

Total parenteral nutrition and fluid/electrolyte therapy in the home

Nine years' experience¹

Ezra Steiger, M.D., F.A.C.S.
Cathleen Marein, R.N., B.S.N.
Patrice Misny, R.N., B.S.N.
Faith Srp, R.N.
Megan O'Neill, B.A.
Jane Paysinger, Pharm. D.

Nine years' experience with total parenteral nutrition and fluid/electrolyte therapy in the home are reviewed. A total of 110 patients could not maintain adequate nutrition, hydration, and/or electrolyte balance due to inadequate bowel length or absorption. Nutrition, clinical examination, and blood studies were corrected, and the patient was instructed in catheter care, fluid compounding and administration, self-monitoring, and management of complications. They were out of the hospital during 90% of their involvement in the program; readmissions were primarily related to underlying disease rather than complications. Home parenteral therapy can reduce or eliminate hospitalization for many patients and may allow return to a near-normal life style.

Index terms: Home care services • Parenteral feeding

Cleve Clin Q 52:317-327, Fall 1985

Home parenteral therapy (HPT), including home parenteral nutrition (HPN) and home fluid/electrolyte therapy (HFE), is a means of providing adequate nutrition and hydration in patients with compromised gastrointestinal (GI) tract function.¹⁻⁹ HPT is used for a variety of diseases resulting in insufficient bowel length and/or abnormal intestinal absorption which render the GI tract incompetent.¹⁰⁻¹⁶ The advent of HPT has allowed successful management of patients in the home for those diseases which otherwise would have resulted in repeated or prolonged hospitalization for correction of nutritional, fluid, and electrolyte imbalances. Prolonged venous access for administration of customized nutrient and electrolyte solutions is achieved through the use of a Silastic right atrial catheter.

¹ Departments of General Surgery (E.S., C.M., P.M., F.S.) and Hospital Pharmacy (J.P.), The Cleveland Clinic Foundation. Submitted for publication Feb 1985; accepted May 1985.

0009-8787/85/03/0317/11/\$3.75/0

Copyright © 1985, The Cleveland Clinic Foundation

Table 1. Home parenteral therapy (HPT) patient data

	No. of patients	% of total HPT patients	Age (yr)		Sex	
			Mean	Range	Male	Female
Crohn's disease	37	33	37	18-64	16	21
Radiation enteritis	24	22	56	40-73	6	18
Mesenteric vascular infarction	13	11	51	15-64	10	3
Small-bowel obstruction secondary to cancer	9	8	52	28-70	3	6
Ulcerative colitis involving the mucosa of other regions	4	4	37	14-69	3	1
Esophagitis secondary to bone marrow transplant	4	4	23	19-32	3	1
Small-bowel obstruction	4	4	53	40-65	2	2
Pseudo-obstruction	3	3	57	45-71	2	1
Malabsorption syndrome	3	3	53	37-66	2	1
Scleroderma of the GI tract	2	2	55	46-63	0	2
Gardner's syndrome (familial polyposis)	2	<2	38	28-48	1	1
Hirschsprung's disease	1	<1	39	...	0	1
Diverticulitis	1	<1	66	...	0	1
Congenital malrotation of the intestine	1	<1	22	...	1	0
Sclerosing cholangitis	1	<1	35	...	1	0
Toxic megacolon	1	<1	17	...	1	0
TOTALS	110	100	43	14-73	51	59

ter.¹⁷⁻²¹ Complete instruction of the patient and family in aseptic technique for catheter care and admixture and administration of parenteral solutions is mandatory. Continued success at home depends on regular patient follow-up and monitoring and compliance with the program.²² The purpose of this nine-year retrospective study was to describe the HPT patient population at The Cleveland Clinic Foundation (CCF) to evaluate their clinical course and to determine the efficacy of the program.

Materials and methods

A total of 110 patients were discharged on HPN or HFE from January 1976 through December 1984 (Table 1). There were 51 males and 59 females in this group, with a mean age of 45.3 ± 15.8 years (range, 14-73 years). Ninety-four patients (85%) required HPN and 16 patients (15%) required HFE. Patients with Crohn's dis-

ease, radiation enteritis, and ischemic bowel disease compromised 67% of the total patient population.

A consult to the hospital Nutrition Support Team (NST) initiated the evaluation for candidacy for HPT. The primary members of the NST included a physician, liaison psychiatrist, nurse, pharmacist, dietitian, and social worker who combined their efforts to thoroughly evaluate, prepare, and follow up the patient. Candidacy for HPT was considered only after dietary modifications, enteral supplements, and anti-diarrheal agents had been found to be unsuccessful in reducing fluid loss. The patient's present nutritional status was assessed and base-line laboratory values obtained to estimate fluid, electrolyte, and calorie requirements. The NST also evaluated the patient's emotional state, cognitive ability, motor skills, and readiness to learn. Team members conferred to determine whether HPT was

Table 2. Length of home parenteral therapy

	1-12 mo	13-24 mo	25-36 mo	37-48 mo	49-60 mo	61-72 mo	73-84 mo	85-96 mo	97-108 mo	Totals
HPN patients	41	16	11	11	6	2	2	1	4	94
HFE patients	10	1	1	1	0	1	2	1	1	16
TOTALS	51	17	12	12	6	3	0	2	5	110

Table 3. Reasons for discontinuation of HPT

Diagnosis	Death			HPT terminated	
	HPT associated (No. of patients)	Underlying disease and/or complicating factors (No. of patients)	Concomitant illness (No. of patients)	Bowel continuity surgically restored (No. of patients)	Bowel continuity medically restored (No. of patients)
Crohn's disease	0	3	5	5	3
Radiation enteritis	0	12*	1	3	0
Ischemic bowel	0	1	3	0	0
Small-bowel obstruction sec- ondary to cancer	0	6	0	1	0
Ulcerative colitis involving the mucosa of other regions	0	0	2	1	1
Esophagitis secondary to bone- marrow transplant	0	0	0	0	4
Pseudo-obstruction	0	0	1	0	0
Small-bowel obstruction	0	2	0	1	1
Malabsorption	0	1	0	1	1
Scleroderma (GI tract)	0	1	0	0	0
Congenital malrotation of bowel	0	0	0	0	1
Sclerosing cholangitis	0	0	0	0	1
Toxic megacolon	0	0	0	1	0
Gardner's syndrome	0	0	0	0	1
TOTALS	0	26 (41%)	12 (19%)	13 (20%)	13 (20%)

* Eight patient deaths were attributable to recurrent cancer.

appropriate and feasible in each case. Placement of a Hickman/Broviac right atrial catheter was done after candidacy for HPT was determined.²³ Instruction in catheter care, solution preparation and administration, and self-monitoring techniques by the NST nurse and pharmacist began once the patient was physically and emotionally prepared to learn.^{24,25} The instruction period lasted an average of two to three weeks and was considered complete when the patient demonstrated competency and understanding of the HPT procedures and management of possible complications. Patients were stabilized on 1 to 6 L of fluid per day and gradually tapered off from a 24-hour to an overnight infusion schedule during this training period. The patients were discharged once training was complete, home-going solutions were stabilized, and both patient and family were psychologically prepared. While at home, 82 patients (75%) mixed their own HPT solutions and 28 (25%) received pre-mixed solutions from the home supply vendor. Follow-up examinations, review of home self-monitoring records, and blood studies were done at two-week intervals, progressing to once a month based on patient stability. Routine laboratory studies included blood glucose, electrolytes,

blood urea nitrogen (BUN), creatinine, calcium, phosphorus, and magnesium levels. Once a month, a complete blood count, prothrombin time, serum zinc level, and liver function tests were done, with vitamins, trace elements, essential fatty acids, and urinary calcium levels being monitored at six-month intervals.

Results

The average length of HPT for all 110 patients in our program was 26.1 months (range, 1–105), totaling 2,435 months (203 years) for HPN patients and 410 months (34 years) for patients receiving HFE (Table 2). The current active patient population, consisting of 39 HPN and 7 HFE patients, has averaged 36 months on HPT. Patients no longer in the program due to discontinuance of therapy or death (Table 3) had been on HPT for an average of 18.5 months.

Eighty-seven patients were rehospitalized a total of 324 times for an average of 3.76 ± 4.52 admissions per patient, totaling 6,661 days of rehospitalization. Twenty-three patients were never rehospitalized. Only 1,793 of the total of 6,661 rehospitalization days were related to HPT (120 admissions), or approximately 15.3 ± 14.2 days per admission. Non-HPT-related admissions

totaled 4,868 days (204 admissions), or approximately 24.4 ± 24.1 days per admission. Thus only 27% of admissions were related to HPT. In addition to rehospitalization, we have noted previously that overall mortality was also related to the underlying disease process rather than to HPT.²⁶ Of 187 catheters inserted, mean catheter life was 15.34 ± 17.57 months (range, 0.25–99). Seventy-one patients (65%) have never required catheter replacement.

Discussion

In reviewing our nine years of experience with the HPT program, we found the minimal rehospitalization time to be noteworthy. Our 110 patients were out of the hospital for 68,790 days, with rehospitalization accounting for only 10% of their time in the program. The most frequent cause for readmission was the patient's primary disease process and its associated complications. The majority of HPT-related rehospitalizations were for actual or suspected catheter sepsis and other catheter-related complications, with only infrequent readmissions for fluid/electrolyte abnormalities. The fact that our patients were able to spend 90% of their time at home is significant and can be attributed to the concerted efforts of the multidisciplinary team, their extensive knowledge of HPT and its complications, and the thorough instruction and follow-up of each patient. Continued work in the study and understanding of HPT and improvement of products and techniques are necessary in order to enhance the efficacy of HPT and further reduce rehospitalizations.

Catheter insertion and care

Prior to the operation, one of the NST nurses shows the patient a catheter, explains the insertion technique, and marks the exit site on the patient's skin with gentian violet. The catheter exits to the right or left of the sternum along the nipple line at a level that can be easily seen by the patient. It should not exit near a draining wound or intestinal drainage bag, so as to minimize the possibility of contamination.

We employ a durable silicone rubber catheter (Hickman or Broviac) which is approximately 90 cm long. The two catheters differ in that the Broviac catheter has a narrower internal lumen than the Hickman catheter (1.0 versus 1.6 mm). Both function and are managed in the same manner, but the Broviac catheter is more likely

to become occluded,²⁷ and blood samples may be more difficult to obtain due to collapse of the internal lumen upon aspiration. On the other hand, the Hickman catheter may theoretically be more thrombogenic because of its larger size.

Bilateral subclavian venograms are obtained prior to catheter placement to determine the availability of vessels in the upper thoracic venous system. Veins usually selected for catheter insertion include the cephalic, internal or external jugular, and the muscular branch of the subclavian. In the event of thrombosis involving the superior vena cava, a saphenous approach to the inferior vena cava may be necessary.²⁸ Subclinical thrombosis of the subclavian vein, occasionally noted on preoperative venograms, would preclude its use for permanent catheter placement; this is seen most commonly in patients who have had multiple temporary catheters placed for in-hospital total parenteral nutrition (TPN) or central venous pressure monitoring. Catheter placement into the azygous system via the intercostal vein is a relatively new approach which has reportedly been successful in children.²⁹ We have also used direct right atrial catheterization via a thoracotomy incision when all other venous access sites have been depleted.³⁰

Catheters must be cared for using aseptic techniques in order to prevent contamination and sepsis. Hickman/Broviac dressings are changed daily by the patient, or more frequently if they become soiled or wet. Localized exit site infections are treated by changing the dressing twice a day and administering intravenous (IV) antibiotics. We have been successful in resolving such infections with this protocol if the organism has not invaded the Dacron cuff within the subcutaneous tunnel. Intravascular catheter sepsis is suspected if positive blood cultures are obtained through the catheter and/or no other source of sepsis can be determined. Antibiotics are usually not necessary if the catheter is removed promptly. A new catheter can be placed in five to seven days if two consecutive sets of peripheral blood cultures prove negative. If the catheter must be removed due to confirmed sepsis, catheter care and mixing procedures are reviewed. Subcutaneously implanted venous access devices such as the Port-a-Cath, Infuse-a-Port, and Mediport have recently been reported to be successful for infusion of HPT fluids,^{31,32} but require daily skin puncture and have not yet been widely accepted.

To prevent the catheter from becoming occluded, it is flushed with 2 mL of heparin (100 U/mL) at the completion of the infusion. Should occlusion occur, patency may be restored by infusing a dilute solution of streptokinase or urokinase. Streptokinase, a protein derived from group C beta hemolytic streptococci, acts by forming a complex with plasminogen which then converts plasminogen to plasmin. Urokinase, an enzyme produced by the human kidney, directly converts plasminogen to plasmin. In both instances, the plasmin then degrades the fibrin within the clots. Anaphylaxis and systemic fibrinolysis are potential risks associated with the use of streptokinase, however, the use of a very dilute solution and aspiration of any residual drug in the catheter minimizes these risks. Urokinase is considered nonantigenic, but the increased cost of this product outweighs that benefit and makes streptokinase the thrombolytic agent of choice for preventing catheter occlusions. Using a tuberculum syringe, a mixture of streptokinase and normal saline, producing a concentration of 2,500 U/mL of streptokinase, is gently instilled in a volume equal to the internal volume of the catheter. The solution should remain in contact with the clot for 30 minutes, after which repeated aspirations of the residual clot and drug are made every five minutes until patency is restored. If the catheter is not patent after several attempts, the solution is left in the catheter for 24 hours.³³

To minimize fibrin formation within the catheter and decrease the incidence of venous thrombosis associated with infusion of hypertonic solutions, 1,000 U of heparin is added to each liter of HPT solution. Venograms should be obtained if thrombosis is suspected. Thrombosis can be confirmed if venous obstruction and collateralization are noted upon injection of contrast material. Distension of veins in the shoulder and anterior chest walls, swelling of the arm, and shoulder pain on the involved side are typical of subclavian vein thrombosis. In addition, neck or throat pain, puffiness of the face, extensive tearing, and rhinorrhea may be noted. While heparin has been found to be ineffective in achieving thrombolysis, success may be attained with local infusion of streptokinase.^{34,35} However, in most cases, treatment proves futile and the catheter must be removed.

HPT patients are provided with a temporary repair kit and instructed in its use should catheter breakage occur. The kit contains all supplies

needed for temporary repair, including a sterile blunted needle whose gauge corresponds to that of the catheter: a 15 gauge for the Hickman catheter, 18 gauge for the Broviac, and 22 gauge for the pediatric Broviac. The catheter is cut with sterile scissors so that the site of the breakage is removed. The blunted needle is inserted into the lumen of the catheter, forming a new end on the original catheter. Patients are told that this repair is only temporary, and that they should make an appointment with the physician who placed the catheter so that it can be permanently repaired; this involves splicing a new end onto the remaining segment of the original catheter. We have not observed any increase in catheter sepsis rates associated with such repairs.³⁶

Patient/family instruction

The goal of the HPT program is to facilitate a smooth transition of the patient from the hospital to the home. In order to achieve the goal, the patient and/or family must be educated to a level of independence and develop a sense of confidence. Thorough evaluation of the patient prior to instruction is needed to determine any cognitive, psychological, and/or physical impairments in order to plan for learning needs and adapt instruction materials as necessary. The patient is trained as the primary care giver; a family member is involved, if willing, but only as a back-up. This not only prevents family members from feeling burdened with the primary responsibility of the patient's care, but also helps to alleviate the patient's loss of autonomy and forced dependency. If absolutely necessary, a family member can be taught to be solely responsible, but never anyone who lives outside the home. Some patients may require total care for HPT; and although resources are available, we have found only a limited number of extended care facilities that would accept patients requiring HPN or HFE.

Instruction can be expedited with the use of a Cath-Train vest, a teaching tool which simulates the patient's own catheter. The vest allows the patient to become proficient in working with the catheter and permits unsupervised practice between lessons. Instructions need to be written in simple concise sentences employing very basic terminology. A format of instructions written in step-by-step fashion, separate from definitions, complications, and discussions, is easier for the

Table 4. Contents of a typical HPN solution

Nutrient	1 L	2 L
FreAmine III 8.5%*	500 mL	1,000 mL
Dextrose 50%	500 mL	1,000 mL
Calcium gluconate injection (0.46 mEq/mL)	9.2 mEq	18.4 mEq
Magnesium sulfate injection (4.06 mEq/mL)	8.1 mEq	16.2 mEq
Potassium chloride injection (2 mEq/mL)	20.0 mEq	40.0 mEq
Potassium phosphate injection (4.4 mEq K/mL)	10.0 mEq	10.0 mEq
Sodium acetate injection (2 mEq/mL)	20.0 mEq	40.0 mEq
Sodium chloride injection (4 mEq/mL)	50.0 mEq	100.0 mEq
Heparin sodium (1,000 U/mL)	1,000 U	2,000 U
Multiple vitamins (N = 12)	10.0 mL	10.0 mL
Multiple trace elements concentrate	1.0 mL	1.0 mL
Selenium injection (40 µg/mL)	120 µg	120 µg

* American McGaw Laboratories, Irvine, Calif.

patient to follow than instructions written in paragraph form or intermingled with diagrams. Memorization of procedures is not necessary as it extends the learning time and increases the patient's anxiety. The patient must be able to simply follow the written instructions. One procedure is demonstrated at a time, practice allowed, and competency nearly mastered prior to advancing to the next procedure. Evaluation is conducted continuously, with immediate feedback provided and appropriate changes made. Instruction is complete when the learner can perform all procedures independently and can respond appropriately when posed with actual or hypothetical problems or complications.

Solution/additive preparation and administration

HPT solutions can be either mixed by the patient or pre-mixed by the home supply vendor. Since pre-mixed HPT solutions did not become available until 1980–1981, prior to this time, all patients had to be instructed in self-mixing procedures. At the CCF, most patients who are expected to be on HPT permanently (or at least more than six months) learn to mix their own dialysis solutions; patients and family members who are unable to learn the mixing techniques, or patients who will only require HPT for six months or less, receive pre-mixed solutions through the home supply vendor. The additional cost of the vendor-prepared solutions is offset by the savings associated with a shorter in-hospital stay for training.

The base solutions and additives used for HPT are basically the same as the TPN and fluid/electrolyte solutions used in the hospital. Fluid composition and nutrient requirements are estab-

lished, based on each patient's individual nutritional assessment, daily weight, intake and output, BUN, and creatinine. HPT patients may require 1–6 L of nutrient/IV crystalloid solutions to maintain homeostasis, depending on the state of functional impairment of the bowel and losses through a GI tract fistula or stoma. While HFE patients require only IV crystalloids, with appropriate additives to maintain fluid and electrolyte balance and adequate renal function, HPN patients need amino acids, dextrose, electrolytes, vitamins, minerals, and trace elements to maintain nutritional homeostasis. Many HPN patients require additional IV crystalloids to control fluid and electrolyte balance when high-output stomas and/or fistulas are present. Most HPN patients must have a minimum of 2 L of nutrient solutions. A typical 2-L HPN formula containing 4.25% amino acids and 25% dextrose as a base solution, along with additives, is shown in *Table 4*. Vitamins and trace elements are added to HPT solutions in accord with the American Medical Association (AMA) Nutrition Advisory Group recommendations for parenteral use (*Tables 5 and 6*).^{37,38} Since all fat- and water-soluble vitamins except K are contained in the single multivitamin preparation, the patients are instructed to take 10 mg of vitamin K intramuscularly twice a month.

Deficiencies of trace elements such as zinc, copper, chromium, and selenium in patients receiving TPN have been documented.^{41–43} Since 1976, our HPT patients have added not only these trace elements but also manganese to 1 L of their daily infusate to maintain adequate levels. Dosages were originally based on the oral recommendations of the recommended dietary al-

Table 5. Intravenous vitamin additives for TPN

Vitamin	Multiple vitamins (N = 12) (per 10 mL)	AMA guidelines for adults ³⁷
A	3,300 IU	3,300 IU
D	200 IU	200 IU
E	10 IU	10 IU
K
C (ascorbic acid)	100.0 mg	100.0 mg
B ₁ (thiamin)	3.0 mg	3.0 mg
B ₂ (riboflavin)	3.6 mg	3.6 mg
Niacin	40.0 mg	40.0 mg
B ₆ (pyridoxine)	4.0 mg	4.0 mg
Pantothenic acid	15.0 mg	15.0 mg
Folacin	0.4 mg	0.4 mg
B ₁₂ (cyanocobalamin)	5.0 µg	5.0 µg
Biotin	60.0 µg	60.0 µg

lowances guidelines, but have been re-evaluated yearly as more definitive requirements³⁹ and clinically manifested deficiency states have appeared. Since 1978–1979, when serum trace mineral analysis using atomic absorption spectrophotometry first became available at the CCF, patient monitoring has progressed to include trace metal analysis every six months as a routine protocol. Dosages of the trace minerals described in *Table 6* have maintained serum zinc and copper levels within normal limits. While chromium levels in our patients tended to be in the very low normal range, manganese levels were consistently elevated, indicating that AMA recommendations for manganese in HPT patients may be excessive. Manganese deficiency has not been reported in HPT patients to our knowledge. Selenium levels tended to be low. In a previous monograph describing selenium use in adult and pediatric patients receiving HPT,⁴⁰ we indicated that 63% of our patients occasionally had low selenium levels. Reviewing a smaller sample of patients (N = 28)

revealed that 39% of them maintained normal levels at the prescribed dosage. When the remaining patients were placed on higher doses, only 35% demonstrated increased selenium levels. However, despite low levels of chromium and selenium and elevated manganese levels for varying periods of time, none of the patients seen during the course of our program has demonstrated clinical trace mineral deficiency^{41–43} or toxicity. Exogenous administration of iron-dextran complex (Imferon) may be given as required to maintain normal hematologic values. However, test dosages should be given prior to infusion of the therapeutic dose to test for and guard against the occurrence of an anaphylactic-type reaction. Regular insulin should be added directly to the HPN solution, if necessary, to control serum glucose levels during infusion: they should be ≤200–250 mg% during the infusion, and return to normal within two hours post-infusion. The cyclic, eight-to-12-hour overnight infusion schedule necessitates the use of a volu-

Table 6. Trace element additives for TPN

Trace element solutions (per mL)	AMA guidelines* ³⁸		
	Stable adults	Catabolic adults	Adults with intestinal losses
Zinc	5.0 mg	2.5–4.0 mg	Additional 2.0 mg Additional 12.2 mg per L of small-bowel fluid lost; 17.1 mg per kg of stool or ileostomy output
Copper	1.0 mg	0.5–1.5 mg	...
Chromium	10.0 g	10.0–15.0 µg	20.0 µg
Manganese	0.5 mg	0.15–0.8 mg	...

* Multiple trace element concentrate, LymphoMed, Melrose Park, Ill.

Table 7. Patients with metabolic bone disease

Case	Age (yr) and sex	Clinical symptoms	Bone biopsy	Urine calcium balance	Serum calcium	Vitamins		Diagnosis
						25-(OH) ₂ D ₂	1,25-(OH) ₂ D ₃	
1	61, F	Multiple spontaneous fractures	Active osteopenia	NA	Below normal	Normal	Normal	Radiation enteritis following treatment for cancer
2	67, F	Bone pain and fractured vertebrae	Active osteopenia	Negative	Normal	High normal	Low normal	Short bowel syndrome, diverticulitis
3	28, M	Spontaneous hip fracture	Not done	NA	Normal	NA	NA	Crohn's disease
4	26, F	Rib fracture	Not done	NA	Normal	NA	NA	Crohn's disease
5	51, F	Bone pain	Normal	Negative	Low normal	Elevated	Below normal	Radiation enteritis following treatment for ovarian cancer
6	64, F	Spontaneous hip fracture	Inactive osteoporosis	Positive	Normal	Elevated	Below normal	Radiation enteritis following treatment for ovarian cancer
7	19, M	Bone pain and compression fracture	Active osteopenia/osteomalacia	Positive	Normal	Elevated	Elevated	Crohn's disease

metric infusion pump with appropriate alarm systems, which assures safe and accurate delivery of solutions while the patient is asleep.⁴⁴⁻⁴⁷

Final in-line filtration of particulate matter and bacteria is achieved by the addition of a 0.22- μ air-eliminating filter to the infusion system.⁴⁸⁻⁵⁰ Solutions may be administered using a Y-tube set for a separated 2 L of fluid, admixture of the entire volume of solution into the appropriate size 2- or 3-L bag, or both. Such set-ups will allow the patient to hang the entire volume of solution at one time, allowing for an uninterrupted night's sleep. During the last hour of infusion, the prescribed infusion rate is reduced by half to taper off the quantity of dextrose infused, thereby preventing reactive hypoglycemia.

In the majority of HPT patients requiring high caloric solutions (HPN group), fat must be provided intravenously in addition to dextrose and protein to prevent essential fatty acid deficiency (EFAD). This condition, characterized by an increase of 5,8,11-eicosatrienoic acid, may develop within two weeks of fat-free parenteral nutrition.⁵¹ Symptoms could include eczematous desquamative dermatitis, hair loss, thrombocytopenia, and possibly impaired wound healing and

hepatic dysfunction.⁵²⁻⁵⁵ Providing 2%–3% of the patient's total calories as linoleic acid can prevent these sequelae.^{56,57} Infusion of 500 mL of 10% fat emulsion over a period of four hours twice a week is generally sufficient for maintaining essential fatty acid levels; however, they should be monitored periodically to adjust for individual patient needs. A 20% fat emulsion is also available for IV use. However, infusion of a 20% fat emulsion over less than the recommended eight hours was associated with priapism in 2 of our male patients. Although vascular thrombosis is suspected, the exact etiology is unclear.⁵⁸

Metabolic bone disease

Metabolic bone disease, characterized by bone pain and spontaneous fractures (nonattributable to physical mishap) and diagnosed pathologically by osteoporosis and osteomalacia bone histology, has been documented in patients receiving HPN.⁵⁹⁻⁶² Previous observations^{59,60,63} have indicated that HPN is a primary causative factor in the development of these histological abnormalities despite the underlying disease. Review of the medical histories of the 110 CCF patients

treated to date revealed metabolic bone disease in 7 (6.4%) (Table 7), documented clinically by bone pain (1 patient), spontaneous fractures (4 patients), or both (2 patients). Patients included 2 men and 5 women, with the latter group including 4 who were post-menopausal and/or had undergone total abdominal hysterectomy at least 10 years earlier; the mean age was 45.5 ± 20.3 years over a range of 26–67. Three patients had gastrointestinal malabsorption secondary to Crohn's disease, 3 had radiation enteritis following treatment for ovarian cancer, and 1 had diverticulitis; all had undergone multiple operations resulting in short-bowel syndrome one to 10 years prior to HPN. Bone pain developed in 3 patients within six months of initial discharge on HPT. Two patients experienced compression fractures of the vertebrae shortly after onset of bone pain. Four patients without previous pain had spontaneous fractures of the hip or ribs eight to 54 months after initial discharge on HPT.

Four women and 1 man in our series underwent tetracycline labeling and subsequent iliac crest bone biopsy to aid in diagnosis and treatment. Biopsy findings, ranging from normal to severe inactive osteoporosis, were correlated with urinary calcium balance and levels of serum calcium, 25-hydroxy vitamin D and 1,25-dihydroxy vitamin D whenever possible. Based on this analysis, recommendations were made for increased IV calcium to maintain a positive urinary calcium balance⁶⁴ and/or increased vitamin D supplementation.^{62,65} Only one patient receiving increased calcium has demonstrated long-term relief of symptoms.

Just as the exact etiology of metabolic bone disease in HPT has remained a mystery, treatment has been varied and experimental.^{59,62,63,65,66}

Length of therapy

Depending upon their underlying disease, degree of GI tract dysfunction, and length of bowel remaining, patients may require HPT on a permanent or temporary basis. In our experience, patients with radiation enteritis or ischemic bowel disease due to mesenteric infarction usually remain on HPT permanently, whereas patients with Crohn's disease and/or short-bowel syndrome with subsequent bowel adaptation may only require HPT temporarily. Both groups of patients are able to avoid prolonged hospitaliza-

tion with HPT and can return home. Patients who have catastrophic enterocutaneous fistulas and require prolonged bowel rest for resolution of inflammation prior to surgery have also been managed with HPT; after three to six months, surgery is performed, if necessary, to resect persistent fistulas and/or re-establish temporarily interrupted GI tract continuity.^{14,67}

Conclusion

The 110 patients taking part in the CCF HPT program were able to spend a significant length of time at home with a low incidence of morbidity and mortality. All had significant underlying disease¹ that would otherwise have required either repeated or permanent hospitalization. A satisfactory fluid balance and nutritional status can be maintained with an overnight infusion of customized solutions. Accurate record keeping, routine laboratory monitoring, regular follow-up visits, and continued communication between the patient, patient's family, and NST members are essential to ensure patient safety and well-being as well as the success of the program.

Ezra Steiger, M.D.
Department of General Surgery
The Cleveland Clinic Foundation
9500 Euclid Ave.
Cleveland, OH 44106

References

1. Dudrick SJ, Wilmore DW, Vars HM, Rhoads JE. Long-term parenteral nutrition with growth, development, and positive nitrogen balance. *Surgery* 1968; **64**:134–141.
2. Ivey M, Riella M, Mueller W, Scribner B. Long-term parenteral nutrition in the home. *Am J Hosp Pharm* 1975; **32**:1032–1036.
3. Bordos DC, Cameron JL. Successful long-term intravenous hyperalimentation in the hospital and at home. *Arch Surg* 1975; **110**:439–441.
4. Riella MC, Scribner BH. Five years' experience with a right atrial catheter for prolonged parenteral nutrition at home. *Surg Gynecol Obstet* 1976; **143**:205–208.
6. Grundfest S, Steiger E, Sattler L, Washko S, Wateska L. The current status of home total parenteral nutrition. *Artif Organs* 1979; **3**:156–160.
7. Steiger E, Grundfest S. A review of home hyperalimentation. *Contemp Surg* 1979; **15**:33–40.
8. Grundfest S, Steiger E. Experience with the Broviac catheter for prolonged parenteral alimentation. *JPEN* 1979; **3**:45–47.

9. Grundfest S, Steiger E. Home parenteral nutrition. *JAMA* 1980; **244**:1701-1703.
10. Byrne WJ, Halpin TC, Asch MJ, Fonkalsrud EW, Ament ME. Home total parenteral nutrition: an alternative approach to the management of children with severe chronic small bowel disease. *J Pediatr Surg* 1977; **12**:359-366.
11. Dudrick SJ, MacFadyen BV Jr, Souchen EA, Englert DM, Copeland EM III. Parenteral nutrition techniques in cancer patients. *Cancer Res* 1977; **37**:2440-2450.
12. Faulk DL, Anuras S, Freeman JB. Idiopathic chronic intestinal pseudo-obstruction. Use of central venous nutrition. *JAMA* 1978; **240**:2075-2076.
13. Fleming CR, McGill DB, Berkner S. Home parenteral nutrition as primary therapy in patients with extensive Crohn's disease of the small bowel and malnutrition. *Gastroenterology* 1977; **73**:1077-1081.
14. Oakley JR, Steiger E, Lavery IC, Fazio VW. Catastrophic enterocutaneous fistulae: the role of home hyperalimentation. *Cleve Clin Q* 1979; **46**:133-136.
15. Stewart GR. Home parenteral nutrition for chronic short bowel syndrome. *Med J Aust* 1979; **2**:317-319.
16. Grischkan D, Steiger E, Fazio V. Maintenance of home hyperalimentation in patients with high-output jejunostomies. *Arch Surg* 1979; **114**:838-841.
17. Scribner BH, Cole JJ, Christopher TG, Vizzo JE, Atkins RC, Blagg CR. Long-term total parenteral nutrition. The concept of an artificial gut. *JAMA* 1970; **212**:457-463.
18. Byrne WJ, Ament ME, Burke M, Fonkalsrud E. Home parenteral nutrition. *Surg Gynecol Obstet* 1979; **149**:593-599.
19. Goldberger JH, Deluca FG, Wesselhoeft CW, Randall HT. A home program of long-term total parenteral nutrition in children. *J Pediatr* 1979; **94**:325-328.
20. Solassol C, Joyeux H, Etco L, Pujol H, Romieu C. New techniques for long-term intravenous feeding: an artificial gut in 75 patients. *Ann Surg* 1974; **179**:519-522.
21. Broviac JW, Cole JJ, Scribner BH. A silicon rubber right atrial catheter for prolonged parenteral alimentation. *Surg Gynecol Obstet* 1973; **136**:602-606.
22. Hughes BA, Fleming CR, Berkner S, Gaffron R. Patient compliance with a home parenteral nutrition program. *JPEN* 1980; **4**:12-14.
23. Lees CD, Steiger E, Hooley RA, et al. Home parenteral nutrition. *Surg Clin North Am* 1981; **61**:621-633.
24. Gullledge AD, Gipson WT, Steiger E, Hooley R, Srp F. Home parenteral nutrition for the short bowel syndrome. Psychological issues. *Gen Hosp Psychiatry* 1980; **2**:271-281.
25. Srp F, Steiger E, Montague N, et al. Patient preparation for cyclic home parenteral nutrition: a team approach. *Nutr Support Serv* 1981; **1**:31-34.
26. Steiger E, Srp F. Morbidity and mortality related to home parenteral nutrition in patients with gut failure. *Am J Surg* 1983; **145**:102-105.
27. Srp F, Montague N, Steiger E, O'Neill M. A comparison of two types of long-term silicone right atrial catheters (RAC) (abst). *JPEN* 1980; **4**:597.
28. Wilson SE, Owens ML. *Vascular Access Surgery*. Chicago, Year Book, 1980, p 24.
29. Newman BM, Cooney DR, Karp MP, Jewett TC Jr. The intercostal vein: an alternate route for central venous alimentation. *J Pediatr Surg* 1983; **18**:732-733.
30. Oram-Smith JC, Mullen JL, Harken AH, Fitts WT Jr. Direct right atrial catheterization for total parenteral nutrition. *Surgery* 1978; **83**:274-276.
31. Baptista RJ, Lahey MA, Bistran BR, et al. Periodic reassessment for improved, cost-effective care in home total parenteral nutrition: a case report. *JPEN* 1984; **8**:708-710.
32. Goodman MS, Wickam R. Venous access devices: an overview. *Oncol Nurs Forum* 1984; **11**:16-23.
33. Hurtubise MR, Bottino JC, Lawson M, McCredie KB. Restoring patency of occluded central venous catheters. *Arch Surg* 1980; **115**:212-213.
34. Rubenstein M, Cregar WP. Successful streptokinase therapy for catheter-induced subclavian vein thrombosis. *Arch Intern Med* 1980; **140**:1370-1371.
35. Smith NL, Ravo B, Soroff HS, Khan SA. Successful fibrinolytic therapy for superior vena cava thrombosis secondary to long-term total parenteral nutrition. *JPEN* 1985; **9**:55-57.
36. Montague N, Srp F, Steiger E. Emergency catheter repairs in the home parenteral nutrition (HPN) patient (abst). *JPEN* 1980; **4**:597.
37. American Medical Association Department of Foods and Nutrition. Multivitamin preparations for parenteral use. A statement by the Nutrition Advisory Group. *JPEN* 1979; **3**:258-262.
38. American Medical Association. Guidelines for essential trace element preparations for parenteral use. A statement by the Nutrition Advisory Group. *JPEN* 1979; **3**:263-267.
39. AMA Department of Foods and Nutrition. Guidelines for essential trace element preparations for parenteral use. A statement by an expert panel. *JAMA* 1979; **241**:2051-2054.
40. Steiger E, Helbley M, Zlotkin SH. Selenium in total parenteral nutrition: clinical experience in two medical centers. Melrose Park, Ill., LymphoMed, 1982.
41. Solomons NW. On the assessment of trace mineral nutriture in patients on total parenteral nutrition. *Nutr Support Serv* 1981; **1**:13-16.
42. Johnson RA, Baker SS, Fallon JT, et al. An occidental case of cardiomyopathy and selenium deficiency. *N Engl J Med* 1981; **305**:1210-1212.
43. Van Rij AM, Thomson CD, McKenzie JM, Robinson MF. Selenium deficiency in total parenteral nutrition. *Am J Clin Nutr* 1979; **32**:2076-2085.
44. Goldfarb IW, Slater H. Techniques of administration. *Am J Intra Ther Clin Nutr* 1981; **8**:25-34.
45. Fabry J. Infusion monitoring devices. *NITA* 1981; **4**:366-367.
46. Pipp TL. Intravenous infusion pump justification and selection and utilization. *Infusion* 1978; **2**:45-58.
47. Turco SJ. Mechanical and electronic equipment for parenteral and enteral use: an update. *Am J Intra Ther Clin Nutr* 1982; **9**:9-43.
48. Schneider PJ. Should inline filters be used when administering TPN solution? *Infusion* 1982; **6**:66-67.
49. National Coordinating Committee on Large Volume Parenterals. Problems and benefits of in-line filtration. *Am J Intra Ther Clin Nutr* 1980; **7**:45-47.
50. Lorenzen BA. Final filtration. *NITA* 1981; **4**:322-327.
51. Goodgame JT, Lowry SF, Brennan MF. Essential fatty acid deficiency in total parenteral nutrition: time course of development and suggestions for therapy. *Surgery* 1978; **84**:271-277.
52. Collins FO, Sinclair AJ, Royle JP, Coats DA, Maynard AT, Leonard RF. Plasma lipids in human linoleic acid deficiency. *Nutr Metab* 1971; **13**:150-167.
53. Richardson TJ, Sgoutas D. Essential fatty acid deficiency in four adult patients during total parenteral nutrition. *Am J Clin Nutr* 1975; **28**:258-263.
54. Paulsrud JR, Pensler L, Whitten CF, Stewart S, Holman

- RT. Essential fatty acid deficiency in infants induced by fat-free intravenous feeding. *Am J Clin Nutr* 1972; **25**:897-907.
55. Caldwell MD, Jonsson HT, Othersen HB. Essential fatty acid deficiency in an infant receiving prolonged parenteral alimentation. *J Pediatr* 1972; **81**:894-898.
 56. Tashiro T, Ogata H, Yokoyama H, Mashima Y, Itoh K. The effect of fat emulsion (Intralipid) on essential fatty acid deficiency in infants receiving intravenous alimentation. *J Pediatr Surg* 1976; **11**:505-515.
 57. Barr LH, Dunn GD, Brennan MF. Essential fatty acid deficiency during total parenteral nutrition. *Ann Surg* 1981; **193**:304-311.
 58. Klein EA, Montague DK, Steiger E. Priapism associated with the use of intravenous fat emulsion: case reports and postulated pathogenesis. *J Urol* 1985; **133**:857-859.
 59. Shike M, Harrison JE, Strutridge WC, et al. Metabolic bone disease in patients receiving long-term total parenteral nutrition. *Ann Intern Med* 1980; **92**:343-350.
 60. Klein GL, Targoff CM, Ament ME, et al. Bone disease associated with total parenteral nutrition. *Lancet* 1980; **2**:1041-1044.
 61. Klein GL, Horst RL, Norman AW, Ament ME, Slatopolsky E, Coburn JW. Reduced serum levels of $1\alpha, 25$ -dihydroxy-vitamin D during long-term total parenteral nutrition. *Ann Intern Med* 1981; **94**:638-643.
 62. Klein GL, Cannon RA, Diament MD, et al. Infantile vitamin D-resistant rickets associated with total parenteral nutrition. *Am J Dis Child* 1982; **136**:74-76.
 63. Shike M, Strutridge WC, Tam CS, et al. A possible role of vitamin D in the genesis of parenteral-nutrition-induced metabolic bone disease. *Ann Intern Med* 1981; **95**:560-568.
 64. Wittine MF, Freeman JB. Calcium requirements during total parenteral nutrition in well-nourished individuals. *JPEN* 1977; **1**:152-155.
 65. Sloan GM, White DE, Brennan MF. Calcium and phosphorus metabolism during total parenteral nutrition. *Ann Surg* 1983; **197**:1-6.
 66. Ott SM, Maloney NA, Coburn JW, et al. The prevalence of bone aluminum deposition in renal osteodystrophy and its relation to the response to calcitriol therapy. *N Engl J Med* 1982; **307**:709-713.
 67. Byrne WJ, Burke M, Fonkalsrud EW, Ament ME. Home parenteral nutrition: an alternative approach to the management of complicated gastrointestinal fistulas not responding to conventional medical or surgical therapy. *JPEN* 1979; **3**:355-359.