THE PULMONARY COMPLICATIONS OF DYSPHAGIA

BY

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INTRODUCTION

This paper is concerned with the pulmonary aspiration phenomena associated with mechanical dysphagia, and the term "dysphagia pneumonitis" is used for the syndrome. Lesions due to central dysphagia and secondary to bronchooesophageal fistulae have been excluded. In 1927 Vinson reported a case of achalasia which was complicated by a lung abscess, and since then 38 others have been described. These, together with a further collection of nine cases hitherto unpublished, form the basis of this article.

After Vinson's paper the majority of reports in the 1930's were published by French otolaryngologists as single unusual histories. But from 1938 onwards more have appeared in the American literature, some have been described by clinicians, and others by radiologists. Weens (1944) collected five and reviewed the literature, Bird-Acosta (1944) described a further three, and Hawes and Soule (1945) two cases. Six have been published in the British journals by Cummins and Williams (1933), Schrier (1938), Robson and Wilkinson (1946), and Wooler (1948); others have been described incidentally in reviews of different conditions. Jackson and Jackson (1933), in discussing pulmonary symptoms of oesophageal disease, cite three cases, and Keefer (1934) reported one aspiration lung abscess in a paper describing seventeen examples of oesophageal carcinoma associated with pulmonary lesions. Harrington (1945) and Lahey (1946), in their large series of pharyngeal diverticula, each mention four cases complicated by lung abscesses.

The association of dysphagia with the presence of non-pathogenic acid-fast bacilli in the sputum and with lipoid pneumonia has led to the publication of a further six cases (Rothstein and Pirkle, 1946; Baldwin, 1942; Thomas and Jewett, 1936; Warring and Rilance, 1943; Cummins and Williams, 1933; Tcherthoff, 1941), and Wooler (1948), in his paper on cardiospasm, refers to three examples of "dysphagia pneumonitis."

Case Histories

The summaries of the nine cases given below include examples of the main causative factors and of the commoner resulting pulmonary lesions.

Case 1. Achalasia complicated by collapse.—A woman aged 65 had thirteen years' history of dysphagia due to achalasia. She was investigated in November, 1947, and a gross mega-oesophagus was discovered (Plate Ia). In January, 1948, she was admitted to hospital with three weeks' history of productive cough, fever, and dyspnoea. A radiograph (Plate Ib) showed a collapsed right lower lobe. Her clinical condition improved and an oesophagogastronomy was performed (N. R. Barrett) in March, 1948. The operation was complicated by total atelectasis on the right side. The upper lobe re-expanded after aspiration of food particles through a bronchoscope, but the lower lobe collapse persisted.

Case 2. Achalasia complicated by pneumonitis with acid-fast bacilli in sputum.—A man aged 41, from 1940 to 1945 complained of slight dysphagia on hurry ing food. In 1945 he developed "pneumonia." Acid-fast bacilli were found in the sputum. A radiograph (Plate Ila) showed bilateral mid-zone infiltration with a suggestion of cavitation on the right side. Rapid clinical and radiological improvement ensued until his radiograph was almost clear six months later. In March, 1948, a Heller's operation was performed (N. R. Barrett) for increasing dysphagia. He had no pulmonary symptoms in the interim, the radiograph was then clear (Plate IIb) and no acid-fast bacilli were found in his oesophagus or sputum.

Case 3. Achalasia associated with "typical" fibrosis.—A man aged 50, whose abnormality was discovered on mass radiography (Eley, 1948), had had slight dysphagia for years with recent dyspnoea and repeated bronchitis. The radiograph (Plate IIIa) is characteristic of the condition; this and his symptoms have now remained unchanged for a year.

Case 4. Carcinoma of lower third of oesophagus and lung abscess.—A man aged 69 gave a three weeks' history of cough and purulent sputum. A radiograph showed a left dorsal-lobar lung abscess with a fluid level. He improved until he was symptom-free one month later. The radiograph now showed no cavity,
THE PULMONARY COMPLICATIONS OF DYSPHAGIA

but there was residual fibrosis in the fissure area. Two
months later his dysphagia became overt and a barium
swallow and oesophagoscopy revealed a carcinoma of
the lower third of the oesophagus. There was no
evidence of a fistula.

Case 5. Pharyngeal diverticulum with patchy
pneumonitis.—A man aged 65 who had had bron-
chitis for years developed "pneumonia" eight years
ago and had had repeated attacks since then. He also
noticed dysphagia for six to eight years, which was
getting gradually worse. In August, 1947, he de-
veloped a patch of infiltration at the apex of his right
lung, and acid-fast bacilli were found in the sputum
on one occasion. The symptoms cleared rapidly, but
the pneumonia reoccurred in December, 1947; he has
remained sputum-negative since then. His diverticu-
lem was excised in June, 1948 (C. Price Thomas).
His bronchitis persists, but there are no other
symptoms, and the infiltration in the radiograph is
now clearing.

Case 6. Pharyngeal diverticulum with lung abscess.
—A man aged 46 complained of one year's history of
dysphagia. In September, 1947, he developed pneu-
monia, and two months later during a second attack
he was diagnosed as having a lung abscess. He was
admitted to hospital in January, 1948, and was shown
to have a pharyngeal diverticulum associated with
a left upper-lobe lung abscess and collapse of the left
lower lobe. One month later he developed a patch of
"pneumonitis" in the right lower lobe which cleared
with chemotherapy. His general condition improved,
and the radiograph cleared, but the left lower
lobe collapse persisted. In February, 1948, the
pouch was excised and he was discharged much
improved one month later. At that time the collapse
and abscess had cleared, leaving areas of pulmonary
fibrosis.

Case 7. "Short" oesophagus associated with
bilateral bronchiectasis.—A child aged 6 gave a history
of frequent coughs, colds, and pain in chest since the
age of 1 year. She had "pneumonia" when aged 2
years and 3 years, and thereafter continuous non-
foetid sputum. She was investigated in 1945 and
found to have almost total bronchiectasis on the right
side. She was seen again six months later when she
had started "vomiting" white phlegm and was not
 gaining weight. Another bronchogram at this time
showed lingual bronchiectasis. One year later the
vomiting was much worse and a barium swallow
showed dilated "short" oesophagus, probably
secondary to peptic ulceration.

Case 8. Achalasia in association with pulmonary
fibrosis.—A man aged 35 who had had an empyema
drained in 1930 developed "influenza" in November,
1940. He was investigated in 1941 and found to have
mega-oesophagus secondary to achalasia and bilateral
mid-zone infiltration of the lungs. During the next
two years the infiltration waxed and waned on alternate
sides due to recurrent attacks of "pneumonitis." In
January, 1943, he was admitted to hospital with a
history of occasional haemoptyses, minimal cough,
and regurgitation. Clubbing was present. The radi-
ograph (Plate III) shows mid-zone shadows and a
mega-oesophagus. The achalasia was treated by
dilatation of the cardia with a hydrostatic bag. The
lung fields remained unchanged on radiography, but
the oesophageal dilatation was improved.

Case 9. Achalasia associated with lipoid pneu-
monia.—A woman aged 19 complained of three
years' history of sickness after meals. Bougies were
passed at intervals with partial improvement for one
year. She then developed rheumatism in the hands,
and radiograph of the chest showed "abnormality
secondary to dysphagia." Two years later radiography
showed lipoid pneumonia (Plate IV), and bouginage
was continued. One year later, in 1945, a Heller's
operation was performed. The diagnosis of lipoid
pneumonia was made from the radiographs and from
a history of inhalation of liquid paraffin.

DISCUSSION

INCIDENCE.—The frequency with which patho-
logical conditions of the oesophagus are associ-
ated with abnormalities of the lungs is difficult to
assess ; there is little doubt that it is considerably
greater than the number of cases recorded in the
literature suggest. Weens (1944) found that in
his series of fifteen cases of achalasia of the cardia,
five developed pulmonary lesions, but Eley (1948)
only found one example in 60,000 mass radiogra-
phs. The eight patients described by Harring-
ton (1945) and Lahey (1946) give an accurate idea
of the incidence in their series of 349 pharyngeal
diverticula.

AETIOLOGY.—The pulmonary changes associ-
ated with dysphagia are due to aspiration of
oesophageal contents into the bronchial tree. The
mechanism of aspiration varies with different
oesophageal lesions. During normal deglutition
the arrival of a bolus of food in the pharynx sets
up a reflex mechanism, part of which is concerned
with the onwards transmission of the food, and
part of which prevents its ingress through the glottis. A lesion which interferes with this pro-
tective reflex makes aspiration almost inevitable.
When deglutition is not taking place the cough
reflex protects the bronchial tree from contami-
nation by liquid in the pharynx or oesophagus.
Any lesion which may cause the retention of
liquid in this area, in the presence of factors
which tend to dull the cough reflex, renders the
patient liable to "dysphagia pneumonitis."
Carcinomata of the upper third of the oeso-
phagus are particularly prone to produce aspira-
tion, as they are usually of the stenotic type
causing early dysphagia, and are often compli-
icated by recurrent nerve palsies. This combination provides the factors of retention of liquid in the pharynx plus interference with the mechanism normally responsible for closure of the glottis during deglutition. Camiel and Loeve (1941) divided their cases of carcinoma of the oesophagus into upper-third and lower two-thirds groups, for the purpose of discussion of pulmonary complications. They demonstrated that cases in the upper third almost invariably died of aspiration pneumonia (eleven cases out of thirteen) secondary to vocal-cord paralysis. They demonstrated the "spill" in progress during a barium swallow, and considered that it would be possible to do this in all cases of upper-third carcinoma with vocal-cord paralysis. The remaining seventeen cases in their series were in the lower two-thirds of the oesophagus, and of these only two gave rise to "dysphagia pneumonitis."

The two conditions most frequently responsible for the presence of retained liquid in the pharynx are achalasias of the cardia and pharyngeal diverticulum. Even in advanced cases of mega-oesophagus secondary to achalasia the column of fluid seldom reaches the glottis when the patient is in the upright position, and thus it is only liable to be aspirated in recumbency. There are cases on record of patients who had to sleep in the upright position to prevent the choking caused by recumbency (Grey Turner, 1946). The fact that regurgitation occurs under these conditions may be due, as has been suggested by Negus (1942), to incompetence of the cricopharyngeal sphincter.

The method by which the contents of the pharyngeal pouches get into the danger area is probably different. Alteration in position of the head from side to side may squeeze the retained contents of the pouch into the pharynx; during the daytime gravity carries the material away from the glottis, but during sleep it flows upwards.

Sargent and others (1934) produced an hypothesis to account for loss of the normal protective reflex of the glottis. They described a patient who was found to have barium in his right lower lobe after a barium swallow. The patient had been unaware of the passage of liquid into his bronchial tree. These authors suggested that repeated small "insults" dulled the normal reflex and gave rise to aspiration during deglutition.

Brock (1946) explains the predilection of lung abscess for the upper lobes by stating that bronchial embolism occurs when the cough reflex is diminished during sleep, recumbency and gravity being responsible for the position of the abscesses. A large proportion of the cases in his series had upper-lobe disease.

Anaesthesia also causes abolition of the cough reflex. Post-operative bronchial embolism may be due to material aspirated from the oesophagus as well as from the much more common sites above the glottis. This occurred in Case 1, in which the right upper-lobe collapse was due to aspiration of oesophageal contents either during or soon after operation.

Price Thomas (1948) suggests that in order to diminish the risk of aspiration in the pre-operative period the pouch or oesophagus should be cleansed with sodium bicarbonate and then emptied by the patient tipping head downwards just before retiring. Emptying the "sump" immediately pre-operatively, either by this method or by oesophagoscopy as practised by Allison, seems a wise precaution.

**Pathology**

**General.**—Certain aspects of the pathology of the pulmonary lesions secondary to the aspiration of oesophageal contents differ from those secondary to aspiration from the upper respiratory tract and mouth. There are two reasons for this: the aspirations of oesophageal contents frequently take the form of repeated small insults to the bronchial tree, rather than of large "spills"; and secondly the material aspirated is less liable to contain pathogenic organisms. Where there is ulceration of the oesophageal lesions, as in the case of carcinomata, pulmonary complications occur earlier than in achalasia where ulceration is a late and inconstant feature.

The pulmonary lesions with which the patients have presented in this series have been as follows:

<table>
<thead>
<tr>
<th>Disease</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung abscess</td>
<td>18</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>6</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>10</td>
</tr>
<tr>
<td>&quot;Lipoid pneumonia&quot;</td>
<td>4</td>
</tr>
<tr>
<td>&quot;Pneumonitis&quot;</td>
<td>8</td>
</tr>
<tr>
<td>Collapse</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>48</strong></td>
</tr>
</tbody>
</table>

As the aetiology of these lesions is fundamentally the same, no hard-and-fast line should be drawn between them; each is a manifestation of an incident in the course of the disease. As pulmonary abscess formation is the most serious complication, the largest number of cases were discovered at this stage. At least five of these abscesses gave rise to residual fibrosis, and two patients who were discovered at the stage of...
THE PULMONARY COMPLICATIONS OF DYSPHAGIA

TABLE I

SUMMARY OF HISTORIES OF PATIENTS WITH PNEUMONITIS

<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Sex</th>
<th>Cause</th>
<th>Effect</th>
<th>Site</th>
<th>Course and comments</th>
<th>Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cummins and Williams</td>
<td>30</td>
<td>F</td>
<td>Achalasia</td>
<td>Pneumonitis</td>
<td>Right</td>
<td>Acid-fast bacilli, clubbing Cleared completely</td>
<td>Dysphagia</td>
</tr>
<tr>
<td>Bird-Acosta</td>
<td>58</td>
<td>M</td>
<td>Achalasia</td>
<td>Pneumonitis</td>
<td>2nd and 5th right interspace</td>
<td>Cleared 1 year</td>
<td>Recurrent pneumonia</td>
</tr>
<tr>
<td>Weens</td>
<td>18</td>
<td>F</td>
<td>Achalasia</td>
<td>Pneumonitis</td>
<td>Both cases</td>
<td>Cleared completely</td>
<td>Dysphagia 2 years</td>
</tr>
<tr>
<td>Jackson</td>
<td>42</td>
<td>M</td>
<td>Achalasia</td>
<td>Pneumonitis</td>
<td>Right lower lobe</td>
<td></td>
<td>Dysphagia</td>
</tr>
<tr>
<td>Jackson</td>
<td>1½</td>
<td>F</td>
<td>Stricture</td>
<td>Pneumonitis</td>
<td>Diffuse</td>
<td></td>
<td>Pneumonia</td>
</tr>
<tr>
<td>Tucker</td>
<td>13</td>
<td>M</td>
<td>Achalasia</td>
<td>Pneumonitis</td>
<td>Right upper lobe</td>
<td></td>
<td>Chronic cough</td>
</tr>
<tr>
<td>Wooler</td>
<td>16</td>
<td>F</td>
<td>Collapse with pneumonitis</td>
<td>Right mid-zone and right lower lobe</td>
<td></td>
<td>Minimal dysphagia</td>
<td></td>
</tr>
<tr>
<td>Case 2</td>
<td>41</td>
<td>M</td>
<td>Achalasia</td>
<td>Pneumonitis</td>
<td>Mid-zones maximal on right inter-space</td>
<td>Acid-fast bacilli, clubbed, Acid-fast bacilli, cleared</td>
<td></td>
</tr>
<tr>
<td>Case 5</td>
<td>65</td>
<td>M</td>
<td>Diverticulum</td>
<td>Pneumonitis</td>
<td>Right second inter-space</td>
<td></td>
<td>Dysphagia and recurrent pneumonia 8 years</td>
</tr>
</tbody>
</table>

fibrosis gave histories suggestive of abscess formation.

Tracheo-bronchitis.—This lesion can cause recurrent cough long before the development of frank pulmonary lesions, and a low-grade tracheo-bronchitis is inevitable whenever irritating material is repeatedly aspirated in small quantities. This is responsible for the long-standing cough often associated with dysphagia. It has been mentioned specifically by Jackson and Jackson (1933) and by Tucker (1933); the former authors proved the presence of tracheo-bronchitis on bronchoscopy.

"Pneumonitis."—The term "dysphagia pneumonitis" is used in the broad sense to cover all pulmonary lesions associated with dysphagia. In this section "pneumonitis" is taken to mean non-specific inflammation of the interstitial tissue of the lung; it has occurred as a separate entity and also in association with fibrosis (Case 8) and with lung abscesses (Case 6). In nine cases (three of which were associated with acid-fast bacilli in the sputum) it has presented as a separate entity, and the impression given by the histories is that the aspirations responsible were minimal, sporadic, and of short duration (as in Case 2).

"Pneumonitis" has been mentioned as occurring in association with fibrosis on five occasions. It cleared subsequently and must have been due to an exacerbation of the process causing the fibrosis. The features common to these patients were that they all cleared up completely, some after treatment of their dysphagia, and all had had single or recurrent attacks of "pneumonia" with fever, cough, sputum, and pain in the chest. The distribution of the lesions was predominantly right-sided and in the mid-zones. The original radiograph of Case 2 is similar to those published by Waring and Rilance, both of whose patients went on to fibrosis. In Case 2 complete clearing took place, and the relative shortness of history and paucity of symptoms accounts for this difference, although it is possible that in all cases of "pneumonitis" some degree of fibrosis takes place, but that only in old-standing ones is it sufficiently dense to be radio-opaque.

Pulmonary abscess.—A major spill of material from the oesophagus, in some cases containing virulent organisms, is the probable cause of development of pulmonary abscesses. Of the eighteen cases collected in this series three have been multiple and ten have been described as foetid. The localization of the lung abscesses has been as follows:

- Right upper lobe ... ... ... 11
- Right middle lobe ... ... ... 0
- Right lower lobe ... ... ... 5
- Left upper lobe ... ... ... 1
- Left lower lobe ... ... ... 3

Of those occurring in the lower lobes four have been in the apical segments; that is, of the total twenty abscesses in eighteen cases, sixteen have occurred in the sites most liable to bronchial embolus during recumbency.

The positions in which a group of patients slept were recorded twice a night in order to see whether
the predominance of right-sided lesions could be accounted for by gravity. The results were as follows:

<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Sex</th>
<th>Cause</th>
<th>Effect</th>
<th>Site</th>
<th>Course and comments</th>
<th>Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vinson</td>
<td>42</td>
<td>M</td>
<td>Achalasia</td>
<td>Putrid abscess</td>
<td>Right lobe</td>
<td>Fibrosis</td>
<td>10 years' dysphagia</td>
</tr>
<tr>
<td>Sampson</td>
<td>40</td>
<td>M</td>
<td>Achalasia</td>
<td>Putrid abscess</td>
<td>Right lobe</td>
<td>Asymptomatic; lung stone, acid-fast bacilli, fibrosis</td>
<td>Lung abscess</td>
</tr>
<tr>
<td>Rothstein and Pirkle</td>
<td>41</td>
<td>F</td>
<td>Achalasia</td>
<td>Abscess</td>
<td>Right lobe; left lower lobe</td>
<td>10 years' dysphagia and regurgitation</td>
<td>Dysphagia and regurgitation</td>
</tr>
<tr>
<td>Keefer</td>
<td>63</td>
<td>M</td>
<td>Carcinoma</td>
<td>Abscess</td>
<td>Right lobe</td>
<td>No full description</td>
<td>Dysphagia</td>
</tr>
<tr>
<td>Baldwin</td>
<td>24</td>
<td>F</td>
<td>Achalasia</td>
<td>Abscess</td>
<td>Right lobe</td>
<td>Acid-fast bacilli, fibrosis, 10 years minimal dysphagia</td>
<td>Lung abscess</td>
</tr>
<tr>
<td>Weens</td>
<td>29</td>
<td>M</td>
<td>Achalasia</td>
<td>Foetid abscess</td>
<td>Right lobe</td>
<td>10 years' dysphagia</td>
<td>Lung abscess</td>
</tr>
<tr>
<td>Bird-Acosta</td>
<td>53</td>
<td>F</td>
<td>Achalasia</td>
<td>Foetid abscess</td>
<td>Right lobe</td>
<td>Fibrosis - &quot;Silent&quot; dysphagia</td>
<td>Dysphagia 9 years</td>
</tr>
<tr>
<td>Lalleman and Ordioni</td>
<td>38</td>
<td>F</td>
<td>Achalasia</td>
<td>Non-foetid abscess</td>
<td>Right lobe</td>
<td>20 years bronchitis, cleared abscess</td>
<td>Dysphagia; short history</td>
</tr>
<tr>
<td>Sergent</td>
<td>64</td>
<td>M</td>
<td>Stricture</td>
<td>Foetid abscess</td>
<td>Right lobe</td>
<td>No fistula</td>
<td>Dysphagia 15 years and regurgitation</td>
</tr>
<tr>
<td>Mallory</td>
<td>45</td>
<td>M</td>
<td>Carcinoma</td>
<td>Foetid abscess</td>
<td>Right lobe</td>
<td>Dysphagia, recurrent productive cough</td>
<td>Dysphagia 16 years</td>
</tr>
<tr>
<td>Pruvost and Leblanc and Chabrol and Cachin Sergent</td>
<td>48</td>
<td>M</td>
<td>Diverticulum</td>
<td>Foetid abscess</td>
<td>Right lobe</td>
<td>Cleared up</td>
<td>Lung abscess</td>
</tr>
<tr>
<td>Walle</td>
<td>7</td>
<td>M</td>
<td>Stricture</td>
<td>Foetid abscess</td>
<td>Right lobe</td>
<td>Dysphagia and regurgitation</td>
<td>Dysphagia and regurgitation</td>
</tr>
<tr>
<td>Jackson</td>
<td>44</td>
<td>M</td>
<td>Stricture</td>
<td>Foetid abscess</td>
<td>Right lobe</td>
<td>Cleared up</td>
<td>Bronchopneumonia</td>
</tr>
<tr>
<td>Case 6</td>
<td>46</td>
<td>M</td>
<td>Diverticulum</td>
<td>Abscess collapse</td>
<td>Left upper lobe</td>
<td>Fibrosis - fibrosis - &quot;silent&quot; dysphagia</td>
<td>Dysphagia 1 year</td>
</tr>
<tr>
<td>Case 4</td>
<td>69</td>
<td>M</td>
<td>Carcinoma</td>
<td>Abscess</td>
<td>Left dorsal lobe</td>
<td></td>
<td>Lung abscess</td>
</tr>
</tbody>
</table>

the straightness of the right main bronchus tends to make aspirated material go to the right side when the patient lies on his back, and thus lesions on the right side should be twice as common as those on the left. In fact the proportion is higher.

In this series not one of the pulmonary abscesses had to be drained; of those in which details were given, three died, five led to fibrosis with disappearance of the cavity (Case 4), two led to chronic abscesses (one with a "lung stone" but no symptoms), and five cleared up completely. The clinical course is different from the other types of aspiration abscess in which chronicity is so much more common.

Abscess formation is an incident in the course of the disease. Where there has been a very long history the likely end result will be fibrosis; where the history has been short complete clearing appears to be more likely.

**Pulmonary fibrosis.**—A prolonged series of aspirations extending over many years gives rise to pulmonary fibrosis, and it is the most frequent end result of dysphagia pneumonitis. Ten cases were first diagnosed at this stage, and five of the pulmonary abscesses
PLATE I.—(a) Case 1, gross mega-oesophagus with mid-zone fibrosis.  (b) Case 1, after collapse of right lower lobe.
(c) Case 2, mega-oesophagus with bilateral mid-zone infiltration with suggestion of cavitation on right side.
(d) Case 2, almost completely clear three years later.
Plate IV.—(a) Case 9, lipoid pneumonia.  (b) Chronic pulmonary tuberculosis in a case which had a Heller's operation for cardiospasm.  Left diaphragm remains high.
THE PULMONARY COMPLICATIONS OF DYSPHAGIA

TABLE III

SUMMARY OF HISTORIES OF PATIENTS WITH PULMONARY FIBROSIS AND LIPOID PNEUMONIA

<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Sex</th>
<th>Cause</th>
<th>Effect</th>
<th>Site</th>
<th>Course and comments</th>
<th>Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hawes and Soule</td>
<td>57</td>
<td>M</td>
<td>Achalasia</td>
<td>Fibrosis</td>
<td>Mid-zone maxillary right</td>
<td>One haemoptysis</td>
<td>Dysphagia and regurgitation</td>
</tr>
<tr>
<td>Hawes and Soule</td>
<td>31</td>
<td>M</td>
<td>Achalasia</td>
<td>Fibrosis</td>
<td>Mid-zone</td>
<td>No pulmonary symptoms</td>
<td>Dysphagia and regurgitation</td>
</tr>
<tr>
<td>Weens</td>
<td>37</td>
<td>F</td>
<td>Achalasia</td>
<td>Fibrosis</td>
<td>Maxillary right</td>
<td>No dysphagia</td>
<td>Dysphagia and regurgitation</td>
</tr>
<tr>
<td>Weens</td>
<td>26</td>
<td>F</td>
<td>Achalasia</td>
<td>Fibrosis</td>
<td>Right maxillary zone both</td>
<td>Clipping</td>
<td>Dysphagia and regurgitation</td>
</tr>
<tr>
<td>Bird-Acosta</td>
<td>39</td>
<td>M</td>
<td>Achalasia</td>
<td>Fibrosis</td>
<td>Mid-zone</td>
<td>Aspirated via bronchoscope</td>
<td>Productive cough 2 yrs</td>
</tr>
<tr>
<td>Waring and Rilance</td>
<td>65</td>
<td>F</td>
<td>Achalasia</td>
<td>Fibrosis</td>
<td>Maxillary right</td>
<td>Acid-fast bacilli</td>
<td>Dysphagia and regurgitation 19 yrs.</td>
</tr>
<tr>
<td>Robson and Wilkinson</td>
<td>13</td>
<td>F</td>
<td>Achalasia</td>
<td>Fibrosis</td>
<td>All zones maxillary right</td>
<td>Post-mortem confirmation</td>
<td>Dysphagia and regurgitation 6 yrs</td>
</tr>
<tr>
<td>Heaton</td>
<td>51</td>
<td>F</td>
<td>Achalasia</td>
<td>Fibrosis</td>
<td>Mid-zones maxillary right</td>
<td>Previous history of abscess</td>
<td>Dysphagia and regurgitation 30 yrs.</td>
</tr>
<tr>
<td>Case 3</td>
<td>50</td>
<td>M</td>
<td>Achalasia</td>
<td>Fibrosis</td>
<td>Mid-zones maxillary right</td>
<td>Recent bronchitis</td>
<td>Dysphagia and regurgitation 20 yrs.</td>
</tr>
<tr>
<td>Case 8</td>
<td>35</td>
<td>M</td>
<td>Achalasia</td>
<td>Fibrosis</td>
<td>Mid-zones maxillary right</td>
<td>Mega-oesophagus and regurgitation</td>
<td>Cough and dyspnoea</td>
</tr>
<tr>
<td>Case 9</td>
<td>19</td>
<td>F</td>
<td>Achalasia</td>
<td>Lipoid pneumonia</td>
<td>Mid-zones</td>
<td>History of use of liquid paraffin</td>
<td>Dysphagia 3 yrs</td>
</tr>
<tr>
<td>Thomas</td>
<td>32</td>
<td>M</td>
<td>Lipoid pneumonia</td>
<td>Lipoid pneumonia</td>
<td>Right mid-zones left base</td>
<td>Recurrent pneumonia</td>
<td>Dysphagia 4 yrs</td>
</tr>
<tr>
<td>Wooler</td>
<td>31</td>
<td>F</td>
<td>Lipoid pneumonia</td>
<td>Lipoid pneumonia</td>
<td>Left lower lobe</td>
<td>Due to liquid paraffin Improved, due to excess milk</td>
<td>Dysphagia and regurgitation “Bronchial carcinoma”</td>
</tr>
<tr>
<td>Tcherthoff</td>
<td>77</td>
<td>M</td>
<td>Diverticulum</td>
<td>Lipoid pneumonia</td>
<td>Left lower lobe</td>
<td>Lipoid pneumonia</td>
<td></td>
</tr>
</tbody>
</table>

led to residual fibrosis. Heaton's (1948) case had an old history of lung abscess, and several others either showed areas on their radiographs suggestive of cavity formation or had histories of febrile attacks associated with cough, sputum, and haemoptyses. The diagnosis has been radiological, the presence of fibrosis alone often giving rise to no symptoms or to a non-productive cough, febrile incidents being associated with superadded "pneumonitis" or abscess formation.

The characteristic radiological picture is one of bilateral mid-zone infiltration greater on the right than on the left, and often sharply demarcated below by the fissures (for example, Plates IIIa and IVa) and maximal at the hilum.

The patient described by Robson and Wilkinson (1946) died after the performance of a Heller's operation; at necropsy the lungs showed widespread nodular fibrosis of all zones, reaching a maximum in the right upper lobe; this suggests that fibrosis may be more diffuse than is apparent radiologically. Fibrosis alone in this series had not given rise to much disability; dyspnoea and clubbing are recorded, but no cases have developed right heart failure.

**Lipoid pneumonia.**—Tcherthoff (1941) reviewed the 140 published cases of this disease; he stated that all were secondary to some form of defective swallowing and gave a long history of injections of some oily substance; mineral oils in particular were responsible. In the final stages mid-zone fibrosis with an excess of oil in the lungs develops. He described one case in which the disease followed excessive ingestion of milk in a man of 77 suffering from a pharyngeal diverticulum. Two others have been described in association with achalasia; Case 9 in this series makes the total up to four. The radiological opacities which are in the mid-zones (Plate IVa) are mainly due to fibrosis and the oily deposits are coincidental. These cases are examples of pulmonary fibrosis secondary to dysphagia with the added factor of a high fat content in the material retained in the oesophagus; sooner or later aspiration of this fatty material leads to the development of lipoid pneumonia.

**Collapse.**—There are two possible causes of collapse; a large "bolus" may be aspirated causing flooding of a bronchus; this was responsible for the upper-lobe and lower-lobe collapse in Case 1. The alternative is that a bronchus becomes occluded by
the dilated oesophagus pressing upon it from behind. Plummer and Vinson (1921) record cases with post-prandial dyspnoea which in their opinion were due to dilatation of the oesophagus, and Bird-Acosta records an enormous mega-oesophagus causing a bulge in the posterior wall of the trachea which was observed at bronchoscopy. Wooler is of the opinion that the latter mechanism is responsible for the collapse, as re-expansion took place after dilatation of the cardiac sphincter in his case.

**CLINICAL FEATURES**

Grey Turner (1946) has stated that achalasia of the cardia may present as a chronic cough. Examples have occurred in this series which presented as pulmonary disease with completely "silent" dysphagia, and there were others in which the pulmonary lesions were revealed only on routine radiography during investigation of dysphagia. Many intermediate examples have been reported. The patients in this series have been divided into three clinical groups: those who presented with major pulmonary and minor dysphagic symptoms; those who presented as dysphagia with subsequent development of pulmonary lesions; and a few cases in which these symptoms appeared almost simultaneously.

The patients with "silent dysphagia" constitute an important group. They may be defined as those who have a definite obstruction to normal deglutition but who have had no subjective difficulty in swallowing. There have been eight described, and unless it is recognized that mechanical obstruction of the oesophagus sufficient to lead to aspiration with consequent pulmonary lesions can exist without symptoms of which the patient is conscious, the real aetiology of the pulmonary lesion may be overlooked. Cases 4 and 7 are examples of "silent dysphagia," but in those described by Pruvost and Leblanc (1935) and Lallemant and Ordioni (1945) the oesophageal lesions were discovered fortuituously when transnasal bronchoscopy was performed. In each, some of the iodized oil went past the glottis; in the first a hitherto unsuspected pharyngeal diverticulum, in the second an achalasia of the cardia which had been symptomless, became apparent. Both these patients had presented with right upper-lobe lung abscesses.

Asymptomatic pulmonary lesions are of less importance, but the presence of radiological changes in the lung fields in association with dysphagia makes active treatment of the latter more necessary in order that serious pulmonary lesions may be avoided. The presence of bilateral mid-zone fibrosis in association with achalasia was recognized in a case described by Hawes and Soule (1945), and it was not until two years later that an haemoptysis with purulent foetid sputum developed.

Eighteen patients presented with predominantly pulmonary symptoms. The eight "silent dysphagias" are included in this number, and the remaining ten had varying degrees of dysphagia, but often they had become accustomed to dysphagia and only admitted its presence on questioning.

A long-standing cough in association with the dysphagia has been a frequent feature, and it may be the only clinical indication of a pulmonary lesion. Jackson and Jackson (1933) are of the opinion that there is a "tussive centre" at the lower end of the oesophagus, and that any lesion or pressure from a column of liquid on this area may be responsible for a reflex cough. It is also probable that even in the early stages of achalasia small aspirations take place which cause tracheobronchitis and an irritating cough. In the four cases in which the onset of pulmonary and dysphagic symptoms has been simultaneous, this mechanism may have been responsible for the early development of the cough.

The remaining twenty-six cases presented with predominantly dysphagic symptoms, although in several the onset of the pulmonary disease was responsible for their seeing a doctor. In four the lung lesion was symptomless and was discovered by the radiologist.

Freeman (1940), discussing the symptomatology of achalasia, states that the average length of history is five years and that the predominant symptoms are dysphagia, pain, and regurgitation. In this series the dysphagia has often been minimal or periodical, despite the presence of enormous mega-oesophagus. Regurgitation, which Freeman regards as a late symptom, has been common; it has often been associated with nocturnal cough and choking, and has been recorded eighteen times. Plummer and Vinson (1921) mention regurgitation as occurring in eighty-nine of 301 cases of achalasia.

Where the length of history of dysphagia in patients suffering from achalasia and with pharyngeal diverticula has been given, it has averaged ten years prior to the onset of pulmonary symptoms. Pulmonary lesions may therefore be regarded as a late complication; but aspiration phenomena complicating carcinomata and strictures appear more rapidly.
THE PULMONARY COMPLICATIONS OF DYSPHAGIA

TABLE IV
SUMMARY OF HISTORIES OF PATIENTS SUFFERING FROM BRONCHIECTASIS

<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Sex</th>
<th>Cause</th>
<th>Effect</th>
<th>Site</th>
<th>Course and comments</th>
<th>Presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schrier</td>
<td>12</td>
<td>F</td>
<td>Achalasia</td>
<td>Bronchiectasis</td>
<td>Right middle and lower</td>
<td>Silent dysphagia</td>
<td>Productive cough 6 years</td>
</tr>
<tr>
<td>Wooler</td>
<td>18</td>
<td>F</td>
<td>Achalasia</td>
<td>Bronchiectasis</td>
<td>Bilateral</td>
<td>Minimal dysphagia</td>
<td>Pneumonia, cough 5 years</td>
</tr>
<tr>
<td>Weens</td>
<td>28</td>
<td>F</td>
<td>Achalasia</td>
<td>Bronchiectasis</td>
<td>Bilateral lower lobes</td>
<td>Mega-oesophagus</td>
<td>Productive cough 15 years</td>
</tr>
<tr>
<td>Reeke</td>
<td>19</td>
<td>F</td>
<td>Achalasia</td>
<td>Bronchiectasis</td>
<td>Bilateral lower lobes</td>
<td>Simultaneous onset</td>
<td>Dysphagia and regurgitation since infancy</td>
</tr>
<tr>
<td>Chabrol and Cachin</td>
<td>46</td>
<td>M</td>
<td>Peptic ulcer of oesophagus</td>
<td>Bronchiectasis</td>
<td>Right lower lobe</td>
<td>Coughs 1 year</td>
<td>Dysphagia 2 years</td>
</tr>
<tr>
<td>Case 7</td>
<td>6</td>
<td>F</td>
<td>Achalasia</td>
<td>Bronchiectasis</td>
<td>Right middle upper lower lobe, left lingual</td>
<td>Silent dysphagia</td>
<td>Pneumonia</td>
</tr>
</tbody>
</table>

Two groups appear to be clinically separate from the majority of the cases. Those which have been associated with bronchiectasis have almost all occurred in young people (five out of six), and in several it was difficult to determine whether or not the dysphagia, which in three was almost silent (Case 7), preceded the bronchiectasis. The average age for onset of symptoms was six years and of presentation 20, whereas in the remainder of this series the average age of presentation was 43 years. The bronchiectasis had the usual distribution in the lower and middle lobes, and it is possible that aspiration which in adults is liable to produce lung abscess gives rise to bronchiectasis in younger patients. In the second group the pulmonary lesions have been associated with acid-fast bacilli in the sputum. It is not stated whether or not the three cases of pulmonary tuberculosis referred to by Plummer and Vinson in relation to achalasia were confirmed on culture and guinea-pig inoculation. The first case in which acid-fast bacilli were found and were recognized as being non-tuberculous was described by Cummins and Williams (1933); since then there have been three more in the literature. Cases 2 and 5 in this series make the total six. All except Case 5 were secondary to achalasia.

The bacilli which Cummins and Williams isolated were non-pathogenic to guinea-pigs. In Baldwin's (1942) case the acid-fast bacilli were non-pathogenic and were found in both vomit and sputum. A "tuberculin" was made from the organisms to which the patient gave a markedly positive reaction, but despite this the organism was considered to be non-pathogenic and was thought to be related to the Timothy grass bacillus. Warring and Rilance (1943) described the next case; the clinical and radiological findings were similar to Baldwin's and to Case 2; the "tuberculin" reaction was positive. The third case was reported by Rothstein and Pirkle (1946); the patient was "tuberculin" positive, but negative to the standard tuberculin tests. The radiographs of two of the patients in whose sputa acid-fast bacilli were found showed cavitation; in the other two the films were similar to that shown in Plate IIIa.

The feature which originally led to the suspicion that the patients were not tuberculous was the presence of few or occasional "showers" of organisms, with persistent copious sputum. Where follow-ups are available the pulmonary lesions have either cleared up completely or have left residual fibrosis in the mid-zones. The cough and sputum have been considerably relieved by the treatment of the dysphagia. Case 2, diagnosed at first as suffering from pulmonary tuberculosis, now shows a clear radiograph (Plate IIb), and was certainly never tuberculous. Case 5 is still doubtful; the acid-fast bacilli were found on only one occasion and were not cultured. He is almost symptom-free and is assumed to be non-tuberculous.

The acid-fast bacilli are derived from the oesophagus, possibly from fruit skins; they are aspirated into the bronchial tree and are non-
pathogenic. The pulmonary lesions they produce are similar to those of pneumonitis in which no acid-fast bacilli have been demonstrated, and the finding of the organism is coincidental. The association of pulmonary tuberculosis with mega-oesophagus is sufficiently uncommon to have been reported when it has occurred, and it is suggested that when aspiration pneumonitis appears the diagnosis of tuberculosis should be confirmed by culture or guinea-pig inoculation. Achalasia and pulmonary tuberculosis can co-exist as in the case illustrated (Plate IVb); this man had had six years of recurrent pulmonary symptoms prior to an acute onset of achalasia.

The causes of dysphagia and their consequences in this series are shown in Table V.

**TABLE V**

**CAUSES AND CONSEQUENCES OF DYSPHAGIA IN THE PRESENT SERIES**

<table>
<thead>
<tr>
<th></th>
<th>Number</th>
<th>Abscess</th>
<th>Bronchiectasis</th>
<th>Fibrosis</th>
<th>Pneumonitis</th>
<th>Collapse</th>
<th>Lipoid pneumonia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Achalasia</td>
<td>34</td>
<td>9</td>
<td>4</td>
<td>10</td>
<td>6</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Pharyngeal diverticulum</td>
<td>5</td>
<td>3</td>
<td>3</td>
<td>10</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Carcinoma of oesophagus</td>
<td>3</td>
<td>3</td>
<td>2</td>
<td>10</td>
<td>8</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Various strictures</td>
<td>6</td>
<td>3</td>
<td>3</td>
<td>10</td>
<td>8</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>48</td>
<td>18</td>
<td>6</td>
<td>10</td>
<td>8</td>
<td>2</td>
<td>4</td>
</tr>
</tbody>
</table>

**REFERENCES**


Thomas, C. Price (1948). Personal communication.


The Pulmonary Complications of Dysphagia

J. R. Belcher

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