- 2 Weseley AC, Douglas GW. Continuous use of chlorothiazide for prevention of toxemia of pregnancy. *Obstet Gynecol* 1962;19:355-8.
- 3 Flowers CE Jr, Grizzle JE, Easterling WE, Bonner OB. Chlorothiazide as a prophylaxis against toxemia of pregnancy. A double-blind study. *Am J Obstet Gynecol* 1962;84:919-29.
- 4 Menzies DN. Controlled trial of chlorothiazide in treatment of early pre-eclampsia. *Br Med J* 1964;i:739-42.
- 5 Fallis NE, Plauche WC, Mosey LM, Langford HG. Thiazide versus placebo in prophylaxis of toxemia of pregnancy in primigravid patients. *Am J Obstet Gynecol* 1964;88:502-4.
- 6 Cuadros A, Tatum HJ. The prophylactic and therapeutic use of bendroflumethiazide in pregnancy. *Am J Obstet Gynecol* 1964;89:891-7.
- 7 Landesman R, Aguero O, Wilson K, LaRossa R, Campbell W, Penaloza O. The prophylactic use of chlorthalidone, a sulfonamide diuretic, in pregnancy. *Br J Obstet Gynaecol* 1965;72:1004-10.
- 8 Finnerty FA Jr, Bepko FJ Jr. Lowering the perinatal mortality and the prematurity rate. The value of prophylactic thiazides in juveniles. *JAMA* 1966;195:135-8.
- 9 Kraus GW, Marchese JR, Yen SSC. Prophylactic use of hydrochlorothiazide in pregnancy. *JAMA* 1966;198:128-32.
- 10 Tervila L, Vartiainen E. The effects and side effects of diuretics in the prophylaxis of toxemia of pregnancy. *Acta Obstet Gynecol Scand* 1971;50:351-6.
- 11 Campbell DM, MacGillivray I. The effect of a low calorie diet or a thiazide diuretic on the incidence of pre-eclampsia and on birth weight. *Br J Obstet Gynaecol* 1975;82:572-7.
- 12 MacGillivray I, Campbell DM. The relevance of hypertension and oedema in pregnancy. *Clin Exp Hypertens* 1980;2:897-914.
- 13 Redman CWG. Treatment of hypertension in pregnancy. *Kidney Int* 1980;18:267-78.
- 14 MacGillivray I. Sodium and water balance in pregnancy hypertension—the role of diuretics. *Clinical Obstetrics and Gynaecology* 1977;4:549-61.
- 15 Gray MJ. Use and abuse of thiazides in pregnancy. *Clin Obstet Gynecol* 1968;11:568-78.
- 16 Lindheimer MD, Katz AI. Sodium and diuretics in pregnancy. *Obstet Gynecol* 1974;44:434-40.
- 17 Chamberlain G, Phillip E, Howlett B, Masters K. *British births*. London: Heinemann, 1970.
- 18 Office of Population Censuses and Surveys Monitor. *Infant and perinatal mortality 1981*. London: Office of Population Censuses and Surveys, 1982.
- 19 Pritchard JA, Walley PJ. Severe hypokalemia due to prolonged administration of chlorothiazide during pregnancy. *Am J Obstet Gynecol* 1961;81:1241-4.
- 20 Goldman JA, Neri A, Ovadia J, Eckerling B, De Vries A. Effect of chlorothiazide on intravenous glucose tolerance in pregnancy. *Am J Obstet Gynecol* 1969;105:556-60.
- 21 Ferris TF. Toxemia and hypertension. In: Burrow GN, Ferris TF, eds. *Medical complications during pregnancy*. 2nd ed. Philadelphia: WB Saunders, 1975:53-104.
- 22 Harley JD, Robin H, Robertson SEJ. Thiazide-induced neonatal haemolysis. *Br Med J* 1964;i:696-7.
- 23 Rodriguez SU, Leikin SL, Hiller MC. Neonatal thrombocytopenia associated with ante-partum administration of thiazide drugs. *N Engl J Med* 1964;270:881-4.
- 24 Crosland DM, Flowers CE Jr. Chlorothiazide and its relationship to neonatal jaundice. *Obstet Gynecol* 1963;22:500-4.
- 25 Minkowitz S, Soloway HB, Hall JE, Yermakov V. Fatal hemorrhagic pancreatitis following chlorothiazide administration in pregnancy. *Obstet Gynecol* 1964;24:337-42.
- 26 Johnston DH, Cornish AL. Acute pancreatitis in patients receiving chlorothiazide. *JAMA* 1959;170:2054-6.
- 27 Menzies D, Prystowsky H. Acute hemorrhagic pancreatitis during pregnancy and the puerperium associated with thiazide therapy. *J Fla Med Assoc* 1967;54:564-5.
- 28 Miller JN. Hyponatremia: a complication of the treatment of the edema of pregnancy. *Obstet Gynecol* 1960;16:587-90.
- 29 Peto R, Pike MC, Armitage P, et al. Design and analysis of randomized clinical trials requiring prolonged observation of each patient. 1. Introduction and design. *Br J Cancer* 1976;34:585-612.
- 30 McIlwaine GM, Dunn F, Howat RCL, Smalls M, Wyllie MM, Macnaughton MC. *The Scottish perinatal mortality survey 1977-1981*. Glasgow: University of Glasgow, 1984.
- 31 Fidler J, Smith V, Fayers P, De Swiet M. Randomised controlled comparative study of methyldopa and oxprenolol in treatment of hypertension in pregnancy. *Br Med J* 1983;286:1927-30.
- 32 Rubin PC, Clark DM, Summer DJ, Low RA, Butters L, Reynolds B. Placebo-controlled trial of atenolol in treatment of pregnancy-associated hypertension. *Lancet* 1983;i:431-4.
- 33 Chesley LC. The remote prognostic significance of the level of blood pressure in pregnancy. *Clin Exp Hypertens* 1980;2:777-801.
- 34 Nelson TR. A clinical study of pre-eclampsia. I. *Br J Obstet Gynaecol* 1955;62:48-57.

(Accepted 21 November 1984)

Clinical importance of enteric communication with abdominal abscesses

S H SAVERYMUTTU, A M PETERS, J P LAVENDER

Abstract

The dynamics of leucocytes in abdominal abscesses were studied using indium-111 autologous leucocyte scanning in 30 patients. Thirteen patients showing enteric drainage of leucocytes on delayed scans were characterised by a lack of abdominal localising signs and a low detection rate by ultrasound (25%). By contrast, 16 of 17 patients without enteric drainage had abdominal signs, and in these patients ultrasound was associated with a higher detection rate (58%). Despite the presence of an enteric route of drainage for the abscess 10 of the 13 patients needed surgical intervention.

These results help explain the wide variation in clinical presentation of abdominal abscesses; suggest that ¹¹¹In leucocyte scanning should be the initial investigation in those patients without focal signs; and show that formal surgical drainage is needed in patients recognised as having enteric communication with abscesses.

Introduction

Despite advances in surgical technique and medical treatment intra-abdominal abscesses remain a common diagnostic problem. Mortality is over 80% in undrained collections^{1 2} but may be decreased to less than 30% with effective surgical treatment. The critical factor determining the prognosis of these patients is the difficulty in localising the abscesses. In many abscesses there are classic clinical features with focal abdominal tenderness, and in these cases ultrasound is a rapid, sensitive diagnostic technique.^{3 4} In some abscesses, however, the clinical presentation may be insidious with minimal signs of localisation, and these pose much greater problems in diagnosis. This wide range in clinical presentation of intra-abdominal abscesses is unexplained.

During early studies using indium-111 leucocyte scanning we observed that in some abscesses with minimal localising signs there was drainage of labelled leucocytes into the bowel on delayed scans, indicating enteric communication with the abscesses.⁵ To investigate whether enteric drainage of pus decompresses the abscess and so accounts for lack of abdominal signs we have examined the incidence of enteric communication with abscesses and related this feature to the clinical presentation.

Patients and methods

From August 1981 to December 1983 patients referred to this hospital's department of diagnostic radiology for routine ¹¹¹In leucocyte scans were included in the study if (a) an abscess was detected by the scan, (b) the diagnosis was confirmed independently, and (c) full clinical details were available for review.

A total of 30 abscesses were studied. Confirmation of the diagnosis

Royal Postgraduate Medical School, Hammersmith Hospital, London W12

S H SAVERYMUTTU, BSC, MRCP, lecturer in medicine
A M PETERS, MD, senior lecturer in radiology
J P LAVENDER, FRCPD, FRCR, senior lecturer in radiology

Correspondence to: Dr S H Saverymuttu, Medicine II, St George's Hospital Medical School, Tooting, London SW17.

was by surgery in 23 cases, at necropsy in two, by witnessing the discharge of pus in four, and by computed tomography (CT) in one. In 24 patients the results were compared with an ultrasound scan performed within seven days of the ^{111}In leucocyte scan. All patients had fever, leucocytosis, or abdominal pain. In 20 patients the abscess was a consequence of surgery.

Leucocyte labelling and scanning—Autologous leucocytes were labelled in plasma with ^{111}In -tropolonate.⁶ In patients with leucocyte counts greater than $12 \times 10^9/\text{l}$ mixed leucocyte preparations were used, and in patients with counts less than $12 \times 10^9/\text{l}$ a pure granulocyte preparation was separated on a discontinuous density gradient. Early scans were performed two to four hours after administration of the labelled cells and delayed scans between 18 and 24 hours. In some cases scans were performed for up to 120 hours. Abnormal activity in early scans equal to or greater than liver or spleen activity was considered to indicate the presence of an abscess. Delayed scans were analysed for evidence of enteric communication with the abscess, recognised by drainage of activity into the bowel. In seven cases faecal excretion of labelled leucocytes was measured in a four day collection. The study was repeated after an interval of seven to 26 days in six patients to determine whether drainage had been associated with spontaneous resolution of the abscess.

Results

ABSCESSES WITH ENTERIC DRAINAGE

On delayed scanning 13 patients showed evidence of an enteric communication with the abscess (figure). Table I lists the sites of the abscesses; the commonest location was paracolic. The degree of drainage varied, nine abscesses showing major clearance of activity into the bowel, while in the remaining four patients most of the activity remained at the original site of localisation. Nine of the 13 patients had no abdominal signs and, with the exception of one patient, all showed major degrees of bowel drainage. The remaining patients had either fever or leucocytosis. Only four patients had abdominal tenderness, three of whom showed minor degrees of bowel drainage. There appeared to be no difference between patients showing enteric drainage and patients without enteric drainage regarding incidence of fever, leucocytosis, positive blood cultures, or use of steroids (table II). The duration of symptoms of leucocytosis before scanning, however, was longer in the enteric drainage group (mean 12.1 days; range 6-18 days) than in patients without enteric communication (mean 6.5 days; range 3-15 days). In this group of abscesses ultrasound scanning gave poor results, detecting only three out of 12 abscesses.

Faecal granulocyte excretion in a four day collection in two patients with major drainage was 42.3% and 24.6%, while in two patients with minor drainage excretion was 12.4% and 6.1% of the injected dose.

Despite enteric drainage 10 abscesses in this group were surgically drained (table III). In six cases a trial of antibiotics had been given but repeat scans seven to 26 days later showed no improvement, with similar degrees of bowel drainage. Two of these patients died within 30 days of surgery. One further patient died in whom surgery was deferred because of the development of acute renal failure. Necropsy was not performed, and this was the only patient whose diagnosis was confirmed by CT. Two further abscesses were not surgically drained. In one case the paracolic abscess clinically resolved completely. The other patient had underlying radiation enteritis with a chronic pelvic abscess draining through both the rectum and vagina. Since she was virtually symptom free we decided that the risks of surgery outweighed the benefits.

TABLE I—Site of abscess

	Enteric communication	No enteric communication
Paracolic	7	3
Pelvic	4	5
Subphrenic	0	2
Subhepatic	0	2
Pancreatic	0	2
Miscellaneous	2	3
Total	13	17

TABLE II—Distribution of clinical features in patients studied. (Percentages in parentheses)

	Enteric communication (n = 13)	No enteric communication (n = 17)
Abdominal tenderness	4 (31)	16 (94)
Previous bowel surgery or underlying bowel disease	13 (100)	10 (59)
Detection by ultrasound	3/12 (25)	7/12 (58)
Fever ($\geq 38^{\circ}\text{C}$)	9 (69)	11 (65)
Leucocytosis ($\geq 12 \times 10^9/\text{l}$)	8 (62)	9 (53)
Positive blood cultures	2 (15)	2 (12)
Steroid treatment	3 (23)	4 (24)

TABLE III—Management and outcome of abscesses

	Enteric communication	No enteric communication
Surgical drainage:		
Immediate	4	13
After failure of antibiotic trial	6*	0
No surgery:		
Spontaneous resolution	1	2
Chronic abscess	1	0
Death	1	2
Total	13	17

*Two patients in this group died within 30 days after surgery.

ABSCESSES WITHOUT ENTERIC DRAINAGE

Seventeen patients had no evidence of enteric communication with abscesses on delayed scans. Sixteen of these patients had abdominal signs, in 12 cases tenderness was localised, and in four it was diffuse. In only one case was a mass palpable, but only after examination under anaesthetic after localisation by ^{111}In leucocyte scan. Ultrasound was more successful in this group, detecting seven out of 12 abscesses. Faecal granulocyte excretion in two patients was lower than in patients showing enteric drainage, 1.2% and 2.4% of the injected dose being recovered in a four day collection.

Thirteen of the 17 abscesses were surgically drained with no mortality (table III). Two pelvic abscesses drained spontaneously through the rectum with complete clinical resolution. Both remaining patients died, and abscesses were confirmed at necropsy. In one of these a decision against surgery was made because the patient was in established chronic renal failure. In the other case laparotomy failed to locate the abscess detected by scanning and subsequently the patient died.

Discussion

Hitherto the dynamics of leucocytes in abscesses have not been studied because of a lack of a suitable method. Enteric communication with abscesses has been shown by barium studies⁷ and more recently by CT.⁸ The degree of drainage, however, cannot be assessed with these techniques. The use of ^{111}In labelled leucocytes to follow leucocyte dynamics is a novel yet logical approach to this problem.

Recent studies in inflammatory bowel disease have validated the technique for studying leucocyte kinetics.^{9,10} There is avid binding of ^{111}In to leucocytes, rapid localisation in inflammatory sites, negligible elution, and essentially no reabsorption from the bowel. Measurement of faecal leucocyte excretion in abscesses with enteric communication yields values comparable to those in patients with active inflammatory bowel disease, while in patients without enteric communication excretion is roughly the same as in the irritable bowel syndrome.¹⁰ Although in many instances scanning suggested almost complete spontaneous drainage of the abscesses, it should be remembered that drainage of leucocytes is a continuous process, in equilibrium with replacement by fresh, unlabelled leucocytes. Repeat studies showed no improvement over periods of up to 26 days. In only one case with enteric drainage was there complete clinical spontaneous resolution of the abscess. At first sight these results showing failure of resolution in the enteric drainage group appear surprising, as these patients were spontaneously decompressing their abscesses. Unlike when abscesses are drained therapeutically percutaneously, however, the enteric communication site also provides an entry site for faecal material, so promoting chronicity.

The close association between absence of abdominal signs and evidence of spontaneous drainage of leucocytes through enteric communications with abscesses suggests that this is the mechanism accounting for the clinical presentation. By contrast, patients without enteric communications had a more classical presentation, all but one showing abdominal signs. The mean time to detection by scanning in the group with enteric drainage was longer than in the group without enteric drainage, presumably reflecting the lack of clinical suspicion associated with the atypical presentation. The proportion of abscesses with occult presentations is not known. In one large series 92 (64%) of 143 abscesses had no localising signs, but there was a degree of preselection, abscesses complicating appendicitis and peptic ulceration being excluded.¹¹ In a recent, smaller series 37.5% of patients had clinically silent abscesses, but again these cases were preselected.¹² The figure in our series for patients without abdominal signs was 33%. This series was also biased, however, as most abscesses diagnosed by ultrasound were drained without referral for ^{111}In leucocyte scanning. This limitation must also be considered when comparing the results of ^{111}In leucocyte scanning and ultrasound. Overall ^{111}In scanning was more accurate than ultrasound, and this was particularly evident in patients with enteric communication. The major reason for poor ultrasound results in the group appeared to be the lack of localising signs.

The mortality in the enteric drainage group (23%) was higher than in the closed abscess group (12%). The implication of this is difficult to judge because of the small numbers but probably it does reflect a true increase in mortality associated with occult presentations and consequently delayed detection. All three patients with enteric drainage who died were in a poor medical state directly attributable to the systemic effects of the abscess. In two immediate surgical intervention after detection of the abscess by ^{111}In leucocyte scanning rather than a trial of antibiotics may have changed the outcome. Of the two patients with closed abscesses who died, one would probably have survived had the abscess been found at laparotomy, while the other had deteriorating renal function before the development of the abscess.

These studies provide an explanation for some of the variations in clinical presentation of abdominal abscesses. Furthermore,

the recognition of an enteric communication with an abscess should direct management towards open surgical drainage rather than the percutaneous method¹³ or a trial of systemic antibiotics. On the basis of these results we propose that ¹¹¹In leucocyte scanning should be the primary investigation in patients without localising signs in whom an intra-abdominal abscess is suspected. Although ¹¹¹In leucocyte scanning is a relatively new imaging technique for detecting abscesses, its ability to visualise a wide range of inflammatory and infective conditions¹⁴⁻¹⁶ has resulted in its routine availability in many centres.

We are grateful to the physicians and surgeons at the Hammersmith Hospital for referring the patients included in this study. The ¹¹¹In chloride was kindly supplied by Amersham International. We are grateful to Miss Sally Barnes for expert help.

References

- 1 Altermeier WA, Culbertson WR, Fullen WD, *et al.* Intra-abdominal abscesses. *Am J Surg* 1973;125:70-9.
- 2 Ariel IM, Kazarian KK. *Diagnosis and treatment of abdominal abscesses*. Baltimore: Williams and Wilkins Co, 1971.
- 3 Taylor KKW, Wasson JF, De Graaf D, Rosenfeld A, Andriole VT. Accuracy

- of grey scale ultrasound diagnosis of abdominal and pelvic abscesses in 220 patients. *Lancet* 1978;ii:83-4.
- 4 Carroll B, Silverman PM, Goodwin DA, McDougall JR. Ultrasonography and indium-111 white blood scanning for the detection of intra-abdominal abscesses. *Radiology* 1981;140:155-60.
 - 5 Peters AM, Saverymutter SH, Karimjee S, Lavender JP. Indium-111 labelled leucocytes in the diagnosis of inflammatory disease. A comparison of 111-In oxine with 111-In acetylacetonate. *Br J Radiol* 1982;55:827-32.
 - 6 Danpure HJ, Osman S, Brady F. The labelling of blood cells in plasma with 111-In tropolonate. *Br J Radiol* 1982;55:247-9.
 - 7 Meyers MA. Diverticular disease. In: Mashak RH, Linder AE, Maklansky D, eds. *Radiology of the colon*. Philadelphia: Saunders, 1980:401-11.
 - 8 Chintapalli K, Thorsen M, Foley W, Unger G. Abdominal abscesses with enteric communication: CT findings. *American Journal of Roentgenology* 1983;141:27-8.
 - 9 Saverymutter SH, Peters AM, Hodgson HJF, Chadwick VS, Lavender JP. 111-Indium autologous leucocyte scanning: comparison with radiology for imaging the colon in inflammatory bowel disease. *Br Med J* 1982;285:255-7.
 - 10 Saverymutter SH, Peters AM, Pepys M, Lavender JP, Hodgson HJF, Chadwick VS. Quantitative fecal leucocyte excretion in the assessment of disease activity in Crohn's disease. *Gastroenterology* 1983;85:1333-9.
 - 11 Fry DE, Garrison N, Heitsch C, Calhoun K, Polk H. Determinants of death in patients with intra-abdominal abscess. *Surgery* 1980;88:517-23.
 - 12 Goldman R, Hunter T, Haber K. The silent abdominal abscess: role of radiologist. *American Journal of Roentgenology* 1983;141:21-5.
 - 13 van Sommenberg E, Ferrucci JT, Mueller PR, Wittenberg J, Simone JF, Malt RA. Percutaneous radiographically guided catheter drainage of abdominal abscess. *JAMA* 1982;247:190-2.
 - 14 Anderson JR, Spence RAJ, Laird JD, Ferguson WR, Kennedy TL. Initial experience with indium-111 autologous leucocyte imaging in patients with acute pancreatitis. *Br Med J* 1983;287:637-8.
 - 15 Gordon I, Vivian G. Radiolabelled leucocytes: a new diagnostic tool in occult infection/inflammation. *Arch Dis Child* 1984;59:62-6.
 - 16 Froelich JW, Swanson D. Imaging of inflammatory processes with labelled cells. *Semin Nucl Med* 1984;14:128-40.

(Accepted 8 October 1984)

SHORT REPORTS

Premature loss of bone in chronic anorexia nervosa

Most patients with anorexia nervosa eventually achieve a reasonable body weight, but some starve themselves for years.¹ After seeing two such patients with severe loss of bone we sought evidence of osteoporosis in other patients with longstanding anorexia nervosa.

Patients, methods, and results

We studied the case notes of 140 patients with anorexia nervosa admitted as inpatients. Evidence of osteoporosis was sought in those aged over 30 who had had the disease for over 10 years. We suspected osteoporosis if fracture had occurred after minimal trauma or vertebral crush fractures were seen on routine chest radiography. We studied three patients further, taking x ray films of the hands and vertebral column and measuring the metacarpal index. Distal forearm bone density was measured by photon absorptiometry.

Details of five patients with anorexia and bone loss

	Case 1	Case 2	Case 3	Case 4	Case 5
Age (years)	47	54	41	42	42
Duration of anorexia (years)	30	14	23	27	15
Usual weight (kg)	35	33	35	24	34
Serum calcium concentration (mmol/l)		2.49	2.34	2.00	2.51
Alkaline phosphatase activity (IU/l)			136	266	157
Luteinising hormone concentration (IU/l)				1.0	3.1*
No of vertebral fractures	2	3	2	0	2
Bone density (mg/cm ²)†			490	400	480
Metacarpal index‡			0.81	0.83	0.81
Trabecular bone volume (%)§			6	9	11

*Measured after patient had suddenly gained weight, reaching 56 kg.

†Normal range (mean (2 SD)) 620 (150) mg/cm².

‡Normal range (mean (2 SD)) 0.87 (0.15).

§Normal range (mean (2 SD)) 21.6 (5.6) %.

Conversion: SI to traditional units—Calcium: 1 mmol/l ≈ 4 mg/100 ml.

Biopsy specimens of the ilium were taken and serum calcium concentration, alkaline phosphatase activity, and luteinising hormone concentration measured.

Six of the patients had starved themselves for more than 10 years. One showed no evidence of fracture and could not be traced. Two had well developed dorsal kyphosis but could not be traced, and their x ray films were inadequate for assessment of osteoporosis. Three showed radiological evidence of vertebral crush fractures. The table gives details of the five

patients with definite bone loss (the two presenting patients and three with vertebral crush fractures). None of the bone biopsy specimens showed evidence of osteomalacia.

Comment

These five patients had exaggerated and premature bone loss. Does chronic anorexia nervosa predispose to osteoporosis, and if so by what mechanism? Three out of six patients who had had anorexia for over 10 years had osteoporosis, and two more had dorsal kyphosis suggesting vertebral bone loss. Such figures suggest an association between the two conditions. Although five patients had menstruated before the onset of anorexia, if anorectic patients never reach maturity this might cause low bone mass. Two patients, however, were well into adult life before the onset of anorexia, and recent work suggests that trabecular bone volume declines from the age of 20 or earlier.²

The causes of most forms of bone loss are controversial, but we suggest various factors. The first is malnutrition resulting from lack of protein for many years. The effects of malnutrition on bone are not clear. Cases of bone disease were described after the world wars, but the changes reported are difficult to assess. Recent work suggests that endosteal resorption of bone occurs in cases of severe malnutrition from lack of protein.³ Although man can adapt considerably to a low calcium intake, it may affect bone mass, and this mechanism might have acted in our patients. At least one of them (case 4), however, drank one to three pints of milk daily. We doubt that intake of vitamin D was important in view of the lack of histological evidence of osteomalacia. A more important factor may be longstanding oestrogen deficiency. Established anorexia is associated with low concentrations of gonadotrophin and oestrogen. Amenorrhoea in our patients had lasted for 14 to 30 years, and oestrogen deficiency can be assumed for most of this time, analogous to premature menopause. Bone loss after oophorectomy is well known and has been found in young women with amenorrhoea from other causes.⁴ Osteoporosis is more common among lean women.⁵ Chronic increases of serum cortisol concentration, which occur in anorectic patients, might exacerbate bone loss.

Chronic anorexia nervosa may be associated with a high prevalence of osteoporosis. If this is confirmed the possibility of using oestrogen replacement should be considered. Shorter periods of anorexia in young patients could predispose to osteoporosis in later life. Finally, patients with anorexia nervosa may be a useful group for studies of pathogenesis of bone loss as the effects of age can be examined and the mechanisms concerned will usually be reversed with successful restoration of weight and resumption of menses.