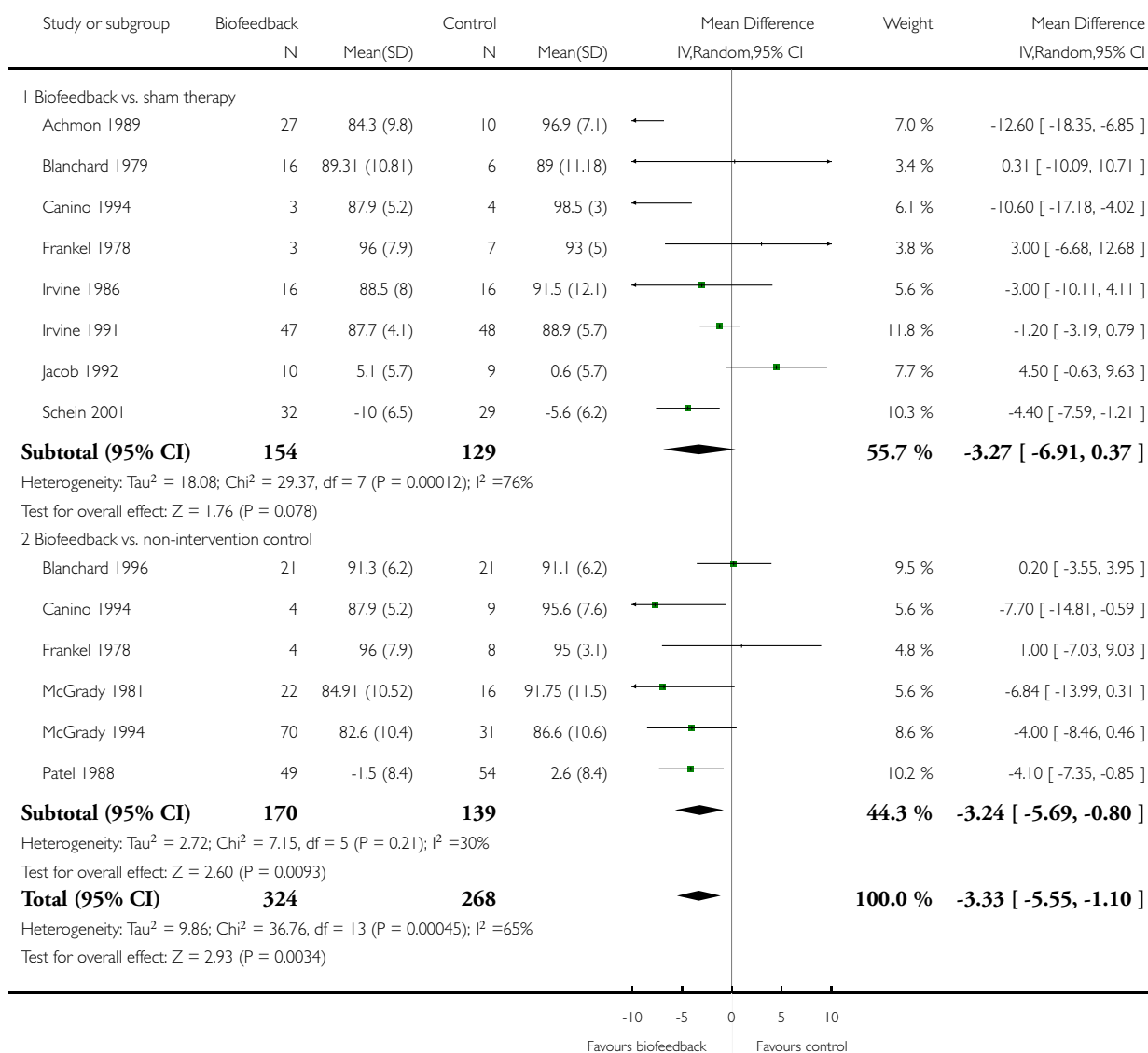


## Analysis 9.2. Comparison 9 Relaxation with biofeedback versus control (subgrouped by type of control), Outcome 2 Diastolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 9 Relaxation with biofeedback versus control (subgrouped by type of control)

Outcome: 2 Diastolic BP

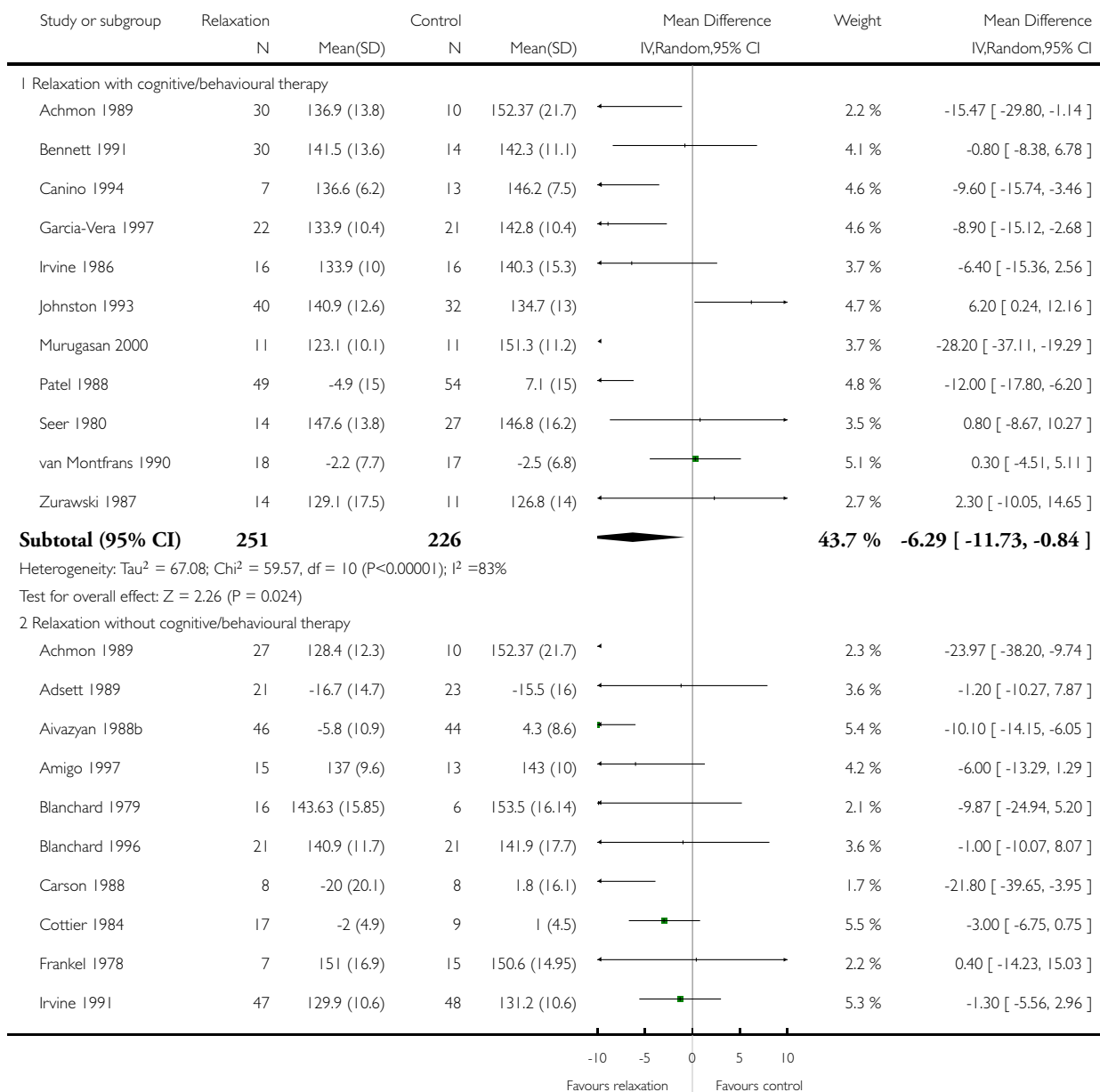


### Analysis 10.1. Comparison 10 Relaxation versus control (sub-grouped by with/without cognitive/behavioural therapy), Outcome 1 Systolic BP.

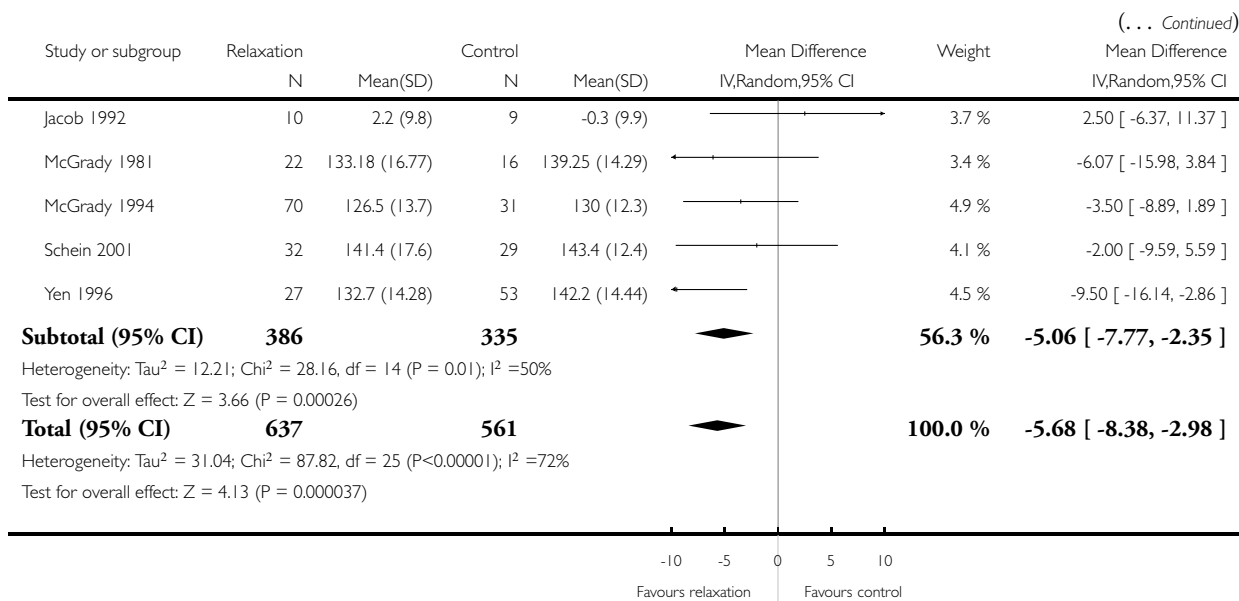
Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 10 Relaxation versus control (sub-grouped by with/without cognitive/behavioural therapy)

Outcome: 1 Systolic BP



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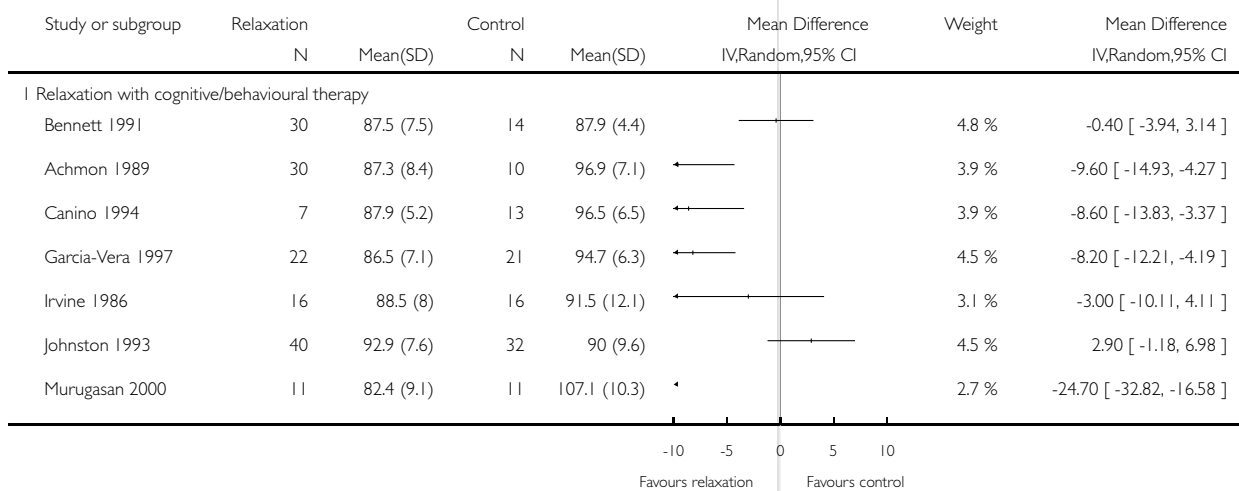


### Analysis 10.2. Comparison 10 Relaxation versus control (sub-grouped by with/without cognitive/behavioural therapy), Outcome 2 Diastolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

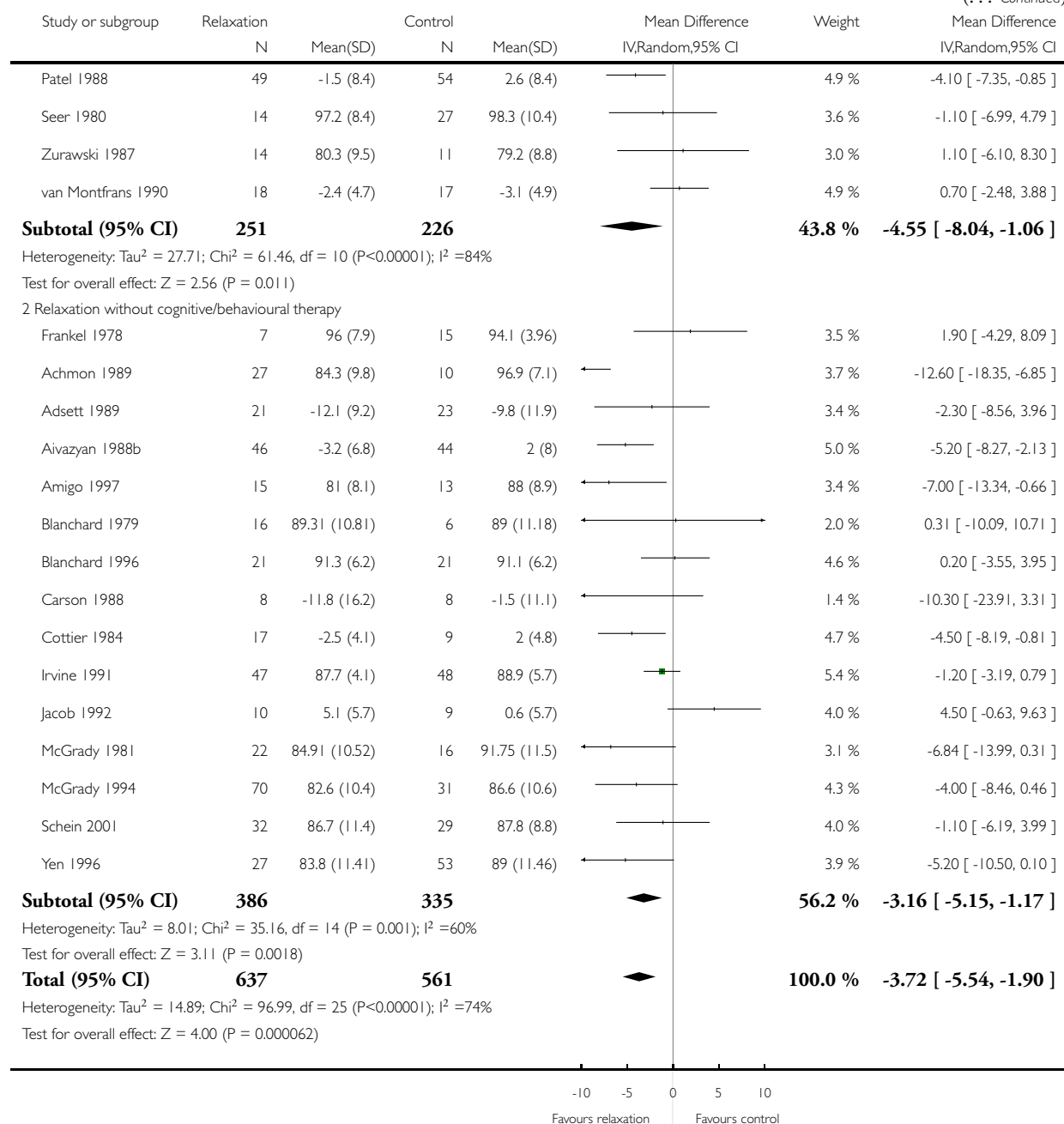
Comparison: 10 Relaxation versus control (sub-grouped by with/without cognitive/behavioural therapy)

Outcome: 2 Diastolic BP



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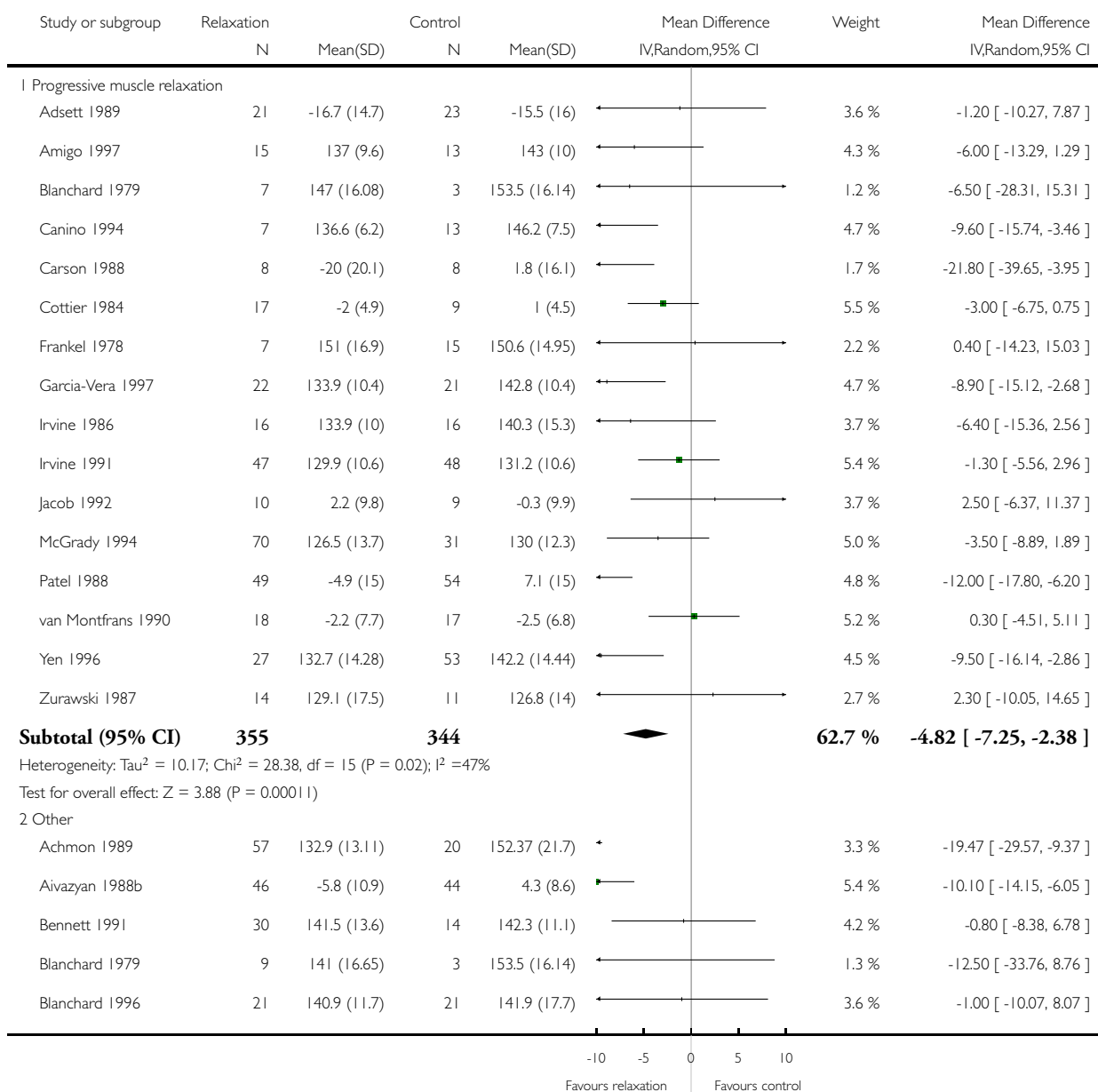


### Analysis 11.1. Comparison 11 Relaxation versus control (sub-grouped by progressive muscle relaxation/other), Outcome 1 Systolic BP.

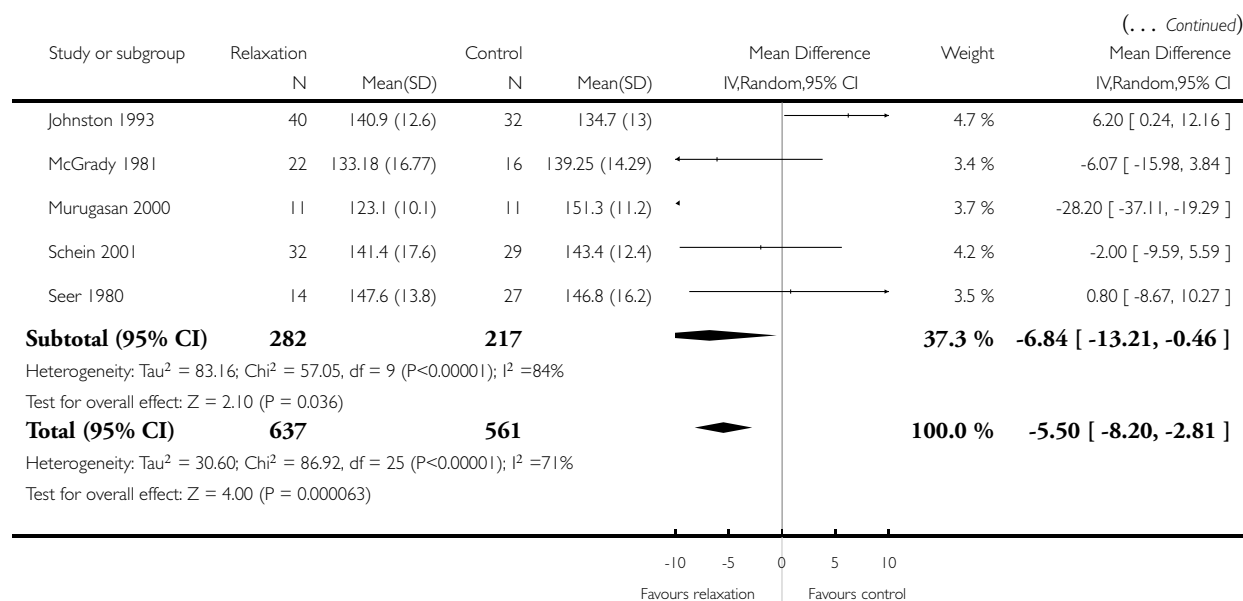
Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 11 Relaxation versus control (sub-grouped by progressive muscle relaxation/other)

Outcome: 1 Systolic BP



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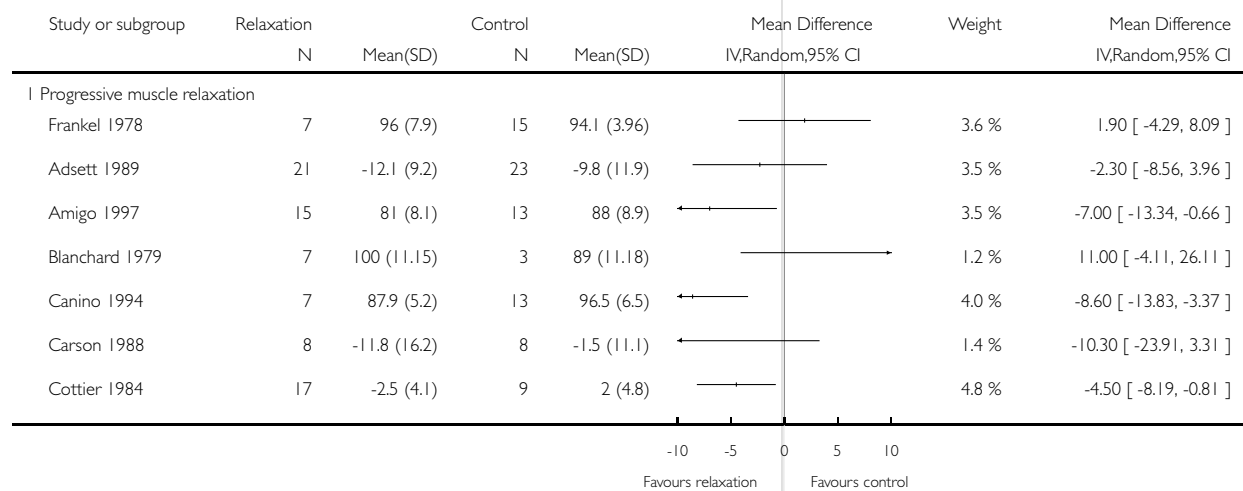


### Analysis 11.2. Comparison 11 Relaxation versus control (sub-grouped by progressive muscle relaxation/other), Outcome 2 Diastolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

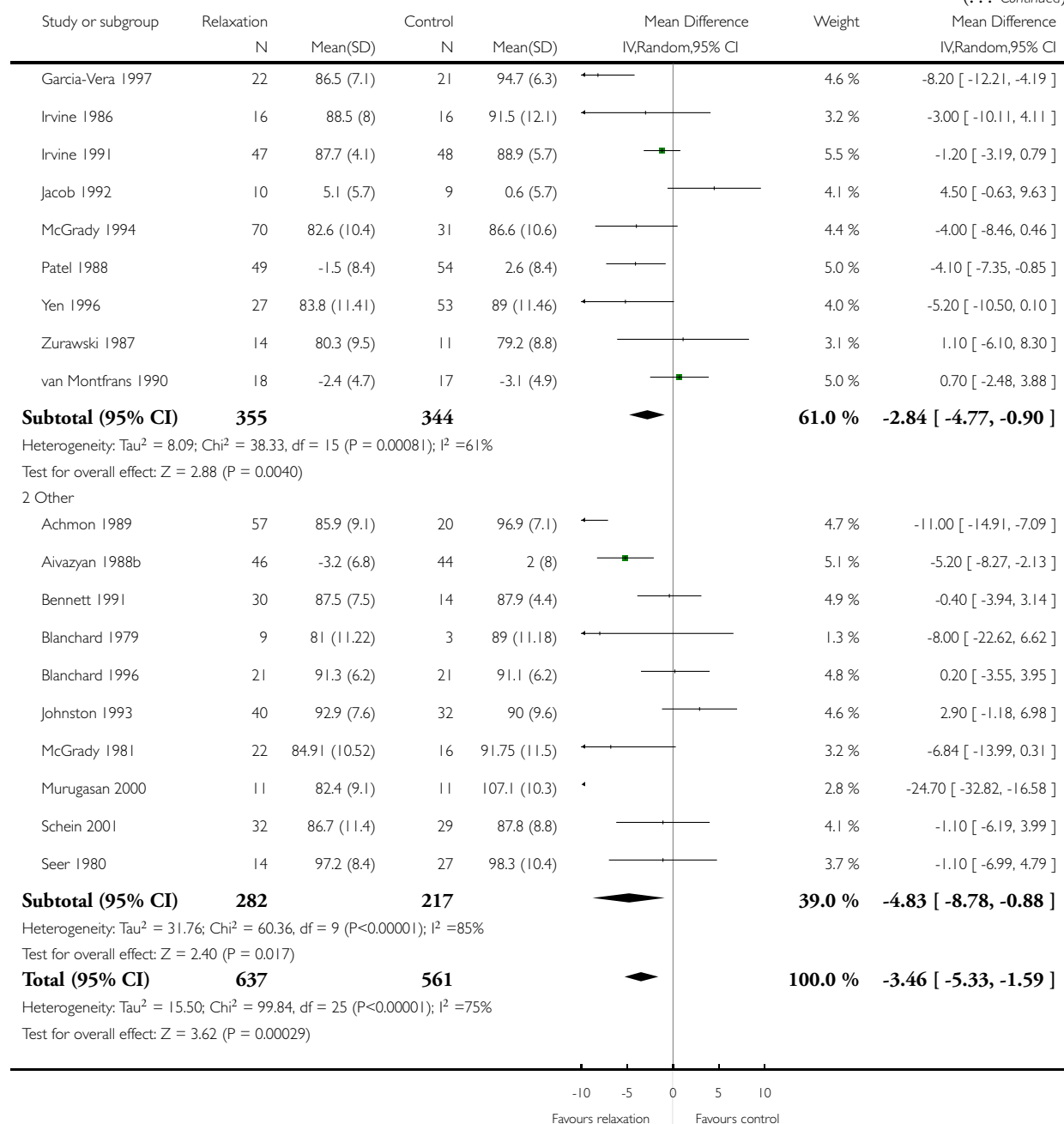
Comparison: 11 Relaxation versus control (sub-grouped by progressive muscle relaxation/other)

Outcome: 2 Diastolic BP



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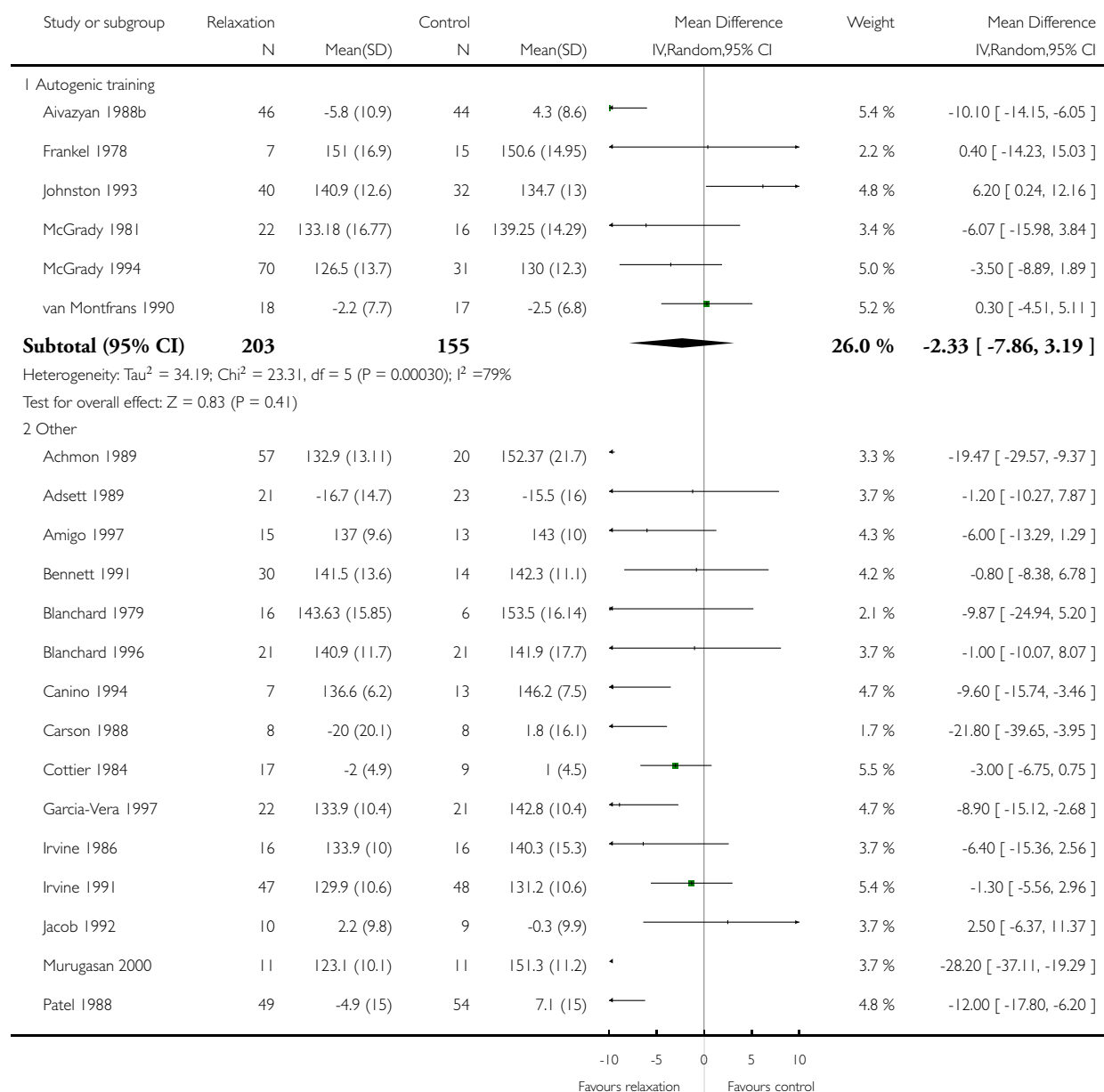


### Analysis 12.1. Comparison 12 Relaxation versus control (sub-grouped by autogenic training/other), Outcome 1 Systolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

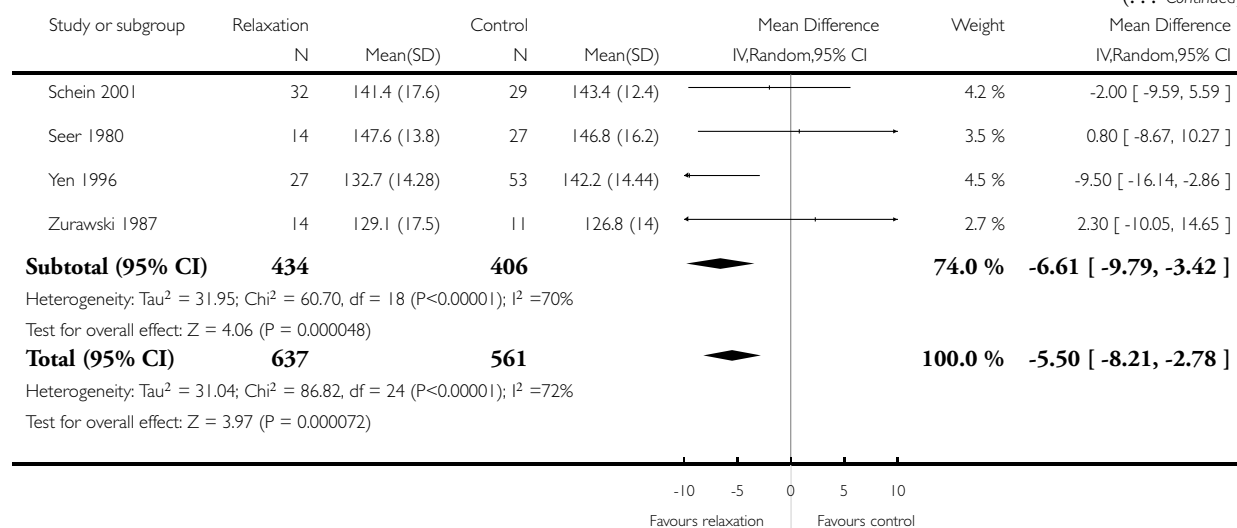
Comparison: 12 Relaxation versus control (sub-grouped by autogenic training/other)

Outcome: 1 Systolic BP



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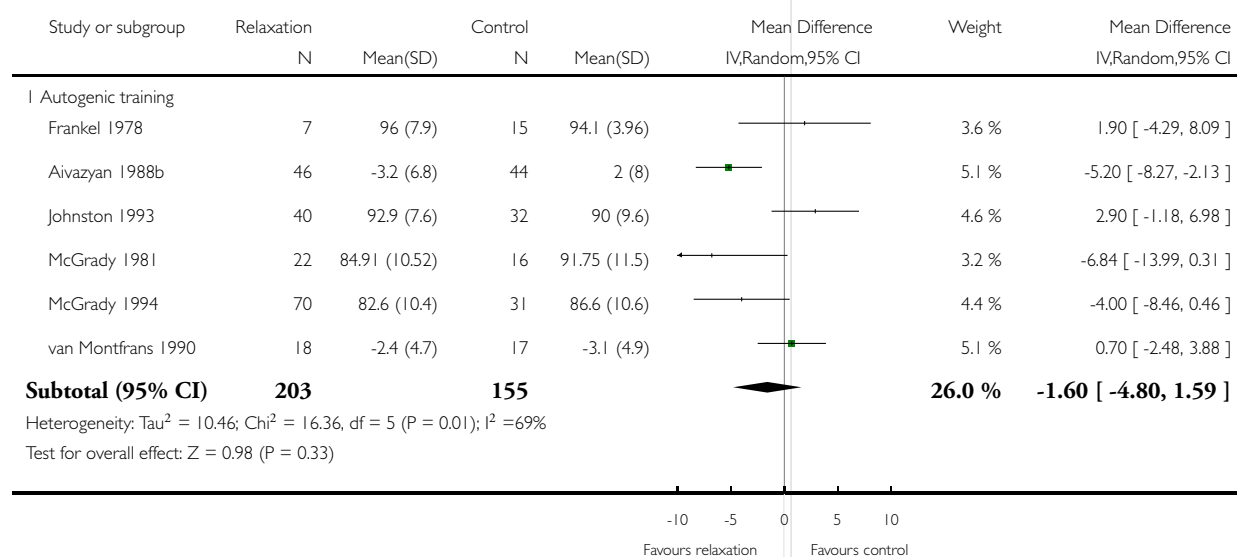


### Analysis 12.2. Comparison 12 Relaxation versus control (sub-grouped by autogenic training/other), Outcome 2 Diastolic BP.

Review: Relaxation therapies for the management of primary hypertension in adults

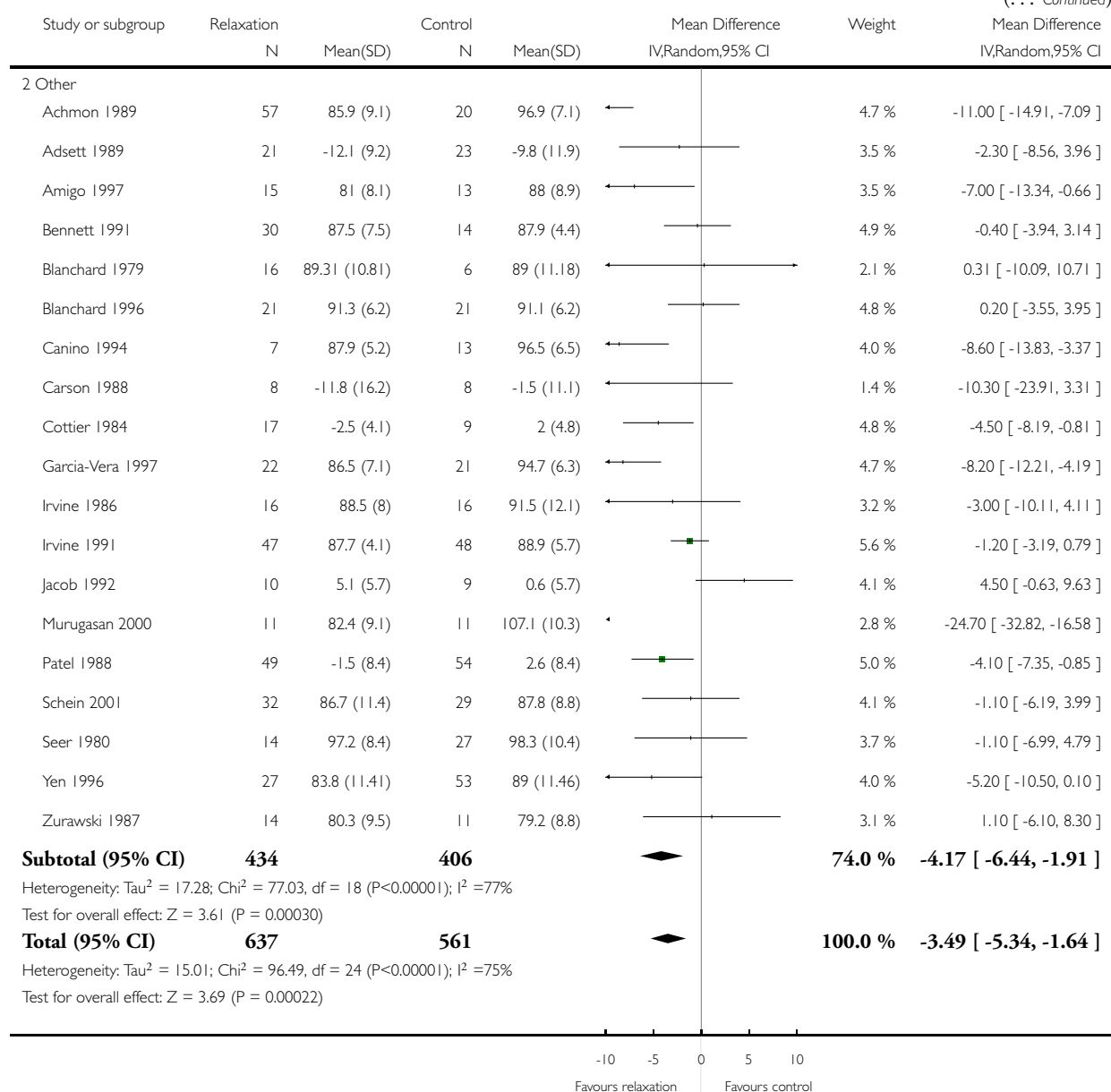
Comparison: 12 Relaxation versus control (sub-grouped by autogenic training/other)

Outcome: 2 Diastolic BP



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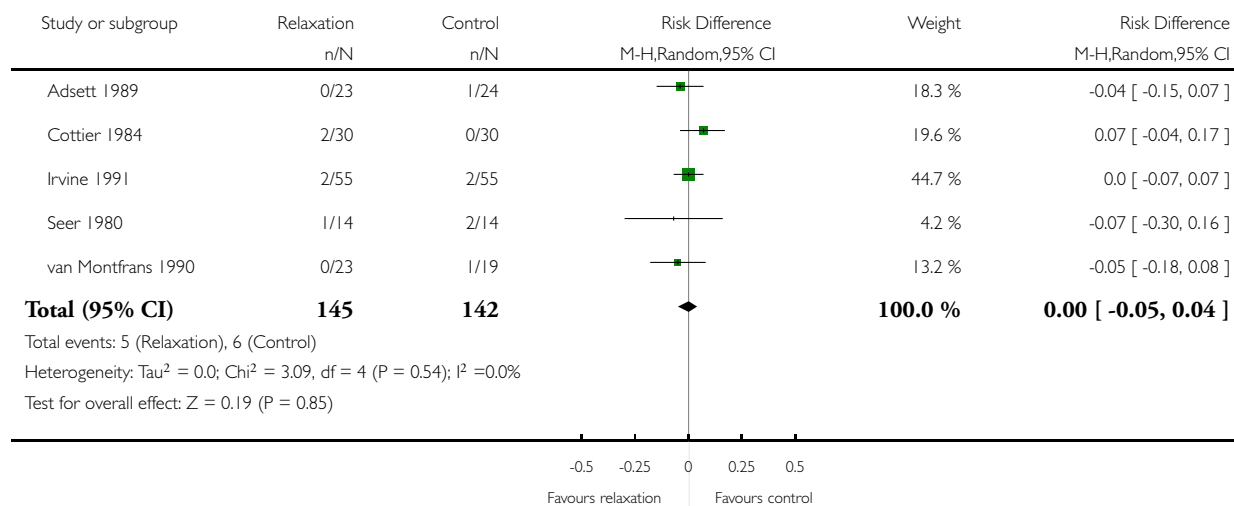


### Analysis 13.1. Comparison 13 Relaxation versus control, Outcome 1 Adverse events - uncontrolled hypertension.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 13 Relaxation versus control

Outcome: 1 Adverse events - uncontrolled hypertension

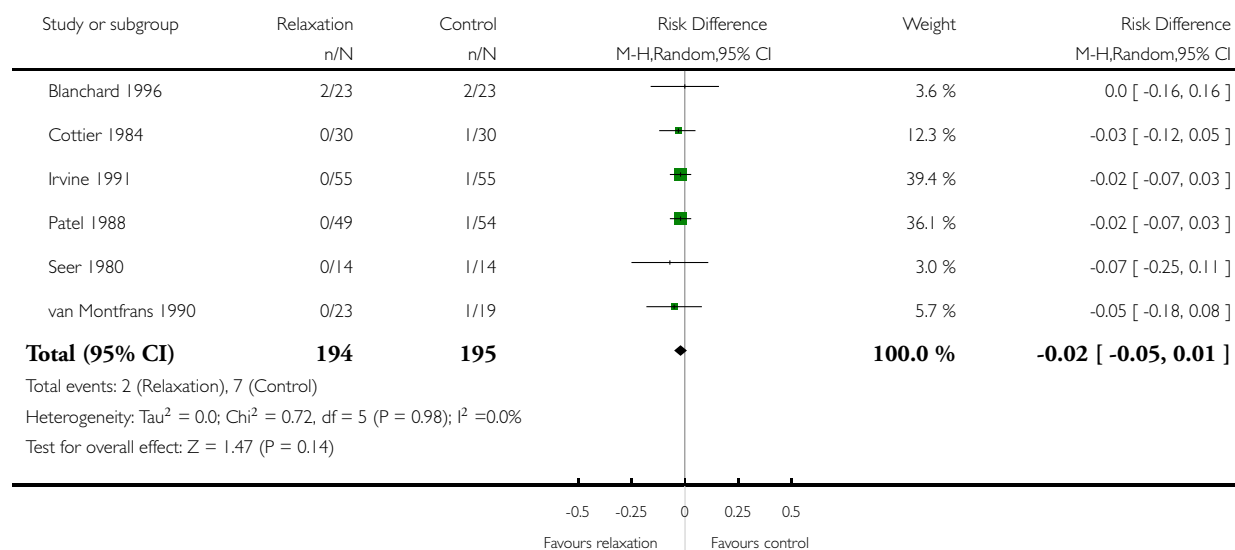


### Analysis 13.2. Comparison 13 Relaxation versus control, Outcome 2 Other adverse events.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 13 Relaxation versus control

Outcome: 2 Other adverse events



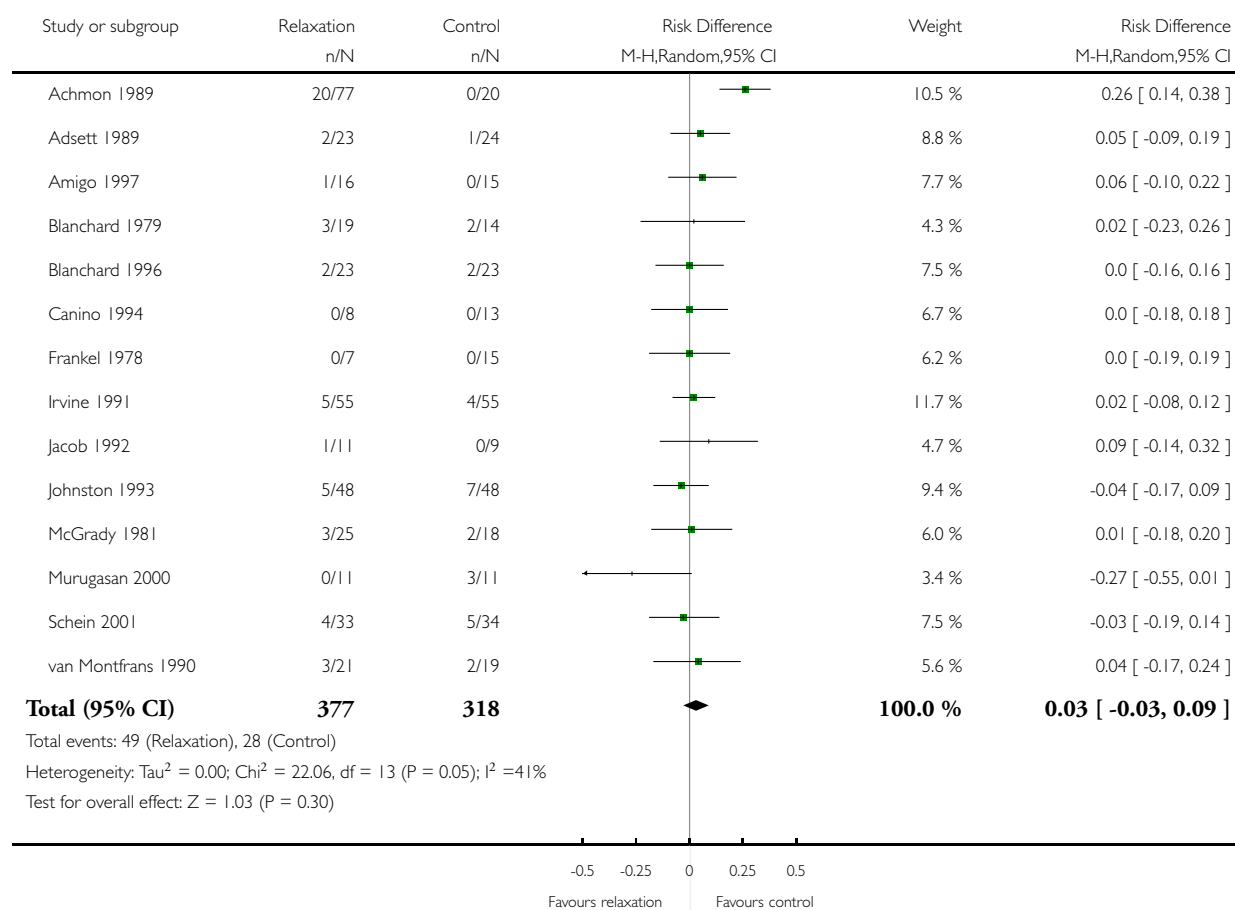


### Analysis 13.3. Comparison 13 Relaxation versus control, Outcome 3 Withdrawal from treatment.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 13 Relaxation versus control

Outcome: 3 Withdrawal from treatment

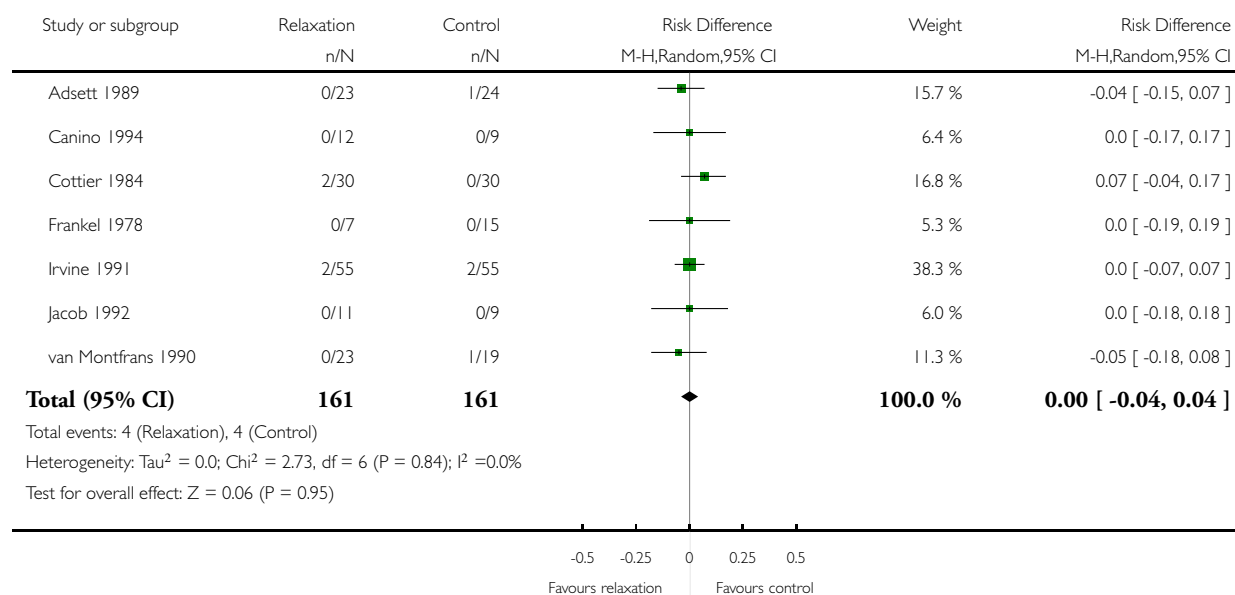


### Analysis 13.4. Comparison 13 Relaxation versus control, Outcome 4 Withdrawals due to adverse events - uncontrolled hypertension.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 13 Relaxation versus control

Outcome: 4 Withdrawals due to adverse events - uncontrolled hypertension

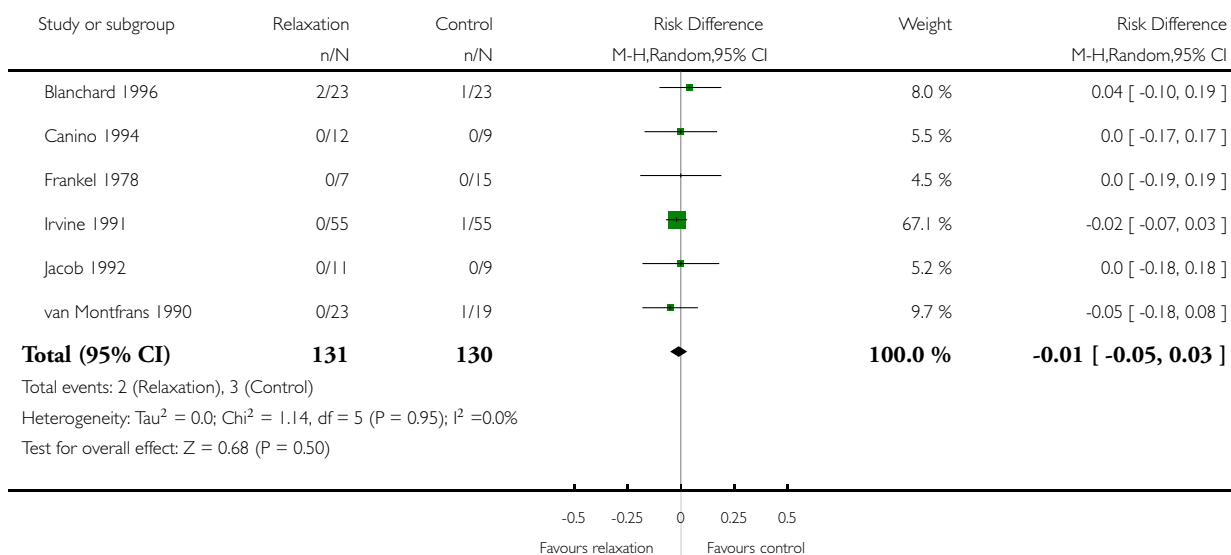


### Analysis 13.5. Comparison 13 Relaxation versus control, Outcome 5 Withdrawals due to other adverse events.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 13 Relaxation versus control

Outcome: 5 Withdrawals due to other adverse events

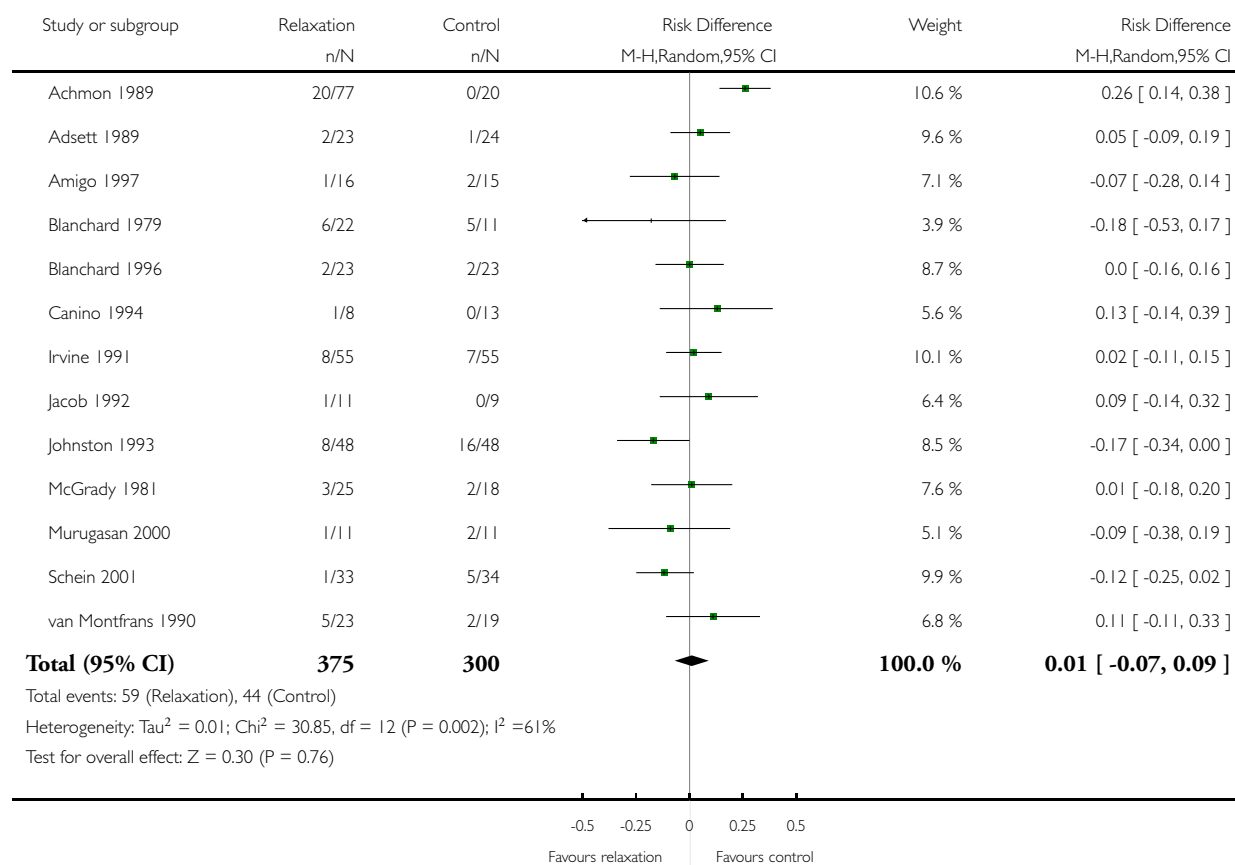


### Analysis 13.6. Comparison 13 Relaxation versus control, Outcome 6 Loss to follow-up.

Review: Relaxation therapies for the management of primary hypertension in adults

Comparison: 13 Relaxation versus control

Outcome: 6 Loss to follow-up



## ADDITIONAL TABLES

Table 1. Types of active treatment and control therapies used in relaxation trials.

Study	In primary meta-anal	Cogn/ Behav/ Meditat	Autogenic/ Relax Resp	Prog musc relaxation	Biofeed-back	Sham therapy	BP monitoring only	No intervention
Achmon 89	Yes	I1			I2	C		
Adsett 89	Yes			I		C		

**Table 1. Types of active treatment and control therapies used in relaxation trials.** (Continued)

Aivazyan 88b	Yes		I				C
Amigo 97	Yes			I		C	
Bennet 91	Yes	I1, I2					C
Blanchard 79	Yes			I2	I1, I2	C	
Blanchard 96	Yes				I		C
Bosley 89	No	I				C	
Canino 94	Yes	I		I	I	C1	C2
Carson 88	Yes			I		C	
Cottier 84	Yes			I			C
Frankel 78	Yes		I	I	I	C1	C2
Garcia-Vera 97	Yes	I		I			C
Hafner 82	No	I1, I2			I2		C
Irvine 86	Yes	I		I	I	C	
Irvine 91	Yes			I	I	C	
Jacob 92	Yes			I	I	C	
Johnston 93	Yes	I	I			C	
Krame-lashvili 86	No		I1		I2		C
Lagrone 88	No			I		C1	C2
McGrady 81	Yes		I		I		C
McGrady 94	Yes		I	I	I		C
Murugasan	Yes	I					C
Patel 88	Yes	I		I	I		C

**Table 1. Types of active treatment and control therapies used in relaxation trials.** (Continued)

Schein 01	Yes				I	C		
Seer 80	Yes	I				C1		C2
van Montfrans 90	Yes	I	I	I		C		C
Yen 96	Yes				I			C
Zurawski 87	Yes	I			I	C		

## WHAT'S NEW

Last assessed as up-to-date: 6 November 2007.

Date	Event	Description
12 November 2008	Amended	Contact details updated

## HISTORY

Protocol first published: Issue 3, 2004

Review first published: Issue 1, 2008

Date	Event	Description
13 August 2008	Amended	Converted to new review format.

## **CONTRIBUTIONS OF AUTHORS**

JM designed the study; HOD and DJN wrote the protocol; FRB performed the searches and managed the references; DJN, FC, FRB and HOD sifted the references; DJN, HOD, FC, JVC, FRB and JM abstracted the data; HOD performed the statistical analysis; HOD, DJN, FC and JVC wrote the review, GAF advised on clinical aspects and interpretation of the review.

## **DECLARATIONS OF INTEREST**

None. The funding source was not in a position to benefit financially from the results of the review.

## **SOURCES OF SUPPORT**

### **Internal sources**

- No sources of support supplied

### **External sources**

- National Institute for Clinical Excellence, UK.

## **INDEX TERMS**

### **Medical Subject Headings (MeSH)**

\*Relaxation Therapy; Hypertension [\*therapy]; Randomized Controlled Trials as Topic

### **MeSH check words**

Humans