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Smoking, drinking, and polycythaemia

The mean packed cell volume is higher in the smoking population than in non-smokers.[1-3] Chronic hypoxaemia, from whatever cause, tends to cause a rise in the packed cell volume.[4,5] The obvious temptation is to invoke hypoxaemia, resulting from lung disease induced by smoking, to explain the high values found in smokers—but is this apparently straightforward explanation correct?

The increase in packed cell volume in chronic hypoxaemia is due mainly to increased erythropoiesis and a rise in the red cell mass. When the red cell mass is above the normal range the condition is termed absolute polycythaemia.[4,5] Evidence on the effect of smoking on plasma volume is conflicting,[6,7] but it is clearer on erythropoiesis and the red cell mass. In a study of patients with hypoxic cor pulmonale Calverley *et al* found a higher mean red cell mass in the smokers than the non-smokers despite the two groups being similar in age, severity of airways obstruction, and resting blood gas values.[7] There was a good correlation of red cell mass with the carboxyhaemoglobin concentration, suggesting that carboxyhaemoglobin might be contributing in some way to the polycythaemic response.

If patients with polycythaemia secondary to chronic hypoxia receive long term treatment with oxygen (15 hours a day or more) the red cell mass falls in those who stop smoking but not in those who continue.[7,8] Possibly the most convincing evidence that smoking itself influences erythropoiesis, independent of hypoxia due to lung disease induced by smoking, is the occasional occurrence of absolute polycythaemia in smokers without overt lung disease, hypoxaemia, or any other apparent cause for polycythaemia.[6,9,10] When such smokers stop smoking the red cell mass falls to normal.[6,9] Oxygen delivery to the imprecisely identified renal oxygen sensor is the main determinant of secretion of erythropoietin,[11] so how might this be affected by smoking?

As much as 16% of the total haemoglobin of heavy smokers may be bound to carbon monoxide and so not be available to bind with oxygen.[12] The affinity of haemoglobin for carbon monoxide is about 250 times that for oxygen; it may be 48 hours after stopping smoking before carboxyhaemoglobin concentrations fall to those seen in non-smokers.[13] The familiar sigmoid haemoglobin-oxygen dissociation curve predicts the percentage saturation of haemoglobin with oxygen that will occur at any given partial pressure of oxygen (Po_2), but when carboxyhaemoglobin is present only that predicted percentage of the available haemoglobin—that is, total haemoglobin less carboxyhaemoglobin—will take up oxygen. At any Po_2 the actual amount of oxygen carried by the blood will thus be lowered by the presence of carboxyhaemoglobin.

The percentage saturation figures produced by most blood gas analysers in clinical use are misleading in smokers: they do not measure the saturation directly but calculate it from the Po_2, Pco_2, and pH, no account being taken of any carboxyhaemoglobin present. Again, only the available haemoglobin (total haemoglobin less carboxyhaemoglobin) will be saturated to this degree. The clinician finding apparently reassuring blood gas results in a smoker should be aware that a respectable arterial Po_2 and calculated saturation may conceal a substantial reduction in oxygen content of the blood compared with that of a non-smoker with an identical arterial Po_2.

As well as occupying some binding sites and reducing the oxygen carrying capacity of the blood, carbon monoxide increases the affinity of the remaining haem sites for oxygen, shifting the haemoglobin-oxygen dissociation curve to the left.[14] At normal or marginally reduced alveolar Po_2 this increased affinity will result in only negligible increases in oxygen loading on to haemoglobin in the lungs, since these values of Po_2 are on the upper portion of the dissociation curve, where it is nearly flat. At tissue Po_2, however—on the steep part of the dissociation curve—the increased affinity of haemoglobin for oxygen will considerably reduce the oxygen released from binding; this may be further reduced by changes induced by carbon monoxide in the shape (rather than position) of the curve.[15] The smoker loses more on the swings of tissue oxygen release than he gains on the roundabouts of oxygen loading in the lungs.

There are good reasons, therefore, why smoking might reduce tissue oxygen delivery and stimulate erythropoiesis. Absolute polycythaemia due purely and simply to smoking may be uncommon, but in patients with a borderline or overtly low arterial Po_2 smoking will be an additional stimulus to erythropoiesis. Nevertheless, the increased packed cell volume associated with smoking is not widely recognised.

Paradoxically, the plethoric appearance of the alcoholic is part of folklore and everyday clinical experience, but firm evidence linking alcohol and polycythaemia is scanty. Acute abuse of alcohol inhibits the release of antidiuretic hormone,[16] and the result may be a high packed cell volume due to a low

plasma volume ("relative polycythaemia"). Some case reports have suggested that chronic alcohol abuse may cause a sustained increase in the packed cell volume by this mechanism,[17 18] but drinkers are usually smokers and the relative influence of the two factors in such cases may be hard to establish.[19]

The evidence that chronic abuse of alcohol might lead to increased erythropoiesis and absolute polycythaemia rather than just a fall in plasma volume is circumstantial. Studies in animals with liver damage have shown that they have higher concentrations of erythropoietin in response to anaemia[20] and hypoxia[21] than animals with normal livers. An outbreak of hepatitis B in a renal unit led to dramatic rises in haemoglobin and fall in transfusion requirements in affected patients during their period of liver dysfunction.[22] These data suggest a link between liver disease and erythropoietin production or metabolism, and high concentrations of erythropoietin have been found in patients with hepatocellular carcinoma.[23] Possibly alcoholics with liver damage might have increased erythropoiesis and a tendency to absolute polycythaemia independent of any relative polycythaemia due to haemoconcentration, but the evidence is lacking.

A further possible mechanism deserves attention: intermittent hypoxia causes polycythaemia in animals,[24] and many authorities believe that hypoxia during sleep may be an important determinant of the degree of secondary polycythaemia developing in patients with chronic obstructive airways disease.[25-27] Alcohol depresses respiration[28]; in patients with obstructive sleep apnoea it increases the frequency and duration of apnoea and worsens desaturation, and it precipitates overt apnoea in snorers.[29] Heavy drinkers might have more pronounced nocturnal hypoxia than abstainers—with the consequent stimulation of erythropoiesis and a tendency to an absolute polycythaemia.

The message is clear regarding smoking in patients with polycythaemia, and measurement of carboxyhaemoglobin concentrations should be part of their investigation. Although polycythaemia is less certainly linked with alcohol, an association seems possible. The smoking and drinking habits of the patient should be borne in mind when a high packed cell volume—or absolute polycythaemia—is either inexplicable or seems to be out of proportion to the degree of any hypoxia present.

JOHN MOORE-GILLON
Lecturer in medicine
T C PEARSON
Reader in haematology

United Medical and Dental Schools,
St Thomas's Hospital,
London SE1 7EH

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Faecal incontinence is not inevitable

Faecal incontinence is one of the more unpleasant consequences of failing health in old age—distressing for the sufferer, and unpleasant for his or her carers. Despite the lack of published evidence geriatricians and the nurses who work with them know from experience that faecal incontinence is not inevitable. Why then is it not prevented?

The main obstacle is ignorance. Tobin and Brocklehurst have recently shown in a controlled trial the effectiveness of simple measures available to any primary care team.[1] From 30 residential homes in Manchester they chose 82 residents with regular faecal incontinence, a 10% prevalence in line with previous studies.[2 3] Those with diarrhoea were excluded. Fifty two residents were randomly selected for treatment and 30 as controls. The two groups were comparable in terms of age, duration of incontinence, mobility, and mental function. Three quarters were mentally impaired. So low were expectations that only two residents had been referred to their general practitioners—yet three quarters had been incontinent for over a year.

Faecal impaction was considered to be the cause of the incontinence when there was a history of continuous faecal soiling in the presence of a loaded rectum and a lax anal sphincter. Neurogenic incontinence was diagnosed when formed stools were passed and rectal examination gave normal results.

With the collaboration of the primary care team patients with faecal impaction were treated with daily enemas until there was no response. They were then given lactulose twice daily and a weekly enema to prevent recurrence. Those with neurogenic incontinence were treated with codeine phosphate to produce constipation. They were then given two enemas a week to produce a bowel movement at a predictable time.

After two months two thirds of the study patients were no longer incontinent compared with only a third of the controls. Those with faecal impaction did better than those with neurogenic incontinence and some patients did not comply fully with the prescribed regimen. In those in whom full compliance was achieved the results were even better,