the calibration factors can be keyed into the monitors to take into account the sensor temperature.

As Davidson and Hosie state, to monitor ventilation satisfactorily it is necessary to measure both the adequacy of oxygenation and the adequacy of elimination of carbon dioxide. Transcutaneous monitoring allows this to be done easily and noninvasively in infants and children of all ages.

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 Davidson JAH, Hosie HE. Limitations of pulse oximetry: respiratory insufficiency—a failure of detection. BMJ 1993; 307:372-3. (7 August.)

### Migraine and risk of stroke

EDITOR,—Christophe Tzourio and colleagues found overall no association between migraine and ischaemic stroke.<sup>1</sup> Their subjects were 212 patients admitted with stroke to two Paris hospitals, and 212 matched controls. Their study setting therefore excludes those stroke patients well enough to remain at home. Within this group one would expect to find patients with infarction in the posterior cerebral artery territory. Visual field defects are the only neurological abnormality in most of these patients,<sup>2</sup> and hence they are usually managed as outpatients.

In their review of nearly 5000 migraine sufferers (aged under 50 years), Broderick et al described the clinical profile of 20 patients with associated infarctions with migraine.3 In most of these the infarcted area and angiographic abnormalities were in the distribution of the posterior cerebral artery. This is consistent with the prominence of visual symptoms in classic migraine. All stroke patients in Tzourio and colleagues' study had either computed tomography or magnetic resonance imaging. Correlation of the infarct location with a history of migraine might have yielded additional important information. In the absence of such information their work perhaps shows that migraine is not a risk factor in hemiplegic stroke, but the inclusion criteria would need to be considerably wider to support their conclusion that migraine and stroke are not associated.

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- Tzourio C, Iglesias S, Hubert J, Visy J, Alperovitch A, Tehindrazanarivelo A, et al Migraine and risk of ischaemic stroke: a case-control study. BMJ 1993;307:289-92. (31 July.)
   Pessin M, Lathi E, Cohen M, Kwan E, Hedges T, Caplan L.
- 2 Pessin M, Lathi E, Cohen M, Kwan E, Hedges T, Caplan L. Clinical features and mechanism of occipital infarction. Ann Neurol 1987;21:290-9.
- 3 Broderick J, Swanson J. Migraine related strokes. Clinical profile and prognosis in twenty patients. Arch Neurol 1987;44:868-71.

# Effect of aspirin and NSAIDs on colorectal adenomas

#### Protective effect may be spurious

EDITOR,—We believe that R F A Logan and colleagues have not dealt adequately with four factors that could have biased the results of their study of the effect of aspirin and non-steroidal anti-inflammatory drugs on colorectal adenomas.<sup>1</sup>

Firstly, patients with positive results of faecal occult blood tests could have been advised by their doctors to stop taking aspirin or non-steroidal anti-inflammatory drugs years previously. When interviewed subsequently they may have been more likely than the controls with negative results to say "no" to questions on drug use, thereby giving rise to a spurious association.

Secondly, the authors state that the protective effect of aspirin and non-steroidal drugs on the development of colorectal adenomas was unknown when the screening programme started to recruit subjects. But reports of the protective effects were published from 1981 onwards.<sup>24</sup> When the interviews began in 1985 the hypothesis under investigation was therefore known in the scientific community and presumably to the authors, given their stature in this work. The authors did not report whether their interviewers were blind to the hypothesis, for this could also introduce bias.<sup>5</sup>

Thirdly, the authors report a 10% false negative result of the faecal occult blood screening programme. Misclassification of cases and controls with negative results on testing could have introduced a further bias, and this would not necessarily result in a diminution of the reported association.

Fourthly, subjects with gastrointestinal disorders such as dyspepsia may be less likely to take aspirin or non-steroidal anti-inflammatory drugs. If they were also more likely to develop colorectal adenoma this might explain the reported association between failure to take aspirin and colorectal adenomas.

The evidence presented in this paper is unconvincing and does not warrant a change in public policy towards the primary prevention of colorectal adenomas and, by implication, carcinoma with aspirin and non-steroidal anti-inflammatory drugs.

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- Logan RFA, Little J, Hawtin PG, Hardcastle JD. Effect of aspirin and non-steroidal anti-inflammatory drugs on colorectal adenomas: case-control study of subjects participating in the Nottingham faceal occult blood screening programme. *BMY* 1993;307:285-9. (31 July.)
   Pollard M, Luckert PH. Treatment of chemically-induced
- intestinal cancers with indomethacin. Proc Soc Exp Biol Med 1981;167:161-4.
- Waddell WR, Loughry RW. Sulindac for polyposis of the colon. *J Surg Oncol* 1983;24:83-7.
   Metger U, Meier J, Uhlschmid G, Weihe H. Influence of various
- 4 Metger U, Meier J, Unischmid G, weine H. Innuence of various prostaglandin synthesis inhibitors on DMH-induced rat colon cancer. Dis Colon Rectum 1984;27:366-9.
- 5 Sitthi-amorn C, Poshyachinda V. Bias. Lancet 1993;343:286-8.

#### Author's reply

EDITOR,—I do not think that any of the four biases suggested by Su Vui Lo and colleagues are likely to have occurred.

Firstly, it is unlikely that subjects with a positive result of a faecal occult blood test would have been advised to avoid aspirin or other non-steroidal anti-inflammatory drugs because of previous occult or overt rectal bleeding. The tests were performed in a trial of screening for colorectal cancer, and few, if any, subjects would have had previous faecal occult blood tests. Furthermore, to avoid unnecessary investigation the trial attempted to exclude subjects with conditions likely to result in a positive result on faecal occult blood testing, such as ulcerative colitis.1 It is also doubtful whether doctors would advise people with overt rectal bleeding, such as blood on toilet paper, to avoid these drugs. Nevertheless, such bleeding was not reported more commonly by the patients with positive results of faecal occult blood tests.

Secondly, as we stated in our paper, we had no prior hypothesis with regard to these drugs and to this extent our interviewers were blinded. Until recently we were not aware of the experimental studies linking non-steroidal anti-inflammatory drugs and carcinogenesis.

Thirdly, non-differential misclassification of case-control status will lead to an underestimate of an association.<sup>2</sup> As we discuss in the paper, differential misclassification such as might arise

from a positive result of a faecal occult blood test being due to use of aspirin would produce results opposite to those that we reported—that is, less use of aspirin and non-steroidal anti-inflammatory drugs in the controls with negative results on faecal occult blood testing.

Fourthly, we know of no evidence linking unspecified dyspepsia with the presence of colorectal adenomas. Whether people taking aspirin or other non-steroidal anti-inflammatory drugs have more or less dyspepsia than others is also controversial; Jones and Tait compared the 12 month prevalence of dyspepsia among people who were and were not taking aspirin and non-steroidal antiinflammatory drugs and found that in each group it was 45%.<sup>3</sup>

Nobody would suggest a change in public policy on the basis of a single study, whatever that policy might be for preventing colorectal adenomas. Other studies, however, have been published since our paper was submitted. A reduced risk of colorectal cancer has been reported among patients with rheumatoid arthritis,<sup>4</sup> and Greenberg *et al* found a halving in the rate of detection of new adenomas in regular users of aspirin compared with non-users in subjects being recruited for a randomised trial of chemoprevention with micronutrients. Clearly more studies are required, not only to confirm those findings but to define the balance of risk and benefit.

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- Hardcastle JD, Chamberlain J, Sheffield J, Balfour TW, Armitage NC, Thomas WM, et al. Randomised, controlled trial of faecal occult blood screening for colorectal cancer. Results for first 107 349 subjects. Lancet 1989;i:1160-4.
- 2 Mertens T. Estimating the effects of misclassification. Lancet 1993;342:418-21.
- 3 Jones RH, Tait CL. Gastrointestinal side effects of NSAIDs: a community-based study. Gut 1993;24(suppl 1):S57.
  A Critly: C. McJ supplies IV. Ebborn A. Klassekra, J. Adami
- Gridley G, McLaughlin JK, Ekbom A, Klareskog L, Adami H-O, Hacker DG, et al. Incidence of cancer among patients with rheumatoid arthritis. *JNCI* 1993;85:307-11.
   Greenberg ER, Baron JA, Freeman DH JF, Mandel JS, Haile R.
- 5 Greenberg ER, Baron JA, Freeman DH Jr, Mandel JS, Haile R. Reduced risk of large-bowel adenomas among aspirin users. *JNCI* 1993;85:912-6.

## Nestlé's marketing policy

EDITOR,—In response to the personal attack by Geoffrey A Fookes of Nestlé<sup>1</sup> I wish to make the following points. Fookes does not deny any of the allegations made in Luisa Dillner's article<sup>2</sup> but tries to deflect criticism by claiming that Nestlé is unaware of its own marketing practices.

We at Baby Milk Action did not waste time pursuing our complaints with Nestlé after its own monitoring commission was disbanded, because we knew that Nestlé disagreed with both our and the commission's interpretation of the World Health Organisation's code on the marketing of baby milk formulas. We had even less confidence in the industry's ombudsman, who was appointed and funded by the very group he was supposed to investigate.

Fookes implies that companies have an interest in the establishment of monitoring procedures but fails to point out that the companies want to be judged only on national codes, not on the much stricter provisions of the international code. Where national code monitoring committees exist we regularly send reports. In countries like Britain, where the baby food industry finances and controls the monitoring committee, it is rare for that committee to find fault with the companies.

Finally, Fookes implies that all criticism of Nestlé's marketing policy comes from a handful of activist groups. Yet millions of people and thousands of church, consumer, health, and development bodies around the world, including