

Amylase activity in tracheobronchial secretions of laryngectomized patients

V. NANDAPALAN, F.R.C.S.*, J. C. McILWAIN, M.D., F.R.C.S.†, J. ENGLAND, F.R.C.S.†

Abstract

The alpha-amylase activity in tracheobronchial secretions of 16 consecutive patients with a total laryngectomy was studied. None of these patients had a tracheopharyngeal fistula or pulmonary disorder which might affect the amylase activity. This study proves the presence of amylase in tracheobronchial secretions of laryngectomized patients with a normal lung at a level between x and y and the quantitative analysis of the amylase activity is discussed. The relevance of investigating laryngectomy patients is because of the nature of the surgery the lower respiratory tract is permanently and physically isolated from any other source of salivary amylase. No similar study of the analysis of amylase in normal lung tissue had been reported before.

This information may be of value in order to detect salivary aspiration in patients with a tracheostomy or endotracheal intubation if the level in the aspirate is in the order of a-b times greater than that found in normal tracheobronchial secretions. ($x = 35$ and $y = 1125$ i.u./l; $a = 31.8$ and $b = 628.6$ i.u./l).

Key words: Amylases; Laryngectomy; Trachea, secretions; Bronchus, secretions

Introduction

Alpha-amylase is a single polypeptide chain enzyme with a molecular weight of 56 000. The principal organs of secretion of this enzyme are the salivary glands (ptyalin – salivary alpha-amylase) and pancreas (pancreatic alpha-amylase). Alpha-amylase activity had also been reported in normal liver (Ende, 1960). Both salivary and pancreatic alpha-amylase act on ingested polysaccharides and hydrolyse 1, 4-alpha linkages sparing 1,6-alpha linkages, terminal 1,4-alpha linkages and the 1,4-alpha linkages next to branching points (Ganong, 1987). The end products of alpha-amylase digestion are oligosaccharides, the disaccharide maltose, the trisaccharide maltotriose, some slightly larger polymers with glucose in 1,4-alpha linkage and alpha-limit dextrans (Ganong, 1987). Both salivary and pancreatic alpha-amylase have similar organic structures but they can be differentiated by electrophoretic activity due to their different molecular size.

In 1938 Takano provided an important clue to the fact that the lung deserved special attention with respect to amylase by his report that amylase activity in the left cardiac blood was greater than that in the right in rabbits. Since then, many reports have appeared describing the occurrence in man of high serum amylase (hyperamylasemia) associated with a variety of pulmonary diseases (Luhr, 1951; McGea-

chin and Adams, 1957). Ende (1960) noted a significant correlation between the presence of neoplastic cells in the pleural or ascitic fluid and the amylase activity in these fluids.

The exact role of amylase in lung parenchyma is still obscure. Berk *et al.* (1978) recorded amylase activity in three normal lung extracts and found its activity clearly exceeded that in normal serum. No comment was made in their study as to whether the amylase in the lung extracts was in fact due to secretion by the lung tissue or due to contamination by the saliva. Furthermore on isoamylase analysis of the lung extracts they found the predominant isoamylase form was the salivary type, with the pancreatic type accounting for only three to eight per cent. Harada and Kitamura (1971) analysed the amylase further and found the majority of the amylase in urine, serum and pleural fluid of patients with lung cancer corresponded to salivary type isoamylase.

The foregoing data provide convincing evidence that amylase does exist in lung tissue and amylase activity in the serum may increase in association with various diseases of the lung. Factual information with respect to amylase activity and the normal lung is lacking in the literature.

The aim of this study was to confirm the presence

From the Departments of Otorhinolaryngology, Royal Liverpool University Hospital*, Prescott Street, Liverpool and Whiston Hospital†, Liverpool.

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of amylase in tracheobronchial secretions and to quantitatively analyse its value.

Patients

Sixteen consecutive patients who had had a total laryngectomy, at Whiston Hospital, for primary laryngeal pathology were recruited for this study. The purpose of selecting such patients was to obtain uncontaminated tracheobronchial secretions. Their total laryngectomy operation had been performed between two and 10 years (mean 4.2 years) earlier than the date of the study. None of the patients had a tracheopharyngeal fistula, and had never been fitted with a speaking valve. The purpose of the study was explained to the patients and their written consent was obtained.

All patients were interviewed regarding lung disorders and they were examined to exclude any such disorders. A routine chest X-ray was performed on each patient to detect any obscure pathology. Patients with evidence of pulmonary infection and pulmonary metastasis were to be excluded but there were none.

Methods

The tracheobronchial secretions were collected by sterile technique, using a sputum trap container attached to a suction catheter (Penine-soft tip intraluminal catheter - 14 F.G.). A sample of oral saliva was collected from each patient into another sterile container at the same time. The containers with the collected specimens were labelled and transported to the laboratory without delay for analysis of alpha-amylase.

The blocked substrate maltoheptoside method was used at 37°C to assess amylase activity in the sample. The substrate *p*-nitro-phenyl-alpha-D-mal-toheptoside was hydrolysed by alpha-amylase into several oligosaccharides of shorter chain lengths. The oligosaccharides were further hydrolysed by glucoamylase and alpha-glucosidase to glucose and *p*-nitrophenol, which absorbs at 405 nm wave length in the spectrometer. The rate of production of *p*-

nitrophenol is directly proportional to the activity of alpha-amylase in the sample.

Results

The results were tabulated to show the patient's amylase activity in saliva and in tracheobronchial secretions, and the percentage ratio (Table I). Table I also shows age and sex of the 16 study patients and the amylase activity in the tracheobronchial secretion and in the saliva.

These figures show that the amylase activity in tracheobronchial secretions ranged widely, except for one patient (no. 12), where the amylase level was 1125 i.u./l. In the other 15 patients amylase levels were below 1000 i.u./l (range: 35-950 i.u./l).

Discussion

The present study shows amylase exists in tracheobronchial secretions but its level was much lower than in saliva. In fact the amylase activity in the saliva of these patients was in the ratio of about 30 to 600 times greater than that of the tracheobronchial secretions. The percentage ratio of tracheobronchial amylase to salivary amylase ranged between 0.8 to 3.15. Berk *et al.*, (1978) found that the amylase activity in their study of three normal lung extracts was between 762 and 1125. In our study the amylase activity in the tracheobronchial secretion was between 35 and 1125 i.u./l.

The preponderance of male patients in the study group reflects the incidence of laryngeal carcinoma in the population. Laryngectomized patients were ideal for our study because salivary contamination of the trachea could not occur in the absence of a tracheopharyngeal fistula. Following total laryngectomy the physiology of respiration is affected but there is no evidence to suggest amylase production is unique in these patients.

In the collection of the specimens no saline was used, as dilution could lead to erroneous results. The specimens were collected in sterile containers and immediately transported to the laboratory for

TABLE I
AGE AND SEX OF THE STUDY PATIENTS AND THEIR AMYLASE ACTIVITIES IN THE TRACHEO-BRONCHIAL SECRETION AND IN THE SALIVA AND THE PERCENTAGE RATIO OF TRACHEO-BRONCHIAL AMYLASE TO SALIVARY AMYLASE

Patient no	Age	Sex	Amylase in tracheo-bronchial secretion (i.u./l)	Amylase in saliva (i.u./l)	Ratio of salivary amylase/ tracheo bronchial amylase
1	58	M	280	27 000	96.4
2	62	M	50	31 000	620.0
3	62	M	56	29 000	517.9
4	63	M	120	25 000	208.3
5	78	M	240	30 100	125.4
6	59	M	880	28 300	32.2
7	63	M	650	20 800	32.0
8	77	M	35	22 000	628.6
9	75	M	56	24 300	433.9
10	62	F	150	28 000	186.7
11	63	M	950	38 400	40.4
12	68	M	1125	36 600	32.53
13	69	M	780	24 800	31.8
14	67	M	700	31 100	44.4
15	62	M	470	28 900	61.5
16	66	M	310	25 000	80.6

analysis, in order to minimize bacterial action on amylase activity.

Luhr (1951) noted that in 11 out of 80 cases of lung diseases (pneumonia and tuberculosis) hyperamylasemia was displayed and strikingly that 30 out of 32 cases of lung carcinoma were associated with hyperamylasemia. By contrast, he found hyperamylasemia in only one case among 55 other types of non-pulmonary cancers. McGeachin and Adams (1957) studied eight patients with primary lung cancer and found three patients with some degree of hyperamylasemia. They also confirmed the occurrence of high serum amylase in pneumonia. Individual cases of lung cancer associated with increased amylase activity in the serum and/or in the urine were reported by Weiss *et al.* (1951) and Sachar (1952).

Ende (1960) noted a significant correlation between the presence of neoplastic cells in pleural and ascitic fluid and the amylase activity in these fluids, and he interpreted the finding as evidence for amylase production by the neoplasm. In the subsequent year the same author reported high amylase levels in pleural fluid associated with various cancers, including cancer of the lung, and offered the suggestion that the lungs may serve as the site of origin for the amylase (Ende, 1961).

Related to Ende's observation Saugier *et al.* (1976) reported that most of the 14 patients they studied without pancreatic disease, but with pleural or ascitic fluid rich in amylase, had carcinoma of the lung.

Hyperamylasemia is also found in various lung disorders other than cancer. Benjamin and Kenny (1974) described marked high serum amylase (hyperamylasemia) and high urinary amylase (hyperamylasuria) associated with acute viral pneumonitis. Heffernon *et al.* (1976) reported that out of 17 heroin addicts with acute pulmonary changes, 13 developed hyperamylasemia.

Otsuki *et al.* (1977) and Moores *et al.* (1977) found hyperamylasemia following open heart surgery and suggested pulmonary cellular hypoxia secondary to inadequate tissue perfusion as the possible causative mechanism.

Clarke *et al.* (1981) studied amylase activity of transtracheal aspirations of 21 patients with moderate to severe chest infections. Their study showed that six out of 21 seriously ill patients showed greatly increased levels of amylase activity in the bronchial secretions compared with those found in the 15 less ill patients.

This study strongly suggests that amylase is present in the normal lung. The levels of amylase activity in normal lung, both mean (428.2 i.u./l) and median (295 i.u./l) values, obtained in our study are considerably lower than the median amylase activity in the trans-tracheal aspirate (2600 i.u./l) of patients with moderate to severe chest infections reported by Clarke *et al.* (1981). The percentage ratio of tracheobronchial amylase to salivary amylase varies only by a small margin (0.8–3.15). This information may be of value for comparison in patients with aspiration of saliva into the airways.

Conclusions

There exists in the isolated tracheobronchial tree of laryngectomized patients a level of amylase at least 30th of that found in saliva. In the event of salivary aspiration the amylase activity in the tracheobronchial secretion is expected to increase by a large amount. This information may be of value for detection of salivary aspiration in patients with a tracheostomy or endotracheal intubation.

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Address for correspondence
Mr J. C. McIlwain
Department of Otorhinolaryngology,
Whiston Hospital,
Prescot, Merseyside L35 5DR.

Fax: 0151 430 1094