ASBESTOS AND LARYNGEAL CARCINOMA

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Summary One hundred male patients with squamous carcinoma of the larynx and one hundred matched controls with various non-malignant diseases were questioned about their exposure to asbestos. Thirty-one patients and three controls had experienced an important degree of exposure to asbestos. The difference between the two groups was statistically highly significant. The average latent interval between first exposure and development of laryngeal carcinoma was 30 years, and the average duration of exposure was 27 years.

Introduction

PULMONARY asbestosis is a recognised occupational hazard of long exposure to asbestos dust. There is now also considerable evidence to support the contention that pulmonary asbestosis plays a definite role in the development of bronchial carcinoma,[1] and, furthermore, the association between mesothelioma of the pleural and peritoneal cavity and exposure to asbestos is widely accepted.[2,3]

The results of a previous study of the relation between exposure to asbestos and cancer of the upper respiratory tract [4] indicated that exposure to asbestos was most likely to be of importance in laryngeal carcinoma. The present study is an effort to investigate in greater depth the importance of asbestos in laryngeal carcinoma.

Patients and Methods

One hundred consecutive male patients with laryngeal carcinoma attending the Liverpool Ear, Nose, and Throat Hospital formed the basis of the study. The controls were a series of one hundred male patients, matched for age, who were attending the hospital for various non-malignant conditions. Both groups were questioned to determine their exposure to asbestos and their smoking-habits. This information was obtained in both groups by personal interview. All the tumours were histologically proven squamous carcinoma.

The age-distribution of the patients and controls is shown in table I, and their smoking-habits are shown in table II.

Results

Out of one hundred patients with laryngeal carcinoma, thirty-one (31%) had important exposure to asbestos, as compared with three (3%) of the control series. The difference between the two groups is statistically highly significant ($\chi^2=30{\cdot}1$, $n=1$, $P<0{\cdot}001$).

There were three types of occupational exposure: lagging of heating equipment (nineteen patients), scaling of boilers (three patients), and unloading raw asbestos on the docks (nine patients) (before the introduction of container traffic).

Analysis of data from these asbestos workers showed that the latent period between first exposure and the development of laryngeal carcinoma ranged from 1 to 54 years, the average being 30 years; the average duration of exposure was 27 years (table III). In those cases of laryngeal carcinoma associated with asbestos, the maximum incidence was in the 51–60 age-group, as compared to the usual maximum incidence in the 61–70 age-group.[5]

There was no difference in the smoking-habits of the patients who were asbestos workers compared to those patients who had no association with asbestos (table II). But, as might be expected, there was a greater percentage of non-smokers among the controls compared with those patients who had laryngeal carcinoma.[5]

TABLE I—AGE-DISTRIBUTION IN PATIENTS AND CONTROLS

Age-group	Patients					Controls
	History of exposure		No history of exposure		Total	
	No.	%	No.	%		
41–50	4	13	8	11	12	13
51–60	13	42	19	28	32	32
61–70	10	32	33	48	43	43
71–80	4	13	8	11	12	12
81–90	·		1	1	1	

TABLE II—SMOKING-HABITS OF PATIENTS AND CONTROLS

—	Patients				Controls (%)
	History of exposure		No history of exposure		
	No.	%	No.	%	
Non-smokers	4	13	4	5	20
1–10 cigs./day	7	23	13	19	22
11–20 cigs./day	11	35	27	39	32
Over 20 cigs./day	9	29	18	26	19
Pipe	..	..	7	10	7

TABLE III—LATENT PERIOD AND DURATION OF EXPOSURE TO ASBESTOS IN PATIENTS WITH LARYNGEAL CARCINOMA

Years	Latent period of exposure	Duration of exposure
0–5	2	4
6–10	0	4
11–15	1	2
16–20	2	0
21–25	3	2
26–30	6	5
31–35	4	2
36–40	2	10
41–45	10	0
46–50	0	1
51–56	1	1

Discussion

Whilst the results quoted above suggest that there is an association between exposure to asbestos and laryngeal carcinoma, it has to be admitted that this conclusion is based on a retrospective study, with all the disadvantages of such a study. A prospective study of the problem would be difficult, however, since the annual incidence of laryngeal carcinoma is of the order of 1/50,000, and it would be difficult to collect a static population of asbestos workers large enough to produce sufficient cases of laryngeal carcinoma for a prospective survey.

In our series of thirty-one cases of laryngeal carci-

noma with a history of exposure to asbestos there was only one patient with pulmonary asbestosis. (There was apical pulmonary fibrosis in five other cases, but this is unlikely to be related to asbestos exposure.) The explanation of this apparent anomaly seems to be that heavy industrial exposure in the past has in most cases resulted in severe asbestosis and early death before the patient could survive long enough for cancer to develop. With the introduction of modern precautions for the protection of asbestos workers, there has been a fall in the incidence of asbestosis and an increase in the number of cases of asbestos-associated cancer.[6] This would be in keeping with a long latent period, of say 30 years, as in our patients.

The patients' smoking-habits were similar, irrespective of their exposure to asbestos. As expected, however, there were more smokers among the patients than among the controls.[3] It may be that smoking is a cofactor in the development of laryngeal carcinoma, but, as previously pointed out,[5] it is unlikely to be very important, since the incidence of laryngeal carcinoma in the twentieth century has declined slightly in the face of an enormous increase in tobacco consumption.

We intend to perform histological examinations for asbestos bodies in patients with laryngeal carcinoma. Unfortunately, however, not many specimens are available because the vast majority of patients with laryngeal carcinoma are treated by radiotherapy. Even if the tumour recurs and the larynx becomes available for study after laryngectomy, it is doubtful whether asbestos bodies could be found—presumably they would lie at the centre of the tumour and thus be sloughed off as the tumour ulcerated during its growth. Even if they survived this insult, they would almost certainly slough out in the healing process after radiotherapy. As a pilot study, we examined serial sections from one larynx, but no asbestos bodies were found.

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Hypothesis

ACUPUNCTURE ANÆSTHESIA

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Summary A possible mechanism to explain acupuncture anæsthesia is presented which takes into consideration the fact that acupuncture needles have to be vibrated or rotated and which is not invalidated by the lack of correlation between the traditional acupuncture sites and the anatomy of the peripheral nervous system. It is suggested that rhythmic stimulation causes areas of the cerebral cortex to become "locked on" to the stimulating rhythm and thus "busy" and unable to react to stimuli in the normal way.

Two hypotheses are currently favoured to explain how acupuncture might induce anæsthesia.[1] The one is hypnotism and the other is the so-called "gate hypothesis" of Melzack and Wall.[2] The latter is based on postulated interactions at cord level between pain stimuli travelling in the A and C fibres of the peripheral nerves resulting from the differences in speed of transmission in the two classes of fibre.

Neither of these accounts for the fact that it appears to be necessary for the acupuncture needles to be vibrated or rotated either manually or electrically.[1] A further hypothesis is presented below which takes this into account.

It is known that some patients suffering from jacksonian epilepsy whose attacks start in a periphery can abort their attacks if they apply a strong stimulus to the limb proximally in the "path" of the ascending sensory or motor disturbance.[3,4] It is postulated that this stimulus causes the cortex in the area surrounding the dysrhythmic focus to become sufficiently refractory to block the spread of the dysrhythmia to the rest of the cortex. The "busy" area of the cortex is partly refractory to further stimulation.

There is another circumstance in which the cortex can be made refractory to random impulses, and that is when a rhythmic stimulus is fed in through a sensory channel. This is most easily demonstrated in the case of the visual cortex, where photic stimulation at appropriate rates may induce a wide variety of subjective experiences not only in the visual fields but also in other sensory modalities. In many subjects fits of varying sorts may be induced and demonstrated electroencephalographically or seen clinically. This forms the basis of the use of photic stimulation in diagnostic electroencephalography.

The phenomenon is not confined to the visual pathways, and similar effects can be demonstrated electroencephographically and occasionally clinically with rhythmic stimulation of the periphery or of the auditory pathways. The rate of stimulation appears to be critical and to vary between about 1 and 25 cycles per second. Moreover, the interval between volleys of stimulation appears to be important in determining the response. Voluntary inhibition or augmentation of the responses can be demonstrated both electroencephalographically and subjectively.[5,6] It appears that, when a sensory stimulus recurs at an appropriate frequency, large areas of the cortex may become "locked on" to that frequency or a harmonic of it and that its normal function may be thereby disturbed.

The analogy with acupuncture anæsthesia is close. The frequency of vibration or rotation of the acupuncture needles is within the appropriate range