

EFFECTS OF SMOKING ON PREGNANCY

SUMMARY

Maternal smoking during pregnancy is associated with decrease in infant birth weight and increased incidence of prematurity, as defined by weight alone, and may be associated with an increased incidence of spontaneous abortion, stillbirth, and neonatal death. Changes in the metabolism of the placenta and in various hematological factors in the newborn infant have been found to be associated with maternal smoking, but the mechanism of the effect of smoking on the outcome of pregnancy remains to be determined.

New studies on the effect of maternal smoking on the outcome of pregnancy have been published since the review of this topic in the 1967 Report (11). In the 1967 review, the literature cited supported a relationship between maternal smoking and low birthweight and prematurity in infants. However, the evidence relating maternal smoking to fetal or neonatal death was not definitive. The addition of new studies has reconfirmed the relationship between maternal smoking and low birth weight and prematurity. The relationship between maternal smoking and spontaneous abortion, stillbirth, and neonatal death has been investigated in several studies. As detailed below, some of the studies reported a statistically significant increase in unsuccessful pregnancies in mothers who smoked when compared with mothers who did not smoke.

EPIDEMIOLOGICAL STUDIES

In a prospective study of more than 2,000 pregnant women, Russell et al. (8) examined the effect of the mother's smoking habits and blood pressure on the outcome of the pregnancy and on the birth weight of the infant. A smoker was defined as one who regularly smoked five or more cigarettes a day. In each blood pressure category the percentage of unsuccessful pregnancies (abortion, stillbirth, neonatal death) was higher for smokers. Although fewer smokers were found in the higher blood pressure categories, women who smoked and had blood pressure levels equal to, or greater than, 150/100 had a rate of unsuccessful pregnancy of 31.4 percent as compared to a rate of 14.5 percent among nonsmokers with the same blood pressure.

levels. Although the number of women in the two groups was small (35 and 138, respectively), the difference observed was statistically significant. For those with blood pressure levels of less than 140/90, the percentage of unsuccessful birth was 6.5 among smokers and 2.7 among nonsmokers; for those with blood pressure levels in the range of 140/90, the percentage was 6.8 among smokers and 4.1 among nonsmokers.

Extrapolating from his series, Russell (7) estimated that one out of every five unsuccessful pregnancies in women who smoked regularly would have been successful if the mother had not smoked regularly during the pregnancy. This statement implies a cause-and-effect relationship between maternal cigarette smoking during pregnancy and abortion and perinatal death. In the absence of proof of a cause-and-effect relationship, the least that can be said is that on the basis of the findings of Russell, et al., one out of every five unsuccessful pregnancies among women who smoke regularly during pregnancy would not have been unsuccessful if these women had the same risk of unsuccessful outcome of pregnancy as women who do not smoke.

In keeping with previous findings, Russell, et al., found that the mean birth weight of the infant was lower for the smoking mothers in each blood pressure category. Various factors were examined as confounding variables for their possible effect on birth weight and the production of spurious associations. These included: social class of consort, maternal age, parity, maternal height, social class of woman's father, educational level, age of consort, maternal attitude toward the pregnancy, work during pregnancy, and sex of offspring. For each variable, the smoking effect was clearly distinguished as a separate effect even when the individual factor was itself associated with smoking (consort's social class, father's social class, and maternal educational level).

A study of increases in the infants' weight and in their head circumference during the early weeks of life revealed that the babies of smoking mothers grew faster than those of nonsmokers through the sixth month after birth. However, the mean weight per week of conception age (duration of pregnancy, plus age after birth) was greater in babies of nonsmokers through the sixth week after birth, the effect not being visible at the sixth month examination. These last two findings support the theory that smoking during pregnancy acts as a retarding influence on fetal growth and that a catching-up phenomenon begins among the babies of smoking mothers at birth when the toxic influence is removed.

In a controlled study of 197 premature births among Negroes, Terris, et al. (9) found a significantly higher prevalence of smoking among the mothers of premature infants. Prematurity was defined as a birth weight of 2,500 grams or less.

Mulcahy, et al. (6) studied the relationship between smoking habit and the outcome of pregnancy in 3,681 women admitted to the Coon Lying-in Hospital in Dublin, Ireland. Besides finding significant lower birth weight for infants born to mothers who smoked, they covered a significant increase in the incidence of neonatal death, stillbirth, and spontaneous abortion. These effects were independent of gestation or parity. No significant difference in the rate of congenital abnormalities was found between the offspring of the smokers and those of the nonsmokers.

Kizer (4) studied the effect of maternal smoking on the outcome of pregnancy in 2,095 patients in Venezuela. He found a significant diminution in the birth weight of infants of smoking mothers and a higher incidence of premature rupture of the membranes, but did not find a difference in the incidence of abortion or perinatal mortality.

Duffus, et al. (2) studied the relationship between smoking during pregnancy and the incidence of albuminuric preeclamptic toxemia in 2,543 married, urban primigravidae attending antenatal clinics in Aberdeen in 1960. Albuminuric preeclampsia is defined as albuminuria in pregnancy in which the urine contains at least 0.25 grams of albumin per liter accompanied by a rise in diastolic blood pressure to 90 mm Hg. or more, on 2 or more days after the 26th week of gestation or progressively during labor. The incidence of albuminuric preeclampsia was lower in smokers than in nonsmokers. Among women with preeclampsia, however, smokers lost more babies in the perinatal period than the nonsmokers. The babies of smokers, both normal and preeclamptic, had a lower mean weight than the babies of nonsmokers. In the preeclamptic group, a greater percentage of the babies of smokers weighed less than 5 pounds. These differences are in keeping with those found in other studies but do not reach statistical significance. The implication is that smoking mothers are less likely to become preeclamptic, possibly by way of blood pressure effects, but are more likely to have their pregnancies result in perinatal death in the event they are preeclamptic.

In a study of 5,843 deliveries in Hungary, Fülöp (3) found a statistically significant increase in premature births among women who smoked during their pregnancies, whether the women were married or unmarried, held a job, or were unemployed. Lacuska, et al. (5) found a higher frequency of premature births and abortions among women who smoked during pregnancy than among nonsmokers, although the differences fell short of statistical significance.

Tokuhata (10) analyzed the fertility history in relation to smoking in groups of married women who died of breast cancer, genital cancer, and various noncancerous diseases. Statistically significant increases were found in both the rate of infertility (as judged by absence of pregnancy) and the rate of miscarriage among women who smoked.

nancy) and in fetal loss (defined as abortions and stillbirths) were found in smokers who died of noncancerous diseases. These differences withstood analysis for a number of possible confounding factors. However, since the sample was made up of women who died in a certain geographical area in a given amount of time, biases may have been introduced. Retrospective findings in a group of dead people are not necessarily the same as findings derived in a prospective study of a living population.

Although by this time the evidence for reduction in birth weight of babies born to smoking mothers is overwhelming, a problem that remains to be solved is why some studies do and others do not appear to show fetal wastage as measured by abortion, stillbirth, and neonatal death. It may be that the method of selection of the population under study, especially the degree to which entire obstetrical histories are included, accounts for this variation.

EXPERIMENTAL STUDIES

Younoszai, et al. (13) compared various hematological factors in the blood of 16 smoking mothers and newborn infants with those of 16 nonsmoking mothers and their offspring. Both groups of infants were delivered at term and appeared clinically well. The smoking mothers had a mean carboxyhemoglobin saturation of venous blood of 8.3 percent as compared to 1.2 percent in the nonsmoking mothers. Corresponding figures for the umbilical vein cord blood were 7.3 percent and 0.7 percent. A mild metabolic acidosis was seen in the infants of smokers. These infants also had a higher mean capillary hematocrit than those of the nonsmoking mothers. The authors point out that the differences, although real, probably are not of clinical significance in the newborn. However, the effect of chronic exposure of the embryo and fetus to carboxyhemoglobin levels and other hematological abnormalities has not been elucidated.

Welch, et al. (12) reported that the placentas from women who smoked during pregnancy show a much greater ability to hydroxylate benzo(a)pyrene than the placentas from women who did not smoke during pregnancy. The placentas from women reporting similar cigarette consumption varied greatly in the degree of BP hydroxylase activity. However, no information is available on the brand of cigarettes smoked or the degree of inhalation, differences which may result in different dosages of BP. It is possible, but not likely, that carcinogens in tobacco smoke reach the fetus in significant amounts. The ultimate effect of the exposure of the human fetus to carcinogenic substances is unknown.

Becker, et al. (1) studied the effect of subcutaneous injections of increasing doses of nicotine on groups of pregnant rats and their off-

spring. They found that the rats receiving nicotine injections consumed less food and gained less weight than control animals and the magnitude of this effect increased when the dose of nicotine was greater. Whereas no other differences were found in the rats receiving lower dosages, those receiving 3.0 mg./kg. or 5.0 mg./kg. daily had offspring which differed from those of the controls in being lighter, having a longer gestation, a higher mortality rate during the first 4 hours of life, and a fetal appearance.

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CHAPTER 5

Smoking and Noncancerous Oral Disease

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SMOKING AND NONCANCEROUS ORAL DISEASE

SUMMARY

The previous reports have not presented findings on noncancerous oral disease. Several recent studies have made a review appropriate at this time. This review of the available literature leads to the conclusion that ulceromembranous gingivitis, alveolar bone loss, and stomatitis nicotina are more commonly found among smokers than among non smokers. The influence of smoking on periodontal disease and gingivitis probably operates in conjunction with poor oral hygiene. In addition there is evidence that smoking may be associated with edentulism and delayed socket healing. While further experimental and clinical studies are indicated, it would appear that nonsmokers have an advantage over smokers in terms of their oral health.

EPIDEMIOLOGICAL AND CLINICAL STUDIES

Periodontal disease is a chronic destructive process affecting the supporting structures of the teeth (gingiva, periodontal fibers, and alveolar bone). It is generally considered inflammatory in nature. Solomon et al. (21) studied data on 3,552 nonsmokers and 3,639 smokers, all white and between the ages of 20 and 79. He found that periodontal disease occurred without significant statistical difference in male and female nonsmokers of the same age, but that smokers of both sexes had a higher prevalence of the disease. The prevalence in female smokers paralleled that in male smokers in the younger age groups but resembled that of the nonsmokers in the older age groups. The author believes that this difference is related to increased smoking in younger women.

Brandtzaeg, et al. (3) examined 206 Norwegian Army recruits between the ages of 19 and 25 and found a trend toward increased periodontal disease with increased smoking. However, when an analysis of covariance was performed, most of the changes in periodontal disease severity were accounted for by changes in oral hygiene. This finding suggests that tobacco consumption may influence the periodontal tissues but only with accompanying changes in oral hygiene.

A seemingly contradictory paper reporting on periodontal disease in 8,206 Ceylonese was published by Waerhaug (25). He found tobacco smokers to have less periodontal disease than nonsmokers. H

pointed out, however, that for many individuals the alternative to smoking tobacco is chewing betel nuts, which is associated with even more periodontitis than cigarettes. Thus, tobacco users are relatively better off.

The relationship of smoking to gingivitis, the initial stage of periodontal disease, has also been studied. Arno, et al. (2) examined 1,346 employees of a manufacturing company in Oslo and found that tobacco smoking was associated with an increase in the prevalence of gingivitis. However, its importance as compared with that of oral hygiene was not a dominating one. Ludwick, et al. (15) studied 2,577 naval enlistees at the Great Lakes Naval Training Center and found no relationship between smoking and simple marginal gingivitis, but a significant one between smoking and ulceromembranous gingivitis (necrotizing ulcerative gingivitis, Vincent's gingivitis, trenchmouth). This is an acute form of periodontal disease of apparent sudden onset, characterized by ulceration of the tips of the interdental papillae, gingival bleeding, pain, and foul odor. In the United States and Europe, it occurs primarily in adolescents and young adults. Bacteria, local factors, systemic factors, and psychogenic factors have been suggested as contributing to its etiology (10).

Pindborg's study (17) of 1,433 Danish Royal Marines between the ages of 16 and 28 revealed that the prevalence of chronic marginal gingivitis was not affected by smoking, but that the prevalence of ulceromembranous gingivitis was much greater in smokers than nonsmokers. A second study by Pindborg (16) of 3,505 Danish military personnel confirmed these findings: nonsmokers had a prevalence of ulceromembranous gingivitis of 2.2 percent, while for those who smoked 10 g. or less of tobacco daily, the prevalence was 7.0 percent, and for more than 10 g. a day it was 9.5 percent.

Smitt (20) found a prevalence of ulceromembranous gingivitis of 2.5 percent in Dutch Navy recruits. In those who smoke 50 g. of tobacco for a week or more, the prevalence was 10.5 percent.

Frandsen, et al. (9) investigated the correlation between the form of tobacco used and occurrence of gingivitis in Danish Marines. He found that 1,848 cigarette smokers and 273 pipe smokers had essentially the same rates of simple marginal and ulceromembranous gingivitis.

Arno, et al. (1) and Herulf (11) have investigated alveolar bone changes in smokers. Arno studied 728 men between the ages of 21 and 45 and found that alveolar bone loss, measured as the percentage of maximum height adjacent to the mesial and distal surfaces of each tooth present, was higher among those with high tobacco consumption. The author suggested that tobacco consumption is a complicating factor in periodontal disease and when accompanied by poor oral hygiene

and unfavorable systemic background may help speed up the destruction of the supporting tissues of the teeth.

Herulf measured interdental bony septa in 389 men and 215 women at the Institute of Dentistry in Stockholm. He, too, found a significant relationship between smoking and bone loss.

The relationship between cigarette smoking and edentulism has been studied by Summers, et al. (22) in a sample of residents of Tecumseh, Mich. Information on 324 dentulous and 84 edentulous people revealed that among males in both groups those with the greatest evidence of periodontal disease smoked significantly more cigarettes than those with medium or little evidence of the disease. Solomon, et al. (23) found significantly more edentulism and advanced periodontal disease in both men and women who smoked cigarettes than in nonsmokers of the same age.

Jackson (12) has cited heavy smoking as a factor in delayed healing of tooth sockets after extraction.

Stomatitis nicotina is a form of palatal leukoplakia (4). It is characterized by raised umbilicated papules with small central red depressions located primarily on the soft palate and the posterior region of the hard palate. The papules represent blocked palatal mucus glands and the red depressions are their inflamed duct orifices. Saunders (14) notes that the lesions begin as tiny red dots and may progress very rarely to ulceration. Although it sometimes occurs in cigar and cigarette smokers, stomatitis nicotina is found most frequently in pipe smokers (4, 5, 19). According to Chapman, et al. (4), pipe smoking points a stream of smoke directly onto the palate, thereby allowing longer contact between it and the smoke than in other forms of tobacco use. The condition disappears with the cessation of smoking (6, 7, 14, 18, 19, 24), though Kerr (13) warns that healing may be slow, sometimes requiring months before no lesions are present.

Thoma (23) observed a patient who wore dentures for over 4 years and showed lesions of stomatitis nicotina only on the part of the palate that was not covered by the prosthesis. He concluded that the changes were due to local surface rather than to systemic influence.

Lewis (14), Saunders (18), and Thoma, et al. (24) advise biopsy to rule out malignancy in advanced cases. Forsey, et al. (8) feel that no association between stomatitis nicotina and cancer has been demonstrated.

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