# Chapter 12

## Other Conditions

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# Chapter 12

## RELATIONSHIP OF PEPTIC ULCER TO TOBACCO USE

There are five retrospective studies on the relationship of peptic (gastric and duodenal) ulcer to smoking, in which data have been obtained about the smoking habits of peptic ulcer patients and various kinds of control groups (1, 2, 7, 14, 18). Also, in one cross-sectional study, the frequency of peptic ulcer has been determined in a population of individuals with varying smoking habits (11).

Tables 1 and 2 summarize the methods used and the results of these studies. These studies demonstrate an association between cigarette smoking and peptic ulcer which appears to be greater for gastric than for duodenal ulcers. The proportion of non-smokers is higher among the controls than among the ulcer patients in every one of these studies.

No differences were noted with respect to the frequency of heavy smokers in the study of Doll (7) and no consistent relationship with amount smoked was observed by Trowel1 (18).

In the cross-sectional study of Edwards, et al. (11), a larger proportion of peptic ulcer cases was found among the cigarette smokers, and this proportion increased with amount of cigarette smoking. The heavy cigarette smokers had a frequency of peptic ulcer twice that of those who had never smoked (12 percent as compared to 6 percent).

No association with pipe smoking was noted (1, 11, 14, 18).

In three prospective studies (Table 3) gastric ulcer has been classified separately from duodenal ulcer. The mortality ratios of cigarette smokers from gastric ulcer are high in all three studies (46/0, 5.1 and 4.3). For duodenal ulcers the mortality ratios are more modest (2.2, 2.3 and 1.1). In the remaining four prospective studies only the combined mortality ratios for gastric and duodenal ulcers are available: their results being based on small numbers of deaths, are erratic but their over-all average mortality ratio is about the same as for the three large studies. Consequently, it appears likely that the excess mortality of cigarette smokers from peptic ulcer can be attributed primarily to gastric ulcer. A breakdown by amount smoked (Chapter 8, Table 23) shows no trend. For cigar and pipe smokers the peptic ulcer mortality ratio (total over five studies) is 1.6 but in view of the small number of deaths this elevation is not statistically significant.

Doll, et al., (7) conducted a clinical trial of the effect of stopping smoking on the healing of gastric ulcers. The results were assessed by measuring radiologically the reduction in the size of the ulcer niche. Patients advised to stop smoking had an average 78% reduction in the size of the ulcer, compared to 57% for those who continued to smoke. In view of the probable existence of other factors which may have concomitantly been introduced in the approach to the smokers, and the complex nature of the healing process, it is difficult to interpret this observation.

TABLE 1.—*Summary of methods used in retrospective and cross-sectional studies of peptic ulcer and smoking*

| Investigator and Year | Country | Sex | Cases | | Controls | | Collection of data |
|---|---|---|---|---|---|---|---|
| | | | No. | Method of Selection | No. | Method of Selection | |
| Barnett, (2) 1927 | U.S.A. | M | 66 Gastric; 178 Duodenal. | Patients admitted between 1913 and 1926. Only cases with complete smoking history selected. | 500 | Selected at random from the general admissions–males, aged 20–60. | 1. Retrospective review of records at Peter Bent Brigham Hospital.<br>2. Ulcer diagnosis probably well established. |
| Trowell, (18) 1934 | England | M | 50 Duodenal | Not stated | 400 | Selected at random from wards of a general hospital. | 1. Interviewed by investigator.<br>2. Ulcer diagnosis confirmed by X-ray and/or surgery. |
| Mills, (14) 1950 | U.S.A. | M | 55 | Not stated | 275 | Sample of population in Columbus, Ohio. | No details given. |
| Allibone and Flint, (1) 1958 | England | M&F | 107 | Consecutive admissions to hospital of patients with gastric and duodenal hemorrhage or perforation. | 107 | Matched by age, sex, and time of admission from acute general surgical emergency admissions. | Patients and controls interviewed by same observer. |
| Doll, Jones, and Pygott (7), 1958 | England | M&F | 327 Gastric; 338 Duodenal. | Ulcer patients in Doll and Hill Lung Cancer Study plus additional patients in Central Middlesex Hospital. | 1,143 | Patients with non-ulcer diseases. Each case matched with 2 control patients of same sex, 5-year age group, and same type of place of residence. Male patients matched by social class. | 1. Same interviewers and questionnaire in cases and controls.<br>2. Ulcer diagnosis probably well established. |
| Edwards, McKeown, and Whitfield (11), 1959 | England | M | 1,737 men aged 60 and over on 11 General Practioners' lists were examined and interviewed by these practitioners. Represents about 84% of all such men on these lists. (9% non-response due to death and/or untraced.) | | | | Of 143 considered to have a peptic ulcer, 53 were confirmed by X-ray. |

TABLE 2.—*Summary of results of retrospective and cross-sectional studies of peptic ulcer and smoking*

| Investigator | Percent Non-smokers | | Percent Heavy Smokers or Average Amounts Used | |
|---|---|---|---|---|
| | Cases | Controls | Cases | Controls |
| Barnett (2) | Total 18<br>Gastric 15<br>Duodenal 20 | 25 | | |
| Trowell (18) | Duodenal 8 | 17 | Cigarettes: 12.0 per day<br>Pipe: 1.6 oz. per week | 11.1 per day.<br>2.15 oz. per week. |
| Mills (14) | 18 | 35 | | |
| Allibone and Flint (1) | 38 | 54 | | |
| Doll et al. (7) | *Gastric*<br>M 1.3<br>F 51.1<br>*Duodenal*<br>M 2.1<br>F 53.7 | 4.7<br>66.8<br><br>5.8<br>62.0 | *Gastric*<br>M 10.6<br>F 1.1<br>*Duodenal*<br>M 10.2<br>F 1.9 | <br>11.3<br>1.1<br><br>12.7<br>1.9 |
| Edwards et al. (11) | Percent of Peptic Ulcer by Smoking Category<br>Never smoked 6.0<br>Formerly smoked 6.7<br>Cigarettes:<br>1–9 per day 9.4<br>10–19 per day 9.8<br>20 plus per day 12.0<br>Pipe 6.5<br>Pipe and cigarettes 8.5 | | | |

TABLE 3.—*Expected and observed deaths and mortality ratios for ulcer of stomach and duodenum* among current cigarette smokers, from seven prospective studies*

| Investigator | Type of Ulcer | Number of Deaths | | Mortality Ratio |
|---|---|---|---|---|
| | | Observed | Expected | |
| Hammond and Horn (13)** | Gastric | 46 | 9 | |
| | Duodenal | 54 | 25 | 2.2 |
| | Both types | 100 | 425 | 4.0 |
| Dorn (8)** | Gastric | 31 | 6.1 | 5.1 |
| | Duodenal | 36 | 15.4 | 2.3 |
| | Both types | 67 | 21.5 | 3.1 |
| Hammond (12) | Gastric | 42 | 9.7 | 4.3 |
| | Duodenal | 32 | 28.9 | 1.1 |
| | Both types | 74 | 38.6 | 1.9 |
| Doll and Hill (6) | Both types | 14 | 0 | |
| Dunn et al., Occupational (9) | Both types | 12 | 23.1 | 0.5 |
| Dunn et al., Legion (10) | Both types | 12 | 1.8 | 6.8 |
| Best et al. (5) | Both types | 54 | 7.9 | 6.9 |

*Includes ISC numbers 540, 541.
**The Hammond and Horn data are from their original published report; the other results listed include more recent data as tabulated for the Committee (see Chapter 8).

Numerous investigators have studied the clinical and physiological effects of smoking on gastric motility and acid secretion in humans with and without peptic ulcer. Great variation of gastric motility and secretion was observed in response to cigarette smoking.

Some workers found inhibition of gastric motility (15, 17). Batterman (3) showed three type of response in normal subjects and ulcer patients after smoking one cigarette. In one-third no effect was observed, another third complete inhibition of motor activity for a time, and in the rest a period of hypermotility was followed by normal or subnormal activity, Smoking appears to produce variable effects also on gastric secretion. In a few studies, gastric secretion increased, while in others no change was observed or there was depression of secretory activity (4, 15, 16, 17). Additional studies of the effect of smoking on gastric secretory activity and motility are needed to explain the biological meaning of the statistical association between cigarette smoking and peptic ulcer.

## CONCLUSION

Epidemiological studies indicate an association between cigarette smoking and peptic ulcer which is greater for gastric than for duodenal ulcer.

## REFERENCES


1. Allibone, A., Flint, F. J. Bronchitis, aspirin, smoking and other factors in the etiology of peptic ulcer. Lancet 2: 179-82, 1958.
2. Barnett, C. W. Tobacco smoking as a factor in the production of peptic ulcer and gastric neurosis. Boston Med Surg J 197: 457-9, 1927.
3. Batterman, R. C. The gastro-intestinal tract. In: Wynder, E. L. ed. The Biologic Effects of Tobacco. Boston, 1955. Chapter 5, p. 133-50.
4. Batterman, R. C., Ehrenfeld, 1. The influence of smoking upon the management of the peptic ulcer patient. Gastroenterology 12: 575-85, 1949.
5. Best, E. W. R., Josie, G. H., Walker, C. B. A Canadian study of mortality in relation to smoking habits, a preliminary report. Canad J Pub Health 52: 99-106, 1961.
6. Doll, R., Hill, A. B. Lung cancer and other causes of death in relation to smoking: A second report on the mortality of British doctors. Brit Med J 2: 1071-81. 1956.
7. Doll, R., Jones, F. A., Pygott, F. Effect of smoking on the production and maintenance of gastric and duodenal ulcers. Lancet 1: 657-62, 1958.
8. Dorn, H. F. Tobacco consumption and mortality from cancer and other diseases. Public Health Rep 74: 581-93, 1959.

9. Dunn, J. E., Linden, G., Breslow, L. Lung cancer mortality experience of men in certain occupations in California. Amer J Pub Health 50: 1475-87, 1960.
10. Dunn, J. E., Jr., Buell, P., Breslow, L. California State Department of Public Health, Special Report to the Surgeon General's Advisory Committee on Smoking and Health.
11. Edwards, F., McKeown, T., Whitfield, A. G. W. Association between smoking and disease in men over sixty. Lancet 1: 196-200, 1959.
12. Hammond, E. C. Special report to the Surgeon General's Advisory Committee on Smoking and Health.
13. Hammond, E. C., Horn, D. Smoking and death rates-Report on forty-four months of follow-up of 187,783 men. II. Death rates by cause JAMA 166: 1294-1308, 1958.
14. Mills, C. A. Tobacco smoking: Some hints of its biologic hazards. Ohio Med J 46: 1165-70, 1950.
15. Packard, R. S. Smoking and the alimentary tract: A review. Gut 1: 171-4, 1960.
16. Schnedorf, J. G., Ivy, A. C. The effect of tobacco smoking on the alimentary tract. JAMA 112: 898-903, 1939.
17. Steigmann, F., Dolehide, R. U., Keminski, L. Effects of smoking tobacco on gastric acidity and motility of hospital controls and patients with peptic ulcer. Amer J Gastroent 22: 399-409, 1954.
18. Trowell, O. A. The relation of tobacco smoking to the incidence of chronic duodenal ulcer. Lancet 1: 808-9. 1934.

## TOBACCO AMBLYOPIA

For more than a century clinicians have attributed certain cases of amblyopia--dimness of vision unexplained by an organic lesion--to the use of tobacco.

The distinguishing characteristic of tobacco amblyopia is a specific type of centrocecal scotoma. Since this disease was defined as a distinct clinical entity for the first time in 1930 (4), the medical literature prior to this date is of relatively little value in the critical evaluation of the problem (3). No epidemiological studies with adequate controls are available to establish for this disease a relative risk among smokers and nonsmokers.

Clinical impressions associate tobacco amblyopia with pipe and cigar smoking and very rarely with cigarette smoking.

It has been suggested that this disease, which is now rare in the United States, occurs mainly in individuals with a nutritional deficiency which presumably renders the retina or optic nerve unduly sensitive to tobacco (1,5).

Objective attempts at experimentation have been extremely rare and most of the literature is related to uncontrolled clinical impressions (2).

## CONCLUSION

Tobacco amblyopia had been related to pipe and cigar smoking by clinical impressions. The association has not been substantiated by epidemiological or experimental studies.

## REFERENCES


1. Heaton, J. M., McCormick, A. J. A., Freeman, A. G. Tobacco Amblyopia: A clinical manifestation of vitamin B-12 deficiency. Lancet 2: 286-90, 1958.
2. Potts, A. M. Special report to the Surgeon General's Advisory Committee on Smoking and Health.
3. Schwartz, J. T. Special report to the Surgeon General's Advisory Committee on Smoking and Health.
4. Traquair, H. M. Toxic Amblyopia, including retro-bulbar neuritis. Trans Ophthal Soc U K 50: 351-85, 1930.
5. von Sallmann, L. Special report to the Surgeon General's Advisory Committee on Smoking and Health.


# SMOKING AND CIRRHOSIS OF THE LIVER

Epidemiological studies have noted an association between cigarette smoking and mortality from cirrhosis of the liver. The mean mortality ratio for cirrhosis of the liver calculated from all prospective studies was 2.2 (Table 19, Chapter 8). The individual ratios in six of these studies ranged from 1.3 in the Canadian veterans study (1) to 4.0 in the California occupational study (3). The earliest prospective study, by Doll and Hill (2) reported no deaths from cirrhosis of the liver among non-smokers.

The small amount of information on the biological effects of nicotine and tobacco smoke on the liver of experimental animals is contradictory (5).

In several studies (4, 6, 7) it has been reported that heavy smokers also tend to drink alcoholic liquors excessively. It is well established that heavy consumption of alcohol and nutritional deficiencies are associated with increased mortality from cirrhosis of the liver. The increased death rate from cirrhosis among smokers may reflect the consumption of alcohol and associated nutritional deficiencies rather than the effect of cigarette smoking.

## CONCLUSION

Increased mortality of smokers from cirrhosis of the liver has been shown in the prospective studies. The data are not sufficient to support a direct or causal association.

## REFERENCES

1. Best, E. W. R., Josie, G. H., Walker, C. B. A Canadian study of mortality in relation to smoking habits. a preliminary report. Canad J Pub Health 52: 99-106, 1961.
2. Doll, R., Hill, A. B. Lung cancer and other causes of death in relation to smoking: A second report on the mortality of British doctors. Brit Med J 2: 1071-81, 1956.
3. Dunn, J. E., Linden, G., Breslow, L. Lung cancer mortality experience of men in certain occupations in California. Amer J Pub Health 50: 1475-87, 1960.
4. Heath, C. W. Differences between smokers and non-smokers. AMA Arch Int Med 101: 377, 1958.
5. Larson, P. S., Haag. H. B., Silvette, H. Tobacco-experimental and clinical studies. A comprehensive account of the world literature. Balliere, Tindall & Cox, London, 1961. p. 319-321.
6. Matarazzo, J. D., Saslow, G. Psychological and related characteristics of smokers and non-smokers. Psycho1 Bull 57: 493, 1960.
7. McArthur, C.. Waldron, E., Dickinson, J. The psychology of smoking. J Abnorm Soc Psycho1 56: 267-275, 1958.


# MATERNAL SMOKING AND INFANT BIRTH WEIGHT

Five retrospective and two prospective studies have shown an association between maternal smoking during pregnancy and birth weight of the infant (2, 4, 5, 6, 8, 9, 10) Women smoking during pregnancy have babies of lower birth weight than non-smokers of the same social class. They have also a significantly greater number of premature deliveries (defined as birth weight of 2,500 grams or less) than the non-smoking controls.

While several studies reported a slightly greater neonatal death rate of the children of smokers (2, 5), others did not demonstrate any significant difference in the fetal and neonatal death rates of the two groups (6, 7).

Studies on alterations of placental morphology and function as a response to smoking are insufficient for judgment. The difference in infant weight may be due to vasoconstriction of the placental blood vessels, (1) or to toxic substances such as CO in the circulation of the smoker and fetus (3).

It is not known whether the lower birth weight of the infants of smokers has any clinical significance. In one of the groups studied (5) there was less need for surgical induction of labor among mothers who smoked.

## CONCLUSIONS

1. Women who smoke cigarettes during pregnancy tend to have babies of lower birth weight.
2. Information is lacking on the mechanism by which this decrease in birth weight is produced.
3. It is not known whether this decrease in birth weight has any influence on the biological fitness of the newborn.

## References

1. Essenberg, J. M., Schwind, J., Patras, A. The effects of nicotine and cigarette smoke on pregnant female albino rats and their offsprings. J Lab Clin Med 25: 708-17, 1940.
2. Frazier, T. M., Davis, G. H., Goldstein, H., Goldberg, I. D. Cigarette smoking and prematurity: a prospective study. Amer J Obstet Gynec 81: 988-96, 1961.
3. Haddon, W. Jr., Nesbitt, R. E. Smoking and pregnancy: carbon monoxide in blood during gestation and at term. Obstet Gynec 18: 262-7, 1961.
4. Herriot, A., Billewicz, W. F., Hytten, F. E. Cigarette smoking in pregnancy. Lancet 1: 771-3, 1962.
5. Lowe, C. R. Effect of mother's smoking habits on birth weights of their children. Brit Med J 2: 673-6, 1959.
6. O'Lane, J. M. Some fetal effects of maternal cigarette smoking. Obstet Gynec 22: 181-4, 1963.
7. Savel, L. E., Roth, E. Effects of smoking on fetal growth. Obstet Gynec 20: 313-6, 1962.
8. Simpson, W. J. A preliminary report on cigarette smoking and the incidence of prematurity. Amer J. Obstet Gynec 73: 808-15, 1957.
9. Villumsen, A. L. Cigarette smoking and low birth weights: A preliminary report. Ugeskr Laeg 124: 630-1, 1962.
10. Yerushalmy, J. Statistical considerations and evaluation of epidemiological evidence. In: James, George and Rosenthal, Theodore eds. Tobacco and Health. Springfield, III. Charles C. Thomas, 1962, p. 208-30.


# SMOKING AND ACCIDENTS

Smoking has been associated with a variety of accidents. Among these, fires have the most obvious and important consequences.

In special study of home accident fatalities in 1952 through 1953, the Public Health Service and the National Safety Council reported that 231 (18%) of 1,274 deaths from fires of known origin were due to cigarettes, cigars or pipes (1).

The Metropolitan Life Insurance Company reported that of 352 deaths in 1956 and 1957 among their policyholders from fires and burns with known causes in and about the home, 57 (16%) were due to smoking (2).

Of physiological responses related to driving, smoking degrades detectably only the differential brightness threshold and this effect increases with amount of smoking (4) The epidemiological data available on the effects of smoking on traffic accidents are inconclusive.

It has been shown that a level of carboxyhemoglobin of 5 percent--a level which is not uncommon among heavy cigarette smokers (3, 6)--depresses visual perception to as great an extent as anoxia at 8,000 to 10,000 feet altitude (4, 5).

## Conclusion

Smoking is associated with accidental deaths from fires in the home. No conclusive information is available on the effects of smoking on traffic accidents.

## References


1. Home Accident Fatalities: 1952-1953. National Office of Vital Statistics, U.S. Public Health Service, 1956. Mimeographed report. Table 12.
2. How fatal accidents occur in the home. Metrop Life Insur Statist Bull 40: 6-8. November-December, 1959.
3. Larson, P. S., Haag, H. B., Silvette, H. Tobacco: Experimental and Clinical studies. Baltimore, Williams and Wilkins, 1961. Carboxy hemoglobin, p. 107-110.
4. McFarland. R. A., Moseley, A. L. Carbon monoxide in trucks and buses and information from other areas of research on carbon monoxide, altitude and cigarette smoking. In: Conference proceedings: Health, medical and drug factors in highway safety. National Academy of Sciences-National Research Council Publication 328, 1954. Sect. 4.17-4.33.
5. McFarland, R. A., Roughton, F. J. W., Halperin, M. H., Niven, J. I. The effects of carbon monoxide and altitude on visual thresholds. J Aviat Med 15: 6, 381-94. 1944.
6. Schrenk, H. H. Results of laboratory tests. Determination of concentration of carbon monoxide in blood. Pub Health Bull 278: 36-49, 1942.