Association, would receive a fresh impetus. Perhaps, as we have seen, our own B.M.A. Commonwealth Conference and Overseas Committee can take the initiative.

But the overwhelming satisfaction of achieving this co-operation—and in what field is there more prospect of success than in medicine and health?—would lie in the substantial step it would represent towards human brotherhood and co-operation. We are already, through the advent of the radio and the aeroplane, one world, for better or for worse. Here is a chance to emphasize the good in contrast to the many opportunities for evil. All the peoples of the world suddenly seem to have grasped the idea that they need not continue in hunger, poverty, ill-health, and ignorance. They know with Isaiah that every valley can be exalted and every mountain and hill laid low, and they are determined in action to bring about the changes they desire. The newly awakened countries may not know clearly what the good life is, or how to achieve it—any more perhaps than we do in the wealthier countries; but they do recognize some of the bad features of their present condition, as we do, and they will try to remedy them. They see in technology the way to grow more food, secure more shelter, and gradually to ameliorate their lot. How proper it is for us, who for the moment—perhaps a very fleeting moment—know a little more about these things, to wish to help; how dismal our failure if we sit at ease in our Zions and do nothing. In terms of practical politics and even of self-interest it may be better to lend a hand—when and where it is wanted—while the brief opportunity is there.

It is not only in medicine and health that Commonwealth-wide co-operation is promising. The British Association meeting at Cardiff in September, 1960, talked about food production and mouths to be fed on the world scale. It was obviously again "one world" where mutual interdependence and co-operation seemed desirable, indeed inescapable. Progress in agriculture and in health go very much together, the one helping the other towards human betterment. It was Lord Bruce of Melbourne who wished to contrive this marriage some years ago at the United Nations. Similarly, in science generally we can hope for valuable results by wider co-operation. Again the process should be an easy one. For the principles on which the scientist works are of universal validity and application, and men meet in scientific discussion on equal terms, without any reference to class, creed, or colour. One world of science is therefore already with us in principle—let us have it in practice and directed towards beneficent ends. The final union of all mankind is the ultimate hope, in the Parliament of Man and the Federation of the World.

The Theme Again

There have been many major proposals and steps towards united efforts in recent years which should encourage us to feel that a comprehensive scheme for health in the Commonwealth is not too visionary and that it might be realized even in five to ten years' time. We still remember the feeling of wild surmise early in the war when Mr. Churchill proposed for us a common citizenship with France. Since the war there have been various plans for economic co-operation, common markets, and so forth between units and nations hitherto considering themselves as inviolable units. Even in defence we have the proposal to vest the Western European atomic weapons in an association of States rather than in one or more individual ones. These plans

have made real progress, and in a few years many things that seemed utterly unrealistic have, in fact, come about. In the same way the current proposals, looked back upon from ten years hence, may seem quite modest. There has been great success already in international co-operation in science, in health, and in education.

Probably the chief obstacle, as always, lies in ourselves. We need—even as doctors—more of the compassion towards our fellow men that is our distinctive mark, so that we will want to spread our net of service wider. We need also the touch of imagination that will enable us to glimpse greater possibilities. The man with the muck-rake in *The Pilgrim's Progress* is too much with us. He was so bent on heaping together the dust and the leaves that he never looked up, and never saw the celestial crown above his head. Let us straighten our backs and look up.

REFERENCES

- ¹ "Commonwealth and Common Health," Sir Arthur Porritt, *Brit. med. J.*, 1960, **1**, 1907.
- ² "Medical Research in the Colonies." Leading Article, *ibid.*, 1960, **2**, 588.
- ³ "Coming Home and Going Abroad." Leading Article, *Lancet*, 1960, **2**, 1013.
- ⁴ "A Time Abroad." Leading Article, *ibid.*, 1959, **1**, 767.
- ⁵ "On Being Seconded." Personal Paper, *ibid.*, 1959, **1**, 778.
- ⁶ W. C. W. Nixon, *ibid.*, 1959, **1**, 831.
- ⁷ "Filling Medical Appointments Overseas." Report of B.M.A. Conference, *Brit. med. J.*, 1958, **2**, Suppl. pp. 220-224. Leading Article, *ibid.*, 1958, **2**, 1279.
- ⁸ "One World in Medicine," T. C. Routley, *Brit. med. J.*, 1958, **1**, Suppl. p. 21.
- ⁹ "Medical Service Within the Commonwealth and Other Countries Overseas," Kenneth R. Hill, *ibid.*, 1961, **1**, 117.

CIGARETTE SMOKING AND ISCHAEMIC HEART DISEASE

BY

B. BRONTE-STEWART, M.D., M.R.C.P.

Physician and Senior Lecturer

(From the Department of Medicine, University of Capetown, and Groote Schuur Hospital, Capetown, South Africa)

Attention was drawn to the association of cigarette smoking and mortality from ischaemic heart disease in two surveys on smoking habits and lung cancer (Hammond and Horn, 1954; Doll and Hill, 1956). The following points are worthy of emphasis: that this finding was an incidental one; that the two surveys were quite independent, one being carried out in the United States of America and the other in Great Britain; and that a graded association existed in that the mortality increased in proportion to the amount smoked. Higher mortalities were seen in those who smoked cigarettes as opposed to pipes or cigars. Despite this, the finding has been the subject of criticism in its statistical aspects (Berkson, 1955).

Epidemiologically, too, there is a relationship between the prevalence of ischaemic heart disease and fashions in tobacco consumption. Since the invention of the cigarette-making machine in 1880 (*Lancet*, 1957) the fashion of pipe smoking has given way to cigarette smoking, and the increasing rate of tobacco consumption in this manner parallels the increasing mortality from ischaemic heart disease (see Fig. 1). In England, too, the higher mortalities of the professional and executive classes seen in the occupational mortality statistics of the Registrar-General (1954) could be attributed to the

fact that economic factors influence smoking habits (Todd, 1957). Men smoke more than women, and the rationing of tobacco during the 1939-45 war years in Europe could be held responsible for the sharp decrease in mortality from circulatory diseases that occurred (Strøm and Jensen, 1951). Another epidemiological feature of ischaemic heart disease—the wide interracial differences in mortality—bears some relationship to differences in the cigarette-smoking habits in those countries from which such information is available (see Fig. 2) (Todd, 1957). Closer examination reveals certain discrepancies. Finland and Japan, for example, do not differ greatly in smoking habits but have vastly different mortality rates. In fact, Fig. 2 may merely emphasize

FIG. 1.—Since the turn of the century tobacco consumption in the form of pipe and cigar smoking has given way to cigarette smoking in the United Kingdom. This rise in cigarette consumption parallels the increase in mortality from ischaemic heart disease.

FIG. 2.—Interracial mortality from ischaemic heart disease in relation to cigarette consumption. A high consumption of cigarettes per adult per annum is seen, particularly in those countries with a high mortality from ischaemic heart disease.

that heavy cigarette smoking is a habit acquired especially by those in the English-speaking world.

Nevertheless epidemiologically there is sufficient evidence to support the findings of the lung-cancer surveys. On examining the association further one is immediately confronted with two major possibilities. Firstly, has the smoking of a cigarette a direct effect by interfering with either coronary blood flow or heart action? Secondly, is the effect indirect in that both cigarette smoking and ischaemic heart disease mortality are related to a third and common factor?

Direct Effect

There is little to suggest that the increased mortality in cigarette smokers is due to the effect of tobacco products on the coronary circulation. The onset of myocardial infarction during or immediately after the smoking of a cigarette has not as yet been related, and the term "tobacco angina" is now less fashionable. Pickering and Sanderson (1945) found no evidence to suggest that smoking produced coronary vasoconstriction, for smoking before or during exercise did not reduce the exercise tolerance in a subject with angina pectoris. They felt that smoking was a factor in producing pain by virtue of its increasing the work of the heart through raising the pulse rate and blood-pressure.

Bargeron *et al.* (1957), by catheterizing the coronary sinus in order to obtain coronary venous blood, measured the effect of smoking on coronary blood flow, coronary vascular resistance, and the myocardial extraction of oxygen, glucose, lactates, pyruvates, and ketones. Their results failed to show any impairment resulting from smoking.

Ballistocardiographic changes occur after the smoking of a cigarette in persons with and without ischaemic heart disease, particularly when they are over the age of 50 years (Russek *et al.*, 1955; Davis *et al.*, 1956). There is more evidence to suggest a relationship of these changes to the peripheral circulation than to alterations in the coronary circulation or myocardium. So far it has not been possible to identify physiological changes in the coronary circulation as a result of cigarette smoking which would account for the statistically higher incidence of coronary disease in smokers (*Brit. med. J.*, 1956).

With respect to blood-clotting, Blackburn *et al.* (1959) studied the effect of smoking on the recalcified plasma "stypven time." The only change of statistical significance before and after smoking was a rise in pulse rate. These studies on the immediate response to a single cigarette may be little guide to the long-term effects of heavy cigarette smoking. Studies on groups with and without ischaemic heart disease revealed that fibrinolysis in the cigarette smokers of each group was more rapid than in the non-smokers, but the differences were not significant (Lackner and Bronte-Stewart, personal observations, 1960).

Indirect Effects

It is indeed possible that the smoking habit and ischaemic heart disease may be related indirectly in that both may be related to a "third and common factor" (*Brit. med. J.*, 1955). Sedentary occupations may allow the opportunity for greater consumption of cigarette

tobacco, and most heavy cigarette smokers agree that their smoking rates increase during periods of mental strain. In order to study these possibilities, analysis of certain data collected from an interracial survey in the Cape Peninsula during 1955 (Bronte-Stewart *et al.*, 1955) was pursued. This survey occurred prior to the widespread adverse publicity given to the cigarette-smoking habit with respect to health.

Material and Methods

The sample consisted of approximately 600 apparently healthy adult men aged 25 to 55 who were actively engaged in their occupations. All who had clinical, haematological, or electrocardiographic abnormalities were excluded from further analysis. Data were obtained from the individuals concerning their income, occupation, smoking habits, and customary diet. The information on the diet was obtained by recall, particular emphasis being laid on specific items of food that contained fat, such as meat, dairy products, eggs, margarine, oils, etc. Quantification was based on the average helping method and the fat content was computed from food tables. Anthropometric measurements were recorded and the cholesterol was chemically determined in the serum and after filter-paper electrophoresis in the alpha and beta fraction (Anderson and Keys, 1956).

In the analysis of the results each racial group was subdivided into three income classes, but the economic scale is such that the high-income Bantu would overlap into a similar economic plane as the low- and medium-income Cape Coloured group, whereas the high-income Cape Coloured subdivision would be on an economic par with the low-income European. Cigarette smokers were analysed separately from pipe smokers and were classified into heavy, medium, and light, if they smoked more than 20 a day, between 10 and 20, and fewer than 10 respectively. Later, owing to small sample size, the light and medium classes were grouped together. No account was taken of whether filter- or cork-tipped or plain cigarettes were preferred. Pipe smokers were classified into heavy, medium, and light, if more than $\frac{1}{4}$ lb. (113 g.) was smoked in a fortnight, if this was smoked in a month, or less than this per month respectively. As there were too few cigar smokers, tobacco chewers, or snuff takers, they were excluded from the analysis.

FIG. 3.—Smoking habits of Capetown's three racial groups. The smoking habits of the Cape Coloured and European are very similar, despite marked differences in death rate from ischaemic heart disease.

Results

Smoking Habits of the Three Racial Groups.—The smoking habits of the Cape Coloured and European are very similar, but the Bantu differ markedly (see Fig. 3). Of the Bantu sampled 81% claimed that they never smoked cigarettes, but over half could be classed as heavy pipe smokers. Conversely there was a high proportion of men who never smoked pipes among the Cape Coloured (73%) and the European (76%), whereas 31% of the former and 39% of the latter were heavy cigarette smokers. As only 5% of the Bantu sample could be classed as heavy cigarette smokers, all Bantu were excluded from further analysis, which meant that the sample consisted now of 179 Cape Coloured and 181 European men.

Economic Privilege and Tobacco Consumption.—In Capetown, tobacco is relatively inexpensive and the supply is plentiful. A packet of 20 cigarettes (king size, filter tip) of average grade cost just less than 1s. 6d. in 1955, while a $\frac{1}{4}$ lb. (113 g.) of pipe tobacco could be bought for less than 2s. 6d. In the European sample,

FIG. 4.—The distribution of obesity is similar in both smokers and non-smokers as measured by the combined arm and scapula skin fold.

economic privilege bore no relationship to the amount of cigarette tobacco smoked. There were as many heavy cigarette smokers among the low-income Europeans as amongst the high (33%), but just more than half (53%) of the middle-income Europeans were heavy cigarette smokers. In the Cape Coloured sample, however, the frequencies with which heavy cigarette smokers were seen increased from 17% in the low-income group to 27% in the medium-income group to 49% among the high-income group.

Cigarette Smoking at Various Ages.—In neither the European nor the Cape Coloured samples was there any significant effect of age on whether they were heavy, medium, light, or non-smokers among the three decades of ages from 25 to 55.

Occupation and the Cigarette-smoking Habit.—Subdivision of the nature of the occupation into five different grades of physical activity ranging from sedentary occupations through intermediate grades to heavy manual labour failed to reveal that the majority of the heavy cigarette smokers belonged to sedentary or light-activity occupational classes. In fact, there were more heavy cigarette smokers amongst the occupational

classes requiring higher grades of physical activity, but the differences seen were not significant on chi-square analysis ($0.2 > P > 0.1$).

Body Build and Cigarette Smoking.—There is no relationship between height or weight and the amount of cigarettes smoked. The distribution of obesity as measured by the sum of the arm (triceps) and scapula skin folds recorded by the use of callipers standardized at 10 g. per square mm. is shown in Fig. 4. A marked difference in the distribution between European and Cape Coloured samples can be seen, but no differences exist between heavy and non-cigarette smokers in either racial group ($0.8 > P > 0.7$ for Europeans; $0.7 > P > 0.5$ for Cape Coloureds).

Blood-pressure and the Cigarette-smoking Habit.—On each individual casual (non-basal) blood-pressure readings were taken; each reading being recorded on first contact with the examining physician. No significant differences at given age existed between the mean blood-pressure levels corrected for arm circumference of the European sample and those of the Cape Coloured sample. In order to increase the sample size these two racial groups were analysed together by the use of regression of blood-pressure (Y) on age (X). This regression was linear over this age range for both systolic and diastolic pressures (see Fig. 5). Neither

FIG. 5.—No significant differences can be seen in the casual blood-pressure levels of smokers and non-smokers throughout the age range studied.

in the steepness of the regression slopes nor in the intercepts could significant differences be found between the heavy smokers and the non-smokers. In fact, throughout the age range the mean blood-pressure of heavy smokers was lower than that of the non-smokers.

Serum Cholesterol Levels.—Comparison was made on each race separately at two age ranges, 25–39 years and 40–55 years. The heavy cigarette smokers in the younger age range had the higher mean serum cholesterol levels. In the 40–55 year range the reverse trend was seen. A consistent trend, however, in both age-groups and in both Cape Coloured and European samples was the distribution of cholesterol between the alpha- and beta-lipoprotein fractions. The percentage of cholesterol in the beta fraction is always higher in the cigarette smokers, and the amount of cholesterol (in mg./100 ml.) in the alpha fraction is consistently lower, giving a higher $\beta:\alpha$ ratio (see Fig. 6). This applies to comparisons between heavy smokers and non-smokers of the same age, the same race, and even the same income group (last-mentioned not shown).

Dietary Fat Intake and the Smoking Habit.—Owing to the consistency of the difference between the serum cholesterol changes, attention was then paid to the dietary-fat intakes of heavy cigarette smokers and non-smokers. Running concurrently with the lower

alpha-lipoprotein cholesterol levels of the heavy smokers are higher intakes of fat—in particular that from animal sources—and this trend is seen consistently when comparisons are made between groups on the same economic plane at given age and within the same race (see Fig. 7). Owing to wide variation, however, the differences between means are not statistically significant on analysis of single group pairs.

Discussion

Epidemiological studies have considerable limitations, but could provide a lead for further study under controlled conditions. The 1955 mortality rate from ischaemic heart disease for men aged 55–59 in the Cape Coloured community was 273 per 100,000, while the figure for the White (European) was 628 per 100,000 (Director of Census and Statistics, personal communication, 1959), yet in this study the smoking habits of these two racial groups are essentially similar. Heavy cigarette smokers were, in fact, more commonly seen among the high-income Cape Coloured than among either the low-income or the high-income Europeans.

FIG. 6.—In young men the serum cholesterol levels are higher in smokers than in non-smokers, but a reverse trend is seen in middle-aged men. The distribution of cholesterol between the beta and alpha lipoprotein fractions shows a consistent difference, however. The smokers in each racial group and in each age range show a higher beta:alpha cholesterol ratio than the non-smokers.

FIG. 7.—A semi-quantitative estimate of the dietary fat intake reveals that smokers in each race and in each age range consume more fat than the non-smokers.

Despite the relatively small sample-size in this survey the findings agree with those of other similar surveys (Gofman *et al.*, 1955; Thomas, 1958; Karvonen *et al.*, 1959). In the rural areas of Finland (Karvonen *et al.*, 1959) the non-smokers tended to be fatter as measured by the arm and scapula skin fold, but in Helsinki there were no differences in body fatness between smokers and non-smokers. Likewise the arterial blood-pressure levels were higher in the non-smokers. This finding is somewhat surprising in view of the immediate effects of smoking a cigarette on the blood-pressure (Burn, 1957) and the evidence that the adrenaline output in man may be increased during smoking (Watts and Bragg, 1956). These findings do not exclude the possibility that the increased susceptibility to ischaemic heart disease in the heavy cigarette smokers could arise from the blood-pressure changes during smoking, as it is well recognized that elevation of the blood-pressure accentuates the severity of atheroma. In the rabbit it has been shown that for this effect not much of a rise in pressure is necessary (Bronte-Stewart and Heptinstall, 1954), nor does it appear that the elevation need be long sustained, as daily alterations in the rabbit's posture with resulting variations in arterial pressure led to marked accentuation of atheroma (Wilens, 1943).

On the other hand, the mechanism for the increased susceptibility of the smokers could operate via the changes in serum lipid found. Apart from higher serum cholesterol levels, patients with ischaemic heart disease have less cholesterol in the alpha-lipoprotein fraction and more in the beta fraction, and hence a higher $\beta:\alpha$ ratio than age-matched controls (Barr, 1953; Nikkilä, 1953; Oliver and Boyd, 1955). In the Capetown survey the heavy cigarette smokers, irrespective of race, age, and economic status, possessed higher $\beta:\alpha$ serum cholesterol ratios. In California, Gofman *et al.* (1955) have likewise reported that their heavy cigarette smokers in the 20-39 age-group had higher serum cholesterol values than non-smokers but not in the 40-59 age-group. In all age-groups, however, the heavy cigarette smokers had the type of ultracentrifugally determined lipoprotein pattern resembling that found in patients with ischaemic heart disease.

In the Finnish survey (Karvonen *et al.*, 1959) higher serum cholesterol values were found in the heavy smokers in all age-groups and at all areas with the exception of the 50-59 age-group in the rural areas. The difference, unlike other studies, appeared to increase with age, and those who smoked the most tended to have the highest serum cholesterol levels. Thomas (1958) has also reported a statistically significant association between smoking and elevated serum cholesterol levels in American medical students. It would appear, then, that in all surveys to date a consistent difference that was found on comparing heavy cigarette smokers and non-smokers appears to lie in the serum lipid levels, whether these individuals have been compared in California, Finland, Baltimore, or Capetown.

The known influence of the diet on the level of cholesterol in the serum led to the analysis of the dietary fat intakes of these individuals. There are no publications that exhibit differing food preferences of individuals differing in their smoking habit, but there is a widely held impression that heavy smokers prefer spicy and savoury food and that with abstinence from smoking not only an increase in appetite follows but the perception of odours, tastes, and flavours becomes

more acute. It is known, for example, that men who stop smoking tend to become fatter (Brožek and Keys, 1957). The data on diet in this survey, however, could only be regarded as semi-quantitative; the information obtained by recall with quantification being based on the average helping method. As the interrogator had no prior knowledge of the manner of subsequent subdivision of the sample, it was unlikely that any bias was exerted toward any particular group. There was, however, sufficient to indicate further study on taste thresholds and food preferences of smokers and non-smokers (Bronte-Stewart, 1956). This had led to the findings reported by Krut *et al.* (1961) and Perrin *et al.* (1961).

Summary

The evidence for and against a relationship between cigarette smoking and mortality from ischaemic heart disease is emphasized and discussed.

Epidemiologically there is some relationship between susceptibility to ischaemic heart disease and the cigarette-smoking habit, but it is pointed out that the relationship need not be causal.

There is little evidence to suggest that cigarette smoking has a direct effect on the coronary circulation or blood coagulability, but the immediate effects of smoking a cigarette can be seen by a rise in pulse rate and a rise in both systolic and diastolic arterial pressures.

On the hypothesis that the cigarette-smoking habit and susceptibility to ischaemic heart disease may be merely related indirectly, in that both would be related to a third and common factor, an analysis was conducted on the multiracial community resident in Capetown.

No relationship could be found between age, income, nature of the occupation, height, weight, or degree of obesity as measured by the skin-fold thickness. The arterial pressures of heavy smokers tended to be slightly lower than those of non-smokers, but the differences were not significant. A highly consistent difference shown was the distribution of cholesterol between the alpha- and beta-lipoprotein fractions. This held true at each age range, for each race, and for each income subgroup. Such differences were paralleled by differences in the customary dietary fat intake, but statistically these differences were not significant.

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REFERENCES

- Anderson, J. T., and Keys, A. (1956). *Clin. Chem.*, **2**, 145.
- Bargeron, L. M., jun., Ehmke, D., Gonlubol, F., Castellanos, A., Siegel, A., and Bing, R. J. (1957). *Circulation*, **15**, 251.
- Barr, D. P. (1953). *Ibid.*, **8**, 641.
- Berkson, J. (1955). *Proc. Mayo Clin.*, **30**, 319.
- Blackburn, H., jun., Orma, E., Härtel, G., and Punsar, S. (1959). *Amer. J. med. Sci.*, **238**, 448.
- Brit. med. J.*, 1955, **1**, 91.
- 1956, **2**, 90.
- Bronte-Stewart, B. (1956). *Brit. med. J.*, **2**, 659.
- and Heptinstall, R. H. (1954). *J. Path. Bact.*, **68**, 407.
- Keys, A., and Brock, J. F. (1955). *Lancet*, **2**, 1103.

- Brožek, J., and Keys, A. (1957). *Science*, **125**, 1203.
 Burn, J. H. (1957). *Proc. roy. Soc. Med.*, **50**, 500.
 Davis, F. W., jun., Scarborough, W. R., Mason, R. E., Singewald, M. L., and Baker, B. M., jun. (1956). *Amer. Heart J.*, **51**, 165.
 Doll, R., and Hill, A. B. (1956). *Brit. med. J.*, **2**, 1071.
 Gofman, J. W., Lindgren, F. T., Strisower, B., DeLalla, O., Glazier, F., and Tamplin, A. (1955). *Geriatrics*, **10**, 349.
 Hammond, E. C., and Horn, D. (1954). *J. Amer. med. Ass.*, **155**, 1316.
 Karvonen, M., Orma, E., Keys, A., Fidanza, F., and Brožek, J. (1959). *Lancet*, **1**, 492.
 Krut, L. H., Perrin, Monica J., and Bronte-Stewart, B. (1961). *Brit. med. J.*, **1**, 384.
Lancet, 1957, **1**, 1348.
 Nikkilä, E. (1953). *Scand. J. clin. Lab. Invest.*, **5**, Suppl. 8.
 Oliver, M. F., and Boyd, G. S. (1955). *Brit. Heart J.*, **17**, 299.
 Perrin, Monica J., Krut, L. H., and Bronte-Stewart, B. (1961). *Brit. med. J.*, **1**, 387.
 Pickering, G. W., and Sanderson, P. H. (1945). *Clin. Sci.*, **5**, 275.
 Registrar-General (1954). *Decennial Supplement, England and Wales, 1951. Occupational Mortality*, Pt. 1, p. 13. H.M.S.O., London.
 Russek, H. I., Zohman, B. L., and Dorset, V. J. (1955). *J. Amer. med. Ass.*, **157**, 563.
 Strøm, A., and Jensen, R. A. (1951). *Lancet*, **1**, 126.
 Thomas, C. B. (1958). *J. chron. Dis.*, **7**, 198.
 Todd, G. F. (1957). "Statistics of Smoking," Research Papers No. 1. Tobacco Manufacturers' Standing Committee, London.
 Watts, D. T., and Bragg, A. D. (1956). *J. appl. Physiol.*, **9**, 275.
 Wilens, S. L. (1943). *Amer. J. Path.*, **19**, 293.

TASTE PERCEPTION IN SMOKERS AND NON-SMOKERS

BY

L. H. KRUT, M.B., Ch.B.

Research Bursar

MONICA J. PERRIN, B.Sc., Dip.Diet.

Research Dietitian

AND

B. BRONTE-STEWART, M.D., M.R.C.P.

Physician and Senior Lecturer

(From the Department of Medicine, University of
Capetown, and Groote Schuur Hospital, Capetown,
South Africa)

Taste is a complex sense. In addition to sensations arising in taste receptors, it is influenced by chemical, tactile, warm, and cold receptors in the mouth, and in particular by olfactory sensations (Houssay, 1955). It is presumably the complexity of the taste sense that has limited its objective study. The subject is, however, simplified by the generally held view that there are but four primary taste modalities: sweet, sour, salt, and bitter (Houssay, 1955; Best and Taylor, 1955; Fulton, 1955; Bell, Davidson, and Scarborough, 1959), and that odour plays no part in the recognition of these pure tastes.

Smoking is popularly held to decrease taste perception. This is attested by almost everyone subject to the habit, and particularly those who have mastered their addiction to it. There are, however, few objective studies on the effects of smoking on taste.

Bronte-Stewart (1956) discussed the relationship between smoking and ischaemic heart disease, and put forward the hypothesis that smoking could affect food preferences via the taste mechanism. To test this hypothesis, taste thresholds for sweet, sour, salt, and bitter in groups of smokers and non-smokers were determined and compared. Taste thresholds were similarly determined immediately before and immediately after the

smoking of a cigarette in a sample of the above groups. In view of the findings, the relationship between the ability to taste phenylthiocarbamide (P.T.C.) and smoking was also examined.

Material and Methods

Two groups of subjects from different walks of life were tested in an identical manner a few weeks apart: (1) 80 medical students, 40 of whom were smokers—the mean age of this group was 20.5 (± 3.1) years; and (2) 76 employees of a local insurance company, 39 of whom smoked, having a mean age of 27.6 (± 9.3 years). Thus a total of 79 smokers and 77 non-smokers were tested. Tests were carried out over several days between 9.30 and 11 a.m. on each of these groups.

Test Procedure.—The subjects were asked to complete a questionnaire giving details of their age, sex, and smoking habits. They were informed of the aim of the experiment and of the taste modalities to be tested. They were told that there would be no fixed order in the substances to be tasted and that tap-water would be used from time to time. The observer recording the thresholds was kept ignorant of the smoking habits of the subjects. These tests were thus done in a "blind" manner. It was arranged that there were approximately equal numbers of smokers and non-smokers having threshold determinations on each day. Stepwise dilutions of sucrose (20–0.15%), citric acid (5–0.15%), sodium chloride (5–0.15%), and quinine hydrochloride (0.1–0.006%) were made up into solutions with tap-water. Each step in the titre series was a multiple of 0.5 of the preceding step. A single drop of a solution was deposited on to the centre of the protruded tongue by means of a pipette. The subject then withdrew his tongue, tasted the solution at leisure, and swallowed the drop. The threshold for each substance was determined in turn by depositing increasing concentrations of each solution, beginning with the most dilute. When two consecutive correct answers were given, the lower concentration was recorded as the threshold in each case. Mouths were rinsed with tap-water after each taste was identified. In this manner it was possible to determine the mean threshold value for each substance in each group of subjects.

Immediate Effects of Smoking.—Sixty of the above individuals, including 29 smokers, were subjected to a second series of tests. Initial threshold determinations were made as above, and the subject then went out for 10 minutes. The smokers all smoked a cigarette, while the non-smokers waited for a similar period until recalled. Each subject was then retested and thresholds were again determined.

Phenylthiocarbamide (P.T.C.).—Titres (0.13–0.00025%) were made up into solution in tap-water. Each step in the titre series was again a multiple of 0.5 of the preceding step. These threshold values were determined in the same manner as those above in 75 subjects, of whom 32 were smokers, drawn from both the student and the insurance groups.

Results

Thresholds for the Four Primary Taste Modalities.—The student group showed no significant differences between smokers and non-smokers in the mean taste thresholds for sweet, sour, or salt. The mean threshold for bitter, however, was significantly higher ($P < 0.001$) in the smokers than in the non-smokers. These findings