Islam with the internet could do much to prevent disease

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Intracranial hypertension and nasal fluticasone propionate

Editor—Bond et al said that nasal fluticasone propionate caused benign intracranial hypertension in a 13 year old boy with a history of Crohn’s disease and subsequently reported this to the Committee on Safety of Medicines.1 We have numerous problems with this hypothesis. Firstly, Bond et al did not confirm the diagnosis of intracranial hypertension as the cerebrospinal fluid pressure, which should be markedly raised, was not measured on any occasion.2 Their conclusions can thus at best be based only on papilloedema, headache, and backache.

The boy was seen by specialists from the ear, nose, and throat department, but no mention was made of the presence or absence of the otological manifestations, which include objective pulsatile tinnitus and low frequency hearing loss, which can be the major or only manifestation of this syndrome.3 Other recognised associated conditions were not excluded, such as hypervitaminosis A, systemic lupus erythematosus, hypothyroidism and its correction, and malnutrition and renutrition, which is not unheard of in patients with Crohn’s disease; the patient was in remission, so both factors could have been at work.4

We have to assume that this teenage patient was not receiving any other drugs such as tetracyclines or isotretinoin (commonly used in the treatment of acne in adolescents), which also have been implicated in the development of benign intracranial hypertension. The condition tends to be self limiting, with a course of less than 12 months in most cases and recurrence in 10%.5 We were therefore concerned that the temporal relation that Bond et al describe may just be the normal course for the condition. It is paradoxical that steroids are implicated as an aetiological factor and are also an accepted treatment. Would Bond et al suggest that nasal steroids could in different circumstances be a useful treatment?

1 Bond JW, Charlton CPJ. Benign intracranial hypertension secondary to nasal fluticasone propionate. BMJ 2001;322:867. (14 Apr)

Ultrasonography may have role in assessing spontaneous miscarriage

Editor—Ankum et al’s regular review on the management of spontaneous miscarriage suggests that medical management is of little benefit in the treatment of incomplete miscarriage.6 The wide variation in reported success rates might, however, be responsible for differences in the use of ultrasonography.

The use of ultrasonography to determine whether there are retained products of conception will exclude about 30% of women from treatment as they will be found to have an empty uterus.7 If ultrasonography is not used at this stage the success rate will be inflated by the inclusion of women having unnecessary treatment. Conversely, the use of ultrasonography to assess completeness after uterine evacuation may reduce the apparent success rates: the finding of intrauterine tissue usually leads to an assumption that the treatment has failed. The clinical course of this finding, however, is unknown, and most of these women will probably complete the spontaneous miscarriage without further intervention.

Analysis of the available studies shows a clear relation between the reported success rates and the time at which the ultrasonography was carried out after treatment (figure). The analysis suggests that the low reported success rates in some studies occurred as a result of an over-reaction to ultrasonographic findings that are of unproved relevance. Initial medical treatment followed by expectant management may be the key to the effective management of incomplete miscarriages. These findings also add weight to the arguments in favour of using expectant management alone. These two regimes need to be compared. The figure may represent the natural course of incomplete miscarriages, but this

In these days of evidence based medicine there are several tests of causation that a hypothesis or proposal should be subjected to, and Bond et al have not done this. All clinicians should be observant of adverse drug reactions, with particular reference to topical nasal steroids and children.

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1 Bond DW, Charlton CPJ. Benign intracranial hypertension secondary to nasal fluticasone propionate. BMJ 2001;322:867. (14 Apr)

Comparison of reported success rates with time at which ultrasonography was carried out after treatment. Numbers refer to references

Needs to be confirmed in a prospective ultrasonographic study of women having expectant management.

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Editor—Modern public health has paid little attention to prevention tailored towards Muslims even though the Islamic population now numbers over 1 billion. Religion is a major component of the social life of many communities throughout the world, but in disease prevention we often use the concept of “one size fits all,” with little recognition of religion or culture. Religion’s positive influences can be incorporated into a strategy for health promotion and disease prevention by using recent developments in information technology.1

Removal of thioridazine from primary care formulary will result in prescribing vacuum

Environ—Recently the Committee on the Safety of Medicines recommended that general practitioners should not prescribe thioridazine. It recommended that prescribing should be limited to second line treatment in schizophrenia and that prescription should be initiated only by a consultant psychiatrist. The reason was that the drug was thought to be a cause of very rare cardiac deaths by widening the QT interval in an electrocardiogram, leading to a greater risk of developing arrhythmias. The evidence base as to substantiate this decision is not, however, sufficient. Thioridazine is the most commonly prescribed phenothiazine in primary care. Its implication in very rare deaths would not be substantiated if number of deaths were directly standardised to volume of phenothiazine prescribed. In 1997 the Royal College of Psychiatrists produced a council report on the association between phenothiazines and sudden death. The report showed that sudden death was associated with all the phenothiazines prescribed in primary care. The phenothiazine associated with most deaths was not thioridazine but chlorpromazine. The report documented that abnormalities in an electrocardiogram are comparatively common in people receiving neuroleptics, occurring in around 25%. It also stated that such changes are commonly considered benign; even now consensus on the clinical significance of prolonged QT, is lacking.

Removing thioridazine from the primary care formulary will lead to a prescribing vacuum, and it is unfortunate that more thought has not been given to which drugs will fill it. As thioridazine has become an adequate substitute for the prescribing of benzodiazepines in conditions of anxiety and irritability, its removal from the primary care formulary will increase the probability of benzodiazepines being overprescribed in primary care. Perhaps the Committee on the Safety of Medicines should have stood by its original advice in 1996 when it suggested that, whenever possible, drugs associated with QT interval prolongation should be avoided in patients who have underlying cardiac disease.

Perhaps the committee singled out thioridazine as a result of the work by Reilly et al, who measured a prolonged QT interval in 495 patients. The cohort had been prescribed a wide variety of psychotropic drugs, and no single drug was immune from being associated with a widening of the QT interval. The paper concluded that QT interval abnormalities are surrogate markers, and the link between psychotropic drugs, arrhythmia, and sudden death should be examined. It is unfortunate that this recommendation was not explored before the committee on the Safety of Medicines advised the removal of thioridazine from the primary care formulary.

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“Coronary heart disease” is not tautologous

Environ—Gilroy repeats a common misconception in suggesting that the term “coronary heart disease” is a new term and that it is a tautology. It is an established North American term, to be preferred to the British synonym “ischaemic heart disease,” which dominates recent editions of the International Classification of Diseases. Ischaemic is imprecise and could, as Friedberg pointed out 35 years ago, apply equally well to heart disease secondary to stenosis of the aortic valve, or even to anaemia. “Coronary” does not mean cardiac but “resembling, or encircling, like a crown.” It was the 17th century anatomists’ descriptive name for the arterial pattern around the heart.

Coronary artery disease is used, confusingly, to mean pathological findings confined within the coronary arteries. These changes are present in almost all adults and are usually symptomless. It is only when changes to the coronary arteries are severe enough to affect the myocardium, to cause coronary heart disease, that patients are truly diseased—from angina, myocardial infarction, sudden death, or heart failure. Coronary heart disease means that the heart as an organ is involved, necessitating secondary prevention, whereas we all have coronary artery disease for decades beforehand and try to slow its progression to heart disease by primary prevention. Heart disease and heart attacks are too non-specific as terms to get by without qualification. Twenty five years ago I tried to introduce “coronary heart attack” as a generic term to cover both cardiac infarction and coronary deaths (often unassociated with infarction), but without success.

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1 Gilroy F. Tautology, or not tautology. BMJ 2001;322:1606, 1608 [June].