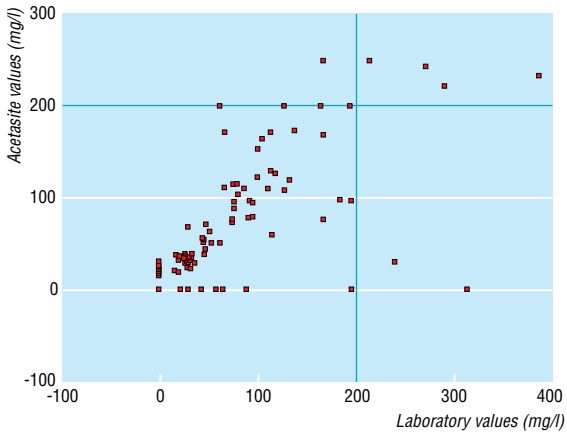
Department of Clinical Biochemistry, Addenbrooke's NHS Trust
I Hamdi, *senior registrar*
Correspondence to: Dr Robinson

paracetamol. Differences between the AcetaSite and laboratory results were spread randomly between high and low concentrations of paracetamol. Treatment with acetylcysteine would have been withheld from three out of the five patients whose results were outside the limits of agreement, had the decision to treat been based only on the result from the AcetaSite test. This might have resulted in a poor outcome.

Impressive performance characteristics are reported in the datasheet for the AcetaSite test and Stat-Site reflectance meter when compared with the GDS enzymatic liquid reagent ($r=0.970$) and the TDX (Abbott) liquid reagent ($r=0.983$). For the data sheet, accuracy was assessed using whole blood, plasma, and serum samples with known concentrations of paracetamol and a small number of clinical samples (n=42). The methodology may partially explain the discrepancy between the results found in our study and those found in preclinical testing; correlation does not assess the degree of agreement but rather the relationship between the two tests.[2] Also, the use of samples with known concentrations in a laboratory environment may not accurately replicate analysis of samples obtained in a clinical setting.



Scattergram of untransformed data comparing results obtained by AcetaSite test with values obtained in the laboratory (mg/l). Dotted lines show concentrations at which treatment with acetylcysteine would be indicated in groups not considered to be at high risk (4 hours after ingestion)

Operator error may explain why some of the results from the AcetaSite test bear little relation to the results found with the laboratory tests. Although the majority of department staff attended two training sessions, difficulties in using the Stat-Site meter were reported by some inexperienced operators. The production of a simple algorithm for using the card and meter reduced the number of difficulties reported.

The rapidity with which the AcetaSite results were available could have been advantageous if the results had been in agreement with the laboratory results. This study found that the AcetaSite test should not replace the standard Quantase assay.

We wish to thank Dr C Palmer for his statistical advice and Miss C Reid for her help in data acquisition.

Funding: The equipment and technical support required for this study were provided by Cambridge Life Sciences.

Conflict of interest: None.

1 Prescott LF, Illingworth RN, Critchley JAAH, Stewart MJ, Adam RD, Proudfoot AT. Intravenous N-acetylcysteine: the treatment of choice for paracetamol poisoning. *BMJ* 1979;2:1097-100.
2 Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1986;i:307-10.

(Accepted 11 March 1997)

Comparison of case fatality in smokers and non-smokers after acute cardiac event

Gabe S Sonke, Alistair W Stewart, Robert Beaglehole, Rod Jackson, Harvey D White

Department of Community Health, Faculty of Medicine and Health Science, University of Auckland, Private Bag 92019, Auckland, New Zealand
Gabe S Sonke, *postgraduate student*
Alistair W Stewart, *biostatistician*
Robert Beaglehole, *professor*
Rod Jackson, *associate professor*

continued over

BMJ 1997;315:992–3

Although smoking is a major modifiable risk factor for acute myocardial infarction, it has also been associated with an up to twofold lower risk of dying in hospital after an acute myocardial infarction.[1 2] We analysed data from a community based register of coronary heart disease to determine whether differences in case fatality (the proportion of those dying) between smokers and non-smokers are restricted to patients who have been admitted to hospital and to evaluate possible explanations for this smoker's paradox.

Subjects, methods, and results

All deaths related to coronary causes and all admitted patients aged 25-64 who met predefined criteria for myocardial infarction or coronary death were identified in Auckland, New Zealand, between 1986 and 1992 as part of the World Health Organisation MONICA (monitoring trends and determinants in cardiovascular disease) project. Study criteria, and methods of case finding and data collection procedures have been published.[3 4] Postmortem examinations were performed on 63% of those who died from cardiac causes. Deaths before admission to hospital, deaths within 28 days after admission, and the total number of deaths were measured. Smoking was determined by direct questioning of surviving patients and of relatives of those who died. Patients were classed as current smokers (those who smoked at least one cigarette a week at the onset of symptoms or gave up smoking less than one month before the index event), ex-smokers (those who had abstained from smoking for at least one month before the onset of symptoms), or non-smokers (those who had never smoked). Logistic regression models were used to assess the effects of smoking on case fatality after adjusting for age, sex, history of myocardial infarction, and history of angina. For those admitted to hospital, adjustments were based

Demographic information, case fatality, crude odds ratio, and adjusted odds ratio by smoking status for acute cardiac events in patients aged 25-64 years, 1986-92

	Non-smokers (n=1088)	Current smokers (n=2166)	Ex-smokers (n=1477)
Mean (SD) age (years)	55.8 (7.2)	53.3 (8.2)	56.7 (6.6)
No (%) men	801/1088 (73.6)	1689/2166 (78.0)	1229/1477 (83.2)
No (%) with previous myocardial infarction	257/1084 (23.7)	436/2153 (20.2)	552/1475 (37.4)
No (%) with previous angina	243/1086 (22.4)	342/2158 (15.8)	301/1476 (20.4)
Case fatality (%)			
Before admission to hospital:	409/1088 (37.6)	831/2166 (38.4)	503/1477 (34.0)
Crude odds ratio* (95% CI) (n=4731)	1.00	1.03 (0.89 to 1.20)	0.86 (0.73 to 1.01)
Adjusted odds ratio* (95% CI)	1.00	1.09 (0.93 to 1.27)	0.79 (0.67 to 0.94)
After admission to hospital:	123/679 (18.1)	157/1335 (11.8)	197/974 (20.2)
Crude odds ratio* (95% CI) (n=2988)	1.00	0.60 (0.47 to 0.78)	1.13 (0.88 to 1.45)
Adjusted odds ratio* (95% CI)	1.00	0.72 (0.55 to 0.95)	0.93 (0.71 to 1.22)
Total:	532/1088 (48.9)	988/2166 (45.6)	700/1477 (47.4)
Crude odds ratio* (95% CI) (n=4731)	1.00	0.88 (0.76 to 1.01)	0.94 (0.81 to 1.10)
Adjusted odds ratio* (95% CI)	1.00	0.97 (0.84 to 1.13)	0.85 (0.72 to 1.00)

*Odds ratio is the estimated odds of dying relative to a non-smoker.

on whether they received thrombolytic treatment. An adjustment for the year of infarction was included to account for time trends in event rates.

Between January 1986 and December 1992, 5106 patients with a definite myocardial infarction or who died from coronary causes were identified. Of these, 2166 were current smokers, 1477 were ex-smokers, and 1088 were non-smokers; information on smoking was missing for 375 patients, 231 of whom died before admission to hospital. Smokers were younger, more likely to be men, and fewer of them had a history of coronary heart disease when compared with non-smokers (table). The ex-smokers were older, more likely to be men, and more of them had previously had a myocardial infarction when compared with non-smokers.

Compared with non-smokers, smokers had a higher risk of dying before hospital admission but this was not significant. The risk of dying after hospital admission was significantly lower in smokers. Overall, there was no significant effect of smoking on total case fatality because smokers who die before admission have a bigger effect on total case fatality than smokers who survive to be admitted. Ex-smokers had lower risks of dying both before and after hospital admission, resulting in an overall reduction in case fatality when compared with non-smokers.

Comment

We found a lower case fatality within 28 days after an acute cardiac event for smokers who had been admitted to hospital when compared with non-smokers; there was a non-significant rise in case fatality before admission to hospital in smokers. The lower case fatality after hospital admission among smokers is balanced by an excess in the number of smokers who died before hospital admission. There was no overall effect of smoking on case fatality from an acute cardiac event.

Adjusting for confounding reduced the apparent beneficial effect of smoking shown in the crude analysis of deaths after admission and increased the magnitude of the detrimental effect of smoking in the analysis of deaths before admission. The apparent decrease in case fatality in smokers after an acute cardiac event is restricted to patients who have been admitted, and the smoker's paradox is largely explained by a greater case fatality before admission to hospital in smokers.

Funding: Health Research Council of New Zealand and the National Heart Foundation of New Zealand.

Conflict of interest: None.

1 Barbash GI, Reiner J, White HD, Wilcox RG, Armstrong PW, Sadowski Z, et al. Evaluation of paradoxic beneficial effects of smoking in patients receiving thrombolytic therapy for acute myocardial infarction: mechanism of the "smoker's paradox" from the GUSTO-I trial, with angiographic insights. *J Am College Cardiol* 1995;26:1222-9.

2 White HD. Lifting the smoke-screen: the enigma of better outcome in smokers after myocardial infarction. *Am J Cardiol* 1995;75:278-9.

3 Tunstall-Pedoe H, Kuulasmaa K, Amouyel P, Arveiler D, Rajakangas AM, Pajak A for the WHO MONICA Project. Myocardial infarction and coronary deaths in the World Health Organisation MONICA project. Registration procedures, event rates, and case-fatality rates in 38 populations from 21 countries in four continents. *Circulation* 1994;90:583-612.

4 Sonke GS, Beaglehole R, Stewart AW, Jackson R, Stewart FM. Sex differences in case fatality before and after admission to hospital after acute cardiac events: analysis of community based coronary heart disease register. *BMJ* 1996;313:853-5.

(Accepted 25 April 1997)

Department of Cardiology, Green Lane Hospital, Auckland

Harvey D White, *cardiologist*

Correspondence to: Professor Beaglehole r.beaglehole @auckland.ac.nz

A memorable patient

Learning how to do it

He was on holiday with his family in the west of Ireland and was breathless. They asked me to see him—it was the 1940s and I had only just got my MD.

They asked me if he was likely to die; I said not, but the next epidemic of flu might carry him off. He had emphysema. But his medical history was interesting. He had been a navy aviator before there was a Fleet Air Arm; he told me that he got his "ticket" in 1912 in a plane with a top speed of 50 mph in level flight.

He was the first person to survive a flat spin, though with many broken bones. He told me that he had been interrogated in detail by someone called Lindemann on what he had done to escape the spin. Lindemann said, "Now I know what to do," took a plane up, put it into a flat spin, and got out of it.

Lindemann (later Lord Cherwell) was Churchill's scientific adviser in the second world war.

T H Flewett, *retired consultant virologist, Solihull*