1·6 (95% confidence interval 1·2 to 2·2). Did the authors collect information on whether non-participants had or had not been exposed to pet birds? On the other hand, the proportion of controls who had kept pet birds in Gardiner and colleagues' study is high, suggesting that this control group may have been overexposed compared with the population of origin of the cases.

Because of the small number of non-smokers among the cases in Kohlmeier and colleagues' study it is not certain that the absence of a significant interaction between bird keeping and smoking in the logistic regression model allows us to rule out a potential multiplicative interaction between these two factors with respect to lung cancer. Some insight into whether bird keeping is an independent risk factor for lung cancer could be obtained if the authors reported the numbers of cases and controls not exposed to either smoking or pet birds, exposed to pet birds but not to smoking, exposed to smoking but not to pet birds, and exposed to both smoking and pet birds.

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1 Kohlmeier L, Arminger G, Bartolomeycik S, Bellach B, Rehm J, Thamm M. Pet birds as an independent risk factor for lung cancer: case-control study. *BMJ* 1992;**305**:986-9. (24 October.)
2 Gardiner AJS, Forey BA, Lee PN. Avian exposure and bronchogenic carcinoma. *BMJ* 1992;**305**:989-92. (24 October.)

EDITOR,—The first two sentences of John Britton and Sarah Lewis's editorial[1] illustrate a common misconception. They suggest that because cigarette smoking accounts for about 80% of deaths from lung cancer the contribution of other causes must be small. The fallacy arises because different causes of disease act in concert and not in competition. Avoidance of any one may be sufficient to prevent illness developing.

The fact that 80% of lung cancers could be eliminated by abolition of smoking does not preclude the possibility that 100% might be prevented by some other public health measure. In the same way, the fact that all cases of a disease are attributable to a genetic defect cannot be taken to imply that the environment makes no important contribution to its aetiology. Phenylketonuria is genetically determined, but it is also "completely" attributable to consumption of foods containing phenylalanine.

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1 Britton J, Lewis S. Pet birds and lung cancer. *BMJ* 1992;**305**: 970-1. (24 October.)

EDITOR,—Two recent papers indicate that contact with pet birds may increase the risk of developing lung cancer.[1,2] In these papers (and in the pioneer study by Holst *et al*[3]) adjustment was made for smoking habits and other variables.[4]

None of the papers adjusted for atopic disease. Atopic allergy is common and is a confounding factor related to both the exposure and the condition under study. People with asthma, hay fever, and related allergies are less likely to keep pets, including pet birds, as they are well aware of the potential hazards. This applies also to all members of their families. People with asthma tend to refrain from smoking as this will aggravate their symptoms. In their households smoking is usually not permitted as passive smoking may also provoke asthmatic symptoms in susceptible people.

Thus lung cancer is not positively related to keeping pets. Rather, the absence of pets protects against lung cancer indirectly, smoking and a familial predisposition to atopy being the confounding variables. Further research on this issue should properly control for asthma and other atopic conditions. This applies to both patients with lung cancer and controls as well as their households.

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1 Gardiner AJS, Forey BA, Lee PN. Avian exposure and bronchogenic carcinoma. *BMJ* 1992;**305**:989-92. (24 October.)
2 Kohlmeier L, Arminger G, Bartolomeycik S, Bellach B, Rehm J, Thamm M. Pet birds as an independent risk factor for lung cancer: case-control study. *BMJ* 1992;**305**:986-9. (24 October.)
3 Holst PA, Kromhout D, Brand R. Pet birds as an independent risk factor for lung cancer. *BMJ* 1988;**297**:1319-21.
4 Britton J, Lewis S. Pet birds and lung cancer. *BMJ* 1992;**305**: 970-1. (24 October.)

## Misuse of anabolic drugs

EDITOR,—H M Perry and B N C Littlepage warn of the increasing use of anabolic steroids among non-competitive athletes.[1] Other authors have attempted to draw doctors' attention to this alarming trend,[2,3] and the government is at last funding research into patterns of use of anabolic steroids in Britain (Department of Health, personal communication).

Surveys of the prevalence of use of anabolic steroids such as those quoted by Perry and Littlepage have been scarce and have chiefly been carried out in the United States. There is growing evidence, however, that the problem is at least as widespread in Britain. I recently conducted a survey (unpublished) of 687 students attending a Scottish college of technology. The response rate was 92%. Eighteen students admitted to current or previous use of anabolic steroids (15/341 (4·4%) male students and 3/292 (1·0%) female students. Fourteen of these had first used these drugs at age 17 or less, and 10 at age 15 or less. Six combined oral and intramuscular routes of administration. Fifteen admitted to having obtained the drugs from an illegal source. The most common reason given for use of these potentially harmful drugs was enhancement of appearance, not athletic performance. Participation in a wide range of sports, many of which are not traditionally associated with use of anabolic steroids (such as football, hockey, and middle distance running), was reported, suggesting that patients who are not "well muscled" should not escape suspicion.

Surveys like this must be repeated on a larger scale and in a range of populations to establish the true extent of the problem. Anabolic steroids are readily available, effective, and relatively inexpensive.[2] If, as this survey suggests, people commonly start using them at school age and nearly one in 20 male college students uses them then they may represent a public health problem that ranks after only alcohol, tobacco, and cannabis use.

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1 Perry HM, Littlepage BNC. Misusing anabolic steroids. *BMJ* 1992;**305**:1241-2. (21 November.)
2 Williamson DJ. Anabolic steroids: Edinburgh's lesser known drug problem. *Edinburgh Medicine* 1991;**65**:6-7.
3 Williamson DJ. Anabolic steroid use outside competition. *Br J Psychiatry* 1991;**159**:161.

EDITOR,—H M Perry and B N C Littlepage highlight many complications due to misuse of anabolic steroids but do not mention thrombosis.[1] We report on a 26 year bodybuilder who suffered pulmonary emboli while taking the anabolic agent methandienone.

The man initially presented with pleuritic chest pain and haemoptysis. A ventilation-perfusion scan showed a mismatched wedge shaped defect, and he was given anticoagulant drugs. At that time he had no apparent risk factors for thrombosis. He was discharged taking 10 mg of warfarin as an outpatient, but the dose proved difficult to titrate, being subject to large day to day variation. Three months later he was readmitted with further pleuritic chest pain. The ventilation-perfusion scan was unchanged, but he confessed to having taken a minimum of 25 mg of methandienone a week over the preceding six months to supplement his weightlifting. The drug's potentiation of warfarin was thought to have accounted for the difficulties in anticoagulation.

There are several case reports associating misuse of anabolic steroids with thrombosis.[2,3] The clinical circumstances of our case and the others reported suggest a causal relation. Potential mediators of the hypercoagulable state include enhanced platelet aggregation, alteration in coagulation or fibrinolytic proteins, and increased vascular reactivity.[4]

Acute thrombotic events in well muscled people should alert doctors to potential misuse of anabolic steroids as the risks are underappreciated.

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1 Perry HM, Littlepage BNC. Misusing anabolic drugs. *BMJ* 1992;**305**:1241-2. (21 November.)
2 Montine TJ. Massive pulmonary embolus and anabolic steroid abuse. *JAMA* 1992;**267**:2328-9.
3 McNutt RA, Ferenchick GS, Kirlih PC, Hamlin NJ. Acute myocardial infarction in a 22-year old world class weight lifter using anabolic steroids. *Am J Cardiol* 1988;**62**:164.
4 Ferenchick GS. Are androgenic steroids thrombogenic? *N Engl J Med* 1991;**322**:476.

EDITOR,—I am surprised that H M Perry and B N C Littlepage do not mention testicular changes in men who use anabolic drugs.[1] High doses of androgens or anabolic steroids suppress the pituitary-testicular axis, commonly giving rise to clinically apparent testicular atrophy and azoospermia or oligospermia.[2] Serum gonadotrophin concentrations are usually undetectable or low normal. These changes are reversible if the man stops using anabolic steroids.

Perry and Littlepage could have enhanced their clinical message by stating that well muscled men with testicular atrophy should be assumed to be taking anabolic steroids until proved otherwise. The same applies to well muscled women with amenorrhoea.

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1 Perry HM, Littlepage BNC. Misusing anabolic drugs. *BMJ* 1992;**305**:1241-2. (21 November.)
2 Knuth UA, Maniera H, Nieschlag E. Anabolic steroids and semen parameters in bodybuilders. *Fertil Steril* 1989;**52**: 1041-7.

EDITOR,—Having recently admitted two patients in their early 20s with problems related to use of anabolic steroids we believe that H M Perry and B N C Littlepage's editorial should have highlighted the need to consider this diagnosis in patients of normal build as well as in well muscled patients.[1] Both our patients were male amateur weightlifters who used anabolic steroids to improve their muscle bulk. Both presented with atypical chest pain, and rhabdomyolysis secondary to excessive physical activity and use of anabolic steroids was subsequently diagnosed.[2] The first patient was of an extremely muscular build and the diagnosis was obvious from an early stage, but the second man was of normal build and had only just started using these substances.