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Psychological treatment of cardiac patients: a meta-analysis

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Mortality;
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Depression;
Distress

Previous reports of the effectiveness of psychological treatments (PTs) for cardiac patients reveal inconsistent results. We determined overall effects and gender differences. Eligible studies were randomized controlled trials, containing a PT arm. The authors identified 43 relevant randomized trials; 23 reported mortality data for 9856 patients. The odds-ratio (OR) for all-cause mortality at follow-up of 2 years or less, comparing PT plus usual care vs. usual care only, was OR 0.72 [95% confidence interval (CI) 0.56–0.94], but weakened with longer follow-up (OR 0.89; 95% CI 0.80–1.10). Mortality benefits only applied to men (OR 0.73, 95% CI 0.57–1.00; OR 1.01; 95% CI 0.87–1.72 for women). Trials initiating treatment at least 2 months after a cardiac event showed greater mortality benefits than those initiating treatment right after the event (OR 0.28; 95% CI 0.11–0.70 vs. OR 0.87; 95% CI 0.86–1.15, respectively). Mortality benefits due to PT were achieved despite small concomitant changes in negative affect. PT of cardiac patients reduces mortality and event recurrence. The mortality benefits appeared only in men even after controlling for age differences. The timing for the initiation of PT may be a critical mediating variable for mortality outcomes.

Psychological treatment (PT) often is a component of cardiac rehabilitation (CR). The term CR describes a broad class of interventions targeting risky behaviours, namely smoking, lack of exercise, poor eating habits, and often also targets psychological distress.[1,2] CR programs that focus on psychological factors are largely similar in that they use cognitive–behavioral interventions to reduce distress and teach psycho-physiological self-regulation skills.[3–5] While previous meta-analytic reviews[6–11] document the effectiveness of multi-component CR programs for reducing mortality and secondary event rates, the benefit of added PTs is still in question. To resolve this question, the current review targets only studies where (i) cardiac patients had been randomized to treatment and control and (ii) where at least one intervention arm permitted the isolation of effects specifically due to PT. Exercise, dietary modification, and smoking cessation were thus excluded.

We begin with a review of previous meta-analyses and then provide an updated meta-analysis of the outcome of PT. We also report gender-specific effects and evaluate the impact of varying program characteristics on outcome. The core features and results from meta-analyses published since the mid-1990s are described in *Table 1*. *Table 1* reveals differences in review methodologies that range from obvious (i.e. publication year) to less obvious discrepancies (i.e. study selection and categorization procedures). Predictably, there is overlap in the respective reference lists; therefore these reviews cannot be considered independent of each other. With respect to classification decisions, Rees *et al.*[9] and Clark *et al.*[11] included interventions where the CR had been amalgamated with psychological and 'other' (i.e. health education) rehabilitation components thus making it impossible to isolate benefits solely attributable to psychological components. Observed rates of mortality and morbidity reductions for PT relative to usual care (UC) control varied considerably with mortality reductions for the treatment groups at follow-up being as high as 71%[10] and as low as 3%[11] at 1-year follow-up. In none of the reviews were the effects of different active PTs statistically different from each other. Importantly, Dusseldorp *et al.*[8] showed that PT reduced cardiac-specific mortality (31% decrease) when distress had been reduced, but that studies in which distress was unchanged also failed to show mortality or morbidity benefits (14% increase).

The observed mortality benefits reported in the previous reviews were not systemically different in long vs. short follow-ups. Two reviews[8,11] reported greater mortality reductions with longer follow-up periods, whereas Linden

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† W.L. had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Table 1 Summary of review features and reported effects of previous meta-analyses

	Linden *et al.*[7]	Dusseldorp *et al.*[8]	Rees *et al.*[9]	Clark *et al.*[11]	Van Dixhoorn and White[10]
Studies included	23	37	36	63	27
Treatment conditions	A clearly identifiable, distinct psychological treatment	Structured health education	Stress management	Exercise only	Relaxation Therapy (single method relaxation and multi-component treatments were aggregated)
		Stress management	Other rehabilitation with psychological components	Risk factor education/ counselling plus exercise	
				Risk factor education/ counselling	
Control conditions	UC (not further defined)	UC (+/− health information)	UC (+/− exercise and/or health education)	UC (not further defined)	UC (not further defined)
Follow-up (FU)	<2 years;>2 years	<1 year; 1–2 years;>2 years	Mortality reported; length of FU not stated	1 year; 2 years; 5 years	Reported in narrative form, no comparison of short vs. long-term FU
Mortality OR (treatment vs. control)	0.57 (FU <2 years)	0.83 (<1 year)	0.86 for any psychological intervention	0.97 (1 year)	0.29 (2 years)
	0.72 (FU >2years)	0.94 (1–2 years)	0.62 for stress management (corresponding FU not stated)	0.53 (2 years)	No difference between type of active treatment
		0.66 (>2 years)		0.77 (5 years)	
		1.14 (when distress was not reduced); 0.69 (when distress was reduced)		No difference between type of active treatment	
Gender-specific outcomes	No	No	No	No	No
Consideration of treatment length	No	No	No	No	Yes
Distress reduction as a moderator	No	Yes	No	No	No
Non-English articles	No	Yes	Yes	No	Yes

et al.[7] reported the opposite trend. The reasons for such inconsistent mortality effects over time are unclear. It is surprising that no meta-analysis has reported tests of gender differences in mortality outcomes although important differences in individual studies were reported as early as 1997.[12] Given these previous conclusions, we sought to test the following hypotheses: (i) Does additional PT reduce mortality and morbidity, over short vs. longer follow-up, for both genders? (ii) Do program characteristics like timing of treatment and effectiveness of the distress reduction differentially affect outcomes?

Methods

Study selection

Study selection steps are shown in *Figure 1* and the studies and supplementary articles ultimately included[12–66] are described in *Table 2*. The search utilized the protocol outlined by the Cochrane Database of Systematic Reviews, using PsycInfo, Web of Science, Ovid Medline, PubMed, and EMBASE databases. In addition to the computer searches for the years 2002–2006, all secondary references from the earlier meta-analyses were followed, and the first author personally contacted key researchers in the field to inquire about relevant unpublished research. We reviewed studies in English, German, Portuguese, Chinese, Russian, Dutch, Swedish, Spanish, and Danish. Where necessary, interpreters with knowledge of psychological research assisted data extraction. Only one of the four foreign language articles provided mortality data.[44] In general, non-English publications provided fewer design and protocol details and the quality of the designs were therefore more difficult to assess. The search terms we used are listed in the appendix.

Inclusion criteria

For inclusion of a trial, it was necessary that one treatment condition involved a predominantly psychological or behavioural intervention. This psychological intervention had to exist against a backdrop of at least one UC control condition so that the additional benefit of psychological intervention could be evaluated. Uncritical

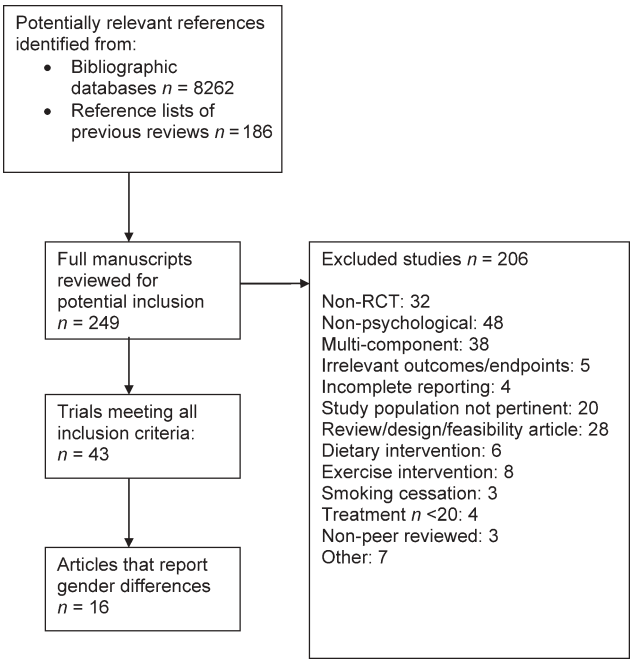

Figure 1 Study extraction and selection process.

acceptance of the term 'usual care' was deemed inappropriate because it harboured considerable heterogeneity in treatment exposure. The identified trials were subjected to a four-category system that promised highest inter-rater agreement. M.J.P. and J.L. then conducted independent categorization of all studies and an inter-rater agreement of 89% was reached (83 agreements out of 93 total decisions). Disagreements were evenly distributed across different pairings of categorization decisions suggesting that no one decision type was particularly problematic; remaining disagreements were resolved in a conference of the authors. This process resulted in initial grouping of studies into two types of control conditions and two active treatment conditions, namely:

(1) Basic UC control. Defined as 'under medical care but not participating in structured exercise and/or lifestyle counseling'.
(2) Multi-component UC control. Intervention including: nutrition counselling, exercise, and/or instruction/education about the disease and medications, typically offered by cardiac nurses, exercise specialists, or case managers. The instruction component was usually short (range 2–6 sessions) and not individually-tailored.
(3) Multi-component PT (active treatment). Given that the term stress management usually refers to a multi-component psychological intervention,[67] all treatments labelled 'stress management' were categorized as multi-component PT, as were all treatments labelled psychological therapy, cognitive–behavioural, or behaviour therapy. To qualify for the multi-component PT category, the intervention had to have at least two-thirds of the following characteristics: (i) majority of treatment exposure was for psychological targets (distress, depression, etc.); (ii) therapists were trained in mental health care at a graduate level; and (iii) treatment targets and choices were individually-tailored.
(4) Biological/self-regulation (BSR) treatments (active treatment). These included meditation, autogenic training, biofeedback, breathing, yoga, and/or muscular relaxation. When self-regulation training was part of multi-component stress management regime, such a study was categorized as PT. We created the BSR category as distinct from the multi-component category because BSR has a more specific psycho-physiological rationale for cardiac function than does 'stress management',[68] it is easier to standardize for service delivery,[10] and can be offered at lower expense than multi-component PT.

We included studies without follow-up (i.e. studies that were excluded in the Cochrane Review[9]) because, in addition to mortality and disease recurrence, we wished to learn about gender and process predictors of immediate outcomes. Given that a single publication may contain more than one active treatment, we only included the one considered most intensive. Excluded were (i) interventions (or conditions within a study) that were not fully randomized; (ii) interventions that had combined psychological and non-psychological components of CR and did not isolate the PT component; (iii) randomized trials without psychosocial or mortality/morbidity outcomes (e.g. those that only measured lipid changes); and (iv) studies where the intervention arm had less than 20 participants (to avoid unreliable findings).

Data extraction and analysis

Information regarding the following outcomes was extracted: all-cause mortality, cardiac mortality, CHD progression and recurrence, CHD risk factors (e.g. blood pressure, lipids), and/or psychological well-being (e.g. depression, hostility/anger, anxiety, social support, quality of life). Because all studies that provided mortality data provided all-cause mortality data but only some differentiated 'all-cause' from 'cardiac' mortality, we chose 'all-cause mortality' for consistency.

Although technically possible, comparisons involving less than 200 patients were not conducted. The number 200 was chosen to minimize random variation in findings and is based on the fact that 200 participants are needed to detect a significant effect of $r>0.3$ with 80% power. Data on mortality and morbidity (i.e. event recurrence) were computed as odds-ratios (ORs), and continuous data were initially computed as Cohen's effect sizes *d*, then converted to *r*. The frequently used Comprehensive Meta Analysis Version 2 software package[69] (www.Meta-Analysis.com) was used for statistical comparisons. The more conservative random effects model was chosen for between-group comparisons because of (i) the known heterogeneity of effects as reported by other reviewers (*Table 1*), and (ii) the fact that the random effects model makes fewer assumptions about shared population and treatment characteristics and are therefore more conservative.[70] The CMA program automatically weighs ORs and effect sizes for sample size, tests for homogeneity of variance, and provides confidence intervals. In a random effects model, very large sample studies have slightly less impact on obtained ORs than is true in a fixed-effects model[70] (p. 215).

For morbidity scores, the following endpoints were summed: rehospitalization for cardiac emergency and new cardiac procedures, new MI, newly diagnosed arrhythmias, or persistent angina. The computed data reflect numbers of patients with a recurring event and not absolute number of events because the latter was often not reported. Results are displayed in tabular format and forest plots are shown for the most critical analyses. Mortality reductions of at least 20% were considered as clinically meaningful even if they did not reach traditional cutoffs of $P<0.05$; this decision was based on the results of Lau *et al.*[71] who have shown that beta-blockers and exercise rehabilitation each brought about reductions of mortality of about 20%, and this rate was of sufficient importance to drive current clinical practice. The OR analyses addressed the following core questions:

(1) Does PT (aggregated across the BSR and multi-component PT types) confer additional benefits relative to UC (aggregated across the two types of UC)?
(2) Do benefits vary by gender? Female patients were an average of 6 years older than male patients, thus age was included as a covariate in these analyses.
(3) Are PTs that are implemented right after a cardiac event (defined as less than 2 months post event) more effective than treatment that began later? The 2 months cutoff was chosen because it reflects the median length of wait times for CR access in Canada.[72]
(4) Can we replicate the results of Dusseldorp *et al.*[8] that patients whose distress (or depression) was not reduced also did not

Table 2 Summary information about study characteristics*

				Experimental group characteristics		
Study, Year (Location)	Cardiac population	Group	Type of intervention	Age (years)	Women (%)	Tx Length/Follow-up (months)
Appels *et al.*,[12] 2005 (Netherlands)	PCI	C	Basic UC ($n = 344$)	53.6	20	6/18
		E	+Multi-component PT ($n = 366$)			
Baumbauer *et al.*,[13] 2005 (USA)	CA, MI, CHD	C	Basic UC ($n = 34$)	59.9	31	2/6
		E	+Multi-component PT ($n = 45$)			
Bishop *et al.*,[14] 2005 (Singapore)	CABG	C	Basic UC ($n = 29$)	54.7	0	1.5/3
		E	+Multi-component PT ($n = 29$)			
Black *et al.*,[15] 1998 (USA)	CHD	C	Multi-component UC ($n = 30$)	60.7	12	2/21
		E	+Multi-component PT ($n = 30$)			
Blom *et al.*,[16] *in press* (Sweden)	MI, PCI, CABG	C	Basic UC ($n = 122$)	62	100	12/17
		E	+Multi-component PT ($n = 113$)			
Blumenthal *et al.*,[17,18] 2002 and 1997[a] (USA)	CHD+MI	C	Multi-component UC ($n = 26$)	58.5	0	4/60
		E	+Multi-component PT ($n = 31$)			
Blumenthal *et al.*,[19] 2005 (USA)	CHD+MI	C	Basic UC ($n = 42$)	63	34	4/0
		E	+Multi-component PT ($n = 44$)			
Burell *et al.*,[20] 1994; Sundin *et al.*,[21] 1994[a] (Sweden)	CHD	C	Multi-component UC ($n = 24$)	54.8	0	12/12
		E	+Multi-component PT ($n = 25$)			
Burgess *et al.*,[22] 1987 (USA)	MI	C	Basic UC ($n = 91$)	62	15	3/10
		E	+Multi-component PT ($n = 89$)			
Claesson *et al.*,[23] 2005 (Sweden)	CHD	C	Basic UC ($n = 86$)	59	100	12/0
		E	+Multi-component PT ($n = 80$)			
Clark *et al.*,[24] 1997 (USA)	CHD	C	Basic UC ($n = 318$)	69.6	61	1/17
		E	+Multi-component PT ($n = 318$)			
Cowan *et al.*,[25,26] 2001 and 1997[a] (USA)	CHD	C	Basic UC ($n = 66$)	60	27	1.5/24
		E	+Multi-component PT ($n = 67$)			
del Pino *et al.*,[27] 2005 (Canary Islands)	CHD	C1	Basic UC ($n = 32$)	49.7	0	9/24
		C2	Multi-component UC ($n = 33$)			
		E	+Multi-component PT ($n = 33$)			
Berkman *et al.*,[28] 2003; ENRICHD,[29] 2001[a]; Schneiderman *et al.*,[30] 2004[a] (USA)	MI	C	Basic UC ($n = 1243$)	61	43	6/12
		E	+Multi-component PT ($n = 1238$)			
Fielding *et al.*,[31] 1979 (UK)	MI	C	Basic UC ($n = 21$)	...	0	1.5/12
		E	+Multi-component PT ($n = 24$)			
Frasure-Smith *et al.*,[32,75] 1997 and 2002[a] (Canada)	MI	C	Basic UC ($n = 684$)	59.3	34	12/60
		E	+Multi-component PT ($n = 692$)			
Frasure Smith *et al.*,[33,34] 1985 and 1989[a] (Canada)	MI	C	Basic UC ($n = 229$)	58	0	12/60
		E	+Multi-component UC ($n = 232$)			
Gallacher *et al.*,[35] 1997 (UK)	CA	C	Basic UC ($n = 225$)	...	0	2.5/6
		E	+Biofeedback/Self-regulation ($n = 227$)			
Gallagher *et al.*,[36] 2003 (Australia)	CHD	C	Multi-component UC ($n = 103$)	67	100	1.5/3
		E	+Multi-component PT ($n = 93$)			

Continued

Table 2 *Continued*

				Experimental group characteristics		
Gillis *et al.*,[37] 1993; Gortner *et al.*,[38] 1988[a] (USA)	Cardiac surgery	C	Basic UC ($n = 81$)	59.2	9	2/4
		E	+Multi-component PT ($n = 75$)			
Gruen *et al.*,[39] 1975 (USA)	MI	C	Basic UC ($n = 37$)	50	...	0.75/4
		E	+Multi-component PT ($n = 38$)			
Guzzetta *et al.*,[40] 1989 (USA)	MI	C	Basic UC ($n = 27$)	57.6	12.5	0.07/0
		E	+Biofeedback/Self-regulation ($n = 53$)			
Ibrahim *et al.*,[41] 1975 (USA)	MI	C	Basic UC ($n = 60$)	54.5	19.5	11.5/6
		E	+Multi-component PT (n=58)			
Janz *et al.*,[42] 2004; Clark *et al.*,[43] 1992[a] (USA)	CHD	C	Basic UC ($n = 143$)	73.0	100	1.5/4
		E	+Multi-component PT ($n = 314$)			
Johansen *et al.*,[44] 2003 (Denmark)	MI	C	Basic UC ($n = 100$)	...	23	.../...
		E	+Multi-component PT (n=100)			
Jones and West,[45] 1996 (UK)	MI	C	Basic UC ($n = 1160$)	...	...	1.75/12
		E	+Multi-component PT ($n = 1168$)			
Kanji *et al.*,[46] 2004 (UK)	PCI	C	Basic UC ($n = 29$)	64.5	37	2/3
		E	+Biofeedback/Self-regulation ($n = 30$)			
Krucoff *et al.*,[47] 2005 (USA)	PCI	C	Basic UC ($n = 192$)	66	29	.../6
		E	+Biofeedback/Self-regulation ($n = 185$)			
Liao *et al.*,[48] 2004 (China)	Heart valve surgery	C	Basic UC ($n = 40$)	38.5	67.5	0.75/0
		E	+Multi-component PT ($n = 40$)			
Ma and Teng,[49] 2005 (China)	CA	C	Basic UC ($n = 50$)	56.5	...	2/0
		E	+Multi-component PT ($n = 50$)			
Mayou *et al.*,[50,51] 2002 and 2005[a] (UK)	MI	C	Basic UC ($n = 58$)	...	20	.../12
		E	+Multi-component PT ($n = 56$)			
Michalsen *et al.*,[52] 2005 (Germany)	CHD	C	Basic UC ($n = 53$)	59	21	12/0
		E	+Multi-component PT ($n = 48$)			
Nelson *et al.*,[53] 1994 (USA)	MI	C	Multi-component UC ($n = 20$)	56.6	0	0.27/6
		E	+Biofeedback/Self-regulation ($n = 20$)			
Rahe *et al.*,[54] 1975; Rahe *et al.*,[55] 1979[a] (USA)	MI	C	Basic UC ($n = 21$)	48.3	8	3/45
		E	+Multi-component PT ($n = 36$)			
Schulte *et al.*,[56] 1986 (Netherlands)	MI	C	Basic UC ($n = 16$)	55	...	2.5/0
		E	+Multi-component PT ($n = 29$)			
Sebregts *et al.*,[57] 2005 (Netherlands)	MI, CABG	C	Basic UC ($n = 98$)	55.6	14	2/9
		E	+Multi-component PT ($n = 106$)			
Stern *et al.*,[58] 1983 (USA)	MI	C1	Basic UC ($n = 29$)	59	11	3/9
		C2	Multi-component UC ($n = 42$)			
		E	+Multi-component PT ($n = 35$)			
Sundin *et al.*,[59] 2003 (Sweden)	PCI, CABG, MI	C	Basic UC ($n = 36$)	58.8	0	12/0
		E	+Multi-component PT (n=32)			

Trzcieniecka-Green and Streptoe,[60] 1996 (UK)	MI, CABG	C	Basic UC ($n = 50$)	59.4	14	2.5/6
		E	+Biofeedback/Self-regulation ($n = 50$)			
Van Dixhoorn *et al.*,[61] 1987 (Netherlands)	MI	C	Multi-component UC (n=47)	55.5	7	1.5/27.5
		E	+Multi-component PT ($n = 43$)			
Van Dixhoorn *et al.*,[62–64] 1989, 1990[a], 1999[a] (Netherlands)	MI	C	Multi-component UC ($n = 80$)	55.4	7	1.5/59
		E	+Multi-component PT ($n = 76$)			
Van Elderen *et al.*,[65] 1994 (Netherlands)	CHD	C	Basic UC ($n = 108$)	55	14	2/7
		E	+Multi-component PT ($n = 109$)			
Wan *et al.*,[66] 2005 (China)	CHD	C	Basic UC ($n = 31$)	59.2	39	0.75/0
		E	+Multi-component PT ($n = 31$)			

Ellipses indicate insufficient data given.
PCI, percutaneous coronary interventions; CA, coronary angina; CHD, coronary heart disease; MI, myocardial infarction; CABG, coronary artery bypass grafting; C1, control subgroup 1; C2, control subgroup 2; E, experimental group; UC, usual care; PT, psychological therapy.
[a]Supplemental data obtained/data used for effect size calculations or intervention characteristics.

benefit in terms of mortality? We used actual observed median effect sizes of distress and depression reduction obtained from the current data to create relevant success/failure groupings.

Non-categorical data

Non-categorical outcome data were either psychological in nature (e.g. anxiety) or reflected indices of biological risk (e.g. blood pressure). The effect sizes were calculated for each outcome within each individual study, weighted for sample size and reported for all studies, aggregated across treatment conditions and genders, and (where possible) reported separately for men and women.

Results

Mortality and morbidity

As the data in *Table 3* indicate, 23 studies provided data on mortality. Patients who received PT benefited in terms of short-term mortality reductions (OR 0.72; 95% CI 0.56–0.94) (*Figure 2*), but this effect became non-significant as follow-up lengthened. Event recurrence was reduced at long-term follow-up (OR 0.57; 95% CI 0.37–0.86) but not at short-term follow-up (OR 0.84; 95% CI 0.70–1.02).

When the analyses were repeated separately for the two genders, only 10 studies met inclusion criteria; these studies described results from 1190 women and 2034 men (*Figures 3* and *4*). Men benefited from PT with a 27% short-term mortality reduction but women did not (OR 0.73 vs. 1.01; 95% CI 0.51–1.05 vs. 0.46–2.23). Note that the observed OR of 0.73 for men was accompanied by a marginal significance level of $P = 0.057$ (*Tables 4* and *5*). There was a similar but weaker pattern of gender differences for morbidity.

Mortality reductions were strong for studies that initiated treatment late (72% reduction in short-term mortality; 95% CI 0.12–0.70) (*Figure 5*), whereas early initiation was not associated with a significant change in mortality (−13%; 95% CI 0.75–1.20) (*Figure 6*). Similarly, there was a significant reduction in short-term mortality in patients in whom distress had actually been reduced (OR 0.46; 95% CI 0.28–0.75); no significant mortality reduction was observed when distress reduction had been unsuccessful (OR 0.67; 95%CI 0.27–1.65). Whether depression was effectively reduced, or was not reduced, had no impact on mortality outcomes (ORs 1.03 and 1.04, respectively, for short-term follow-up).

Biological risk factor and psychological variables (Tables 4 and 5)

Univariate *F*-tests (aggregated for both genders and weighted for sample size) revealed that PT was statistically superior to control for reduction of heart rate (the difference in *r*-scores (r) was −0.21; a minus score indicates superiority of PT over control), and it improved both social support (r, −0.16) and quality-of-life (r, −0.34). Treated women showed significant reductions of distress and improved social support, whereas treated men showed greater reduction of depression and improved social support. Irrespective of significance, all signs in front of the effect sizes point in the same direction, namely superiority of treatment over control.

Additional computations of zero-order correlations revealed that older participants showed less reduction in depression (r, −0.48; $P = 0.014$) and anxiety (r, −0.43; $P = 0.057$). Obtained correlation coefficients for reductions in various types of negative affect were highly consistent,

Table 3 Odds-ratios for mortality and morbidity outcomes: psychological treatment vs. usual care

Sub-analysis	Follow-up	Mortality				Morbidity			
		K	*N*	OR (CI, 95%)	Homogeneity of variance I^2 (*P*)	*K*	N	OR (CI, 95%)	Homogeneity of variance I^2 (*P*)
All studies									
	≤2 years	23	9856	0.72 (0.56–0.94)	34.0 (0.06)	22	7831	0.84 (0.70–1.02)	41.9 (0.021)
	>2 years	6	4727	0.89 (0.69–1.14)	39.4 (0.14)	6	5872	0.57 (0.37–0.86)	71.5 (0.004)
Gender									
Women	≤2 years	4	1190	1.01 (0.46–2.23)	58.8 (0.06)	4	1121	0.88 (0.67–1.15)	0.0 (0.61)
	>2 years	2	1557	1.30 (0.95–1.79)	25.0 (0.25)	1	1084	1.01 (0.80–1.29)	0.0 (1.00)
Men	≤2 years	6	2042	0.73 (0.51–1.05)	17.5 (0.30)	6	2023	0.84 (0.64–1.11)	32.2 (0.19)
	>2 years	3	2761	0.83 (0.67–1.03)	0.0 (0.94)	3	1914	0.82 (0.68–0.99)	2.0 (0.36)
Total/Overall	≤2 years	10	3232	0.77 (0.56–1.07)	49.7 (0.46)	10	3144	0.86 (0.71–1.04)	2.0 (0.18)
	>2 years	5	4318	0.96 (0.80–1.15)	46.8 (0.02)			...	...
Treatment initiation									
≤2 months post-event	≤2 years	14	8522	0.87 (0.66–1.15)	37.5 (0.08)	11	6432	0.80 (0.61–1.04)	62.5 (0.003)
	>2 years	5	4527	0.95 (0.75–1.20)	31.1 (0.21)	4	5616	0.63 (0.39–1.03)	73.6 (0.010)
>2 months post-event[a]	≤2 years	6	639	0.28 (0.12–0.70)	0.0 (0.98)	7	659	0.94 (0.61–1.46)	0.0 (0.64)
Distress reduced?									
Yes[a]	≤2 years	3	754	0.46 (0.28–0.75)	0.0 (0.53)	3	687	0.79 (0.57–1.08)	0.0 (0.38)
No[a]	≤2 years	4	603	0.67 (0.27–1.65)	0.0 (0.71)			...	...
Depression reduced?									
Yes[a]	≤2 years	3	1071	1.03 (0.78–1.36)	0.0 (0.65)	4	1274	0.93 (0.74–1.18)	0.0 (0.51)
No[a]	≤2 years	6	4173	1.04 (0.79–1.37)	2.0 (0.40)	5	3946	0.92 (0.76–1.12)	0.0 (0.49)

Odds-ratios <1 indicate a reduction in mortality/morbidity due to psychological treatment.

K, number of studies included in analysis; *N*, sample size; OR, odds ratio; *P*, level of significance; CI, confidence interval; I^2, homogeneity of variance statistic; ellipse, insufficient data to complete analysis.

[a]No data for >2 years.

positively inter-correlating with *r*-coefficients ranging from 0.50 to 0.95, thus indicating that changes in various indices of psychological well-being moved in a synchronous manner.

Discussion

Key findings

Together our findings reveal that PT offered in addition to UC reduces mortality for at least the first 2 years. Our results also provide the first meta-analytic evidence of gender differences in outcomes, and illustrate for the first time that treatment program characteristics differentially affect outcome. Overall, PT of cardiac patients reduced mortality by 27% for follow-up of 2 years or less and reduced event recurrence at follow-up longer than 2 years by 43%. There were no mortality benefits for women (OR 1.01 and OR 1.30, for short- and long-term follow-up, respectively). PT initiated within 2 months of the cardiac event produced no significant mortality benefits (−13%, n.s.), whereas studies that recruited cardiac patients later reported much greater benefits (−72%, $P = 0.01$) at less than 2-year follow-up.

We replicated Dusseldorp *et al.*'s[8] conclusion that successful reduction of distress was necessary for mortality benefits to occur (<2 year mortality reduction of 54%, $P = 0.002$) and that studies that failed to reduce subjective distress correspondingly failed to produce significant mortality benefits (−33%, $P = 0.38$); however, the difference observed in the current analysis is considerably smaller than the one originally reported by Dusseldorp *et al.* Failure or success in the reduction of depression did not affect mortality, although this finding is in contrast to a re-analysis of the ENRICHD findings where it had been shown that successful reduction of depression also accounted for observed mortality benefits.[73]

Overall, the conclusions are largely consistent with the ones drawn in the earlier reviews (*Table 1*). The previously observed reductions in mortality attributable to PT ranged from −3% to −71% and places the values obtained in this review—for follow-up of 2 years or less—approximately in the centre of this spectrum. We posit that the beneficial findings reported here are attributable to clean definitions of treatments to be included, to wide sampling of relevant studies, and to exclusion of unreliable small-*n* studies thus reducing opportunities for publication bias. Similar to previous reviews,[7–9] the observed mortality and morbidity benefits were paired with only small-to-moderate-sized reductions in negative affect and biological risk factors. In light of the high costs of cardiac surgery and extended hospital stays, an investment of about $2000 for PT (average treatment exposure was 16.3 h, range 0.67–52.5 h) is not a prohibitive expense.

The largest trial to date (ENRICHD[28]) was characterized by a significant reduction in depression that, however, failed to achieve superiority over controls because the UC control group had also improved. Original to our analysis is the finding that studies which initiate psychological PT soon after the cardiac event (as ENRICHD did) show little benefit

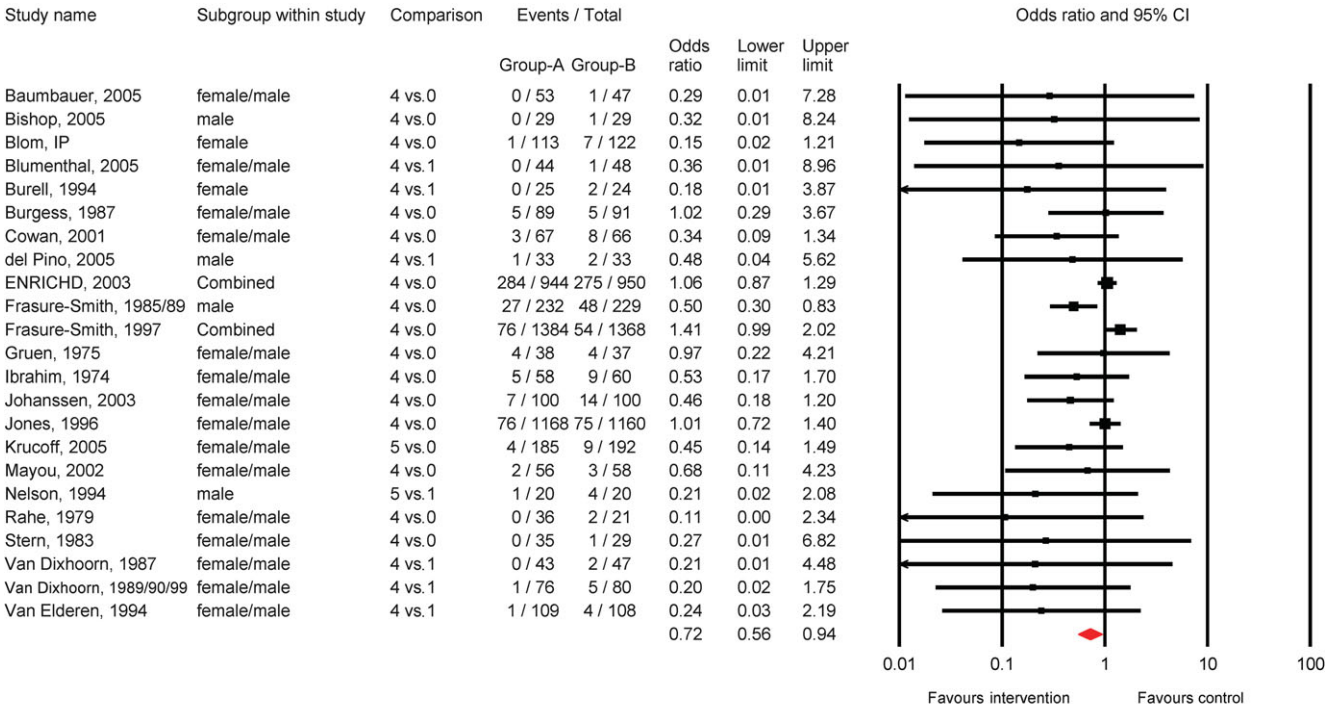

Figure 2 Overall effect on mortality (<2 years).

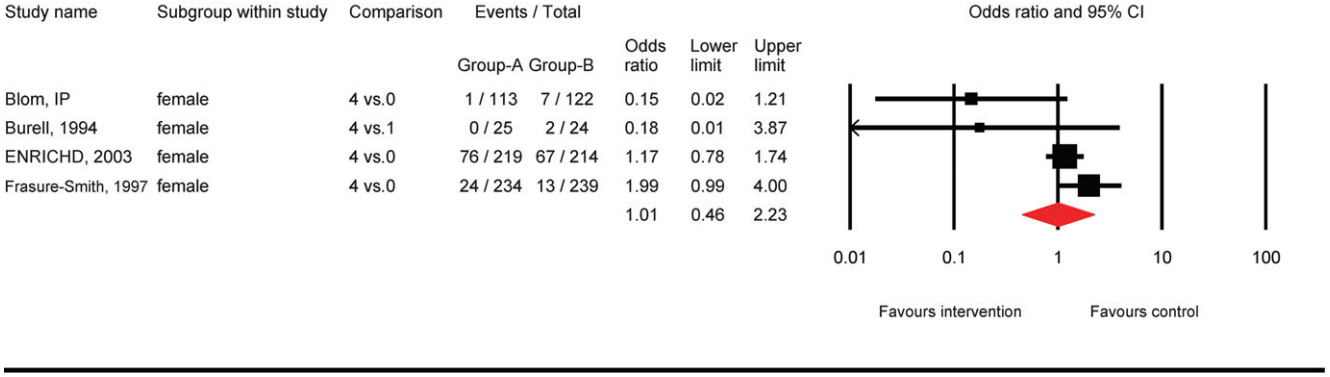

Figure 3 Effect on mortality in females (<2 years).

compared with UC controls, whereas those that start PT at least 2 months post-event showed an impressive mortality reduction of 72% in the first 2 years. It is possible that patients recruited late differ in a number of ways from those recruited early. We speculate that early recruitment may capture a subgroup of patients (in treatment and control groups alike) who possess excellent resources and resilience, and who will often recover even without professional help. This latter observation is consistent with the results of Schrader *et al.*[74] who showed that the prevalence of depression in post-myocardial infarction patients changes over time, revealing spontaneous improvement in some, but also worsening of depression in other patients. Therefore, screening for residual distress and depression and subsequent treatment provision might be more cost-effective than screening and offerings of PT very early after a cardiac event. This interpretation needs to be gauged against the fact that the question of effectiveness of PT as a function of timing of treatment has never been directly compared within a single study, and we believe that such a comparison is urgently needed to assure that our suggested explanation is accurate and that resulting decisions for clinical practice can be based on solid empirical data.

Limitations

Some of the conclusions drawn are hampered by the inevitable shortcomings of meta-analyses. Although we did control statistically for the existing age difference of men and women, this cannot be seen as a strong test due to the fact that in a meta-analysis only the mean age for total samples is provided, hence creating possible range restriction problems. Note, however, that for our analyses the range of mean ages was 38.2 to 73 years and thus not overly narrow.

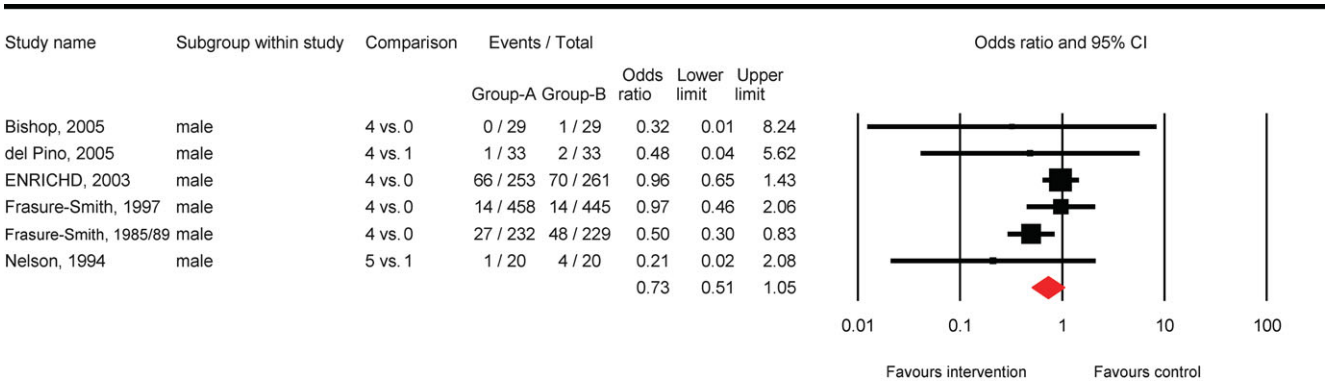

Figure 4 Effect on mortality in males (<2 years).

Table 4 Effect sizes *r* for change in biological risk factor variables

	Biological indices				
	Systolic BP, *k*=/*n*= fail-safe *n*, *r*	Diastolic BP, *k*=/*n*= fail-safe *n*, *r*	Heart rate, *k*=/*n*= fail-safe *n*, *r*	Total cholesterol, *k*=/*n*= fail-safe *n*, *r*	HDL, *k*=/*n*= fail-safe *n*, *r*
Both genders[a]	5/774	5/774	5/402 40	3/569	1/452
Treatment	−0.03	−0.09	−0.18*	−0.19*	−0.02
Control	0.08	0.03	0.03	0.00	−0.13

Fail-safe *n* are reported only for significant differences ($P < 0.05$).
HDL, high-density lipoprotein; BP, blood pressure; *k*, number of studies; *n*, total sample size.
[a]Insufficient data to report gender differences on these biological outcomes.
*$P < 0.05$, significant superiority of psychological treatment over control.

Table 5 Effect sizes *r* for change in psychological variables

	Psychological indices						
	Depression, *k*=/*n*= fail-safe *n*, *r*	Anxiety, *k*=/*n*= fail-safe *n*, *r*	Distress, *k*=/*n*= fail-safe *n*, *r*	Quality-of-life, *k*=/*n*= fail-safe *n*, *r*	Perceived support, *k*=/*n*= fail-safe *n*, *r*	Vital exhaustion, *k*=/*n*= fail-safe *n*, *r*	Anger/Hostility, *k*=/*n*= fail-safe *n*, *r*
Both genders	23/8914	21/5987	14/2841	13/2971 155	5/2719 40	4/1297	10/1440
Treatment	−0.30	−0.17	−0.36	−0.21*	−0.28*	−0.35	−0.19
Control	−0.21	−0.11	−0.20	−0.13	−0.12	−0.25	−0.03
Women	6/1286	2/669	3/692 16	...	1/1084 1	...	...
Treatment	−0.28	−0.18	−0.27*	...	−0.44*	...	...
Control	−0.23	−0.25	−0.10	...	−0.14	...	...
Men	8/3097 81	5/1526	3/963	...	2/1455 7	4/1297	10/1440
Treatment	−0.28*	−0.11	−0.76	...	−0.29*	−0.35	−0.19
Control	−0.17	−0.03	−0.53	...	−0.14	−0.25	−0.03

Fail-safe *n* are reported only for significant differences ($P < 0.05$).
k, number of studies; *n*, total sample size; ellipse, insufficient data to complete analysis.
*$P < 0.05$, significant superiority of psychological treatment over control.

There is widespread consensus that publication bias may exist in any meta-analysis and thus paint an overly positive picture of outcomes due to the fact that unsuccessful studies may never get published. In this meta-analysis, publication bias does not appear to be a major threat in that the three largest trials[29,45,75] had been described in

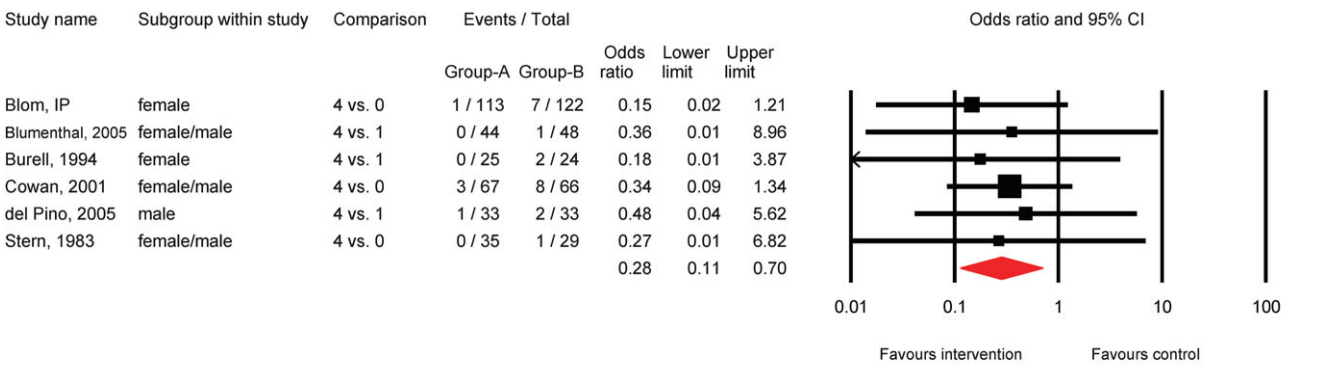

Figure 5 Effect of mortality in patients recruited late in the rehabilitation process.

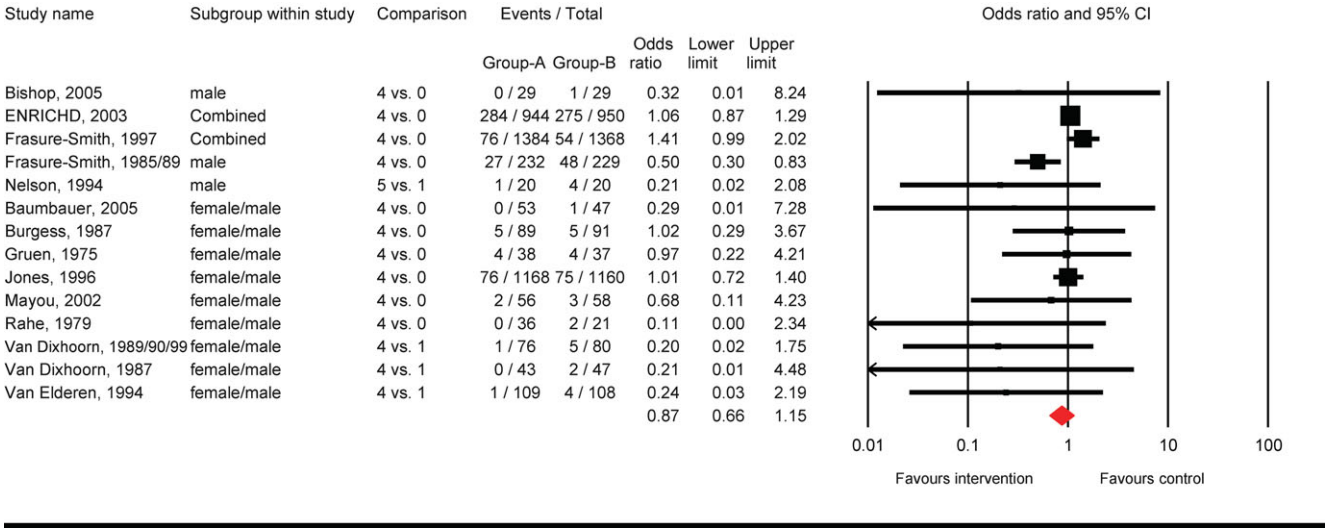

Figure 6 Effect of mortality in patients recruited early in the rehabilitation process.

major peer-reviewed journals and had provided 71% of the patients available for analyses on mortality outcomes. All three trials reported essentially null findings and this, in turn, runs counter to a claim of positively biased outcomes. In a related fashion, potential hetereogeneity of outcomes places constraints on the ease of interpretation of findings; our analysis is no exception. The reported results were mostly based on homogeneous outcomes (*Table 3*) but that is not equally true for all reported analyses. Unfortunately, the meta-analytic researcher cannot change this, only report the relevant statistics and ask for caution in interpretation.

We have conducted a number of additional analyses on various aspects of treatment program design but these were unfortunately statistically underpowered and did not allow meaningful conclusions. Therefore, remaining, unresolved questions are:

(1) Is type of PT critical? Previous reviews (*Table 1*) suggest that it may not be of importance. In our review, only two studies were found in the 'pure' relaxation/biofeedback category and their samples were small to moderate in size ($n = 40$ and $n=377$); the mortality benefits obtained for <2 years (OR=0.38, CI 0.13–1.10, n.s.) looked promising although inconclusive. We posit that resolving this question is of critical importance for cost-effective service delivery.
(2) Is distress screening needed to avoid floor effects? Evidence for other outcome research suggests that it may be important.[76]
(3) How much treatment is needed for lasting benefits? Strong support for lengthy interventions was provided in an earlier, influential, large sample rehabilitation study.[77]
(4) Does PT add more or less benefit when the quality of UC is high vs. low? In this meta-analysis, data from these two types of control groups were ultimately aggregated into one control group because analyses separated for two types of control groups had been underpowered and thus inconclusive.

Recommendations

We suggest assessing distress and depression repeatedly throughout the CR process and offering PT primarily to those who continue to struggle with adjustment for months after the critical event. When PT is initiated, it

should continue until distress is clearly reduced. An *a priori* fixed length of treatment (as is typical in clinical trials) may be unsuitable for clinical practice. Furthermore, we think it is urgent to develop PTs for female cardiac patients that meet their unique needs (i.e. emotional processing, being listened to, and attention to family role issues[16,23]). While men respond well to direct advice about required lifestyle changes, evidence has suggested that women do not.[78] This gender-specific treatment need may in part be a consequence of the greater age and social isolation of female cardiac patients.[78] A promising direction for effective PT of women is suggested via two Swedish interventions where the program had been specifically tailored for women participants.[16,23] Given that the sample sizes of these two studies were substantially smaller than those of two large studies where women had failed to reap benefits from PT,[28,30,32,75] the ultimate value of gender-tailored programs needs to be determined via replication in different health care environments. Nevertheless, these Swedish programs provide a beacon for further PT research with female cardiac patients.

In sum, our findings largely concur with those of earlier narrative and meta-analytic reviews. We were able to show for the first time that women with cardiac illness do not benefit from traditional PT and that early treatment initiation may not be effective.

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Author contributions: Study concept and design: W.L., J.L.; Acquisition of data: W.L., M.J.P., J.L.; Analysis and interpretation of data: W.L., M.J.P.; Drafting of the manuscript: W.L., M.J.P.; Critical revision of the manuscript for important intellectual content: W.L., M.J.P., J.L.

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Appendix

Appendix 1 Search terms employed in online database search

Patient characteristics	Psychological interventions	Outcomes
hostil*	autogen* train*	anxiety
myocard* infart*	biofeedback	distress
myocard* adj5 infarct	cardiac rehab*	mortality
MI	relax* train*	morbidity
heart adj5 surgery	psychosocial nursing therap*	cholestrol
coronary adj5 by pass	hypno*	overweight
percutaneous transluminal coronary angioplasty	relax* therap*	weight
PTCA	progressive muscle relax*	obesity
CHD	meditat*	smoking
coronary adj5 disease	psychologic* adj5 intervention	depression
heart adj5 disorder	stress manag*	anger
cardiovascular adj5 disorder	counsel*	quality of life
type A behavior	psychoeducat* adj5 intervention	well-being
myocardial adj5 ischemi*	relax*	blood pressure
myocardial adj5 ischaemi*	therapy	psychopathol*
angina	autogen*	social isolation
ischemi* adj5 heart	behavior adj5 modif*	social conflict
ischaemi* adj5 heart	behaviour adj5 modif*	social domination
coronary adj5 angioplast*	behavior adj5 therap*	negative affect
coronary adj5 thrombo*	behaviour adj5 therap*	job stress
myocardial revascularization	psychoeducat*	career stress
angioplasty	self adj5 manage*	stress
coronary adj5 arter*	patient adj5 educat*	nutrition
	health adj5 educat*	food
	stress adj5 manage*	risk adj5 reduction
	cognit* adj5 therap*	risk adj5 modific*
	anxiety adj5 manage*	
	relax* techniques	
	psychotherap*	
	psycholog* adj5 treatment	
	muscle adj5 train*	
	lifestyle adj5 modific*	
	self adj5 monitor*	
	cognitive adj5 restructur*	
	physical adj5 exercise	

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