Trained in pathophysiology, diagnosis, and treatment, doctors find themselves spending more time thinking about issues like management, improvement, finance, law, ethics, and communication. Luke Filde's 19th century painting of a contemplative doctor alone with a sick child might now be replaced by a harassed doctor trying to park his car to get to a meeting on time. The gratification that comes from curing a sick child is different from that which comes from being part of the meeting that agrees to take an abused child into care. Christian Koeck—a doctor, professor of health policy, and member of the *BMJ* editorial board—thinks the problem goes deeper. He thinks the intellectual model of medicine is wrong and that instead of being trained simply to apply the natural sciences to peoples' health problems doctors should also be trained as change managers. That way they can help people adjust to the sickness, pain, and death that are central to being human.

Another way to think about doctors' unhappiness is to think of the change in the contract between doctors and patients. We hear much about doctors changing from being authorities to being partners with patients, and some find this transition unsettling. But perhaps the change is deeper still. Maybe we are changing from what has become a bogus contract between doctors and patients to something more real (see box). Doctors are often acutely aware of the limitations of what they can do, whereas patients—partly through the exaggeration of doctors—have inflated ideas of the power of medicine. Negative media coverage might represent the world's waking up to the limitations of doctors and medicine, and—though it's uncomfortable now—it may lead to a much more honest, adult, and comfortable, relationship.

Richard Smith *editor, BMJ*

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3 Wise J. Milburn to shift power to health staff. *BMJ* 2001;322;1083.

Is transmitted drug resistance in HIV on the rise?

It seems so

Papers p 1087

The transmission of drug resistant variants of HIV-1 has the potential seriously to limit the therapeutic options of newly infected patients. The selection of HIV drug resistant variants among individuals who are already receiving treatment also clearly limits both the size and duration of the viral supression induced by drug treatment.[1 2] Reports from North America and Europe indicate that up to 14% of recently infected patients have been infected with a strain of virus bearing well characterised drug resistance mutations (in 1-10% of cases) or reduced susceptibility to a particular drug (2-14% of cases).[3–5] Temporal trends in the transmission of drug resistance for these populations are not yet available, but a paper from the United Kingdom in this week's *BMJ* suggests an increase in the risk of being infected with drug resistant HIV virus between 1994 and 2000 (p 1087).[6]

Estimates of the likelihood of transmission vary depending on the type of exposure and the magnitude of viral load in the HIV infected partner.[7] An incomplete understanding of the biological factors that influence viral transmission further limits the accuracy of projected estimates of transmitted drug resistance. In order to interpret the relative prevalence rates of drug resistance among recently infected subjects we must consider the route of exposure (mucosal or blood borne), possible geographical variations, detection assay type (genotype *v* phenotype), susceptibility threshold (for phenotypic assays) or type of mutations considered (for genotypic assays), and perhaps HIV subtype (non-B *v* B *v* recombinant subtypes). Available assays generally identify only the resistance profile of the predominant viral variant in the infected subject. In the absence of drug selection pressure, reversion to a more replication competent, perhaps drug susceptible, variant may occur, which may in turn preclude the detection of drug resistant variants. Prevalence estimates of transmitted drug resistance in newly infected patients should not therefore be generalised to patients with established infection who have not yet started treatment with antiretroviral drugs, who may harbour drug resistant variants within archived latent reservoirs of virus that may re-emerge in the presence of drug selection pressure.

In the study this week from the UK Collaborative Group on Monitoring the Transmission of HIV Drug Resistance, 69 subjects who developed HIV infection during 1994–2000 were evaluated for resistance within 18 months of their infection; none had received treatment with antiretroviral drugs at the time of resistance testing.[6] Genotypic resistance was detected in 14% of the subjects, 3% with mutations conferring drug resistance to all three of the available classes of antiretroviral drugs. These estimates are consistent with previous reports of transmitted drug resistance in recently infected subjects.[3–5] These investigators also identified an increase in the prevalence of transmitted drug resistance during the period of study, with drug resistant variants detected in 27% of subjects identified in 2000. Significant increases in the prevalence of transmitted drug resistance have been reported from North America during this same period.[8]

The clinical importance of transmitted drug resistance, particularly using different thresholds of susceptibility, has not been established. However, among patients already established on treatment there is generally good correlation between genotypic and phenotypic markers of resistance and virological responses to treatment.[9]

Methods to improve drug adherence and targeted HIV prevention messages may ultimately reduce the risk of transmitted drug resistance. However, the study this week from the UK group clearly identifies the urgency that needs to be associated with these steps.

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Drug resistance testing in all recently infected individuals is needed to monitor changes in the prevalence of transmitted drug resistance among different risk groups and to optimise initial treatment choices.

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Dysfunctional breathing and asthma

It is important to tell the difference

General practice p 1098

General practitioners and emergency departments from time to time see patients with asthma who appear very breathless, with fast deep breathing and wheeziness, who complain of tingling lips and hands and who recover quite rapidly after breathing in and out of a paper bag and then using a few puffs of salbutamol. Asthma and anxiety with dysfunctional breathing are both common conditions and they often coexist. Indeed, a paper in this week's issue suggests a very high prevalence of dysfunctional breathing among patients with asthma.[1] There are reasons to doubt the prevalence suggested by this paper, but the overlap between anxiety and asthma nevertheless creates a problem for patients and their doctors since we seem not to be very good at telling the difference.

Several studies have shown that patients with asthma have significantly higher anxiety scores than normal and are more likely to have clinically diagnosed panic disorder.[2 3] Conversely, patients with panic disorders, hyperventilation, or "overbreathing" may have unidentified airways reversibility.[4] Demeter investigated 47 patients referred for hyperventilation syndrome using methacholine challenge and reversibility testing and judged 38 of them to have asthma.[5] The hyperventilation symptoms were eliminated in 29 with a combination of explanation and bronchodilators.

Thomas et al sent the Nijmegen questionnaire, an instrument designed to identify the so called hyperventilation syndrome, to all adults in one general practice with a diagnosis of asthma and at least one prescription for an asthma drug in the previous year (p 1098).[1] Of those who responded 29% had scores indicative of dysfunctional breathing. The authors suggest that "a large minority of patients may be experiencing avoidable morbidity because of inappropriate diagnoses and ineffective treatment." Their study reminds us of a very real and important problem but overestimates its size: we do not believe that nearly a third of patients in general practice with a diagnosis of asthma have been wrongly diagnosed.

Firstly, the 16 item Nijmegen questionnaire was not validated in an asthmatic population. Its 91% sensitivity and 95% specificity for physician diagnosed hyperventilation syndrome were shown in a study comparing a group of physician diagnosed non-asthmatic hyperventilators with a group of non-asthmatic normal controls.[6] Several of the questions relate to symptoms such as shortness of breath, pain and constriction in the chest, and feeling tense—symptoms common to asthma and dysfunctional breathing. This necessarily impairs the ability of the questionnaire to make the latter distinction.

Secondly, the very existence of a discrete "hyperventilation syndrome" has been questioned by research looking for, and failing to find, reliable correlations between panic, overbreathing, and hypocapnia. One reviewer of this work suggests that hyperventilation syndrome is a chimera.[7] Clinicians may respond that, though unable to define a chimera, they know one when they see one. Nevertheless, our understanding of the interaction between physical symptoms, physiological disturbances, and cognitive perceptions in anxiety—and in asthma—remains limited.

What should we do about the overlap between the symptoms of asthma and of anxiety? Firstly, because straightforward misdiagnosis is possible we must perform careful and repeated history taking, examination, and physiological measurements—particularly peak flow diaries. We should not assume that an earlier diagnosis was correct, especially when computerised records carry terse definitive-looking diagnoses from earlier years without providing the information on which the diagnosis was based.

Secondly, we must routinely assess the extent and effect of the anxieties of our asthmatic patients. We can then seek to allay them and avoid stepping up asthma treatments inappropriately when anxiety, hyperventilation, or laryngeal dysfunction are the problem, not worsening asthma: Hyland showed that a higher rate of corticosteroid prescribing was significantly associated with higher levels of panic or fear independent of lung

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