Treatment of cigarette smoking with short-term high-dosage corticotrophin therapy: preliminary communication¹

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Summary: Uncontrolled studies have indicated that the smoker's 'lift' can be simulated clinically by short-term, high-dosage corticotrophin therapy. Following such treatment, smokers previously prevented by the withdrawal syndrome from giving up smoking were able to do so easily. All 10 patients reported here were still not smoking after seven months.

Introduction

Anti-smoking clinics using a variety of methods report a long-term success rate of between 10% and 20% (Higenbottam & Chamberlain 1984). No significant difference has been shown to exist between the success rate following the use of nicotine gum and that of plain gum (Jamrozik *et al.* 1984). A search of relevant medical literature has revealed no previous attempt to infer a clinical approach to the management of tobacco addiction from the known physiology of nicotine.

Nicotine inhalation causes a temporary rise in blood sugar which coincides with the smoker's 'lift' (Haggard & Greenberg 1934). The rise in blood sugar is the result of an increased output of glucocorticoids by the adrenal cortex in response to stimulation by endogenous corticotrophin secreted by the anterior pituitary gland in response to unphysiological stimulation with nicotine (Kershbaum *et al.* 1968). It is possible, therefore, that the smoker's 'lift' is initiated by endogenous corticotrophin. It is also possible that in smokers the capacity of the anterior pituitary gland to produce normal quantities of corticotrophin is impaired by repeated unphysiological stimulation with nicotine. This would result in secondary hypoadrenocorticism and a consequent reduced output of glucocorticoids with a tendency to hypoglycaemia. The latter would result in a reflex increase in adrenaline production which would tend simultaneously to correct the hypoglycaemia and cause feelings of nervous tension. The hunger caused by the low blood sugar and the nervous tension caused by the extra circulating adrenaline may be the basis of the smoking withdrawal syndrome.

The smoking withdrawal syndrome has been shown to resolve when endogenous corticotrophin is secreted in response to inhaled nicotine (Kershbaum *et al.* 1968). The withdrawal syndrome might, therefore, also resolve in response to exogenous corticotrophin therapy. If this were the case, the relief from withdrawal symptoms brought about by corticotrophin therapy would enable patients to abstain from smoking. At the same time, the anterior pituitary gland might recover its capacity to produce adequate corticotrophin as a result of no longer being impeded by the adverse effects of repeated unphysiological stimulation with nicotine.

Methods

During July 1984, 10 smokers were treated with high-dosage short-term corticotrophin therapy, each being given an initial intramuscular injection of 180 iu corticotrophin gelatin (Acthar Gel). This was found to reduce considerably or abolish the smoking withdrawal syndrome. As a result, all patients were able to stop smoking or to reduce their cigarette consumption. Three patients had a slight exacerbation of withdrawal symptoms after 3–6 days and

¹ Accepted 20 March 1985. US patent applied for

Table 1. Patients treated with corticotrophin for cigarette smoking (7 month follow up)

Patients age/sex	No. smoked per day	Years smoked	Day of booster	Weight gain in stones (kg)
36/F	20	19	—	1 (6.3)
38/M	40	20	—	$\frac{1}{2}$ (3.2)
45/M	25	25	3	—
37/M	25	17	—	$1\frac{1}{2}$ (9.5)
24/M	10	10	—	$\frac{1}{2}$ (3.2)
38/F	25	5	6	—
50/M	10	25	—	—
70/M	80	50	6	—
49/F	60	30	—	1 (6.3)
52/F	20	33	6	—

were given a second injection of 80 iu Acthar Gel; this abolished their withdrawal symptoms altogether.

Results

All patients except one stopped smoking after the first injection. A second injection in 3 patients who had a slight exacerbation of withdrawal symptoms after a few days enabled them not to resume smoking. One patient who was accustomed to smoking 80 cigarettes a day was able to reduce to 30 cigarettes after the first injection and to cease smoking altogether after the second.

After seven months, none of the patients had resumed smoking although 50% had gained weight (Table 1). No other side effects were reported.

Discussion

Although these preliminary results appear promising they should be regarded with caution, since the trial was uncontrolled and the validity of the results was not confirmed with estimations of blood carboxyhaemoglobin levels.

Continuing observations on the management of cigarette smoking are indicating that attention should also be paid to clinical nutrition and to the smoking habit. A tendency to hypoglycaemia has been shown to occur following cessation of smoking (Glauser *et al.* 1970), and the nutritional regimen is therefore designed to reduce this (Tintera 1955). The smoking habit is controlled by the utilization of substitutes for oral gratification, such as plain chewing gum and small snacks (Health Education Council 1984).

It is concluded that while the preliminary results of short-term low-dose corticotrophin therapy have been encouraging, the clinical approach to the treatment of smoking should also encompass other factors such as stress control and a balanced diet. Further clinical studies are in progress to determine an optimal clinical regimen.

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