Annals of the Royal College of Surgeons of England (1984) *vol.* 66

The prognosis and possible cause of severe primary lymphoedema*

JOHN H N WOLFE FRCS
Senior Surgical Registrar,
St Thomas' Hospital

Key words: LYMPHOEDEMA PRIMARY; PROGNOSIS OF LYMPHOEDEMA; LYMPH NODE FIBROSIS; LYMPHOGRAPHIC PELVIC OBSTRUCTION

Summary

We studied 372 patients with primary lymphoedema in order to predict the extent and severity of the disease. We found that the limits of oedema were defined early in the process and that the loss of distal lymphatics alone did not lead to severe oedema. Severe lymphoedema was associated with pelvic lymphatic 'obstruction' on lymphography and 26% of these patients eventually required surgery.

Lymphography suggested that the 'obstruction' was related to lymph nodes and inguinal node biopsies were taken at the time of lymphography in 72 patients. In patients with pelvic lymphatic 'obstruction' we found a severe nodal fibrosis which was not apparent in those with distal lymphatic disease alone. This fibrosis was not related to episodes of cellulitis and since it was present in the early stages of the disease it is unlikely to be due to slow obliteration of distal lymphatics. Furthermore it could not be reproduced by ligating either afferent or efferent lymphatics of the rabbit popliteal lymph node.

This suggests that severe primary lymphoedema may develop as a result of disease of the pelvic lymph nodes.

Introduction

The cause of primary lymphoedema remains obscure, and the management of the individual patient has been hampered by difficulties in judging the prognosis. The variability in the clinical course of lymphoedema is confusing; most patients have symptoms that can be controlled by conservative measures but some eventually develop severe lymphoedema. In these severely affected patients it has been unclear whether the oedema slowly spreads or whether the extent of the oedema is defined early in the process.

If the clinically severe form of the disease could be recognised early in the process this might allow more rational treatment and a more confident reassurance of mildly affected patients.

Allen *(1)* reviewed 300 cases of lymphoedema in 1934. In 124 patients no cause for the lymphoedema was found and these he classified as 'primary'. Following the introduction of lymphography by Kinmonth in 1952 *(5)* a classification of primary lymphoedema based on radiographic abnormalities of the distal lymphatics emerged. He used the terms 'aplasia', 'hypoplasia', and 'hyperplasia'. It later became apparent that many patients with severe lymphoedema had adequate distal lymphatics, and the possibility that the disease started more proximally was raised *(7)*. We therefore explored the possibility that a primary lymph node process may cause lymphoedema.

* Based on Hunterian lecture delivered on 2nd December 1982 at St Thomas' Hospital, London

Address for correspondence: St. Thomas' Hospital, London, SE1 7EH

The Editor would welcome any comments on this paper by readers

The studies reported here were intended firstly to examine the clinical features of lymphoedema in order to determine whether an accurate prognosis could be given. And secondly to explore the role of lymph node disease in the causation of lymphoedema.

Clinical study

SUBJECTS AND METHODS

Four hundred and twenty nine patients with primary lymphoedema of the legs were treated between 1970 and 1978; 345 of these are still being followed as out-patients. Forty five persons who live at a distance were sent a questionnaire to discover any changes in symptoms since their last examination; 27 questionnaires were returned. An additional 39 patients could not be traced. Therefore 372 patients were included in the study.

Progression of the lymphoedema was assessed by serial measurements and photography of the limb. Lymphographic progression of the disease could be assessed in 20 patients who had lymphograms on two occasions. For the purposes of assessment the leg was divided into three compartments: ankle, below knee and thigh. Extension of the disease was considered to have occurred if oedema developed in compartments that had been unaffected during the first year of symptoms. Extension was separated into two components: further extension of oedema in the same limb, and subsequent involvement of the other leg. Increase in girth was measured in affected areas.

CLASSIFICATION OF PATIENTS

The patients were classified according to their lymphangiograms into the following groups (Fig. 1) *(14)*:

(1) Distal obliteration alone (91 patients).
(2) Pelvic 'obstruction' with or without distal lymphatic obliteration (153 patients; 60 had pelvic 'obstruction' alone and the rest also had distal obliteration).
(3) Hyperplasia: bilateral disease (29 patients).
(4) Reflux into megalymphatics (12 patients).

Aplasia was not separately classified since this is probably an extreme form of distal obliteration in most patients. True aplasia is very rare and was not seen in this study.

It is now our policy to perform an inguinal lymphogram on all patients in whom distal lymphatics are obliterated. If the pelvic lymphatics are inadequate only a few small lymphatics are seen, ie, the patient has pelvic 'obstruction' and distal obliteration. However 38 patients in this study could not be classified since they had a failed foot lymphogram and no inguinal lymphogram; 49 patients did not

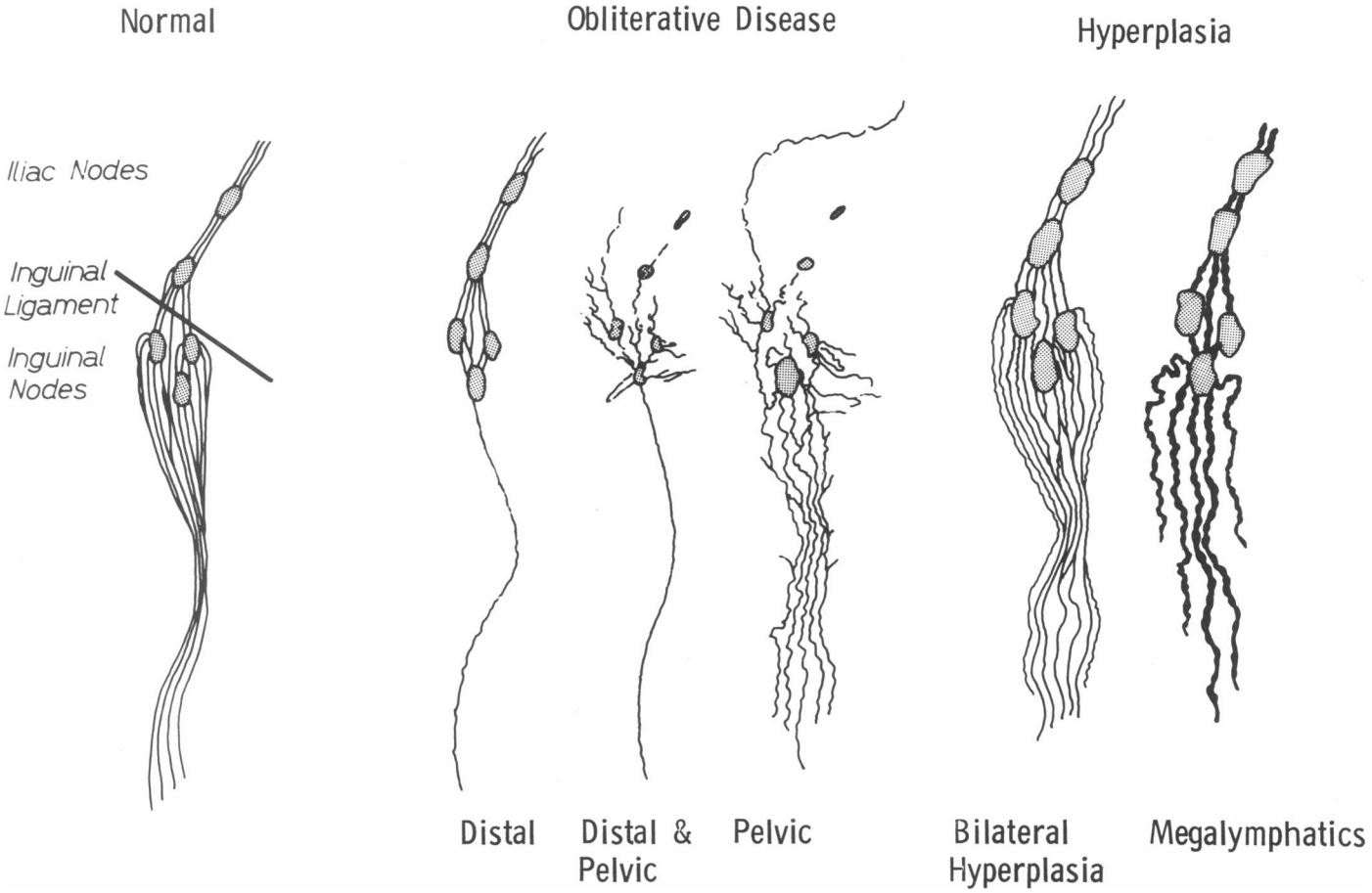

FIG. 1 Lymphographic classification of primary lymphoedema.

have lymphography since it was not considered clinically indicated.

LYMPH NODE HISTOLOGY

In addition to lymphography a superficial inguinal node biopsy was performed in 72 patients: 22 had distal lymphatic obliteration; 38 had pelvic obstruction with or without distal lymphatic obliteration; 11 had bilateral hyperplasia, and three had reflux into megalymphatics. In all cases an attempt was made to cut the node in a similar transverse plane through the hilum. The histological findings were compared with 30 normal inguinal lymph nodes obtained with the informed consent of patients undergoing surgery on varicose veins (21 patients) or peripheral arteries (9 patients). All inguinal nodes from five fresh necropsies were also studied to ensure that there was no major histological variation in lymph nodes taken from a single patient.

The nodes were assessed for fibrosis; the area of fibrosis in each section was traced using a calibrated, computerised light pen and monitor. The area of fibrosis was recorded as a percentage of the whole area of the lymph node section. This method was highly reproducible, but each trace was performed five times and a mean reading taken. A mean of 9 sections were studied for each lymph node.

Results

The median age of onset of clinical lymphoedema was 19 years in the patients with the 'obliterative' types of disease but younger in those with bilateral hyperplasia (13 years) and megalymphatics (11 years). Congenital lymphoedema was present in 10% of patients.

The duration of symptoms at the time of follow up was similar in the different groups: distal obliteration, 15 years; proximal obstruction with or without distal obliteration, 15 years; bilateral hyperplasia, 14 years; and reflux into megalymphatics, 12 years. Thus these appear to be variants of the disease and not different stages of the same process. There was an overall preponderance of women (72%) in the study, but they were not uniformly distributed between the groups. Most of those with distal hypoplasia were female (90%). But only 66% of those with pelvic 'obstruction' and 37% of those with hyperplasia were female. A family history of primary lymphoedema was less common in those patients with pelvic 'obstruction' (12%) than in those patients with distal obliteration alone (22%). Associated congenital anomalies were present in 6% of patients and were more common in patients with hyperplasia (22%).

CLINICAL FEATURES RELATED TO LYMPHANGIOGRAMS

The clinical features are given in Table 1. Whole limb lymphoedema was common with pelvic 'obstruction' (62%) and was very uncommon with distal obliteration alone (2%). It was interesting that in 10 patients with pelvic 'obstruction' the oedema started in the thigh or groin and only subsequently affected the lower leg. Unilateral lymphoedema was more common in patients with pelvic 'obstruction' with or without distal obliteration (88 patients, 51%), and less common in those with distal obliteration alone (27 patients, 30%).

Pelvic 'obstruction' is therefore associated with the severe form of lymphoedema, usually affects one limb, and affects men in one third of cases. In contrast distal obliteration alone is usually associated with milder, bilateral lower leg oedema; 90% of these patients were female.

The prognosis was closely related to the lymphographic findings. Pelvic 'obstruction' was the most likely cause of severe lymphoedema, as shown by the 26% of patients who eventually required surgery (Table 1). Distal obliteration was the least severe form of lymphoedema, since only one patient required surgery.

Recurrent attacks of cellulitis occurred in 27% of the patients and were more common in those patients with

TABLE I *Clinical data according to lymphangiographic classification*

	Distal obliteration	*Pelvic obstruction ± distal obliteration*	*Bilateral hyperplasia*	*Refluxing megalymphatic*
Number of patients	91	153	29	12
M/F ratio	1:9	1:2	3:2	3:2
Whole leg lymphoedema	2%	62%	24%	75%
Unilateral clinical lymphoedema	30%	51%	10%	83%
Extension in same limb following 1st year	5%	8%	10%	8%
Involvement of opposite limb following 1st year	5%	9%	6%	16%
Increase in girth at 20 years	36%	43%	42%	66%
Leg reducing surgery required	1%	26%	3%	25%

evidence of pelvic 'obstruction' (25%) than in those with distal obliteration alone (15%).

PROGRESSION OF LYMPHOEDEMA

The anatomical limits of clinical lymphoedema were usually defined early in the process and extension of lymphoedema was unusual after the first year of symptoms (Fig. 2). Further extension of the disease in the same limb occurred in only 27 patients (14%) between the first and fifth years, and in only three patients (2%) after five years. In no patient with distal obliteration alone did lymphoedema extend into the thigh after the first year of symptoms. Oedema of the other leg subsequently developed in 34 patients (9%).

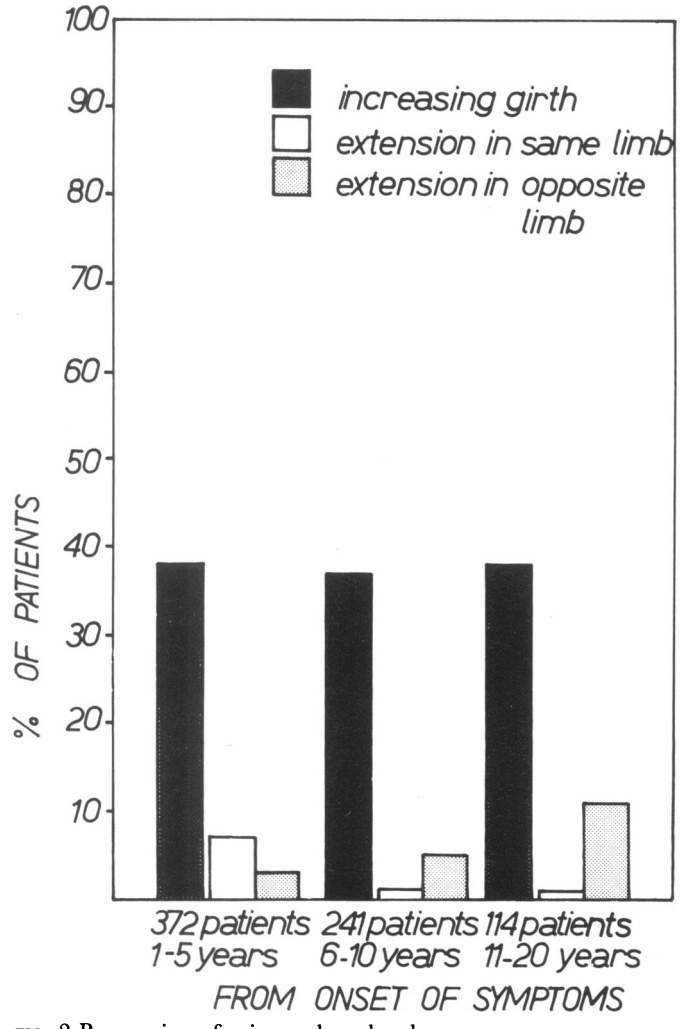

FIG. 2 Progression of primary lymphoedema.

Although the extent of clinical lymphoedema was established early in the process, there was an inexorable increase in the girth of the leg in 37% of patients (Fig. 2). All the patients had been advised about conservative treatment (hygiene, leg massage, elevation of the leg while resting, and support stockings), but their compliance varied. The efficacy of conservative treatment in avoiding an irrevocable increase in leg swelling cannot be assessed from this study.

In 20 patients a second lymphogram was performed after a mean interval of 31.5 months (range 1–96 months). The initial lymphogram showed pelvic obstruction with adequate distal lymphatics in 13 patients and bilateral hyperplasia in 7. No patient had evidence of distal obliteration.

On the subsequent studies, no change in the lymphographic appearance was found in 7 patients but the remaining 13 patients showed definite changes. Seven patients had features indicative of increasing lymphatic obstruction: increased numbers of collateral vessels in groin and pelvis, increased tortuosity and dilatation of the peripheral lymph trunks and increased backflow of contrast in the dermal lymphatics. A further 6 patients showed a reduction in the number of distal lymph vessels. Of the 13 patients in whom the second lymphogram showed evidence of increase in distension or distal obliteration, 10 had worsening of the limb swelling. Where the lymphograms were unchanged, 5 of the 7 patients did not experience any clinical deterioration. None of the 20 patients had cellulitis between the first and second lymphogram.

LYMPH NODE HISTOLOGY

The normal inguinal lymph nodes varied considerably in their appearance. Some fibrosis limited to the hilar region was a normal feature. Pericortical fibrosis (thickening of the fibrous capsule) was likewise present in a few 'control' lymph nodes. Lymphoid tissues involutes with age and fibrolipomatosis was a feature of many lymph nodes taken from older patients.

Taking these normal variations into consideration, the most striking difference in the lymph nodes from patients with lymphoedema was an extensive fibrosis (Fig. 4). From observations on some of the less affected nodes the fibrotic process appeared to emanate from the hilum and initially affect the medullary sinuses. It was associated with obliteration or recanalisation changes in many of the adjacent lymph vessels.

The amount of lymphoid tissue in the nodes was reduced, but what remained appeared normal. A few lymph nodes showed very active germinal centres, but this was unusual. Even in patients with larger and clinically palpable inguinal nodes or a very short history of lymphoedema there was no evidence of an acute inflammatory process. Fibrin is a potent stimulus for fibrosis but no interstitial deposition of fibrin was seen in the sections stained with picro Malory violet.

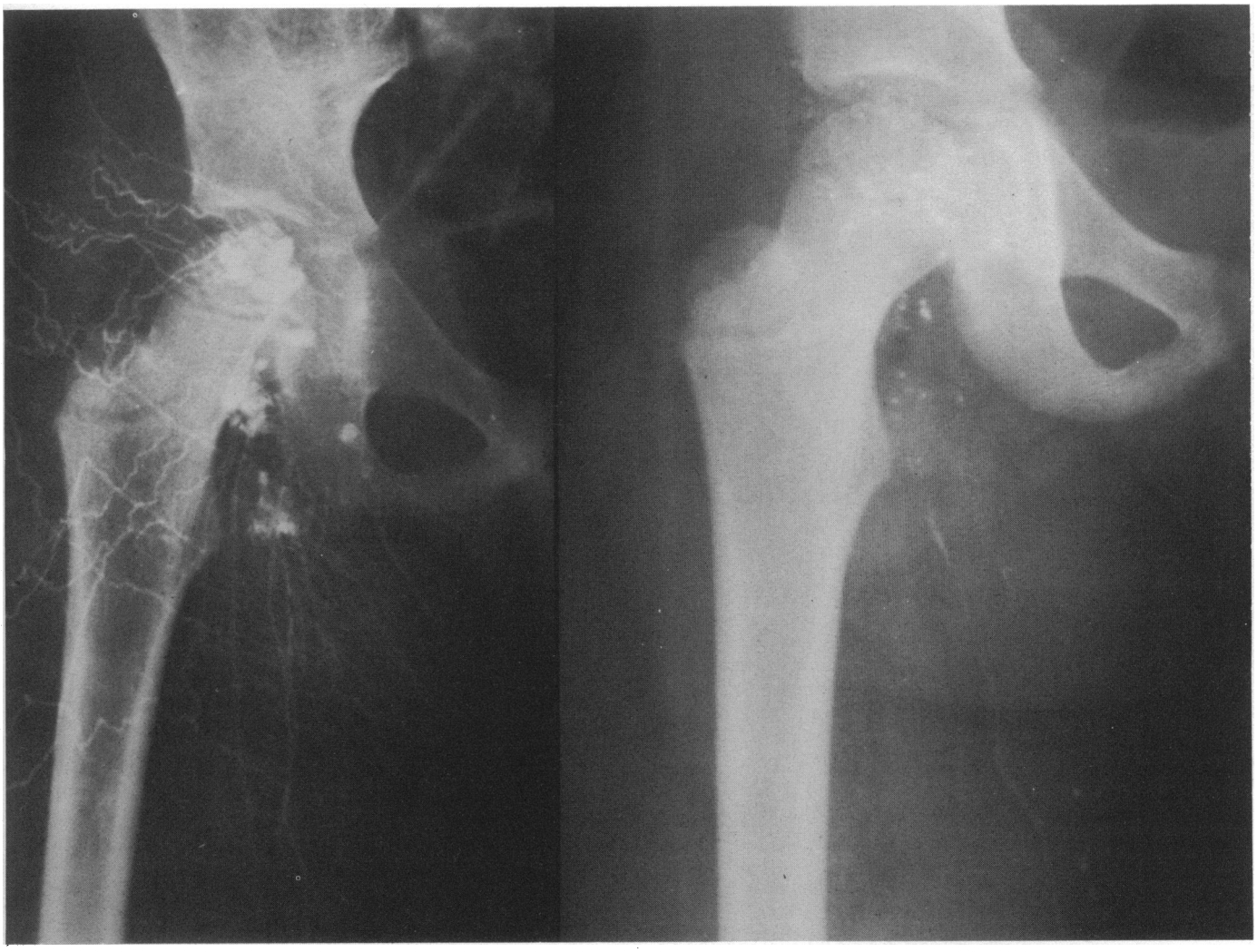

FIG. 3 Patient age six. Three year history of swelling of the whole of the right leg. First lymphogram (left) shows severe obstruction in the inguinal region with multiple collateral lymphatics. Nine months later the second lymphogram (right) shows only a single efferent lymphatic after infusion of the same volume of contrast. Residual contrast from the first study can still be seen in the groin. (Reproduced by permission of *Lymphology.*)

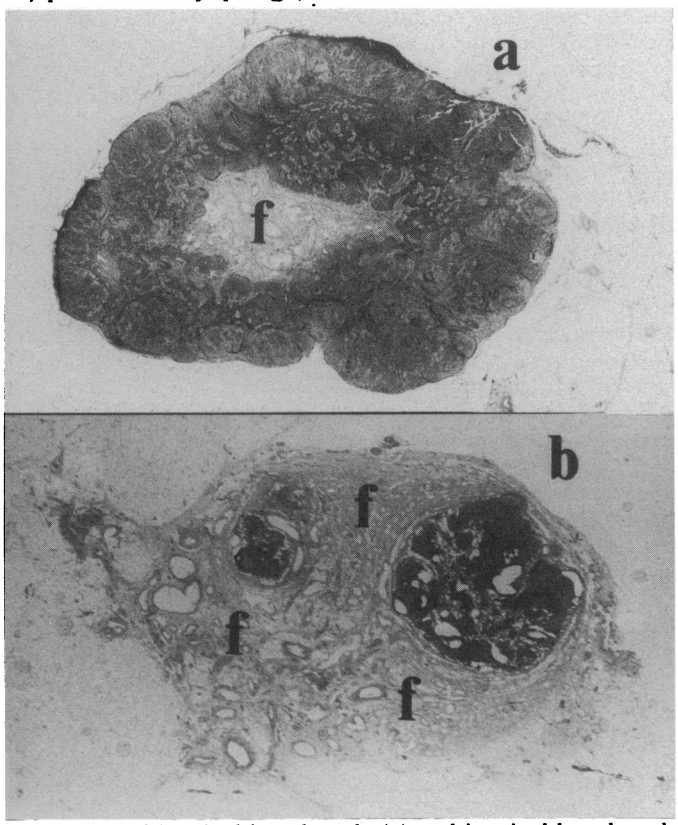

FIG. 4 Normal inguinal lymph node (a) and inguinal lymph node from patient with primary lymphoedema (b) showing extensive fibrosis (f).

In 2 patients with Milroy's disease (hereditary congenital lymphoedema) the nodes were small and moderately fibrosed (27% and 18% fibrosis respectively). The changes were similar to those seen in the other patients.

MEASUREMENT OF LYMPH NODE FIBROSIS

When the lymph nodes were separated into groups according to lymphographic findings it became apparent that patients with distal obliteration alone had a normal mean area of fibrous tissue (Table II). The patients with pelvic 'obstruction' had significantly more fibrosis than any other category ($p<0.01$, Student's t test and covariance analysis). The patients with bilateral hyperplasia also had an abnormal amount of fibrosis in the inguinal nodes. The fibrosis cannot be attributed to lymph node atrophy since the fibrotic nodes were not smaller than the normal nodes (Table II).

TABLE II *Inguinal lymph node fibrosis*

Lymphogram	*Mean area of node sections* (mm^2)	*Mean % fibrosis within node*
Normal	26	10%
Distal obliteration	29	11%
Proximal obstruction ±distal obliteration	39	38%*
Bilateral hyperplasia	64	28%
Refluxing megalymphatics	18	11%

*$p<0.01$ (Student's t test or covariance analysis).

In 7 patients an inguinal node taken from the clinically normal limb showed significantly less fibrosis than the node from the affected limb ($p = 0.01$ by the paired t test). There was, however, slightly more fibrosis in these nodes than in the control nodes. The nodal fibrosis was very similar in the two limbs of patients with bilateral lymphoedema (16 pairs); and was also similar in two nodes taken from the same lymphoedematous limb (13 pairs).

THE ROLE OF INFECTION

It might be postulated that the lymph node fibrosis was secondary to attacks of cellulitis and ascending lymphangitis, but only 20 patients gave a history of previous episodes of clinical cellulitis. The mean area of fibrosis in these patients was 30% and in those with no previous attacks of clinical cellulitis it was 29%, a negligible difference. Fibrosis could thus not be related to episodes of clinical cellulitis.

DURATION OF DISEASE AND DEGREE OF FIBROSIS

Another point to be considered is whether the fibrosis in the lymph nodes could follow progressive obliteration of the afferent lymphatic vessels feeding them. The median duration of clinical lymphoedema before biopsy was 10 years in those patients with less than 10% fibrosis and 5.5 years in those with more than 30% of fibrosis. This suggests that fibrosis is unrelated to the duration of clinical oedema.

LYMPH VESSEL HISTOLOGY

In general the changes in the foot lymphatics were very similar to those in the afferent and efferent inguinal lymphatics. There was considerable intimal proliferation and hypertrophy of the muscular layer. The lumen often showed recanalisation changes and was obliterated in patients with 'aplasia'.

Animal study

METHODS

Forty eight rabbits were studied and were divided into 4 groups. Each rabbit had either the afferent or efferent lymphatics of one popliteal node ligated and a sham operation performed on the contralateral limb. Half of these rabbits had lymphography to assess the efficacy of ligation, alterations in lymphatic drainage and any changes in lymph node area. The rest had no Lipiodol injected since this oil produces an oleogranulomatous reaction *(4)* which resolves *(2,3)*. In the rabbits in whom no lymphography was performed Patent Blue Violet was injected subcutaneously to ensure that lymphatic ligation was complete.

The effect of ligation was studied by cinelymphangiography, serial lymph node area measurements, lymph node volumes and lymph node histology.

Lymph node volumes were estimated at the time of operation by measuring length, width and depth with Vernier calipers and applying the formula for an ellipsoid. This method allowed us to measure lymph node volumes without removing the node. Following lymphography lymph node areas were measured every 2 weeks by tracing the area of the node as seen on lymphadenograms.

Results

One rabbit died 3 weeks after surgery and a second rabbit was excluded from consideration due to technical failure of the procedure. All the wounds healed well by primary intention and in all cases a second lymphogram was achieved. The rabbits did not develop clinical oedema as a result of the lymphatic ligation.

RESULTS OF AFFERENT LYMPHATIC LIGATION

Thirteen of these rabbits had lymphography which showed adequate ligation. Regenerated lymphatics were subsequently shown in 4 cases.

The rate of flow between the site of cannulation and the lumbar nodes was significantly more rapid as a result of bypassing the popliteal lymph node (mean rate 0.63 mm/sec $\pm$ 0.37 S.D.; control 0.40 mm/sec $\pm$ 0.16 S.D.).

Following lymphatic ligation there was a rapid initial decrease in the area of the lymph nodes and this did not recover during the course of the experiment. This reduction in lymph node area was borne out by a significant reduction in lymph node volume. This was true whether or not Lipiodol had been infused ($p < 0.005$ using the Mann Whitney U test).

Perinodal fibrosis was a striking feature in 15 of the nodes. There was marked shrinkage of the medulla. The cortex of the lymph nodes was usually congested with large numbers of lymphocytes in the sinuses but the germinal centres appeared similar to the normal contralateral node. There was no evidence of an increase in the amount of central fibrosis in any lymph node from this group.

In summary, despite the dramatic reduction in lymph node size following afferent lymphatic ligation, there was no increase in the amount of central fibrosis.

RESULTS OF EFFERENT LYMPHATIC LIGATION

Fourteen rabbits had lymphography following efferent lymphatic ligation; with the exception of one rabbit, regeneration followed ligation (Fig. 5). The mean transit time of Lipiodol through the popliteal node was nevertheless twice as long as in the normal control studies (mean 134 sec $\pm$ 74 S.D., control 76.8 sec $\pm$ 52.9 S.D.). There was an initial increase in the lymph node area which started to resolve as the weeks passed. However the lymph nodes remained larger than normal throughout the experiment. There was an increase in the volume of nodes following efferent lymphatic ligation whether or not they had Lipiodol infused ($p < 0.02$ using the Mann Whitney U test).

The nodal swelling following efferent lymphatic ligation was produced by stagnation of lymph. The lymphatic sinuses were dilated and contained scanty numbers of lymphocytes.

In 6 nodes there was some evidence of fibrosis along the medullary sinuses, but this was minimal and difficult to discern from some of the normal nodes. Four of the 6 nodes that had this fibrosis were not infused with Lipiodol so that the oil cannot be incriminated as the cause.

In summary lymph node stasis due to obstruction proximal to the lymph node may produce some medullary fibrosis but obstruction distal to the lymph nodes does not have the same effect and in neither situation are histological changes produced that bear any similarity to the changes seen in the nodes from patients with lymphoedema.

Discussion

Early recognition of various forms of primary lymphoedema should lead to more rational treatment and our results show that the pattern of disease is usually defined at the outset. We were surprised that only a small proportion of patients developed new areas of oedema after the initial year of symptoms. Whole leg oedema developed in only one of 124 patients after the first year of symptoms. This early demarcation of the limits of oedema implies that distal lymphatic obliteration does not slowly progress proximally, and that pelvic 'obstruction' rapidly produces mild oedema in the areas that may later become severely affected.

We have found that patients with 'hypoplasia' have a number of obliterated lymphatics and we have therefore substituted the term 'obliterative disease' for 'hypoplasia'. The primary defect does not appear to be numerical lack of lymphatic collecting vessels but rather a thrombosis of those present.

Many patients have in the past been classified as 'distal aplasia' on the basis of a failed foot lymphogram. By performing a direct inguinal injection we now differentiate between patients with distal disease alone, and a good prognosis and those who also have evidence of pelvic obstruction, and a poor prognosis.

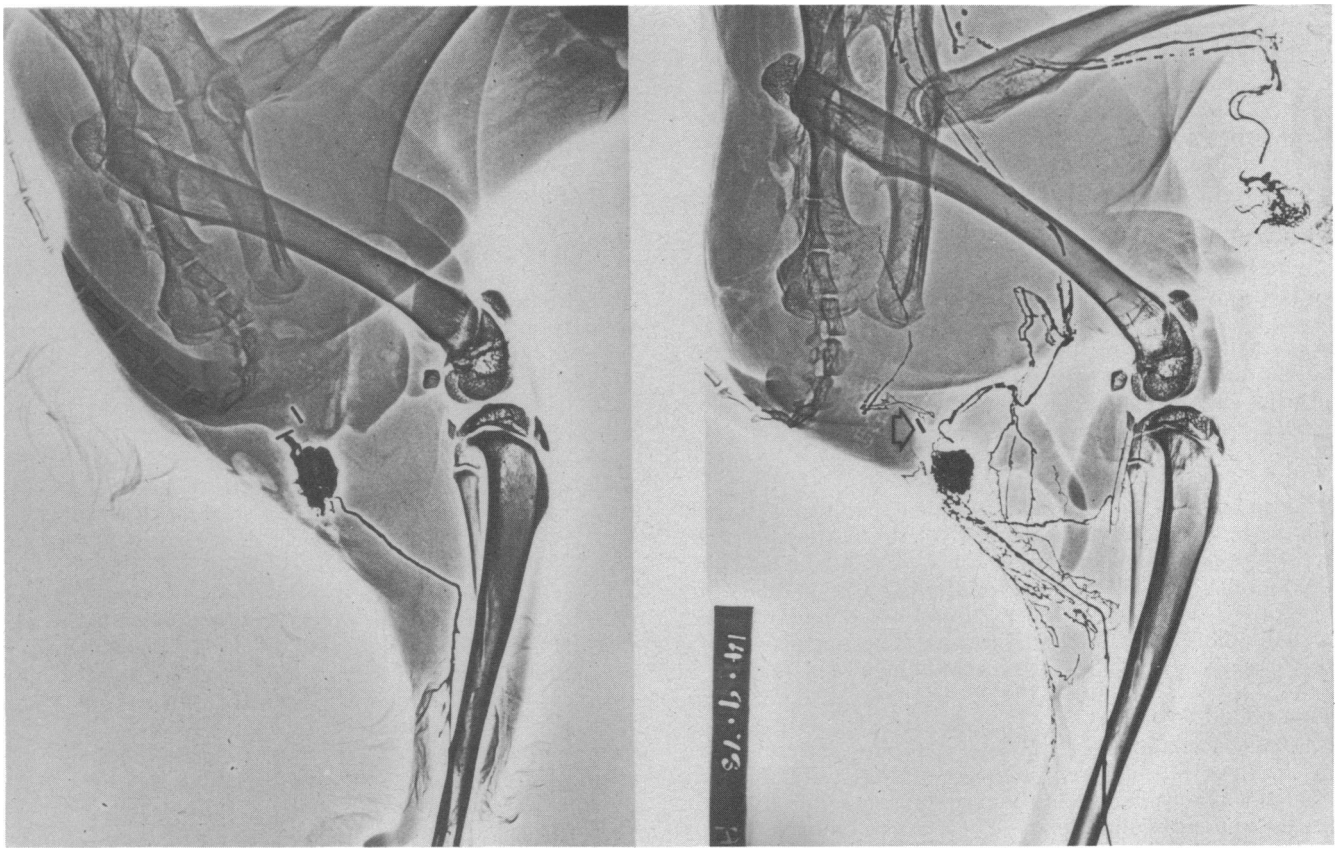

FIG. 5 Lymphangiogram in a rabbit immediately following efferent lymphatic ligation and 14 weeks later. The second lymphogram shows regeneration of lymphatics through and around the popliteal node. The filling of distal collateral lymphatics suggest continuing obstruction. (Reproduced by permission of *Lymphology*)

Mild bilateral lymphoedema associated with distal lymphatic obliteration is well controlled by conservative measures and these patients, who are often females presenting between the age of 15 and 25, can be reassured that progressive swelling is extremely unlikely. Lymphography may be useful in making the diagnosis, but will not affect management since surgical intervention is not indicated. In many of these patients lymphography is therefore unnecessary. A recognisable group of patients have a rapidly progressive disease that starts in the pelvis. These patients usually have unilateral whole leg oedema, although the oedema in the thigh may be mild in the initial stages. They may present when the distal lymphatics are patent and this allows a 'lymph drainage' operation to be considered early in the course of the disease *(8, 11)*. Progressive obliteration of distal lymphatics occurs if the 'obstruction' cannot be relieved, and these changes are associated with an increase in leg swelling.

For any significant advance to be made in the management of severe primary lymphoedema the underlying cause of the 'obstruction' must be elucidated. It may then become possible to treat it before the trapped proteins distal to the obstruction produce irreversible changes in the subcutaneous tissues.

The concept of lymph node obstruction is well recognised in filariasis, and is also considered to be the cause of lymphoedema secondary to tuberculosis and other chronic infections *(10)*. Mowlem in 1948 *(9)* suggested that lymph node obstruction was the cause of primary lymphoedema but had no lymphographic or other supportive evidence. This isolated remark went unnoticed once lymphography had revealed the gross abnormalities of the distal lymphatics. These latter findings influenced the direction of further studies, but more recently the possibility of a primary lymph node abnormality has been reconsidered *(7)*.

Our histological studies revealed a severe lymph node fibrosis that cannot be explained by episodes of clinical cellulitis or by slow atrophy due to inadequate lymph supply. This fibrosis correlated with a clinically severe form of lymphoedema in which the lymphogram revealed evidence of pelvic 'obstruction'.

The cause of the fibrosis remains unknown. In a study on rabbit popliteal lymph nodes we were unable to reproduce the extensive fibrosis. This suggests that the fibrosis in the lymph nodes secondarily affects the lymphatics, rather than distal lymphatic disease producing the lymph node fibrosis. The experiments could be criticised for the short time interval between the ligation of lymphatics and the removal of the lymph nodes; but since the lymphatics slowly regenerated we considered that most of the effect of lymphatic ligation would be seen in the early period. This lack of fibrosis has since been confirmed *(12)* using a time interval of up to 210 days in experiments similar to our own.

There is considerable evidence for a congenital factor in the development of primary lymphoedema *(6)*, but this fails to explain why the majority of patients develop clinical symptoms in early adulthood, and why patients with bilateral lymphographic abnormalities sometimes have only unilateral oedema. In the 7 patients in whom a lymph node biopsy was also taken from the clinically normal leg the lymph node was significantly less fibrotic although it was more fibrotic than normal.

It could be postulated that since the fibrosis was closely related to the clinical severity of the disease, a secondary insult produces lymph node fibrosis in a congenitally inadequate lymphatic system and this results in clinical oedema. Alternatively the fibrosis may slowly progress from birth and only later result in clinical symptoms.

This study was performed under the guidance of the late Professor J B Kinmonth. Without his extensive clinical experience, the consent of his patients, and his consistent support it would not have been possible. Mr D L Rutt gave valuable technical assistance and Professor J R Tighe gave helpful advice on the histology.

References

1 Allen EV. Lymphoedema of the extremities. Classification, etiology and differential diagnosis; a study of 300 cases. Arch Intern Med 1934;54:606–24.
2 Bourquin J. Histologische Grundlagen des lymphadenograms. Radiologe 1968;8:150–3.
3 Bujar H. Functional interpretation of lympography according to contrast medium persistence in the lymph nodes. Rev Roum Med Int 1973;10:388–95.
4 Fischer HW, Zimmermann GR. Roentgenographic visualization of lymph nodes and lymphatic channels. Am J Roentgen 1959;81:517–34.
5 Kinmonth JB. Lymphangiography in man. A method of outlining lymphatic trunks at operation. Clin Sci 1952;11:13–20.
6 Kinmonth JB. The lymphatics, diseases, lymphography, and surgery. London: Arnold, 1982:128–9.
7 Kinmonth JB, Eustace PW. Lymph nodes and vessels in primary lymphoedema. Ann Roy Coll Surg Engl 1976;58:278–84.
8 Kinmonth JB, Hurst PA, Edwards JM, Rutt DL. Relief of lymph obstruction by use of a bridge of mesentery and ileum. Br J Surg 1978;65:829–33.
9 Mowlem R. The treatment of lymphoedema. Br J Plast Surg 1948;1:48–55.
10 Ngu VA, Konstam P. Chronic lymphoedema in Western Nigeria. Br J Surg 1964;51:101–10.
11 Nielubowicz J, Olszewski W. Surgical lymphatic venous shunts. Br J Surg 1968;55:440.
12 Vlahos L, Theophanosis P, Tsakraklides S, Pontifex G. Lymph node changes after experimental interruption of the lymphatics. In: Bartos V, Davidson JW, eds. Advances in Lymphology. Prague: Avicenum, 1982;302–6.
13 Wolfe JHN. Diagnosis and classification of lymphoedema. In Rutherford, RB, ed. Vascular Surgery, 2nd Ed, WB Saunders, Philadelphia, 1984.

The Editor wishes to thank the following who are among those who have kindly acted as referees for papers:

John Alexander-Williams
Rosemary Baillod
Les Berger
Evelyn Boesen
Doreen Brown
Norman Browse
Kevin Burnand
Donald Campbell
Brendan Devlin
Robert Dick
James Dickson
Adrian Fox
Phyllis George
Lionel Gracey
John Groves
Peter Hardwick
Bill Heald
James Hinton
Ken Hobbs
John Hopewell
Hilary Howell
Crawford Jamieson
Alan Johnson
Iain Laws
Adam Lewis
Ken Lindsay
John Lumley
Colin Madgwick
Robert Maurice-Williams
Robert Morgan
Paul Noone
Santilal Parbhoo
Roy Pounder
Dick Rushman
Imrich Sarkany
Gerald Shaper
Robert Shaw
Robert Shields
Lewis Spitz
Martin Thomas
Hamish Thomson
James Thomson
Derek Wilson

If your name was inadvertently left out, please jog our memory.