LUNG LESIONS IN RHEUMATOID ARTHRITIS

BY

A. ARONOFF, M.D., B.Sc.

E. G. L. BYWATERS, M.B., F.R.C.P.

AND

G. R. FEARNLEY, M.D., M.R.C.P.

From the Department of Medicine, Postgraduate Medical School, London, W.12

Pulmonary changes, varying from pleural effusions, pleural thickening, mottling, reticulation, and chronic fibrosis have been observed in patients with "collagen diseases" (Weir, 1939; Elkeles, 1944; Miller and Daley, 1946; Tumulty and Harvey, 1949; Murphy *et al.*, 1941; Weiss *et al.*, 1943; Littler, 1953). A number of authors have noted similar changes in patients with rheumatoid arthritis (Ellman, 1947; Ellman and Ball, 1948; Hart, 1948; Leys and Swift, 1949; Schlesinger, 1949; Bloom and Rubin, 1950; Christie, 1954; Harris, 1954), where no other specific cause for these changes could be uncovered. Ellman and Ball (1948) coined the terms "rheumatoid lung" and "rheumatoid disease" to indicate that these pulmonary changes were another manifestation of connective-tissue involvement similar to that seen in the joints and in nodules of patients with rheumatoid arthritis, "thus indicating the widespread nature of the process in this condition."

More recently Ellman and Cudkowicz (1954) described a group of signs and symptoms in these patients which they labelled the "rheumatoid lung syndrome," characterized by cough, fever, and dyspnoea, frequently associated with an exacerbation of arthralgia. The x-ray changes observed were multiform, as described in other collagen diseases. The degree of dyspnoea was often greater than the clinical signs and x-ray findings suggested. Fever, leucocytosis, and arthralgia might or might not accompany the onset of pulmonary symptoms. The signs and symptoms were unaffected by anti-rheumatic or antibiotic therapy and resolved slowly, leaving residual fibrosis and pleural thickening.

Caplan (1953) described, in coal-miners, a syndrome of nodular lung fibrosis which was associated with rheumatoid arthritis in more than 50% of cases; and it was also suggested that the prevalence of rheumatoid arthritis was increased amongst men with complicated pneumoconiosis (Miall *et al.*, 1953). In a further study Miall (1955) has raised again the question of an association between rheumatoid arthritis and tuberculosis. His data suggest that a relationship between the two diseases may exist, and that this relationship is due to some alteration in tissue reaction which is often of genetic origin and perhaps a component of the so-called "rheumatoid diathesis."

In our experience at the Postgraduate Medical School over the last 15 years we have not noticed any specific lung lesions in patients with rheumatoid arthritis, and we have not encountered any instances of the "rheumatoid lung syndrome." We have learnt to recognize lung lesions in allied diseases such as the fibrosis of scleroderma (Shuford *et al.*, 1953) and the interstitial infiltrations of periarteritis nodosa and lupus erythema-

tosus (Rakov and Taylor, 1942). We recognize, but have not personally encountered, the very rare occurrence of rheumatoid nodules in the lung, of the type described by Raven *et al.* (1948). We have also, with Dr. Griffith Pugh, investigated a patient with a lung lesion associated with Sjögren's syndrome, described by Ellman and Weber (1949). Rheumatoid arthritis is a disease that primarily affects the joints—a local disease, affecting some joints and not others, whose economic and human importance depends upon its predilection for synovial membrane. It is a common disease affecting, according to Kellgren *et al.* (1953), approximately 2% in some samples of the population, and many of our patients have had non-specific lung lesions. We have therefore analysed our clinical and pathological experience to determine how often lung lesions occur in patients with rheumatoid arthritis and to see how this compares with the incidence in other hospital material.

Results

Table I shows that in a consecutively collected series of 253 rheumatoid arthritis patients coming to hospital between 1939 and 1948 and followed to the present time the following clinical diagnoses were made. In 180 there was no evidence of chest disease clinically. In the remainder the most common lesions observed were bronchitis (including pneumonia), bronchitis and emphysema, emphysema alone, bronchiectasis, and old apical tuberculosis. Basal rales were recorded in nine cases, indicative of congestive failure or

TABLE I.—Clinical Diagnosis Pertaining to Lungs (Rheumatoid Arthritis Follow-up Series, 1939-48) 253 Cases (In a Number of Cases Several Diagnoses were Recorded)

No apparent disease	180
Bronchitis alone	26
" + asthma	3
" + emphysema	12
Emphysema alone	8
Bronchiectasis	6
Pulmonary tuberculosis	4
" fibrosis	3
{ Bilateral	3
{ Unilateral	3
{ Honeycombing ¹	3
{ Reticulation ²	2
Silicosis	2
Xanthomatosis ³	1
Basal rales (congestive)	9
Carcinoma	2
Old pleuritis	2
Pulmonary embolus	1

¹ A. D. (Case 2). H. K. (chronic bronchitis and emphysema). A. W. (infiltration due to chronic lymphatic leukaemia).

² D. S. (Case 4). M. C. (rheumatoid arthritis with some features resembling acute lupus erythematosus).

³ E. P. (Case 3).

TABLE II.—Radiological Lung Changes During Total Period of Hospital Observation

	R.A.F.U.	Controls
Total cases	253	
Cases x-rayed	130	130
not x-rayed	123	—
No lesions	68	76
Bronchitis ⁴	20	10
Emphysema	13	4
Bronchiectasis	4	2
Unilateral apical tuberculosis	8	15
Bilateral	6	7
Pulmonary fibrosis	2	—
{ Reticulation ⁵	4	—
{ Honeycombing ⁶	2	—
Silicosis	2	—
Pleural thickening	8	4
" effusion	3	4
Pulmonary congestion	7	2
Carcinoma ⁷	2	8
Pulmonary embolus	1	0
Spontaneous pneumothorax	0	1
Haemosiderosis	0	1

⁴ Includes "pneumonitis" (infective, with subsequent clearing).

⁵ D. S. (Case 4). M. C. (rheumatoid arthritis, with some features resembling acute lupus erythematosus).

⁶ A. D. (Case 2). E. P. (Case 3). H. K. (chronic bronchitis and emphysema). A. W. (infiltration due to lymphatic leukaemia).

⁷ Includes metastatic carcinoma to lung.

hypostatic congestive changes. This result does not impress us as very different from what might be expected in a general hospital population.

Examination of the x-ray films in 130 cases of this series in which radiological examination of the chest was indicated on clinical grounds revealed a variety of miscellaneous conditions (Table II), but most of them could be accounted for by a current episode of non-specific pulmonary infection, congestive failure, or established underlying disease such as silicosis, old tuberculous infection, or neoplastic disease. Six cases were diagnosed as pulmonary fibrosis, two cases showing reticulation pattern and four showing marked honeycombing. Of the films showing honeycombing, one (A. W.) proved to be lymphatic infiltration in a case of chronic lymphatic leukaemia, and another (H. K.) was due to chronic bronchitis and bronchiectasis with bullous formation and fibrous-tissue reaction. Of the reticulations, one case (M. C.) was due to pulmonary infiltration in a case of possible disseminated lupus erythematosus with rheumatoid arthritis (referred to in the post-mortem series). The three remaining cases presented some difficulty, and are detailed later. In none of these rheumatoid patients with clinical or radiological lesions could we establish any temporal correlation with an exacerbation of their arthritis process.

For comparison with this rheumatoid series of x-ray films we chose a similar number of films from consecutive non-rheumatoid arthritis patients sent up for chest x-ray examination between April and June, 1954, excluding hospital staff,

TABLE III.—*Post-mortem Lung Lesions in 42 Cases of Rheumatoid Arthritis, 1936-54*

	R.A.	Controls
No lesions	11	23
Bronchitis	16	7
Emphysema	10	9
Bilateral apical lesions	15	4
Unilateral "	3	3
Bilateral adhesions	18	9
Unilateral "	9	3
Miliary tuberculosis	1	0
Silicosis	1	0
Bronchiectasis	5	0
Terminal bronchopneumonia	9	6
" carcinoma	4	14
Total cases	42	42

tuberculosis and antenatal clinic cases, and patients below the age of 20. The spring control period would, if anything, enhance any undue incidence of lung lesions in the rheumatoid arthritis series. However, it will be seen from Table II that the overall incidence of lung disease was slightly greater among the rheumatoid patients than among controls selected in this way. The same sort of changes were, however, seen in both series and in similar proportions.

Finally, we have examined the lung changes found at necropsy in 42 patients with rheumatoid arthritis dying between 1936 and 1954 in this hospital. Table III shows the lung lesions listed by the pathologist and based on the macroscopic and, where indicated, the microscopical examination of the lungs. (We have excluded 12 cases—8 with ankylosing spondylitis; 3 with rheumatic heart disease, thought to be examples of Jaccoud's syndrome; and 1 case of arthritis with features of both rheumatoid arthritis and disseminated lupus erythematosus in which malignant hypertension and uraemia were also present, and in which there were fibrinous exudates in the lung fields.) Again no specific lung lesion was found and no nodules were seen.

For comparison, we assembled a control series by selecting from the serially num-

bered post-mortem examinations the next necropsied patient of the same age, decade, and sex as each rheumatoid arthritis patient.

The most frequent lesions observed in both series (Table III) were bronchitis, emphysema, apical lesions, pleural adhesions, and bronchiectasis. Again, there seems to be a higher incidence of all lung lesions, with the exception of emphysema and carcinoma, in the rheumatoid arthritis series than in the non-rheumatoid arthritis group. The frequency, however, with which these main lesions appear relative to each other seems to be the same in both series, with the possible exception of bronchiectasis.

We conclude that there is no specific lung lesion common enough to appear in 42 consecutive post-mortem cases of rheumatoid arthritis—a finding similar to that of Baggenstoss and Rosenberg (1943) from a survey of 30 such cases. The apparently increased incidence of bronchitis, bronchiectasis, and old apical tuberculous lesions in the rheumatoid patient is quite striking, and may be due to a real increased susceptibility of these patients to pulmonary infections, as suggested by Hollander (1953). Alternatively, this apparently higher incidence in the rheumatoid arthritis series than in our control material may be because people with two diseases are sent to hospital more frequently than people with one, particularly if one of the diseases makes them very difficult to nurse adequately under home conditions. Finally, it must be borne in mind that all data based on hospital patients may be invalidated, as Mainland (1953) has pointed out, not only by the conditions of selection of controls but also by the unconscious selection which operates every time any patient is sent to a particular hospital.

Case Histories Illustrating Diagnostic Difficulties

The following case histories indicate the greatest difficulties we have encountered in arriving at a satisfactory diagnosis of the pulmonary changes. (Cases 2, 3, and 4 were in the follow-up series 1939-48.)

Case 1

J. M., a male packer, was quite well until 1941, when (aged 41) he developed rheumatoid arthritis involving the hands, feet, and shoulders. His arthritis was never severe and was easily controlled with aspirin and codeine. In 1950 a typical rheumatoid nodule appeared on the posterior aspect of the left ulna. In 1951 pulmonary symptoms first showed when he was admitted to hospital for an acute episode of fever, cough, and pleuritic pain. Left pleural effusion and pericardial effusion were diagnosed. E.S.R. on admission was 101 mm. an hour (Westergren) and fell to 34 mm. on discharge two months later. Investigations for

FIG. 1.—J. M., Case 1. Postero-anterior and left lateral chest x-ray films taken in March, 1954, showing what was then believed to be pleural thickening but which subsequent investigation indicated to be a collapsed lower lobe due to bronchial carcinoma.

tuberculosis and carcinoma were negative. There was no associated exacerbation of his arthritis at this time. He was discharged relatively symptom-free with x-ray signs thought to be due to residual pleural thickening at the left base.

He had a slight cough, which increased in severity and then became productive of mucopurulent sputum: four to six months after discharge he developed persistent streaky haemoptyses and increasing dyspnoea on exertion. In December, 1953, he was reinvestigated, but again no cause was found for the x-ray appearances and haemoptyses. In May, 1954, haemoptysis and dyspnoea became progressively severe. At this time he had a mild exacerbation of his arthritis. In September, 1954, he was readmitted to hospital because of haemoptysis, chest pain on coughing, and weight loss.

A chest x-ray film (identical with that taken in March, 1954, see Fig. 1) showed what was then believed to be left pleural thickening with fine reticulation in both lung fields, which had progressed slightly since 1951. Other investigations: E.S.R., 55 mm.; differential agglutination titre (D.A.T.), 1:64. A biopsied nodule (right ulna) showed characteristic changes of rheumatoid arthritis. Studies for L.E. cells were negative, as were repeated cultures for tubercle bacilli. Mantoux positive 1:1,000. A bronchogram revealed slight dilatation of bronchi and some compression of left posterior segment of the bronchus to the left lower lobe. Bronchoscopy finally revealed a squamous-cell carcinoma in the left lower lobe bronchus, which was confirmed by biopsy. Re-examination of the chest film at this time indicated that what had been considered to be pleural thickening was probably collapse of the left lower lobe due to bronchial neoplasm.

Case 2

A.D. first developed rheumatoid arthritis in 1941 at the age of 44, involving ankles, knees, arms, and wrists. He had been a sign-writer since 1914. In May, 1942, while in hospital for treatment, no pulmonary symptoms were found. E.S.R. 19 mm. an hour. In September, 1947, a thyroid adenoma was removed. Chest x-ray examination at this time revealed moderate bilateral apical fibrosis with emphysematous changes at both bases. The process was well established and must have been developing for some time. He had only a slight cough and minimal shortness of breath. His arthritis in the preceding years had been mild and was considered to be "mildly active" when seen in 1947. In August, 1949, the rheumatoid arthritis was slightly worse; D.A.T., 1:64. In September, 1951, the arthritis was still

troublesome, and chest x-ray examination revealed progressive pulmonary fibrosis. He was given a 32-day course of corticotrophin, which had only a transient effect on his arthritis and no effect whatever on his pulmonary lesions.

He was readmitted in August, 1953, with increasing dyspnoea, cyanosis, and clubbing of the fingers. An x-ray film (Fig. 2) showed apical fibrosis with honeycombing throughout both lung fields. E.S.R., 60-80 mm. an hour; Mantoux positive 1:1,000. Search for acid-fast organisms and malignant cells was persistently negative. ¹³¹I uptake over lungs was nil, ruling out a possible metastatic thyroid lesion. Biopsy of rheumatoid nodule showed typical findings. He was discharged with a diagnosis of idiopathic pulmonary fibrosis. He died soon after, outside hospital. A necropsy was not done.

Case 3

E.P., aged 58, was first admitted in 1949 with melaena and haematemesis, and later the same year for coronary thrombosis. She had severe rheumatoid arthritis, which had been present for 17 years. There were palpable glands in both axillae and the right supraclavicular fossa. Breath sounds were distant. B.P. was 150/110. Hands showed *main-en-lorgnette* deformity and ulnar deviation of fingers; there was marked limitation of movement of shoulders, elbows, and knees, and a nodule on the right elbow showing non-specific changes only on biopsy. A knee biopsy was compatible with rheumatoid arthritis.

An x-ray film of the chest (Fig. 3) revealed multiple ill-defined circular areas of increased density throughout both lung fields. A skull x-ray film revealed a large osteolytic defect in the left temporal region and smaller ones in left fifth rib. There was collapse of the ninth dorsal vertebra. Investigations for tuberculosis, primary or metastatic carcinoma, hyperparathyroidism, and leukaemia were negative. Plasma cholesterol, calcium, and phosphorus were normal; alkaline phosphatase, 54 to 22 K.A. units; E.S.R., 60 mm.; colloidal gold test, 5; D.A.T., 1:32. Mantoux positive 1:100.

It was considered that the lesions resembled xanthomatosis. Past history and a review of old x-ray films taken in 1932 revealed that at that time she had almost exactly similar pulmonary lesions for which she received a "virtual death warrant" and deep x-ray therapy. Her rheumatoid arthritis began a short time after this treatment for her pulmonary lesions. She died with coronary thrombosis. No necropsy was done. The pulmonary and skeletal lesions were attributed to a xanthomatous process. This, however, was not proved.

FIG. 2a

FIG. 2b

FIG. 3

FIG. 2.—A.D., Case 2. Postero-anterior views of chest. (a) September, 1947, showing apical fibrosis with basal emphysema. (b) August, 1953, showing progression of fibrosis with honeycombing of both lung fields. FIG. 3.—E.P., Case 3. Postero-anterior view of chest in November, 1951, revealed multiple circular opacities in both lung fields, known to have been present in 1932, when a diagnosis of malignant secondary defects in the lungs had been made. Note osteolytic lesions in left fifth rib.

Case 4

D.S. was affected with blindness due to congenital microphthalmia in the left eye and corneal opacities in the right eye. In 1940, at the age of 56, she developed rheumatoid arthritis involving the hands, wrists, knees, and feet. Her arthritis progressed slightly over the years, but was never very severe, and she was able to dress herself with only slight difficulty. E.S.R. varied between 5 and 30 mm. an hour. She had had an episode of pleurisy 15 years previously, which preceded the onset of her arthritis. There was a positive family history of tuberculosis. In 1949 multiple papillomata were excised from the tongue. In 1952 she received x-ray therapy for senile keratosis of the face, and a year later a chest x-ray film (Fig. 4) revealed diffuse mottling and streaking of both lung fields. She had no symptoms

FIG. 4.—D.S., Case 4. Postero-anterior view of chest, 1953, showing diffuse mottling of both lung fields, most marked at bases.

referable to the respiratory system, no history of pulmonary infections or any exacerbations of her arthritis. In January, 1953, after a bout of influenza, she developed diabetes insipidus, which was readily controlled with pitressin tannate. The chest x-ray film remained unchanged. Investigations for acid-fast bacilli and carcinoma were negative. E.S.R., 30 mm.; D.A.T., 1:128. Mantoux positive 1:1,000. Lymph-node biopsy showed reticular and follicular cell hyperplasia.

In view of the sudden appearance of diabetes insipidus and its frequent association with Boeck's sarcoid and xanthomatosis, and in view of the otherwise negative investigations, a presumptive diagnosis of Boeck's sarcoid or xanthomatosis was made to explain the pulmonary findings.

Comment

These four cases illustrate the difficulty encountered in determining the aetiology of pulmonary fibrosis. In each case the patient had been observed for many years. The only associated disease was rheumatoid arthritis of varying severity. In the face of repeatedly negative findings and by the process of exclusion it was easy, but intellectually unsatisfactory, to implicate the rheumatoid process.

In Case 1 the pattern of events could well have fitted the "rheumatoid lung syndrome." Two and a half years after the onset of pulmonary signs and symptoms a squamous-cell carcinoma was found. It is reasonable to assume that this lesion could well account for the course of events in this patient.

Case 2 had rapidly developing pulmonary fibrosis, which was noted at the time of surgical removal of a thyroid adenoma five years after the onset of rheumatoid arthritis. The lesion was well advanced prior to operation and progressed post-operatively until the patient was finally incapacitated. This patient's arthritic disease was relatively mild. Investigations neither revealed any cause for the

pulmonary fibrosis nor suggested even a presumptive alternative diagnosis. There was no evidence of scleroderma. It was considered that, in view of the relatively low-grade rheumatic process, there was probably no relationship between his arthritic disease and the pulmonary fibrosis.

Case 3, with severe rheumatoid arthritis of 17 years' duration, when first seen in 1949 was found to have a widespread mottling in both lung fields. Investigation failed to uncover a causative agent. The pulmonary lesion was considered at that time to be possibly related to her rheumatoid disease. Old x-ray films taken in 1932 showed a similar picture before the onset of rheumatoid arthritis. A causal relationship between the two processes was therefore possible at the onset. Latterly she developed osteolytic lesions in the skull and ribs, which suggested a diagnosis of xanthomatosis.

Case 4, an elderly woman with mild rheumatoid arthritis, was found to have pulmonary reticulation on chest x-ray examination 10 years after the onset of her arthritis. No previous chest films were available for comparison. Her arthritis was never severe and she had no respiratory complaints. At no period in the recorded history was there an episode which could be characterized as the "rheumatoid lung syndrome." The relation of the pulmonary lesions to her arthritis could, however, not be ruled out. She later developed diabetes insipidus, which suggested that the pulmonary changes might have been due to sarcoidosis or xanthomatosis.

In each case a "rheumatoid" aetiology of the pulmonary lesions was entertained. Subsequently an alternative diagnosis was considered more probable. Thus pulmonary lesions are only rarely associated with rheumatoid arthritis. When observed, a careful search should be made to rule out transient or persistent pulmonary infiltration seen in scleroderma, disseminated lupus erythematosus, rheumatic fever, asthma, angioneurotic oedema, allergic states, periarteritis nodosa, Löfller's syndrome, Boeck's sarcoid, atypical pneumonia, congestive failure, and organizing albuminoid exudates of uraemic states. Frequently no aetiological agent can be found. Continued and prolonged observations will often bring to light the underlying process.

Summary

The case histories of 253 patients with rheumatoid arthritis, chest x-ray findings of 130 of these and of 130 non-rheumatoids, and post-mortem findings of 42 rheumatoid cases and 42 controls were reviewed to determine the incidence of pulmonary lesions in a rheumatoid and non-rheumatoid series of patients.

1. No specific lung lesion was found which could characteristically be associated with rheumatoid arthritis.

2. The clinical and post-mortem incidence of various non-specific pulmonary lesions appeared to be rather greater in the rheumatoid patients than in the control series, but no one particular lesion was outstanding.

3. In patients with clinical and radiological evidence of pulmonary disease, no temporal correlation was observed with exacerbations of rheumatoid disease.

4. In this comparatively small group of patients studied, no example of what has been described as "rheumatoid lung" or "rheumatoid lung syndrome" was observed.

5. Four cases are cited in detail, illustrating what have been in our experience the most difficult of the problems of differential diagnosis of lung lesions in rheumatoid arthritis.

Our thanks are due to Professor J. H. Dible and his staff for the use of their pathological observations, to the late Professor Duncan White and Dr. Robert Steiner and their staff for

radiological data, to Dr. M. D. Milne for permission to include Case 1, and to Dr. C. M. Fletcher and other colleagues at the Postgraduate Medical School for advice.

REFERENCES

- Baggenstoss, A. H., and Rosenberg, E. F. (1943). *Arch. Path. (Chicago)*, 35, 503.
- Bloom, J., and Rubin, J. H. (1950). *Canad. med. Ass. J.*, 63, 355.
- Caplan, A. (1953). *Thorax*, 8, 29.
- Christie, G. S. (1954). *Aust. Ann. Med.*, 3, 49.
- Elkeles, A. (1944). *Brit. J. Radiol.*, 17, 368.
- Ellman, P. (1947). *Proc. roy. Soc. Med.*, 40, 332.
- and Ball, R. E. (1948). *British Medical Journal*, 2, 816.
- and Cudkowicz, L. (1954). *Thorax*, 9, 46.
- and Weber, F. P. (1949). *British Medical Journal*, 1, 304.
- Harris, L. H. (1954). *Lancet*, 2, 119.
- Hart, F. D. (1948). *British Medical Journal*, 2, 996.
- Hollander, J. L. (1953). *Comroe's Textbook of Arthritis and Allied Conditions*, 5th ed., p. 153. Kimpton, London.
- Kellgren, J. H., Lawrence, J. S., and Aitken-Swan, J. (1953). *Ann. rheum. Dis.*, 12, 5.
- Leys, D. G., and Swift, P. N. (1949). *British Medical Journal*, 1, 434.
- Littler, T. R. (1953). *Rheumatism*, 9, 24.
- Mainland, D. (1953). *Amer. Heart J.*, 45, 644.
- Miall, W. E. (1955). *Ann. rheum. Dis.* In press.
- Caplan, A., Cochrane, A. L., Kilpatrick, G. S., and Oldham, P. D. (1953). *British Medical Journal*, 2, 1231.
- Miller, H. G., and Daley, R. (1946). *Quart. J. Med.*, 15, 255.
- Murphy, J. R., Krainin, P., and Gerson, M. J. (1941). *J. Amer. med. Ass.*, 116, 499.
- Rakov, H. L., and Taylor, J. S. (1942). *Arch. intern. Med.*, 70, 88.
- Raven, R. W., Weber, F. P., and Price, L. W. (1948). *Ann. rheum. Dis.*, 7, 63.
- Schlesinger, B. (1949). *British Medical Journal*, 2, 197.
- Shuford, W. H., Seaman, W. B., and Goldman, A. (1953). *Arch. intern. Med.*, 92, 85.
- Tumulty, P. A., and Harvey, A. M. (1949). *Bull. Johns Hopk. Hosp.*, 85, 47.
- Weir, D. R. (1939). *Amer. J. Path.*, 15, 79.
- Weiss, S., Stead, E. A., Warren, J. V., and Bailey, O. T. (1943). *Arch. intern. Med.*, 71, 749.
- (1951) found in Bristol that women in Social Classes I and II breast-fed longer than those in Social Classes IV and V, but their figures did not include cases of failure during the first four weeks.
- Spence *et al.* (1954), in the "thousand families" survey in Newcastle-upon-Tyne, found 42.5% of mothers in Social Classes I and II, 34% in Social Class III, and 29% in Social Classes IV and V wholly breast-feeding after three months. Westropp (1953), studying 574 mothers in Oxford "who regularly attended the Oxford Child Health Survey for at least a year" found that 66% of those in Social Classes I and II, 54% in Social Class III, and 44% in Social Classes IV and V completely breast-fed for four months. Douglas (1950), using material gathered in the 1946 Maternity Survey of England and Wales, found that wives in the professional and salaried classes breast-fed more often for three months than the wives of manual workers; in his investigation a baby was considered to be breast-fed until it was completely weaned. On the other hand, Dykes (1949), in a survey of all infants born in Luton in 1945, found no social class difference in the duration of breast-feeding, and Dummer (1949) found no clear-cut differences in breast-feeding performance between occupational groups.
- Women delivered in nursing-homes appear to breast-feed longer than those delivered in hospitals, who in turn breast-feed longer than women delivered at home (Hughes, 1948; Dummer, 1949; Dykes, 1949; Douglas, 1950; Campbell and Cheeseman, 1954; Spence *et al.*, 1954). The possibility that social class differences may have accounted for these findings, at least in part, was not investigated by those authors.

CLINICAL AND CHEMICAL STUDIES IN HUMAN LACTATION

X. THE MAINTENANCE OF BREAST-FEEDING

BY

F. E. HYTTEN, Ph.D., M.B., B.S.

AND

A. M. THOMSON, M.B., Ch.B., B.Sc., D.P.H.

Obstetric Medicine Research Unit (Medical Research Council), Midwifery Department, University of Aberdeen

In all parts of Britain the official policy is to promote breast-feeding whenever possible, yet its success seems to vary greatly from one area to another. For example, at the end of three months the proportion of women fully breast-feeding varied from 24% in Newbiggin-by-the-Sea (Hughes, 1948) to 58% in Luton (Dykes, 1949).

It is not easy to understand why such variations occur. In any case, exact comparison between different centres is often impossible because results are expressed in different ways, and the populations studied are not fully described in terms of social class, age, parity, etc. This paper describes how such factors affect breast-feeding rates in Aberdeen. The incidence of successful breast-feeding is defined as the proportion of babies wholly fed on the breast during the first three months of life. After three months mothers commonly introduce other feeds, and it is difficult to know what contribution breast milk is making to the child's total food intake.

Discussion of the literature is confined mostly to those British publications in which comparable information is available. Since there is some evidence (Ross and Herdan, 1951) of considerable changes in breast-feeding habits, only publications of the past ten years are quoted.

Factors Affecting Breast-feeding

Social Status

Most authors agree that breast-feeding is usually more successful in the upper social classes. Ross and Herdan

Maternal Age

Westropp (1953) found the duration of breast-feeding to decrease with age in primiparae, but Douglas (1950), using the national sample, found that maternal age had little effect when parity and social class were taken into account, although "young mothers appear to be more likely to stop breast-feeding in the first three months." Miller (1952) found no difference in the incidence of full breast-feeding at three months in primiparae aged above or below 35 or in multiparae aged above or below 40; only patients who left hospital breast-feeding were studied.

Parity

Dykes (1949), Douglas (1950), and Spence *et al.* (1954) found parity to have little or no influence on breast-feeding, but Westropp (1953) found that second and third babies were breast-fed longer than first or fourth and later babies, except in Social Classes I and II, where the breast-feeding performance improved progressively with increasing parity. Miller (1952) found that, of those leaving hospital breast-feeding, slightly more primiparae than multiparae were still breast-feeding at three months.

Physique and Health

Westropp (1953) found that maternal height, weight, and height/weight ratio did not affect the duration of breast-feeding. Miller (1952), however, found that considerably fewer women under 5 ft. (152 cm.) were breast-feeding at three months than taller women, and that somewhat more women "over the average weight for their height and age" were breast-feeding at three months than those who were underweight. Westropp found that women whose health was "uniformly good after delivery" breast-fed more successfully than those whose health was impaired.