BIFA British Intestinal Failure Alliance Special Interest Group
September 2026
BAPEN

British Intestinal Failure Alliance (BIFA) Position Statement

Haematological and Biochemical Monitoring of Adult Patients receiving Home Parenteral Nutrition

Gavin Mercer-Smith, Colette Kirk, Lisa Gemmell, Chris Mountford, Jeremy Nightingale, Nick Thompson and the BIFA Committee

BIFA Recommendations

1. All haematological and biochemical monitoring of HPN (Home Parenteral Nutrition) patients should be individualised and may change with their clinical condition. The point at which patients become stable post discharge will vary.
2. Routine blood tests, including standard electrolytes, chloride, bicarbonate (as a measure of acid-base balance), calcium, magnesium, phosphate, renal and liver function tests, glucose, full blood count, ferritin and CRP should be performed monthly for the first 3 months after discharge. If stable this may then be 3-4 monthly.
3. When discharged patients should have their prothrombin time, cholesterol and triglyceride, HbA1c, vitamin D & B_{12} and folate concentrations checked. These should then be monitored at least 6-monthly.
4. Patients who are to receive long-term HPN should have baseline vitamins A&E, zinc, copper, manganese and selenium concentrations checked and then monitored 6-monthly.
5. If the CRP is significantly raised (>25mg/l) then transferrin saturation should be used as a measure of iron status. In this situation do not measure zinc, copper, selenium or vitamins A, D or E.
6. When measuring zinc, copper, manganese or selenium use a trace element-free collection tube.
7. If the Triglyceride concentration is elevated a fasting concentration should be repeated.
8. Urine sodium concentration is useful for assessing salt balance in patients with short bowel syndrome and a 24-hour urinary oxalate collection for assessing risk of renal stone formation in patients with a short bowel with colon in continuity.

*Disclaimer: BAPEN Position Statements/Guidelines have been prepared as guidance only to assist qualified healthcare professionals in the decision-making processes surrounding nutritional care. Users of these materials may only do so on the condition that they exercise their own professional knowledge and skills when applying such guidance to specific circumstances. Anyone without the appropriate qualifications must seek the advice of a qualified healthcare professional before taking, or refraining from, any action on the basis of the policies or guidance. BAPEN does not (i) owe a duty of care to users of the policies or guidance who are not qualified healthcare professionals; and (ii) cannot accept liability to anyone using these policies or guidance

Background

The essential components of a diet/feed are macronutrients (carbohydrate, fat and protein), water, minerals (includes those often referred to as electrolytes (Na, K, Cl, HCO3, Ca, Mg, P) and trace elements (e.g. Cu, Zn, Mn and Se) and vitamins (water and fat soluble). The macronutrient status is mainly monitored by a clinical examination (weight, BMI, muscle mass) and lipid measurements in the blood. Liver, kidney and bone/haematological functions, in addition to mineral and vitamin status can be assessed from blood tests. This document relates to patients having parenteral nutrition at home though may be used for those also having parenteral fluid. It related primarily to adults.

There is a scarcity of evidence-based literature on monitoring in HPN and recommendations are based mostly on expert opinion. Monitoring is crucial in order to maximise the benefits of HPN, to prevent and treat complications, and to secure and improve the quality of life of the patient. It is the responsibility of a multidisciplinary team with expertise in HPN. We have assumed that patients going home are stable and not at risk of refeeding syndrome. Wengler et al. studied monitoring practices for HPN across Europe and concluded that the majority of centres monitored stable HPN patients at three-monthly intervals.[1] In the future more remote monitoring of patients is likely to be required to reduce the need for patients to travel to an Intestinal Failure centres. It is therefore important that monitoring protocols are clear, safe but not burdensome.

NICE guidelines from Nutrition support for adults: oral support, enteral tube feeding and parenteral nutrition (CG 32), Feb 2006, revised Aug 2017.[2] "People in the community having parenteral nutrition … should receive an individualised care plan which includes … a monitoring plan." Patients should be reviewed by appropriately skilled teams at a specialist hospital clinic every 3–6 months. Monitoring should be more frequent during the early months of home parenteral nutrition, or if there is a change in clinical condition.

Zinc and copper, folate and B12 should be checked at baseline and then every 2-4 weeks, however it isn't clear what the interval should be for those at home. Selenium at baseline and then dependent on concentrations and if suspected deficiency (glutathione peroxidase is a preferred test if available). Iron and Ferritin at baseline and then every 3-6 months. Manganese every 3-6 months and vitamin D every 6 months.

BAPEN guidelines (2016): These guidelines suggest that stable patients are monitored between monthly and 3-monthly, depending on the patient's condition. Electrolytes (Sodium, Potassium & Magnesium), bone profile (Calcium & Phosphate), infection markers (such as C Reactive Protein and white blood cells) and liver function tests should be taken at baseline and then at each planned follow up. Cholesterol and triglycerides may be reviewed weekly initially, reducing to 3-monthly once stable to monitor the risk of potential hyperlipidaemia.

Trace elements (zinc, copper, selenium and manganese) and vitamins (A, D, E, B12, folate) should be checked at baseline if there is previous evidence of malnutrition. This should then be repeated 3 monthly in long-term patients to detect deficiencies or raised concentrations. It is important to interpret these results with caution and monitor clinical symptoms, as serum concentrations can be reduced or raised when inflammatory markers are raised during the acute phase response, and may not reflect total body stores.

ESPEN guidelines (2020): Monitoring should be more frequent during the early months of HPN, or if there is a change in the patient's clinical condition. In clinically stable patients on long-term HPN haematology and biochemistry

(haemoglobin, ferritin, albumin, CRP, electrolytes, venous blood gas analysis, kidney function, liver function and glucose) should be measured at all scheduled reviews (e.g. every three to six months). In patients on long-term HPN, clinical signs and symptoms as well as biochemical indexes of vitamin and trace metal deficiency or toxicity should be evaluated at least once per year. Vitamin and trace metal deficiency may take more time to develop and to present clinical signs and symptoms, so that a six to twelve-month interval of assessment is appropriate. Prospective studies of the impact of different monitoring regimens on outcomes (including QoL) of HPN are warranted.

Baseline tests should be include full blood count, CRP, glucose sodium, potassium, chloride, bicarbonate, magnesium, phosphate, calcium and renal function tests including urea and creatinine. Measurements should be weekly or monthly until stable and then performed regularly. Once stability has been achieved, this might be 3-4 monthly.

Liver function tests, including INR and albumin should be measured monthly and can be reduced to 3-4 monthly when stable. Serum iron and ferritin should be measured every 3-6 months with other micronutrients every 6-12 months (zinc, selenium, copper, vitamins A, E, D, B12, and folic acid). Serum manganese should be checked annually.

Commercial micronutrient preparations

In general, micronutrients for HPN patients are provided in the form of fixed combinations within multivitamin or multi-trace element solutions. This restricts prescribing options and makes it difficult to adjust the dose of individual micronutrients. Numerous observational studies and case reports have identified high concentrations of manganese, copper and chromium in patients receiving HPN, likely due to contamination of PN products, and low concentrations of selenium and zinc.[3,4]

Effect of inflammatory response on micronutrient status

During the acute phase response, quantified by measuring C-reactive protein (CRP), serum concentrations of iron, zinc, selenium and vitamin A decrease and copper concentrations increase.[5] These alterations in concentrations likely represent redistribution rather than true deficiency and this may actually be a beneficial and adaptive response. For example, reduced concentrations deprive microorganisms of vitamins and trace elements essential for growth and replication, and limits free radical production. During the resolution phase of sepsis, serum concentrations return to previous concentrations. The interpretation of zinc requires additional consideration as 90% of zinc is bound to albumin and concentrations will therefore be reduced when albumin decreases.

Micronutrients such as vitamins B_{12} and folate appear unaffected by the inflammatory response and therefore low concentrations are likely to represent true nutritional deficiencies. Further research is needed to examine the inter-relationships between the inflammatory response and circulating concentrations of vitamins C, D and E.

Limitations of these recommendations

There is a paucity of research in the area of monitoring in HPN, particularly around micronutrient monitoring, much of the available evidence is based on lower grade studies/ poor quality studies. Scoping of studies published in Medline (Ovid) and EMBASE was conducted with no restrictions on dates, however, limits to 'Humans' and 'English Language' were applied. A combination of key terms was used: parenteral nutrition *, intestinal failure *, vitamin*, retinol, tocopherol *, zinc, copper, selenium*, micronutrient*. No studies were found looking at monitoring or replacing

Vitamin A or E, Zinc, Copper or Selenium in stable patients being treated with home parenteral nutrition, demonstrating the sparsity of research within this patient group.

Overview of measurements:

Standard Electrolytes and Urea & Creatinine

Sodium: Standard laboratory methods can be subject to pseudohyponatraemia due to the electrolyte exclusion effect which can be caused by severe hyperproteinaemia and hyperlipidaemia - in these cases alternative methodology is recommended and should be discussed with the laboratory. There is a need to interpret with knowledge of fluid status - sodium is closely linked to water homoeostasis and changes in extracellular water volume will cause a change in sodium concentration. [6] Urinary sodium (and chloride) can be used to aid in detecting a salt-retaining state with concentrations <20mmol/l indicating this, however concentrations above this do not exclude hypovolaemia. [7]

Potassium: To avoid spurious hyperkalaemia avoid delays in separation, fist clenching and can result from haematological conditions such as thrombocytosis and leucocytosis (suggest use lithium heparin tube if this is a concern).

Chloride: Generally increases or decreases in direct relationship to sodium, but may change without any change in sodium when there are disturbances of acid-base balance.[6]

Bicarbonate (total carbon dioxide): Dissolved carbon dioxide in the sample will escape into the air over time causing a decrease in the result however in a normal serum sample the stability of bicarbonate is 16 hours [8]. If there is concern about acid-base balance then an arterial blood gas may provide more information however this is more invasive, requires immediate analysis and is rarely performed.

Calcium: Sample should ideally be taken without the use of a tourniquet.[6] Assay detects total serum calcium, free (unbound) calcium can be measured using point of care analyser (direct ion selective electrode). The calculation for adjusted calcium is invalid when albumin <20g/L, in these cases, direct measurement of free calcium can be performed using a blood gas analyser.

Phosphate: There is a risk of hypophosphataemia from some intravenous iron infusions; it is proposed that intravenous iron mediates an increase of serum fibroblast growth factor 23 (FGF23) which is phosphaturic.[9]
Magnesium: Detects total serum magnesium, free magnesium can be measured by point of care analysers (direct ion selective electrode).

Urea: Gives an indication of protein (nitrogen) intake (dietary or parenterally) in addition to renal function. As a general test of renal function urea is of limited value - creatinine (which relates to muscle mass) and eGFR is preferable. It can be difficult to ascribe a cause to abnormal urea concentrations as they are affected by rate of synthesis (protein turnover), volume of distribution (hydration status) and rate of excretion - urea tends to rise before creatinine in early pre-renal failure.

Creatinine: Superior to urea as a test of general renal function, serum creatinine concentration can be used together with demographic information to calculate the estimated glomerular filtration rate (eGFR), wide normal reference interval due to large inter-individual variation. High muscle mass can cause slightly elevated creatinine concentrations in those with normal renal function.

Bilirubin (total): Clinically jaundice tends to become apparent when bilirubin is > 3 x upper limit of normal.

Glucose: If considering a diagnosis of diabetes mellitus this is either by measurement of HbA1c or oral glucose tolerance testing.

Suggested monitoring frequency:

- BAPEN – 1-3 monthly [10]
- NICE – guidance offers no distinction between in/outpatient setting [2]
- ESPEN (2020) – weekly or monthly, 3-4 monthly when stable [11]
- AuSPEN – 3 monthly [12]

Liver enzymes

Alanine aminotransferase (ALT) & Aspartate aminotransferase (AST): In fatty liver disease, an activity ratio AST/ALT of >2 suggests alcohol as a cause; a ratio of ≤1 is suggestive of a non-alcoholic cause.[6] ALT is preferred over AST as an indicator of liver cell damage as it is more specific.[6]

Alkaline phosphatase (ALP): ALP is approximately equally of hepatobiliary and bone origin; other sources are small intestine, placenta and kidney.[6] To investigate the source of elevated ALP, adding GGT can help distinguish hepatobiliary origin or with an isolated ALP elevation - serum electrophoresis distinguishes alkaline phosphatase isoenzymes.

Gamma-glutamyltransferase (GGT): GGT can be used to confirm hepatic origin of elevated serum alkaline phosphatase (ALP) activity, and can suggest compliance with abstinence in patients with alcohol related liver disease.[6]

Suggested monitoring frequency:

- BAPEN – 1-3 monthly [10]
- NICE – guidance offers no distinction between in/outpatient setting [2]
- ESPEN (2020) – monthly, 3-4 monthly when stable [11]
- AuSPEN – 3 monthly [12]

Proteins

Total protein & Albumin: In inflammatory states concentration of albumin decreases but immunoglobulins increase. Albumin is a poor guide to nutritional status.[6] High or low albumin concentrations are frequently multifactorial, with more than one mechanism being responsible e.g. hydration status, inflammation, hepatic impairment. Used in the interpretation of adjusted calcium concentration. Total protein = albumin + globulin, the major component of globulin component is immunoglobulins. Suggested monitoring frequency:

- BAPEN (assumed as part of “liver function tests”) - 1-3 monthly [10]
- NICE – not given [2]

- ESPEN (2020) – monthly, 3-4 monthly when stable [11]
- AuSPEN (assumed as part of "liver function tests") – 3 monthly [12]

C–reactive protein (CRP): concentration increases with inflammation and so assists interpretation of protein, trace element and vitamin results.[2] Suggested monitoring frequency:

- BAPEN – 1-3 monthly [10]
- NICE – guidance offers no distinction between in/outpatient setting [2]
- ESPEN (2020) – weekly or monthly, 3-4 monthly when stable [11]
- AuSPEN – "Inflammatory markers if ongoing inflammatory disease" – no frequency given [12]

Full blood count and prothrombin time (PT)

Full blood count comprises haemoglobin, white blood cell count (and differential), red cell count (and indices) and platelet count. Inflammation can cause increases in white cell and platelet populations. Mean cell volume (MCV) is used to classify anaemia into microcytic, normocytic or macrocytic.

PT measurements help determine synthetic liver function.[13] Prothrombin time is prolonged in patients treated with warfarin (INR may be reported in warfarin monitoring). Increases in prothrombin time can indicate vitamin K deficiency.[14] Not all intravenous vitamin preparations contain vitamin K, and patients who are unable to have an oral intake are at risk of deficiency.

Suggested monitoring frequency:

- BAPEN – white cell count 1-3 monthly, no mention of PT [10]
- NICE – full blood count and mean cell volume - guidance offers no distinction between in/outpatient setting [2]
- ESPEN (2020) – weekly or monthly, 3-4 monthly when stable [11]
- AuSPEN – 'haematology screen' 3 monthly, with INR if anticoagulated [12]

Lipids: Cholesterol & Triglycerides

Inflammation can cause decrease in concentration of cholesterol.[6] Secondary hyperlipidaemia is a complication which can be associated with parenteral nutrition due to a variety of mechanisms.[15] Raised concentrations of lipids should be confirmed on repeat measurement with triglyceride, HDL-cholesterol and non-HDL (or LDL)-cholesterol. LDL cholesterol is not routinely measured directly but is derived. Triglyceride concentration can increase 2-3 fold after a meal.[14]
Suggested monitoring frequency:

- BAPEN – 3 monthly [10]
- NICE – not given [2]
- ESPEN (2020) – not given [11]
- AuSPEN – "longer-term patients should have a lipid screening" – no frequency given[12]

If patients who are HPN dependent are managed with lipid-free parenteral nutrition then testing for Essential Fatty Acids might be appropriate however this is not routine practice.

Vitamins

Vitamin D: Vitamin D2 = ergocalciferol & Vitamin D3 = cholecalciferol, assay detects the main storage form of vitamin D - 25OH-D2 and 25OH-D3, therefore should request total 25-OH vitamin D. 1-25 dihydroxy vitamin D is not routinely analysed. Inflammation can cause concentration decrease.[17] Deficiency is defined as 25OHD < 30 nmol/L, Insufficiency: 30 – 50 nmol/L & Sufficiency: > 50 nmol/L.[17] Low calcium and elevated alkaline phosphatase (ALP) may be the first indication of vitamin D deficiency.[19] Suggested monitoring frequency:

- BAPEN – 3 monthly[10]
- NICE – 6 monthly [2]
- ESPEN (2020) – every 6-12 months [11]
- AuSPEN – not given [102]
- Joint collaboration of RC Pathologists & Assoc Lab Medicine - Repeat after 3-6 months on replacement if baseline was low and potential absorption defect[20]

Folate (B9) & Vitamin B_{12} (cyanocobalamin): Serum concentration of folate indicates recent intake rather than tissue stores - red cell folate reflects whole body status however this is not routinely available.[13,19] Deficiency of either is associated with megaloblastic anaemia (requires measurement of full blood count). Suggested monitoring frequency:

- BAPEN – 3 monthly [10]
- NICE – guidance offers no distinction between in/outpatient setting [2]
- ESPEN (2020) – every 6-12 months [11]
- AuSPEN – not given [12]

Vitamin A & Vitamin E: Limited testing availability of the assay requiring specialist laboratory, inflammation causes concentration decreases which can also occur if samples are not protected from light.[5] Concentration does not provide ideal assessment of vitamin A status as serum concentration may not decline until liver stores have been critically depleted but low concentrations may be observed with adequate hepatic stores owing to decreased availability of binding proteins. Severe vitamin A deficiency can lead to night blindness. Vitamin A deficiency is also associated with vulnerability to measles and gastrointestinal or respiratory infections and may lead to anaemia (mechanism unknown).[13] If serum lipids are elevated the results of Vitamin E analysis are reported as a molar ratio (vitamin E is bound to lipoproteins). Vitamin E is involved in antioxidant and immune system defences and therefore deficiency may lead to impaired immunity. Deficiency can cause neuropathy and myopathy.[14]

Suggested monitoring frequency:

- BAPEN – 3 monthly [10]
- NICE – not given [2]
- ESPEN (2020) – every 6-12 months [11]
- AuSPEN – not given [12]

Trace elements

Iron & Ferritin: Inflammation causes concentration of Ferritin to increase and Fe concentration decreases.[5] Large variation in reference intervals, due to different methods of analysis and large age and gender differences, however intra-individual variation is small.[6] Ferritin is the most useful indicator of iron deficiency - ferritin stores can be significantly decreased before any fall in iron occurs, a combination of chronic disease and iron deficiency may result in a normal ferritin concentration. In this situation iron can be measured with transferrin and transferrin saturation to assist interpretation of iron status. Suggested monitoring frequency:

- BAPEN – not given [10]
- NICE – 3-6 monthly [2]
  - Also endorsed by the Joint collaboration of RC Pathologists & Assoc Lab Medicine national minimum re-testing interval report[20]
- ESPEN (2009) – every 3-6 months [11]
- AuSPEN – "iron status" 6 monthly [12]

Zinc: Use trace element-free tube and transport to laboratory as soon as possible for rapid separation of serum from red blood cells. Serum concentrations do not accurately reflect whole body concentrations.[16] There is limited testing availability requiring specialist laboratory, inflammation causes concentration decrease, as it is bound to albumin which is likely to be reduced.[5] If a patient has chronic inflammation and there is clinical concern about possible zinc deficiency then measurement might be appropriate with some adjustment made for low levels related to low albumin concentrations. High concentrations of copper or iron in the diet may decrease zinc intestinal absorption resulting in low serum zinc. Conversely, chronic ingestion of oral zinc supplements may induce reversible anaemia and leucopenia secondary to relative copper deficiency caused by reduced intestinal absorption.[21] Zinc deficiency can cause perioral and perineal rash.[14, 22] Low concentrations can impair wound healing and cause diarrhoea.[13,14] It can also cause a change in taste and some patients complain of food tasting of metal or cardboard.[14] Deficiency can also lead to crinkly hair growth.[14]

Provision of adequate zinc intravenously to achieve a positive zinc balance is associated with improvement in nitrogen balance.[13] Suggested monitoring frequency:

- BAPEN – 3 monthly [10]
- NICE – guidance offers no distinction between in/outpatient setting [2]
- ESPEN (2020) – every 6-12 months [11]
- AuSPEN – 6 monthly [12]

Copper: Use Serum trace element-free tube. There is limited testing availability requiring specialist laboratory, inflammation causes concentration increase.[5] Copper deficiency can cause a microcytic anaemia (that is unresponsive to iron therapy) and neutropaenia.[21, 22] Mainly biliary excretion so patients with cholestatic jaundice are at risk of copper accumulation. Copper deficiency can result from excess iron or zinc ingestion through interference with copper absorption.[21] Suggested monitoring frequency:

- BAPEN – 3 monthly [10]
- NICE – guidance offers no distinction between in/outpatient setting [2]

- ESPEN (2020) – every 6-12 months [11]
- AuSPEN – 6 monthly [12]

Manganese: Use a whole blood sample trace element-free tube with EDTA anticoagulant. Individual laboratories might request an empty EDTA tube is sent with the sample from the same batch to check for contamination. There is limited testing availability requiring specialist laboratory. Manganese is ubiquitous within the environment, including potentially in the metal needle used to withdraw blood, to reduce this potential for contamination the sample for trace element analysis should not be the first one taken. Inflammation does not affect concentrations.[23] Concentrations > 360 nmol/L indicates manganese retention.[13] HPN often contains a fixed combination of multivitamins and trace elements and tends to contain high concentrations of manganese which exposes long-term HPN patients to a risk of toxicity which can result in manganism (a Parkinsonian-like neurodegenerative disorder).[3,4,14] Excretion is primarily via bile so accumulation can result if liver disease/cholestasis is present.[13] Suggested monitoring frequency:

- BAPEN – 3 monthly [10]
- NICE – 3-6 monthly [2]
- ESPEN (2020) – annually [11]
- AuSPEN – 6 monthly [12]

Selenium: Use a Serum trace element-free tube and transport to lab as soon as possible for rapid separation of serum from red blood cells. There is limited testing availability requiring specialist laboratory. Inflammation causes concentration decrease.[5] Toxicity may occur with concentrations > 5.1 µmol/L.[13] Serum concentration reflects recent intake, red cell glutathione peroxidase is a superior index of long-term intake but is not the preferred procedure in routine laboratories.[13,21] Selenium is an important component of enzymes involved in antioxidant functions, thyroid hormone metabolism and male fertility. Selenium deficiency may contribute to cardiomyopathy, hypothyroidism, and an impaired immune system. Excessive provision may result in hair loss, 'garlic breath', nail changes and peripheral neuropathy.[14, 24]

Suggested monitoring frequency:

- BAPEN – 3 monthly [10]
- NICE – "Further testing dependent on baseline" [2]
- ESPEN (2020) – every 6-12 months[11]
- AuSPEN – 6 monthly [12]

Chromium: This is needed for pancreatic function and is not routinely measured.

Iodine: This is not routinely measured however sufficient amount is necessary for normal thyroid function and so annual Thyroid function tests eg with Thyroid stimulating hormone (TSH) would be appropriate.

Urine

Random urinary sodium concentration is useful for determining hydration and sodium balance. It is most useful in jejunostomy patients or patients with a high output stoma or fistula, see above.

Patients with a jejunum in continuity with a functioning colon should have annual 24 hour urinary oxalate excretion measured as this can predict those likely to develop calcium oxalate renal stones which can be prevented with treatment.

Table 1
Haematological and biochemical monitoring of patient having HPN and considered clinically stable

	3-4 monthly	6 monthly	annually
Blood count	x		
Prothrombin time (INR)		x	
Renal function*			
(Na, K, Cl, HCO3, urea, creatinine)	x		
CRP	x		
Liver function			
Alk P, GGT, ALT, AST, Bil, T prot, albumin	x		
Bone chemistry (Mg, Ca, P)	x		
Glucose/HbA_1C	x		
Cholesterol and triglyceride,		x	
Vitamins (A, D, E, B_{12}, folate)**		x	
Ferritin/Fe/TIBC		x	
Trace elements			
Zn, Cu, Se, Mn		x	
Urine [Na] *	x		
24 hr urine oxalate (J-C patients only)			x
Thyroid Function tests, TSH			x

*: HCO3, chloride and urinary Na if stable may be done annually

**: Vit A&E may be measured annually unless results are abnormal.

Not measured in UK: chromium and Vit C/other B vitamins

Note: These are suggested time intervals for stable patients. If results are abnormal or the clinical situation changes they may be done more frequently.

References

1. Wengler A, Micklewright A, Hébuterne X, Bozzetti F, Pertkiewicz M, Moreno J, et al. Monitoring of patients on home parenteral nutrition (HPN) in Europe: A questionnaire based study on monitoring practice in 42 centres. Clinical Nutrition. 2006 Aug;25(4):693–700.

2. Nutrition support for adults: oral nutrition support, enteral tube feeding and parenteral nutrition; CG 32 [Internet]. 2006. Available from: nice.org.uk/guidance/cg32

3. Kirk C, Gemmell L, Lamb CA, Thompson NP, Mountford CG, Toole BJ. Elevated Whole-Blood Manganese Levels in Adult Patients Prescribed "Manganese-Free" Home Parenteral Nutrition. Nutrition in Clinical Practice 2019; Oct 22, doi: 10.1002/ncp.10431.

4. Btaiche IF, Carver PL, Welch KB. Dosing and monitoring of trace elements in long-term home parenteral nutrition patients. Journal of Parenteral and Enteral Nutrition. 2011 Nov;35(6):736–47.

5. Galloway P, Mcmillan DC, Sattar N. Effect of the inflammatory response on trace element and vitamin status. Ann Clin Biochem. 2000;37:289–97.

6. Association for Laboratory Medicine- Analyte Monographs [Internet]. Available from: https://labmed.org.uk/our-resources/science-knowledge-hub/analyte-monographs.html

7 Sherman RA & Eisinger RP. The Use and Misuse of Urinary Sodium and Chloride Measurements. JAMA 1982;247(22):3121-24.

8 Boyanton BL & Blick KE. Stability studies of twenty four analytes in human plasma and serum. Clinical Chemistry. 2002;48(12):2242-47.

9. Coppolino G, Nicotera R, Cernaro V, Calimeri S, Leonardi G, Cosentino S, et al. Iron Infusion and Induced Hypophosphatemia: The Role of Fibroblast Growth Factor-23. Ther Apher Dial. . 2019 Sep 4. doi: 10.1111/1744-9987.13435.

10. British Association for Parenteral and Enteral Nutrition. Parenteral Nutrition Monitoring [Internet]. 2016. Available from: https://www.bapen.org.uk/nutrition-support/parenteral-nutrition/monitoring

11. Pironi L et al. ESPEN Guideline on home parenteral nutrition. Clinical Nutrition, https://doi.org/10.1016/j.clnu.2020.03.005

12. Gillanders L, Angstmann K, Ball P, Chapman-Kiddell C, Hardy G, Hope J, et al. AuSPEN clinical practice guideline for home parenteral nutrition patients in Australia and New Zealand. Nutrition. 2008;24(10):998–1012.

13. Burtis CA, Bruns DE. Tietz Fundamentals of Clinical Chemistry and Molecular Diagnostics. Seventh. Sawyer BG, editor. Elsevier; 2015.

14. Austin P, Stroud M. Prescribing Adult Intravenous Nutrition. Pharmaceutical Press; 2007.

15. Raman M, Almutairdi A, Mulesa L, Alberda C, Beattie C, Gramlich L. Parenteral nutrition and lipids. Vol. 9, Nutrients. MDPI AG; 2017.

16. Marshall W, Lapsley M, Day A, Ayling R. Clinical Biochemistry. Third. Marshall W, Lapsley M, Day A, Ayling R, editors. Churchill Livingstone; 2014.

17. Blaauw R, Osland E, Sriram K, Ali A, Allard JP, Ball P, et al. Parenteral Provision of Micronutrients to Adult Patients: An Expert Consensus Paper. Journal of Parenteral and Enteral Nutrition. 2019 Mar 1;43(S1):S5–23.

18. Francis R, Aspray T, Fraser W, Gittoes N, Javaid K, Macdonald H, et al. Vitamin D and Bone Health: A Practical Clinical Guideline for Patient Management National Osteoporosis Society Practical Guides for a breakfree future. First. The National Osteoporosis Society; 2013.

19. Gidden F, Shenkin A. Laboratory Support of the Clinical Nutrition Service. Vol. 38, Clin Chem Lab Med. 2000.

20. Lang T, Croal B. G147 National Minimum Retesting Intervals in Pathology 2021:. https://labmed.org.uk/resource/g147-national-minimum-retesting-intervals-in-pathology.html.

21. Ayling R, Marshall W. Nutrition and Laboratory Medicine. Harris B, Lapsley M, editors. ACB Venture Publications; 2007.

22. Fessler TA. Trace Elements in Parenteral Nutrition: A Practical Guide for Dosage and Monitoring for Adult Patients. Nutrition in Clinical Practice. 2013 Dec;28(6):722–729

23. Zemrani B, McCallum Z, Bines JE. Trace element provision in parenteral nutrition in children: One size does not fit all. Vol. 10, Nutrients. MDPI AG; 2018.

24. Shenkin A. Selenium in Intravenous Nutrition. Gastroenterology. 2009;137(5 SUPPL):S61–9.