VI

Tobacco

Well before 1492 the use of tobacco and its effects were known to the original inhabitants of what was to become the Americas. Europeans first made its acquaintance during Columbus' first voyage of discovery when two of his sailors observed natives in Cuba smoking the leaf.[1] Columbus was offered a bunch of the leaves, which were subsequently brought back to Europe. By the middle of the sixteenth century tobacco was being grown in Western Europe and had become generally available in countries fronting the Atlantic Ocean. Consumption increased rapidly from the mid-sixteenth century onwards.[2] Its initial introduction into England is traditionally attributed to Sir Walter Raleigh, although the story of his alarmed servant extinguishing the fire in his master's pipe with a bucket of water is probably apocryphal. As with sugar, mercantilist restrictions on trade with foreign countries delayed importation of tobacco into England on any scale until after the 1607 establishment of the Virginia colony, where plantations were developed using West African slave labour. Its popularity was enhanced initially because it was thought by some contemporary medical writers to have medicinal value, for chest diseases in particular.[3] These beliefs were countered to some extent by the classical 1604 *Counterblaste to tobacco* by King James I. Concerned with the physical and mental well-being of his subjects, he denounced smoking as "a custom loathsome to the eye, hateful to the nose, harmful to the brain, dangerous to the lungs".[4] His criticisms were remarkably prescient and today they would be regarded as eminently justified. However, attempts at prohibition having proved ineffective, English governments turned to the obvious alternative. They taxed all tobacco imports at the port of landing. Growing tobacco at home was prohibited and existing plants uprooted in order that revenue from import duties would not be foregone.[5] Yearly government statistics relating to imports are therefore available and reflect consumption. The economic disruptions produced by the Civil War temporarily reduced the availability of tobacco, but after the monarchial restoration of 1660 its importation, distribution and consumption rose very rapidly,[6] notwithstanding the royal censure by the now long dead James I.

Despite some marked year to year fluctuations, the documented net tobacco imports (total imports less re-exports) rose dramatically, from 25,000 lbs in 1603 to

[1] Jordan Goodman, *Tobacco in history*, London, Routledge, 1993, p. 37.
[2] Ibid., pp. 37, 59.
[3] Ibid., pp. 44, 61.
[4] Quoted in David Harley, 'The beginnings of the tobacco controversy: puritanism, James I, and the royal physicians', *Bull Hist Med*, 1993, **67**: 28–50, p. 43.
[5] Stanley Gray and V J Wykott, 'Tobacco trade in the eighteenth century', *Southern Econ J*, 1940–41, **7**: 1–26, p. 15.
[6] Ibid., pp. 19, 20.

over 18,000,000 lbs towards the end of the century.[7] If anything, the official figures are underestimates because smuggling was rampant.[8] By 1670, 25 per cent of the population was smoking at least one pipeful a day, annual consumption per head having risen from 0.01 lbs in the decade 1620–1629 to 2.30 lbs in the period 1698–1702 and then tended to decline slightly, to within the range of 1.56 to 2.00 lbs during the years 1738–52.[9]

Two major problems need to be addressed before possibly accepting the introduction and increasing use of tobacco as a factor that contributed to the initial emergence and subsequent rise in incidence of angina pectoris during the late eighteenth century. Firstly, tobacco was sometimes chewed and, during the reign of Queen Anne, taking it in the form of snuff became quite widespread.[10] Secondly, the twentieth-century association of coronary heart disease with tobacco smoking is associated almost exclusively with the use of cigarettes, whereas pipes alone were used in the eighteenth century. Pipe smoke is inhaled at a lower temperature than that of cigarettes and is alkaline, in contrast to cigarette smoke, which is acidic; the chemical content of the two forms of smoke therefore differ somewhat. Pipe tobacco products are absorbed more readily from the mouth into the bloodstream and the need to draw the smoke into the lungs is less. This could account for differences in localization of their carcinogenic effects. As a cause of death, the ratio of mouth to lung cancers is greater among pipe than among cigarette smokers.[11] Either way, however, nicotine is absorbed and pipe smokers obtain the same mildly stimulating, calming and euphoric effects that cigarette smokers sense. Because of this, pipe smokers might be expected to suffer the same undesirable systemic cardiovascular effects of nicotine as cigarette smokers, notably the sympathetic nervous stimulation and epinephrine release with a rise in heart rate and blood pressure.[12] Pipe smokers also absorb carbon monoxide with its potential for damaging vascular endothelium and inducing hypoxaemia. Noel Hickey and his co-workers found a mean carboxyhaemoglobin level of 3.2 per cent among thirteen pipe smokers, somewhat less than the 4.3 per cent in seventy-nine cigarette smokers who were comparable with regards to the extent of their tobacco consumption, but significantly greater than the 0.56 per cent average among non-smokers.[13]

The differences between the characteristics and the effects of pipe and cigarette smoke are counterbalanced to some extent by their similarities once absorbed into the bloodstream, so that reasons for any differing systemic effects on cardiovascular disease incidence are not readily apparent. Nevertheless, pipe smoking has been

[7] Ibid., pp. 18–23.
[8] Goodman, op. cit., note 1 above, p. 60.
[9] Ibid., pp. 60, 72.
[10] George Macaulay Trevelyan, *English social history*, 3rd ed., London, Longmans, Green, 1947, p. 315.
[11] E Cuyler Hammond, 'Smoking in relation to death rates of one million men and women', *Nat Cancer Inst Monogr*, 1966, **19**: 127–204, p. 151, 158.
[12] Joel G Hardman and Lee E Limbird (eds), *Goodman & Gilman's The pharmacological basis of therapeutics*, 9th ed., New York, McGraw-Hill, 1996, p. 192.
[13] Noel Hickey *et al.*, 'Cigar and pipe smoking related to four year survival of coronary patients', *Br Heart J*, 1983, **49**: 423–6, p. 425.

Table VI.1
Male CHD death rates/100,000 person years: non-smokers and pipe smokers (mortality ratios in parentheses)

	Number of Subjects		
Age	45–54	55–64	65–74
Never smoked regularly	150 (1.00)*	542 (1.00)*	1,400 (1.00)*
Current pipe smoker	141 (0.94)	647 (1.19)	1,396 (1.00)

* By definition.

Source: E Cuyler Hammond, 'Smoking in relation to death rates of one million men and women', *Nat Cancer Inst Monogr*, 1966, **19**: 127–204, p. 145.

implicated only inconsistently as a risk factor for coronary heart disease. Epidemiological studies relating tobacco consumption to any disease, cardiovascular or otherwise, are dependent very largely on information provided by the subjects themselves with all its attendant limitations. More importantly, people who have never smoked anything but a pipe are nowadays few in numbers, so that meaningful statistical studies become difficult and pipe smokers are often grouped together with cigar smokers in epidemiological investigations. Hickey and his colleagues observed the impact of smoking a pipe on the four-year survival of patients who had suffered either a myocardial infarction or an episode of unstable angina prior to entry into the study. During the follow-up 12.3 per cent of the pipe smokers suffered a CHD death, in contrast to 9.4 per cent of the non-smokers. With small and unbalanced numbers, 28 and 299 respectively, the differences show a trend but fail to reach statistical significance.[14] In an extensive study, E Cuyler Hammond was able to incorporate sufficient numbers of exclusive pipe smokers for overall comparison with non-smokers and for subgroup analyses. When compared to men who had never smoked regularly, no statistically significant differences in CHD mortality could be found (Table VI.1). However, in the combination of negative results with adequacy of numbers, this investigation is unique.[15] In contrast, a study involving British civil servants showed that men who were exclusively pipe smokers had a 40 per cent higher coronary heart disease mortality than did "never smokers". Although the pipe smokers numbered only 492 out of a sample of 19,018, these results were statistically significant.[16]

Exposure to secondary smoke was frequent in the eighteenth century. Ventilation of many homes and buildings was poor. Early in the eighteenth century, a tax based on the number of windows in a house had been imposed and as a result many windows were bricked up. Because coal fires heated only adjacent parts of the rooms, the need to keep the remaining windows closed in winter was often compelling. As

[14] Ibid., p. 424.
[15] Hammond, op. cit, note 11 above, p. 145.
[16] Yoav Ben-Shlomo *et al.*, 'What determines mortality risk in male former cigarette smokers?', *Am J Public Health*, 1994, **84**: 1235–42, p. 1237.

Table VI.2
Exercise duration to angina. Effect of exposure to tobacco smoke. Patients serving as their own controls

Room status	Exposure status	Time to angina (seconds)	P value
Well ventilated	Smoke free	232.3 ± 68.4″	<0.001
	Smoke exposure	181.1 ± 52.4″	
Unventilated	Smoke free	233.7 ± 64.8″	<0.001
	Smoke exposure	145.8 ± 36.9″	

Adapted from data published by W S Aronow, 'Effect of passive smoking on angina patients', in *N Engl J Med*, 1978, **299**: 21–4, p. 22.

a consequence air exchange and clearance of atmospheric tobacco smoke were presumably reduced. Exposure to environmental tobacco smoke was also a hazard in places such as coffee and ale houses where men gathered socially.

The differences in characteristics of pipe and cigarette smoke are lessened appreciably when they are inspired secondarily. The two forms are then at similar temperatures and inhaled in the same way. The harmful effects of secondary smoking are now well attested. A Judson Wells noted that the smoke-laden atmosphere in an indoor environment could contain as much as 50 parts per million of carbon monoxide, and commonly result in blood carboxyhaemoglobin concentrations of 2 to 4 per cent.[17] Wilbert S Aronow exercise tested non-smoking patients with stable angina pectoris in a room which was smoke free one time but on a second occasion contained the exhalations of three healthy volunteers, each of whom had smoked five cigarettes during the two hours before the exercising patients began their tests. Aronow found that when the subjects were stress tested in a smoke filled atmosphere they developed angina earlier in the course of the exercise and at a lower heart rate-blood pressure product. The effect of the smoky environment was greater when the room was kept unventilated (Table VI.2).[18]

A United States survey by Dale P Sandler and his co-workers showed that, when compared to male non-smokers living in a smoke-free home, the relative risk of coronary heart disease among non-smoking men living in a smoke-laden home atmosphere was significantly increased, with a risk ratio of 1.31 (CL 1.05-1.64). The results were obtained after adjustment for age, marital status, education and quality of housing.[19] Findings in the Boston Nurses Survey also confirmed an association between secondary smoking and coronary heart disease, both in its incidence and in the mortality. In a ten-year follow-up of 32,046 women aged thirty-two to

[17] A Judson Wells, 'Passive smoking as a cause of heart disease', *J Am Coll Cardiol*, 1994, **24**: 546–54, p. 549.
[18] W S Aronow, 'Effect of passive smoking on angina pectoris patients', *N Engl JMed*, 1978, **299**: 21–4, p. 22.
[19] Dale P Sandler *et al.*, 'Deaths from all causes in non-smokers who lived with smokers', *Am J Public Health*, 1989, **79**: 163–7, p. 165.

Table VI.3
Relative risks associating ischaemic heart disease morbidity and mortality with passive cigarette smoking. Meta-analysis results.
(95% confidence intervals in parentheses)

	Sex	No. of studies	Relative risk*
Morbidity	Women	6	1.51 (1.16–1.97)
	Men	4	1.28 (0.91–1.81)
Mortality	Women	8	1.23 (1.11–1.36)
	Men	5	1.25 (1.03–1.51)

* Relative risk with no exposure 1.0.

From A Judson Wells, 'Passive smoking as a cause of heart disease', reprinted with permission from the American College of Cardiology, *J Am Coll Cardiol*, 1994, **24**: 546–54, pp. 549–50.

sixty-one at entry into the study, the relative risk of contracting any form of CHD was 1.91 (CL 1.113.28) among non-smokers who were regularly exposed either at home or at work. The differences remained significant after adjustment for other traditional risk factors. There was a dose-response gradient between frequency of self-reported passive smoking and cardiovascular risk.[20] A far-reaching review by Wells combined Sandler's study with seven others in a meta-analysis. This too showed secondary smoking to be associated with a significant increase in the relative risk of coronary heart disease morbidity among women and an increase that approached significance among men (Table VI.3). More than the men, the women in that era were in the smoke laden home atmosphere for longer periods. The greater effect of home exposure to tobacco smoke on non-smoking women is thus readily explained. The relative morbidity risk with passive smoking was significantly increased in both sexes.[21] The significantly greater impact of secondary smoking on male mortality as opposed to morbidity may reflect an effect on the severity of the disease as well as on its incidence. There is support from animal studies for this possibility. Karin Przyklenk produced myocardial infarction in dogs by coronary artery ligation. When compared to controls, the myocardial infarcts were larger among the dogs that had been previously exposed to tobacco smoke.[22]

Taking snuff and chewing tobacco are exceedingly rare in the twentieth century and the need to establish their effects on cardiovascular health have been minimal and therefore gone unstudied. Either would spare users the harmful effects of tobacco smoke, but they would still be exposed to the systemic effects of nicotine. The consequences of absorbing nicotine without smoke were shown by Lennart Kaijser and B Berglund who studied the effects of chewing 4 mg pieces of nicotine gum in eight healthy non-smokers aged between twenty-one and fifty-two years. In addition

[20] Ichiro Kawachi *et al.*, 'A prospective study of passive smoking and coronary heart disease', *Circulation*, 1997, **95**: 2374–9, p. 2376.
[21] Wells, op. cit., note 17 above, p. 550.
[22] Karin Przyklenk, 'Nicotine exacerbates postischemic contractile dysfunction of "stunned" myocardium in the canine model. Possible role of free radicals', *Circulation*, 1994, **89**: 1272–81, p. 1277.

to confirming the previously known rise in heart rate and blood pressure, these investigators studied the effects of raising myocardial oxygen demand by pacing the hearts of their subjects at increasing rates, coronary arteriovenous oxygen differences and haemodynamics being recorded throughout. They found that following administration of the gum, myocardial contractility increased and oxygen requirements rose more than would be expected from control observations of a pacemaker induced rise in the rate-pressure product. Additionally, when compared to the control observations, there was a relative increase in coronary circulatory resistance and the coronary arterial blood flow increase with pacing was blunted after chewing of the gum.[23] Because of these haemodynamic effects, smoke-free nicotine usage, as with snuff or chewing tobacco, has the potential to lower the threshold for angina in patients with established coronary heart disease.

In conclusion, there is good historical evidence to indicate that tobacco consumption increased very considerably during the lead time for the eighteenth-century appearance of angina pectoris as a new clinical entity. One suspects that there is some connection between the two and that despite insufficient epidemiological support, grounds exist for incriminating the use of snuff, chewing tobacco and pipe smoking. Recent observations have established that exposure to secondary smoke is a risk factor for coronary heart disease, and this association may be one way in which eighteenth-century pipe smoking and emergence of angina pectoris might have been linked.

[23] Lennart Kaijser and B Berglund, 'Effect of nicotine on coronary blood-flow in man', *Clin Physiol*, 1985, **5**: 541–52, pp. 549–50.