Hot tea and increased risk of oesophageal cancer

Allowing tea to cool for five minutes before drinking is advisable

MATTTILDA/FOTOLIA

RESEARCH, p 876

David C Whiteman NHMRC principal research fellow, Queensland Institute of Medical Research, PO Royal Brisbane Hospital, QLD 4029, Australia
david.whiteman@qimr.edu.au

Competing interests: None declared.

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

Cite this as: *BMJ* 2009;338:b610
doi: 10.1136/bmj.b610

In the linked case-control study, Islami and colleagues assess the association between how people drink their tea and the risk of oesophageal squamous cell carcinoma.[1]

Cancers of the oesophagus kill more than 500 000 people worldwide each year, with the bulk of the disease occurring in discrete populations in Asia, Africa, and South America.[2 3] Despite recent increases in the incidence of adenocarcinomas of the oesophagus in industrialised nations,[4 5] the most common subtype of oesophageal cancer worldwide is oesophageal squamous cell carcinoma. Tobacco and alcohol are the main causal factors related to oesophageal squamous cell carcinoma in the West, but they are not implicated in non-Western populations that have very high rates of this disease. Nutritional deficiency,[6] viral infection,[7] and dietary toxins[8] have all been postulated as causal factors, although none can fully explain the extraordinary excess of cases of oesophageal squamous cell carcinoma seen in these populations.

An intriguing hypothesis is that repeated thermal injury to the oesophageal epithelium may initiate carcinogenesis, but the limited evidence to date is inconclusive. Previous studies have indicated that consuming hot maté (a South American herbal beverage) or hot alcoholic liqueurs increases the risk of oesophageal squamous cell carcinoma.[9-12] However, testing this hypothesis has been hampered by the difficulty of separating the effect of heat from the confounding effects of ingested products already known or thought to cause this cancer (alcohol, tobacco, and maté). A further challenge has been to obtain valid and reliable estimates of the temperature at which participants typically swallow food or beverages because all studies have relied on self reports of intake.

Islami and colleagues' study is the most compelling test to date of the thermal injury hypothesis for oesophageal squamous cell carcinoma in humans. The study was conducted in a population in northern Iran with a high incidence of the disease. The investigators first elicited the tea drinking preferences of 300 people with oesophageal squamous cell carcinoma and a matched group of 571 controls drawn from the same geographical area. When compared with cancer-free controls, the patients were more than twice as likely to report drinking their tea "hot" (odds ratio 2.07, 95% confidence interval 1.28 to 3.35) rather than "warm or lukewarm" and were eight times more likely to drink their tea "very hot" (8.16, 3.93 to 16.9). The findings were similar for rapidity of intake—people who drank their tea within two minutes had a five times higher risk than those who waited more than four minutes before drinking their tea. These are substantial increases in the relative risk of cancer, and although observational studies are prone to well known forms of bias, the investigators minimised methodological error as much as possible.

An important potential weakness was that the estimates of tea drinking temperature were based on self reports by the participants. To tackle this problem—and in an attempt to calibrate the reports of warm, hot, and very hot tea—the investigators conducted a second study in which they measured the actual temperature of the tea consumed by nearly 50 000 residents of the same province of Iran. Taken together, these studies provide persuasive evidence that drinking tea at temperatures greater that 70°C markedly increases the risk of oesophageal squamous cell carcinoma.

Strengths of the study include the high rates of case ascertainment and control participation, which lessen the chance that biased selection of study participants introduced systematic errors. In addition, the almost universal consumption of tea in this population, coupled with the low rates of exposure to tobacco and alcohol, greatly reduces the likelihood of confounding that has bedevilled earlier studies. So although this study was conducted in a population with atypical and possibly unique patterns of exposure to substances that are commonly consumed, the findings are relevant to clinicians and researchers in many settings.

At the level of basic science, this report lends support to the notion that thermal injury may be a cause of epithelial cancers. The mechanism through which heat promotes the development of tumours warrants further exploration and might be given renewed impetus on the basis of these findings.

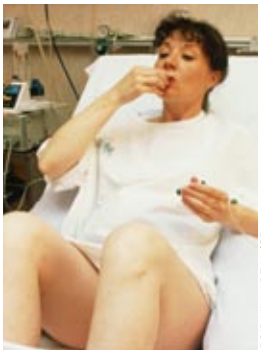
Given that randomised trials are unlikely to be conducted, any health advice must rely on these types of observational data. Replication of these findings is therefore desirable, but in the meantime, a precautionary approach should be taken in the region in which the study was conducted. Indeed, the consumption of hot drinks is common worldwide, although perhaps not at the scalding temperatures seen in Iran. It is therefore possible that thermal injury may underlie, at least in part, a proportion of cases of oesophageal squamous cell carcinoma elsewhere. It is difficult to imagine any adverse consequences of waiting at least four minutes before drinking a cup of freshly boiled tea, or more generally allowing foods and beverages to cool from "scalding" to "tolerable" before swallowing. These findings are not cause for alarm, however, and they should not

reduce public enthusiasm for the time honoured ritual of drinking tea. Rather, we should follow the advice of Mrs Beeton, who prescribes a five to 10 minute interval between making and pouring tea, by which time the tea will be sufficiently flavoursome and unlikely to cause thermal injury.

1 Islami F, Pourshams A, Nasrollahzadeh D, Kamangar F, Fahimi S, Shakeri R, et al. Tea drinking habits and oesophageal cancer in a high risk area in northern Iran: population based case-control study. *BMJ* 2009;338:b929.
2 Ferlay J, Bray F, Pisani P, Parkin DM. *GLOBOCAN 2002: cancer incidence, mortality and prevalence worldwide*. Lyon: International Agency for Research on Cancer, 2004.
3 Garcia M, Jemal A, Ward EM, Center MM, Hao Y, Siegel RL, et al. *Global cancer facts and figures 2007*. Atlanta, GA: American Cancer Society, 2007.
4 Blot WJ, McLaughlin JK. The changing epidemiology of esophageal cancer. *Semin Oncol* 1999;26:2-8.
5 Pohl H, Welch HG. The role of overdiagnosis and reclassification in the marked increase of esophageal adenocarcinoma incidence. *J Natl Cancer Inst* 2005;97:142-6.
6 Wei WQ, Abnet CC, Qiao YL, Dawsey SM, Dong ZW, Sun XD, et al. Prospective study of serum selenium concentrations and esophageal and gastric cardia cancer, heart disease, stroke, and total death. *Am J Clin Nutr* 2004;79:80-5.
7 Sitas F, Urban M, Stein L, Beral V, Ruff P, Hale M, et al. The relationship between anti-HPV-16 IgG seropositivity and cancer of the cervix, anogenital organs, oral cavity and pharynx, oesophagus and prostate in a black South African population. *Infect Agent Cancer* 2007;2:6.
8 Isaacson C. The change of the staple diet of black South Africans from sorghum to maize (corn) is the cause of the epidemic of squamous carcinoma of the oesophagus. *Med Hypotheses* 2005;64:658-60.
9 Rolon PA, Castellsague X, Benz M, Munoz N. Hot and cold mate drinking and esophageal cancer in Paraguay. *Cancer Epidemiol Biomarkers Prev* 1995;4:595-605.
10 Castellsague X, Munoz N, De-Stefani E, Victora CG, Castelletto R, Rolon PA. Influence of mate drinking, hot beverages and diet on esophageal cancer risk in South America. *Int J Cancer* 2000;88:658-64.
11 Sewram V, De Stefani E, Brennan P, Boffetta P. Mate consumption and the risk of squamous cell esophageal cancer in Uruguay. *Cancer Epidemiol Biomarkers Prev* 2003;12:508-13.
12 Launoy G, Milan C, Day NE, Faivre J, Pienkowski P, Gignoux M. Oesophageal cancer in France: potential importance of hot alcoholic drinks. *Int J Cancer* 1997;71:917-23.

Eating a light diet during labour

Does not seem to worsen obstetric outcomes



BSIP VILLAREAL/SPL

RESEARCH, p 880

Soo Downe director of ReaCH, Research in Childbirth and Health Group (ReaCH), University of Central Lancashire, Preston PR1 2HE
sdowne@uclan.ac.uk

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

Cite this as: *BMJ* 2009;338:b732
doi: 10.1136/bmj.b732

The obstetric textbook *Midwifery by Ten Teachers*, published in 1931, states that "the patient should be encouraged to take light food during the first stage of labour."[1] In 2000, the *Guide to Effective Care in Pregnancy and Childbirth* noted, "that food and drink should be withheld once labour has commenced is almost universally accepted in hospital care."[2]

In the linked randomised controlled trial, O'Sullivan and colleagues assess the effect of allowing women to eat a light diet during labour on the spontaneous vaginal delivery rate.[3] The authors provide evidence in their introduction that professional attitudes and clinical practices in relation to eating during labour still vary greatly within and between countries. Some maternity units limit oral intake to ice chips and drinks of water for all women in labour. This is to minimise the risk of pulmonary aspiration (Mendelson's syndrome) in women who may require an emergency caesarean under general anaesthesia. Other units permit non-particulate carbohydrate intake, such as sports drinks. Yet others advocate free access to food and drink during labour, on the basis that labour requires intense physical activity, and that restriction of nutritional intake will inhibit its progress. It is generally accepted now that minimal use of general anaesthetics during labour, and the application of the correct anaesthetic technique, are the best ways to minimise the risk of Mendelson's syndrome. The most recent guidelines on intrapartum care for healthy women and babies from the National Institute for Health and Clinical Excellence (NICE) conclude that women in established labour may eat a light diet unless they have received opioids, or they develop risk factors that make a general anaesthetic more likely.[4]

However, little good quality evidence is available to support or refute this conclusion. The only Cochrane review in this general area focuses specifically on the treatment of ketosis in labour, and it found no relevant good quality trials.[5] As O'Sullivan and colleagues note, five trials of calorific intake in labour were undertaken before their study, but the results of none of them were conclusive. Given the uncertainty in this area, their study is long overdue.

These authors found no significant difference in spontaneous vaginal delivery (44% in intervention and control groups; relative risk 0.99, 95% confidence interval 0.90 to 1.08) or duration of labour.[3] The results reinforce what has already been shown in many observational studies. The study is therefore an excellent starting point for future clinical policies, but it should not stop future debate.

Clinicians may worry that the changing health profile of pregnant women, and particularly rising rates of obesity, may increase the very small but real risk of Mendleson's syndrome in the future. People who are keen to promote women's choice will note that women's views and experiences are not reported in the trial. Although a light diet during labour may have no obvious clinical benefit, women may find that the morale boost of eating and drinking is a positive component of their labour experience.

Some factors pertaining to the trial itself may limit the generalisibility of the findings. The authors based the sample size for their primary outcome on a baseline spontaneous vaginal delivery rate of 60%. In the event, the rate of spontaneous vaginal delivery in both arms of the study was 44%. This rate is very low and much lower than the United Kingdom's national average of around 65% in 2005-6, when the trial was in progress.[6] The study participants also had higher rates of caesarean section, epidural usage, and use of oxytocin for induction or augmentation of labour than is seen in many settings in the UK today. Although these data may partly be attributed to the women being nulliparous, policies and practices in the unit where the study was undertaken may have

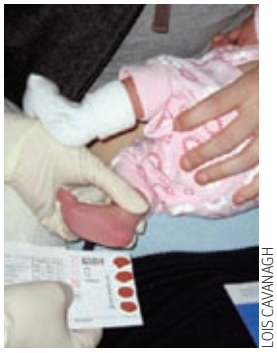
limited the incidence of spontaneous vaginal delivery to a level that was beyond the influence of nutritional policies.

O'Sullivan and colleagues' findings offer the best evidence yet in this area, even if it is in a particular type of maternity context. The results reinforce the guidance in the NICE intrapartum guidelines, but they may not fully resolve the clinical debate. Future research could investigate women's views and experiences of eating and drinking in labour, and the effect of a policy of a light diet on outcomes in settings where rates of normal birth, and of intrapartum interventions, are likely to be closer to the national average.

1 Berkeley C, Fairbairn JS, White C, eds. *Midwifery by ten teachers*. 4th ed. London. Arnold, 1931.
2 Enkin M, Keirse MJNC, Neilson J, Crowther C, Duley L, Hodnett E, et al, eds. *A guide to effective care in pregnancy and childbirth*. Oxford: Oxford University Press, 2000.
3 O'Sullivan G, Liu B, Hart D, Seed P, Shennan A. Effect of food intake during labour on obstetric outcome: randomised controlled trial. *BMJ* 2009;338:b784.
4 National Institute of Clinical Excellence. *Intrapartum care—care of healthy women and their babies during childbirth*. 2007. www.nice.org.uk/nicemedia/pdf/IPCNICEGuidance.pdf.
5 Toohill J, Soong B, Flenady V. Interventions for ketosis during labour. *Cochrane Database Syst Rev* 2008;(3):CD004230.
6 Information Centre for Health and Social Care. *NHS Maternity Statistics, England: 2005-06*. 2007. http://www.ic.nhs.uk/statistics-and-data-collections/hospital-care/maternity/nhs-maternity-statistics-2005-06.

Screening for MCAD deficiency in newborns

Saves lives and is now offered to every baby in England and Northern Ireland



LOIS CAVANAGH

Clodagh Loughrey consultant chemical pathologist, Belfast Health and Social Care Trust, Belfast City Hospital, Belfast BT9 7AB
clodagh.loughrey1@belfasttrust.hscni.net
Michael J Bennett director, metabolic disease laboratory, Children's Hospital of Philadelphia, Department of Pathology and Laboratory Medicine, Philadelphia, PA 19104-4399, USA

Competing interests: None declared.
Provenance and peer review: Commissioned; not externally peer reviewed.
Cite this as: *BMJ* 2009;338:b971
doi: 10.1136/bmj.b971

Newborn screening in England and Northern Ireland has recently been extended to include screening for medium chain acylcoenzyme A dehydrogenase (MCAD) deficiency within the entire newborn population. MCAD deficiency is the most common inherited disorder of mitochondrial fatty acid oxidation in people from northern Europe. This autosomal recessive metabolic disease affects about one in 10 000 people in the United Kingdom,[1] and it has a common mutation (985A>G) with a carrier rate of around one in 65.[2] Homozygosity for this mutation has not been found in black or Asian ethnic groups that have been screened in England, which suggests that MCAD deficiency caused by the 985A>G mutation is a disease of white ethnic origin.[2]

Individuals with undiagnosed MCAD deficiency typically present clinically with failure of fatty acid oxidation after fasting and an inability to generate energy during periods of increased energy demand. This may manifest as symptomatic hypoglycaemia through hepatic encephalopathy (similar to that seen in Reye's syndrome) to sudden unexpected infant death, which may be classified as sudden infant death syndrome.[3-5] Most cases present before 2 years of age (mean age 13 months), although a variable spectrum of disease is increasingly recognised, with both neonatal and adult presentation being reported.[6 7]

About a third of affected individuals will remain asymptomatic throughout life but may be at risk of metabolic decompensation in periods of critical energy supply—for example, during infection or prolonged fasting.

About 25% of patients with undiagnosed MCAD deficiency die at or shortly after the first presentation.[7] A further large group of undiagnosed patients presents too late to prevent long term neurological disability. However, if the diagnosis is made early, children with this deficiency can expect to lead a full and normal life, with simple dietary treatment aimed mainly at the avoidance of fasting.

Since the first descriptions of MCAD deficiency in the early 1980s, laboratory methods for diagnosis have improved greatly. The advent of tandem mass spectrometry has increased our diagnostic ability and helped augment our understanding of the clinical and biochemical spectrum of the disorder. Tandem mass spectrometry has been developed to such a degree that it can easily screen whole populations using appropriate biomarkers. During the past decade, measurement of newborn bloodspot concentrations of octanoylcarnitine by tandem mass spectrometry has emerged as a primary screen for MCAD deficiency that has high sensitivity and specificity.[8] Furthermore, multiple analytes can be measured simultaneously using the multiple reaction monitoring capabilities of the technique. Thus phenylalanine, the screening marker for phenylketonuria, can be measured at the same time as octanoylcarnitine and outdated methodology can be replaced.

Newborn screening for MCAD deficiency has already been incorporated into screening programmes in other Western nations. Guided by expert opinion from the membership of the American College of Medical Genetics, the US model for expanded newborn screening for metabolic diseases has focused on a core group of 29 conditions, 22 of which can be identified using the multiple reaction monitoring function, and 22 additional secondary targets all identified by this function.[9] Australia also has a universal programme that includes many other conditions in addition to MCAD deficiency and phenylketonuria.[10] Other European countries have comprehensive programmes with variable numbers of conditions that are screened for.[11] Outcome data from Australia have shown great benefit from the inclusion of MCAD deficiency in screening programmes.[10] Studies that have also included rare conditions, and could therefore not be justified without the inclusion of MCAD deficiency, are awaited.

A UK wide collaborative study to evaluate a pilot MCAD deficiency screening service in six sites across England was commissioned by the Department of Health in 2004.[2] This was followed by a fast track review of the newborn screening policy in 2006, and—on the basis of this pilot's findings and clinical outcome data from Australia—the National Screening Committee recommended that screening should be introduced. This led

to a statement from the health minister in February 2007 that MCAD deficiency screening would be offered to every newborn infant in England by March 2009 (www.newbornbloodspot.screening.nhs.uk).

To date, more than 1.5 million newborn babies have been screened in the six pilot sites using the heelprick sample collected at 5-8 days of age and measuring octanoylcarnitine as a single biomarker. The findings of this pilot study show the validity and usefulness of screening at this age in an NHS setting. Predictive value is high—194 babies screened positive, and 152 (78%) were confirmed to have MCAD deficiency (personal communication, Carol Dezateux, 2009). No plans have been made to introduce this screening in Wales, but Northern Ireland will implement screening in April 2009 and Scotland will follow suit by 2011.

A recent statement from the House of Lords Science and Technology Committee stated that, "government expenditure on research and development should result in more effective translation of health research into health and economic benefits in the UK."[12] The journey from discovery of MCAD deficiency, through development of new technology, epidemiological research, evaluation of pilot screening studies, the sharing of experience globally, and the development of clinical standards by multidisciplinary teams that led to the implementation of newborn screening is an excellent illustration of translational medicine, the process that "leads from evidence based medicine to sustainable solutions for public health problems."[13]

1 Blakemore AIF, Singleton H, Pollitt RJ, Engel PC, Kolvraa S, Gregersen N, et al. Frequency of the G985 MCAD mutation in the general population. *Lancet* 1991;337:298-9.
2 Khalid JM, Oerton J, Cortina-Borja M, Andresen BS, Besley G, Dalton RN, et al. UK Collaborative Study of Newborn Screening for MCADD. Ethnicity of children with homozygous c.985A>G medium-chain acyl-CoA dehydrogenase deficiency: findings from screening approximately 1.1 million newborn infants. *J Med Screen* 2008;15:112-7.
3 Kølvraa S, Gregersen N, Christensen E, Hobolth N. In vitro fibroblast studies in a patient with C6-C10-dicarboxylic aciduria: evidence for a defect in general acyl-CoA dehydrogenase. *Clin Chim Acta* 1982;126:53-67.
4 Coates PM, Hale DE, Stanley CA, Corkey BE, Cortner JA. Genetic deficiency of medium-chain acyl coenzyme A dehydrogenase: studies in cultured skin fibroblasts and peripheral mononuclear leukocytes. *Pediatr Res* 1985;19:671-6.
5 Howat AJ, Bennett MJ, Variend S, Shaw L. Deficiency of medium chain fatty acylcoenzyme A dehydrogenase presenting as the sudden infant death syndrome. *BMJ* 1984;288:976.
6 Rinaldo P, Matern D, Bennett MJ. Fatty acid oxidation disorders. *Annu Rev Physiol* 2002;64:477-502.
7 Grosse SD, Khoury MJ , Greene CL, Crider KS, Pollit RJ.The epidemiology of medium chain acyl-CoA dehydrogenase deficiency: an update. *Genet Med* 2006;8:205-12.
8 Chace DH, Hillman SL, Van Hove JL, Naylor EW. Rapid diagnosis of MCAD deficiency: quantitative analysis of octanoylcarnitine and other acylcarnitines in newborn blood spots by tandem mass spectrometry. *Clin Chem* 1997;43:2106-13.
9 Watson MS, Mann MY, Lloyd-Puryear MA, Rinaldo P, Howell RR, eds. Newborn screening: towards a uniform screening panel and system. *Genet Med* 2006;8:1-252.
10 Wilcken B, Haas M, Joy P, Wiley V, Chaplin M, Black C, et al. Outcome of neonatal screening for medium-chain acyl-CoA dehydrogenase deficiency in Australia: a cohort study. *Lancet* 2007;369:37-42.
11 Bodamer OA, Hoffmann GF, Lindner M. Expanded newborn screening in Europe 2007. *J Inherit Metab Dis* 2007;30:439-44.
12 House of Lords Science and Technology Committee. *Genomic medicine.* 2008. www.parliament.uk/documents/upload/stGMGovernmentResponse.pdf.
13 Lean MEJ, Mann JI, Hoek JA, Elliot RM, Schofield G. Translational research: from evidence-based medicine to sustainable solutions for public health problems. *BMJ* 2008;337:a863.

Practice based commissioning in the UK

Reinvigoration will require more than just extra funding

Stephen Gillam general practitioner, Luton LU1 1HH
Richard Q Lewis director, Ernst and Young LLP, London SE1 2AF

Competing interests: RQL acted as an adviser to the King's Fund practice based commissioning team and as a consultant to the Department of Health in relation to the NHS next stage review.
Provenance and peer review: Commissioned; externally peer reviewed.

Cite this as: *BMJ* 2009;338:b832
doi: 10.1136/bmj.b832

An editorial in the *BMJ* a year ago asked whether practice based commissioning was "the sick man of the NHS reforms."[1] Practice based commissioning has been a central part of the government's health policy since April 2005, when interested practices were first entitled to indicative budgets. The paucity of achievements described in a recent assessment by the King's Fund suggests that the health of this patient is little improved.[2] The government has recently clarified its vision for practice based commissioning, but will this be enough to deal with its persistent weaknesses?[3]

Commissioning is a mechanism for managing financial risk while matching services supplied to patients needs (or demands) in a quasi-market where patients do not pay for services directly. However, nearly two decades of experimentation in the English NHS have provided little evidence that any form of commissioning has greatly affected hospital services. Commissioning led by primary care has delivered some benefits in primary and intermediate care and some improvements in the responsiveness of elective hospital care, but are they sufficient to justify its continued existence?[4 5]

Primary care commissioning capitalises on the pivotal role of general practitioners as "gatekeepers" to hospital services and their supposed knowledge of local services. Budgetary responsibilities were aligned with clinical decision making when the prototype, general practitioner fundholding, was introduced in 1990.[6] Fundholding harnessed the entrepreneurial flair of general practitioners through financial incentives to reduce unnecessary use of care, promote new community based services, and negotiate lower prices for and faster access to hospital treatment. Over time, concerns about the inequitable nature of fundholding and its high transaction costs led to the evolution of more collective forms of primary care commissioning.

The King's Fund researchers undertook wide ranging interviews within four primary care trusts, and their findings echo those of an earlier report from the Audit Commission.[7] Practice based commissioning is stalling—a view that is implicitly acknowledged by the government in Lord Darzi's review last summer.[8]

Collaborative working between practices and their local primary care trusts has improved, but signs of service development remain few—although they have recently increased.[9] Of course, practice based commissioning is a work in progress, and advances may be imminent. On the other hand, the King's Fund's report

may underestimate many factors that weaken interest in practice based commissioning. For practices, the absence of strong incentives in the face of increasing financial insecurity means that the imperatives of the quality and outcomes framework may take priority. For primary care trusts, the latest developmental preoccupation is "world class commissioning," a multifaceted programme intended to increase their strategic effectiveness,[10] which is diverting their interest, in the short term at least, from supporting practice consortiums.

However, all manifestations of primary care commissioning have been beset by common weaknesses. These include a lack of clinical engagement, organisational immaturity, insufficient support from management, limited public involvement or accountability, and lack of information on which to base commissioning decisions.[11] The right formula with which to tackle these weaknesses remains, as yet, stubbornly out of reach. This raises the question of whether these deficits are simply intrinsic.

So where do we go from here? The King's Fund team is judiciously pragmatic and proposes a "matrix" model that recognises the multilayered nature of commissioning and places different responsibilities at different levels. This would give general practitioners real budgets while retaining population wide commissioning with primary care trusts. Other "solutions" abound. The Royal College of General Practitioners' proposal for practice federations (associations of practices and community primary care teams—for example, in the form of a social enterprise or limited company—that aim to develop health services) may provide much needed critical mass and the ability to develop expertise.[12] There may be a role for general practitioners with a special interest in public health and commissioning to strengthen management capacity.[13]

New "integrated care organisations" are about to be piloted.[14] These will offer real budgets to practices in return for the responsibility to manage health and population care by providing or commissioning new service models that unify primary care, community care, social care, and some forms of secondary care. Integrated care organisations offer the prospect of much stronger incentives for general practitioners and other professionals to shape local services.

Practice based commissioning is clearly not about to be dismantled, even with a change of government, but its next iteration needs to deliver more. The Department of Health has reconfirmed its commitment to practice based commissioning and has clarified practices' entitlements to timely information, management support, local incentives, and rapid decision making by primary care trusts. An investment of £1m (€1.1m; $1.4) has been made to pump prime practical support for practice based commissioners and their primary care trusts. These initiatives are a welcome starting point. But if tangible results remain elusive, evidence based policy makers will wonder whether this patient needs palliative care not reinvigoration.

1 Lewis R, Mays N, Curry N, Robertson R. Implementing practice based commissioning. *BMJ* 2007;335:1168.
2 Curry N, Goodwin N, Naylor C, Robertson R. *Practice-based commissioning. Reinvigorate, replace or abandon?* King's Fund, 2008. www.kingsfund.org.uk/publications/the_kings_fund_publications/pbc.html.
3 Department of Health. *Clinical commissioning: our vision for practice-based commissioning.* 2009. www.dh.gov.uk/en/Managingyourorganisation/Commissioning/Practice-basedcommissioning/DH_095694.
4 Smith JA, Mays N, Dixon J, Goodwin N, Lewis R, McClelland S, et al. *A review of the effectiveness of primary care-led commissioning and its place in the UK NHS*. The Health Foundation, 2004. www.library.nhs.uk/commissioning/ViewResource.aspx?resID=273592.
5 Mays N, Mulligan J-A, Goodwin N. The British quasi market in health care: a balance sheet of the evidence. *J Health Serv Res Policy* 2000;5:49-58.
6 Secretaries of State for Health. *Working for patients*. Cm 555. London: Stationery Office, 1989.
7 Audit Commission. *Putting commissioning into practice; implementing practice based commissioning through good financial management*. 2007. www.audit-commission.gov.uk/Products/NATIONAL-REPORT/67664124.../Puttingcommissioningintopractice_22Nov2007REPORT.pdf.
8 Department of Health. *NHS next stage review, our vision for primary and community care.* 2008. www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_085937.
9 Department of Health, Practice Based Commissioning GP Survey, 5 December 2009 (http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsStatistics/DH_091372 last accessed 6 March 2009).
10 Department of Health. *World class commissioning: competencies*. 2007. www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_080958.
11 Lewis R, Gillam S. Quality in primary care commissioning. *Qual Primary Care* 2007;15:367-72.
12 Cohen D. RCGP recommends federations but dismisses polyclinics. *BMJ* 2007;335: 585. doi:10.1136/bmj.39342.589294.DB.
13 Bradley S, McKelvey SD. General practitioners with a special interest in public health; at last a way to deliver public health in primary care. *J Epidemiol Community Health* 2005;59:920-3.
14 Department of Health. *Integrated care pilot programme—prospectus for potential pilots.* 2008. www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_089338.

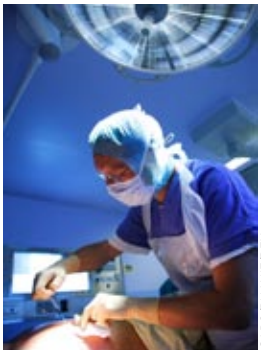
Minimally invasive surgery for breast cancer

May be trading better cosmetic outcomes for worse rates of cure

Both oncological principles and aesthetic outcomes need to be considered when planning surgery for breast cancer. As breast conserving therapy has evolved over the past 30 years from a radical idea to an accepted standard, and as options for breast reconstruction have increased, the surgical literature has become increasingly devoted to new techniques to improve cosmetic outcomes. "Minimally invasive breast surgery" and "oncoplastic breast surgery" are the new buzzwords among breast surgeons. But what do they mean? And more importantly, how should such procedures be evaluated?

Oncoplastic surgery has been described as "the seamless joining of the extirpative and reconstructive aspects of breast surgery performed by a single surgeon,"[1] and those in the field agree that the purpose of oncoplastic surgery is to improve cosmetic outcomes.[2] Minimally invasive breast surgery includes techniques of breast conserving surgery, mastectomy, sentinel node biopsy, and axillary dissection. Although initially breast conserving surgery and sentinel node

Cite this as: *BMJ* 2009;338:b557
doi: 10.1136/bmj.b557



GARO/PHANIE/REX

Monica Morrow chief of breast service and Anne Burnett Windfohr chair in clinical oncology, Memorial Sloan Kettering Cancer Center, New York, NY 10065, USA
morrowm@mskcc.org

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

biopsy were themselves considered minimally invasive, the term has more recently been used to describe endoscopic breast and axillary surgery,[3 4] which aim to improve cosmetic outcomes by using a smaller incision. Oncoplastic surgery and minimally invasive surgery are separate but overlapping fields, and advocates of both disciplines could be asked the same key question—what level of efficacy or oncological safety must be demonstrated before advocating and performing a new procedure outside the setting of a clinical trial? In other words, just because something is technically possible is it necessarily appropriate?

One recent case series described 33 patients who had endoscopic assisted skin sparing mastectomy.[3] Patients had extensive ductal carcinoma in situ, small invasive cancer with an extensive intraductal component, or small multicentric cancer, but no further details about selection criteria were provided. No information was given on the status of the anterior or posterior margins, a topic of particular interest with this technique, because 24% of the patients required excision of the nipple as a result of a positive margin. The absence of any local recurrences at 51 months was reassuring but hardly definitive in such a small sample. In spite of these uncertainties, the authors concluded that the technique provides excellent cosmetic results without reducing oncological safety, and that the procedure is suitable for patients who cannot have breast conserving therapy.

The study was reported by the lay press under the title "Breast cancer surgery that reduces risk and improves cosmetic appearance: new research."[5] Such a title suggests to patients that this is a proved technique that they should seek.

A second case series of 20 patients described a technique of endoscopic partial mastectomy for inner quadrant tumours that used a transaxillary, retromammary approach.[4] The minimum resected volume of the breast was 25%, and all margins were negative. The authors concluded that the procedure could become a single standard method for conserving the breast.

In oncoplastic surgery large resections are carried out and the breast tissue is often rearranged to minimise the resulting defect. This may make it difficult or impossible to deliver a boost dose of radiation to the tumour bed, a procedure that has been shown in a randomised trial to reduce the incidence of local failure.[6] It is unclear whether this will have a negative effect on local control given the small number of patients treated and the relatively short follow-up.[2] But local failure rates as high as 7-9% at five years[7 8] in patients treated with oncoplastic techniques raise concern when local recurrence at 10 years in multi-institutional studies is less than 7%.[9] Randomised controlled trials have shown that neoadjuvant chemotherapy increases the number of women who can have breast conserving therapy without increasing local recurrence or decreasing survival,[10] and that patient ratings of the cosmetic outcome of conventional breast conserving therapy and breast reconstruction after skin sparing mastectomy are generally high. Insisting that new procedures aimed at improving cosmetic outcomes are evaluated for oncological safety therefore does not deprive women of treatment options.

Failure to demand a rigorous evaluation of oncological outcomes as well as cosmetic ones runs the risk of losing some of the gains in survival seen in the past decade. Until relatively recently, local treatment was dismissed as unimportant to survival. The Early Breast Cancer Trialists Collaborative Group overview shows that for every four local recurrences avoided at five years, one death from breast cancer is prevented at 15 years.[11] As systemic treatment continues to improve, uncontrolled local disease will possibly have an even greater effect on mortality.

We must ensure that surgical approaches designed to improve cosmetic outcomes do not increase local failure and the risk of subsequent death from breast cancer. In addition, the developing fields of oncoplastic surgery and minimally invasive breast surgery require rigorous assessment of patient reported outcomes.[12] This will ensure that new procedures that potentially prolong surgery, that may increase the use of resources, and that are associated with uncertain oncological safety actually improve outcomes that are important to patients.

The local treatment of breast cancer is based on the results of numerous high quality clinical trials and is therefore a model for evidence based care. As we attempt to advance from good to great cosmetic outcomes it is important that we remember this.

1 Malycha PL, Gough IR, Margaritoni M, Deo SV, Sandelin K, Buccimazza I, et al. Oncoplastic breast surgery: a global perspective on practice, availability, and training. *World J Surg* 2008;32:2570-7.
2 Rainsbury RM. Surgery insight: oncoplastic breast-conserving reconstruction—indications, benefits, choices and outcomes. *Nat Clin Pract Oncol* 2007;4:657-64.
3 Ito K, Kanai T, Gomi K, Watanabe T, Ito T, Komatsu A, et al. Endoscopic-assisted skin-sparing mastectomy combined with sentinel node biopsy. *Aust NZ J Surg* 2008;78:894-8.
4 Yamashita K, Shimizu K. Transaxillary retromammary route approach of video-assisted breast surgery enables the inner-side breast cancer to be resected for breast conserving surgery. *Am J Surg* 2008;196:578-81.
5 Medical News Today. *Breast cancer surgery that reduces risk and improves cosmetic appearance: new research.* 2008. www.medicalnewstoday.com/articles/124117.php.
6 Bartelink H, Horiot JC, Poortmans PM, Struikmans H, Van den Bogaert W, Fourquet A, et al. Impact of a higher radiation dose on local control and survival in breast-conserving therapy of early breast cancer: 10-year results of the randomized boost versus no boost EORTC 22881-10882 trial. *J Clin Oncol* 2007;25:3259-65.
7 Clough KB, Lewis JS, Couturaud B, Fitoussi A, Nos C, Falcou MC. Oncoplastic techniques allow extensive resections for breast-conserving therapy of breast carcinomas. *Ann Surg* 2003;237:26-34.
8 Nos C, Fitoussi A, Bourgeois D, Fourquet A, Salmon RJ, Clough KB. Conservative treatment of lower pole breast cancers by bilateral mammoplasty and radiotherapy. *Eur J Surg Oncol* 1998;24:508-14.
9 Wapnir IL, Anderson SJ, Mamounas EP, Geyer CE Jr, Jeong JH, Tan-Chiu E, et al. Prognosis after ipsilateral breast tumor recurrence and locoregional recurrences in five national surgical adjuvant breast and bowel project node-positive adjuvant breast cancer trials. *J Clin Oncol* 2006;24:2028-37.
10 Fisher B, Bryant J, Wolmark N, Mamounas E, Brown A, Fisher ER, et al. Effect of preoperative chemotherapy on the outcome of women with operable breast cancer. *J Clin Oncol* 1998;16:2672-85.
11 Clarke M, Collins R, Darby S, Davies C, Elphinstone P, Evans E, et al. Effects of radiotherapy and of differences in the extent of surgery for early breast cancer on local recurrence and 15-year survival: an overview of the randomised trials. *Lancet* 2005;366:2087-106.
12 Pusic AL, Chen CM, Cano S, Klassen A, McCarthy C, Collins ED, et al. Measuring quality of life in cosmetic and reconstructive breast surgery: a systematic review of patient-reported outcomes instruments. *Plast Reconstr Surg* 2007;120:823-37; discussion 38-9.