SCIENTIFIC LETTER

Over 20 000 avoidable coronary deaths in England and Wales in 2000: the failure to give effective treatments to many eligible patients

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Many evidence based cardiological treatments reduce coronary heart disease (CHD) deaths. These treatments together explained over 40% of the substantial fall in CHD deaths between 1981 and 2000.[1] However, the CHD National Service Framework (NSF) recognised in 1999 that barely half of all eligible patients actually received effective treatments for myocardial infarction (MI), angina, or heart failure. Uptake rates were consistently worse among women, the elderly, and the deprived.[2 3] This study therefore examined the reduction in CHD deaths potentially achievable through increasing treatment levels in England and Wales.

METHODS

The previously validated cell based IMPACT model was used to combine data on (1) numbers of patients in specific CHD groups; (2) the prescription rates for all standard CHD treatments in 2000; and (3) the effectiveness of these treatments, defined as survival benefit over a minimum of one year, from the largest and most recent meta-analyses or randomised controlled trials.[1] Cumulative benefit from polypharmacy in individual patients was estimated by the Mant and Hicks formula, where relative benefit = 1 − (1 − treatment A) × (1 − treatment B) × (1 − treatment C), etc. Compliance (concordance) for medical treatment was assumed to be 100% while patients were in hospital, 70% among symptomatic patients with angina or heart failure, and 50% among patients with hypertension or increased cholesterol. Uptake level was defined as prescription rate times adherence.[1]

Having estimated the actual reduction in CHD deaths in 2000, we then used the IMPACT model to examine the consequences of increasing the uptake (prescription) rates of specific medical treatments in each disease category to reach 80% of all eligible patients (100% was considered unrealistic).[2]

The corresponding calculation was performed for revascularisation, assuming that coronary artery bypass graft (CABG) surgery and percutaneous transluminal coronary angioplasty (PTCA) procedures in 2000 were increased by 80% (substantially more than the NSF targets).[2]

Multiway sensitivity analyses were then performed by using the analysis of extremes method.[1] Minimum and maximum mortality reductions were generated by 95% confidence intervals from meta-analyses for treatment efficacy and from minimum and maximum plausible values for patient numbers, treatment uptake, and adherence.[1]

All data sources, clinical definitions and *International classification of diseases* codes are detailed on our website (www.liv.ac.uk/PublicHealth/sc/bua/IMPACT-Model-Appendices.pdf).

RESULTS

In 2000, specific medical and surgical treatments in England and Wales were estimated to prevent or postpone about 25 805 deaths for at least one year (minimum estimate 17 110, maximum estimate 49 040) (fig 1, table 1). However, uptake (prescription) rates were generally mediocre. For instance, treatment rates among MI survivors averaged 56%

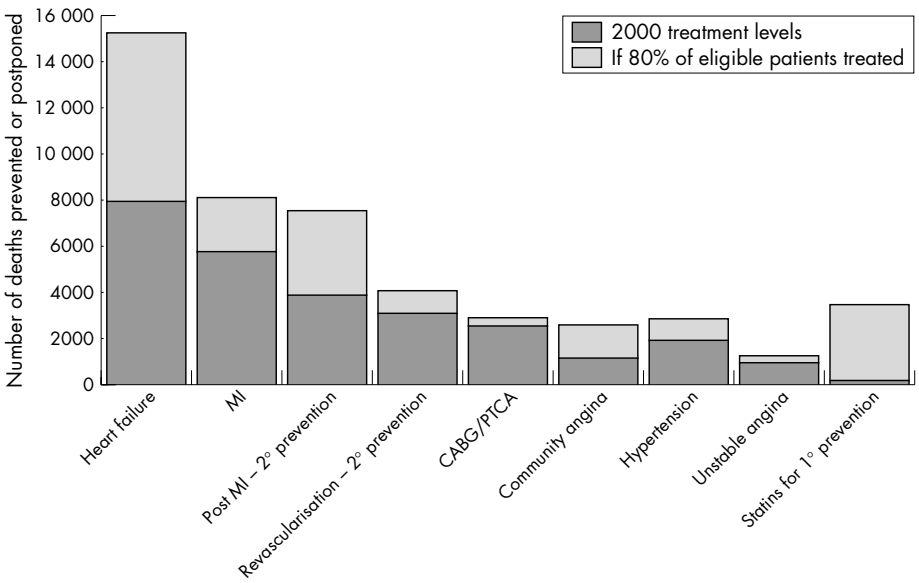

Figure 1 Estimated coronary heart disease mortality reductions in 2000 and potential gains if specific treatments reached 80% of eligible patients. CABG, coronary artery bypass graft; MI, myocardial infarction; PTCA, percutaneous transluminal coronary angioplasty.

Table 1 Coronary heart disease mortality reduction in England and Wales in 2000: effect of increasing treatment levels to reach 80% of eligible patients

Treatment	Eligible patients	Treatment Level in 2000	Treatment Efficacy (RRR)	Deaths prevented or postponed* In 2000	Gain if 80% treatment level	Total gain	Minimum estimate	Maximum estimate
Acute myocardial infarction	66195			5755	2370	11%	1329	3414
Community resuscitation	3045	0.046	0.11	799	381			
Hospital resuscitation	7280	0.99	0.21	1453	0			
Thrombolysis†		0.47	0.21	1321	50			
Aspirin		0.94	0.15	1949	0			
Primary angioplasty‡		0.01	0.28	38	1331			
β Blockers		0.04	0.04	21	197			
ACE inhibitors		0.19	0.07	172	409			
2° prevention after infarction	313380			3845	3695	18%	2741	4865
Aspirin		0.56	0.15	1242	67			
β Blockers		0.34	0.23	969	721			
ACE inhibitors		0.19	0.23	442	916			
Statins		0.25	0.29	459	644			
Warfarin§		0.04	0.15	100	250			
Rehabilitation		0.23	0.27	673	1057			
2° prevention after revascularisation	157840			3055	985	5%	561	1638
Aspirin		0.56	0.15	821	99			
β Blockers		0.35	0.23	568	148			
ACE inhibitors		0.22	0.23	349	268			
Statins		0.34	0.29	677	203			
Warfarin§		0.04	0.15	54	117			
Rehabilitation		0.35	0.27	586	152			
Angina revascularisation				2495	400	2%	270	560
CABG surgery	187415	1.00	0.31	1935	276		233	381
Angioplasty¶	112405	1.00	0.08	559	124		36	181
Unstable angina	67375			915	305	1%	224	419
Aspirin and heparin		0.59	0.27	467	165			
Aspirin alone		0.30	0.15	234	0			
Gp IIB/IIIA inhibitors and clopidogrel		0.48	0.09	211	141			
Chronic stable angina	2114670			1100	1475			
Aspirin		0.58	0.15	995	370	2%	234	790
Statins		0.07	0.29	105	1105	5%	958	1471
Heart failure in hospital	34690			4755	3350	16%	2178	6206
ACE inhibitors		0.62	0.26	1848	595			
β Blockers		0.31	0.37	1278	1044			
Spironolactone		0.10	0.30	348	990			
Aspirin		0.50	0.15	870	119			
Statins		0.21	0.29	412	700			
Community heart failure	242090			3210	3935	19%	1020	3048
ACE inhibitors		0.56	0.26	1536	34			
β Blockers		0.15	0.37	550	1595			
Spironolactone		0.10	0.30	206	965			
Aspirin		0.29	0.15	585	579			
Statins		0.17	0.36	333	763			
Hypertension treatments	13352870	0.53	0.11	1885	945	4%	438	1586
Statins for 1° prevention	7630760	0.03	0.29	145	3295	16%	1078	5493
Total				25805	20910	100%	11030	33495

*Deaths prevented were calculated by multiplying the age specific case fatality rate by the estimated relative risk reduction; †60% maximum uptake assumed; ‡40% maximum uptake assumed; §20% maximum uptake assumed for warfarin if 80% of patients were taking aspirin; ¶Assuming relative risk reduction (RRR) of 8%, equivalent to coronary artery bypass graft (CABG) surgery for two vessel disease.
ACE, angiotensin converting enzyme; Gp, glycoprotein.

for aspirin, 34% for β blockers, and 25% for statins. Similarly, patients with heart failure managed in the community averaged just 56% for angiotensin converting enzyme inhibitors, 17% for statins, and 15% for β blockers (fig 1, table 1). Increasing treatment rates to reach 80% of eligible patients could have prevented or postponed about 20 910 additional deaths (minimum estimate 11 030; maximum estimate 33 495). Of the 20 910 fewer deaths, 4680 (22%) would have resulted from increasing secondary prevention after an acute MI or revascularisation, and 7285 (35%) fewer deaths would have resulted from increases in heart failure treatments for patients in the community and in hospital (fig 1, table 1).

Extending primary prevention statin treatment to 80% of the 7.6 million ''healthy'' people with total cholesterol concentrations above 6.2 mmol/l would have prevented about 3295 deaths, representing 16% of the total gain, compared with 2370 (11%) fewer deaths from initial treatments for acute MI, 945 (4%) from treatments for hypertension, and 1475 (7%) from increases in aspirin and statins for patients with angina managed in the community (fig 1, table 1).

Only 400 (2%) additional deaths would have been prevented by an 80% increase in revascularisation procedures in 2000, and just 305 (1%) fewer deaths would have resulted from increased treatments for unstable angina. Irrespective of whether best, minimum, or maximum values were used in sensitivity analyses, the major potential gains consistently came from secondary prevention and heart failure, followed by statins and initial infarction treatments (fig 1, table 1).

DISCUSSION

In 2000, barely half the patients with cardiac disease actually received the appropriate treatment in England and Wales, much as elsewhere in Europe.[3] If just 80% of eligible patients with CHD had received the medical treatments indicated,

then over 20 000 extra deaths could have been prevented or postponed, almost doubling the mortality reduction actually achieved, consistent with older studies.[4]

Furthermore, almost two thirds of the total potential additional benefit would have come from focusing on secondary prevention and heart failure in primary care. Because absolute benefit is greater in older groups, they have the most to gain. The 2003 general medical services contract will now reward the identification of eligible patients and the creation of CHD registers in every general practice. Such incentives may substantially increase treatment uptakes. The increasing enthusiasm for chronic disease management programmes and nurse led primary care clinics focused on secondary prevention and cardiac rehabilitation should also help. The situation in 2005 may therefore be substantially better than that in 2000.

We generously assumed that CABG surgery and PTCA procedures in 2000 were increased by 80%. This was substantially more than the NSF had achieved by 2003 (some 6000 additional procedures over 1999 rates).[2] Relatively few deaths were prevented. However, revascularisation is being increasingly seen as a symptomatic intervention for improving quality of life, rather than simply for saving lives.[2]

All analytical models have limitations.[1] The IMPACT model was confined to CHD and did not explicitly consider patients with stroke or peripheral disease. Patients with diabetes were considered only in terms of their established CHD. The IMPACT model also assumed that efficacy, the mortality benefits reported in randomised controlled trials, can be generalised to effectiveness in unselected patients in clinical practice. A constant relative risk reduction, independent of the level of risk, was also assumed. Overestimation of the true treatment benefits therefore remains possible. Further explicit assumptions were required to cover deficiencies in the UK CHD data, which remain lamentably patchy and mixed.[5] Sensitivity analyses were therefore essential to examine the effect of varying these underlying assumptions and hence test the robustness of the model.[1] Maximum and minimum estimates were generally narrow. Furthermore, the relative contribution of each intervention remained remarkably consistent. This study focused on mortality reduction. Further research is now required on life years gained, symptom relief, quality of life, cost effectiveness, and the potential reduction in serious non-fatal events such as recurrent MI, stroke, or heart failure often leading to repeated hospitalisation.[2]

In conclusion, future national strategies should maximise the delivery of appropriate treatments to all eligible patients with CHD and prioritise secondary prevention and heart failure.

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