Holding Smokers Accountable for Heart Disease Costs

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Abstract This article discusses escalating health care costs in terms of their principal component, cardiovascular diseases. The role of cigarette smoking is highlighted throughout. A Smoker's Accountability Trust is proposed for the prevention of nonfatal myocardial infarction. It is demonstrated that this approach can contain costs by controlling the root causes of their acceleration. (Circulation. 1994;90:1029-1032.)

Key Words • hypertension • myocardial infarction • heart failure • smoking

Of the estimated $900 billion spent annually on health care, approximately one sixth is for cardiovascular diseases. Not only are cardiovascular diseases the largest component of the overall health care budget but, more importantly, they represent the fraction that is rising most rapidly. Unless corrective measures are taken soon, the rate of rise in the near future may be nonlinear.

Defining the Problem

The acceleration of cardiovascular disease costs can best be understood when it is viewed in terms of two factors, unmodifiable and modifiable. The unmodifiable factors are those related to aging. The US Census Bureau has recently projected the subset of the population 45 to 64 years old to grow to 80 million by the years 2010 to 2015. This represents an abrupt increase of 35%. The implications concerning cost containment relate principally to hypertension and myocardial infarction (MI).

It is well known that as the population ages, the prevalence of hypertension increases. There is a marked break point between those <45 and those >45 years old. A similar phenomenon is seen for MI for men 45 to 64 and women >65 years old. Inasmuch as approximately one third of the population will soon be >45 years old, it is important that we redouble our efforts to modify the factors described below.

The modifiable parts of the problem consist of hypertension, especially as it relates to MI, along with hyperlipidemia and cigarette smoking. Roughly 50% of all MIs occur in hypertensive people. The JNC-V report recently reclassified hypertension. Accordingly, it is estimated that approximately 50 million Americans are hypertensive. Of these, only 21% are thought to be optimally treated. Although this represents a slight improvement from previous observations, it leaves room for much improvement. That report also notes that at this time 30 million people are classified as high normal. This means that between 50 and 80 million people have the endothelial cell defect associated with hypertension that predisposes to MI. By the year 2015, these numbers will increase.

Forty percent of hypertensive people have coexisting hyperlipidemia. It is estimated that 22 million have both. This doubles the risk for MI. It was recently reported that of those diagnosed with cardiovascular disease, only 30% are on treatment for hyperlipidemia.

It is estimated that 25% to 30% of the American population smoke cigarettes. The Framingham Heart Study found that cigarettes are an independent predictor for the development of coronary heart disease. If hypertensive people with coexisting hyperlipidemia also smoke cigarettes, their risk for coronary heart disease doubles again.

Figs 1 and 2 provide insight as to the financial consequences of neglecting these opportunities for early intervention in MI for the past 40 years. Fig 1 shows trends in mortality for coronary heart disease, cerebrovascular disease, and hypertension (N. Haase, personal communication, 1992). Cardiovascular operations and procedures (defined as coronary arteriography, percutaneous transluminal coronary angioplasty, and coronary bypass operations) from 1975 through 1990 were added to this. After that, I superimposed mortality figures for congestive heart failure (CHF) for the total US population for the years 1965 through 1983.

The first observation that one should make from this is that stroke mortality has dropped considerably more than mortality for coronary heart disease. Not shown here, but very important, is the fact that stroke morbidity has also dropped. If you are a patient, this is good news. If you are a health economist, however, this is bad news. Patients who formerly suffered strokes are now having MIs instead. Relative to stroke, survivors of MI require "high-tech" care. This explains the rapid growth of procedures as of 1975.

Another point to be made by this figure concerns the near-exponential increase in fatal cases of CHF. These data were examined because the leading diagnosis-related group for Medicare admissions is CHF. Fatal CHF is only a small part of the picture. CHF prevalence is 100-fold greater.

The disturbing feature of this trend in CHF is the mystery that surrounds its pattern of growth. This is
because it was initially felt that the origin of heart failure was hypertension in 75% of the cases. Therefore, it was expected that with increased treatment of hypertension, the number of new cases of CHF would level off or decline in a fashion similar to that illustrated for stroke. In other words, if increasing the treatment of hypertension increases control of blood pressure and reduces stroke, the same control of blood pressure should have reduced the number of new cases of fatal and nonfatal CHF. This is because control of blood pressure is thought to prevent hypertensive cardiomyopathy.

This mystery may now be slowly being unraveled, as shown in Fig 2, which depicts the pathogenesis of congestive heart failure in terms of hypertension and coronary heart disease as attributable causes during 40 years of follow-up by the Framingham Study. The interesting feature here is the steep line upward, which shows cases of CHF in 1958 attributable to coronary heart disease rising from 22% all the way up to 67% in 1988. This is further defined by recent reexamination of this group as depicted on the left side of the figure. As can be seen, during the second half there was a marked increase in the number of cases of CHF due to MI, especially in women in the age group 60 to 69 years, in which the number doubled. The failure to significantly impact on MI relative to stroke over a comparable period of time in which hypertension was treated principally with diuretics has implications for diuretics as a possible cause of this shift in pathogenesis. Other possible reasons to explain this pathogenetic shift will be discussed in the section that follows. Regardless, fatal MI is decreasing, nonfatal MI is increasing, and total MI is unchanged. It is thought that today’s new cases of CHF arise principally from an ever-expanding pool of survivors of MI. This would explain why CHF has behaved in this paradoxical fashion. The longer we neglect opportunities to prevent nonfatal MI, the more CHF hospitalizations will increase in the future.

The implications for future cost containment should these trends continue can be seen by projecting costs for the past 5 years to 2015 using linear growth assumptions. When this is done, the result is $255 billion per year in 2015 for cardiovascular diseases alone. This does
not take into account previously discussed projections for population growth for those \( \geq 45 \) years old.

**How Did We Get Into This Predicament?**

Four explanations are generally given to explain why hypertensive therapy prevents stroke but not MI. These are reviewed in detail elsewhere.\(^{25,26}\) I would like to focus on one of these, discussed earlier, regarding diuretics and why they may be changing the pathogenesis of heart failure, because it is well known that diuretics alter lipids and increase insulin resistance.\(^{27}\) Insulin resistance states are thought to be associated with increased levels of plasminogen activator inhibitor type 1 (PAI-1).\(^{28,29}\) Increased levels of PAI-1 have been found to be associated with increased risk for MI and coronary mortality.\(^{30-32}\) On this basis, one could argue that diuretics should be first-line agents only in the elderly when\(^{25}\) and only when\(^{18}\) their safety and efficacy have been demonstrated, since the elderly do not live long enough to be exposed to the risk from diuretics seen by 25-year-olds. This is especially the case inasmuch as other drugs useful in the treatment of hypertension are not detrimental to lipids or PAI-1 or may be beneficial,\(^{33}\) especially in patients whose retreatment risk is increased due to smoking.

Which of these theories best explains why hypertensive therapy decreases stroke without significantly impacting on MI in the nonelderly? This is difficult to determine. Probably a combination is involved, and diuretics are not solely to blame. Failure to control cigarette smoking has to be another consideration, since cigarettes increase risk for MI more than for stroke.\(^{34}\)

**Solving the Problem**

As a remedy for the previously described problem, I recently proposed a new approach to legislation involving cigarette taxation. This is based on the experience of the past 10 years in Canada. It is an attempt to improve on their initially positive results by targeting revenues from cigarette taxation to the prevention of nonfatal MI.

Before I describe the key features of this proposal, a bit of background on the Canadian experience is in order. Beginning around 1980, the taxes on cigarettes were gradually raised so that by 1991, $3.30 of the price of a pack\(^{35}\) was taxes. Over that same time period, the percentage of Canadians 15 to 19 years old who smoked dropped from 43% to 17%.\(^{35}\) The Canadians estimate a saving in lives of between 670,000 and 1.4 million.\(^{35}\) In addition, the overall number of smokers dropped by 50%.\(^{35}\) Of interest, a recent survey of fatal CHF in Canada for the years 1970 to 1989 revealed a decline beginning around 1980 to 1981 and extending to 1989.\(^{36}\)

During the years 1981 to 1991, tax revenues from cigarette sales rose from $2 billion to $7.2 billion despite smuggling in the border states.\(^{35}\) This represented a net gain of $5.2 billion for a tax hike of $3.30 per pack over 10 years. Most importantly, with gradual tax increases, there were no falloffs in revenues secondary to the increase in prices due to taxation, as the Tobacco Institute propagandizes. If one considers that $5.2 billion/y in cigarette revenues are now generated from 26 million Canadians, that number for 260 million Americans could grow to 52 billion new dollars per year.

If we were to target this money to the 79% of hypertensive people who are undiagnosed or suboptimally treated along with the 70% of patients with hyperlipidemia and coronary disease who are untreated, we could not only reduce nonfatal MI by reducing cigarette consumption but also aggressively intervene in a preventive capacity through the Public Health Service. We would not have to use thiazide diuretics to treat 25-year-olds with hypertension for 40 years for reasons of cost. We could also fund research that would specifically target the problem of nonfatal MI, especially as it relates to the treatment of hypertension. With the large amount of money left over, we could ask those individuals who choose to smoke despite warnings to the contrary to pay for their bypass and other revascularization procedures on the “layaway plan.” This is especially for teenagers who insist on smoking and who are at highest risk for future events.\(^{34}\) This would obviate the necessity of nonsmokers’ having to subsidize the indiscretions of smokers through global budgets.

To accomplish these objectives, I have proposed that revenues from cigarette taxation be placed in a specific trust titled “The Smoker’s Accountability Trust.”\(^{37}\) A full copy of the transcript of this testimony given on November 19, 1993, to the Ways and Means Committee can be obtained from this office. This transcript also lists the many advantages of this approach. Two that I would like to highlight here are as follows: (1) It (The Smoker’s Accountability Trust) contains costs regardless of whether patients quit or continue to smoke, ie, establishes a servomechanism with a negative feedback loop; and (2) it represents the first health care reform proposal that addresses the principal root causes responsible for cost acceleration.

In summary, for the past 10 to 15 years, serious efforts at containing health care costs have been largely unsuccessful. This is because the problem has been incompletely defined in narrow financial terms. The underlying root causes have been given secondary consideration at best or no consideration at all. The net effect has been like “rearranging deck chairs on the Titanic.” Any approach at health care reform that fails to address the root causes described here runs the risk of failure secondary to “volume overload.” The Smoker’s Accountability Trust can contain costs by controlling the root causes responsible for their acceleration.

**References**

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