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Article *in* The Lancet · January 2010

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scans¹ and some patients do get transferred back to district hospitals with neurological deficit (albeit improving). However, if there is any doubt as to the possibility of recurrence, postoperative CT is done. After transfer, patients are treated by doctors in referring hospitals or in the community and receive a CT scan if there is a clinical concern. Except those with entirely normal appearances, all CT scans done in the district hospitals are sent for review by the neurosurgical unit. As a rule, when symptoms recur or deteriorate (after an initial improvement), or if a new neurological deficit occurs, and CT scan shows recurrence, a patient is transferred and drainage is done.

We agree with Rahimi-Movaghar and colleagues that assessment of patients by use of a questionnaire has limitations. However, the modified Rankin score has been validated and is widely used. We also agree that it may result in small haematomas being missed. But, it was not designed as a diagnostic tool and was not used in this study as such. Moreover, there is no reason to suspect that the modified Rankin score, as used in this study, would lead to a bias.

Contrary to Yi-hui Ma and Zhou Fei's statement, we did report the frequency of medical and surgical complications in the drain and no-drain groups (Results, paragraph 9). With regard to their query about association between clinical and laboratory variables and recurrence, in our cohort *only* the use of a drain was associated with recurrence and *not* the presence of coagulopathy, platelet dysfunction, scores on the Glasgow coma scale and modified Rankin scale, or neurological deficit.

Steroids were very rarely used in our cohort and an investigation of this variable would have not yielded any useful results. We are familiar with the study by Sun and colleagues² and

agree that with Ma and Fei that the question of the role of steroids in the management of chronic subdural haematoma is an important one and needs to be further investigated.

We declare that we have no conflicts of interest.

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- 2 Sun TF, Boet R, Poon WS. Non-surgical primary treatment of chronic subdural haematoma: preliminary results of using dexamethasone. *Br J Neurosurg* 2005; **19**: 327–33.

Adverse health effects of non-medical cannabis use

In their Review of the adverse health effects of non-medical cannabis use, Wayne Hall and Louisa Degenhardt (Oct 17, p 1383)¹ cite a study by Mittleman and colleagues² which suggests that smoking marijuana may be a rare trigger of myocardial infarction. However, in that study, only 37 of 124 marijuana-smoking patients reported smoking within 24 h of infarction and more than half the whole study group were cigarette smokers. Mittleman and colleagues do acknowledge limitations to their study.

The Review ignores contrary evidence of the beneficial effects of cannabinoids on the cardiovascular system. These include a protective role in atherosclerosis progression and in cerebral and myocardial ischaemia. Acute exposure to cannabinoids is associated with tachycardia and a small pressor effect, whereas longer-term use is associated with bradycardia and hypotension.³ Such cardiovascular tolerance can occur

within 2 days of frequent exposure but disappears quickly when cannabis use is stopped.⁴

Cannabis smoking, rather than $\Delta 9$ -tetrahydrocannabinol per se, may be the villain. At this juncture, it is fair to say that the jury remains out on cannabinoids and the heart.⁵

I declare that I have no conflicts of interest.

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- 1 Hall W, Degenhardt L. Adverse health effects of non-medical cannabis use. *Lancet* 2009; **374**: 1383–91.
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I found Wayne Hall and Louisa Degenhardt's Review of the adverse health effects of cannabis¹ both interesting and informative, but was dismayed by the term "non-medical cannabis use" in the title. This title insinuates that the adverse consequences discussed pertain only to non-medical users of cannabis, which, I am sure, must not have been Hall and Degenhardt's intention. It also conveys the impression that medical use of cannabis is a cogent and established entity, even though it is still controversial. Hall and Degenhardt do not mention whether the studies included in the Review distinguished between medical and non-medical cannabis users, and hence the use of the term does not seem to be justified.

Cannabis is the most commonly used illicit drug and those who use it for purely medical reasons make up only a very small proportion of the total number of users. Also, medical



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use of cannabis has been shown to be significantly associated with non-medical use of cannabis.² Hence, it can be safely assumed that medical users are also likely to experience the adverse consequences of cannabis use.

Cannabis is widely perceived by the public as being safe and is growing in popularity. Concurrently, there is a move towards relaxation of the criminal penalties associated with the recreational use of cannabis—ranging from the downgrading of criminal penalties in the UK to the possibility of full legalisation in Canada and Switzerland. In such a scenario, a discussion of the adverse consequences of the use of cannabis is welcome because it can help put the issue in perspective. But the needless use of the term “non-medical” in the title deprecates Hall and Degenhardt’s efforts to highlight the adverse effects of cannabis as a potential public-health menace.

I declare that I have no conflicts of interest.

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Authors’ reply

We agree with William Tormey that the cardiovascular risks of cannabis smoking have not been well characterised by the limited research done to date. We also agree that cannabis smoking is currently not a major contributor to cardiovascular disease, because it is much less common than cigarette smoking is in middle-aged adults. Nonetheless, we believe that the possible cardiovascular risks of cannabis smoking need to be better understood because: (1) cannabis smoke is very similar to tobacco smoke; (2) most recreational

users smoke the drug; (3) cannabis smoking is persisting into middle age among adults who started use in the 1970s and 1980s; and (4) older users probably use cannabis more episodically and so are less likely to develop tolerance to the stimulatory effects that tetrahydrocannabinol has on a cardiovascular system that may already be impaired by high blood pressure, atherosclerosis, and so on.

We disagree with Preeti Parakh that our use of “non-medical” in the title undermines our efforts “to highlight the adverse effects of cannabis”. We used “non-medical use” (rather than the even more contentious term “recreational”) to limit our task to reviewing the adverse effects of the patterns of cannabis use that cause greatest community concern and have the largest potential adverse public health effect—ie, the use of cannabis by adolescents and young adults seeking to experience its euphoric effects. Our title carries no implication that these adverse consequences are confined to non-medical users.

All studies in our Review were of non-medical cannabis users. Medical cannabis use is very rare by comparison with non-medical use, and is often only short-term (eg, to manage nausea in cancer chemotherapy). Its risks have not been as well studied as those of non-medical use.¹ Nonetheless, it is a reasonable hypothesis that medical cannabis users who smoke the drug on a regular basis over months or years face similar risks of adverse effects to those of non-medical users.

We declare that we have no conflicts of interest.

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- Degenhardt L, Hall WD. The adverse effects of cannabinoids: implications for use of medical marijuana. *CMAJ* 2008; **178**: 1685–86.

How much lift to the UPLIFT study?

In the UPLIFT study (Oct 3, p 1171),¹ Marc Decramer and colleagues compare the newer anticholinergic tiotropium to placebo in patients with chronic obstructive pulmonary disease (COPD). One could have predicted with a fair degree of certainty that the drug would be effective since earlier trials have consistently shown similar outcomes. Therefore, by comparing the new anticholinergic to placebo, Decramer and colleagues guaranteed a positive outcome. A clinically more useful answer might have been obtained if tiotropium had been compared with ipratropium—the only other established drug of its class.

Also, Decramer and colleagues consider only patients with Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage II COPD, and do not make any comparison with those who had stage III and stage IV disease for whom the outcome is more important.

To date, smoking cessation is the only persuasive intervention that retards the rate of decline of forced expiratory volume in 1 s (FEV₁) in patients with COPD.² A systematic review³ confirmed the effect of smoking cessation on COPD-related morbidity and mortality, and concluded that “in smokers aged 35 years with mild to moderate COPD, smoking cessation initially increases the FEV₁, and subsequently the rate of FEV₁ decline in sustained quitters reverts to the age-related decline seen in never-smokers in the background population”. So, contrary to Decramer and colleagues’ conclusion that “treatment of COPD should begin at an early stage of the disease”, we must find a better strategy for smoking cessation.

The mechanisms by which tiotropium is effective in reducing the rate of decline of postbronchodilator FEV₁ and in preventing exacerbation of COPD could provide a clue for future clinical research.