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Protecting breast feeding from breast milk substitutes

The WHO code is widely violated and needs monitoring and supporting

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In all societies breast feeding is one of the most important measures to improve child health. An important component of the global effort to protect breast feeding is the WHO's international code of marketing of breast milk substitutes. In this week's issue an interagency group on breast feeding monitoring produces compelling evidence that the code is widely violated (p 1117).[1]

The World Health Organisation estimates that 1.5 million deaths a year could be prevented by effective breast feeding protection.[2] A recent systematic review estimated that in a low income country with a postneonatal mortality rate of 90 per 1000 children, artificial feeding would produce an excess of postneonatal deaths per million births ranging from 11 290 (13%) to 112 900 (59%) at prevalences of artificial feeding at 6 months of 10% and 100% respectively.[3] In the industrialised world a failure to breast feed increases the risk of childhood diseases,[4] impairs child development,[5] and may increase the risk of adult disease.[6]

The international code, devised in 1981, reaffirmed in 1996, and endorsed by the manufacturers, was developed to protect mothers and health workers from commercial pressure by manufacturers of breast milk substitutes. It forbids provision of free samples to mothers or health facilities (except for professional research), because of the negative impact on breast feeding.[7] It also forbids inducements to health workers, because recipients are more likely to promote a particular product[8] and remain passive in promoting breast feeding.

Since the code was introduced widespread violations by companies have been reported by various agencies, but companies have dismissed such evidence as unreliable, anecdotal, or distorted by activists. The report in this week's issue is a large, systematic, and random survey of mothers and health professionals that quantifies the level of violations in Bangladesh, Poland, Thailand, and South Africa.[1] It seems to vindicate previous reports by the International Baby Food Action Network.[9] One tenth of all mothers interviewed (range 0-26%) and a quarter of all facilities visited (8-50%) had received free samples of milk, bottles, or teats—none of them for research purposes. Violating information was received by 30% of health facilities (15-56%), and 11% of health workers surveyed had received gifts (2-18%), three quarters of which bore a company brand name.

How reliable are these estimates? While minor methodological criticisms may be made, the study used random sampling, several interviewers, subset validation, and had internal consistency (the country with legislation had the least free samples and that with no code had suffered the most violations. The study probably underestimates the size of the problem in developing countries, where failure to breast feed carries the highest risk of mortality. From the 48 least developed countries,[10] only Bangladesh was included, a country with a good recent record on breast feeding protection.

It will be depressing, but predictable, if manufacturers dismiss this paper. Like tobacco companies, their promotional activities may be regulated only when they face substantial claims for damages from consumers. Meanwhile there is much that individual countries and monitoring agencies can do.

Firstly, governments should incorporate the articles of the WHO code into national legislation. Encouragingly, China, Brazil, and India (nearly half the world's population) have incorporated most of the code into legislation, although by September 1997 only 17 countries had approved laws that put them fully in compliance with the code.[11] Hopefully the British government will listen to the recently formed UK Baby Milk Law Working Group and bring the code into national law.

Secondly, monitoring for overt violations should be more systematic. Even when overt violations are documented in countries with legislation, legal action against companies is often too difficult to implement. Nevertheless, systematic monitoring for violations is important for evaluating trends, mobilising public opinion, and deterring overt promotional activities.

The marketing departments of manufacturers are also innovative. Anecdotally, covert promotional methods have been reported, especially targeted at doctors, who have an important effect on the timing and choice of a breast milk substitute: unsigned, non-prescription slips with specific company names for mothers with the ill defined "insufficient milk syndrome"; free vitamin drops in containers identical to formula milk products; "anonymous" donations to national paediatric societies; complimentary textbooks or journals, particularly for residents; funds to attend scientific meetings; and electioneering support for candidates in national society elections known to be passive to breast feeding promotion and code monitoring. Future monitoring should evaluate such covert promotional activities.

Thirdly, doctors must be aware that companies try to gain "endorsement by association," or at least passivity towards their products, from prestigious national bodies. Interestingly, neither the UK's Royal

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College of Paediatricians and Child Health nor the British Nutrition Foundation, both of which accept financial support from milk substitute companies, were part of the interagency group. By contrast, in 1994, while hosting the eighth Asian Congress of Paediatrics, the Indian Academy of Paediatrics declined a large donation from the industry. Such voluntary decisions by national paediatric societies represent a welcome move by paediatricians to protect breast feeding.

Fourthly, as with antismoking campaigns, legislation and monitoring are only part of a broader strategy needed to protect mothers, regardless of how they feed their infants. Positive approaches to breast feeding for mothers are equally (if not more) crucial. These measures include training midwives and doctors in lactation counselling (including guidance for HIV positive mothers, for whom breast feeding may be contraindicated), "breast feeding advertisements," extending the "baby friendly" hospital initiative, and financial support for advocacy groups that support health promotion for mothers. Such positive attempts to protect breast feeding, and to counter company propaganda, remain a challenge largely unfulfilled by health workers and professional bodies.

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Neurological channelopathies

Dysfunctional ion channels may cause many neurological diseases

Disorders of ion channels (channelopathies) are increasingly being identified, making this a rapidly expanding area of neurology. Ion channel function may be controlled by changes in voltage (voltage gated), chemical interaction (ligand gated), or by mechanical perturbation. The first disorders recognised as channelopathies were the voltage gated channelopathies causing inherited muscle diseases: the non-dystrophic myotonias and familial periodic paralyses. Paramyotonia congenita is due to mutations in the gene coding for the α1 subunit of the sodium channel, while Thomsen's disease (autosomal dominant myotonia congenita) and Becker's disease (autosomal recessive myotonia congenita) are allelic disorders associated with mutations in a gene coding for skeletal muscle chloride channel. Familial hyperkalaemic periodic paralysis is due to mutations in the same sodium channel gene as that affected in paramyotonia congenita, while familial hypokalaemic periodic paralysis results from mutations in the gene coding for the α1 subunit of a skeletal muscle calcium channel.[1]

The first demonstration that channelopathies could affect nerves as well as muscles came in 1995, when researchers discovered that episodic ataxia type 1, a rare autosomal dominant disease, results from mutations in one of the potassium channel genes.[2] The impairment of potassium channel function, which normally limits nerve excitability, results in the rippling of the muscles (myokymia) of the face and limbs seen in this disease. Episodic ataxia type 2, also autosomal dominant, is not associated with myokymia but responds dramatically to acetazolamide, an unexpected feature it shares with many channelopathies. The suspicion that it too might be a channelopathy was confirmed when mutations in a gene coding for the α1 subunit of a brain specific calcium channel were found.[3] Mutations in this same gene can also cause familial hemiplegic migraine and spinocerebellar degeneration type 6.[4] It is unclear how different mutations of the same gene can give rise to such different phenotypes. In the case of myotonia congenita and familial hyperekplexia, point mutations in the same gene can result in either autosomal recessive or dominant inheritance.

Ligand gated channelopathies that have recently been described include familial startle disease, which is due to due to mutations of the α1 subunit of the glycine receptor, and dominant nocturnal frontal lobe epilepsy, which is due to mutations of the α4 subunit of the nicotinic acetylcholine receptor.[5,6] A gene for familial paroxysmal choreoathetosis has been mapped to a region of chromosome 1p where a cluster of potassium channel genes is located.[7]

Channelopathies may be acquired as well as inherited. Recognised causes include toxins and autoimmune phenomena. The marine toxin ciguatoxin, which contaminates fish and shellfish, is a potent sodium channel blocker that causes a rapid onset of numbness, intense paraesthesia and dysaesthesia, and muscle

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weakness.[8] Antibodies to peripheral nerve potassium channels may result in neuromyotonia (Isaac's syndrome).[9] Lambert-Eaton myasthenia, which is associated with small cell carcinoma of the lung in 60% of cases, is caused by autoantibodies directed against a presynaptic calcium channel at the neuromuscular junction and against multiple calcium channels expressed by lung cancer cells.[10] The neurophysiological abnormalities seen in Guillain-Barré syndrome, chronic inflammatory demyelinating polyneuropathy, and multiple sclerosis, traditionally regarded as the result of demyelination, could also be explained by sodium channel dysfunction. The transient nature of some symptoms in multiple sclerosis and the rapid recovery that is sometimes seen in multiple sclerosis and Guillain-Barré syndrome are more consistent with a temporary channelopathy mediated by antibodies than a longer process of demyelination and remyelination. In fact, cerebrospinal fluid from patients with Guillain-Barré syndrome or chronic inflammatory demyelinating polyneuropathy does cause a transient decrease in neuronal sodium currents.[11 12]

All these channelopathies have surprisingly similar clinical features. Typically, there are paroxysmal attacks of paralysis, myotonia, migraine, and ataxia precipitated by physiological stresses. A channelopathy may cause an abnormal gain of function (such as myokymia, myotonia, and epilepsy) or an abnormal loss of function, (such as weakness or numbness) depending on whether loss of channel function leads to excessive membrane excitability or to membrane inexcitability.

Ion channels consist of multiple subunits, each with very similar structure but different electrophysiological characteristics. The differing neuronal expression and combination of these subunits into complexes gives rise to enormous diversity in the properties and distribution of ion channels, which is reflected in the variety of diseases that make up the neurological channelopathies. Many of the channelopathies respond predictably to membrane stabilising drugs such as mexilitine, as well as to acetazolamide. The neuronal specificity of ion channels allows the potential for targeted drug therapy akin to the selective receptor agonists and antagonists currently available: 3,4-diaminopyridine, a potassium channel blocker, can relieve symptoms in patients with Lambert-Eaton syndrome and improves leg strength in patients with multiple sclerosis.[13 14] Specific channel modulating drugs are currently being developed for migraine, chronic pain, and cardiac dysrhythmias and these may be useful for neurological channelopathies.

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Antidepressant discontinuation reactions

Are preventable and simple to treat

Discontinuation reactions from antidepressants have been recognised since the drugs were first introduced[1] and can occur with all the major classes of antidepressants.[2 3] This phenomenon has important implications for antidepressant prescribing, particularly as these drugs are increasingly used in disorders other than depression. Nevertheless, antidepressant discontinuation reactions have received little systematic study and many clinicians are unaware of them.[4]

The incidence of discontinuance reactions is unclear owing to the lack of research and of an accepted definition of what constitutes a discontinuation reaction. Antidepressants vary in their propensity to cause reactions,[5] and reactions are more common after abrupt termination and longer courses of treatment.[6 7] Given this background, the reported incidence has varied from 0%[6] to 100%.[8] One of the few double blind placebo controlled studies found that in the two weeks after a 12 week treatment period adverse events, mostly mild or moderate, occurred in 35% of patients treated with paroxetine compared with 14% of controls.[9] Although this study was carried out in patients with panic disorder, with certain

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antidepressants reactions probably occur in a significant minority of patients of all diagnostic categories when they stop treatment.

Discontinuation reactions are distinct from recurrence of the primary psychiatric disorder. They usually start abruptly within a few days of stopping the antidepressant (or, less commonly, of reducing its dose) and are short lived, resolving within one day to three weeks.[5 6] In contrast, depressive relapse is uncommon in the first week after stopping an antidepressant: symptoms tend to build up gradually and become chronic. Discontinuation symptoms are varied and differ depending on the class of antidepressant. Common symptoms include gastrointestinal disturbance (nausea, abdominal pain, diarrhoea), sleep disturbance (insomnia, vivid dreams, nightmares), general somatic distress (sweating, lethargy, headaches), and affective symptoms (low mood, anxiety, irritability). Although there is some overlap with anxiety and depressive disorders,[2] many discontinuation symptoms are distinct. With the serotonin reuptake inhibitors the commonest symptom appears to be dizziness/light headedness, with sensory abnormalities—including numbness, paraesthesia, and electric shock-like sensations—also well recognised.[6] Discontinuation reactions usually resolve within 24 hours of reinstating antidepressant treatment,[6] whereas in depressive relapse the response is slower.

Discontinuation symptoms do not in themselves indicate drug dependence. Dependence is a syndrome,[10] and diagnosis requires several other features, such as tolerance, inability to control drug use, primacy of drug taking behaviour, and continued use despite harmful consequences. Antidepressants are not associated with these other features and are not drugs of dependence. The common lay belief that antidepressants are addictive probably contributes to the significant undertreatment of depressive illness.[11] It is important not to foster this belief inadvertently—one reason that "discontinuation reaction" is a better term than "withdrawal reaction."

Discontinuation reactions are clinically important for several reasons. Firstly, although most are mild and short lived, a minority are severe or chronic and cause considerable morbidity.[2] Secondly, if the reaction is misdiagnosed, inappropriate treatment may result. For example, a reaction after stopping antidepressants may be misdiagnosed as a relapse of the psychiatric illness, leading to unnecessary reinstatement of the antidepressant. A reaction after covert non-compliance may lead to the erroneous conclusion that a higher dose or a switch to another antidepressant is needed. Many discontinuation symptoms are physical and may prompt investigations to identify the cause. These scenarios waste money, put the patient at unnecessary risk, and lead to a more negative prognosis that may have social implications. Finally, if the phenomenon is not explained, the patient who recognises the association between the antidepressant and the discontinuation symptoms may comply poorly with further antidepressant treatment.

To reduce the likelihood of discontinuation reactions the *British National Formulary* recommends that antidepressants that have been continuously prescribed for eight weeks or more should not be stopped abruptly but gradually reduced over four weeks.[12] Given current knowledge this seems reasonable, though anecdotal reports suggest that tapering may be unnecessary when switching between serotonin reuptake inhibitors.[7] Patients need to be educated that antidepressants are non-addictive, doses must not be omitted, and courses not stopped abruptly. If recognised, discontinuation reactions are not a serious problem. Clinicians should consider the diagnosis when faced with unexpected physical or psychological symptoms in patients who have just stopped taking antidepressants or are apparently still on treatment: only a few days' medication needs to be missed to precipitate a reaction, and antidepressant non-compliance is common and often covert unless specifically inquired about.

If antidepressant treatment is still required, restarting the antidepressant will rapidly resolve the discontinuation symptoms. If antidepressants are no longer clinically indicated treatment depends on severity. Most cases are mild and require only reassurance. Symptomatic treatment, such as a short course of a benzodiazepine for insomnia, may help with more troublesome symptoms. If severe the antidepressant should be restarted and tapered down gradually—occasionally very gradually. In summary therefore, discontinuation reactions are a significant problem only when strategies for prevention and recognition are ignored.

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Doctors in training: wasteful and inefficient?

Not if the training is properly structured and supervised

The United States government seems almost certain to decrease funding for graduate medical education in teaching hospitals. At the same time competition in healthcare delivery is forcing hospitals to cut costs. If teaching medical graduates increases hospital costs and if trainees cause waste and inefficiency in patient care hospitals may choose to eliminate their teaching programmes. As hospitals throughout the developed world are being subjected to market pressures they need to know the contribution and the costs of training doctors. Is the widely held belief that doctors in training cause waste and inefficiency true?

The costs of a graduate medical education programme include the salaries of trainees and their teachers, administrative support, space, supplies, and equipment. The programme for 30 residents in internal medicine at this hospital costs about $5m (£3.3m). In the United States the revenue for residency training programmes comes from several sources: the federal government's Medicare programme, income generated by the teaching faculty in patients' fees and research grants, and sometimes endowment funds. For our programme, the total revenue is about $6.5m (£4.3m), almost 90% of which comes from Medicare. The surplus is mainly due to an indirect reimbursement for graduate medical education from Medicare. This payment to teaching hospitals (begun in 1983) was intended to cover the indirect costs of education, in particular the greater need for medically trained faculty and other staff, the administrative and space burdens of the programmes, the greater proportion of patients with low income because of inner city locations, and the less efficient patient care provided by trainees.[1,2]

Proponents of the current system would claim that our hospital has no surplus because it is consumed by the indirect costs of the programme. Others, after careful analysis, have concluded that the current indirect reimbursement formula provides more than what is needed to cover the indirect expenses of a training programme.[2] If this analysis is correct many teaching hospitals have probably used their surplus to expand the numbers of doctors on their staff or to support research. The proposed cuts in Medicare will force important reductions at these institutions.

Faced with these cuts in federal reimbursement, hospitals can legitimately debate whether the financial burden of the residency programmes is worth bearing. Prominent in such a debate is the issue of whether patient care is wasteful and inefficient when performed by trainee doctors. Teaching hospitals may not be able to survive in a competitive market if they are saddled with both reduced federal funding for education and higher costs through residents providing inefficient patient care. Most studies show that care in teaching hospitals is more expensive than in non-teaching hospitals.[3-5] However, to compare hospitals properly adjustments must be made for case mix, severity of disease, and the socioeconomic characteristics of patients. These adjustments are difficult to make,[6,7] but when attempts are made the difference between teaching and non-teaching hospitals is diminished.[8,9]

Our hospital is a 625 bed hospital for adults in urban Minneapolis, Minnesota, which has a high proportion of managed care patients. Our residency programme is successful, as shown by the fact that our residents have scored in the top 10% of all residency programmes in the American Board of Internal Medicine certifying examination for the past seven years. Our medicine service is organised into a teaching and non-teaching component, although there is no geographical separation and all patients use the same nursing stations and ancillary services. Patients are admitted to the teaching service by a designated group of 90 admitting faculty internists. Since the teaching service has a limited capacity, however, and is often closed to new admissions, all the admitting physicians often have patients on the non-teaching service whom they care for directly without help from the residents. In an unpublished study in 1985 we showed that an aggregate of patients with pneumonia, heart failure, and acute myocardial infarction admitted to the teaching service had equivalent hospital charges and lengths of stay to those admitted to the non-teaching service after we had adjusted crudely for severity of disease. In 1990 Udvarhelyi et al published a study from our institution looking only at acute myocardial infarction.[10] After careful and detailed adjustment for disease severity and demographic factors, they found that the patients admitted to the teaching service had slightly shorter mean lengths of stay and charges. In 1991 we studied a larger group of diagnoses and after crude adjustment we again found that the patients admitted to the teaching service had slightly shorter lengths of stay and lower charges.

Although our studies were not randomised clinical trials, they support the concept that under some circumstances residents can help provide care that is as cost effective as care provided by staff physicians working alone. Patient care that is provided mostly by residents, with only distant supervision, however, cannot achieve both high quality and cost effectiveness. If attending physicians are competent and appropriately supervise the residents and if the residents have a controlled workload, excellent formal education, and a supportive environment, patient care can be of high quality and efficiency.

It may not be easy to achieve these results. In a randomised trial in a single hospital Simmer et al showed that traditional resident teams provided care that was less efficient than that provided by attending staff only.[11] The challenge for each teaching institution that wants to survive is to find the changes in structure, schedules, supervision, and education that will help residents provide quality and efficiency in patient care. If the challenge is not met it will not be our residents' fault, but the fault of our institutions and our teachers.

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Rehabilitation for older people

At risk in the new NHS

Fifty years ago, in a brief but powerful paper, Marjorie Warren laid down the guiding principles of what was to become the specialty of geriatric medicine.[1] She emphasised the process of rehabilitation—to help elderly people regain their best possible functional independence. How is the rehabilitation of older people faring in the reformed NHS?

Elderly care medicine, like many other acute specialties, has come under considerable pressure. An established pattern of rising admissions, disproportionate to demographic changes, has been compounded by a steady reduction in acute hospital beds. The solution to these conflicting trends has been to press for shorter lengths of stay, making rehabilitation especially vulnerable. Two index conditions which provide an insight into contemporary rehabilitation for older people are stroke and fractured neck of femur.

The Department of Health's targets listed in the *Health of the Nation* drew particular attention to stroke, and we now know, from research, how a comprehensive stroke service should work. There is particular confidence that properly organised hospital care of stroke improves outcome. Yet, despite the opportunities for stroke patients arising from these positive findings, a recent survey of British doctors showed that disorganised stroke care prevails.[2] The inertia is partly due to the focus of health planners on emergencies and waiting lists and partly due to increased demands on general physicians and geriatricians. Even more important has been the difficulty in constructing a service contract for commissioners of stroke care; a well defined specification for the organisation and delivery of the service has proved elusive.[3]

The Audit Commission's recent report on the care of older people with fractured neck of femur emphasised that effective collaboration between elderly care physicians and orthopaedic surgeons leads to improved outcomes.[4] This view is supported by research and by audit findings. Yet two recent surveys of British orthopaedic departments showed that only a few had an effective system for shared care of patients with fractured neck of femur.[5 6] A decade ago a similar minority of districts offered formal orthogeriatric liaison.[7] This does not seem to be an interprofessional issue: most orthopaedic surgeons believe orthogeriatric liaison is desirable.[6]

Thus, in two important marker conditions, rehabilitation is still inadequate for many elderly people despite evidence of clinical effectiveness, national guidance, and widespread agreement. In a further, more general sense the provision of rehabilitation is also being undermined. In the 1980s there was a popular trend to integrate acute care and rehabilitation within single, multipurpose wards. Continued pressure for high throughput, however, has undermined this widely adopted model of care. In effect these wards have quietly withdrawn from an emphasis on rehabilitation and become dominated by providing acute care.

The erosion of rehabilitation for older people in our acute hospitals might be understandable if it was part of a strategy to develop rehabilitation at home. But this has not been the case, and widespread concern exists that rehabilitation based in the community continues to be underdeveloped and underfunded.[8] Work is now urgently needed to rehabilitate rehabilitation for elderly people. Not to do so is an injustice to older people and their families. It is also costly, as failure to achieve optimal rehabilitation increases the need for home support and risks avoidable institutional care.

What should purchasers and providers be doing to correct this? A recent policy report from the Department of Health offers a new opportunity for a collaborative re-examination of rehabilitation services.[9] Health authorities are now obliged to produce local policies and guidelines for continuing health care. The report urges health authorities to include "explicit protocols and eligibility criteria for rehabilitation." This comment is welcome, but there is a need for greater commitment from purchasers and providers and a clearer statement of their mutual responsibilities.

A simple remedy would be a return to designated rehabilitation wards designed to provide the time and space elderly people need to recover from acute illness. This approach would also help restore the threatened role of the rehabilitation nurse. A wider, more fundamental and strategic change may be required, however. A pressing need exists to determine which particular configuration of elderly care services is best for patients. For example, to what extent does rehabilitation prosper when located in community trusts compared with acute hospital trusts? Development of

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rehabilitation based in the community has particular appeal and fits in with the concept of an NHS led by primary care.[10] The new found interest in community hospitals also has potential to sustain rehabilitation for older people but needs to be developed from a policy based on opinion to one based on evidence.

Marjorie Warren described "the proper care and rehabilitation" of older people.[1] In our health service today there is little to suggest that rehabilitation is in robust health. Uncertainties remain over how the service should be commissioned, how it should be divided between primary and secondary care, and the respective roles of social services and health. A new and purposeful strategy is urgently required to prevent further drift and deterioration in rehabilitation services for older people.

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The *BMJ*'s website scales up

Now it provides free access to full text

Three years ago, it was hard to find a medical journal on the internet. Now most have websites, providing selections from their paper journals in electronic form. This week the *BMJ* joins the *Lancet* and a host of specialist journals in taking the obvious next step: providing the full text of the paper journal online. Soon most other medical journals interested in their long term survival will follow suit.

If "surfing the net" has become the defining catchphrase of the age then the wave we caught three years ago has turned out to be a tsunami. Since the world wide web was first used commercially the number of websites has grown exponentially. When the *BMJ* launched its original website in May 1995 there were fewer than 20 000 other websites. Last month the total was 2.1m.[1] The number of people online has also been growing exponentially—to an estimated 107m.[2] The widespread adoption of webTV should increase this figure by at least an order of magnitude.

The *BMJ* embraced the world wide web so avidly because it looked like fun and offered an almost miraculous escape from the limitations of paper publication. Costly and cumbersome, the printing presses and binding lines take 30 hours to churn out the 117 000 copies of the paper *BMJ* each week. The Royal Mail takes another day or two to deliver copies to most addresses in the United Kingdom. Further afield, the delays rapidly escalate—four days to get journals to continental Europe, two to three weeks to Australia. The resources we devote to increasing topicality are wasted for most of our non-UK readers.

By comparison, the electronic journal is available to all countries with world wide web access at 00.01 (GMT or BST) on the Friday preceding the cover date. The geographical reach is a marketer's dream—we are attracting hundreds of readers to the electronic journal from countries with only a handful of paper subscribers. Some 40% of the 20 000 visitors to our website each week "rarely or never see the paper journal."[3]

Unlike the paper journal, the website suffers no constraints on space. This allows us to satisfy the previously irreconcilable demands of readers (for brevity) and authors and other researchers (for detail). We have already begun to publish shorter versions of articles in the paper journal, while posting more extended accounts on the website and are considering augmenting these further. In the case of research articles, that might mean including protocols, fuller descriptions of methods, raw data and the computer programs used to analyse them, and full documentation of the peer review process.

Freed of limitations on space, we need no longer limit ourselves to publishing only a third of the letters we receive, four to five months after the paper to which they refer. Within the next week or two, comments boxes will be appended to each article on the website, allowing readers to email their comments back to us. We intend to post these on the website within 48 hours. Locating responses to articles currently entails thumbing through a stack of journals; on the website all responses will be linked electronically to the relevant article.

Web technology allows all articles on the website—and not just letters—to be found easily. A "search engine" can scour the entire site in seconds for a word, bibliographic citation, or issue date. Our searchable full text archive extends back to July 1997; by the end of this year it will run from January 1994. Readers will be able to read any article online or to print out near perfect copies of the paper version. The irritations of missing or damaged *BMJ*s in libraries, the expense and delays of using document delivery services, disappear when users

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can be assured of the existence of a full text archive. Gone is the pile of journals in offices and studies.

We don't expect readers to stop reading their paper journals, and we envisage paper and electronic versions of the *BMJ* running in tandem for the foreseeable future. Reading words on a page is much easier than on screen. Louis Rossetto, publisher of *Wired*, asks audiences to imagine living in a world of computer monitors and then coming across paper for the first time. "Wow, this is fantastic!" he suspects would be the response. "It's cheap, it's light, it's tough; I can roll it up and stick it in my pocket; I can read it on the beach, in the bath, and in bed. I can jot notes on it, tear off the bits I want: this is epoch shattering!"

Few who have trawled through the massive printed volumes of *Index Medicus* could muster up this level of enthusiasm for paper, and for the past year we have helped visitors to our website locate studies in other journals by providing them with direct access to Medline. New features devised by the National Library of Medicine allow us to extend this service. Readers will now be able to link directly from references appearing in *BMJ* articles to their Medline abstract.

While bringing ever more information to the desk top we cannot provide users with any more time to digest it. So our new site includes features to maximise the efficiency of users' visits. We currently email the journal's table of contents to 10 000 people, but from next week users will be able to opt for only articles on topics that they have nominated. The alerting email will allow links back to a page that includes not only the reference to the article in question but also details of all articles from the *BMJ* on the topic, information on relevant books and specialist journals published by the BMJ Publishing Group, and jobs from our classified supplement in the appropriate specialty. In time, we hope to link to trusted resources off site. All visitors to the site will have access to these collected resources.

Initially, our website was updated once a week, coinciding with publication of the paper journal. Over the past year we have begun to post more material between issues, and this trend is likely to continue with news and letters. Some journals are posting stories to their websites as soon as they are publishable—that is, after acceptance and technical editing. Following their lead would reduce by 2-4 months the current delay between acceptance and eventual publication in the *BMJ*. The idea of the journal as a collection of articles that rolls off the presses each week gives way to the idea of an archive, which is continually updated. So when is the moment of publication? Lawyers say, the moment when something is made public, whatever the medium.

Three years ago, we thought of the website as providing a taster for the paper version. We published the table of contents, abstracts, and the occasional full text article. Since then we have added more full text sections and have begun to publish some material exclusively on the web. Soon the paper journal will be a pared down version of only the most important material that has appeared on the website.

The camera moved

Although this account may suggest we have a clear idea of where we're heading, we haven't. We are poised at the beginning of a profound shift in how information is disseminated but our "paper mindset" blinkers us to the possibilities of the new medium.[4 5] This is usually the case with a new medium, which is handled like old media until its unique properties are recognised and exploited. The first film directors' idea of cinema was to set up a stationary camera in front of a stage play, recording it from the point of view of a single theatregoer. And then around 1908, something occurred of crucial importance to the history of cinema: the camera moved. Eventually, that led to close ups, tracking shots, reaction shots, and the action shown from different points of view—all those features that differentiate a cinematic from a theatrical experience. The internet awaits its camera moving moment.

But before the electronic *BMJ* assumes much more importance in our plans we will have to find a business model to support it. Even without the substantial costs of paper, print, binding, and postage the journal still costs millions of pounds to produce. Our current strategy is to make the electronic journal as attractive as possible so that readers will eventually be prepared to pay for content. And if we can attract a large enough audience then we hope that advertisers will follow. Certainly, some American journals have attracted large sums in sponsorship from the pharmaceutical industry.

The 2% of the world's population who are online may be suffering from an information glut, but what of the 75% who have yet to hear a telephone dial tone, let alone get online? Subbiah Arunachalam, India's premier information scientist, says that the internet will widen the information gap between the developed and the developing world before it reduces it. Poor connections mean that even those with internet access "must spend hours downloading material that would take only minutes for those in the developed world with the best access" (p 1116).[6]

Paradoxically, the world wide web might do more to level the playing fields for the information poor than any number of out of date medical journals and books sent to the developing world. Medical libraries in these countries seem far more likely to acquire computers with internet access than they do to fill their shelves with a critical mass of current information. Several publishers—including the BMJ Publishing Group—are already thinking of how they might provide the developing world with access to their journals. A few lines of computer programming could allow free (or heavily discounted) access to computers from selected countries and, unlike discounts on paper subscriptions, the gesture would cost publishers nothing.

Those of us from whose tongues the words "world wide web" trip most easily have the greatest obligation to ensure that this wonderful new creation lives up to its name.

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