STUDIES IN
THE PHYSIOLOGY OF SWEATING IN CYSTIC FIBROSIS

II: ELEVATED NIGHT SWEATING RATES*

BY

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Since the discovery of high sodium chloride in the sweat of children with cystic fibrosis by di Sant’Agnese and others, many investigators have established this abnormality as a consistent finding (di Sant’Agnese, Darling, Perera and Shea, 1953; Shwachman, Leubner and Catzel, 1955; Sibinga and Barbero, 1961). A number of these reports have described a normal sweating rate in patients with cystic fibrosis (Gochberg and Cooke, 1956; Vink, 1957) when sweat was collected under thermal stimulation (di Sant’Agnese, Darling and Perera, 1953). Physicians and mothers, however, have frequently noted that children with cystic fibrosis sweat more profusely than other children. Parents describe multiple changes of bed clothing at night because of this profuse sweating. This apparent contradiction seemed to warrant investigation of spontaneous sweating as contrasted to the previously studied thermal and drug-induced sweating. This different approach to the physiological process of sweating might reveal abnormalities in threshold and sensitivity of the secretory mechanism.

Method

Collections of sweat on patients with cystic fibrosis and normal children of similar size were done simultaneously as follows: A round aluminium collection chamber of 2.5 cm. diameter (Schwartz, Thaysen and Dole, 1953) was glued on the skin between the scapulae. A number of pre-weighed discs of filter paper were inserted and sealed in the collection chamber, which was made airtight with a special cover. The apparatus was strapped in place with tape. By changing the filter paper at one- or two-hour intervals an accurate picture of the amount (as determined by weight) of sweat secreted was obtained. No extraneous stimulation was used. The environmental temperature was between 75° and 95° F. and relative humidity was between 80 and 95%.

Results

Fig. 1 shows the sweating rates of a child with cystic fibrosis and a normal child of equal weight (ages 4 years and 2 years 9 months) over a two-day period. Activity and temperature were accurately recorded. This experiment was continued over five days, and the pattern of sweating was identical from day to day. During the day the sweating rate in the normal child was greater than in the patient with cystic fibrosis. These differences were closely related to healthy physical activity. During the night after going to sleep, however, the sweating rate of the patient with cystic fibrosis invariably surpassed that of the normal child.

The periods during which the sweating rate of the cystic fibrosis patient exceeded that of the normal child are listed in Table 1. Similar trends in the 24-hour sweating pattern were observed on two other patients with cystic fibrosis during a number of days and compared with two control children.

These observations prompted an investigation of night sweating rates between 11 p.m. and 7 a.m. The total amounts collected at the same time on

| TABLE 1 |
| QUANTITIES OF SWEAT COLLECTED IN PATIENT WITH CYSTIC FIBROSIS AND CONTROL CHILD ON SUBSEQUENT DAYS |

<table>
<thead>
<tr>
<th>Days</th>
<th>Time</th>
<th>Quantities of Sweat</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>p.m.</td>
<td>Cystic Fibrosis Case (mg.)</td>
</tr>
<tr>
<td>1</td>
<td>11.30 until 8</td>
<td>714</td>
</tr>
<tr>
<td>2</td>
<td>11</td>
<td>120</td>
</tr>
<tr>
<td>3</td>
<td>10</td>
<td>394</td>
</tr>
<tr>
<td>4</td>
<td>10</td>
<td>491</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>177</td>
</tr>
</tbody>
</table>

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In studying sweating rates one must distinguish carefully between the findings in different age groups. Many perfectly healthy young children will exhibit night sweating to a certain degree, while in adults this phenomenon seems more closely related to pathological conditions. Some of the control children in this study were showing obvious night sweating, and were selected for that reason.

The significantly increased rate of sweating of the patients with cystic fibrosis measured during the night in these experiments can be related to certain other observations. It has been known for a long time that various pulmonary diseases, specifically tuberculosis, are associated with night sweating. This is frequently interpreted as a sign of a low grade fever. It is also stated to occur with fever in advanced bronchiectasis (Harrison, Adams, Bennett, Resnik, Thorn and Wintrobe, 1958) and many other pulmonary diseases (Cecil and Loeb, 1959). The patients with cystic fibrosis investigated in this study did not exhibit any fever, neither was there any sign of rickets which may be accompanied by night sweating. Increased heat production due to the laborious respirations might well result in sweating; however, a marked increase in sweating rate was observed during the night in our patients with cystic fibrosis when the respiratory requirements were less rather than increased. The markedly increased basal metabolic rate in acute respiratory failure does not result in sweating (Harrison et al., 1958), and some of our patients have sweated less in extreme respiratory insufficiency. Again no explanation for the increase during the night is found.

Congenital cardiac disease has been associated with night sweating and is said to disappear after cardiac surgery. Cyanosis is a feature in both cardiac disease and cystic fibrosis; however, two of the patients with cystic fibrosis were mild cases, one of whom had no respiratory involvement. Hyponatraemia may result in increased sweating rate (Amatruda and Welt, 1953). Blood sodium levels determined on several of the patients were normal and all cystic fibrosis patients were on supplementary salt intake. This last factor is not considered to be the cause of the observed difference in sweating rate. The mechanism for the sweating

![Graph](image-url)
that occurs with peripheral vascular collapse is not established with certainty and like many of the earlier examples have been classified as neuro-vegetative reactions. Kuno (1956) assumes an increase in excitability of the thermal sweat centre in the hypothalamic region by fall-out of the tonic inhibitory action from the cerebral cortex during sleep. The findings on the sodium concentration of spontaneous sweat need further supplementation and will be the subject of further study.

The intrinsic abnormality of the sweat glands in cystic fibrosis might, as such, be responsible for this observation. On the other hand, an increased excitability of the hypothalamic sweating centre in cystic fibrosis cannot be ruled out at present. The purpose of this paper is to call attention to the abnormality in rate of secretion under these conditions. Further clarification of the many factors involved in such spontaneous nocturnal sweating is needed.

**Summary**

The pattern of spontaneous sweating over subsequent 24-hour periods was studied in three patients with cystic fibrosis and three control children. A marked difference in spontaneous night sweating rates was documented in 10 patients with cystic fibrosis and 10 control children. This finding may represent a non-specific abnormality in cystic fibrosis, but clearly shows another deviation of sweat gland function, possibly related to the specific pathophysiological mechanism responsible for the elevation of electrolytes in the sweat of patients with cystic fibrosis.

**REFERENCES**


