Prevention. How much harm? How much benefit? 1. Influence of reporting methods on perception of benefits

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Abstract • Résumé

Before a physician or a patient can decide whether a preventive program is worth while, each must understand the nature and degree of its benefits and the frequency and magnitude of its adverse effects. Preventive interventions can be divided into two major categories: those with infrequent or minor adverse effects and those with adverse effects that are frequent or serious. Accident prevention, avoidance of high-risk behaviour and healthy lifestyle choices such as breast-feeding and moderate exercise are associated with few adverse consequences. By contrast, screening populations for disease, risk classification for the purpose of selective preventive interventions, dietary intervention and prophylactic drug treatment may lead to more frequent and serious adverse effects. When assessing whether the benefits of a preventive intervention outweigh the harm, one must be aware that the methods used to report benefits of clinical trials may distort the reader's perception of their magnitude. The relative reduction of morbidity or mortality rate often grossly exaggerates benefits and should never be used as a basis for clinical decision making. More realistic ways of recording benefits are the absolute reduction of morbidity or mortality rate, the number of patients that need to be treated to avoid one adverse event, and the total cohort mortality rate.

Pour pouvoir se prononcer sur la valeur d'un programme de prévention, un médecin ou un patient doit comprendre la nature et l'ampleur de ses avantages, ainsi que la fréquence et l'ordre de grandeur de ses effets indésirables. Il y a deux grandes catégories d'interventions préventives : celles qui ont des effets indésirables peu fréquents ou mineurs et celles dont les effets indésirables sont fréquents ou sérieux. La prévention des accidents, l'évitement des comportements à risque élevé et le choix de modes de vie sains comme l'allaitement et l'exercice modéré ont peu de répercussions indésirables. Par ailleurs, le dépistage des maladies dans la population, la classification des risques aux fins d'interventions préventives sélectives, les interventions alimentaires et la prophylaxie médicamenteuse peuvent avoir des effets indésirables plus fréquents et plus sérieux. Lorsqu'il faut évaluer si les avantages d'une intervention de prévention l'emportent sur les préjudices, il faut savoir que les méthodes de production de rapports sur les avantages d'études cliniques peuvent déformer la perception que le lecteur a de leur ordre de grandeur. La réduction relative du taux de morbidité ou de mortalité exagère souvent et considérablement les avantages, et il ne faut jamais fonder sur cette réduction la prise de décisions cliniques. La réduction absolue du taux de morbidité ou de mortalité, le nombre de patients qu'il faut traiter pour éviter un événement indésirable et le taux de mortalité de la cohorte totale sont des façons plus réalistes de faire état des avantages.

Canadians devote an enormous amount of time, energy and resources to disease prevention. Patients and physicians are inundated with advice on how to live healthier, longer lives. How legitimate is this advice? Which preventive interventions have actually been proven to work? What is the magnitude of benefit? What is the cost of prevention in terms of psychological, social and physical effects? Is informed consent obtained for participation in preventive programs? This series of articles will present a framework for answering these questions. This article deals with how reporting methods influence physicians' perceptions of benefits; the second will discuss 10 other ways in which the degree of benefits may be misconstrued; the third will deal with the harm

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Subsequent articles in this series will appear in the June 15, July 15 and Aug. 15 issues of CMAJ.

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that preventive interventions may cause; and the last will discuss the ethics of prevention and informed consent.

Determining the balance between the benefits and harm of preventive programs

Before a physician undertakes or a patient accepts a specific preventive intervention, he or she should be able to answer the following four questions.

- Is there any proven benefit from the intervention?
- If there is, how great is it?
- Are there any adverse effects of the intervention?
- If there are, what are they, how serious are they and how often do they occur?

If the answers to these questions are available, a rational decision can usually be made. If the benefits are great and the adverse effects minimal, most would support the intervention. Most would probably also support a preventive intervention even if its benefits are small or unproven, provided there are no significant adverse effects. But only a fool would participate in a program in which the benefits are unproven or small but the adverse effects are proven and large. The most difficult decision arises when the benefits of a program are proven and great but the adverse effects are also proven and serious.

Types of preventive interventions

Many types of preventive interventions are now used. For each, it is important to know how great the benefits are and how frequent and serious the adverse effects are. Any preventive measure, no matter how innocuous it seems, may have adverse effects. People have been injured by seat belts.[1] Contact dermatitis of the penile skin[2] or of the vulvar and vaginal mucosa[3] has resulted from exposure to latex condoms. People in poor physical condition have died of myocardial infarction during vigorous exercise.[4,5] And, no doubt, someone wearing a broad-brimmed hat to prevent the sun from damaging his or her skin will fail to see a rock slide and be killed as a result. However, for these and many other preventive measures, such events are rare. As a result, there is little controversy about promoting them, even if proof of benefit is not always conclusive. Types of programs that most people would put into this category are accident prevention, avoidance of high-risk behavior and healthy lifestyle choices such as breast-feeding and regular moderate exercise (Table 1).

Routine childhood and adult vaccinations may be included in a second category in which both the benefits and the infrequent serious adverse effects are generally well known and well publicized.[6-9]

A third category of preventive interventions includes those in which the detrimental effects, even when well documented, are often poorly publicized, and the benefits are not always as great as the proponents claim. Examples of such interventions are screening or case-finding, risk classification for selective preventive interventions, dietary interventions and prophylactic prescription of drugs (Table 2). This series focuses exclusively on this category of preventive programs.

Categorizing a preventive program as having more frequent or more serious adverse effects than other types of preventive programs does not imply that that the program is not worth while. It simply means that the physician and the patient should ensure that they are aware of all of the benefits as well as all of the adverse effects before deciding whether to participate.

Table 1: Preventive programs with proven or uncertain benefits but with minimal adverse consequences

Type of program	Example
Accident prevention	Using seat belts Using infant car seats Using bicycle and motorcycle helmets "Baby proofing" a home to prevent injuries in infants Lowering water-heater temperature to prevent scalding
Avoidance of high-risk behaviour	Practising safe sex Drinking moderately Avoiding drinking and driving Avoiding or stopping smoking Avoiding the use of illicit drugs Limiting exposure to the sun
Healthy lifestyle choices	Breast-feeding Regular moderate exercise

Table 2: Preventive programs with proven or uncertain benefits but poorly publicized adverse consequences

Type of program	Example
Screening or case-finding to detect early disease	Digital rectal examination[10] Screening mammography[11] Papanicolaou smears[12] Testing stools for occult blood[13-15] Prostate-specific antigen testing[16] Cholesterol-level testing[17-20] Maternal serum screening for markers of birth defects[21,22]
Risk classification for selective preventive interventions	Identifying women as being at a high risk of complications of pregnancy[23,24] Identifying patients as being at a high risk of cardiovascular disease[25]
Dietary intervention	Low-energy, low-fat or high-fibre diets[26-30] Prescription of antioxidants[31,32]
Prophylactic drug treatment	Acetylsalicylic acid[33-36] Cholesterol-lowering drugs[17,18] Hormone replacement therapy[37,38]

METHODS OF REPORTING THE BENEFICIAL EFFECTS OF PREVENTIVE INTERVENTIONS

To decide whether the benefits of a preventive program outweigh its disadvantages, it is necessary to have a firm grasp of the magnitude of the benefits. This is often difficult to determine because of the way beneficial results are reported.

The four standard methods of reporting the beneficial results of a screening or therapeutic program are the following.[39]

- Relative reduction of morbidity or mortality rate.
- Absolute reduction of morbidity or mortality rate.
- Number of patients that need to be treated for 1 year to prevent one adverse event.
- Total cohort mortality rate.

RELATIVE REDUCTION OF MORBIDITY OR MORTALITY RATE

The most common and deceptive method of reporting the benefits of preventive programs is the relative reduction rate. For example, a cholesterol-lowering program may report a 40% lower rate of myocardial infarctions in the group that underwent treatment, or a screening mammography program may show a 30% lower rate of death in the screened population. Such figures, even if statistically significant, may or may not be clinically significant. This is best illustrated by two hypothetical examples.

Suppose that a drug is being assessed for its ability to prevent myocardial infarctions in two different populations of patients. In the first hypothetical study the drug is given daily for 5 years to 1000 men who are known to be at a high risk of having myocardial infarction, and a comparable group of 1000 men is given a placebo. At the end of 5 years, 500 myocardial infarctions have occurred in the placebo group and 250 in the group receiving treatment. Taking the drug has clearly resulted in a 50% decrease in the rate of myocardial infarctions. This is the relative reduction rate. Furthermore, since the drug has prevented 250 infarctions during 5 years in the group that was treated, the result appears to be clinically significant in a high-risk population of patients.

The second hypothetical study involves men with no known risk of having a myocardial infarction. As in the first study, 1000 men are given the drug daily for 5 years, and a comparable group is given a placebo. At the end of 5 years, four myocardial infarctions have occurred in the placebo group and two in the group receiving treatment. As in the first study, the relative reduction rate of myocardial infarction is 50%, which is a pretty impressive figure. However, since only two myocardial infarctions have actually been prevented over 5 years, this small benefit may well be outweighed by the adverse effects of the treatment. If so, the results would not be clinically significant.

The first lesson to be drawn from these examples is that relative reduction of morbidity or mortality rates tell us nothing about the clinical usefulness of the intervention. Also, the clinical usefulness of a preventive intervention is greater in a population with a high prevalence of disease than in one with a low prevalence.

ABSOLUTE REDUCTION OF MORBIDITY OR MORTALITY RATE

The absolute reduction rate provides a much better idea than the relative reduction rate of the real size of any true beneficial effect. In the first example, 250 myocardial infarctions were prevented among the 1000 men who were treated. Since a quarter of the men treated avoided myocardial infarctions, the absolute reduction rate is 25%. By contrast, two infarctions were prevented among the 1000 men treated in the second example, so the absolute reduction rate in that case is only 0.2%. The drug is clearly clinically useful in the first case, but it is probably not in the second.

NUMBER NEEDED TO TREAT

A third reporting method is the number of patients one would need to treat for 1 year in order to prevent one adverse outcome. In the first example, 20 men would have to be treated for 1 year to prevent one myocardial infarction, whereas in the second example 2500 men would have to be treated for 1 year to achieve the same effect. The way these figures are derived can be illustrated by the first example.

Over a 5-year period, 250 myocardial infarctions were prevented by treating 1000 men. Therefore, over a 1-year period, the number of infarctions prevented would be one fifth of 250, or 50. The number of men who would need to be treated to prevent one infarction would be 1000 divided by 50, which equals 20.

When presented this way, it seems likely that the drug is clinically useful in the high-risk population but not in the low-risk population.

TOTAL COHORT MORTALITY RATE

The fourth method of reporting beneficial effects is to give the total cohort mortality rate in the group receiving treatment and in the control group. This is a vital piece of information because, even if the drug tested decreases the rate of death from one disease such as myocardial infarction, but does not decrease the overall death rate, its value is questionable at best. One explana-

tion for such a discrepancy is that adverse effects of the drug have resulted in an increased rate of death from other causes.

Importance of the reporting method

Although this discussion may seem academic, numerous studies show that it is not.[40–45] Physicians are very much influenced by how results are reported. For example, studies conducted in Switzerland[43] and Italy[44] showed that physicians were far more likely to prescribe cholesterol-lowering drugs when they were presented with relative morbidity and mortality rates than they were when presented with the absolute rates or the number of patients that needed to be treated to prevent one adverse outcome.

To illustrate further the importance to clinicians and patients of the way benefits are reported, I present examples culled from the literature on screening programs for breast cancer and colon cancer and on cholesterol-lowering programs.

Breast-cancer screening programs

In 1993, Liddell[46] reviewed the results of five major breast-cancer screening programs involving women over 50 years of age. For each study, she compared the relative reduction of mortality rate to the number of patients that needed to be screened to save one life (Table 3). The relative reduction rates tend to make one enthusiastic about these programs; expressed as the number of patients that need to be screened to save one life, the inference is somewhat discouraging.

Harris and Leininger[52] compared the relative reduction of mortality rate with the estimated absolute reduction rate in seven randomized controlled trials of breast-cancer screening. They found that the overall relative reduction of mortality rate varied between 15% and 30%. In absolute terms, this meant that two to four deaths from breast cancer were prevented among 1000 women screened regularly over 10 years, yielding an absolute reduction of mortality rate of 0.2% to 0.4%. Of these seven studies, proof of effectiveness reached statistical significance in only three. Harris and Leininger also calculated that, among women 50 to 70 years of age, the number of mammograms required to save one life was between 1700 and 5000.

Table 3: Two methods of reporting results of five trials of breast-cancer screening*

Study	Relative reduction of mortality, %	No. of patients that need to be screened to save one life
Health Insurance Plan of Greater New York[47]	32.0	> 3 000
Two counties[48]	40.0	> 5 000
Edinburgh[49]	20.0	> 12 000
Malmo[50]	21.0	> 41 000
Canadian National Breast Screening Study[51]	2.5	> 100 000

*Adapted from Liddell.[46]

Screening for occult blood in the stool to prevent colon cancer

Studies of colon-cancer detection through screening for occult blood in the stool also show the striking difference between relative and absolute reduction of mortality rates. In 1993 Mandel and associates[13] were the first to report a statistically significant decrease in deaths from colon cancer as the result of annual hemoccult testing. In a cohort of patients who submitted six stool specimens annually for 13 years, the rate of death from colon cancer was 33% lower than that in controls; however, this translated into an absolute reduction of mortality rate of 0.3%.[13]

Cholesterol-lowering programs

As all physicians know, lipid-lowering programs have been shown to reduce the incidence of heart attacks. However, scrutiny of the reported results shows how large the benefit really is.

The Lipid Research Clinics Coronary Primary Prevention Trial (LRC trial)[53] was widely reported to show a reduction in the incidence of fatal myocardial infarctions of 24% (a relative reduction of mortality rate). The study involved 3806 middle-aged men who had hypercholesterolemia but had no known coronary artery disease. They were treated for 7 to 10 years with either cholestyramine or a placebo. Among the 1906 men who received cholestyramine, there were 30 deaths from coronary artery disease, whereas in the placebo group of 1900 men, there were 38 such deaths. This difference of eight deaths works out to the reported relative reduction in death from coronary artery disease of 24%; in absolute terms, 1.6% of men in the treated cohort died of myocardial infarction, as did 2.0% of those in the placebo group. The difference of 0.4% is the absolute reduction of mortality rate. The number of men who had to be treated to obtain this result was large. Over the 7- to 10-year period there were eight fewer deaths from coronary artery disease in the group taking cholestyramine, which is approximately one death prevented each year. This means that approximately 1900

men had to be treated for 1 year to prevent one death. Furthermore, there was no decrease in the total cohort mortality rate in the group taking the drug.

The Helsinki Heart Study was hailed by many because it was reported to show a reduction in the incidence of cardiac events of 34%.[54] Like the LRC trial, it involved middle-aged men with hypercholesterolemia but with no known coronary artery disease. Over a 5-year period, a cohort of 2051 men received gemfibrozil and a control cohort of 2030 men received a placebo. At the conclusion of the study, 56 men taking gemfibrozil had had a cardiac event, as had 84 men in the control group, yielding the widely reported relative reduction rate of 34%. However, the absolute rate of difference in cardiac events was 1.3%. If one analyses the report further and looks for the effect of gemfibrozil on deaths from cardiac events, the absolute difference was small; there were 6 such deaths in the group receiving treatment and 10 in the control group, for a relative reduction rate of 40% but an absolute reduction rate of only 0.2%.

The Helsinki Heart Study data may also be presented on the basis of how many men had to be treated to prevent a specific number of adverse cardiac events. Since 28 adverse events were prevented by treating 2051 men for 5 years, approximately 5 events were prevented each year. Therefore, in 1 year, 5 men benefitted from taking gemfibrozil and 2046 did not. Another way of putting this is that the number of men requiring treatment for 1 year to prevent one cardiac event was 410. If one looks only at deaths from cardiac events, 2460 men would have had to take gemfibrozil for 1 year in order to prevent one such death. Even this small benefit is of questionable significance because there was no decrease in the total cohort mortality rate.

The most recent report on the efficacy of treating men who have no symptoms of heart disease with cholesterol-lowering drugs is the pravastatin study conducted in Scotland.[55] In this study approximately 3300 men received 40 mg of pravastatin daily. Not only was there a reduction in nonfatal and fatal myocardial infarctions in the group receiving treatment, but, for the first time, there was a documented decrease in the total cohort mortality rate in a group of men with hypercholesterolemia but no symptoms who were taking cholesterol-lowering drugs. The figures presented were a relative reduction of 31% in the rate of definite nonfatal and fatal myocardial infarctions and a 22% relative reduction in the rate of death from all causes in the cohort receiving treatment. However, a careful reading of the text reveals that the absolute reduction of definite nonfatal and fatal myocardial infarctions was 2.4% and that more than 200 men had to be treated for 1 year to prevent one such adverse event. The absolute reduction of mortality rate in the total cohort was 0.9%, and the treatment of 555 men for 1 year was required to prevent one death.

The absolute reduction rates from these studies, as well as the number of patients that need to be treated to prevent one adverse event, reinforce Rose's[56] statement of a decade ago that the expectation of benefit for any one person participating in a preventive program is low and that, therefore, the benefit may easily be outweighed by the risks involved.

Conclusion

Attempting to measure the benefits of preventive programs is often difficult, particularly if the reporting methods used are relative reduction of morbidity or mortality rates. Even when absolute reduction of morbidity or mortality rates, or numbers of patients that need to be treated, are given, the clinical importance of the benefits may still be unclear. Some of the reasons for this will be reviewed in the next article in this series (to appear in the June 15 issue), which will discuss 10 pitfalls in describing benefits (other than the reporting methods discussed in this article) that may confuse readers of the literature on prevention.

References

1. Hendey GW, Votey SR: Injuries in restrained motor vehicle accident victims. *Ann Emerg Med* 1994; 24: 77–84
2. Shenot P, Rivas DA, Kalman DD et al: Latex allergy manifested in urological surgery and care of adult spinal cord injured patients. *Arch Phys Med Rehabil* 1994; 75: 1263–1265
3. Kint B, Degreef G, Dooms-Goossens A: Combined allergy to human seminal plasma and latex: case report and review of the literature. *Contact Dermatitis* 1994; 30: 7–11
4. Mittleman MA, Maclure M, Tofler GH et al: Triggering of acute myocardial infarction by heavy physical exertion — protection against triggering by regular exertion. Determinants of Myocardial Infarction Onset Study investigators. *N Engl J Med* 1993; 329: 1677–1683
5. Willich SN, Lewis M, Löwel H et al: Physical exertion as a trigger of acute myocardial infarction. Triggers and Mechanisms of Myocardial Infarction Study Group. *N Engl J Med* 1993; 329: 1684–1690
6. Al-Mazrou A, Scheifele DW, Soong T et al: Comparison of adverse reactions to whole-virion and split-virion influenza vaccines in hospital personnel. *Can Med Assoc J* 1991; 145: 213–218
7. Miller E, Goldacre M, Pugh S et al: Risk of aseptic meningitis after measles, mumps, and rubella vaccine in UK children. *Lancet* 1993; 341: 1541
8. Stratton KR, Howe CJ, Johnston RB Jr: Adverse events associated with childhood vaccines other than pertussis and rubella. Summary of a report from the Institute of Medicine. *JAMA* 1994; 271: 1602–1605
9. Farrington P, Pugh S, Colville A et al: A new method for ac-

tive surveillance of adverse events from diphtheria/tetanus/pertussis and measles/mumps/rubella vaccines. *Lancet* 1995; 345: 567–569
10. Scardino PR, Weaver R, Hudson MA: Early detection of prostate cancer. *Hum Pathol* 1992; 23: 211–222
11. Lerman C, Trock B, Rimer BK et al: Psychological and behavioral implications of abnormal mammograms. *Ann Intern Med* 1991; 114: 657–661
12. Mayeaux EJ, Harper MB, Abreo F et al: A comparison of the reliability of repeat cervical smears and colposcopy in patients with abnormal cervical cytology. *J Fam Pract* 1995; 40: 57–62
13. Mandel JS, Bond JH, Church TR et al: Reducing mortality from colorectal cancer by screening for fecal occult blood. *N Engl J Med* 1993; 328: 1365–1371
14. Mandel JS, Church TR, Ederer F: Screening for colorectal cancer. [letter] *N Engl J Med* 1993; 329: 1353–1354
15. Habr-Gama A, Waye JD: Complications and hazards of gastrointestinal endoscopy. *World J Surg* 1989; 13: 193–201
16. Marshall KG: Screening for prostate cancer. How can patients give informed consent? *Can Fam Physician* 1993; 39: 2385–2390
17. Muldoon MF, Manuck SB, Matthews KA: Lowering cholesterol concentrations and mortality: a quantitative review of primary prevention trials. [review] *BMJ* 1990; 301: 309–314
18. Kaplan JR, Manuck SB, Shively C: The effects of fat and cholesterol on social behavior in monkeys. *Psychosom Med* 1991; 53: 634–642
19. Young SN: Cholesterol, heart disease and the brain: an opportunity in research and a disaster in public health education? [editorial] *J Psychiatr Neurosci* 1993; 18: 1–3
20. Forrow L, Calkins D, Allshouse K et al: Effects of cholesterol screening on health perceptions. [abstract] *Clin Res* 1989; 37: 818A
21. Sorensen JR, Levy HL, Mangione TW et al: Parental response to repeat testing of infants with false-positive results in a newborn screening programme. *Paediatrics* 1984; 73: 183–187
22. Burton B, Dillard RG, Clark EN: The psychological impact of false positive elevations of maternal serum alpha-fetoprotein. *Am J Obstet Gynecol* 1985; 151: 77–82
23. Hall PF: Rethinking risk. [editorial] *Can Fam Physician* 1994; 40: 1239–1244
24. Klein M: Family physician maternity care. Outcomes when family physicians provide most community-based care. [editorial] *Can Fam Physician* 1995; 41: 546–548
25. Horowitz M, Hulley S, Alvarez W et al: News of risk for early heart disease as a stressful event. *Psychosom Med* 1980; 42: 37–46
26. Pamuk ER, Williamson DF, Serdula MK et al: Weight loss and subsequent death in a cohort of US adults. *Ann Intern Med* 1993; 119: 744–748
27. Blair SN, Shaten J, Brownell K et al: Body weight change, all-cause mortality, and cause-specific mortality in the multiple risk factor intervention trial. *Ann Intern Med* 1993; 119: 749–757
28. Reubin A, Muller DC, Sorkin JD: Long-term effects of change in body weight on all-cause mortality. A review. *Ann Intern Med* 1993; 119: 737–743
29. Kamrath RO, Plummer LJ, Sadur CN et al: Cholelithiasis in patients treated with a very-low-calorie diet. *Am J Clin Nutr* 1992; 56 (1 suppl): 255S–257S
30. Wilson GT: Relation of dieting and voluntary weight loss to psychological functioning and binge eating. *Ann Intern Med* 1993; 119: 727–730
31. The Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group: The effect of vitamin E and beta carotene on the incidence of lung cancer and other cancers in male smokers. *N Engl J Med* 1994; 330: 1029–1035
32. Kowalski TE, Falestiny M, Furth E et al: Vitamin A hepatotoxicity: a cautionary note regarding 25,000 IU supplements. *Am J Med* 1994; 97: 523–528
33. The SALT Collaborative Group: Swedish aspirin low-dose trial (SALT) of 75 mg aspirin as secondary prophylaxis after cerebrovascular ischaemic events. *Lancet* 1991; 338: 1345–1349
34. Silagy CA, McNeil JJ, Donnan GA et al: Adverse effects of low-dose aspirin in a healthy elderly population. *Clin Pharmacol Ther* 1993; 54: 84–89
35. Leibovici A, Lavi N, Wainstok S et al: Low-dose acetylsalicylic acid use and hemoglobin levels. Effects in a primary care population. *Can Fam Physician* 1995; 41: 64–68
36. Weil J, Colinjones D, Langman M et al: Prophylactic aspirin and risk of peptic ulcer bleeding. *BMJ* 1995; 310: 827–830
37. Bergkvist L, Adami H-O, Persson I et al: The risk of breast cancer after estrogen and estrogen-progestin replacement. *N Engl J Med* 1989; 321: 293–297
38. Colditz GA, Hankinson SE, Hunter DJ et al: The use of estrogens and progestins and the risk of breast cancer in postmenopausal women. *N Engl J Med* 1995; 332: 1589–1593
39. Jaeschke R, Guyatt G, Shannon H et al: Basic statistics for clinicians: 3. Assessing the effects of treatment: measures of association. *Can Med Assoc J* 1995; 152: 351–357
40. Laupacis A, Sackett DL, Roberts RS: An assessment of clinically useful measures of the consequences of treatment. *N Engl J Med* 1988; 318: 1728–1733
41. Naylor CD, Chen E, Strauss B: Measured enthusiasm: Does the method of reporting trial results alter perceptions of therapeutic effectiveness? *Ann Intern Med* 1992; 117: 916–921
42. Smith GD, Egger M: Who benefits from medical interventions? *BMJ* 1994; 308: 72–74
43. Bucher HC, Weinbacher M, Gyr K: Influence of method of reporting study results on decision of physicians to prescribe drugs to lower cholesterol concentration. *BMJ* 1994; 309: 761–764
44. Bobbio M, Demichelis B, Giustetto G: Completeness of reporting trial results: effect on physician's willingness to prescribe. *Lancet* 1994; 343: 1209–1211
45. Beaulieu MD: Statistiques. Sommes-nous à leur merci? *L'omnipracticien* 1994; 2 (22): 1,3
46. Liddell MJ: Mass mammographic screening — the other side of the argument. *Aust Fam Physician* 1993; 22: 1168–1175
47. Shapiro S, Venet W, Strax P et al: Ten-to-fourteen year effect of screening on breast cancer mortality. *J Natl Cancer Inst* 1982; 69: 349–355
48. Tabar L, Fagerberg CJ, Gad A et al: Reduction in mortality from breast cancer after mass screening with mammography. Randomised trial from the Breast Cancer Screening Working Group of the Swedish National Board of Health and Welfare. *Lancet* 1985; 1: 829–832
49. Roberts MM, Alexander FE, Anderson TJ et al: Edinburgh

trial of screening for breast cancer: mortality at seven years. *Lancet* 1990; 335: 241–246
50. Andersson I, Aspegren K, Janzon L et al: Mammographic screening and mortality from breast cancer: the Malmo mammographic screening trial. *BMJ* 1988; 297: 943–948
51. Miller AB, Baines CJ, To T et al: Canadian National Breast Screening Study: 2. Breast cancer detection and death rates among women aged 50 to 59 years. *Can Med Assoc J* 1992; 147: 1477–1488
52. Harris R, Leininger L: Clinical strategies for breast cancer screening: weighing and using the evidence. *Ann Intern Med* 1995; 122: 539–547
53. Lipid Research Clinics Program: The Lipid Research Clinics Coronary Primary Prevention Trial results: 1. Reduction in incidence of coronary heart disease. *JAMA* 1984; 251: 351–364
54. Frick MH, Elo O, Haapa K et al: Helsinki Heart Study: primary-prevention trial with gemfibrozil in middle-aged men with dyslipidemia. *N Engl J Med* 1987; 317: 1231–1275
55. Shepherd J, Cobbe SM, Ford I et al: Prevention of coronary heart disease with pravastatin in men with hypercholesterolemia. *N Engl J Med* 1995; 333: 1301–1307
56. Rose G: Sick individuals and sick populations. *Int J Epidemiol* 1985; 14: 32–38

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Colorado Springs, Colo.
Thelma Edwards, director of program development, National Stroke Association, 1000–8480 E Orchard Rd., Englewood CO 80111-5015; tel 303 771-1700, ext. 20, fax 303 771-1886

June 6–8, 1996: Quality of Life: an International Conference for Families and Professionals on Developmental and Related Disabilities
Toronto
Quality of Life Conference—Surrey Place Centre, c/o Continuing Medical Education, University of Toronto, Faculty of Medicine, Rm. 121, 150 College St., Toronto ON M5S 1A8; tel 416 978-2719, fax 416 971-2200; a.lind@utoronto.ca

June 6–9, 1996: General Practice Psychotherapy Association 9th Annual Educational Conference : Developing Psychotherapy Skills for Use in General Practice
Mississauga, Ont.
Dr. Greg Dubord, chairman, 1996 General Practice Psychotherapy Association Educational Conference, PO Box 225, First Canadian Place, Toronto ON M5X 1B5; tel 416 391-4040, fax 416 203-6585

June 8–11, 1996: American Diabetes Association 56th Annual Meeting and Scientific Sessions
San Francisco
Meeting Services Department, American Diabetes Association, 1660 Duke St., Alexandria VA 22314; tel 800 232-3472, ext 2453 or 2330; fax 703 683-1351; meetings@diabetes.org

June 8–12, 1996: American Association for Cancer Research Special Conference in Cancer Research — Inducible Genomic Responses
Stevenson, Wash.
Special Conference Registration, American Association for Cancer Research, Ste. 816, Public Ledger Building, 150 S Independence Mall W, Philadelphia PA 19106-3483; tel 215 440-9300, fax 215 440-9313

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