metal's angina are both given as contraindications in the datasheet for sumatriptan; the datasheet also includes advice against the concomitant use of ergotamine. Willett and colleagues' report therefore clearly highlights the need for these warnings and emphasises that sumatriptan should be avoided if there is a history of ischaemic heart disease or undiagnosed chest pain.

W M CASTLE
V E SIMMONS

International Drug Surveillance,
Glaxo Group Research Limited,
Middlesex UB6 0HE

1 Willett F, Curzen N, Adams J, Armitage M. Coronary vasospasm induced by subcutaneous sumatriptan. *BMJ* 1992;**304**:1415. (30 May.)

AUTHORS' REPLY,—Though we agree that the case that we reported highlights the need for warnings against using sumatriptan in ischaemic heart disease and known cases of coronary vasospasm, we believe that we clearly implied that the injection of sumatriptan given in this case did cause coronary vasospasm, which is commonly thought to be the underlying mechanism of so called Prinzmetal's angina. We therefore believe that we expressed this diagnosis clearly.

MARY ARMITAGE
NICK CURZEN
FRANCES WILLETT

Royal Bournemouth Hospital,
Bournemouth BH7 7DW

EDITOR,—In their report of a case of coronary vasospasm after subcutaneous administration of sumatriptan F Willett and colleagues state that the Committee on Safety of Medicines has received only one similar report.[1] The Netherlands Centre for Monitoring of Adverse Reactions to Drugs has received reports of 12 similar cases, mostly after oral intake (table).

All patients experienced chest symptoms, almost invariably within one hour after administration of sumatriptan. Symptoms varied from substernal tightness to severe cramping angina-like pain radiating to the left arm and hand. In one patient a transient increase in blood pressure to 200/120 mm Hg was noted, which later fell to 160/90 mm Hg. Four reporting medical practitioners classified the symptoms as anginal, and two of them notified these as "classical" or "real" angina pectoris. In all patients symptoms resolved without further treatment.

Electrocardiograms were normal in cases 3, 5, and 8 but were obtained after the chest symptoms had resolved. An echocardiogram and results of an exercise test performed after the first episode of chest pain in case 8 were normal. Except for one patient, who was said to have had a similar reaction to ergotamine in the past, none of the patients had had similar episodes before using sumatriptan and none developed such symptoms after stopping it.

Early studies suggested that serotonin-1 ($5\text{-}HT_1$) receptors are largely confined to the cranial circulation. Serotonin induces contraction of isolated epicardial coronary arteries,[2] which seems to be unopposed.[3] As serotonin-2 receptors are more common than serotonin-1 receptors in coronary arteries the effect of sumatriptan on coronary vasculature seems to be relatively mild.[4] Even so, sumatriptan elicited a vasoconstrictive response 30% of that to serotonin.[3]

Although this may not be clinically relevant in most patients, Chester *et al* suggested that when atherosclerotic changes decrease the luminal cross sectional area of the artery, problems may arise as the response to serotonin-1 is maintained in the area distal to an atherosclerotic occlusion.[3] This may be important, as the enhanced vasoconstrictive response of atherosclerotic isolated epicardial coronary arteries to histamine[2] is also seen to serotonin.[5] A study by the manufacturer of sumatriptan in 10 patients with existing or suspected coronary artery disease showed an average constriction of coronary arteries of 13·9% 10 minutes after subcutaneous administration of 6 mg.[5] Although aortic and pulmonary artery pressures were raised, cardiac output did not change. Although no electrocardiographic abnormalities were noted, only one patient experienced chest tightness (Glaxo, unpublished data).

The case reported by Willett and colleagues shows that ST elevation may occur after subcutaneous sumatriptan,[1] and this Dutch series shows that oral intake may also be followed by anginal pain.

B H C STRICKER

Netherlands Centre for Monitoring of
Adverse Reactions to Drugs,
PO Box 5406,
2280 HK Rijswijk,
The Netherlands

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Harm minimisation for drug misusers

EDITOR,—John Strang and Michael Farrell suggest that maintenance programmes with oral methadone may reduce the harm that drug misusers do to themselves.[1] Colin Brewer and colleagues chastise the authors for being too timid and criticise the many British clinicians who are reluctant to prescribe long term maintenance treatment with generous dosages of methadone.[2] Unfortunately, in their contributions none of these workers consider the harm that long term maintenance policies can do to those other than the drug misusers accepted for treatment.

I work as a general practitioner in a part of the country where drug misusers are commonly treated with long term maintenance programmes. Methadone is most commonly prescribed, but dihydrocodeine, diazepam, and temazepam are also often used. A greater emphasis is placed on achieving a stable lifestyle than on working towards a life without drugs of misuse.

One result of this policy has been a flood of drugs on to the black market as misusers sell them, either to gain money to buy the drugs they really want or to convert them into a regular weekly income. Drugs, originally prescribed legally, are now readily available in shopping centres, school playgrounds, and pubs; adults actively look for new children to supply so that the market is continually expanding. The money to pay for these drugs is nearly always raised by crime.

Whether or not long term maintenance policies can be justified, those who operate them have a strong obligation to see that as few drugs as possible leak into the rest of the community. Work showing that maintenance policies benefit the recipients is of little value if many more people are drawn into drug misuse as a result.

IAN McKEE

Edinburgh EH13 0RA

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Farmer's hip

EDITOR,—I was interested in Peter Croft and colleagues' finding that farmers are at increased risk of osteoarthritis of the hip[1] because, like others,[2-5] I have observed an increased incidence of hip replacement among my patients who are or have been farmworkers (table).

The controls were men of the same age (to the nearest five years) as the farmers who were on my practice list. Their occupation varied but was not necessarily sedentary: they were local government employees, teachers, shop assistants, police, gamekeepers, and builders. Those aged over 65 were retired businessmen and had previously lived outside Ryedale. Most had not engaged in manual jobs requiring the lifting of heavy loads.

The prevalence of hip replacement among men who had farmed for more than 10 years was 14 times and eight times greater than that in controls for those aged over 50 and over 60 respectively. These figures are similar to those found in moorland Staffordshire and lowland Cheshire.[1]

Croft and colleagues say that a question as yet unanswered is whether risks relate particularly to

Details of 12 cases of chest symptoms after administration of sumatriptan reported to Netherlands Centre for Monitoring of Adverse Reactions to Drugs

Case No	Age and sex	Dose and route	Latent period (min)*	Symptoms notified by reporting doctor	Recurrence of symptoms after rechallenge†	Other drugs
1	36, F	100 mg orally	30-45	Substernal pressure and discomfort, drowsiness, "shaky"	ND	Terfenadine 60 mg
2	38, F	100 mg orally	30	Substernal pressure and pressure in shoulders and neck	>10 times	Carbamazepine 600 mg, lactulose, hydroquinone hydrobromide dihydrate 100 mg
3	61, F	100 mg orally	About 30	Anginal pain radiating to left arm	2 times	Isosorbide dinitrate 5 mg
4	46, F	100 mg orally	15	Substernal pressure and chest pain, sweating	3 times	Oral contraceptive
5	53, M	100 mg orally	About 30	Anginal pain	ND	None
6	44, M	100 mg orally	30	Substernal chest pain, palpitations, pain in throat	ND	None
7	27, F	100 mg orally	30	Substernal chest pain, malaise, paraesthesia, heaviness of arms	ND	None
8	33, F	6 mg subcutaneously	1-5	Angina pectoris radiating to left arm and hand, dyspnoea	2 times	Propranalol 60 mg
9	45, F	100 mg orally	Same day	Substernal pressure, muscle stiffness	ND	None
10	19, F	6 mg subcutaneously	30-60	Chest pain, dyspnoea, nausea	ND	Oral contraceptive
11	50, F	6 mg subcutaneously	1-5	Anginal pain radiating to the jaw, hypertension	ND	Clonidine 100 μg, aspirin 600 mg
12	36, F	100 mg orally	30	Substernal chest tightness	ND	None

*Between first intake and onset of symptoms as notified by reporting doctor. †ND=Rechallenge not done.

Prevalence of hip replacement among farmers and controls by age

Age (years)	Men who had farmed for >10 years			Controls		
	No assessed	Cases of hip replacement	Prevalence (%)	No assessed	Cases of hip replacement	Prevalence (%)
50-55	20	2	10	17		
56-60	24	2	8	19		
61-65	23	4	17	15		
66-70	27	4	15	20	1	5
71-76	26	5	19	12		
>60	76	13	17	47	1	2
All	120	17*	14	83	1	1

*Five (29%) farmers required bilateral replacements.
Incidence of hip replacement among men over 60 in practice=8·4% and among women over 60=1·3%.

activities at an early age. Many of my farmworkers began their work at the age of 14, when their hip joints were not fully mature. Interestingly, Murray and Duncan observed: "Those areas of the world in which middle aged men are particularly afflicted by degenerative disease of the hip largely correspond to those in which competitive athletic activities emerged."[5]

ROBERT J CUNNINGHAM

Hovingham,
North Yorkshire YO6 4LQ

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EDITOR,—The Body Fluids Research Group welcomes the finding by Peter Croft and colleagues that farmers are at a greater risk of suffering from osteoarthritis than the normal population.[1] In Northern Ireland farming is a common occupation and, with the benefit of a centralised orthopaedic service, we have observed a definite clustering in farming communities for many years. One region, Rathfriland, where the incidence is unusually high, led our research group to consider mechanical events as an aetiological factor in this disease.[2]

We have proposed a new mechanism for producing damage to articular surfaces—namely, the phenomenon of cavitation.[3] This term describes potentially destructive bubble activity in fluid systems. Our hypothesis is that predictable mechanical events occur in normal joints which if accentuated will initiate the idiopathic development of osteoarthritis of the hip. We investigated the familiar cracking of joints by vibration arthrometry and postulated that these were due to cavitation.[4] Tension was applied to the synovial joint and was sufficient to break the synovial fluid, manifesting as cavitation. Cavitation seems to disrupt the surface of hyaline cartilage, particularly the collagen surface of chondrocyte lacunae.[5] We propose that cavitation in vivo can take two forms[5]: that which produces the familiar cracking sound (macrocavitation), and microbubble activity, which may occur because of the bubbles remaining in the synovial fluid after the crack (microcavitation).[6]

We related the high incidence of osteoarthritis of the hip in our farming communities either to high tension and shear forces produced while working in wet clay soils planting, harvesting, and stacking, particularly the potato crop, or to genetic factors producing a stickier than normal synovial fluid, both mechanisms inducing cavitation at a higher level than normal.

We believe that these factors must continue to be investigated because of the potential to prevent the disease process. We welcome the evidence that mechanical events in synovial joints may provide new insight into this common and costly disease.

R A B MOLLAN
K A ROSS
J M DUFFY

Body Fluids Research Group,
Queen's University of Belfast,
Department of Orthopaedic Surgery,
Musgrave Park Hospital,
Belfast BT9 7JB

1 Croft P, Coggon D, Cruddas M, Cooper C. Osteoarthritis of the hip: an occupational disease in farmers. *BMJ* 1992;**304**: 1269-72. (16 May.)
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Making air crashes more survivable

EDITOR,—Daphne Gloag and Ian R Hill give misleading views of progress on improving passengers' survival in aeroplane accidents.[1,2] The quoted survival statistics refer only to accidents in which some fatalities occurred; no account is therefore taken of improvements that may have led to an accident becoming fully survivable rather than partially so.

The overall fatal accident rate for jet aeroplanes in the United Kingdom has fallen from about 1·5 per million flying hours in the early 1960s to about 0·2 per million since the mid-'80s, and worldwide there has been a similar reduction. That improved cabin safety has played its part in this is shown by the fact that the rate of fatal accidents involving fire has halved in this period. The number of deaths in fires has improved even more dramatically from 11 per million hours to less than four per million.

In pursuit of continued improvements in survival in accidents the Civil Aviation Authority in the past seven years has taken 16 major regulatory initiatives. These include improvements to the resistance of cabin materials to fire, equipment to detect and fight fires, access to exits, and emergency procedures and training. Significant further improvements in survival will be hard to achieve and expensive, so Gloag is incorrect in suggesting that "much could be done now at little cost." Aeroplane landing and take off speeds are, after all, about twice those of fast motorway traffic.

In 1988 the Civil Aviation Authority adopted new requirements regarding the strength of seats, which constituted a major advance on the previous standards, but we accept that the accident at Kegworth highlighted the need for still further improvements to seats and their attachments as well as a general need for greater "impact friendliness" in the cabin.

Research is needed to support new international standards, and computer simulation of the accident at Kegworth has led to recommendations for a new brace position. We have commissioned additional research to confirm that this will not reduce overall safety levels, and that work will be completed in July.

The question of smoke hoods for passengers has tended to dominate discussions about cabin safety in recent years, with the proponents maintaining an effective and high profile lobby. The easiest response to this campaign would have been for the Civil Aviation Authority to make their provision mandatory. We are firmly convinced, however, that smoke hoods would cause more lives to be lost than would be saved. Accordingly, we do not require them and discourage airlines from offering them. Many fire safety specialists in other aviation authorities, fire services, research organisations, the airline industry, and various representative bodies support us in this.

Extensive full scale research in the United States and experience of accidents suggest strongly that there is not more than 30 seconds between when a survivor without a smoke hood would be incapacitated by the toxic gases and when one with a smoke hood would be overcome by the fire itself. However good the design of a smoke hood and the necessary briefing on its use given to passengers, it will still be unfamiliar; we are concerned that in a crash, with passengers experiencing shock and perhaps panicking, there could be delays in putting smoke hoods on. Furthermore, it would require only one or two people to get into difficulty with their smoke hoods to jeopardise the whole evacuation. We believe that evacuation is the first priority and that any risk of delay or hindrance should be minimised.

The Civil Aviation Authority has not, however, given up its search for improved fire protection—far from it. We are, for example, working with the United States Federal Aviation Administration on a cabin water mist system, which looks promising in its ability to delay the spread of fire, reduce temperatures, and wash out some toxic species. There are no easy options. More needs to be, and is being, done, and the survivability of aircraft accidents has already been much improved.

K EDGINGTON

Medical Division,
Civil Aviation Authority,
Gatwick Airport South,
West Sussex RH6 0YR

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Life as a junior hospital doctor's spouse

EDITOR,—I sympathise with Janet Cade's view of life as a junior hospital doctor's spouse.[1] My wife and I had been courting since my second year in medical school. We decided to wait until after the preregistration year to get married to make sure that our relationship could stand the strain. In our three and a half years of marriage we moved house six times. The bulk of this work usually fell to my wife despite her demanding nursing career.

For three years I have been continually studying for higher examinations; for 16 months of this I have been on 1 in 2 rotas. I am not in a hard pressed specialty, but the toll on our social life was great. Communication was limited over the evening meal before the night stretched away into revision, but we were happy, planning our futures and planning children. With our last move my wife could not find a suitable job for her experience and skills and so settled into being a housewife with all the stresses that job implies. Studying continued, and with it our communication dwindled. At