Deception among smokers

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Summary and conclusions

Subjects in two different clinical trials who had been advised to stop smoking were asked if they had done so. Some 22% of subjects (11 out of 51) in the first trial and 40% (33/82) in the second trial who said they had stopped smoking were found to have raised carboxyhaemoglobin concentrations. Deception appears to be common in people trying to stop smoking.

Introduction

Results of trials attempting to help people to stop smoking usually depend on statements on whether they have done so. In this respect, however, smokers may be untruthful.[1-5] We have investigated the extent of this deception in two groups of subjects by measuring carboxyhaemoglobin (COHb) concentrations in venous blood. We compared the results with those in known non-smoking members of hospital staff.

Subjects and methods

The following three groups of subjects were studied.

Group A consisted of 91 patients who had survived a myocardial infarction and were taking part in a multicentre secondary prevention trial evaluating dipyridamole and aspirin; 81 were men and 10 women, and their ages ranged from 41 to 76 (mean 60) years. They were followed up by MBW during the year after infarction. Routine advice on smoking had been given both in hospital and while attending an outpatient clinic. Blood samples were obtained during the trial for haematological and biochemical screening, including COHb estimation. Part of a questionnaire asked whether they were smoking.

Group B came from a trial of nicotine chewing gum as an aid to stopping smoking.[6] A total of 140 volunteers completed the one-month course, of whom 86 were men and 54 women; their ages ranged from 16 to 72 (mean 46) years. They attended the clinic weekly and were given advice and encouragement from REM to stop smoking. A blood sample was taken at each visit for COHb estimation.

In neither group were subjects aware of the purpose of the blood tests.

Group C served as controls and consisted of 161 members of hospital staff who were known to be non-smokers.

All blood samples were analysed in duplicate for COHb concentration with an IL 182 CO-Oximeter. The instrument was fully calibrated before each batch of measurements. Zero COHb calibration was obtained by tonometry using blood from a non-smoker in an IL 237 tonometer with oxygen for 18-24 hours.

Results

The COHb concentrations in group C ranged from 0·3% to 1·6% (mean 1·05% ± SD 0·26%). A concentration of 1·7% and over was set as the critical value for smoking (see figure).

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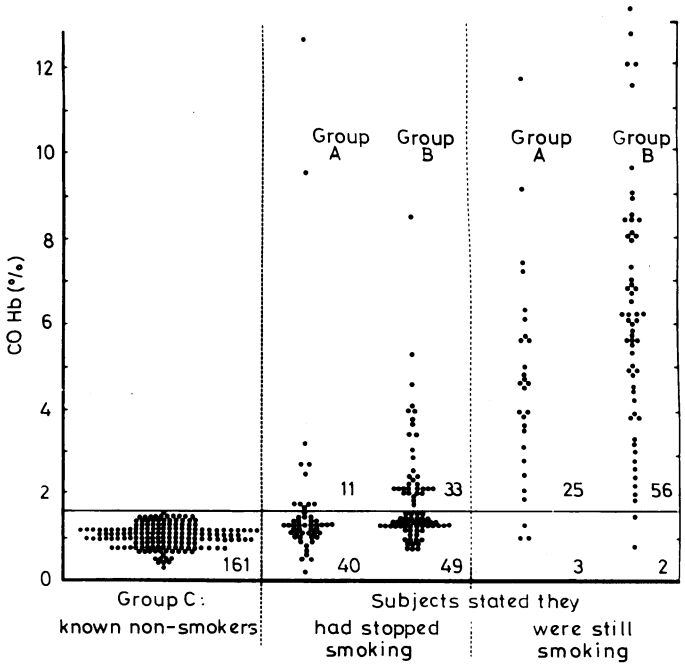

COHb concentrations in 161 known non-smokers and 219 subjects who had stated whether or not they had stopped smoking. Group A = 79 patients in coronary heart disease study. Group B = 140 volunteers in nicotine chewing-gum study. Bar represents upper limit of normal range (1·6%).

Of the 91 subjects in group A, 12 were lifelong non-smokers and were excluded from the study. Of the remaining 79, 28 stated that they were smoking and 51 that they were not smoking. Eleven of the 51 (22%) who stated that they were not smoking had COHb concentrations of 1·7-12·6% (mean 3·8%). Of the 82 subjects in group B who claimed not to be smoking, 33 (40%) had COHb concentrations ranging from 1·8% to 8·5% (mean 2·9%).

Twenty-five of the 28 subjects (89%) in group A and 56 of the 58 (97%) in group B who were still smoking had COHb concentrations of 1·9-13·4%. The other five had values of 0·8-1·5%, probably because they were light smokers or did not inhale.

Discussion

Our findings suggest that many people who say they have stopped smoking may, in fact, continue to smoke. Our recorded incidence of deception was higher than that reported by other workers. Jones *et al* thought that only one out of 21 taxi drivers was untruthful, but they used a critical COHb value of 3·0%.[1] Russell *et al* considered that only one out of 11 subjects who claimed abstinence was still smoking since his COHb concentration was 6·0%, but no critical level was given.[3]

Our conclusions depend on the accuracy of the COHb estimations, the critical COHb value in non-smokers, and the exclusion of other causes of raised COHb concentrations. We find that results with the IL 182 CO-Oximeter are accurate and reproducible to within 0·2%.[7] Our setting of 1·6% as the upper limit of normal for COHb in non-smokers was based on two factors. Firstly, in our control group the highest COHb value recorded was 1·6%, which occurred in a single subject. Secondly, the upper 95% confidence limit (mean + 2 SD) was 1·5%. This critical value for COHb may be questioned because of the possibility of deception among the controls. If some of them had deceived us and were smoking then the true critical value might

be even lower. This would have increased the deception rates in the other two groups.

COHb concentrations may be raised by factors other than smoking. These include leaking motor-car exhausts, pollution in streets and tunnels, industrial and domestic pollution, and, rarely, haemolytic anaemias. Non-smoking police on point-duty in Fleet Street were found to have only slightly raised values (mean 1·9%).[8] Only heavy exposure to the smoke of others causes raised COHb concentrations in non-smokers.[9] We have no reason to believe that any of these factors were present in our study. If any had been, however, all groups should have been affected to some extent.

A sensitive nose and a sceptical attitude towards patients who assert that they have stopped smoking will detect many who have not. No great clinical acumen is needed to detect those with stained fingers or smoky breath. But for others more discerning methods may be required. For this purpose COHb estimation is a valuable objective test. The half life of COHb in resting subjects is about four hours, and it may take 24 hours for normal values to be reached after stopping smoking. Few heavy smokers can stop this long even if forewarned. When raised COHb concentrations are found in those claiming they do not smoke other sources of exposure should be sought, though in our experience these are rare.

We found that a high proportion of subjects who claimed to have stopped were still smoking. Thus COHb estimation acted as a useful lie-detector. Patients confronted with a raised COHb concentration often remember the cigarettes smoked that they had "forgotten." The subjects in groups A and B were not aware that their veracity was being tested when the blood samples were taken.

If our findings are typical then the results of trials depending on patients' statements that they had stopped smoking need to be reassessed. For instance, among those attending the stop-smoking clinic (group B) 59% claimed to have stopped smoking after one month, but in only 32% was this confirmed by COHb estimation.

Studies showing improved prognosis in ex-smokers may have underestimated the true benefit of stopping smoking by including some people who continued to smoke. Likewise results of anti-smoking clinics and other cessation trials may have been worse than actually reported. Probably, of course, many subjects whose stated cessation was not confirmed had considerably reduced their consumption. This is supported by the results in our group B, in which the mean COHb concentration in the 33 "unconfirmed" subjects fell from 7·6% to 2·9%. This may be compared with the "confirmed" subjects, whose mean concentration fell from 6·2% to 1·3%. Stopping smoking must remain the criterion of success, since heavy cigarette smokers who reduce but do not stop smoking often return to their previous level of consumption.

Although we used 1·7% as the critical value for COHb concentration, other laboratories will probably use different values. Methods and instruments for measuring COHb are not standardised, and each centre should produce its own normal range.

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Amniotic fluid cell morphology in early antenatal prediction of abortion and low birth weight

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In 25 amniotic fluid samples containing normal amounts of AFP distinctive new patterns of RA cells were observed, termed fetal distress cells. These pregnancies resulted in five spontaneous abortions and 20 infants with birth weights under 2500 g. Fetal distress cells were not detected in any of the remaining 133 samples. One pregnancy was terminated because of a chromosomal abnormality, and there were seven twin pairs not recognised on ultrasonography before amniocentesis. The remaining 125 pregnancies went to term, resulting in infants with birth weights exceeding 2500 g.

The results suggest that RA-cell morphology will prove to be of value in the early antenatal prediction of spontaneous abortion and low birth weight.

Summary and conclusions

The morphology of rapidly adherent (RA) amniotic fluid cells was examined in 201 pregnant women referred for amniocentesis because of two sequential high serum α-fetoprotein (AFP) concentrations. Out of 43 amniotic fluid samples containing increased amounts of AFP, 42 had neural or peritoneal cells predominating among the RA cells, the outcome being an infant with a neural-tube defect or exomphalos. In the other case with a raised amniotic fluid AFP concentration but only anterior placental cells the infant was normal.

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Introduction

Maternal serum α-fetoprotein (AFP) screening has proved valuable in the early antenatal diagnosis of fetal neural-tube defects.[1-3] A raised serum AFP concentration, however, may