Effect of tobacco smoking on the nervous system / by Sir Humphry Rolleston, Bart., K.C.B., Physician in ordinary to His majesty the King ; Regius Professor of physic in the University of Cambridge.

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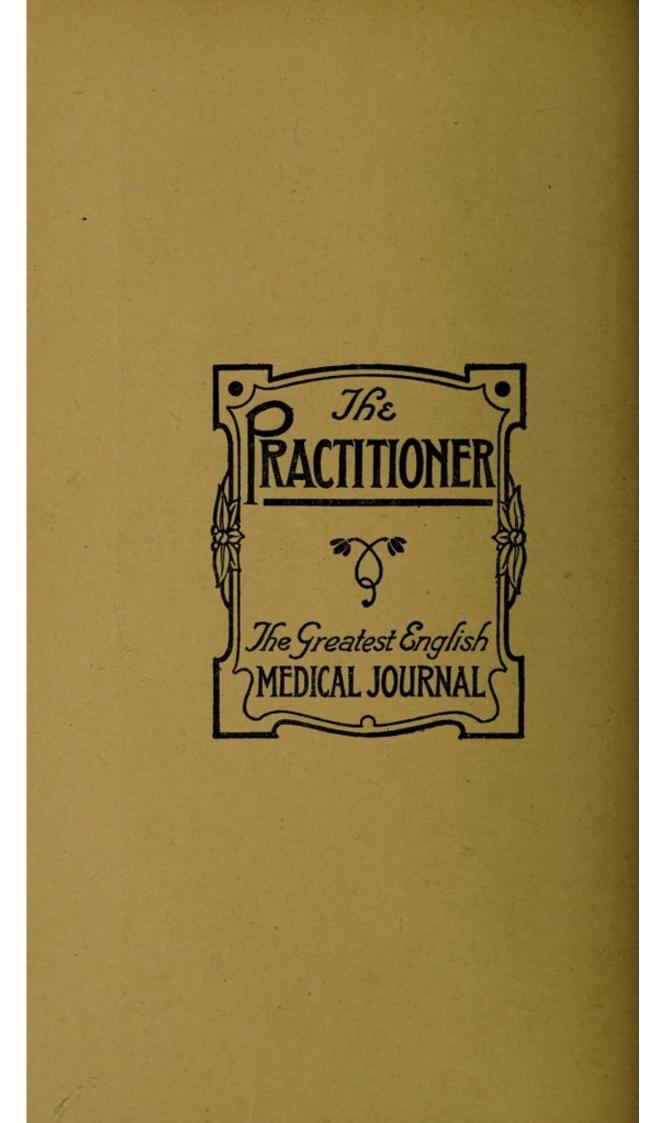
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Reprinted from "The Practitioner" for January, 1927.

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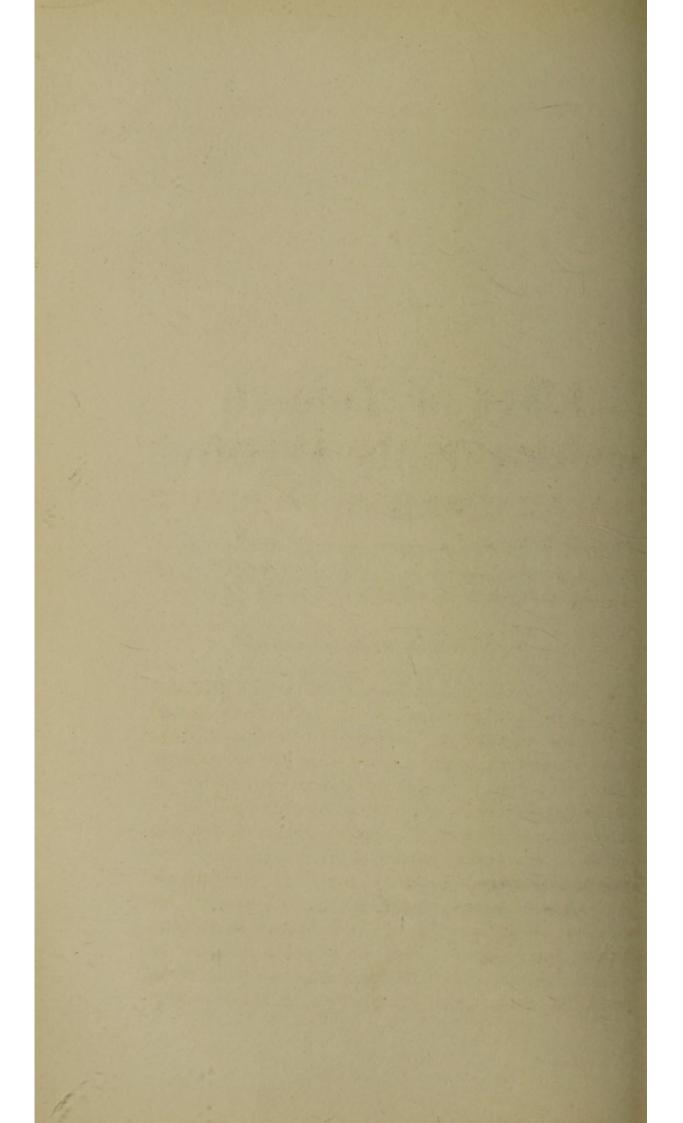


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THE relation of tobacco-smoking to squamouscelled carcinoma of the lip is indirect; though the responsibility of oft-repeated irritation of the muco-cutaneous margin of the lip by the hot stem of a short clay pipe in causing carcinoma has now lost its former importance, as clay pipes have dropped out of fashion, the interest of the sequence remains, for it is similar to the kangri cancer of Kashmir, due to the irritation produced by the kangri, an earthenware bowl heated by wood charcoal, on the skin of the abdomen and thighs (Neve¹). In Japan kairo burn cancer, due to heat alone, the possible influence of volatile substances derived from charcoal being eliminated, has been described (Itoh²). In those so predisposed—and syphilis may, as in carcinoma of the tongue, be such a factor—the irritation of cigarettes smoked to the bitter end should not be neglected as a possible factor in the causation of cancer of the lip.

The hot smoke of certain tobaccos, and here personal idiosyncrasies play their part, produces in some persons soreness of the tongue, and reflexly increased salivation. The smoker's patch of leukoplakia is well known, but syphilis is a powerful auxiliary, for among Fournier's ³ 324 cases of leukoplakia 80 per cent. were syphilitic, though other estimates of the luetic factor are lower, down to 50 per cent.; smoking, however, is undoubtedly a very constant antecedent in leukoplakia, for among Fox's ⁴ 40 cases of leukoplakia, 2 only, both women, were non-smokers. Cases going on to carcinoma are probably complicated by syphilitic disposition.

Smoking does not favour dental caries; in fact, it has been thought to have some bactericidal effect and so may tend to prevent it; but in excess it may set up gingivitis, and in those prone to much deposition of "tartar" it helps to cause oral sepsis. Small doses of nicotine, by their action on the nervous mechanism of the salivary glands, increase the secretion of saliva, whereas large doses have the opposite effect, thus accounting for "a mouth" the morning after a strong cigar. In the presence of oral sepsis increased salivation may wash down more micro-organisms into the stomach and so cause gastritis.

The irritating effects of furfurol, pyridine, and ammonia, which are said to be more especially due to cigarette-smoking, on the pharyngeal mucous membrane may lead to air-swallowing and so to flatulence; according to Huchard, tobacco-dyspepsia may cause anginoid pains (gastro-tabacosis angina). On the other hand, the presence of a pipe held between the teeth renders swallowing very difficult and thus indirectly diminishes the liability to aerophagy, and so, if apology

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be needed, may justify smoking, though not of cigars or cigarettes, after a meal.

As nicotine first inhibits and later increases the contraction of smooth muscular fibres, the question arises whether or not the unstriated muscle of the œsophagus is affected by smoking, and whether or not the condition variously known as idiopathic dilatation of the œsophagus, cardiospasm, or achalazia, is in any case really due to smoking. This possibility is seldom if ever mentioned; but has it been seriously considered? The universality of smoking and the rarity of œsophageal symptoms have probably been regarded as a sufficiently strong negative reply.

The late Dr. Walter Verdon,⁵ who told me that he suffered from angina pectoris and was the "Dr. X" of the book he wrote to prove that the seat of the primary cause of angina was in the muscle of the œsophagus and stomach, mentioned œsophageal spasm and hypertonus in this connection; but he did not consider that moderate smoking of mild tobacco was a cause of angina pectoris, and himself continued to smoke.

Hiccup, whatever the mechanism may be, irritation of the diaphragm due to gastric distension or reflex irritation through the vagus and phrenic, is sometimes excited by smoking, and it may be worth while to mention the often effective, if very simple, method of cure advocated by the late Sir Lauder Brunton, of sipping water so that each drop is swallowed separately, which he considered acted by temporarily paralysing the vagus.

The effect of smoking in staving off the discomfort of hunger, which depends on gastric peristalsis, is probably due to the action of nicotine in inhibiting the movements of the stomach for an hour or so after taking food, as has been shown by skiagraphy (Daniélopolu⁶), and it is quite possible that dilatation of the stomach in heavy smokers may be thus explained. It is said that sulphocyanide of potassium or of sodium is increased in the saliva of smokers and inhibits protein digestion, and that nicotine diminishes the secretion of pepsin and rennin.

Although peristalsis of the body of the stomach is inhibited, the pylorus may be spasmodically contracted by excessive smoking, especially of cigars, and so hyperchlorhydria results, with symptoms simulating duodenal ulcer, which indeed it thus disposes to. This is more prone to occur in men about fifty years of age with diminished tolerance to tobacco. Excessive smoking is thus given as one of the causes of nervous dyspepsia, and no doubt the relative sensitiveness of the individual is an important factor in determining whether or not a heavy smoker will pay the penalty of gastric symptoms.

Ryle,⁷ who finds that genuine tobacco-dyspepsia is rare, points out that it differs from other toxic and infective dyspepsias in being pressor instead of depressor, for, as it is due to sympathetic inhibition, it allows exaggerated vagal action. According to Hurst,⁸ excessive smoking, by increasing hyperchlorhydria and hypertonus of the stomach with more rapid expulsion of food after a meal into the duodenum, especially in persons with a hypersthenic stomach, favours the production of duodenal ulcer, and he ascribes the preeminence of the medical over the other professions, as regards the incidence of duodenal ulcer, to irregular meals and excessive smoking.

The acute gastric symptoms of nausea, vomiting and collapse in tyros in tobacco-smoking are accompanied by diarrhœa due to increased peristalsis. As tobacco may cause headache and occasionally angina pectoris, presumably from coronary spasm, it is possible that attacks of the angina abdominis, described by Lauder Brunton and Williams ⁹ (1912) and by Parkes Weber ¹⁰ as due to arteriosclerotic mesenteric arteries, may be

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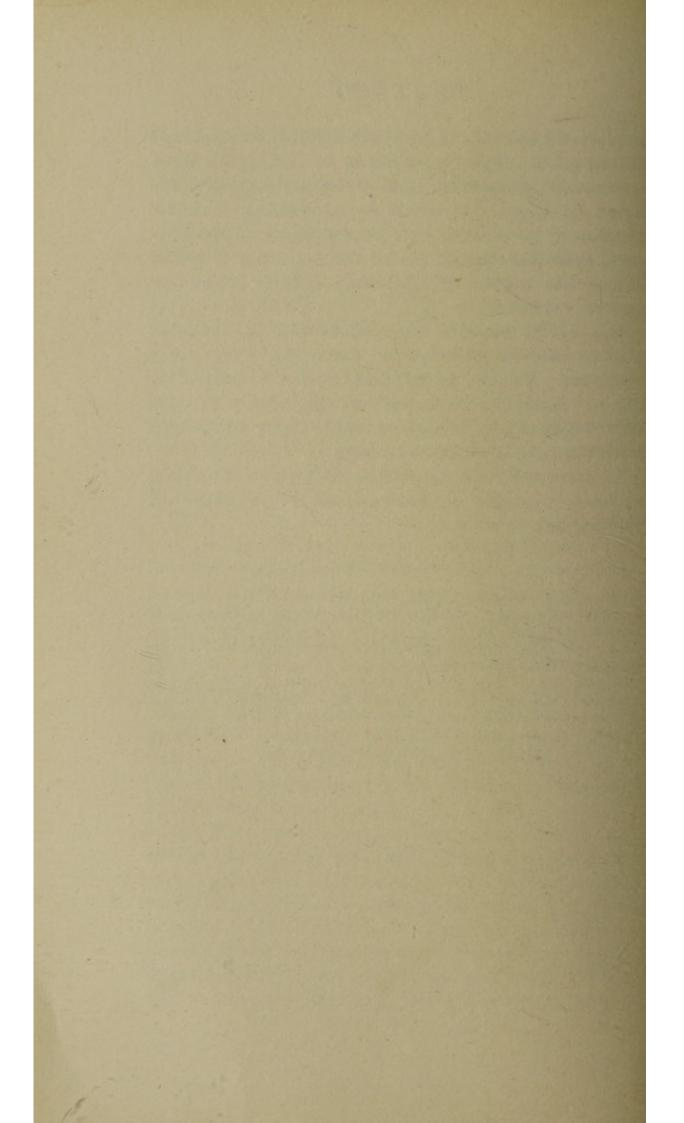
precipitated so to speak by spasm induced by smoking; but if so, it might be difficult to distinguish such angiospastic pain from that of hyperchlorhydria referred to above. It would be interesting to know whether or no nicotine ever causes spasm of the ileocæcal sphincter, but neither of this event nor of spasm of the bile ducts causing biliary colic is there any positive evidence.

The mildly laxative effect of tobacco in seasoned smokers may be offered as an excuse for a pipe after breakfast. On the other hand, spastic constipation, due to spasm of various parts of the colon with colic very resistant to treatment other than abstinence from tobacco, is occasionally seen.

In company with alcoholism and coffee drinking, excessive smoking is incriminated as a cause of pruritus ani.

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