

A Psychophysiological Re-evaluation of Eysenck's Theory Concerning Cigarette Smoking

Part I. The Central Nervous System

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SUMMARY

According to Eysenck, extraverts are characterized by inhibited cortical activity accompanied by prominent alpha brain rhythms. This prevents efficient cortical functioning. They have a 'stimulus hunger' in order to increase cortical efficiency. Assuming that nicotine is a stimulant drug, Eysenck puts forward his theory that extraverts will also have a 'stimulus hunger' for the nicotine in cigarettes and will therefore smoke more than introverts, to whom the reverse applies. Implicit in Eysenck's theory is a positive, causal relationship between the amount of alpha brain rhythms and the number of cigarettes smoked.

Inspection of the literature, however, indicated that small doses of nicotine stimulate the nervous system, whereas large doses tend to inhibit it. Eysenck's theory was therefore challenged by the alternative hypothesis that light smokers are characterized by prominent alpha brain rhythms and smoke for stimulation. Heavy smokers are, however, characterized by a small amount of alpha activity (overactivated cortex which also prevents efficient functioning), and therefore smoke for inhibition to enhance their cortical efficiency and thus their alpha activity.

The results were reconcilable with this hypothesis. The positive relationship implied by Eysenck's theory only held good for light and moderate smokers. Heavy smokers probably smoke for cortical inhibition.

S. Afr. med. J., 52, 237 (1977).

Eysenck¹ uses the concepts of inherent cortical excitation and inhibition to put forward a theory concerning smoking behaviour. Moderate excitation has a facilitative action on the cerebral cortex in mediating perception, attention and mental work in general. Such a brain is optimally qualified to deal with incoming information and reacts appropriately. Inhibition, however, prevents efficient cortical functioning, rendering the brain less able to cope. The same applies to a highly activated cortex.

Eysenck^{2,3} states that, although nicotine is a stimulant drug, its psychopharmacological effect in each case is determined by the level of cortical excitation or inhibition. It stimulates an inhibited cortex, whereas it has a paradoxically inhibitory effect on a highly activated cortex.

According to Eysenck, extraverts are characterized by inhibited cortical activity accompanied by prominent alpha brain rhythms. They have a 'stimulus hunger' in order to increase cortical arousal and thus to enhance cortical efficiency. They will therefore smoke heavily. The more inhibited the cortex (and therefore the more prominent the alpha rhythm), the more they smoke in order to maintain an optimal level of cortical arousal.

Introverts, with high cortical excitation, avoid stimulation in order to decrease cortical arousal and enhance cortical efficiency. They smoke few cigarettes for the paradoxically inhibitory effect. Eysenck explains that owing to their high cortical excitation, the stimulating action of nicotine easily brings them to the arousal threshold, resulting in paradoxical cortical inhibition.

The condition of the central nervous system (CNS) therefore determines whether an individual will be a light or heavy smoker. Eysenck *et al.*^{4,5} found a significant positive relationship between the degree of extraversion and the number of cigarettes smoked.

Eysenck's assumption that nicotine is a stimulant drug whose psychopharmacological effect is solely determined by the level of cortical excitation or inhibition is, however, open to question. Inspection of the literature indicated that the action of nicotine is dose-dependent. Small doses stimulate the nervous system, whereas large doses tend to inhibit it.⁶⁻⁸

It seems unlikely that individuals with high cortical arousal can obtain sustained cortical inhibition by smoking few cigarettes. Although the paradoxical effect is possible, it seems more probable that they will obtain inhibition by the intake of large doses of nicotine (heavy smoking).

With this evidence in mind, a reformulation of Eysenck's theory seemed desirable. Therefore, the aims of the present investigation were as follows:

1. Since small doses of nicotine stimulate the CNS and large doses tend to depress it, the hypothesis was tested that, should there be a causal relationship between the amount of alpha brain rhythms and the number of cigarettes smoked, it would, in contrast to Eysenck's theory, be as follows:

Light smokers have a large amount of alpha brain rhythms (inhibited cortex), and therefore smoke for cortical excitation, which reduces the amount of alpha brain rhythms. Heavy smokers, on the other hand, have a small amount of alpha brain rhythms (overactivated cortex) and therefore smoke for cortical inhibition, which enhances the amount of alpha rhythms.

2. In association with this hypothesis it was investigated whether non-smokers, light smokers and moderate-to-

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Date received: 25 January 1977.

heavy smokers differ in the reactivity of their CNS to stimulation.

SUBJECTS AND METHODS

Subjects

The subjects studied were 36 White male second- and third-year psychology students at the University of Stellenbosch, divided into 3 groups of 12: group I — non-smokers (subjects who had never smoked up to the time of testing); group II — light cigarette smokers (less than 12 cigarettes a day); group III — moderate-to-heavy cigarette smokers (12 and more cigarettes a day).

The ages of the subjects ranged from 19 years 2 months to 26 years 1 month, with a mean age of 21 years 4 months and a standard deviation of 19.1 months.

Apparatus and Method

As part of a more extensive study, a 6-channel Grass Model 5 polygraph was used to obtain, among other physiological variables, a continuous recording of the EEG of the right occipital-temporal region of each subject. This channel was also connected to a Faraday electronic low-frequency waveform analyser which analysed the brain rhythm frequencies in every successive 10-second epoch of the EEG. The frequency histogram of the analyser (in the form of a series of spikes) was written out simultaneously with the EEG on the same recording paper. The amplitude of each spike denoted the summation in time of the voltage amplitude of a particular frequency component of the original EEG data.

During this recording each subject was presented with 14 stimuli, while reclining on a bed with his eyes closed. The first 8 were auditory stimuli. Stimuli 1-5 and 7-8 were identical and consisted of a staccato sound resembling machine-gun fire, presented at an intensity of 78 db. Stimulus 6 was a door bell with an intensity of 68 db. These stimuli were presented every 50 seconds and each lasted 5 seconds.

Stimuli 9-14 were words differing in emotional loading, which are connected with cigarette smoking to different degrees (e.g. 'lung cancer'), as well as cognitive tasks of different complexity (e.g. counting from 100 backwards in threes). This was followed by a 2-minute rest period during which the subject relaxed, with his eyes still closed.

The overhead light was off and room temperature was kept constant at 22°C throughout the polygraphic recording.

Treatment of Data

For each stimulus, the prestimulus level of amount of alpha rhythms (in the epoch prior to stimulation) and the response level (in the epoch during stimulation) were obtained for each subject. For a specific epoch this was obtained by measuring the height (in millimetres) of the analyser spike for each frequency from 8 to 13 Hz, multiplying each height with the frequency concerned and adding these numbers.

These scores were converted to Lacey's Autonomic Lability Scores (ALS),⁹ as it was indicated that these scores also apply to the reactivity of the CNS.¹⁰ The formula

$$ALS = 50 - 10 \left[\frac{Z_y - Z_x r_{xy}}{\sqrt{1 - r_{xy}^2}} \right]$$

was used to ensure that a large decrease in the amount of alpha rhythms resulted in a large score. In this formula Z_x and Z_y represent the subject's standardized prestimulus level score (X) and reaction level score (Y), whereas r_{xy} represents the correlation between all X and Y scores of all the subjects.

This conversion was performed to solve the problems implicated by Wilder's law of initial values,¹¹ which states that the response to agents stimulating the function under investigation depends to a large extent on the initial level of that function. If that level is low, there is a tendency to a marked increase; if the level is average this tendency is less marked; if the initial level is high there will often be a minimal or no increase and quite often a paradoxical drop in the function. The reverse applies to inhibitory agents.

To compare the magnitude of responses within or between persons, some adjustment must therefore be made to eliminate or equate the influence of the prestimulus level on the response magnitude. Working on a regression model, ALS proposes a solution for this problem. It reflects the extent of the difference between the response level and the estimated response level based on the mean responses of the other subjects who had the same prestimulus level.

The ALSs were mainly subjected to profile analysis by multivariate statistical methods. For this purpose, Hotelling's T^2 -statistic was employed to analyse the differences between the profiles of the means of the groups.¹² The raw scores for the prestimulus and response levels were, however, also analysed in detail. Owing to the differences in the nature of stimuli 1-8 and 9-14, the scores for these two groups of stimuli were analysed separately.

The mean number of alpha waves during the 2-minute rest period was calculated for each subject by determining the amount of alpha waves in each of the 12 epochs as described previously. These 12 scores were added and divided by 12.

RESULTS AND DISCUSSION

The profile analyses indicated that group 1 showed a significantly higher degree of reactivity in the amount of alpha waves than group 2 to stimuli 1-8 ($T^2 = 42.3$; $P < 0.05$). For group III, the Bravais-Pearson correlation between the number of cigarettes smoked and the amount of alpha waves in the rest period was significantly negative ($r = -0.748$; $P < 0.01$). For groups II and III combined, this correlation was again significantly negative ($r = -0.449$; $P < 0.05$).

In order to interpret these results in terms of a possible causal relationship between the amount of alpha waves and the number of cigarettes smoked, it was necessary to

compare the amount of alpha waves between stimuli (as reflected by the epoch prior to each stimulus) and during stimulus presentation with the amount of alpha waves during the rest period. When this was done, inspection of the individual scores of the subjects revealed an important phenomenon. Concerning the amount of alpha waves, group III (moderate-to-heavy smokers) formed 2 distinct subgroups. The heavy smokers of group III (20+ cigarettes/day, referred to as group IIIB with $N = 4$) had fewer alpha waves than the moderate smokers (12-18 cigarettes/day, referred to as group IIIA with $N = 8$).

The mean amount of alpha waves for groups I, II, IIIA and IIIB during the different stages of the experimental procedures are shown in Table I and are graphically presented in Fig. 1.

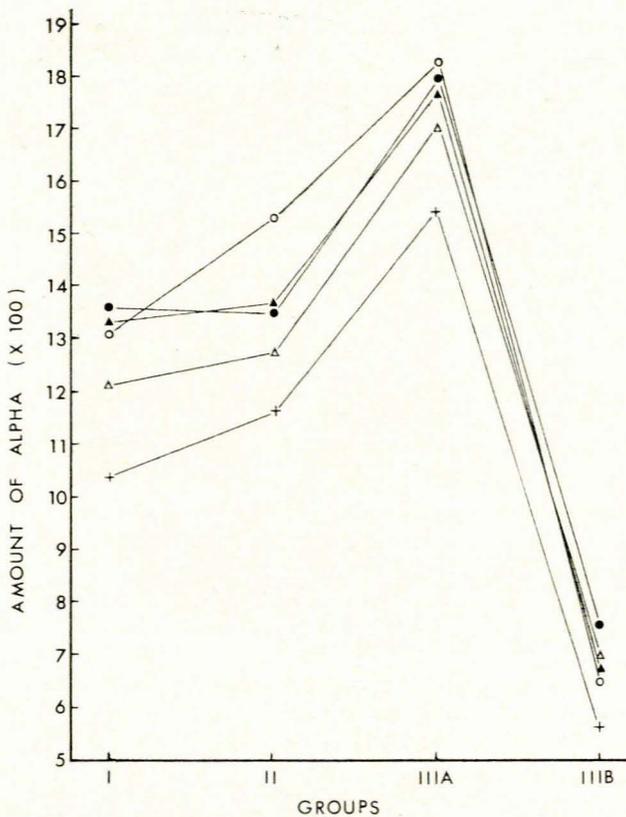


Fig. 1. Mean amount of alpha waves (○—○ rest period; ●—● between stimuli 1-8; △—△ during stimulation (1-8); ▲—▲ between stimuli 9-14; +—+ during stimulation (9-14)).

The median test indicated that the increase in the amount of alpha waves from non-smokers to light smokers to moderate smokers was significant ($\chi^2 = 7.5$; $P < 0.05$). The Mann-Whitney U-test yielded a significant difference in the amount of alpha waves between groups IIIA and IIIB ($U = 1$; $P < 0.01$).

Another important phenomenon was the significant difference in group II between the amount of alpha waves in the rest period and in the intervals between stimulation by stimuli 9-14 ($t = 2.58$; $P < 0.05$). The corresponding t -value regarding stimuli 1-8 was, however, insignificant. The other groups showed no significant variability in the amount of alpha waves. It therefore seems reasonable to describe group II as labile as far as this variable is concerned.

The existence of an optimal level of amount of alpha waves and the idea that smokers, by inhaling nicotine, change their amount of alpha waves (and therefore the degree of excitation of the brain) in the direction of that optimal level, is implicit in Eysenck's theory. It is difficult to determine where the optimal level lies. On theoretical grounds the level of the non-smokers (group I) was regarded as the optimal level for interpreting the present results.

In view of the results obtained it seems that, should there exist a causal relationship between the amount of alpha rhythms and the number of cigarettes smoked, such a causality could be stated as follows:

Light smokers (group II) are very labile as far as the amount of alpha waves is concerned. They are therefore highly susceptible to high levels of alpha waves (inhibition), but also to moderate levels of alpha waves (excitation). During their normal daily routine they are subjected to fairly continuous stimulation. Their level of alpha waves therefore coincides with that of non-smokers (and therefore the optimal level). Whenever stimulation abates, the amount of alpha waves increases rapidly, and to reduce it they smoke a cigarette. They therefore smoke for stimulation, but then only during a state of relaxation, when more alpha waves are present. During their normal daily routine this seldom happens and they therefore smoke few cigarettes. Since they smoke infrequently, they develop hardly any tolerance to the psychopharmacological action of nicotine, and each cigarette stimulates to an efficient degree.

Moderate smokers (group IIIA) have a large amount of alpha waves (more than the other groups), but they are also less reactive to relatively mild stimulation (like the intervals between stimuli). To more intense stimulation

TABLE I. MEAN DEGREE OF ALPHA WAVES

Stimuli	Location of measurement	Group I	Group II	Group IIIA	Group IIIB
1 - 8	Between stimuli	1 357,2	1 345,3	1 807,4	757,9
	During stimulation	1 213,9	1 275,9	1 701,8	700,0
9 - 14	Between stimuli	1 324,2	1 366,6	1 777,2	664,2
	During stimulation	1 030,6	1 156,5	1 531,7	557,6
	Rest period	1 310,1	1 535,5	1 825,2	655,6

they, however, show higher reactivity (basically the same as group II). Even during stimulation by stimuli 9-14 they have the same amount of alpha waves as group II during the *rest period*, and in all 5 situations they have a larger amount of alpha waves than group I ever reaches. These individuals therefore frequently require a high degree of stimulation to reach the optimal level ('stimulus hunger') and thus smoke a moderate amount of cigarettes. They therefore also smoke for the stimulation afforded by nicotine, but smoke more than group II, since they require more stimulation to reach the optimal level. The fact that they smoke more cigarettes probably results in the development of more tolerance to the action of nicotine, and they thus have to smoke more to retain the effect.

Heavy smokers (group IIIB) have a very small amount of alpha waves in comparison to the other groups, and also show little reactivity to stimulation. The latter fact can probably be related to Wilder's law of initial values. They already have such a small amount of alpha waves (overactivated brain) that they can hardly show any further decrease. The fact that they have so little alpha wave activity makes it seem unlikely that they will crave more stimulation. It therefore seems reasonable to expect them to smoke a *large* number of cigarettes for the *inhibitory effect of large doses of nicotine* in order to enhance their amount of alpha waves to the optimal level. Here, too, tolerance to the inhibitory action of nicotine

will develop, compelling them to smoke much more to retain the effect.

This interpretation of the present results thus indicates that the positive causal relationship between the amount of alpha waves and the number of cigarettes smoked, which is implicit in Eysenck's theory of 'stimulus hunger', only holds good for light and moderate smokers. Heavy smokers probably form a unique group whose behaviour is directed towards 'stimulus reduction'. On the whole, the results of this investigation were reconcilable with the tested hypothesis.

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(A list containing some 120 references concerning smoking behaviour and the action of nicotine is available from the author.)

The Hazards of Subclavian Vein Catheterization

Practical Considerations and an Unusual Case Report

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SUMMARY

A case of intrapleural displacement of a piece of catheter as a complication of subclavian vein catheterization is reported. A brief review of the literature concerning complications of subclavian catheterization is presented, together with a logical classification and methods of prevention.

S. Afr. med. J., **52**, 240 (1977).

Subclavian vein catheterization provides convenient and rapid access to the venous system and has been widely

used for rapid infusion of intravenous fluids, for monitoring of central venous pressure, and for the infusion of hyperosmolar solutions used in intravenous alimentation.

Complications can occur, and in recent years many articles have appeared reporting either a series of subclavian catheterizations and the associated complications¹⁻⁶ or case histories describing individual complications.⁷⁻¹⁰

Subclavian catheterization complicated by free migration of a broken catheter across the pleural cavity has not previously been reported.

CASE REPORT

A 38-year-old woman, para 9, was admitted in obstructed labour of 48 hours' duration. She had severe abdominal pain with rigidity and guarding. Her haemoglobin level was 10.5 g/100 ml, systolic blood pressure 80 mmHg

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Date received: 4 February 1977.