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The Development of a Simulation Model of the Treatment of Coronary Heart Disease

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Abstract. A discrete event simulation models the progress of patients who have had a coronary event, through their treatment pathways and subsequent coronary events. The main risk factors in the model are age, sex, history of previous events and the extent of the coronary vessel disease. The model parameters are based on data collected from epidemiological studies of incidence and prognosis, efficacy studies, national surveys and treatment audits. The simulation results were validated against different sources of data. The initial results show that increasing revascularisation has considerable implications for resource use but has little impact on patient mortality.

Keywords: simulation, coronary artery disease, treatment modelling

1. Introduction

Coronary heart disease is the leading cause of death and morbidity in most Western countries. In England, 1.4 million people suffer from angina while 300,000 people have heart attacks and 110,000 die of heart problems every year [12]. A government report [13] has set a target for the reduction in death rates from heart disease and strokes amongst people, aged under 65 years, of at least 33% by 2010. The National Service Framework [12] for coronary heart disease aims to improve the quality and consistency of services in a number of key areas by setting targets for the treatment and prevention of various aspects of coronary heart disease.

Policy makers need to understand the implications of different preventive and treatment strategies in terms of changes in mortality, morbidity and other benefits and costs in order to plan services. Models enable such information to be brought together and presented in an intelligible form. This paper describes the development of a model of patients who have had a coronary event, of their treatment, survival and of any subsequent coronary events. It is being developed in parallel with a model of prevention in the general population [1] and the two models will eventually be linked.

The challenge was to develop a comprehensive but simple model of a complex system such that it could be used for policy evaluation and planning. It was important for the model to follow patients through the whole treatment process because one part of the system influences others. For example, lives saved in one part of the system, will increase the demands for services in another. However, there are many different routes that can be taken through the system and various different treatments available. With the help of a multi-disciplinary team and a wide range of data, we have developed a working model for testing and evaluation. The purpose of this paper is to present the model, the data that were used and some preliminary results.

1.1. Clinical aspects of coronary heart disease

Angina is a chest pain associated with the narrowing of the coronary arteries which is typically induced by exercise. A heart attack or myocardial infarction occurs if there is a complete blockage due to thrombosis (blood clotting) in a narrowed coronary artery; in some cases this leads to sudden death (cardiac arrest). Often as a result of a myocardial infarction some of the heart muscle dies and this may lead to heart failure and subsequent ill health. Cardiac arrests are sometimes due to abnormal heart rhythms (arrhythmias) occurring without any evidence of a myocardial infarction.

Patients with angina are usually given drug treatment for symptom relief (e.g., nitrates, beta-blockers, calcium antagonists). Investigations which patients may be offered include electrocardiograms (ECGs), exercise tests and angiograms. An angiogram is a type of X-ray examination, designed to show up any narrowing of the coronary arteries, called stenoses. It is these stenoses which influence the patient's survival and which guide future treatment. A simple classification of disease is to count the number of vessels (from zero to three) that are more than 50% stenosed and to identify blockages in the key vessels: the left main stem and the left anterior descending arteries.

Some patients, following investigations, may be offered revascularisation to improve the flow of blood in the coronary arteries. The two alternatives are coronary artery bypass grafting and percutaneous transluminal coronary angioplasty. Treatment aims to give symptomatic relief and, if possible, to extend life expectancy. A bypass graft is a surgical procedure using veins or arteries to bypass narrowing or blockages in the coronary arteries. An angioplasty is a method for widening the artery by the temporary insertion of a catheter with a tiny balloon, which may be inflated to reduce the blockage. Many angioplasties use stents, which are tiny metal cages inserted into the artery to hold it open; these are left in place.

Unstable angina presents as angina pain which occurs with increasing frequency and severity and may persist when a patient is at rest. Patients with unstable angina are at high risk of a myocardial infarction or sudden death. They are admitted to hospital urgently and are treated with anti-clotting drugs such as heparin. Some are given emergency angiograms and revascularisation. Once patients stabilise, they are discharged from hospital.

Those with myocardial infarctions are at a high risk of death and are usually admitted to hospital as emergencies. Those who survive to admission to hospital are usually given thrombolysis (medication to disperse the clots) and aspirin, as quickly as possible. A few patients require emergency angiography and revascularisation but most are discharged without. Patients who have had a myocardial infarction retain an increased risk of further myocardial infarctions and death. Some develop stable angina and may receive non-urgent angiography and revascularisation.

Patients surviving coronary events may be offered secondary prevention. This includes the provision of various drugs, such as aspirin and cholesterol lowering statins, which reduce the subsequent risk of coronary events. Patients may also be given advice to change their lifestyle (for example, about smoking, diet and exercise).

1.2. Existing models

Weinstein et al. [34,35] developed a model which has been widely cited. It is a state-transition model that divides the population into homogeneous sub-populations defined by known prognostic factors. A new state or stratum needs to be created for each category, such as age or gender, and for each event, such as a treatment mode. This approach causes the number of strata to be very large (5400 in the Demographic-Epidemiological sub-model alone) and even so, the model does not include congestive heart failure or angioplasty. Bonneux et al. [3] developed a model which is similar to Weinstein's but includes heart failure as a prevalent state. The POHEM model [36] is a simulation approach which builds up individual life histories of health and interventions. It concentrates on the prevention part of the system. Mui's simulation model [24] for the Australian population is also concerned with prevention rather than treatment.

Bensley [2] has developed a population model of coronary events and revascularisation but it only follows patients through one event or one set of events and is not concerned with their long term survival. Davies [6] used a discrete event simulation model to evaluate revascularisation that was hospital, rather than population based. The team decided to develop a population based discrete event simulation model, *de novo*, which followed patients though the system until they reached the age of 85 years or died.

2. The model

Discrete event simulation follows individuals with different characteristics throughout the system. It has the advantage that it is possible to sample from any parametric distribution or histogram and resource use can be constrained to reflect actual practice. The simulation was written using the POST (Patient Oriented Simulation Technique) software with a Delphi interface [7]. The main advantage of using POST over other discrete event simulation software is that the entities in the simulation (in this case patients) can each have more than one future event in the calendar of future events, and can thus take part in more than one activity at once. If one activity interrupts another, then the interrupted activity can be found and de-scheduled quickly and easily, for example, death interrupts all activities.

The model provides a user-friendly interface so that it is easy to change patient numbers, characteristics, risks and transition rates between treatments. The simulation may be run with a pool of pre-existing patients or with no pre-existing patients, referred to in this paper as prevalent and incident populations, respectively.

Figure 1 shows the transitions between the disease states of angina, myocardial infarction, unstable angina and death and the possible interventions. Heart failure and arrhythmias are not explicitly included in the model at present.

New patients enter the model with stable angina, unstable angina or myocardial infarction, randomly, at a rate determined by the incidence rate of disease. Upon entry to the model, a patient entity is given the following attributes: age, gender, vessel disease, time before non-cardiac death and time to age 85. The risk of a patient dying from non-cardiac causes is independent of anything that may happen to them in the model. Those with stable or unstable angina have the additional attribute of the time to myocardial infarction or death and those with stable angina have a further attribute of the time to unstable angina. The sampled future times for myocardial infarction or death, and for unstable angina, depend on, age and vessel disease. The times are entered into the calendar of future events. Patients who progress to cardiac, non-cardiac death, or reach the age of 85, leave the model.

We assumed that the risks of unstable angina or of myocardial infarction or death, increase with age, with more severe vessel disease and with a history of previous myocardial infarctions. These different risks and any changes in risk due to revascularisation were independent of each other and were multiplied by the baseline risks to change the projections of myocardial infarction and death. Time was sampled from a

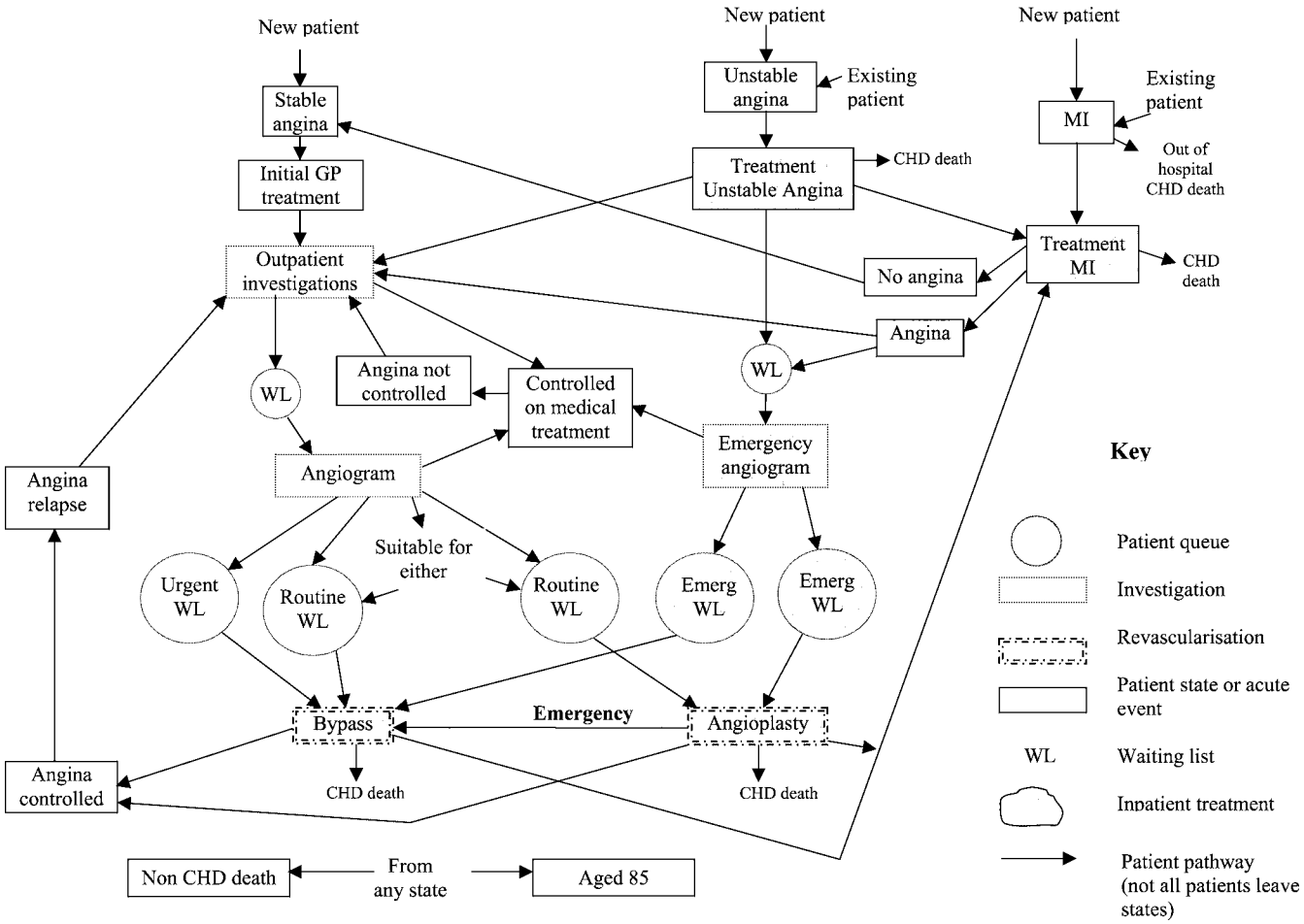

Figure 1. Flowchart of the pathways in the coronary heart disease treatment model. GP = general practitioner, MI = myocardial infarction, CHD = coronary heart disease, WL = waiting list.

time-updated Gompertz distribution, as used in the prevention model [1]. The hazard function is:

$$h(t) = r_1 r_2 r_2 \exp(at + b),$$

where t is age, $\exp(b)$ is the risk for individuals with one or two vessel disease at age zero (which is close to zero), a explains the increasing hazard with time, and r_1, r_2 and r_3 are the relative risks arising from vessel disease, prior history and interventions.

2.1. Stable angina

New angina patients are generated in the initial general practitioner (GP) state and are assumed to be treated medically, with some patients having their symptoms controlled and some remaining uncontrolled. Some transfer to the outpatient investigation state, either immediately or at a later date. Following the outpatient investigations, a proportion of patients join a waiting list (a queue in simulation terminology) for an angiogram and those who do not, go to the medical treatment state. After an angiogram, the extent of the vessel disease determines what happens to the patient next. The rules for doing this can be changed in the simulation input. Patients are allocated to a bypass graft, angioplasty, either of these or to the medical treatment state.

Some patients with the more severe disease, i.e., three vessel disease or two vessel disease with left anterior descending or left main stem artery blockages, join the urgent bypass graft queue. A patient suitable for either a bypass graft or an angioplasty is allocated to the shortest of the two routine queues. All of those waiting for treatment are assumed to receive medical treatment to control their angina until the operation or intervention takes place.

Either intervention gives rise to a small immediate risk of myocardial infarction or death. Some of those receiving angioplasties, require an immediate emergency bypass graft. For those with more severe vessel disease, bypass grafting changes the long term risk of myocardial infarction or death. The future events of myocardial infarction or death are thus rescheduled in the calendar. Patients progress from the angina controlled state to the angina relapse state (i.e., recurrence of symptoms), some immediately and some over the longer term. After angina relapse or the failure of medical treatment, patients return to the outpatient investigation state from where they may be returned to the angiogram queue and re-assessed for treatment. Patients are limited to two bypass grafts and six angioplasties.

Patients who have had a recent myocardial infarction or an unstable angina attack may enter or re-enter the stable angina system in the initial GP state, the outpatient investigation state or via an emergency angiogram. They are treated in the same way as new onset angina patients but have higher probabilities of a myocardial infarction or death.

2.2. Unstable angina

Patients entering the unstable angina state may either come from one of the stable angina states or may be new cases. They are admitted to hospital and a proportion of them will have an immediate myocardial infarction or will die. Those who respond to treatment and stabilise are discharged with stable angina and join the outpatient investigation state. Those who do not stabilise are put in a queue for an emergency angiogram, which in the simulation, takes place within two days. Following the angiogram, they may be put in the queue for an emergency bypass graft or angioplasty, depending on their vessel disease. Patients in the emergency queues are given priority over all other patients in other queues. On entering the stable angina system, patients are given a new sampled time for unstable angina.

2.3. Myocardial infarction

Patients who have a myocardial infarction or sudden death, some of which are due to arrhythmias, may be an existing patient or be new to the system. Of those who survive to be admitted to hospital, there is a further probability of dying in hospital. Of those who get angina whilst in hospital, some are referred for an emergency angiogram and subsequent emergency treatment and some are transferred to the outpatient investigation state in the stable angina system. Those who acquire angina later, enter the initial GP state. On discharge from hospital, patients are given new sampled times to unstable angina and to myocardial infarction or death.

2.4. Model output

Results are collected, each year and in total, on the following events: unstable angina admissions, myocardial infarctions, cardiac deaths in and out of hospital, non cardiac deaths, those reaching age 85, outpatient investigations, angiograms, bypass grafts and angioplasties. Results are collected on the number of patients in the following prevalent states: stable angina with no myocardial infarction, stable angina following a myocardial infarction and post myocardial infarction without angina. The stable angina states were subdivided into: the initial GP state, the angiogram and treatment queues, those controlled on medical treatment, those not controlled on medical treatment, the angina controlled state, and the angina relapse state (see figure 1) and the time spent in each of these sub-states are also collected.

3. Model data

Table 1 shows that parameter estimates were derived from a number of sources, including: searches of electronic databases (Medline, Embase, Cochrane), use of citations, access to national and local audit data and discussions with cardiac specialists. We attempted to collect parameter estimates stratified by the most important and feasible prognostic factors, such as, age, sex, past coronary heart disease history and vessel disease. In many case studies or audits, even these simple factors were not broken down sufficiently (especially for the elderly) and it was necessary to make assumptions and extrapolations from the data available. The data were based as far as possible on the most recent information available but, because treatment and survival change with time, they are likely to provide a less optimistic view of the future than will prove to be true.

3.1. Patient data

The proportion of the population in each age group was derived from the England and Wales census data. The non-cardiac deaths were sampled from probability distributions, conditional on a patient's initial age, derived from Office of National Statistics [27].

All incidence data were taken from the Bromley study [33] but it was scaled up because the standardised mortality ratio for coronary heart disease for Bromley was low (84), indicating a lower than average mortality rate compared to the national rate. Unfortunately, no Bromley data were available for the 75–84 year age band and so data had to be found from other sources and related to the Bromley data. The other sources were the Framingham data set [21] for stable angina and myocardial infarctions and the Hospital Episode Statistics dataset [17] for unstable angina.

The vessel disease makeup of all new patients is not generally known because only a selection of patients are referred for angiograms. The data for the stable angina and myocardial infarction patients were based on an old study, in which all angina patients received angiograms [25]. This, however, appears consistent with angiogram data from more recent studies. The vessels disease make-up for unstable angina patients was from the FRISC II study [29].

For those with stable or unstable angina, we needed an attribute of the time to myocardial infarction or death. Yusuf et al. [37] presented data on mortality related to vessel disease but little on myocardial infarction, whilst Pocock [28] had data on non-fatal myocardial infarction but provided no breakdown of mortality by vessel disease. We combined information from the two studies to provide a breakdown by vessel disease and history.

There appears to be no evidence, as yet, that an angioplasty improves long term prognosis whereas a bypass graft has been shown to improve prognosis for three vessel disease, or for two vessel disease with affected left anterior descending or left main stem arteries [37]. The changed risks were related to the baseline risks by relative risks.

Table 1
Summary of baseline data.

Variable	Value	Source
New onset angina incidence, % of age band per year	M[0.238, 0.548, 0.655, 0.300][a] F[0.098, 0.357, 0.333, 0.600]	[21,33]
New onset unstable angina incidence, % of age band per year	M[0.043, 0.080, 0.190, 0.210][a] F[0.029, 0.030, 0.039, 0.048]	[17,33]
New onset myocardial infarction incidence, % of age band per year	M[0.225, 0.359, 0.71, 1.01][a] F[0.03, 0.165, 0.236, 0.59]	[21,33]
Vessel distribution for new onset angina, % of patients	[52.0, 16.5, 16.0, 12.7, 2.8][b]	[25]
Vessel distribution for new onset unstable angina, % of patients	[14, 29, 26, 23, 8][b]	[29]
Vessel distribution for new onset myocardial infarction, % of patients	[5.0, 24.2, 28.2, 36.4, 6.2][b]	[25]
Annual probability of progression from angina to MI/sudden death[c] at age t, $\exp(at + b)$, one vessel disease	$a = 0.0458$, $b = -6.574$.	[4,28,37]
Relative risks of progression from angina to MI/sudden death (same risks used for progression to unstable angina)	[0.1, 1, 1, 1.78, 4.19][b] Previous MI = 2.45	[37]
Annual probability of progression from stable angina to unstable angina at age t, $\exp(at + b)$, one vessel disease	$a = 0.0458$, $b = -6.491$	[28]
Patients who have a MI whilst in hospital after unstable angina attack, %	3.9	[30]
Patients who die whilst in hospital after unstable angina attack, %	1.5	[30]
Patients who have emergency angiogram after unstable angina attack, %	10	[30]
Patients who die outside of hospital after an MI, %	[25, 29, 37, 50][a]	[26]
Patients who die in hospital after MI, given they survived to hospital, %	[4, 15, 25, 46][a]	[26]
Angiograms per million per year	2100	[8]
Bypass graft per million per year	450	[20]
Angioplasty per million per year	600	[8]
Operative mortality of bypass graft, %	2.3	[20]
Operative non fatal MI of bypass graft, %	5	[8]
Operative mortality of angioplasty, %	0.9	[20]
Operative non fatal MI of angioplasty, %	3.2	[20]
Emergency bypass graft rate after angioplasty, %	1.1	[8]
Benefit of bypass graft, relative survival ratios to MI/ sudden death (bypass graft versus no bypass graft)	[1, 1, 1, 1.56, 2.33][b]	[37]
Benefit of angioplasty, relative survival ratios to MI/ sudden death (angioplasty versus no angioplasty)	[1, 1, 1, 1, 1][b]	[28]
Referral rate from initial GP state to hospital outpatient investigations, % referred within 3 years	59	[14]
Referral rate from initial GP state to hospital outpatient investigations, % referred per year after 3 years	2.2	[5]
Referral rate from hospital outpatient investigations to angiogram, % referred	69	[8]

[a] These vectors refer to the four age ranges [45–54, 55–64, 65–74,75–84] and gender [M,F], where only one vector is given this is a combined figure for male and female.
[b] These vectors refer to vessel disease (VD) [0 VD, 1 VD, 2 VD, 3 VD, left main stem].
[c] MI = myocardial infarction.

Pocock [28] provided progression rates from stable angina to unstable angina. The in-hospital non-fatal myocardial infarction and mortality rates of unstable angina patients were taken from a national study of hospitalised unstable angina cases [30].

For those with myocardial infarction or sudden death (including arrhythmias), deaths, both out of hospital and inside hospital were derived from the UKHAS study (1998) [26]. No values were available for the age group 75–84. These ranges were estimated using the Nottingham Heart Attack Register (1997) [15].

3.2. Referral and interventions

The availability of angiograms, angioplasties and bypass grafts was based on rates published by the British Cardiovascular Intervention Society [8] for 1997 and British Society of Cardiothoracic Surgeons [20] for 1998, respectively.

The immediate transfer of stable angina patients in the initial GP treatment state to the outpatient investigation was based on Gill et al. [14] and the longer term transfer to outpatient investigations, Clarke et al. [5]. Those referred for angiogram were assumed to arrive at a rate that filled the capacity available under the guidelines above [8]. The criteria for treatment after an angiogram were based on guidelines as laid out in Scottish Intercollegiate Guidelines Network [32], de Bono et al. [9] and the European Society of Cardiology [19].

3.3. Assumptions

Although, there are many risk factors for the prognosis of coronary heart disease, the impact of these, such as the presence of diabetes, are not fully understood. Vessel disease was

chosen as an attribute because it is known to be an important prognostic factor and is a guide to the type of revascularisation that may be needed. Other factors will be considered in future work.

Doctors do not know the extent of a patient's vessel disease until after an angiogram. Because the simulation allocates vessel disease on patient entry, it can give the patient entities with severe vessel disease a correspondingly increased risk of a myocardial infarction or death, regardless of whether or when they receive an angiogram. In practice, referral for outpatient investigation and then to angiogram will be based on age, anginal symptoms, quality of life, exercise test results and patient preferences. In the results presented here, we have assumed that more patients with severe vessel disease are referred for outpatient investigations and for angiograms than those with less severe vessel disease [16,22]. This can be justified by a weak link between symptoms and vessel disease [18].

The entry to the myocardial infarction state, includes sudden deaths due to myocardial infarctions or arrhythmias. The probabilities of these sudden deaths, out of hospital or inside, were assumed to be independent of vessel disease and gender. The only other deaths due to coronary heart disease in the model occur after revascularisation or after an unstable angina admission (see figure 1). These deaths were also assumed to be independent of age, vessel disease and gender.

These assumptions will be reviewed and refined as the model is developed.

3.4. Difficulties in acquiring model data

The assumptions and the model input are dependent on epidemiological data. Much are of good quality and internally consistent but the limitations of this type of data should be recognised. Different methodologies may give rise to conflicting results. Self-reported prevalence may, for example, be very different from an analysis of medical records. A further problem is that trials may exclude particular categories of patients, such as those with complications, who are clearly of interest in the model. The trials cannot, therefore, be generalised to the whole population for use in a model.

Data may not be available for all the categories required by the model. It is then necessary to make extrapolations from data from other, less appropriate studies. Where data appear to be absent altogether, it may be difficult to structure the model as well as to run it. For example, it has not been possible to incorporate the role of exercise testing in increasing the likelihood of detecting serious vessel disease in combination with other variables. Even where good data appear to be available, the diagnostic coding may be inaccurate or inappropriate for our purpose.

We have attempted to overcome these difficulties and have validated the model against additional data sets.

4. Validation

The Health Survey for England 1994, 1998 [10,11] (HSE) surveyed representative cohorts of the population and, amongst many other questions, asked them to report whether they had ever been diagnosed with angina. The resulting calculations of angina prevalence were compared to output collected from the model, counting all those who had ever had angina during the simulation run. In comparing model output with data from the HSE (1994), the model predicts total angina prevalence to within 12% (see figure 2). It underestimates for men by 1% and women by 20%. In the HSE sample, there were about 15,000 individuals who had or who had had angina. In estimating prevalence for the whole population, the 95% confidence intervals for age bands ranged from $\pm 1\%$ for 45–54 year age group to about $\pm 3\%$ in the 75–84 year old age group. The model estimates fall within the confidence intervals of the HSE (1994) data for most of the age bands. The HSE (1998) shows an overall increase of about 16% over HSE (1994) for the 45–84 year age group. Consequently, compared to the more recent study, the model may be underestimated by a further 16% for men and women. The reason for this large apparent increase in the HSE results over the space of four years has yet to be explained.

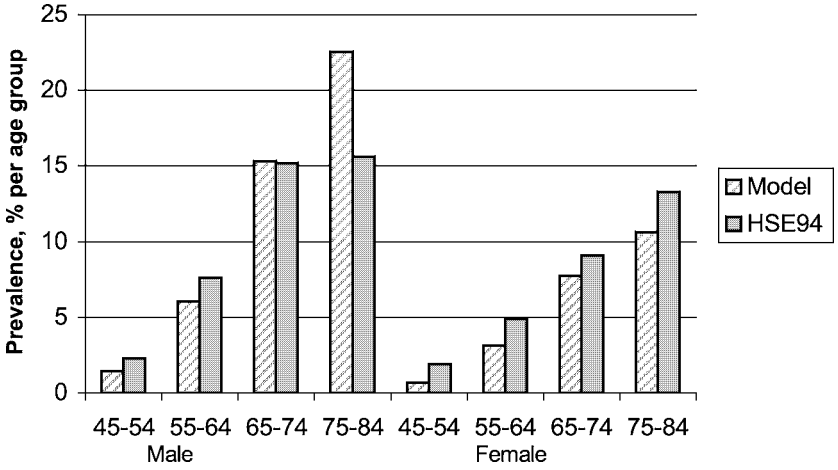

Figure 2. Angina prevalence for a population of 100,000; model estimates compared to the Health Survey for England 1994 (HSE 94).

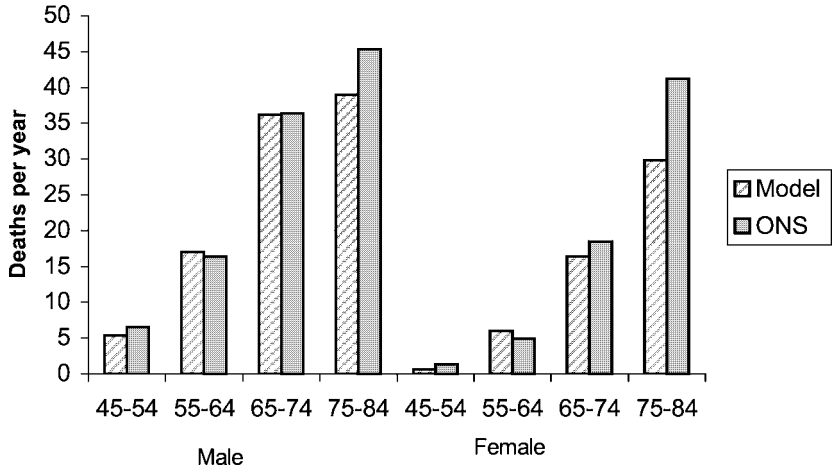

Figure 3. Cardiac deaths, ICD codes 410–414, per year for a population of 100,000; model estimates compared to the Office for National Statistics data (ONS).

The GP Morbidity data set [31] is a record of all patients who have visited their general practitioner within the previous year and gives information on those reporting with angina. The HSE also asked patients about the occurrence of angina within the previous year. A comparison between the studies indicates that the GP Morbidity Data set underestimates one-year angina prevalence by 28% for men and 40% for women as compared to the HSE (1994). Clearly there are problems with the definition of angina and its prevalence, but our model appears to be making conservative assumptions about the prevalence of disease compared to the HSE (1998) and generous assumptions compared to the GP Morbidity data.

Cardiac deaths in the model were validated against mortality data from the Office for National Statistics [27], based on death certificates. Compared to these data, the model underestimated deaths by an average of 12% (see figure 3). The majority of the underestimate is for the females and for the older age ranges, particularly the 75–84 age range (underestimated by 28%). Reported data are known, however, to be less reliable in the older age bands. The Framingham study [23] in the US, for example, estimated that between 12 and 30% of the diagnoses on death certificates in this age band had wrongly been attributed to heart disease. The variance between the model and the death certificate data may, therefore, be largely due to poor reporting of the causes of death on the death certificates.

5. Results

The simulation was run with a prevalent population, derived from the steady state output of previous simulation runs. In the example shown below, the simulation was run for 10 simulated years, using incidence values derived from a "typical" population of 250,000 people.

These results, using the data described above, were compared to a scenario whereby the revascularisation resources were increased. Referrals to the angiogram queue were increased by 50%, capacity for angiograms was increased by 50% from 2080 to 3120 per year per million population, and capacity for revascularisation for each of bypass grafts and angioplasties was doubled from 416 to 832 per year per million population. This scenario was designed to give an indication of the likely effect of exceeding the National Service Framework target of 750 per million for each type of revascularisation [12].

Table 2
Results from the simulation run, scaled up to a population of one million.

Results per year	Baseline	Increased revascularisation
Coronary heart disease deaths	1423	1404
Non fatal myocardial infarctions	1622	1620
Angiograms, per million	2085	3026
Bypass grafts, per million	416	828
Angioplasty, per million	416	828

Table 2 shows the interventions performed, the cardiac deaths and myocardial infarctions. The number of revascularisations performed per year per million increased to meet the capacity for angiograms, bypass grafts and angioplasties. What was particularly interesting was that the increased number of revascularisations was shown to have little effect on the myocardial infarction and death rates.

6. Discussion

We have developed a credible discrete event simulation model of the progress of patients after a coronary event. This model has been developed with a multi-disciplinary team including operational researchers, epidemiologists, health economists, public health doctors and cardiac experts. The paper concentrates on the activities and interventions of patients with stable angina but, nonetheless, individuals flow through the whole model, some progressing to an unstable angina episode or a myocardial infarction and some of them returning again to

stable angina. Changes in risk in one part of the model affect the activities and outcomes in another.

Discrete event simulation can incorporate constraints, such as the availability of angiograms, angioplasties and surgery, and can thus examine the effects of changing levels of resources and of different treatment policies on patient numbers and on adverse outcomes such as myocardial infarctions and deaths.

The collection of relevant and accurate data for modelling purposes is problematic. Nevertheless, validation against different datasets showed a reasonable fit for angina prevalence and cardiac death for age groups up to 75 years. The fit was, in all cases, better for men than for women. We hope that it will be possible to clarify the reasons for these differences.

The initial results show that increasing the number of revascularisations appears to have little effect on the myocardial infarction and death rates. This is not entirely surprising because bypass graft and angioplasty are treatments which are used mainly for symptomatic relief. Furthermore, the interventions themselves lead to an immediate risk of myocardial infarction or death.

We are continuing to develop the model by refining the parameters and, most importantly, by linking the output to costs to facilitate an analysis of the cost effectiveness of particular interventions. The model will include secondary prevention, such as the use of aspirin and anti-cholesterol agents. We aim to extend the model to include angina symptoms, heart failure and arrhythmias. The next step will be to link the treatment model with a prevention model which is being developed in parallel [1]. Using the combined model it will be possible to evaluate the effects of changes in prevention strategies in the general population, in secondary prevention and in different treatment interventions, on overall resource use, morbidity and mortality.

The model describes the transfer of patients between different aspects of heart disease and can evaluate the effects of changes in policy or improvements in risk factors on overall resource use, morbidity and mortality.

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References

[1] H. Babad, C. Sanderson, B. Naidoo, I. White and D. Wang, The development of a simulation model of primary prevention strategies for Coronary Heart Disease, Health Care Management Science 5 (2002) 269–274.

[2] D.C. Bensley, P.S. Watson and G.W. Morrison, Pathways of coronary care – a computer-simulation model of the potential for health gain, IMA Journal of Mathematics Applied in Medicine and Biology 12 (1995) 315–328.

[3] L. Bonneax, J.J. Barendregt, K. Meeter, G.J. Bonsel and P.J. van der Maas, Estimating clinical morbidity due to ischemic heart disease and congestive heart failure: The future rise of heart failure, American Journal of Public Health 84 (1994) 20–28.

[4] The Bypass Angioplasty Re-vascularisation Investigation (BARI) Investigators, Comparison of coronary bypass surgery with angioplasty in patients with multivessel disease, The New England Journal of Medicine 335 (1996) 217–223.

[5] K.W. Clarke, D. Gray and J.R. Hampton, Implication of prescriptions for nitrates: 7 year follow up of patients treated for angina in general practice, British Heart Journal 71 (1994) 38–40.

[6] R. Davies, Simulation for planning services for patients with coronary artery disease, European Journal of Operational Research 71 (1994) 323–332.

[7] H.T.O. Davies and R. Davies, Simulating health systems: Modelling problems and software solutions, European Journal of Operational Research 87 (1995) 35–44.

[8] M.A. de Belder, H.H. Gray, L.D.R. Smith and A.F.R. Rickards, *Cardiac Intervention Procedures in the United Kingdom in 1997* (British Cardiovascular Intervention Society, 1998).

[9] D.P. de Bono and A. Hopkins, The investigation and management of stable angina, Journal of the Royal College of Physicians of London 27 (1993) 267–273.

[10] Department of Health, *Health Survey for England 1994*, Findings, Vol. I (HMSO, 1996).

[11] Department of Health, *Health Survey for England 1998*, Findings, Vol. I, Survey Methodology and Documentation, Vol. II (The Stationary Office, 2000).

[12] Department of Health, *National Service Framework: Coronary Heart Disease, Modern Standards and Service Models* (The Stationary Office, London, 2000).

[13] Department of Health, *Saving Lives: Our Healthier Nation* (The Stationary Office, London, 1999).

[14] D. Gill, R. Mayou, M. Dawes and D. Mant, Presentation management and course of angina and suspected angina in primary care, Journal of Psychosomatic Research 46 (1999) 349–358.

[15] R.J. Goldberg, T. Young, D. Gray, A.M. Skene and J.R. Hampton, Inpatient deaths from acute myocardial infarction 1982–1992: Analysis of data in the Nottingham heart attack register, British Medical Journal 315 (1997) 159–164.

[16] R.A. Henderson, C.L. Raskino and J.R. Hampton, Variations in the use of coronary arteriography in the UK: The Rita trial coronary arteriogram register, Quarterly Journal of Medicine 88 (1995) 167–173.

[17] Hospital Episode Statistics, Department of Health, London (1998) (personal communication).

[18] H.N. Hultgren and P. Peduzzi, Relation of severity of symptoms to prognosis in stable angina pectoris, American Journal of Cardiology 54 (1984) 988–993.

[19] D.G. Julian, M.E. Bertrand, A. Hjalmarson, K. Fox, M.L. Simoons, L. Ceremuzynski, A. Maseri et al., Management of stable angina pectoris. Recommendations of the Task Force of the European Society of Cardiology, European Heart Journal 18 (1997) 394–413.

[20] B.E. Keogh and R. Kinsman, *National Audit Cardiac Surgical Database Report* (The Society of Cardiothoracic Surgeons of Great Britain and Ireland, London, 1998).

[21] D.J. Lerner and W.B. Kannel, Patterns of coronary heart disease morbidity and mortality in the sexes: A 26-year follow-up of the Framingham population, American Heart Journal 111 (1986) 383–390.

[22] R. Lim, I. Kreidieh, L. Dyke, J. Thomas and D.S. Dymond, Exercise testing without interruption of medication for refining the selection of mildly symptomatic patients for prognostic coronary angiography, British Heart Journal 71 (1994) 334–340.

[23] D.M. Lloyd-Jones, D.O. Martin, M.G. Larson and D. Levy, Accuracy of death certificates for coding coronary heart as the cause of death, Annals of Internal Medicine 129 (1998) 1020–1026.
[24] S. Mui, Projecting coronary heart disease incidence and cost in Australia: Results from the incidence module of the cardiovascular disease policy model, Australian and New Zealand Journal of Public Health 23 (1999) 11–19.
[25] Natural history of coronary artery disease, Bulletin of New York Academic Medicine 48 (1972) 1109–1125.
[26] R.M. Norris, *Sudden Cardiac Death and Acute Myocardial Infarction in Three British Health Districts*: The UK heart attack study (British Heart Foundation, London, 1998).
[27] Office for National Statistics, *Annual Abstract of Statistics, 1998* (The Stationary Office, London, 1999).
[28] S.J. Pocock, R.A. Henderson, A.F. Rickards, J.R. Hampton, S.B. King, C.W. Hamm et al., Meta-analysis of randomised trials comparing coronary angioplasty and bypass surgery, Lancet 346 (1995) 1184–1189.
[29] F. Ragmin, L. Wallentin, E. Swahn, F. Kontny, S. Husted, B. Lagerqvist and E. Stahle, Long-term low molecular mass heparin in unstable coronary artery disease: FRISC II prospective randomised multicentre study, Lancet 354 (1999) 701–707.
[30] Royal Brompton hospital, PRAIS UK dataset (1999) (personal communication).
[31] Royal College of General Practitioners, Morbidity statistics from general practice, 1991–1992 (OPCS, London, 1994).
[32] Scottish Intercollegiate Guidelines Network, *Coronary Revascularisation in the Management of Stable Angina Pectoris* (Royal College of Physicians, Edinburgh, 1998).
[33] S.J. Sutcliffe, A.K. Sutcliffe and D.A. Wood, The clinical incidence of fatal and non-fatal coronary heart disease in the population, Heart 8 (Supplement 1) (1999) (Abstract) (personal communication).
[34] J. Tsevat, M.C. Weinstein, L.W. Williams, A.N.A. Tosteson and P.A. Goldman, Expected gains in life expectancy from various coronary heart disease risk factor modifications, Circulation 83 (1991) 1194–1201.
[35] M.C. Weinstein, P.G. Coxson, L.W. Williams, T.M. Pass, W.B. Stason and L. Goldman, Forecasting coronary heart disease incidence mortality and cost: The Coronary Heart Disease Policy Model, American Journal of Public Health 77 (1987) 1417–1426.
[36] M.C. Wolfson, POHEM – a framework for understanding and modelling the health of human populations, WHO Statistics Quarterly 47 (1994) 157–175.
[37] S. Yusuf, D. Zucker, P. Peduzzi, L.D. Fisher, T. Takaro, J.W. Kennedy et al., Effect of coronary artery bypass graft surgery on survival: Overview of 10-year results from randomised trials by the Coronary Artery Bypass Graft Surgery Trialists Collaboration, Lancet 344 (1999) 563–570.