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Gulf war illness

New American research provides leads but no firm conclusions

It is six years since the end of the Persian Gulf war, and we are just beginning to see the publication of scientific research addressing the long term health of those who took part. The four papers on this topic in last week's *JAMA*[1-4] are therefore of considerable interest. Service men and women deployed in the Gulf were exposed to several potentially serious physical and psychological stressors. These include immunisations, pyridostigmine prophylaxis, pollution from oil fires, and the liberal use of pesticides, a list that continues to grow. The campaign took place in inhospitable surroundings and was conducted under the threat of exposure to some of the most fearsome weapons yet invented. Some adverse effects on health are therefore unsurprising.

The first of the *JAMA* papers is a survey of all veterans, both deployed and non-deployed, from the state of Iowa.[1] It is the first population based survey of its kind and achieved an impressive response rate (76%). It provides strong evidence of a health problem associated with service in the Gulf,[5] since the military staff who were deployed were twice as likely to report symptoms as those not deployed (14.7% *v* 6.6% reporting two or more problems). The biggest differences in reporting rates were for cognitive dysfunction, fibromyalgia, depression, anxiety, alcohol misuse, respiratory problems, and chronic fatigue. There was no increase in risk of more specific illnesses such as cancers. These findings are similar to those reported in veterans of previous conflicts.[6] The researchers also asked about the whole range of possible exposures. Each exposure was associated with each outcome, suggesting either that each has the capacity to cause a wide range of problems or that the data were susceptible to recall bias.

The other three papers, from Robert Haley and colleagues, funded by the Perot foundation in Texas, tackled the same problem but came to different conclusions.[2-4] The authors have a commendably clear hypothesis, that the cause of the veterans' ill health was exposure to anticholinesterase inhibitors. Their sample consisted of a previously studied single Naval Reserve Construction Battalion ("Seabees"). Only 41% participated, but, of these, 70% reported serious health problems and all but one attributed them to service in the Gulf. The subjects completed a detailed questionnaire about their symptoms and service experiences. The researchers attempted to organise the different symptoms using factor analysis.[2] The first analysis suggested two syndromes, which the authors described as resembling chronic fatigue syndrome and post-traumatic stress disorder. Subsequent analyses suggested six syndromes, but a reliability test on split halves of the sample showed that only the first three syndromes were stable.

Haley *et al* went on to relate each of the syndromes to self reports of different hazardous exposures.[3] The most robust syndrome—labelled "impaired cognition," which included distractibility, memory problems, and fatigue—affected 5% of the veterans. It occurred in five of the 19 subjects (26%) who used flea collars to prevent insect bites but only seven of the 229 (3%) who did not. The second syndrome,"confusion ataxia," also included symptoms such as problems with thinking and reasoning and a diagnosis of post-traumatic stress disorder. Surprisingly, this was not associated with exposure to pesticides but was associated with recall of particularly severe side effects from pyridostigmine prophylaxis. Those currently experiencing joint and muscle pains and pins and needles ("arthromyoneuropathy") were more likely to report use of insect repellents containing N,N-diethyl-m-toluamide.

The researchers concluded that all of these syndromes are evidence of organophosphate induced delayed polyneuropathy. They claimed to rule out any contribution from psychological and stress related conditions on the basis of responses to a personality inventory. They also rejected recall bias since the reported associations were specific to cholinesterase inhibitors—unlike a larger study in similar units.[7] Dr Haley told a press conference that "the syndromes are due to subtle brain, spinal cord and nerve damage, but not stress," contradicting the conclusions of the American presidential advisory commission, which reported on the same day.[8]

Extrapolating directly from reports of physical symptoms to specific aetiologies is perilous—somatic symptoms are common and usually remain medically unexplained.[9] Haley and his team did not assess whether similar symptom clusters are present in samples of non-Gulf war veterans; one suspects at least some might be.[10] The same symptoms could be related to exposure to low levels of organophosphates[11] and high levels of stress.[12] The mistake is to assume that the two are mutually exclusive. Recent animal experiments have suggested that the two may be synergistic; agents that are without risk in normal situations may be more hazardous either in combination[13] or in battlefield conditions.[14]

The symptoms reported by the 21 veterans in Haley *et al*'s second group ("confusion-ataxia") were also attributed, by 18 of the subjects, to exposure to chemical weapons. The issue of whether there actually was large scale exposure to such weapons is controversial. If it turns out that there was no such exposure then the symptoms could be put down to the natural terror that such a perceived threat would induce.

In the final paper, Haley's team looked for specific evidence of neurological damage.[4] They took the 23 most severely ill veterans and compared them with unaffected battalion members, a departure from standard case-control methodology. They used a variety of different approaches, resulting in 165 different comparisons. No diagnostic abnormalities were found on neurological examination or investigation. However, abnormalities and asymmetries were reported in sensory evoked potentials, eye movements, and neuropsychological test batteries. There were no significant abnormalities detected using functional or structural neuroimaging, and visual evoked potentials were normal. The authors concluded that the tests provided evidence of generalised injury to the nervous system. Possible confounders, such as excess alcohol use,[1] were not addressed.

As the accompanying editorial makes clear,[15] these papers are an important start but some of the conclusions may be a bridge too far. They are hypothesis generating rather than hypothesis testing. It is premature to assume that all the findings necessarily result from a specific exposure unique to service in the Gulf. What we need are comparisons of sick Gulf veterans with sick, rather than normal, controls. Sick veterans of other conflicts would also provide an informative comparison group. We should also take care before generalising the results to British veterans. Reserve troops may be more vulnerable to varioius outcomes because of differences in training, expectations, age, fitness, and combat exposure, and, according to the Iowa study, they were more likely to report exposure to a variety of agents including pesticides.[1] Compared with the United States, Britain made far less use of reservists.

No single health problem has yet emerged as a focus for aetiological studies. Despite the reassuring findings on mortality from all 700 000 American veterans[16]—the only increase found was in deaths by accidents—it is premature to exclude at this stage an association with diseases with long incubations such as cancer. An increase in more ill defined conditions such as chronic fatigue syndrome now seems likely but not certain. The issue of reproductive health still needs to be addressed, since some veterans and their spouses have given birth to children affected by various congenital abnormalities. Again, this is not prima facie evidence for a link with Gulf service.

The scientific case for large scale epidemiological studies remains overwhelming. Even when there is a consensus about the existence of any particular adverse outcome, linking it with particular exposures years after the event will be far from easy, as Haley *et al*'s work shows. For this, contemporary records detailing individual exposures are desirable but may be difficult to obtain. Some will cry "cover up," although the military authorities would argue that their task was to fight a war not collect data for subsequent epidemiological studies.

In Britain, the Ministry of Defence, the Medical Research Council, and the United States Department of Defence have commissioned three epidemiological studies of possible adverse effects of Gulf war service. The London School of Hygiene and Tropical Medicine will be studying reproductive outcomes, while we at King's College, London, and Professor Nicola Cherry and colleagues at the University of Manchester will be looking at the pattern of illness in the veterans. In the meantime it is essential that attention continues to be paid to individual veterans. While the Ministry of Defence has been criticised for its slowness to commission systematic research, it deserves credit for its response to the individuals affected. The universally praised medical assessment programme, begun by Group Captain William Coker, recognises the need for every person with health problems that may be related to service in the Gulf to receive a high quality comprehensive medical assessment.

After six years many fundamental questions remain unanswered. But there is now widespread recognition of the need for a swift, rigorous, and effective post-conflict illness surveillance system.[8 17] Let's hope we never have to use it.

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Outbreaks of *E coli*

Prevention may rest on time hallowed public health principles, rather than research

See p 249

Verotoxin producing *Escherichia coli* causes severe gastrointestinal disease, often with frank blood in the stool (haemorrhagic colitis). In about 2-7% of cases infection is complicated by haemolytic uraemic syndrome,[1] which has a mortality of 3-5%. The current outbreak of infection with *E coli* 0157 in Scotland,[2] described as "the second largest recorded outbreak anywhere," is the subject of an interim report by Professor Hugh Pennington's expert group, produced with commendable speed.

Perhaps inevitably, the group has been able to discharge some parts of its remit better than others. With various legal proceedings pending, the first part of its remit—to examine the specific "circumstances which led to the outbreak"—receives only a brief reference. There are no descriptive or other data. Regarding the second part of its remit—"the implications for food safety and the general lessons to be learned"—the group identifies four key areas: research, surveillance, enforcement, and procedures for handling outbreaks.

Surveillance is crucial, and the report makes two recommendations. The first is to strengthen requirements to report infectious diseases: in the short term by incorporating compulsion in the NHS purchaser-provider contractual arrangements, and in the long term by reform of the law, which most people recognise as overdue.[3] However, the best incentive for clinicians and laboratories to report infections is if the data they provide is fed back to them in a timely fashion as information that they can use to prevent and treat disease.

The report's second recommendation is to improve the efficiency of the surveillance process. Suggested improvements include testing all faeces from people with diarrhoea for *E coli* 0157 and electronically linking the surveillance network. Both are practicable. Welsh laboratories have examined all specimens for *E coli* 0157 since 1990,[4] and a recommendation went out in England in 1995.[5] Electronic surveillance systems introduced via the Public Health Laboratory Service have been in use throughout Wales since 1990[6] and are being introduced in England.

What sort of action might be taken on enforcement? The report recommends, firstly, structural alterations to food premises to ensure better physical separation of raw and cooked meats; secondly, a selective licensing system for food premises; and, thirdly, better targeted and more frequent inspections. Contamination of cooked meats from raw meat has been a well recognised cause of food poisoning outbreaks for a depressingly long time. Unfortunately, the absence of any data on the outbreak leaves us unable to judge whether the proposals in the report, reasonable enough on microbiological and food hygiene grounds, would have prevented it. Also, although it was not the group's remit to produce a handbook of verotoxin producing *E coli* infection, anyone whose sole knowledge of the organism comes from the report might be unaware of other important aspects of prevention.

Other foods besides meats have caused outbreaks and other premises besides butchers' shops have been the setting, notably catering premises. Outbreaks have been ascribed to handling soiled potatoes,[7] apple juice,[8] yoghurt,[9] and unpasteurised milk.[10] Direct transmission from animals occurs.[11] Person to person spread occurs, particularly in preschool children, and outbreaks have occurred in nurseries.[12] Guidance to prevent such human transmission exists, and public health authorities should act promptly when informed of cases.[13 14]

The prevention of outbreaks of *E coli* 0157 infections may rest principally on the judicious application of time honoured principles of public health rather than on research, to which the report gives prominence. Scotland has one of the highest reported rates of infection with *E coli* 0157 in the developed world,[10] a higher proportion of Scottish cases are associated with outbreaks as opposed to being sporadic, and certain health boards, notably Grampian, are disproportionately affected. The reason for these differences, identified over a decade of careful surveillance by the Scottish Centre for Infection and Environmental Health, are not obvious. Examining these differences, as well as detailed data from the outbreak, is likely to bring into focus the areas for preventive activity. This report marks a sensible starting point.

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Pig transplants postponed

Until we know more about graft rejection, physiology, and infectivity

Britain's long awaited report on the ethics of xenotransplantation has now appeared.[1] Following on from last year's report from the Nuffield bioethics committee,[2] its main conclusion is that clinical trials of xenotransplantation are not appropriate at the present time as there is insufficient knowledge of the immune response to a xenograft, the physiological behaviour of a xenograft, and the risk of infection across species. Both reports conclude that it is ethical to use pigs as a source of donor tissues, provided that the pigs are well cared for, but not to use primates other than for carefully controlled research. However, in recommending that clinical trials should not yet be carried out the advisory group has taken a stronger line.

Is this recommendation reasonable given that hyperbole in the lay press might have led us to believe that pig hearts would be transplanted into humans any day now? The answer is definitely, yes, until our knowledge is more complete.

What then are the unresolved problems?[3] The first is graft rejection, the most dramatic form of which is hyperacute rejection. This is mediated by complement in the presence of natural cytotoxic antibodies in humans directed against a pig xenoantigen, gal a(1,3) galactose.[4 5] This major hurdle has been largely overcome with the development of pigs expressing a human transgene for complement regulatory factors. Other methods for preventing this destruction of the xenograft include removing the antibody by immunoabsorption with gal antigen, blocking complement with soluble complement receptor 1 (CR1), producing "knockout" pigs which do not express the gal antigen (so far achieved only in the mouse), or expressing the H antigen of the human blood group O instead of the gal antigen.[6-12]

However, while hyperacute rejection can be prevented by one or a combination of these approaches,[13] there still remain significant immunological problems to be overcome. For example, the activation of the endothelium by antibody leads to a procoagulant state in the transplanted organ, with potential for thrombosis. This is followed by an almost certainly strong cellular immune response, which results in acute rejection.[14]

Also unknown are the physiological problems of xenotransplantation. For example, will the pig kidney produce erythropoietin that will function in humans, and will the denervated pig heart provide a satisfactory working heart in upright humans? However, most concern has been expressed about the possibility of transmitting infection (especially viruses) from pigs to humans. The new report states that the risks of infection with bacteria, fungi, parasites, and prions are ethically acceptable provided that donor animals are bred in a specific pathogen free environment, but it rightly stresses that prion related diseases remain latent for long periods of time. Certainly we need to know much more about porcine viruses, particularly retroviruses which may be non-pathogenic in the pig but are not necessarily so in humans; Weiss has recently shown that a pig retrovirus can infect human cells in vitro.[15] Such concerns justify the call for further research before clinical trials are considered.

See p 247

The report's recommendations may disappoint workers in the field, as well as patients convinced by exaggerated reports in the press (no doubt often commercially driven) that pig transplants would start tomorrow. The report correctly points out that premature clinical use of xenotransplantation could harm the allotransplant programmes on which we must depend for many years yet, if not forever.

Two other recommendations are to be applauded: the establishment of a national regulatory authority to oversee clinical xenotransplantation, and more support for research in all aspects of xenotransplantation. Research into transplantation biology, both xeno and allo, has not been high on research councils' lists of priorities—a deficiency that needs correcting if clinical xenotransplantation is to become a reality.

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Cervical carotid or vertebral artery dissection

An underdiagnosed cause of stroke in the young

See p 291

The major causes of stroke in young adults and children differ from those in older people.[1] Dissection of the internal carotid and vertebral arteries in the neck accounts for about a fifth of strokes in the young compared with about 2.5% in older patients.[1]

Dissection of intracranial blood vessels is rare and has a worse prognosis than extracranial dissections.[2] Arterial dissection occurs when blood tracks into the vessel wall along a specific line of cleavage. This may be subintimal, causing luminal narrowing or occlusion, or subadventitial, when a pseudoaneurysm may form. The cause is rarely established and may differ according to the artery affected. The incidence of arterial dissection is increased in patients with fibromuscular dysplasia, migraine, or hypertension; in smokers; and in those taking oral contraceptives. It is commonly associated with trauma or manipulation to the neck.

Patients with dissection of the arteries in the neck may present with any combination of craniocervical pain (in 50-80% of cases), cerebral ischaemia, cranial nerve palsy, Horner's syndrome, and pulsatile tinnitus. Pain often precedes neurological features, usually by hours or days but occasionally by weeks.[3] The pain has been described as stabbing, pulsating, aching, "thunderclap," sharp, or "unusual." It is usually localised to the neck, head, eye, or face, with some tendency for the pain of carotid artery dissection to localise anteriorly and that of the vertebral artery to localise posteriorly.

Ischaemic neurological features occur in 30-80% of all dissections and may present as transient attacks (often stereotyped) or completed stroke, which develops in as many as 20% of cases.[2] Strokes are most likely to occur within 24 hours of the onset of symptoms, with almost all occurring within 10 days.[4] Early suspicion and establishment of the diagnosis is therefore crucial for preventing stroke.

The differential diagnosis of craniocervical pain and neurological deficit in a young patient includes migraine. The potential difficulties in diagnosing dissection in patients with migraine is demonstrated in the grand round in this week's *BMJ* (p 291).[5] Neurological deficit without pain produces a wider differential diagnosis,[1 2] but dissection should be considered in any patient presenting with craniocervical pain with or without neurological features.

Establishing the diagnosis of arterial dissection relies on imaging. The preferred method has been angiography, with dissection having characteristic appearances such as a "string sign," pseudoaneurysm, double lumen, or an intimal flap. However, Doppler ultrasound can be highly sensitive in diagnosis—particularly for the carotid arteries,[6] where accuracy approaches 95%—and is valuable in monitoring evolution. For dissection of the vertebral arteries, standard Doppler appearances are less specific, but this may improve with duplex colour flow imaging.[7] Combined magnetic resonance angiography and imaging of the neck allows both the luminal narrowing and intramural clot to be seen, and, where available, this may become the imaging method of choice.[6]

The aim of treatment is to prevent neurological deficit. The mechanism of ischaemia after dissection probably results from distal microemboli.[8] Medical treatment aimed at reducing propagation of clot and distal embolisation has included use of antiplatelet drugs and anticoagulation. However, there have been no prospective randomised controlled trials of any form of treatment. Early anticoagulation with heparin seems to reduce the chance of a complete stroke developing.[2] We recommend anticoagulation with intravenous heparin as soon as possible after the start of symptoms and diagnosis. Anticoagulation may be worth starting even as long as one month after presentation.[3] Provided there are no contraindications on computerised tomography of the brain, anticoagulation should probably also be started even after neurological signs have appeared, as there is a tendency for strokes after dissection to follow a "stuttering" pattern. Anticoagulation is usually continued with warfarin until the vessel has recanalised, as determined by repeated Doppler ultrasound studies. Recanalisation may occur at any time from two days after presentation, but in our experience it may still not have occurred as long as 18 months later.

The prognosis for other features of cervical arterial dissection is good. Most cranial nerve palsies recover completely and cranial pain usually resolves within three to four days, although in some patients it may become chronic.[3]

Dissection of the major arteries in the neck is an important and probably underdiagnosed cause of stroke and craniocervical head pain. Familiarity with the clinical features and rapid performance of Doppler ultrasound scanning allow a rapid diagnosis to be made in nearly all cases. Early treatment with anticoagulation may significantly improve the outcome. However, no formal trials of anticoagulation in this important condition have been performed.

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Antiphospholipid (Hughes') syndrome

A treatable cause of recurrent pregnancy loss

See p 253

The antiphospholipid syndrome, first described in 1983,[1] is now recognised as an important prothrombotic disorder associated with a specific group of antibodies. Its main clinical feature is thrombosis, both venous and arterial (especially recurrent cerebral ischaemic attacks). Other features include mild thrombocytopenia, chorea, heart valve disease, livedo reticularis, and, most commonly, recurrent pregnancy loss.[2] The importance of the syndrome in general medicine, especially in vascular and neurological disease, is now acknowledged.

The syndrome has had various names. Hughes originally studied it in patients with systemic lupus erythematosus but recognised that most patients "had atypical lupus, or no lupus at all"—hence the concept of "primary" antiphospholipid syndrome.[3] In the early 1990s it was found that a phospholipid binding protein, β_2 glycoprotein I, was required for the binding of antibodies to phospholipids. More recently, other such proteins have been shown to be involved.[4] The complex and possibly indirect links between antiphospholipid antibodies and thrombosis led to the suggestion that the name of the syndrome be changed to "Hughes' syndrome."[5]

One of the major features of the syndrome in women is pregnancy loss, most typically in the second trimester. Some women suffer six or more miscarriages before the diagnosis is made. Even worse, in others the diagnosis remains "infertility." Even in patients with systemic lupus erythematosus it is now recognised that most of the excess fetal loss occurs in association with antiphospholipid antibodies.[6]

Recurrent pregnancy loss, defined as three or more spontaneous consecutive miscarriages, affects 1-2% of women. There are many known causes. What proportion of this major problem does antiphospholipid syndrome represent? The published studies suggest a figure between 7% and 25%. Even if the lower percentage is true (and our own bias is towards the lower end of the range) the annual incidence of pregnancy loss among women in Britain alone because of this potentially treatable condition must be huge.

The causes of pregnancy failure may be numerous, but the consensus is that thrombosis leading to placental insufficiency is the central mechanism. Logically, therefore, antithrombotic treatment is preferred to corticosteroids (once widely recommended). The current choice is aspirin or heparin, or both.

The use of low dose aspirin has never been subjected to randomised clinical trial, although there are several non-randomised studies suggesting that 75 mg daily is an effective treatment.[7 8] Furthermore, low dose aspirin has been reported to reduce pregnancy loss in experimental antiphospholipid syndrome in mice.[9] In a recent study pregnancy outcome improved from 19% to 70% after treatment with low dose aspirin in all patients and subcutaneous heparin in those with previous thrombosis.[10]

Heparin does not cross the placenta and is not known to cause any adverse fetal effects. However, long term use of heparin in pregnancy has been associated with osteoporosis in the mother. Many centres are now using low molecular weight heparin, since it has increased bioavailability and a longer half life and can therefore conveniently be given once daily.[11] It seems to have no greater deleterious effect on bone density than occurs physiologically during pregnancy.[12] Two prospective, randomised studies have shown that heparin plus low dose aspirin provides a significantly better outcome than low dose aspirin alone.[13 14] One, published in this week's *BMJ* (p 253), included a high proportion of patients who were positive for lupus anticoagulant[13]; the other, from Dallas, Texas, excluded such individuals.[14] The clinical conclusions, however, are similar.

The improved outlook for successful pregnancy in patients with antiphospholipid or Hughes' syndrome is a medical achievement worth celebrating. In the immediate future better antithrombotic regimens are likely. Most importantly, there will be increased awareness of the syndrome (and its potential for treatment) by physicians, obstetricians, and general practitioners.

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Time to look again at sight tests

Those at greatest risk of glaucoma are not the most likely to attend for sight tests

There is no formal screening programme for glaucoma in Britain, and current practice can best be described as opportunistic surveillance, with no attempt to define clearly, or cover, the entire population at risk. To facilitate this surveillance, certain sections of the population are exempt from sight test charges, including first degree relatives of people with glaucoma, diabetic patients, and people receiving income support. However, probably about half of cases of glaucoma remain undiagnosed,[1] and patients continue to present late in the course of the disease.

Detecting presymptomatic chronic glaucoma requires two independent events. Firstly, an individual must attend for a sight test, and, secondly, the practitioner must use appropriate tests: intraocular pressure measurement, optic disc assessment, and visual field testing—preferably all three.[2] We know roughly how often optometrists perform tests for glaucoma from work done by the International Glaucoma Association,[2] but until recently we lacked information on who consults optometrists and how often—particularly since charging for sight tests began in 1989. The controversy over whether charging has affected rates of glaucoma detection remains unresolved.[3]

Most patients referred to ophthalmologists for glaucoma testing come from optometrists (via general practitioners), so we can use attendance for tests as an indicator of the frequency of glaucoma testing. According to Crick and Tuck, optometrists check intraocular pressure in only half of their customers and test the visual field in only a tenth.[2] Thus sight tests are probably more than twice as common as glaucoma testing.

The general household survey for 1990-4 provides invaluable information.[4] The survey covers the population in private households in Britain each financial year. The sample size is between 23 000 and 24 000 people a year, and the past four years have included sections on use of spectacles, contact lenses, and attendance for sight testing. Although it has been suggested that people tend to underestimate the time since their previous sight test,[5] the crude percentage of sight testing in the population has increased over the survey period, from 27% in 1990-1 to 32% in 1993-4 (not age standardised). The percentage of women has been consistently higher than men. In the most recent survey (1993-4) a third of women (34%, 95% confidence interval 33% to 35%) had sight tests in the previous 12 months compared with 28% (28% to 29%) of men.

The incidence of glaucoma increases with age (from 0.08/1000 per year in white people in their early 40s, rising to 1.46/1000 per year in the over 80s). However, the data from the general household survey do not show the same trend for the likelihood of sight testing. This increased from 28% of those aged 25-34 having sight tests to 40% of those aged 45-54, but thereafter decreased so that 37% of those over 65 reported having had a sight test in the previous year. The peak in the fifth decade is likely to be due to presbyopia—the need for reading glasses, which usually starts in the fifth decade but stabilises by the seventh. Those aged over 65 are less likely to need more powerful reading correction and so have less incentive to seek sight testing. The survey's findings therefore indicate that the population subgroup at greatest risk of glaucoma are not the most likely to attend for sight tests.

The survey also reveals differences between socioeconomic groups. The highest percentages of sight testing are found in professionals, with 39% of professional men and 40% of professional women attending compared with 22% of unskilled men and 29% of unskilled women. Stratification shows that this difference is not accounted for by age.

The fact that those aged over 65 are at higher risk of glaucoma but are not the most likely to seek sight testing must be a matter of concern for a government which has proposed to offer free sight tests to those at increased risk of blinding diseases. The single largest relative risk of developing glaucoma is increasing age—exceeding the risk of a positive family history.[6] The next largest factor is race, which also exceeds family history: in people of African origin, glaucoma is four or more times commoner than in white Europeans and is the commonest cause of blindness.[7]

So what can be done? We are not advocating a nationwide glaucoma screening programme as this entails questions of implementation, cost, and diagnostic accuracy that have not yet been satisfactorily answered. However, the present system is clearly failing to detect large amounts of preventable blindness, and other options need to be explored—including free eye tests for elderly people, an educational campaign to increase public awareness of the need for regular glaucoma assessment, and locally based initiatives in areas with high proportions of Afro-Caribbean people. Finally, any strategy that increases glaucoma case detection in the community has to be backed up by adequate resources. Only then will an already stretched hospital eye service be able to cope with the resulting workload.

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London's mental health services in crisis

Needs more than just more money

See p 262

In 1992 the King's Fund organised the first London commission on the future of London's health services. The commission's recommendations, taken up in the Tomlinson report,[1] led to radical and controversial changes in London's health services. In Spring 1997, a second London commission will issue its recommendations on the future of mental health services. *London's Mental Health*, published this week by the King's Fund, summarises the evidence on which the Commission will base its deliberations.[2]

London's Mental Health is a comprehensive overview of the capital's mental health services prepared by its foremost experts. This giant document may be reduced to four propositions: first, that London has uniquely high requirements for psychiatric services; second, that psychiatric services in inner London are "near collapse"; third, that the reason for this collapse is underfunding from central government, arising from a failure to take into account London's uniquely high requirements; and, fourth, that the solution is to increase substantially spending on London's psychiatric services across the board. Each proposition is worth considering in more detail.

The report establishes beyond doubt that London has a uniquely high requirement for psychiatric services. Inner London is at the extreme end of the spectrum on practically all sociodemographic variables known to be associated with high rates of severe mental disorder. These variables include unemployment (16.5% of adults in London, 9.2% in Britain), social deprivation (London includes the six most deprived districts in England), social isolation, homelessness, and concentrations of ethnic minorities with high incidences of psychoses.

The report also convincingly establishes that inner London's inpatient services are at breaking point (although, as Shepherd *et al* show in this week's *BMJ* (p 262), this is not solely a London problem[3]). Admission rates in inner London have increased substantially over the past five years and are higher than anywhere else in the country, including other deprived inner city areas. Bed occupancy rates have occasionally reached 125%, and, perhaps not surprisingly, levels of violence among inpatients are the highest in the county. Vast sums of money are being wasted funding extracontractual referrals and providing "hotel" facilities for patients for whom no suitable alternative accommodation is available. The report is less successful in establishing how far community services are under pressure. Waiting times for community visits are unacceptably long, but otherwise, since the advent of the internal market, there is "a lack of interpretable and accurate information."[2]

The report is also less successful at proving that London's psychiatric services are underfunded. To prove that London is losing out, the report applies a sophisticated but questionable methodology comparing actual levels of service with levels predicted by a computer program (MINI). Despite the inherent difficulties in this type of analysis, the report's authors wholeheartedly embrace the conclusion that underfunding is the sole cause of London's difficulties. This attitude provides a convenient excuse for not examining how far the actions of London's purchasers and providers are responsible. Is no one struck by the paradox that the demand for acute in patient care is at its height at a time when London's community services have never been more extensive or "innovative"? Could there possibly be any connection between London's "full implementation" of the care programme approach and rising admission rates? Could there be a link between London having pioneered court diversion schemes and the level of violence in its acute wards or its massive appetite for medium secure beds? Is there a relation between the explosion of "back protecting" bureaucratic practices after 11 public inquiries and the appallingly slow response times to real emergencies? The report chooses not to address these obvious but uncomfortable questions.

Non-Londoners reading this report will conclude that what London needs is a hard headed citywide strategic plan to reduce the pressure on inpatient beds and thus release funds for other service developments. This plan would have three main elements: first, investment to develop services that are known to reduce admissions (such as assertive community treatment, acute day hospitals, and home based care) and increase discharges (such as supported accommodation); second, an urgent review of London's community services aimed at ensuring that they practise in ways proved to reduce admission rates; and, third, a moratorium on developing services that are not likely to affect admission rates until spending on extracontractual referrals is under control.

Unfortunately, what *London's Mental Health* proposes is simply a massive injection of someone else's money into everything from liaison psychiatry to the training of general practitioners. It remains to be seen how far the second London commission will accept this robust defence of the status quo.

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