Cardiovascular risk tables

Estimating risk is not the problem, using it to tailor treatment to individuals is

CORDELIA MOLLOY/SCIENCE PHOTO LIBRARY

RESEARCH, p 1475

Thierry Christiaens professor of general practice, Department of General Practice and PHC & Heymans Institute of Pharmacology, Ghent University, Ghent, Belgium
thierry.christiaens@ugent.be

Competing interests: None declared.

Provenance and peer review: Commissioned; not externally peer reviewed.

BMJ 2008;336:1445-6
doi: 10.1136/bmj.a480

In the linked study, Hippisley-Cox and colleagues develop and validate the second version of the QRISK cardiovascular disease risk algorithm (QRISK2), an attempt to more accurately estimate cardiovascular risk in patients from different ethnic groups in England and Wales.[1]

The advent of the first Framingham risk tables in the early 1990s was a challenge for most doctors. Since the second world war the management of cardiovascular risk has been part of the core business of general practice, but the single risk model dominated. Hypertension, diabetes, and hypercholesterolaemia were islands, each with its own experts fighting for bigger kingdoms by pushing for ever stricter boundaries and demanding more attention.

Framingham taught us to look at the different risk factors, and provided a major lesson: a cumulative average risk could be more important than one peak. Yet soon the extrapolation of these US tables to European populations seemed to overshoot the real risk in these groups.[2 3] The SCORE tables used the same risk factors to calculate corrected European cardiovascular mortality.[4]

More recently the ASSIGN[5] and now the QRISK tables tried to incorporate some other known risk factors, especially deprivation and family history. Again, a major step: for several decades the medical community has had to face the troubling fact that cardiovascular morbidity and mortality are strongly and independently related to deprivation.[6 7] If we ignore this we overestimate the risk for rich people (and overtreat them) and underestimate that for poor people. It's probably naive to think that we can close the gap in cardiovascular risk just by giving more statins to poor people.

If epidemiologists could estimate cardiovascular risk accurately, would it solve our problems in managing patients? Not at all. Risk calculation itself is based on evidence. However, using risk calculation in managing patientsrelies on consensus. When does a "risk" become a "high risk"? At what moment does a high risk justify starting lifelong drug treatment? The SCORE tables are useful, but when the European Guidelines tried to implement these tables and defined a 5% risk of death within the coming 10 years as high risk (comparable to a 20% risk in the Framingham tables),[8] it led to an enormous medicalisation of many healthy elderly people, as proved by the Nordic Risk Group.[9] Nearly all Norwegian men aged 60 years and older and allwomen aged 65 years and older were classified as at "high risk"—in a population with one of the highest life expectancies in the world.

To use an absolute risk score as a threshold for starting drugs is dangerous and not evidence based. It is therefore surprising that the recent NICE guidelines strongly recommend statins for anyone with a cardiovascular risk score of 20 or more in the Framingham tables.[10]

Age is such an important risk factor for developing cardiovascular problems within the next 10 years that all risk tables are misleading. Becoming older is by far the strongest predictor for morbidity and mortality—this is a biological fact. By looking at the risk tables, anyone can see what happens: by age 65, a large group has reached the 20% risk threshold, and lipid lowering drugs are prescribed for the rest of their lives.

A non-smoking man of 70 with a systolic blood pressure of 130 mm Hg and a total cholesterol concentration of 5 mmol (far below the median cholesterol concentration in most European countries) is at high risk according to the SCORE criteria. Unfortunately, most of the trials of statins include only a few people older than 70.[11] The PROSPER trial, which specifically looked at this elderly population, showed that the primary composite endpoint (cardiovascular death or non-fatal infarction or cerebrovascular accident) was lowered by only 15% (48 people have to be given statins for three years to prevent one event), a marginally significant gain for cardiovascular death (relative risk 0.76, 95% confidence interval 0.58 to 0.99; NNT 112 for three years) and no effect at all on total mortality.[12] In contrast, a male smoker aged 50 with a systolic blood pressure of 145 mm Hg and a total cholesterol of 6.5 mmol/l is at low risk on SCORE.

A better way of using risk tables would be to compare the risk of an individual with the minimal risk of people of the same sex and age. Treatment should be considered when he or she has, for example, three times that minimal risk for his or her age and sex. This will prevent overtreatment of elderly people whose high risk is related to age and undertreatment of younger people who are at high risk. For our two examples the treatment options would be totally different.

All attempts to make risk tables more accurate, as done by Hippisley-Cox and colleagues in the QRISK2 algorithm,[1] are necessary and should be welcomed. However, this is not the key problem. We have to fundamentally rethink how to use risk tables when making treatment decisions in practice, taking into consideration the medicalisation of healthy older people and the correct use of drugs.

1 Hippisley-Cox J, Coupland C, Vinogradova Y, Robson J, Minhas R, Sheikh A, et al. Predicting cardiovascular risk in England and Wales: prospective derivation and validation of QRISK2. *BMJ* 2008;337:doi: 10.1136/bmj.39609.449676.25
2 Brindle P, Emberson J, Lampe F, Walker M, Whincup P, Fahey T, et al. Predictive accuracy of the Framingham coronary risk score in British men: prospective cohort study. *BMJ* 2003;327:1-6.
3 Ferrario M, Chiodini P, Chambless LE, Cesana G, Vanuzzo D, Panico S, et al. Prediction of coronary events in a low incidence population: assessing accuracy of the CUORE cohort study prediction equation. *Int J Epidemiol* 2005:34:413-21.
4 Conroy RM, Pyörälä K, Fitzgerald AP, Sans S, Menotti A, De Backer G, et al. Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project. *Eur Heart J* 2003;24:987-1003.
5 Woodward M, Brindle P, Tunstall-Pedoe H, SIGN group on risk estimation. Adding social deprivation and family history to cardiovascular risk assessment: the ASSIGN score from the Scottish heart health extended cohort (SHHEC). Heart 2007;93:172-6.
6 White C, Galen FV, Chow YH. Trends in social class differences in mortality by cause. *Health Stats Q* 2003;20:25-36.
7 Tunstall-Pedoe H, Woodward M. By neglecting deprivation, cardiovascular risk scoring will exacerbate social gradients in disease. *Heart* 2006;92:307-10.
8 De Backer G, Ambrosioni E, Borch-Johnsen K, Brotons C, Cifkova R, Dallongeville J, et al. European guidelines on cardiovascular disease prevention in clinical practice. Third Joint Task Force of European and Other Societies on Cardiovascular Disease Prevention in Clinical Practice. *Eur J Cardiovasc Prev Rehabil* 2003;10(suppl 1):S1-78.
9 Getz L, Sigurdsson JA, Hetlevik I, Kirkengen AL, Romundstad S, Holmen J. Estimating the high risk group for cardiovascular disease in the Norwegian HUNT 2 population according to the 2003 European guidelines: modelling study. *BMJ* 2005;331:551-4.
10 Cooper A, O'Flynn N, on behalf of the Guideline Development Group. Risk assessment and lipid modification for primary and secondary prevention of cardiovascular disease: summary of NICE guidance. *BMJ* 2008;336:1246-8.
11 Prospective Studies Collaboration. Blood cholesterol and vascular mortality by age, sex, and blood pressure: a meta-analysis of individual data from 61 prospective studies with 55000 vascular deaths. *Lancet* 2007;370:1829-39.
12 Shepherd J, Blauw GJ, Murphy MB, Bollen EL, Buckley BM, Cobbe SM, et al. Pravastatin in elderly individuals at risk of vascular disease (PROSPER): a randomised controlled trial. *Lancet* 2002;360:1623-30.

Measuring deaths from conflict

New method is promising but is still likely to underestimate deaths

VIETNAM MEMORIAL/DAVID ZAITZ/PHOTONICA/GETTY

RESEARCH, p 1482

Richard Garfield Henrik Bendixen professor of clinical international nursing, School of Nursing, Columbia University, Box 6, New York City, NY 10032, USA

Competing interests: None declared.

Provenance and peer review: Commissioned; not externally peer reviewed.

BMJ 2008;336:1446-7
doi: 10.1136/bmj.a146

In the linked study, Obermeyer and colleagues challenge conventional thinking about deaths related to war and force us to re-evaluate some well established assumptions about these deaths.[1] Deaths in combatants and non-combatants are always underestimated during conflicts between armed groups in poor countries that are not national armies.[2] Even in middle income developing countries, counts that are purported to be precise fail to include most of those killed.[3 4]

Obermeyer and colleagues used demographic data from world health surveys collected before and after conflicts in 13 countries over the past 50 years. The surveys collect information from one respondent for each household about sibling deaths, including whether the deaths were related to war. The data are then compared with those obtained through passive reports (mainly from eyewitnesses and the media). This method eliminates some of the ambiguity rampant in this highly politicised field.

Obermeyer and colleagues estimate that 5.4 million (95% confidence interval 3.0 to 8.7) deaths occurred as a result of war in 13 countries from 1955 to 2002; the numbers ranged from 7000 in the Republic of Congo to 3.8 million in Vietnam. The estimates were about three times higher than those obtained from passive reports.

Limitations of the analysis include the relatively small number of surveys analysed (13) and the fact that five of them were based on relatively small national samples. Also, because undercounting varies greatly between conflicts, the confidence intervals are wide. The pattern of undercounting was not consistent—some countries even overcounted the number of deaths.

Good quality data on the epidemiology of violence often become available only years after the killing has ended. Real time surveillance systems that can count most deaths as they occur are needed to solve this problem. In poor and unstable countries, where almost all wars now occur, such systems are rare.

Despite rigorous efforts to correct for under-reporting, Obermeyer and colleagues could not correct for household members who chose not to report deaths. How the relevant questions were asked in face to face interviews can greatly influence the results obtained. Similarly, the total number of deaths in war may be grossly underestimated by multiyear demographic modelling. Half a million deaths can occur unnoticed when demographic models do not count actual deaths but depend on projections from count data that are decades old.

Finally, the study only includes violent deaths. In the poorest countries, where most conflicts now occur, a rise in deaths from infectious diseases often dwarfs the number of violent deaths during a conflict. For all these reasons, Obermeyer and colleagues' study is likely to underestimate the importance of conflict as a cause of death.

To reduce casualties from violence or disease we need current data, as well as assessments of their inadequacies. A lack of such assessments has fuelled controversies over estimates of deaths in non-combatants that are based on data from field epidemiological studies in Iraq, Darfur Sudan, and the Democratic Republic of Congo. But we should not despair. A generation ago little controversy existed over such figures because epidemiological studies like these did not even exist.

The news about war related deaths in the world these days is both good and bad. The good news is that fewer combatants die today than at any time in the past 100 years, and the number and intensity of military conflicts have declined considerably since 1994.[5] The bad news is that most excess deaths in

areas of conflict in developing countries occur in non-combatants, and these deaths are often not counted, so we cannot be sure that the total number of war related deaths has also dropped.

The method pioneered by Obermeyer and colleagues is promising, however. When stability returns to current or recent hotspots where epidemiological study is difficult—such as Somalia, southern Sudan, and Iraq—we may yet be able to count the lives and deaths of these people. As the authors state in their introduction, the importance of war as a public health problem and a social problem makes this imperative.

1 Obermeyer Z, Murray CJL, Gakidou E. Fifty years of violent deaths related to war from Vietnam to Bosnia: analysis of data from world health survey programme. *BMJ* 2008 doi: 10.1136/bmj.a137.
2 Human Security Centre. *War and peace in the 21st century.* New York: Oxford University Press, 2005. www.humansecurityreport.info/HSR2005_HTML/Cover/index.htm.
3 Tapp C, Burkle FM, Wilson K, Takaro T, Guyatt GH, Amad H, et al. Iraq war mortality estimates: a systematic review. *Conflict Health* 2008;2:1752-85.
4 US Government Accountability Office. *Darfur crisis: death estimates demonstrate severity of crisis but their accuracy and credibility could be enhanced.* 2006. www.gao.gov/new.items/d0724.pdf
5 Garfield R. Epidemiology of war. In: Levy B, Sidel V. *War and public health.* 2nd ed. New York: Oxford University Press, 2007:23-37.

Seroprotection against serogroup C meningococcal disease

Is higher if vaccination is given in the second decade of life rather than in the first

RESEARCH, p 1487

Lucieni O Conterno coordinator of infectious diseases discipline and clinical epidemiology unit, Marilia Medical School, ZIP 17 519 030 Marilia, São Paulo, Brazil
Paul T Heath reader in paediatric infectious diseases and honorary consultant, Child Health and Vaccine Institute, St George's, University of London, London SW17 ORE

Competing interests: PTH has conducted clinical trials on behalf of St George's, University of London, which were sponsored by vaccine manufacturers, including manufacturers of meningococcal vaccines, and has received funds from vaccine manufacturers to attend scientific meetings.
Provenance and peer review: Commissioned; not externally peer reviewed.

BMJ 2008;336:1447-8
doi: 10.1136/bmj.39577.487558.BE

Neisseria meningitidis is a leading cause of bacterial meningitis worldwide. Most cases in developed countries are caused by endemic disease. The incidence is around 1-2 per 100 000,[1-3] with rates among infants as high as 20 per 100 000.[2] Children younger than 2 years have the highest incidence of meningococcal disease, with a second peak between 15 and 24 years. Most cases are caused by serogroups A, B, C, W-135, and Y. Serogroups C and B predominate in temperate countries.[1-3]

In the accompanying study, Snape and colleagues evaluate the persistence of serum bactericidal antibody against meningococcal serogroup C in a large cohort of adolescents originally immunised with serogroup C meningococcal conjugate vaccines at 6-15 years of age.[4] These vaccines, now used in many countries, were licensed on the basis of immunogenicity rather than clinical efficacy.[5] Despite the high public profile of meningococcal disease, it is relatively rare, which makes traditional efficacy studies prohibitive and impractical. The approach to licensing this vaccine was therefore novel and was based on its ability to induce serum bactericidal antibody at titres known to correlate with clinical protection. Since the licence was granted, effectiveness studies have confirmed the validity of this approach and have allowed further fine tuning of these serological correlates of protection.[5-8]

A systematic review conducted in 2006 confirmed that serogroup C meningococcal conjugate vaccines are highly immunogenic in all age groups.[9] However, a decline in serological protection over time was noted in children who were vaccinated in infancy, a phenomenon also seen with the *Haemophilus influenzae* type b conjugate vaccines. The relevance of this finding was not clear, as the presence of immunological memory implied that a rapid immune response should occur when antigen was encountered. This might result in clinical protection, even in the absence of detectable circulating antibody. But meningococcal disease has a short incubation period, so the speed of response is crucial. Subsequent observations on vaccine failures and analysis of vaccine effectiveness (for both the meningococcal and influenza vaccines) now support the need for persistence of bactericidal antibody.[10] In the United Kingdom, the public health response to this has been the recent addition of a booster dose of these vaccines in the second year of life.

Snape and colleagues found that five years after immunisation, 84.1% (95% confidence interval 81.6 to 86.3) of 987 participants had bactericidal antibody titres ≥1:8. However, geometric mean titres were significantly lower in 11-13 year olds (147; 115 to 188) than in 14-16 year olds (300; 237 to 380) and 17-20 year olds (361; 253 to 513) ($P<0.0001$ for both comparisons). Protective titres were achieved in around 10% fewer of the 11-13 year olds than in the older groups. The authors conclude that antibody titres five years after immunisation are higher if children are vaccinated in the second decade of life rather than the first.[4]

These data emphasise the importance of age at vaccination for conjugate vaccines with regard to protection and persistence, and they have implications for the number of vaccine doses needed at different ages. The increased immune response seen in the second decade of life is difficult to explain. The authors suggest that the older participants may have had more natural exposure to serogroup C meningococci just before or after they were immunised and so were better primed (or boosted) by carriage, with better primary responses and better persistence. In the era before vaccination, carriage was highest in the adolescent age group (albeit only at around 0.5%).[11] Study participants over the age of 15 were therefore vaccinated around the time of maximum carriage and exposure. Seroepidemiological studies of group C (pre-vaccine) and group B meningococci also show sharp increases in antibody titres in adolescent age groups, which lends support to this argument.

The authors' second hypothesis is that immune responses mature in the second decade of life, so that primary and persistent vaccine responses are enhanced. They found no literature to support this with respect to conjugate vaccines, but it may be possible to test this hypothesis in the next phase of the UK meningococcal vaccine programme.

The meningococcal vaccination programme has been a great success—high levels of direct and indirect vaccine protection have been recorded; carriage and invasive disease have declined significantly[12]; for the first time ever, no one under 19 years old has died of this disease in the past year; and the feared replacement by serogroup B disease has not occurred. This experience may help other countries to define the best strategy to prevent serogroup C disease, taking into account their own epidemiological reality.

However, concerns about ongoing control of disease as children in certain age groups get older and perhaps lose their antibodies—as shown by Snape and colleagues[4]—remind us that continued high quality surveillance must continue, even long after disease seems to have been controlled.

1 Sharip A, Sorvillo F, Redelings MD, Mascola L, Wise M, Nguyen DM. Population-based analysis of meningococcal disease mortality in the United States: 1990-2002. *Pediatr Infect Dis J* 2006;25:191-4.
2 Perrocheau A, Taha MK, Levy-Bruhl D. Epidemiology of invasive meningococcal disease in France in 2003. *Euro Surveill* 2005;10:238-41.
3 De Wals P, Deceuninck G, Boulianne N, De Serres G. Effectiveness of a mass immunization campaign using serogroup C meningococcal conjugate vaccine. *JAMA* 2004;292:2491-4.
4 Snape MD, Kelly DF, Lewis S, Banner C, Kibwana L, Moore CE, et al. Seroprotection against serogroup C meningococcal disease in adolescents in the United Kingdom: observational study. *BMJ* 2008; doi: 10.1136/bmj.39563.545255.AE.
5 Miller E, Salisbury D, Ramsay M. Planning, registration, and implementation of an immunisation campaign against meningococcal serogroup C disease in the UK: a success story. *Vaccine* 2001;20(suppl 1):S58-67.
6 Goldschneider I, Gotschlich EC, Artenstein MS. Human immunity to the meningococcus. I. The role of humoral antibodies. *J Exp Med* 1969;129:1307-26.
7 Andrews N, Borrow R, Miller E. Validation of serological correlate of protection for meningococcal C conjugate vaccine by using efficacy estimates from postlicensure surveillance in England. *Clin Diagn Lab Immunol* 2003;10:780-6.
8 Borrow R, Andrews N, Goldblatt D, Miller E. Serological basis for use of meningococcal serogroup C conjugate vaccines in the United Kingdom: reevaluation of correlates of protection. *Infect Immun* 2001;69:1568-73.
9 Conterno LO, Silva Filho CR, Rüggeberg JU, Heath PT. Conjugate vaccines for preventing meningococcal C meningitis and septicaemia. *Cochrane Database Syst Rev* 2006;(3):CD001834.
10 Auckland C, Gray S, Borrow R, Andrews N, Goldblatt D, Ramsay M, et al. Clinical and immunologic risk factors for meningococcal C conjugate vaccine failure in the United Kingdom. *J Infect Dis* 2006;194:1745-52.
11 Maiden MC, Stuart JM, UK Meningococcal Carriage Group. Carriage of serogroup C meningococci 1 year after meningococcal C conjugate polysaccharide vaccination. *Lancet* 2002;359:1829-31.
12 Ramsay ME, Andrews NJ, Trotter CL, Kaczmarski EB, Miller E. Herd immunity from meningococcal serogroup C conjugate vaccination in England: database analysis. *BMJ* 2003;326:365-6.

Effects of quality improvement collaboratives

Are difficult to measure using traditional biomedical research methods

RESEARCH, p 1491

Peter K Lindenauer director and associate professor of medicine, Center for Quality and Safety Research, Baystate Medical Center and Tufts University School of Medicine, Springfield MA, 01199, USA
peter.lindenauer@bhs.org

Competing interests: None declared.
Provenance and peer review: Commissioned; not externally peer reviewed.

BMJ 2008;336:1448-9
doi: 10.1136/bmj.a216

In the linked study, Schouten and colleagues report a systematic review of the effectiveness of quality improvement collaboratives in improving the quality of care. They conclude that the evidence supporting these collaboratives is positive but limited and their effects are difficult to predict.[1]

Despite limited evidence, the quality improvement collaborative is one of the most popular methods for organising improvement efforts at hospitals and ambulatory practices worldwide. Quality improvement collaboratives in health care date back to the mid-1980s, and some of the earliest and most successful examples include the Northern New England Cardiovascular Disease Study Group, the US Veterans' Affairs National Surgical Quality Improvement Program, and the Vermont Oxford Network. These ongoing initiatives have improved care and saved many lives at participating hospitals.[2-4]

In the 1990s, the Institute for Healthcare Improvement, the pre-eminent quality improvement organisation in the United States, popularised a quality improvement model they called the breakthrough series.[5] Whereas earlier quality improvement collaboratives were limited to a single domain (such as cardiac surgery), the breakthrough series method has been applied to a wide range of topics, from improving access in primary care to reducing adverse drug events among patients in hospital.

Quality improvement collaboratives bring together quality improvement teams from multiple sites across a region or country to focus on a common problem. Over one or two years (or many years in the earliest collaboratives) experts in clinical and performance improvement provide the group with periodic instruction and encourage the teams to share lessons learnt and best practices. The model has taken hold largely on its face validity—the idea that improvement teams are likely to be more effective when working together rather than in isolation—and it has been replicated many times across the US and Europe.

Several years ago our hospital joined a quality improvement collaborative to reduce the occurrence of postoperative infections in patients undergoing major surgery. Together with more than 50 hospitals throughout the US and its territories, we identified several specific quality measures and targets; for example, we sought to ensure that all patients received prophylactic antibiotics within one hour of the opening surgical incision.

At each of several "learning sessions" we received instruction from national leaders in perioperative

care and training from quality improvement experts in how to apply the "plan-do-study-act" quality improvement paradigm to surgical care. After the initial meeting, each hospital presented their progress, achievements, and lessons learnt. How to apply these lessons at home was then discussed.

At the end of the 18 month project we had made dramatic improvements in several key process of care measures, but little headway in others, and our postoperative infection rate had not improved. Some hospitals across the collaborative struggled to make even small improvements, whereas others described impressive gains and substantial reductions in infection.

Unfortunately, neither the quality improvement collaborative for surgical infection prevention nor hundreds of others that have been carried out over the past two decades are included in the systematic review by Schouten and colleagues. This cannot be blamed on the authors, who scanned more than 1000 journal abstracts to find 175 articles worth reviewing in detail. Of the 72 published studies that reported on the outcomes or effectiveness of a quality improvement collaborative, 60 (82%) used an uncontrolled study design, generally relying on a simple before and after approach that could not account for secular trends; relied on self report rather than third party chart review; and suffered from generally poor quality data management procedures. The remaining 12 reports represented nine studies, including two randomised controlled trials; seven showed at least some positive effects on process or outcome measures, while two were entirely negative. Even in this highly restricted group, most studies had methodological weaknesses that would be considered problematic outside of the field of quality improvement research. Of the two randomised controlled trials, one showed no benefit, whereas the other showed improvement in two process of care measures but not in outcomes.

Although the review is original it does have several important limitations. Firstly, it is debatable whether the nine studies included represent the global experience with quality improvement collaboratives, and thus whether the findings can be extrapolated to future collaboratives. Secondly, the small number of high quality studies makes it impossible to evaluate which characteristics of these collaboratives are associated with success. For example, the kinds of clinical conditions that are most suited to the approach, the attributes of a successful faculty, the ideal mix of team members, the number of sessions needed and how they should be structured, and the time period over which the quality improvement collaborative should take place.[6]

The third concern is whether aggregating the findings of a heterogeneous group of studies on quality improvement collaboratives makes much sense. To state that quality improvement collaboratives are modestly beneficial seems analogous to saying that, in general terms, drugs have beneficial effects on disease. Although this may be true, it hides the fact that some drugs improve outcomes for patients with certain conditions (for example, aspirin for secondary prevention of coronary artery disease) more than they do for others (for example, cholinesterase inhibitors for Alzheimer's dementia).

A more fundamental question is whether the methods used in traditional biomedical research are sufficient to evaluate quality improvement collaboratives. Undoubtedly, randomised controlled trials are the optimal approach to test the efficacy of drugs. But, unlike most pharmacological trials in which a study coordinator ensures that patients are treated according to a strict protocol, this is not usually the case for quality improvement initiatives, which take place in a less controlled environment. Research into quality improvement that reports only the mean improvement in participants and controls misses an opportunity to explore important contextual factors that might have explained why two hospitals can have such different experiences when participating in the same quality improvement collaborative.

Future research should focus on the behaviours and actions of the participants themselves, such as how the executive sponsors tried to ensure that the team was successful, what role the doctor and nurse champions played in winning the support of their colleagues, and how information technology was used for the benefit of the project.[7 8] While lip service has been paid to the need for these kinds of studies, they remain few and far between.[9-11]

1 Schouten LMT, Hulscher MEJL, van Everdingen JJE, Huijsman R, Grol RPTM. Evidence for the impact of quality improvement collaboratives: systematic review. *BMJ* 2008; doi: 10.1136/bmj.39570.749884.BE.
2 Horbar JD. The Vermont Oxford Network: evidence-based quality improvement for neonatology. *Pediatrics* 1999;103(1 suppl E):350-9.
3 Khuri SF, Daley J, Henderson W, Hur K, Demakis J, Aust JB, et al. The Department of Veterans Affairs' NSQIP: the first national, validated, outcome-based, risk-adjusted, and peer-controlled program for the measurement and enhancement of the quality of surgical care. National VA surgical quality improvement program [see comment]. *Ann Surg* 1998;228:491-507.
4 O'Connor GT, Plume SK, Olmstead EM, Morton JR, Maloney CT, Nugent WC, et al. A regional intervention to improve the hospital mortality associated with coronary artery bypass graft surgery. The Northern New England Cardiovascular Disease Study Group [see comment]. *JAMA* 1996;275:841-6.
5 Kilo CM. A framework for collaborative improvement: lessons from the Institute for Healthcare Improvement's breakthrough series. *Qual Manag Health Care* 1998;6:1-13.
6 Ovretveit J, Bate P, Cleary P, Cretin S, Gustafson D, McInnes K, et al. Quality collaboratives: lessons from research [see comment]. *Qual Saf Health Care* 2002;11:345-51.
7 Hulscher MEJL, Laurant MGH, Grol RPTM. Process evaluation on quality improvement interventions. *Qual Saf Health Care* 2003;12:40-6.
8 Mittman BS. Creating the evidence base for quality improvement collaboratives [see comment]. *Ann Intern Med* 2004;140:897-901.
9 Cretin S, Shortell SM, Keeler EB. An evaluation of collaborative interventions to improve chronic illness care. Framework and study design. *Eval Rev* 2004;28:28-51.
10 Marsteller JA, Shortell SM, Lin M, Mendel P, Dell E, Wang S, et al. How do teams in quality improvement collaboratives interact? *Jt Comm J Qual Patient Saf* 2007;33:267-76.
11 Shortell SM, Marsteller JA, Lin M, Pearson ML, Wu S-Y, Mendel P, et al. The role of perceived team effectiveness in improving chronic illness care.[see comment]. *Med Care* 2004;42:1040-8.

Continuous publication

The next logical step

Fiona Godlee editor, *BMJ*, London WC1H 9JR
Tony Delamothe deputy editor, *BMJ*, London WC1H 9JR
Jane Smith deputy editor, *BMJ*, London WC1H 9JR
jsmith@bmj.com
Competing interests: FG, TD, and JS are employees of the *BMJ*.

BMJ 2008;336:1450
doi: 10.1136/bmj.a491

The *BMJ* is about to undergo another shift in the way that it publishes its content, which we hope will provide benefits for both readers and authors. From the beginning of July we will be publishing content continuously on bmj.com. All our articles will be published on line as they become ready, so bmj.com will update several times a day. Once published, articles will then be selected for a subsequent print issue.

Why are we doing this? It's a logical extension of what we've been doing for some time with online first publication of research, and it will give all articles the benefit of faster publication.[1] This makes most sense in the context of research, news, and other topical items, but all authors appreciate seeing their work published as soon as possible.

Continuous publication also gives readers more flexibility in the way they engage with our content: as a continuous stream or in a weekly "package," or both. And it will allow us to tailor the print journal—which is read largely by UK readers—to their needs.

The *BMJ* is the first major medical journal to move to continuous publication, but within publishing generally it is not alone. Broadcasters have long been posting news continuously on their websites, and many newspapers now post their articles online as soon as they are written, in advance of the next morning's paper edition. People's online behaviour suggests that their interest is primarily in individual articles and not in the print issues, or indeed even the journal in which they appear. The *BMJ* has for some years been taking full advantage of the web, giving authors all the space they need for their research papers and providing shorter versions in print. With the shift to continuous publication for all our content, we will carry an increasing amount of content that is online only, allowing us to serve our growing audiences around the world with content that is most relevant to their specific interests and needs.

The move creates various routes into our content for print and online readers. Print readers will have readable, relevant, clustered content presented to them in a preselected weekly package, with the possibility, as now, to go deeper into that content and to respond to it on bmj.com. For readers who like to use the print issue to find content on bmj.com, the current print issue will still be displayed on the home page, and previous print issues can be browsed in the archive. And PDFs of print issues can be printed off, in section-sized chunks.

Online users can browse current content via a rolling table of contents of all articles published in the past seven days. This will still be organised by section (editorials, news, research, etc).

An important aspect of the change is in the way that articles will be organised and cited. The online "publish ahead of print" model that we have been using for research articles assumed that they would eventually be published in a print issue, and the ultimate citation for that article derived from that print issue. Thus, when it was first published, an online first article had a year and a doi (a unique identifier for a digital object)—for example, *BMJ* 2008 doi:10.1136/bmj.012334.5678.BM—but when it later appeared in print, the definitive citation for that article became the traditional one of year, volume, and page number: *BMJ* 2008;336:123-5.

From now on, each article will have only one, permanent, citation and it will no longer derive from print. The citation will be year, volume, e-locator (a unique identifier for that article)—for example, *BMJ* 2008;337:a145—and this is what will appear in Medline, PubMed, and other bibliographical indexes. We will print this citation on every item we publish, in print and online, and authors will need to use it when they cite these *BMJ* articles.

Highwire Press, who provide our web platform and have built the tools that enable us to publish continuously, predict that in a few years' time "everyone will be doing it." We hope that our authors and readers will see the benefits, and as always we welcome your feedback.

1 Delamothe T. Is that it? How online articles have changed over the past five years. *BMJ* 2002;325:1475-8.