Functional Blood Chemistry Analysis—Secrets to Getting the Most from Your Patients’ Blood Test Results

Seminar Notes

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Another program in the “Four Quadrants of Functional Diagnosis” Seminar Series
Acknowledgments

- We would like to give special thanks to Harry Eidenier, PhD of Biotics Research for introducing us to functionally oriented Blood Chemistry interpretation.
- We would also like to thank the following for their contributions to our understanding of Blood Chemistry and CBC analysis.
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  - Alexander Schauss, Ph.D.
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  - Guy Schenker, DC
  - Harold Loomis, DC
  - John Sherman, ND
  - Russell Marz, ND
  - Dickson Thom, DDS, ND.

Objective of today’s Seminar

1. To demonstrate a system of Blood Chemistry and CBC analysis for the early detection of dysfunction.
2. To differentiate between functional diagnosis versus pathological diagnosis.
3. To teach you to recognize key metabolic patterns in blood chemistry and CBC panels.

Functional Vs. Pathological

<table>
<thead>
<tr>
<th>FUNCTIONAL</th>
<th>PATHOLOGICAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Views body as inter-connected system</td>
<td>1. Mechanistic view of body</td>
</tr>
<tr>
<td>2. Identifies imbalances and dysfunction in physiology</td>
<td>2. Identifies and treats disease</td>
</tr>
<tr>
<td>3. Treats the underlying cause</td>
<td>3. Treats symptoms</td>
</tr>
<tr>
<td>4. Early predictor of dysfunction</td>
<td>4. Later stage treatment of disease</td>
</tr>
<tr>
<td>5. Health measured along a wellness spectrum</td>
<td>5. Health as absence of disease</td>
</tr>
<tr>
<td>6. Identifies multiple therapeutic options</td>
<td>6. Provides little therapeutic options</td>
</tr>
</tbody>
</table>
How do we typically gather data from our patients?

1. Intake form
2. History
3. Physical Exam
4. Tests run in office
   - Routine urinalysis
   - Other tests (pregnancy test, strep throat etc..)
5. Out-sourced labs
   - Blood Chemistry and CBC
   - Other tests (hormone panels, saliva testing, hair analysis etc..)

4 Quadrants of Functional Diagnosis

<table>
<thead>
<tr>
<th>HISTORY</th>
<th>IN-OFFICE LABS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Assessment Questionnaire</td>
<td>1. Urinalysis</td>
</tr>
<tr>
<td>• An essential tool for taking</td>
<td>2. Saliva testing</td>
</tr>
<tr>
<td>initial history and following</td>
<td>3. Other “Terrain” tests</td>
</tr>
<tr>
<td>patients through tx.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PHYSICAL ASSESSMENT</th>
<th>OUT-SOURCED LABS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Traditional PE</td>
<td>1. Chem. Screen and CBC</td>
</tr>
<tr>
<td>2. Physical evaluation using</td>
<td>2. Specialized Labs</td>
</tr>
<tr>
<td>functional testing</td>
<td></td>
</tr>
</tbody>
</table>

Four Essential Questions

Gathering data from all Four Quadrants allows you to answer 4 essential questions:
1. Is there a dysfunction or imbalance in the body?
2. What are the sources of the dysfunction or imbalance?
3. What individualized treatment is needed?
4. When has function been restored?

Biochemical Individuality

1. These four questions respect the biochemical individuality of your patients.
2. We recognize the complexity of human health and physiology.
3. Assessment and treatments should be reflective of each patient’s unique biochemical, physical, emotional, and energetic wellbeing.
“A doctor who prescribes an identical treatment for an identical illness in two individuals and expects an identical development may be properly classified as a social menace”

Lin Yutang (ancient philosopher and healer)

Why Blood Chem Analysis?

1. A detailed blood chemistry analysis helps us uncover multiple nutritional and metabolic deficiencies.
2. It helps clarify potential organ and system dysfunction.
3. Blood chemistries can also be used as a “gateway” test for other testing i.e. markers for digestive dysfunction may prompt you to order a GI Health panel or Comprehensive Digestive Stool Analysis.

Blood Chem & CBC Analysis-
Standard Approach

1. The conventional or standard laboratory reference ranges are designed to identify and diagnose disease states and pathology.
2. The normal reference values can change from year to year depending upon the prevalence of disease in the general population.
3. This leaves a large number of the population testing in a range that is considered “normal” as compared to the rest of the population.
4. From our perspective they are a far cry from being functionally optimal.

1. The functional approach to chem screen and CBC analysis is oriented around changes in physiology and not pathology.
2. We use ranges that are based on optimal physiology and not the “normal” population.
3. This results in a tighter range, which will increase the sensitivity while reducing specificity.
4. We increase our ability to detect patients with changes in physiological function.
5. We can identify the factors that obstruct the patient from achieving optimal physiological, biochemical, and metabolic functioning in their body.

Where do these optimal ranges come from?

1. Many of these ranges used to be the “normal” ranges 20 to 30 years ago.
2. Also, many of these optimal ranges and patterns are the result of information gathered from thousands of patient’s using a system called the “Biochemical Biopsy”.
3. We have studied with and have incorporated ranges from a number of sources including Lyn August, MD; Patricia Kane, PhD; Jim Said, ND, DC; Joseph Montante, MD; and Harry Eidenier, PhD.
4. We have also incorporated our clinical observations using this method of analysis for the last 7 years.

Blood Chem & CBC Analysis: Patterns and Trends

1. It is important to look at the patterns and trends between tests as opposed to looking at just the specific elements in isolation.
2. We will teach you to recognize key metabolic trends and patterns in chem. screen and CBC panels.
3. The trends and patterns between the individual tests can provide important clues to the underlying dysfunction and an understanding of the potential cause.

Where Blood Chemistries Fit in the Scope of Practice

1. It is important to remember that none of the ranges or patterns presented today are absolute.
2. They should be viewed as prognostic guidelines.
3. Functional analysis of blood chemistry and CBCs must be viewed with all of the other diagnostic data gathering methods available to physicians:
   • History
   • Physical examination
   • In-office tests
   • Out-sourced labs
   • Other diagnostic tools available to the licensed practitioner
**Seminar Organization: Foundational Hierarchy**

1. This seminar is designed to show you how to approach blood chemistry and CBC analysis from a functional perspective.
2. The material is organized in a specific and very deliberate way to represent the foundations of health.

**Foundations of Health**

1. Proper nutritional status/diet
2. Good hydration
3. Optimal digestion & toxin elimination
4. Mineral and vitamin balance
5. Essential fatty acid status
6. Reduced oxidative stress
7. Metabolic and hormonal balance

**Approaching the Functional Analysis**

1. Before beginning the analysis it is important to look at the blood test to see what tests are outside the normal reference range.
2. Then transfer the data onto the Blood chemistry and CBC Tracking form.
3. Begin the functional assessment paying attention to the particular markers that correspond to the foundational hierarchy.

**Gastrointestinal Markers**
Gastrointestinal System Reference Ranges

1. The Gastrointestinal system can be assessed by looking at the following elements:

<table>
<thead>
<tr>
<th>Element</th>
<th>Lab Range</th>
<th>Optimal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Protein</td>
<td>60 – 85</td>
<td>69 – 74</td>
</tr>
<tr>
<td>Globulin</td>
<td>20 – 39</td>
<td>24 – 28</td>
</tr>
<tr>
<td>Albumin</td>
<td>35 – 55</td>
<td>40 – 50</td>
</tr>
<tr>
<td>BUN</td>
<td>1.79 – 8.93</td>
<td>3.57 – 5.71</td>
</tr>
<tr>
<td>Creatinine</td>
<td>53.0 – 132.6</td>
<td>70.7 – 97.2</td>
</tr>
<tr>
<td>Phosphorous</td>
<td>0.81 – 1.45</td>
<td>0.97 – 1.29</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0 – 7%</td>
<td>0 – 3%</td>
</tr>
</tbody>
</table>

Background

1. The serum proteins in the blood are most helpful for uncovering digestive dysfunction and protein deficits in the body.
2. Nutrient deficits are mostly dietary protein deficits that are either from lack of amino acids or poor breakdown of available amino acids.
3. It can be very helpful to follow serum blood proteins as you institute a digestive protocol or increase dietary protein.

Total Protein - Background

1. Total serum protein is composed of albumin and total globulin.
2. Conditions that affect albumin and total globulin readings will impact the total protein value.
3. A normal total protein is possible even if the albumin or globulin levels are abnormal.
4. Serum protein is affected by protein digestion, absorption and assimilation.
5. Serum protein should be checked to screen for functional digestive problems, nutritional deficiencies, and dehydration.

Total Protein – Clinical Implications

HIGH

1. An increased serum protein (> 74g/L) is more likely to be an increase in the globulin fraction than the albumin level, unless there is dehydration that is causing the relative albumin increase.
2. Other causes:
   - Hypochlorhydria
   - Diet low in protein
   - Malnutrition
   - Poor protein breakdown
   - Amino acid need
   - Digestive dysfunction and/or inflammation

LOW

1. Hypochlorhydria
2. Diet low in protein
3. Malnutrition
4. Poor protein breakdown
5. Amino acid need
6. Digestive dysfunction and/or inflammation
7. Liver dysfunction

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**Globulin - Discussion**

1. Total globulin can be used to assess inflammatory, degenerative and infectious processes.
2. These processes are associated with an increased production of antibodies, which are synthesized from globulins.
3. With accompanying subjective indicators, a total globulin is helpful to confirm an underlying digestive problem of an inflammatory or infectious nature.
4. Total globulin is more useful for the detection of digestive insufficiency than a frank nutrient deficiency.

**Globulin – Clinical Implications**

- **HIGH**
  - Hypochlorhydria
  - Liver cell damage
  - Oxidative stress
  - Heavy metal toxicity

- **LOW**
  - Digestive dysfunction and/or inflammation
  - Immune insufficiency

**Albumin - Discussion**

1. We will look here at Albumin’s role in identifying nutrient and protein deficits in the body.
2. Albumin is a blood protein and will be affected by both digestive dysfunction and decreased protein and nutrients.
3. A deficient nutritional state leads to a decreased albumin level in the serum primarily from lack of available amino acids.

**Albumin – Clinical Implications**

- **HIGH**
  - Dehydration

- **LOW**
  - Digestive dysfunction due to HCL need
  - Nutrient and protein deficit
  - Liver dysfunction
  - Oxidative stress
**BUN- Background**

- BUN is formed from urea, an element formed almost entirely by the liver from both protein metabolism and protein digestion.
- The amount of urea excreted as BUN varies with the amount of dietary protein intake.
- Increased BUN may be due to an increased production of urea by the liver or decreased excretion by the kidney.

**BUN- Discussion**

- The BUN is a test that is predominantly used to measure kidney function.
- However, it is very useful as a measure of digestive function.
- BUN is useful as a first indicator of renal insufficiency especially if all the other renal indicators are normal.

**BUN- Clinical Implications**

**HIGH**
- Renal disease
- Renal insufficiency
- Dehydration
- Hypochlorhydria
- Diet- excessive protein intake
- Adrenal hyperfunction
- Dysbiosis
- Edema
- Anterior pituitary dysfunction

**LOW**
- Diet- low protein
- Malabsorption
- Pancreatic insufficiency
- Liver dysfunction

**Eosinophils- Discussion**

1. Eosinophils are often increased in patients that are suffering from intestinal parasites.
2. Eosinophils help remove and breakdown the by-products of protein catabolism.
3. They have the ability to ingest antibody-antigen complexes and become active in later stages of inflammation.
4. They are not effective against bacteria but are against parasitic disorders.
**Gastrointestinal Dysfunction**

1. The following are some of the functional problems in the gastrointestinal system that can be assessed using Blood chemistry and CBC analysis

   - Hypochlorhydria
   - Digestive dysfunction/Gastric inflammation
   - Dysbiosis
   - Pancreatic insufficiency
   - Parasites
   - Protein deficiency or impaired protein digestion

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**Hypochlorhydria**

1. Hypochlorhydria is a condition of decreased output of hydrochloric acid and pepsin from parietal cells of stomach

2. Hypochlorhydria is often due to a need for the following nutrients:
   - Chloride (low serum chloride)
   - Zinc (ALP will generally be decreased)
   - Thiamine (CO₂ will generally be decreased with an increased anion gap).

3. These are primary nutritional factors required for the synthesis of hydrochloric acid.

4. Paradoxically, adequate HCL is needed to properly absorb these nutrients as well.

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**Hypochlorhydria- Pattern**

1. An increased total globulin (>28) is often associated with a decreased production of hydrochloric acid in the stomach (Hypochlorhydria)

2. Hypochlorhydria is possible with an increased globulin level (>28) and a normal or decreased total protein (<69) and/or albumin (<40).

3. Hypochlorhydria is probable if globulin levels are increased (>28) along with an increased BUN (>5.71), a decreased or normal total protein (<69) and/or albumin (50) and/or decreased serum phosphorous (<0.97)

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**Hypochlorhydria- Other Patterns**

- Other values that may be reflective of a developing or chronic hypochlorhydria include:
  - Increased or decreased Gastrin
  - An increased MCV (>90) and MCH (>31.9),
  - Decreased or normal calcium (<2.30) and Iron (<8.96)
  - Decreased CO₂ (<25)
  - Increased Anion Gap (>12)
  - Decreased alkaline phosphatase (<70)
Functional Testing for Hypochlorhydria

1. Gastro-test
2. Nutritional Physical Examination
   • Reflex point testing for stomach
   • Used to identify treatment options

Digestive dysfunction/inflammation - Pattern

- Low total serum protein is associated with primary digestive inflammation or inflammation secondary to HCL insufficiency with a low total protein.
- Suspect digestive inflammation with a decreased protein (<69), a decreased total globulin (<24), decreased serum phosphorus (<0.97), an increased BUN (>5.71), and a decreased Creatinine (<70.7).
- The likelihood of digestive inflammation increases with an increased serum gastrin, and an increased basophil count (>1%) and ESR (♀>10, ♂>5).

Digestive Inflammation - Acute and Chronic

- A decreased serum protein (<69) is associated with digestive inflammation.
- Chronic digestive inflammation:
  - May be due to colitis, enteritis, Crohn’s etc.
  - Will compromise protein breakdown and absorption
  - This can lead to a widespread protein deficiency in the body and a decreased level of the inflammatory immunoglobulins, hence the decreased total globulin level (<24) and therefore a low total serum protein (<69).
- Acute digestive inflammation may lead to an increased globulin level (>28) due to the increased production of inflammatory immunoglobulins.
Blood Chemistry and CBC Analysis - A Functional Perspective

**Digestive Inflammation - Acute and Chronic**
- A decreased serum protein (<69) is associated with digestive inflammation.
- **Chronic** digestive inflammation:
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- **Acute** digestive inflammation may lead to an increased globulin level (>28) due to the increased production of inflammatory immunoglobulins.

**Dysbiosis**
1. An increased BUN is a useful indicator for dysbiosis.
2. BUN will be increased from two different mechanisms.
   - In the large intestine, putrefactive action of increased bacterial overgrowth on nitrogenous materials releases significant quantities of ammonia, some of which will be converted into urea by the liver leading to increased BUN levels (>5.71).
   - A significant amount of urea travels from the liver to the colon and is acted upon by gut microflora, which recirculate the nitrogen. Increased catabolism of this nitrogen in the colonic environment will increase BUN.

**Leaky Gut Syndrome**
1. An increased Alkaline phosphatase is associated with a Leaky gut syndrome.
2. Alkaline phosphatase is a mix of isoenzymes, one of which can have an intestinal origin.
3. Increased alkaline phosphatase levels (>100) may be seen in conditions involving the intestinal mucosa e.g. leaky gut syndrome, ulcers, colitis, and malabsorption.
4. Serum Uric acid (>351) and serum gastrin levels may also be elevated.

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Dr. Dicken Weatherby
Blood Chemistry and CBC Analysis - A Functional Perspective

Pancreatic Insufficiency

- A decreased BUN (<10) is associated with a pancreatic insufficiency.
- A decreased level of digestive enzymes secreted from the pancreas, especially protease, can lead to a functional protein deficit.
- This functional protein deficit in turn will lead to lower levels of protein catabolism and low BUN levels.
- You may also see elevated GGT (>30) and a decreased total WBC count (<5.0)

WBCs and Pancreatic Insufficiency

1. A decreased WBC can be caused by a pancreatic insufficiency.
2. The body can respond to pancreatic insufficiency by using phagocytic white cells to do the job of breaking down food and clearing food residue from the body.
3. This is known as leukocytic auto digestion and can cause a decreased white count.

Intestinal Parasites - Pattern

1. An increased eosinophil count is associated with intestinal parasites.
2. Suspect Intestinal parasites with the following patterns:
   - Increased Eosinophils (>3)
   - Increased Basophils (>1)
   - Increased Monocytes (>7)
3. If this pattern is seen, intestinal parasites should be investigated with appropriate stool analysis or treated presumptively.

Protein Malnutrition and Deficiency

- Protein malnutrition/amino acid need is due primarily to the lack of available essential amino acids from the diet. (high refined carb diets, unbalanced vegan/vegetarian diets etc...)
- Expect to see a decreased Albumin count (<40) and you may also see BUN (<3.57), Creatinine (<70.7), and Total Protein (<69)
- Review diet and consider hypochlorhydria support
Hepato-Biliary and Fatty Acid Markers

Hepato-Biliary Function-Reference Ranges

1. Hepato-Biliary function can be assessed by looking at the following elements:

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<thead>
<tr>
<th></th>
<th>Lab Range</th>
<th>Optimal Range</th>
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<tbody>
<tr>
<td>GGTP</td>
<td>1 – 70</td>
<td>10 – 30</td>
</tr>
<tr>
<td>Alk Phos</td>
<td>25 – 140</td>
<td>70 – 100</td>
</tr>
<tr>
<td>Bilirubin-total</td>
<td>1.7 – 20.5</td>
<td>1.7 – 20.5</td>
</tr>
<tr>
<td>Bilirubin-direct</td>
<td>0 – 3.4</td>
<td>0 – 3.4</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>0.34 – 1.7</td>
<td>0.79 – 1.24</td>
</tr>
<tr>
<td>Total Cholesterol</td>
<td>3.36 – 5.20</td>
<td>3.9 – 5.69</td>
</tr>
</tbody>
</table>

GGTP- Background

1. GGTP is one of the main liver enzymes
2. It is useful in elucidating both a developing liver/gallbladder dysfunction and a developed liver/gallbladder pathology
3. GGTP is liberated into the bloodstream following cell damage or destruction.
4. GGTP is found in highest quantities in the gallbladder, prostate and pancreas

GGTP- Discussion

1. GGTP, SGOT/AST, and SGPT/ALT can be used to determine the locus of a problem, depending on which of the liver enzymes is elevated.
2. If GGTP increased above SGPT/ALT and SGOT/AST consider that the problem or area of involvement is possible outside the liver but inside the biliary tree (i.e. gall bladder, common bile duct and pancreas).
3. GGTP is a much more sensitive and specific marker for Hepato-Biliary dysfunction than other markers, such as Alk Phos and SGPT/ALT for some conditions (cholangitis, cholecystitis, obstructive jaundice).
4. GGTP is generally increased above the other liver enzymes in alcoholism.
GGTP – Clinical Implications

- HIGH
- Dysfunction located outside the liver and inside the biliary tree
- Biliary obstruction
- Biliary stasis/insufficiency
- Liver cell damage
- Alcoholism (GGTP production induced by alcohol)
- Acute/chronic Pancreatitis
- Pancreatic insufficiency
- Obesity (elevated as high as 50%)

Alkaline Phosphatase - Clinical Implications

- HIGH
- Biliary obstruction
- Liver cell damage
- Bone: loss/increased turnover or bone growth and/or repair
- Leaky gut syndrome
- Herpes zoster
- Metastatic carcinoma of the bone

Alkaline Phosphatase - Background

1. Alkaline phosphatase (ALP) is a group of isoenzymes that has a maximal activity at a pH of 9.0 – 10.0 and originate in the following tissues:
   - Bone
   - Liver
   - Intestines
   - Skin
   - Placenta

2. In the liver, ALP is formed by liver and biliary mucosal cells, and is excreted in the bile
3. Increased ALP levels can occur with any liver dysfunction
4. ALP is especially sensitive to any type of obstruction in the biliary tract, both intra and extra-hepatic, both severe and mild.

Bilirubin - Background

1. Formed from the breakdown of hemoglobin from red blood cells, by the reticuloendothelial cells of the spleen and bone marrow.
2. It is transported from these cells to the liver where it is conjugated (made water soluble) and excreted via the gall bladder in the bile.
3. Increased serum levels of bilirubin occur with excessive red blood cell destruction or a problem in the liver or gallbladder that prevents the normal excretion of bilirubin.
4. Oxidative stress can cause red blood cell destruction.
Blood Chemistry and CBC Analysis-
A Functional Perspective

Bilirubin- Discussion
1. Total bilirubin is usually the only value reported on a standard chemistry screen.
2. If the levels are elevated, consider ordering the direct (conjugated) and Indirect (unconjugated) values.
3. We recommend obtaining the direct and indirect values on your routine chemistry screens.
4. This will assist in determining if the cause of an increased total bilirubin is due to pre-hepatic situations (increased hemolysis) or post-hepatic problems (biliary obstruction)
5. If either the indirect or the direct has been reported, subtract that number from the total bilirubin and you will have the missing value.

Total Bilirubin – Clinical Implications

HIGH
▪ Biliary stasis
▪ Biliary tract obstruction or calculi
▪ Oxidative stress
▪ Thymus dysfunction
▪ Liver dysfunction
▪ RBC hemolysis
▪ Gilbert’s syndrome

LOW
n/a

Direct Bilirubin
1. Direct bilirubin is the bilirubin that has been conjugated and is excreted in the bile.
2. An increase in direct or conjugated bilirubin is usually associated with a dysfunction or blockage in the liver, gallbladder or biliary tree.

HIGH
▪ Biliary tract obstruction
▪ Biliary calculi/obstruction (usually extra hepatic)

LOW
n/a

Hepato-Biliary and Fatty Acid Dysfunction
1. The following are some of the functional problems associated with the gallbladder and fatty acid metabolism that can be assessed using Blood chemistry and CBC analysis
   ▪ Biliary dysfunction
     ▪ Biliary insufficiency
     ▪ Biliary stasis
     ▪ Biliary obstruction (intrahepatic or extrahepatic)
   ▪ Fatty acid insufficiency
   ▪ Fat metabolism problems

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Biliary Dysfunction

1. A moderate rise in GGTP and other elements can alert us to a developing problem within the biliary tree that is more functional in nature:
   • Biliary insufficiency
   • Biliary stasis
   • Mild obstruction due to either liver congestion or biliary tree blockage
   • Severe obstruction due to major gallstone formation

Biliary Insufficiency

1. Biliary insufficiency is the inability of the liver cells to produce adequate amounts of bile due to:
   • Changes in metabolism (excess hydrogenated oils, excess refined foods, oxidative stress, low fat diets)
   • Diseases affecting function (steatosis causing damage to hepatocytes, hepatitis)
   • Diseases that reduce levels of hepatocytes (advanced cirrhosis)
2. Biliary insufficiency will cause an increase in GGT, Bilirubin, Cholesterol, Alk phos.
3. One of the hallmark signs of biliary insufficiency is tan or clay colored stools.

Biliary Stasis

1. Biliary stasis is a condition marked by a thickening of the bile.
2. Biliary stasis is due to:
   • Supersaturation of the bile caused by increases in cholesterol secretion
   • Decrease in bile acid formation
   • Decrease in phosphatidylcholine secretion
3. If left unchecked biliary stasis will most likely lead to stone formation.
4. Consider biliary stasis to be the earliest stage of biliary obstruction.

Biliary Stasis or Insufficiency- Pattern

1. Suspect biliary insufficiency or biliary stasis if the GGTP is increased (>30).
2. Bilirubin levels may also be elevated (>20.5) along with alkaline phosphatase (>100) and total cholesterol (>5.69). SGOT/AST and SGPT/ALT may be normal or increased (>30).
3. Many cases of biliary insufficiency or stasis will show normal lab values.
4. In these situations suspect biliary stasis or insufficiency if there are strong subjective indicators.
**Some of the complications of Biliary Insufficiency/Stasis**

1. Accumulation of toxins that would normally be excreted in bile.
2. Digestive disorders because factors necessary for digestion are not secreted into the bowel.
3. Inadequate absorption of fat-soluble vitamins A, D, E, and K, and other nutrients absorbed from food.
4. Essential fatty acid deficiency
5. Greatly increased risk of gallstones and biliary obstruction.
6. Further liver damage due to increased pressure on liver.

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**Biliary Obstruction**

1. Biliary obstruction causes cholestasis, a condition of impaired bile flow.
2. Biliary obstruction can occur within the liver itself (intrahepatic) or outside of the liver (extrahepatic).
3. Intrahepatic obstruction is usually caused by damage to the hepatocytes. This may be mild or severe.
4. Extrahepatic obstruction is most often due to a common calculi and usually occurs on a spectrum of mild to severe.
5. GGTP and Alk Phos can be useful in determining the severity of possible obstruction, along with the obvious signs and symptoms.

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**Intra-Hepatic Biliary obstruction**

1. Conditions that cause damage to the liver cells (alcohol-induced injury, viral hepatitis, EBV, infectious mononucleosis, fatty liver congestion, CMV) can cause intra hepatic biliary obstruction.
2. Obstruction in the small biliary channels between the liver cell groups that form the functional units of the liver and create a situation of liver dysfunction.
3. Serum bilirubin levels will be elevated (>20.5) along with both conjugated (>3.4) and unconjugated fractions (>17.1).
4. The unconjugated fraction may be increased due to the liver’s decreased ability to conjugate bilirubin due to liver cell damage.

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**Intra-Hepatic Biliary obstruction Pattern**

1. Suspect intra-hepatic biliary obstruction when the total bilirubin is elevated (>20.5) with increased GGTP (>30), SGPT/ALT (>30), alkaline phosphatase (>100) and/or LDH (>200).
2. Suspect that there is probably some kind of cellular damage occurring in the liver.
**Biliary Obstruction-Extrahepatic**

1. Biliary obstruction usually has a genesis in biliary stasis. This provides the seed for calculi formation.
2. Mild elevations of GGT and Alk phos may be due to a small blockage in the biliary tree.
3. Significant elevations in GGT (greater than 5 times normal reference range) are usually caused by calculi obstructing the biliary tree, liver cell damage, or excess alcohol consumption.
4. Expect Alk phos to be elevated >140 with severe obstruction due to gallstones.

**Severe Biliary Obstruction- Pattern**

1. If GGTP (>85) and alkaline phosphatase (>140) are increased along with a normal or increased SGOT/AST (>55) and SGPT/ALT (>55), biliary obstruction with possible calculi is probable.
2. Biliary obstruction with possible calculi becomes even more likely with an increased total bilirubin (>20.5) and direct bilirubin (>3.4).

**Alcohol Use- Pattern**

1. Increased GGTP without an increase in the other liver enzymes suggests excessive alcohol consumption.
2. If GGTP is increased (>30) along with an increased serum triglyceride level (>1.24), excess alcohol use should be ruled-out.
3. Excessive alcohol use can seriously affect the liver function, therefore we may also see elevated SGOT/AST and SGPT/ALT levels (>30), but the GGTP will usually be higher.

**Fatty Acid Insufficiency**

1. Suspect fatty acid insufficiency with gallbladder dysfunction.
2. You are likely to see decreased triglycerides (<0.79) and/or cholesterol levels in patients that have fatty acid insufficiency.
3. Do not just treat this with fatty acid supplementation.
4. The problem may be due to a lack of bile salts from an underlying biliary stasis or insufficiency.
5. Poor fat metabolism will show an opposite pattern with increased cholesterol (>5.69) and triglycerides (>1.24).
Mineral Balance - Calcium

<table>
<thead>
<tr>
<th></th>
<th>Lab Value</th>
<th>Optimal Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium</td>
<td>2.13 – 2.70 mmol/L</td>
<td>2.30 – 2.50</td>
</tr>
</tbody>
</table>

Calcium - Background

1. The majority of the calcium in the body (98-99%) is stored in the bone and teeth, which act as a major functional store.
2. The body will use this reservoir to maintain the calcium blood levels, which are tightly regulated within a narrow range.
3. Calcium occurs in two forms in the body:
   - Ionic
   - Non-Ionic
4. Ionic calcium is the form of serum calcium that can is used by the body for its many physiological and metabolic functions.

Ionized Calcium - Functions

1. Ionized calcium is used by the body for inflammation and tissue repair, particularly the leukocytes for phagocytosis.
2. It is a vital component of the interstitial matrix facilitating cell to cell adhesion, communication, and cell membrane stability.
3. Ionized calcium is also important in vascular integrity and blood clotting.
4. Other important functions of calcium include:
   - Muscle contraction
   - Transmission of nerve impulses

Calcium Regulation

1. Ionized calcium levels are regulated by vitamin D and parathyroid hormone (PTH), which is produced by the parathyroid gland.
2. PTH is the most important hormone in calcium regulation.
**Calcium Absorption**

1. Calcium absorption is dependent on an optimal acidity of the stomach and is affected by the amount of phosphate and magnesium present.
2. Actual absorption occurs in the upper part of the small intestine.
3. Calcium affects the amount of protein absorption and helps move fats through the intestinal wall.

**Special Note on Calcium**

1. Before considering the clinical implications listed below, please check the serum albumin level to make sure that a decrease in serum albumin is not the cause for a relative serum calcium decrease.
2. Much of the serum calcium is bound to the albumin in the blood.
3. A decreased albumin is the most common cause of a decreased calcium level.
4. For every decrease in albumin by 1 g/L, calcium should be corrected upward by 0.02 mmol/L.

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**Calcium – Clinical Implications**

**HIGH**
- Parathyroid hyperfunction
- Thyroid hypofunction
- Impaired cell membrane health
- Vitamin D (excess ingestion)
- Poor fat emulsification
- Osteoporosis

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**Parathyroid Hyper-Function**

1. Parathyroid hyperfunction will cause an increase in PTH level, which can lead to a significantly increased serum calcium.
2. If the serum calcium is significantly increased (>2.50) with a decreased phosphorous (<0.97) parathyroid hyperfunction is possible.
3. Alkaline phosphatase levels may also be increased (>100), along with a normal or decreased serum or RBC magnesium.
**Parathyroid Hyper-Function**

1. If you suspect parathyroid hyperfunction, follow-up with a serum parathyroid hormone test.
2. If parathyroid hormone levels are also increased presume clinical hyperparathyroidism exists.
3. Hyperparathyroidism may be due to space-occupying lesions on one or more of the glands.
4. Surgical removal may be necessary to determine if there is a neoplasm.

**Calcium – Clinical Implications**

- **LOW**
  - Calcium need
  - Parathyroid hypofunction
  - Hypochlorhydria

**Parathyroid Hypo-Function-Pattern**

1. Parathyroid hypofunction will lead to decreased PTH levels that can cause decreased serum calcium.
2. If calcium is decreased (<2.30) along with a high phosphorous level (>1.29), parathyroid hypofunction is possible. Alkaline phosphatase levels may also be normal or decreased (<70).
3. Follow-up with a serum parathyroid hormone test.
4. If parathyroid hormone levels are also decreased presume clinical hypoparathyroidism exists.

**Calcium deficiency?**

1. There is a predominant belief that calcium deficiency is very common and that most patients would benefit from daily supplementation.
2. A calcium need has more to do with an inability to absorb and utilize dietary calcium than a simple calcium deficiency.
3. Calcium regulation in the body is determined by a number of co-factors that are necessary for adequate digestion, absorption and utilization of calcium.
4. When subjective indicators or lab testing (serum calcium <2.30) identify a potential calcium need, the lack of synergistic co-factors that aid in calcium metabolism is often the main problem.

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Calcium need and/or a need for its co-factors

1. Digestion
2. Vitamin D- increases blood calcium levels
3. Essential Fatty Acid- increase tissue levels
4. Acid-Alkaline balance
5. Macro-minerals (phosphorous, potassium and magnesium)
6. Trace minerals (manganese, boron, copper, zinc)
7. Hormones
   • Calcitonin
   • Parathyroid hormone
   • Sex hormones (Progesterone, Testosterone, Estrogen)

Mineral Balance - Serum Phosphorous

<table>
<thead>
<tr>
<th>Mineral</th>
<th>Lab Value</th>
<th>Optimal Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phosphorous</td>
<td>0.81 – 1.45 mmol/L</td>
<td>0.97 – 1.29</td>
</tr>
</tbody>
</table>

Phosphorous - Background

1. The majority (85%) of the body's phosphorous is stored, in combination with calcium, in the bone.
2. The remainder of the phosphorous is in the cells.
3. The phosphorous is essential for:
   • Bone matrix and hydroxyappetite metabolism
   • Phospholipid and nucleic acid formation
   • Metabolism of glucose and lipids
   • Acid-base regulation
   • Storage and transfer of energy in the form of Adenosine Tri-Phosphate (ATP) and creatine phosphate.

Phosphorous - Discussion

1. Phosphorous levels, like calcium, are regulated by parathyroid hormone (PTH)
2. The net effect of PTH is to decrease serum phosphorous.
3. Phosphorous is a general indicator for digestive dysfunction.
4. Decreased phosphorous levels are associated with hypochlorhydria.
**Blood Chemistry and CBC Analysis—
A Functional Perspective**

**Phosphorous and Calcium**

1. Phosphorous levels should always be evaluated in relation to calcium because of the inverse relationship that exists between the two minerals.
2. An excess in one of the minerals will cause an increase in renal excretion of the other.
3. Phosphorous levels will be increased when calcium levels are decreased and vice versa.

**Calcium/Phosphorous ratio**

1. It is important for the body to maintain a ratio of 10 parts calcium to 4 parts phosphorous in the serum.
2. A low calcium/phosphorous ratio
   - Favors the binding of calcium to phosphorous to form calcium phosphate, decreasing the levels of ionized calcium.
2. A high calcium/phosphorous ratio
   - Favors deposition of calcium into the soft tissue, which decreases the availability of ionized calcium reducing the serum calcium reading.

**Phosphorous— a calcium antagonist?**

1. Phosphorus is often assumed to be a calcium antagonist.
2. This may be true in its supplemental form but organic phosphorous from whole and unrefined foods is naturally buffered with minerals and vitamins that act as synergistic co-factors to increase calcium metabolism.
3. Organic phosphorous should be considered as an essential synergistic co-factor that aids in calcium metabolism.

**Phosphorus— Clinical Implications**

- Diet- excessive phosphorous consumption
- Parathyroid hypofunction
- Bone growth and/or repair
- Renal insufficiency
Diet- excessive Phosphate consumption

1. Serum phosphorous levels may be increased (>1.29) in people who drink a lot of soda.
2. Phosphoric acid is a common additive in sodas and can lead to excessive levels of ingested phosphorous.
3. This can cause significant problems with the calcium/phosphorous ratio, leading to decreased serum calcium and problems with bone metabolism.

Diet - high in refined Carbohydrates

1. Phosphate crosses the cell membrane with glucose.
2. Plasma levels may be decreased after a meal high in refined carbohydrates.
3. A diet high in refined carbohydrates and sugars will deplete phosphorus stores and other important co-factors for carbohydrate metabolism.

Phosphorus – Clinical Implications

- LOW
  - Parathyroid hyperfunction
  - Hypochlorhydria
  - Diet- high in refined carbohydrates
  - Hyperinsulinism
  - Bone growth and repair

Bone Growth (Children) & Bone Repair (Fractures)

1. An increased phosphorous is a normal finding in times of increased bone growth and bone repair.
2. Serum phosphorus will often be > 1.61 with children up to 18 years of age (bone growth). Similar increases may be seen during bone repair (fractures).
3. Expect to see changes in Alkaline Phosphatase levels as well.
**Mineral Balance - Iron**

<table>
<thead>
<tr>
<th></th>
<th>Lab Value</th>
<th>Optimal Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Iron</td>
<td>5.37 – 30.45 μmol/L</td>
<td>8.96 – 17.91</td>
</tr>
<tr>
<td>Ferritin</td>
<td>11 – 193 μg/L</td>
<td>11 – 193</td>
</tr>
<tr>
<td>TIBC</td>
<td>44.8 – 62.7 μmol/dL</td>
<td>44.8 – 62.7</td>
</tr>
</tbody>
</table>

**Iron Metabolism**

1. The majority of dietary iron is in the ferric form.
2. Ferric iron has to be reduced into ferrous iron after ingestion in order to be absorbed.
3. This process requires stomach acid and vitamin C.
4. Iron is absorbed primarily in the duodenum and jejunum.
5. Once absorbed, iron travels in the blood attached to a beta globulin molecule called transferrin.
6. The majority of the iron is taken up by red blood cell precursors in the bone marrow.
7. About 60% of the remainder is stored in the bone marrow, liver and spleen as ferritin, and 40% as hemosiderin.

**Iron Discussion**

1. Serum iron reflects iron bound to serum proteins.
2. The most predominant of which is transferrin.
3. At any one time about 1/3rd of the transferrin is saturated with iron.
4. Serum iron levels will begin to fall somewhere between the depletion of the iron stores and the development of anemia.
5. Serum iron is a useful test for assessing iron deficiency anemia and monitoring its treatment. It can also be helpful in monitoring conditions of iron overload.

**Ferritin Discussion**

1. Main storage form of iron in the body.
2. The most sensitive test to detect iron deficiency.
3. Decreased serum ferritin levels parallel tissue ferritin levels, which in turn reflects the decreased iron storage found in iron deficiency anemia.
4. In most situations the serum ferritin level will occur before changes in serum iron, development of anemia or changes in RBC morphology.
5. The body will do whatever it takes to keep the serum levels of iron at an optimal level.
**TIBC- Discussion**

1. Total iron Binding Capacity is an approximate estimation of the serum transferrin level.
2. Transferrin is the protein that carries the majority of the iron in the blood.
3. The TIBC is useful for helping determine the cause of an anemia, monitoring treatment and for monitoring conditions of iron overload.

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**Iron Metabolism Dysfunction**

1. The following are some of the functional problems associated with iron metabolism that can be assessed using Blood chemistry and CBC analysis:
   - Iron Excess
     - Hemochromatosis
     - Iron Conversion problems
     - Excess iron Consumption
   - Iron Deficiency

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**Increased Iron Levels**

1. Increased iron levels in the body may be due to the following:
   - Hemochromatosis
   - Hemosiderosis
   - Iron conversion problems
   - Excess iron consumption

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**Hemochromatosis and Hemosiderosis**

1. The hallmark of hemochromatosis and hemosiderosis is the excess absorption of iron by the body.
2. Hemochromatosis is an hereditary iron storage disease, which leads to deposition of excess iron in the tissues, especially the liver.
3. Hemochromatosis should be differentiated from hemosiderosis, which is the non-hereditary form of the disease.
4. Hemochromatosis is more common in males, with a clinical onset between the ages of 40-60.
5. The disease can lead to liver damage, cirrhosis, diabetes, liver enlargement, and bronzing of the skin.
Hemochromatosis- Pattern

1. Laboratory changes seen in hemochromatosis include the following:
   • An increased serum iron (>39.4)
   • A decreased TIBC (<44.8)
   • An increased % transferrin saturation (usually > 60%),
   • An increased ferritin level (often >1000 µmol/L).
   • SGOT/AST is usually elevated (>40).

Iron conversion problems

1. If the serum iron is normal or increased (> 17.9) with a decreased RBC (♀ <3.9, ♂ <4.2), HGB (♀ < 135, ♂ < 140) or HCT (♀ < 0.37, ♂ <0.40), consider that there may be an inability of the body to convert inorganic iron (the type found in serum iron) into hemoglobin (organic iron).
2. There may be a concomitant need for B12, folic acid, B6, and/or copper.

Excess Iron Consumption

1. One can consume excess amounts of iron. The following represent some of the common sources:
   • Elevated levels of iron in the drinking water
   • Iron cookware, especially when used to cook acidic foods e.g. tomatoes
   • Consumption of iron containing supplements
2. All of the above are often the reason for an increased serum iron or ferritin.
3. These causes of an increased serum iron should be ruled out before hemochromatosis/iron overload is assumed.

Iron Deficiency Anemia

1. Iron deficiency anemia is the most prevalent anemia worldwide
2. The major causes are:
   • Dietary inadequacies
   • Malabsorption and/or hypochlorhydria
   • Increased iron loss
   • Increased iron requirements e.g. pregnancy
Iron Deficiency Anemia-Pattern

1. Consider iron deficiency if there is:
   • Decreased HCT (♀ <0.37, ♂ <0.40) and/or HGB (♀ <135, ♂ <140)
   • Decreased MCV (<82), MCH (<28), and MCHC (32)
   • A decreased serum iron (<8.96), ferritin (10), % transferrin saturation
   • An increased RDW (>13)

2. Iron deficiency anemia may be secondary to hypochlorhydria if serum phosphorous is decreased (<0.97) and serum globulin is increased (>28) or decreased (<24).

Iron Deficiency Anemia-Iron and other Indices

1. Many physicians make the mistake of only ordering RBC and indices when investigating iron excess or iron deficiency anemia.

2. Without the total serum iron and other iron tests, such as ferritin, TIBC and % transferrin saturation the degree of iron deficiency anemia or iron excess cannot be appreciated.

3. Also, ordering serum iron without a serum ferritin and TIBC has very little clinical value.

Mineral Balance-Copper, Molybdenum, and Zinc

<table>
<thead>
<tr>
<th></th>
<th>Lab Value</th>
<th>Optimal Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uric Acid</td>
<td>131 – 458</td>
<td>208 – 351</td>
</tr>
<tr>
<td>Alk Phos</td>
<td>25 – 140</td>
<td>70 – 100</td>
</tr>
</tbody>
</table>

Copper Deficiency

- Low Uric acid levels (♀ <178, ♂ < 208) have been associated with a potential copper deficiency.
- Copper is required for:
  - The formation of hemoglobin
  - Adrenal hormone synthesis
  - Bone matrix
  - Several enzymes of detoxification formed in the liver
  - The formation of high density lipoproteins (HDL).
Clinical indications of a potential Copper deficiency

1. Fatigue and energy problems (chronic adrenal problems)
2. Chronic iron deficiency anemia (may actually indicate a copper problem as copper is required for proper iron uptake and utilization)
3. Poor skin & connective tissue integrity (easy bruising, ruptured blood vessels, aneurysms etc.)
4. Osteoporosis or poor bone density
5. Bone and Joint abnormalities
6. Impaired immune function

Copper toxicity

- NOTE: Copper is an extremely toxic element when found in excess. Do not supplement without reasonable cause.
- If you suspect copper deficiency confirm levels with a WBC intracellular level as serum and hair mineral testing are not reliable for this element.

Molybdenum Deficiency

1. Xanthine oxidase, the enzyme that converts purine bases into uric acid, contains molybdenum.
2. It has a wide distribution in the body, occurring in breast milk, small intestine, kidneys and the liver, where it is integral to phase II liver detoxification pathways.
3. Low levels of molybdenum, which is fairly common, will compromise Xanthine oxidase activity leading to a decreased Uric acid (<208).

Molybdenum deficiency-Pattern

- Suspect molybdenum deficiency if there is a decreased uric acid level (♀ <178, ♂ ≤ 208) and a normal MCV (82 – 89.9) and MCH (28 – 31.9)
- Rule out Molybdenum deficiency if the following indications are present:
  - Sensitivity to exhaust fumes and gases
  - Sensitivity to perfumes and fragrances
  - Sensitive to MSG, sulfites used in food preparation (red wines, hot dogs etc...)
**Zinc deficiency**

1. Decreased levels of Alkaline Phosphatase (<70) have been associated with zinc deficiency.
2. Alkaline phosphatase is a metalloenzyme, which is a family of zinc dependent enzymes.
3. WBC and/or RBC zinc levels may also be decreased along with a low normal or decreased total WBC (<5.0).
4. Zinc is an essential minerals for:
   - Tissue growth and skin integrity
   - Immune system
   - Cellular processes of translation, transcription and translation
   - Essential Fatty acid metabolism
5. Follow-up a decreased alkaline phosphatase with a zinc taste test.

**Clinical Signs and Symptoms of Zinc Deficiency**

1. White spots on nails
2. Reduced sense of smell or taste
3. Cuts are slow to heal
4. Acne
5. Susceptible to colds, infections and flu
6. BPH

**Electrolyte Balance**

1. A relative electrolyte balance can be determined by the following method:
2. Add chloride and CO2, then subtract this figure from the sodium value.
3. Although similar to the Anion Gap, this formula can be helpful in assessing the electrolyte balance in your patients
4. The net value for good electrolyte balance should be between 9 – 18.
   \[
   (\text{Na}^+) - (\text{Cl}^- + \text{CO}_2) = 9 \text{ to } 18 \text{ optimally}
   \]

**Vitamin Balance**
Anemia is a condition in which there is a decreased amount of hemoglobin, a decreased number of circulating RBCs, and a decrease in the hematocrit. Anemia is a symptom not a disease and the cause of an anemia must be sought out. Many of the causes of anemia can be correlated to nutritional deficiencies:
- Deficiencies of iron
- Deficiencies of vitamins (B12, folate, B6, C) and copper
Anemia can also be caused by increased RBC hemolysis and a decrease in production of RBCs.

**Vitamin Balance and Anemia**

**Vitamin Balance- Markers**

1. Vitamin balance can be assessed by looking at the following elements, which will be out of balance in cases of anemia:
   - RBC count
   - MCV
   - MCH/MCHC
   - Hemoglobin
   - Hematocrit
   - Anion gap

**Vitamin Balance**

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<thead>
<tr>
<th>Element</th>
<th>Lab Value</th>
<th>Optimal Value</th>
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<tbody>
<tr>
<td>RBC Count</td>
<td>♂: 4.6-6.0</td>
<td>4.2 – 4.9</td>
</tr>
<tr>
<td></td>
<td>♀: 3.9-5.5</td>
<td>3.9 – 4.5</td>
</tr>
<tr>
<td>HCT</td>
<td>♂: 0.40 – 0.52</td>
<td>0.40 – 0.48</td>
</tr>
<tr>
<td></td>
<td>♀: 0.36 – 0.47</td>
<td>0.37 – 0.44</td>
</tr>
<tr>
<td>HGB</td>
<td>♂: 135 – 180</td>
<td>140 – 150</td>
</tr>
<tr>
<td></td>
<td>♀: 125 – 160</td>
<td>135 – 145</td>
</tr>
<tr>
<td>MCV</td>
<td>81.0 – 99.0</td>
<td>82.0 – 89.9</td>
</tr>
<tr>
<td>MCH</td>
<td>26.0 – 33.0</td>
<td>28.0 – 31.9</td>
</tr>
<tr>
<td>MCHC</td>
<td>32.0 – 36.0</td>
<td>32.0 – 35.0</td>
</tr>
<tr>
<td>Anion Gap</td>
<td>6 – 16</td>
<td>7 – 12</td>
</tr>
</tbody>
</table>

**Hematocrit- Background**

1. The hematocrit (HCT) is expressed as a percentage of the volume of red blood cells in a known volume of centrifuged blood.
2. The hematocrit will usually parallel the RBC count when the cells are of a normal size. This pattern does not hold true when the RBCs are small (microcytic) or large (macrocytic).
MCV- Background

1. The MCV is a measurement of the volume in cubic microns of an average single red blood cell.
2. MCV indicates whether the red blood cell size appears normal (normocytic), small (microcytic) or large (macrocytic).
3. If the MCV is greater than 89.9, the red cells are macrocytic.
4. If the MCV is less than 82.0, the red cells are microcytic.
5. If the MCV is within the optimum range, the red cells are normocytic.

MCH and MCHC- Background

1. MCH is a calculated value and is an expression of the average weight of hemoglobin per red blood cell.
2. MCHC is a calculated measurement of the average concentration of hemoglobin in the red blood cells using HCT and hemoglobin.
3. MCHC is the value that gives us the chromicity.
4. Low MCHC indicates hypochromic cells associated with iron deficiency anemia.

RDW- Background

1. The RDW is an electronic index.
2. It is essentially an indication of the degree of anisocytosis (abnormal variation in size of red blood cells).
3. It is not a helpful test for those who do not have anemia.
4. Although the RDW will increase with vitamin B12 deficiency, folic acid and iron anemia, it is increased most frequently with vitamin B12 deficiency anemia.

Vitamin Insufficiency

1. The following are some of the functional problems associated with balance that can be assessed using Blood chemistry and CBC analysis:
   - B12/folate deficiency
   - B6 deficiency
   - Vitamin C need
   - Thiamine insufficiency
B12/Folate Deficiency Anemia

1. Vitamin B12 and/or folate deficiency will cause a megaloblastic anemia (large cells)
2. B12 and folic acid are needed for proper nucleus development.
3. In situations of B12/folate deficiency the cytoplasm of the erythrocyte continues to expand until the nucleus has reached its proper size.
4. This leads to large red blood cells (macrocytes)

B12/folic acid deficiency

1. Deficiency may occur for a number of different reasons:
   - Decreased ingestion: vegan diet
   - Impaired absorption: Intrinsic factor deficiency, pernicious anemia, malabsorption, HCl need, hypochlorhydria
   - Competitive parasites
   - Increased requirements: Chronic pancreatic disease, pregnancy, hyperthyroidism
   - Impaired utilization: enzyme deficiencies, abnormal binding proteins

B12/Folate Deficiency Anemia- Pattern

1. If the MCV is > 89.9 in conjunction with an MCH of >31.9, increased RDW (>13), MCHC (>35), and LDH (especially the LDH-1 isoenzyme fraction), and a decreased uric acid level (♀ <178, ♂ <208) the probability of vitamin B-12 or folic acid anemia is high. RBCs and/or HGB and/or HCT will be decreased.
2. This finding should be confirmed with a serum or urinary methylmalonic acid (vitamin B-12) and serum or urinary homocysteine (folic acid and vitamin B-6).

LDH and B12 Deficiency Anemia

1. Serum LDH levels are elevated in about 85% of those with megaloblastic anemia, which is primarily caused by a deficiency in vitamin B12 and/or folate.
2. The megaloblastic changes cause an increased destruction of the red blood cells in the marrow, which causes an increase in LDH.
**Folate Deficiency & Hyper segmented Neutrophils**

1. The presence of hyper segmented neutrophils (5 or more lobes in more than 5% of all neutrophils) has been reported to be more sensitive and reliable than an elevated MCV in detecting megaloblastic anemia and is not affected by coexisting iron deficiency.

2. If MCV is >97 oral supplementation may be ineffective. B12 injections may be needed.

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**Vitamin B6 Deficiency**

1. Vitamin B6, in its active form of pyridoxyl-5-phosphate (P-5-P), is essential for the effective operation of the transferase enzymes.

2. A deficiency in P-5-P from alcoholism, malnutrition, poor assimilation, deficiencies in the diet, etc. will cause decreased levels of aminotransferase enzymes, such as GGTP, SGPT/ALT, and SGOT/AST in general circulation.

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**Vitamin B6 Deficiency - Pattern**

1. B6 deficiency is likely if there is a decreased SGOT/AST (<10) and a concomitant deficiency in GGTP (<10) and/or SGPT/ALT (<10), enzymes that also need B6 for optimum activity.

2. B6 deficiency will also impact red blood cell activity leading to a decreased MCV (<82) and/or MCH (<28) and a normal serum iron and ferritin level.

3. This situation leads to a B6 deficiency anemia.

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**Vitamin C Need**

1. Vitamin C deficiency is common due to lifestyle, diet, pregnancy, strenuous exercise, disease, smoking, stress and inappropriate supplementation.

2. Vitamin C is a complex of nutrients, with co-factors, such as bioflavanoids, certain key chelated ascorbate minerals & copper, which help with absorption & utilization in the body.

3. Continued supplementation with isolated and/or synthetic ascorbic acid can lead to long term vitamin C deficiency.
**Vitamin C Need- Pattern**

1. Albumin will frequently be decreased (<40g/L) along with a decreased HCT (<0.37 in women and or 0.4 in men), HGB (<135 g/L in women and <140 in men), MCH (<28), MCHC (<32), and serum iron (<8.96 µmol/dL).
2. There will also be an increased MCV (>90), alkaline phosphatase (>100), and fibrinogen.
3. The lingual ascorbic acid test is a simple screening tool for tissue levels of vitamin C.
4. Use it to follow up on vitamin C insufficiency.

**Thiamine Need**

1. An increased Anion Gap and a decreased CO₂ is associated with a thiamine need.
2. Thiamine plays an essential role in:
   - Energy transformation- oxidative decarboxylation of pyruvate into acetyl-CoA
   - Synthesis of high energy intermediates e.g. NADPH
   - Membrane and nerve conduction- thiamine is thought to be involved in membrane function and stability
3. Thiamine deficiency is common in the United States, despite the fortification of processed grains and cereals

**Causes of Thiamine deficiency**

1. Increased consumption of thiamine deficient foods
   - Refined foods, especially grains
   - Thiamine levels decrease in food during storage
   - Thiamine is destroyed during cooking
   - Thiamine is inactivated by chlorine
2. Alcohol consumption (one or more drinks per day is enough to induce a thiamine deficiency)
3. Drugs: oral contraceptives will deplete many B-vitamins including Thiamine and Folate.

**Indicators of Thiamine need**

1. The following are early signs of thiamine deficiency:
   - Mood and personality changes
   - Depression and Anxiety
   - Irritability
   - Mental dullness
2. Carbohydrate sensitivity and hypoglycemia
3. Nerve inflammation: burning or numbness of extremities
4. Excess Alcohol
5. Cardiovascular signs: Low blood pressure, rapid resting heart rate
6. Chronic HCL need
7. PMS
Chronic Thiamine Need

1. Patients with a chronic need for thiamine may have a heavy metal body burden, particularly mercury, which interferes with thiamine and its role in the Kreb’s cycle.
2. With chronic thiamine need consider an additional need for the following important co-factors of thiamine metabolism:
   - Zinc
   - Magnesium
   - Manganese
   - Essential fatty acids

Thiamine Need- Pattern

1. Thiamine need may be present if there is an increased Anion gap (>12), a decreased CO₂ (<25), and a low normal HCT and HGB.
2. Thiamine deficiency can be assessed by checking red blood cell Transketolase levels.
3. RBC transketolase is an enzyme that cannot be produced in the absence or deficit of thiamine. This is an expensive test.

Nutrient Deficiency Anemia Summary

<table>
<thead>
<tr>
<th></th>
<th>RBCs</th>
<th>HCT</th>
<th>HGB</th>
<th>MCV</th>
<th>MCH/ MCHC</th>
<th>Iron</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron def.</td>
<td>↓</td>
<td>↓</td>
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<td>↓</td>
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</tr>
<tr>
<td>B12/folate</td>
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<tr>
<td>Copper</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↑/ N</td>
<td></td>
</tr>
<tr>
<td>Vitamin C</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td></td>
</tr>
</tbody>
</table>

Blood Sugar Regulation

The Adrenal glands, Pancreas and Liver working to normalize Blood Glucose
Blood Sugar regulation
Pancreas Markers

1. Blood sugar regulation of the pancreas can be assessed by looking at the following elements:

<table>
<thead>
<tr>
<th>Test</th>
<th>Lab Range</th>
<th>Optimal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Glucose</td>
<td>3.61 – 6.38</td>
<td>4.44 – 5.55</td>
</tr>
<tr>
<td>Hemoglobin A1C</td>
<td>&lt; 0.07</td>
<td>0.041 – 0.057%</td>
</tr>
<tr>
<td>LDH</td>
<td>1 – 240</td>
<td>140 - 200</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>0.34 – 1.7</td>
<td>0.79 – 1.24</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>3.36 – 5.20</td>
<td>3.9 – 5.69</td>
</tr>
<tr>
<td>HDL</td>
<td>1.03 – 2.32</td>
<td>&gt;1.42</td>
</tr>
<tr>
<td>LDL</td>
<td>1.55 – 3.36</td>
<td>&lt;3.1</td>
</tr>
</tbody>
</table>

Blood Glucose- Background

1. Blood glucose is traditionally viewed in terms of diabetes.
2. When viewed with other tests we can use blood glucose as a marker for the beginning stages of blood sugar dysregulation.
3. It is important to remember that a single fasting blood glucose level is not a good screening tool for diabetes.
4. Follow-up abnormal readings with a glucose insulin tolerance test to confirm a diagnosis.

Blood Glucose Regulation

1. Blood Glucose regulation involves an intricate system of inter-connecting hormones:
   • **Insulin**- decreases blood glucose by transporting glucose into the cells by increasing the permeability of glucose through the cellular membrane.
   • **Glucagon**- increases blood glucose by increasing glycogen breakdown in the liver
   • **Other hormones**: The following hormones tend to act on the liver to elevate blood glucose:
     - Epinephrine
     - Cortisol
     - Thyroxine

Hemoglobin A1C-Background

1. The normal lifespan of a red blood cell is about 120 days
2. Glucose combines with the hemoglobin, in a process called glycosolation, to produce a substance called glycohemoglobin.
3. The longer blood glucose levels remain high the greater the amount of glycosylation.
4. Hemoglobin A1C shows the average levels of blood glucose in a 2-3 month period before the test.
5. There is a decreased likelihood of patients developing the long-term complications of diabetes when Hemoglobin A1C levels are kept close to normal.
LDH - Background
1. LDH represents a group of enzymes that are involved in carbohydrate metabolism.
2. LDH is found in almost every tissue where it is primarily involved in the catalytic conversion of pyruvate into lactate, a major product of exercising muscle cells and red blood cell metabolism.
3. A decreased LDH (<140) is a common finding in reactive hypoglycemia.

Syndrome X/Metabolic Syndrome
1. The underlying factor in Syndrome X/Metabolic Syndrome is elevated insulin levels and insulin resistance.
2. Syndrome X/Metabolic Syndrome starts when the body can no longer handle the over-consumption of refined carbohydrates and sugars day in and day out, which causes hyperinsulinemia that leads to insulin resistance.
3. Insulin resistance occurs when the insulin released from the pancreas is no longer able to “unlock” the door that allows the glucose to move into the cell.
4. It is also caused by the over-consumption of trans fatty acids in the form of hydrogenated oil.

Functional Disorders of Blood Sugar Regulation
1. Syndrome X/Metabolic Syndrome and Insulin Resistance
2. Early stage Hyperglycemia
3. Hypoglycemia
   - Reactive
   - Adrenal
   - Liver glycogen storage problem
4. Hypoadrenalism
5. Adrenal Stress

Syndrome X: The Road to Diabetes
Individuals progress through several stages of insulin resistance and glucose intolerance before becoming diabetic.

- Normal Glucose Tolerance
- Hypoglycemia (Hyperinsulinemia with normal cellular activity)
- Insulin Resistance (Hyperinsulinemia with increasing cellular resistance)
- Diabetes: Type II
**Syndrome X: the problems of excessive insulin**

1. Excessive insulin or hyperinsulinemia causes the following:
   - Oxidative stress from free radical production
   - Interferes with delta-6-desaturase
   - Increasing risk for the development of atherosclerosis, type II diabetes, HTN, obesity, some forms of dementia, and other diseases.
   - Insulin resistance: cellular resistance to insulin causes increasing levels of triglycerides, cholesterol and LDL,
   - It may take years to become severe.

**Syndrome X/Metabolic Syndrome- Pattern**

The syndrome is characterized by the following:
- High serum triglycerides (>1.24)
- High cholesterol (>5.69)
- Decreased HDL cholesterol (<1.42)
- Increased serum insulin
- High blood pressure
- Increased Blood glucose (>5.55)
- Increased weight gain and obesity

---

**Syndrome X/Metabolic Syndrome- Treatment**

1. We play an essential role in preventing this cascade from occurring by paying attention to the early warning signs:
   - Slowly increasing blood glucose, cholesterol and triglycerides
   - Decreasing HDL levels
   - Slowly increasing blood pressure and weight gain.

2. It is essential to reduce your patient’s intake of refined carbohydrates, sugars, trans fatty acids and other harmful compounds.

3. By doing this with your patients in their 20s and 30s, you can reverse insulin resistance and stand a very good chance of preventing diabetes and coronary heart disease when they’re older.

**Early Stage Hyperglycemia**

1. Increasing blood glucose levels are an early sign of hyperglycemia and are a sign of possible diabetes.

2. A diagnosis of diabetes mellitus requires a fasting plasma glucose level of > 6.4 on more than one occasion.

3. Elevated blood glucose above the optimal range is a sign that this person may be moving down that road.

4. If blood glucose is increased follow-up with Hemoglobin A1C or glycated hemoglobin.
**Early Stage Hyperglycemia- Pattern**

1. If serum glucose (>5.55) and Hemoglobin A1C (> 0.057) are both elevated, hyperglycemia is probable.
2. Serum triglycerides are often higher than the total cholesterol level in diabetes.
3. Urinary glucose may be increased, HDL levels decreased (<1.42), BUN (>5.71) and creatinine (>97.2) frequently increased with the renal damage associated with diabetes.
4. Follow-up with appropriate testing to confirm the diagnosis e.g. oral glucose insulin tolerance testing.

**Hypoglycemia- Reactive**

1. A low fasting glucose is an indication of blood sugar dysregulation whilst fasting
2. This can be an indication of either an adrenal or a liver connection.
3. Although not diagnostic, a decreased fasting blood glucose along with a decreased LDH (<140) is a common finding in reactive hypoglycemia.
4. Hemoglobin A1C levels may also be decreased (<0.041).
5. LDH is an important enzyme for pyruvate metabolism in glycolysis and is associated with pancreatic function and glucose metabolism.

**Hypoglycemia- Liver Glycogen Problem**

1. A decreased ability of the liver to store and/or produce glycogen can be a major cause of fasting low blood sugar and hypoglycemia, especially if SGPT/ALT is high (>30).
2. The liver requires stimulation from the adrenal glands to keep blood sugar levels up during the fasting state.
3. Patients with this type of hypoglycemia have to have some type of stress in their lives in order to keep going.
4. They need the cortisol release to stimulate the liver to release glycogen.

**Hypoglycemia- Pattern**

1. Suspect hypoglycemia if you see the following pattern:
   - Decreased blood glucose (<4.44)
   - Decreased LDH (<140)
   - Decreased Hemoglobin A1C (<0.041)
2. Suspicion increases with the following strong clinical indicators:
   - Strong craving for sweets (especially true for reactive hypoglycemia)
   - Crave coffee or sweets in the afternoon
   - Sleepy in the afternoon, fatigue relieved by eating
   - Headache and/or shaky if meals are skipped or delayed
   - Irritable before meals
**Blood Sugar regulation Adrenal Markers**

**1.** Blood sugar regulation of the adrenals can be assessed by looking at the following elements:

<table>
<thead>
<tr>
<th>Element</th>
<th>Lab Range</th>
<th>Optimal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium</td>
<td>3.5 – 5.3 mmol/L</td>
<td>4.0 – 4.5 mmol/L</td>
</tr>
<tr>
<td>Sodium</td>
<td>135 – 145 mmol/L</td>
<td>135 – 142 mmol/L</td>
</tr>
</tbody>
</table>

**Potassium- Background**

**1.** Potassium is the main intracellular cation and acts as the primary intracellular pH buffer.

**2.** The majority of potassium (90%) is found inside the cell, with only small amounts found in other tissues, such as bone and blood.

**3.** Intracellular potassium concentration can be as much as 15 to 20 times greater than the serum/plasma.

**4.** Potassium levels will increase with cellular damage, which causes the potassium to leach into the extracellular fluid.

**Potassium Functions**

**1.** It is essential for the body to maintain optimum serum levels even though a small concentration is found outside of the cell.

- Potassium plays an essential role in nerve conduction
- The maintenance of osmotic pressure
- Muscle function
- Cellular transport via the sodium-potassium pump
- Acid-base balance (especially as potassium bicarbonate in the kidneys regulation of pH).

- Potassium, along with calcium and magnesium, controls the rate and force of cardiac muscle contraction and thus controls cardiac output.

**Potassium Regulation**

**1.** Potassium concentration is greatly influenced by the mineralcorticoid adrenal hormones, especially aldosterone.

**2.** Potassium excretion is controlled primarily by aldosterone's effects on the kidney.

**3.** Increased aldosterone levels causes an increased excretion of potassium from the urine and a low serum potassium.

**4.** Potassium levels can be a used as a marker for adrenal dysfunction.
**Potassium (K⁺) - Clinical Implications**

<table>
<thead>
<tr>
<th>HIGH</th>
<th>LOW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenal hypofunction</td>
<td>Adrenal stress</td>
</tr>
<tr>
<td>Dehydration</td>
<td>Drug diuretics</td>
</tr>
<tr>
<td>Tissue destruction</td>
<td>Benign essential hypertension</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td></td>
</tr>
</tbody>
</table>

**Sodium- Background**

1. Sodium is the most prevalent cation in the extracellular fluid, constituting 90% of the electrolyte fluid.
2. Some of the functions of sodium include:
   - Acid-base balance (sodium acts as the chief base of the blood)
   - Maintenance of osmotic pressure
   - Nerve conduction
   - Renal, cardiac and adrenal functions
   - Cellular transport via the sodium-potassium pump
   - Sodium maintains the acidity of the urine.

**Sodium regulation**

1. The body has many complex mechanisms for regulating plasma and extracellular sodium.
2. One of the main mechanisms of control is the adrenal cortex via the mineralcorticoid aldosterone.
3. Aldosterone allows the body to hold onto sodium by causing a decreased excretion of sodium from the urine. As such, sodium levels can be a marker for adrenal dysfunction.
4. Other mechanisms of sodium regulation include:
   - Renal blood flow
   - Carbonic anhydrase enzymatic activity
   - Renin enzymatic secretion
   - Antidiuretic hormone (ADH)/ Vasopressin from posterior pituitary

**Sodium (Na⁺) - Clinical Implications**

<table>
<thead>
<tr>
<th>HIGH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenal Stress</td>
</tr>
<tr>
<td>Cushing’s disease</td>
</tr>
<tr>
<td>Dehydration</td>
</tr>
</tbody>
</table>
**Functional Disorders of Blood Sugar Regulation**

1. Hypoadrenalism
2. Adrenal Stress

---

**Potassium and Sodium-Adrenal Hypofunction**

1. Adrenal hypofunction can cause a decrease in the secretions of both the glucocorticoid and mineralocorticoid hormones.
2. A decrease in aldosterone, the major mineralocorticoid, from adrenal hypofunction will cause:
   - A decrease in the amount of renal potassium excretion, which will cause an increased serum potassium (>4.5).
   - An increase in the amount of renal sodium excretion, which will lead to decreased serum sodium (<135).

---

**Potassium and Sodium-Adrenal Stress**

1. Adrenal Stress can cause an increase in the secretions of both the glucocorticoid and mineralocorticoid hormones.
2. An increase in aldosterone from adrenal stress will cause:
   - An increase in the amount of renal potassium excretion, which will cause a decrease in serum potassium (<4.0).
   - A decrease in the amount of renal sodium excretion or increased sodium resorption, which will cause an increase in serum sodium (>142).

---

**Adrenal Hypofunction-Pattern**

1. Adrenal hypofunction is possible if:
   - Potassium levels are increased (>4.5)
   - Sodium is normal or decreased (<135)
   - Chloride values will often follow sodium

2. Other values that may be out of balance include:
   - Increased triglyceride (>1.24) and cholesterol levels (>5.69)
   - Decreased aldosterone and Cortisol levels

3. Urinary chloride will be increased.
4. Adrenal hypofunction can be confirmed with salivary cortisol studies.
Adrenal Stress Pattern

1. Adrenal stress is possible if:
   • Potassium levels are decreased (<4.0)
   • Sodium is normal or increased (>142)
   • Chloride values will often follow sodium
2. Other values that may be out of balance include:
   • Decreased triglyceride (<0.79) and cholesterol levels (<3.9)
   • Increased aldosterone and cortisol levels.
3. Urinary chloride will be decreased.
4. Adrenal stress can be confirmed with salivary cortisol studies.

Sodium and Addison’s disease

1. In its pathological state, severe hypoadrenia from Addison’s disease will cause increased sodium excretion by decreasing sodium reabsorption from the kidney, leading to severely decreased serum sodium levels.
2. The effects on the body’s ability to balance electrolytes and acid-base balance is enormous, and can push the patient into an “Addisonian crisis”.

Hydration Status

Dehydration

1. Dehydration is a very common problem and is often the primary cause of certain values being outside the optimal range
2. Therefore, dehydration should be factored into your blood chemistry and CBC analysis.
3. Insufficient water is the most common cause of dehydration and is endemic to the population.
4. Both dehydration and over-hydration put significant stress on the kidneys.
5. Adequate hydration is necessary for basic chemistry reactions, digestion, electrolyte balance, hormone transport, renal and cardiac function.
6. An increased albumin (>50) is a sign of dehydration.
Dehydration

1. If you suspect dehydration from history, clinical findings or a blood chemistry screen, evaluate for diuretic use and adequate intake.
2. Over the counter and prescription drugs, botanical medicines, caffeine etc… are common findings.
3. Perform in-office hydration test
   • Find palpable vein in hands
   • Raise to heart level and observe change

Dehydration patterns

1. Short-term dehydration
   - Suspect a short-term (acute) dehydration if there is an increased hemoglobin ($\text{♀} > 145$, $\text{♂} > 150$) and/or hematocrit ($\text{♀} > 0.44$, $\text{♂} > 0.48$) along with an increased RBC count ($\text{♀} > 4.5$, $\text{♂} > 4.9$).
   - Dehydration causes hemo-concentration of the blood sample, leading to a relative increase in these elements. A relative increase in sodium ($>142$) and potassium ($>4.5$) can be noted as well.
2. Long-term dehydration
   - Suspect a long-term (chronic) dehydration if any of the above findings are accompanied by an increased albumin ($>50$), increased BUN ($>5.71$) and/or serum protein ($>74$).

Liver Function

Liver Function - Markers

1. Liver Function can be assessed by looking at the following elements:

<table>
<thead>
<tr>
<th>Element</th>
<th>Lab Range</th>
<th>Optimal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>SGPT/ALT</td>
<td>0 – 45 U/L</td>
<td>10 – 30</td>
</tr>
<tr>
<td>Albumin</td>
<td>35 – 50 g/L</td>
<td>40 – 50</td>
</tr>
</tbody>
</table>
**SGPT/ALT - Background**

1. SGPT/ALT is found in highest quantities in the liver, skeletal muscle, heart, and kidney.
2. SGPT/ALT levels will rise in conditions or situations that cause damage to the hepatocytes.
3. If SGPT/ALT is increased above GGTP and SGOT/AST consider that the problem or area of involvement is possible inside the liver itself.

**SGPT/ALT – Clinical Implications**

- **HIGH**
  - Dysfunction located in the liver
  - Fatty liver (later development)
  - Liver dysfunction
  - Biliary tract obstruction
  - Excessive muscle breakdown or turnover
  - Cirrhosis of the liver
  - Liver cell damage

**Albumin - Background**

1. Albumin is one of the major blood proteins.
2. Serum albumin levels are affected by the health of the liver, which is the primary site of albumin production.
3. Albumin plays a major role in the following:
   - Water distribution
   - Maintaining colloid-osmotic pressure between blood and tissue fluid
   - The transport of hormones etc. in the body (also various drugs).
4. Albumin levels are affected by liver and/or digestive dysfunction.
5. A decreased albumin can be an indication of liver dysfunction, malnutrition, or digestive dysfunction due to HCl need.

**Decreased Albumin Levels**

1. **Liver dysfunction**
   - Albumin is produced almost entirely by the liver, liver dysfunction will affect albumin production and serum albumin levels (<40).

2. **Oxidative stress**
   - Decreased albumin (<40) can be a strong indicator of a frank or developing oxidative stress and excess free radical activity.
Albumin – Clinical Implications

LOW
- Liver dysfunction
- Oxidative stress
- Hypochlorhydria
- Vitamin C need
- Edema
- Digestive dysfunction

Liver Dysfunction

1. One can determine the severity of liver dysfunction by the level of SGPT/ALT.
2. SGPT/ALT, due to its concentration in the hepatocytes, is key in monitoring situations that can cause damage to the liver.
3. A level above the normal reference range is indicative of an emerging process of hepatocyte damage.
4. Levels above the optimal range is indicative of an emerging liver dysfunction.

Liver Dysfunction

1. The following are some of the functional problems associated with the liver that can be assessed using Blood chemistry and CBC analysis:
   - Liver cell damage
   - Liver dysfunction
   - Fatty liver- early and late stage
   - Oxidative stress
2. Functionally oriented liver problems are extremely common and should be evaluated based upon early prognostic indicators.
3. A moderate rise in SGPT/ALT can alert us to a more functional problem within the liver:
   - Developing liver dysfunction
   - Liver congestion (fatty liver)
   - Detoxification/ Oxidative stress issues
   - Conjugation problems
   - Liver cell damage
3. The liver should always be viewed in the context of the hepatobiliary tree and the involvement of SGOT/AST and GGTP.
Fatty Liver (Steatosis)

1. Steatosis is caused by the accumulation of fat in the functional units of the liver.
2. Non Alcoholic Steatotic hepatitis is one of the most common causes of elevated liver enzymes.
3. Fatty liver will cause extensive liver cell damage, so early detection is essential.
4. The beginning stages of fat accumulation in the liver is one of the main causes of “liver congestion”, which can cause a decreased SGPT/ALT level (<10).
5. Later stage steatosis will cause a marked increase in SGPT/ALT.

Causes of Fatty Liver Development

1. A decreased SGPT/ALT is associated with the early development of fatty liver (liver congestion).
2. Some of the reasons for fatty liver to develop include:
   • Diet: fiber deficient diet, trans fatty acids, refined foods, excessive calories
   • Free radical exposure and oxidative stress
   • Obesity
   • Excessive alcohol consumption
   • Prescription drugs (e.g., steroids)
   • Iron overload
   • Solvent exposure

Early Development of Fatty Liver- Pattern

1. If the SGPT/ALT is decreased (<10) with an increased total cholesterol (> 5.69), LDL (>3.1) and triglycerides (>1.24), and a decreased HDL level (<1.42), then the early development of fatty liver is possible.
2. Fatty liver and liver congestion increases the risk of insulin resistance, hypertension, Syndrome X, and type II diabetes mellitus.
3. Fatty changes to the liver tissue can impair the liver’s detoxification ability and increase oxidative stress activity.

Fatty Liver- late Stage

1. Advanced steatosis will cause the SGPT/ALT to be elevated as much as 4 times the upper limit of normal.
2. If the SGPT/ALT is increased above the SGOT/AST and GGTP levels, liver dysfunction due to advanced fatty liver is probable.
3. Consider it more likely if the LDH (>240) and ALP (>140) levels are also increased.
4. Suspect an early development of fatty liver if the SGPT/ALT levels are decreased (<10).
Liver cell damage

1. Liver cell damage will cause a greatly elevated SGPT/ALT along with an elevation in the other liver enzymes
2. Some of the causes of such an increase are:
   - Cirrhosis of the liver
   - Hepatitis: both chronic and acute
   - Infectious mononucleosis, cytomegalovirus
   - Alcoholic liver damage
   - Chemical damage
   - Hepatic necrosis
3. Further testing to identify the cause of the greatly elevated levels is needed.

Oxidative Stress

1. The chem. screen can be used to assess the oxidative stress burden on your patients
2. Oxidative stress is associated with exposure to environmental pollutants, inflammatory diseases and low antioxidant status and is a major factor in chronic disease.
3. A sudden decreased cholesterol for a patient who historically had not had low cholesterol is a strong diagnostic indicator of an increased oxidative stress and potential neoplastic development.
4. Decreased levels of cholesterol (<3.9) put the body at risk for developing oxidative stress, especially lipid peroxidation, and increases the chance of free radical induced diseases.

Cholesterol as an Antioxidant

1. Cholesterol has strong antioxidant activities in the body.
2. Cholesterol, in its unoxidized form, acts as an antioxidant and a free radical scavenger in the body, and as such functions as a natural cell membrane protector.
3. It protects cells from cancer and other free-radical induced diseases.
4. Thus low levels of cholesterol present an increased risk for oxidative stress.
5. In its oxidized state, cholesterol can act as a pro-oxidant and a free radical producer.

Oxidative Stress- pattern

- Oxidative stress should be investigated if a total cholesterol level is suddenly below its historical level, and is seen with:
  - Decreased albumin (<40) and platelet level
  - Decreased lymphocyte count (<20)
  - Increased total globulin (>28) and Uric acid level (>351)
  - Increased Ferritin, bilirubin and LDL levels
**RBC Hemolysis and Oxidative Stress**

1. Oxidative stress can cause an increased destruction of red blood cells; in these situations you will see an elevated bilirubin level.
2. The level of total bilirubin will rise when the level of indirect or unconjugated bilirubin exceeds the liver's ability to clear it from the blood.
3. The direct or conjugated fraction remains normal or slightly elevated.

**Kidney and Genito-urinary Function**

**Oxidative Stress- Other Tests**

1. Investigation of oxidative stress can be accomplished in the office by using the Oxidata Free Radical Test.
2. The test can help determine whether the oxidative activity is too low for optimal physiological function or whether the levels of oxidative stress are high, indicating free radical activity in the body.
3. Other tests to consider include: Acid Phosphatase, serum protein Electrophoresis, CEA, Anti-malignin Antibody, HCG, Alpha Fetoprotein, and Serum Protein Electrophoresis.

**Kidney and Genito-urinary Function Markers**

1. Kidney and Genito-urinary function can be assessed by looking at the following elements:

<table>
<thead>
<tr>
<th>Test</th>
<th>Lab Range</th>
<th>Optimal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>BUN</td>
<td>1.79 – 8.93</td>
<td>3.57 – 5.71</td>
</tr>
<tr>
<td>Creatinine</td>
<td>53.0 – 132.6</td>
<td>70.7 – 97.2</td>
</tr>
<tr>
<td>Alk Phos</td>
<td>24 – 140</td>
<td>70 – 110</td>
</tr>
<tr>
<td>Sodium</td>
<td>135 – 147</td>
<td>135 – 142</td>
</tr>
</tbody>
</table>

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www.BloodChemistryAnalysis.com
**BUN- Discussion**

1. The BUN is a test that is predominantly used to measure kidney function.
2. BUN is useful as a first indicator of renal insufficiency especially if all the other renal indicators are normal.

**Creatinine- Discussion**

1. A disorder of the kidney and/or urinary tract will reduce the excretion of creatinine and thus raise blood serum levels.
2. Creatinine, unlike BUN, is not affected by gender or the amount of dietary protein consumed.
3. Conditions that cause excess protein breakdown (tissue destruction, inflammation, cancer etc.) will not cause increased creatinine levels.

**BUN- Clinical Implications**

- Renal disease
- Renal insufficiency
- Dehydration
- Hypochlorhydria
- Diet- excessive protein intake
- Adrenal hyperfunction
- Dysbiosis
- Edema
- Anterior pituitary dysfunction

**Creatinine – Clinical Implications**

- BPH
- Urinary tract congestion
- Renal disease
- Renal insufficiency
- Uterine hypertrophy
- Creatinine supplementation
- Dehydration
Kidney and Genito-urinary Dysfunction

1. The following are some of the functional problems associated with the kidney and genito-urinary system that can be assessed using Blood chemistry and CBC analysis

   - Renal Disease
   - Renal insufficiency
   - Edema
   - BPH
   - Urinary tract congestion

Renal disease - Pattern

1. An Increased BUN (>8.93), serum creatinine (>132.6), BUN/Creatinine ratio (10-20), Urine specific gravity (1.010 - 1.016), Uric acid (>535), serum phosphorous (>1.45), LDH (>240) and SGOT/AST (>40) is indicative of renal disease.

2. If this pattern is seen, impaired renal function should be ruled out and referred to a qualified practitioner.

3. An elevated BUN above its optimal value (>5.71) found in isolation of the pattern above is more indicative of renal insufficiency.

Renal Insufficiency - Background

1. Renal insufficiency is a slow decrease in kidney function that occurs long before one sees overt renal disease.

2. The kidneys function as much more than a filter for waste and toxins.

3. They regulate fluid and mineral balance, help regulate blood pressure, secrete hormones (erythropoietin, rennin, angiotensin, prostaglandins), and regulate acid-base balance amongst other functions.

4. There are many factors that will cause stress or damage to the kidney affecting these vital functions and contributing to renal insufficiency.

Factors Contributing to Renal Insufficiency

1. The following factors can be very stressful or cause overt damage to the kidneys contributing to renal insufficiency:
   - Dehydration
   - High protein intake, processed foods, sugar, caffeine, alcohol etc.
   - Drugs, both over the counter and prescription
   - Heavy metals, especially cadmium and mercury, which slowly destroy the glomeruli
   - Sub-acute, long-term, low-grade and chronic infections
**Renal Insufficiency - Kidney/Liver connection**

1. Impaired liver function, especially its detoxification function can lead to increased kidney stress.
2. The kidney will often take on many of the detoxification tasks of the liver when the liver becomes compromised.
3. The body will often use the skin as a secondary route of detoxification and as such skin problems that have no known cause are often associated with renal dysfunction.
4. Skin problems may be an indication that the kidney is no longer processing metabolic waste correctly.

**Renal Insufficiency & Hypertension**

1. Renal insufficiency is a very common reason for a mild to moderate elevated blood pressure.
2. The kidney plays an important role in blood pressure control via the rennin-angiotensin system.
3. Many patients with essential hypertension are immediately put on medications to "normalize" blood pressure.
4. These BP medications can further worsen an over-looked underlying kidney insufficiency.
5. Always rule out renal dysfunction with hypertension of unknown etiology.

**Renal insufficiency Pattern**

1. Suspect renal insufficiency if there is an increased BUN level (>5.71) with a normal or increased serum Creatinine (>97.2), a normal to increased Uric Acid (♀ >327, ♂ >351), and an increased serum phosphorus (>1.29).
2. LDH and SGOT/AST will usually be normal.

**Edema**

1. Edema is rarely primary and is most often secondary to other metabolic disturbances e.g. renal dysfunction, food and environmental sensitivities, cardiac muscle stress or endocrine dysfunction.
2. An increased BUN (>5.71) and is associated with edema.
3. When albumin is decreased (<40), osmotic pressure is disturbed and fluid can leak into intracellular spaces, leading to edema.
4. Serum sodium levels may also be decreased (<135) with edema.
Edema

1. Hyponatremia (a decreased serum sodium level) is often reflective of a relative excess of body water rather than a low total body sodium.
2. This can lead to an accumulation of excess water in the body.
3. The following are some of the conditions implicated by this pattern:
   - Congestive heart failure
   - Hypothyroidism
   - Nephritis/Kidney disease

Clinical Note

1. An increased BUN above 5.71 should be viewed as a sign of renal dysfunction. In cases of renal dysfunction the serum creatinine will most likely be elevated.
2. If the serum creatinine is not above 97.2 consider that the problem may be due to an anterior pituitary dysfunction and not renal dysfunction.
3. Anterior pituitary dysfunction is commonly overlooked and this topic will be explored in greater detail in the thyroid sections.

Urinary Tract Congestion

1. Congestion in the urinary tract may cause a back-up into the kidneys leading to an accumulation of creatinine in the blood.
2. This may be indicative of a chronic inflammatory, infectious or obstructive process (i.e. BPH, prostatitis, chronic UTIs, uterine hypertrophy, uterine congestion).
3. Creatinine levels above 97.2 can be used as a prognostic indicator for potential problems in the uterus or prostate
4. This is more likely if other causes for a creatinine increase have been ruled-out e.g. renal disease, prescription drugs.
5. An increased PSA will often show a prostate condition in its later stages.
6. Creatinine levels will be more affected than BUN

BPH- Pattern

- If the serum creatinine is >97.2 in a male over 40 years old, prostatic hypertrophy must be considered.
- Often the creatinine will increase long before the PSA increases.
- The likelihood of BPH increases when there is an increased Creatinine level (>97.2), along with a normal BUN and electrolytes, and an increased monocyte count (>7) and LDH isoenzyme #4, which has a prostatic origin.
BPH – Creatinine vs. PSA

- An increased PSA will often show a prostate condition in its later stages.
- The creatinine changes should be viewed as an early prognostic indicator for potential BPH.
- The following tests may also be indicated with the above pattern:
  - A microscopic examination of the urine for prostate cells.
  - A urinalysis indicating infection.
  - A manual examination of the prostate.

Uterine Hypertrophy or congestion- Pattern

- A chronic inflammatory, infectious or obstructive process of the uterus or urinary tract (Uterine hypertrophy, Uterine inflammation, UTI) can cause blockage of the urinary tract.
- This can cause a back-up of creatinine into the kidneys, impacting normal renal function.
- Suspect urinary tract obstruction or congestion due to uterine hypertrophy or inflammation if there is an increased creatinine level (>97.2), along with a normal BUN and electrolytes.

Thyroid Panel- Introduction

1. Understanding your patients’ thyroid problems can be very complicated.
2. Primary hypothyroidism, which manifests as low T4 and high TSH is well understood.
3. Secondary hypothyroidism, manifesting with low T4 and Low TSH levels is less understood but becoming more common.
4. Thyroid hormone problems secondary to peripheral metabolism are the least understood of the thyroid problems.
**Tests on a Thyroid Panel**

1. We recommend the following tests be on your thyroid panel to maximize your functional thyroid analysis
   - TSH
   - Free T3
   - Free T4

2. We also recommend that you have the following on your general metabolic panel
   - TSH
   - Total T4

3. If you suspect a thyroid problem from the metabolic panel add Free T4 and Free T3 levels.

4. We no longer order FTI, T3 Uptake etc. as Free T3 and Free T4 levels are adequate.

5. All results on a thyroid panel are best analyzed collectively.

---

**Thyroid Panel Ranges**

<table>
<thead>
<tr>
<th></th>
<th>Lab Range</th>
<th>Optimal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>TSH</td>
<td>0.4 – 5.5</td>
<td>1.3 – 2.0</td>
</tr>
<tr>
<td>Free T3</td>
<td>3.59 – 6.56 pmol/L</td>
<td>3.0 – 3.25 pmol/L</td>
</tr>
<tr>
<td>Free T4</td>
<td>9.1 – 31.0 pmol/L</td>
<td>12.90 – 19.31 pmol/L</td>
</tr>
<tr>
<td>Total T3</td>
<td>1.23 – 3.53 nmol/L</td>
<td>1.84 – 1.91</td>
</tr>
<tr>
<td>Total T4</td>
<td>61.8 – 169.9 nmol/L</td>
<td>96.53 – 104.25</td>
</tr>
</tbody>
</table>

---

**Review of Thyroid Hormones**

1. Thyroid gland, in response to stimulation from TSH produces T4, T3 and rT3

2. The synthesis of these hormones requires tyrosine and iodine

3. The synthesis of these hormones within the thyroid gland itself are controlled by a feedback mechanism within the hypothalamic-pituitary-thyroid axis
Review of Thyroid Hormones

4. T4 is the major secretory product of the thyroid.
5. All T4 found in circulation is generated in thyroid unless exogenously administered.
6. Production of T3 and rT3 in thyroid is negligible compared to peripheral conversion.

TSH- Discussion

1. TSH is useful for the determination and potential differentiation of hypothyroidism and hyperthyroidism.
2. TSH is the most sensitive test on a blood chemistry screen for primary clinical hypothyroidism.
3. If there is a clinical picture of hypothyroidism, yet the TSH is normal then investigate other causes such as sub-clinical hypothyroidism, iodine insufficiency, selenium deficiency.

What is a “normal” TSH level?

1. Most US labs have a normal TSH reference range of 0.5 to 5.5.
2. In early 2001, the American Association of Clinical Endocrinologists stated: “Even though a TSH level between 3.0 and 5.0 is in the normal range, it should be considered suspect since it may signal a case of evolving thyroid underactivity.”

What is a “normal” TSH level?

1. The February 2002 issue of the Journal of Clinical Endocrinology and Metabolism said that among the disease-free population (those who do not have any presence of thyroid antibodies, or diagnosed thyroid disease), the mean TSH level is 1.5.
2. It is our assertion that the physiological optimal TSH level is between 1.3 to 2.0, and that levels above that may represent hypothyroid function.
T4 - Discussion
1. T-4 released from thyroid is primarily bound to thyroid binding globulin (TBG).
2. Only about 0.03 – 0.05% of circulating T4 is in a free form.
3. T4 is either converted to T3 or rT3, or eliminated via conjugation, deamination or decarboxylation in the liver.
4. It is estimated that about 70% of T4 produced in the thyroid is eventually deiodinated in peripheral tissues into either T3 or rT3 via the deiodinase enzyme that cleaves an iodine molecule from the quaternary form.
5. Most labs routinely measure total circulating T-4.
6. We recommend that you check Free T4, which is more available for tissue receptors and provides a more accurate measurement for thyroid assessment.

T3- Discussion
1. T-3 is the considered the most metabolically active thyroid hormone.
2. T-3 is 4 -5 times more metabolically active than T-4 and its systemic effects and half-life are shorter.
3. Although some is produced in thyroid, approximately 80 – 85% is produced outside the thyroid, primarily by conversion of T4 in the liver and kidneys.
4. Within the liver and kidney, the enzyme responsible for the peripheral conversion of T3 is a selenium dependent enzyme called 5'-deiodinase.
5. Similar to T4, the majority of T3 is in a bound form.
6. Free T3 represents approximately 8 – 10% of circulating T3.
7. Free T-3 is more available for tissue receptors and provides a more accurate measurement for thyroid assessment.
8. T4 can also be converted in reverse T3 (rT3) in the peripheral tissues.

rT3- Discussion
1. Reverse T3 (rT3) is a metabolically inactive thyroid hormone.
2. Small amounts of rT3 are made within the thyroid; however, 95% of rT3 is produced from peripheral conversion of T4.
3. The enzyme responsible for this conversion is 5'-deiodinase and is not believed to be dependent on selenium.
4. Under normal conditions, 45 – 50% of the daily production of T4 is transformed into rT3.

rT3- Discussion
5. Although an understanding of rT3 is limited, it is thought to be devoid of hormonal activity and acts as a major competitive inhibitor of T3 activity at cellular level.
6. Current research also suggests that rT3 has an inhibitory effect on 5'-deiodinase, suggesting it might interfere with peripheral conversion of T4 into T3.
7. The production of rT3 is subject to a range of environmental, lifestyle and physiological influences.
Free T4 and Free T3 Values

1. Free T4 and Free T3 values can be expressed in either ng/ml or pg/dl
2. The optimal range for Free T4 is 12.9 – 19.31 pmol/L
3. The optimal range for Free T3 may be expressed in either ng/ml (3.0 – 3.25) or in pg/dl (300 – 325)

Factors influencing peripheral metabolism of thyroid hormone

1. As the liver has a primary influence on circulating levels of thyroid hormones, the health and function of the liver plays a critical role in thyroid hormone function.
2. The following have been shown to influence deiodination leading to decreased circulating T3 levels and increased circulation of rT3:
   - Liver dysfunction
   - Liver disease
   - Oxidative stress, antioxidant insufficiency and lipid peroxidation within the liver
   - Heavy metals (cadmium and lead)
3. The health of the kidney will also have a strong influence on circulating levels of thyroid hormone.

Factors influencing peripheral metabolism of thyroid hormone

4. A number of lifestyle factors have a significant impact on peripheral metabolism of thyroid hormones.
5. The following have been shown to influence deiodination leading to decreased circulating T3 levels and increased circulation of rT3:
   - High stress and elevated cortisol levels
   - Selenium deficiency
   - Poor nutrition
   - Fasting
   - Calorie restriction
   - Lack of exercise
   - Alcohol intake
6. There will be a point at which enough of these factors will lead to significant thyroid dysfunction.

Thyroid Dysfunction

Some functional thyroid problems that can be assessed using Blood chemistry analysis

1. Clinical hypothyroidism
   - Primary hypothyroidism
   - Secondary hypothyroidism due to Anterior pituitary dysfunction
2. Subclinical Hypothyroidism
   - Low T3 syndrome
   - Euthyroid sick syndrome
   - Low body temperature (“Wilson’s Thyroid Syndrome”)
3. Hyperthyroidism
4. Iodine insufficiency
5. Selenium insufficiency
Primary Hypothyroidism

1. There are a number of different classifications of clinical hypothyroidism, depending on the endocrine gland that is dysfunctional.
2. In primary hypothyroidism the problem is located in the thyroid gland itself, which fails to produce thyroid hormone.
3. Primary hypothyroidism is often preceded by autoimmune thyroid disease.
4. If you have a patient with suspected thyroid disease you should screen for thyroid antibodies.

Secondary Hypothyroidism

1. In secondary hypothyroidism the problem is due to an anterior pituitary hypofunction, which fails to produce optimum levels of TSH to stimulate the thyroid.
2. Thyroid hypofunction secondary to an anterior pituitary hypofunction (Secondary Hypothyroidism) is getting more common.
3. Anterior pituitary hypofunction is a common problem and one that is frequently mistaken for thyroid hypofunction (the subjective indications are usually identical and the patient's axillary temperature will frequently be below normal).

Primary Hypothyroidism-Pattern

1. TSH levels increased above 2.0
2. Normal or decreased total T4 level (<96.53) and/or T-3 (<1.85), free T4 <12.9, free T3 <3.0
3. Increased cholesterol (>5.69) and triglyceride level (>1.24)

Secondary Hypothyroidism-Anterior Pituitary: Pattern

1. Suspect anterior pituitary dysfunction if the subjective indications of thyroid hypo-function are present and the following pattern is seen:
   • A decreased TSH (<1.30)
   • A decreased T-3 uptake (<0.27)
   • A normal T-4, T-3 and FTI
2. The likelihood increases if serum triglycerides are elevated (>1.24) and the total cholesterol levels are increased (>5.69).
Thyroid Antibody Studies

1. Consider running anti-thyroid antibody studies (i.e. anti-thyroglobulin antibody, thyroid anti-microsomal and thyroid peroxidase antibody) with known or suspected thyroid abnormality.
2. Differentiation between multiple auto-immune conditions (i.e. Hashimoto’s, Grave’s disease and sub-acute thyroiditis) is based upon these antibody titers.
3. With Hashimoto’s and Grave’s disease the titers will be significantly elevated.
4. With sub-acute thyroiditis, the levels are usually slightly increased.

Problems using TSH alone as a marker for Hypothyroidism

1. TSH is often insensitive to mild or borderline cases of hypothyroidism.
2. In many cases TSH levels may be within normal limits yet the patient is suffering from all the classic signs and symptoms of low thyroid.
3. Look at the following to help diagnose subclinical hypothyroidism (low T3 syndrome, Euthyroid sick syndrome, or Wilson’s Thyroid Syndrome):
   • Basal body temperature
   • Achilles return reflex
   • Iodine status
   • Free T3 and Free T4 levels
   • History, and clinical signs and symptoms.

Euthyroid Sick Syndrome

1. Euthyroid sick syndrome is used to describe non-thyroidal illness.
2. It is a condition of normal thyroid gland activity with a reduced peripheral 5’-deiodination conversion of T4 into T3 due to liver or renal dysfunction or disease.
3. There will be an increase in rT3
4. Associated with high cortisol and malnutrition states
5. Very similar to Low T3 syndrome except an underlying dysfunction is present that affects thyroid metabolism.

Euthyroid Sick Syndrome Pattern

1. Low total (<1.84) and Free T3 levels (<3.0)
2. Total T4 levels are usually normal
3. Free T4 levels are normal
4. TSH is either normal or decreased (<1.3)
5. Other findings on blood chem screen with evidence of liver or renal dysfunction:
   • Decreased albumin (<40), increased BUN (>5.71), Increased creatinine (>97.2), decreased potassium (<4.0) and increased sodium (142) (high cortisol), increased SGPT/ALT (>30)
Low T3 Syndrome

1. Low T3 syndrome is another syndrome used to describe non-thyroidal illness.
2. It is a condition of normal thyroid gland activity with a reduced peripheral 5'-deiodination conversion of T4 into T3 with no underlying liver or renal dysfunction or disease.
3. There will be an increase in rT3.
4. Low T3 syndrome is likely to be due to many of the conditions that affect the peripheral conversion of T4 into T3 with a rise in reverse T3 levels (stress, malnutrition, low calorie diets, lack of exercise etc.)

Low T3 Syndrome- Pattern

1. Low total (<1.84) and Free T3 levels (<3.0)
2. Total T4 levels are usually normal
3. Free T4 levels are normal
4. TSH is either normal or decreased (<1.3)
5. Other findings consistent with conditions that decrease peripheral conversion of T4 into T3

Iodine Insufficiency

1. Iodine is an essential nutrient for the production of thyroid hormone.
2. Although thought of as rare, iodine deficiency is actually quite common as there are many reasons for its deficiency:
   • A poor diet
   • Exposure to many halogen compounds can interfere with iodine metabolism (i.e. chlorine, bromine, fluoride).
3. These common compounds render normal iodine uptake extremely difficult and may displace normal stores.

Iodine Insufficiency- Pattern

1. In iodine insufficiency the total T4 will often be decreased (<96.53) as will the free T4 (<12.9), the total T3 is often increased (> 3.25) and there is usually a normal or mildly elevated TSH (>2.0)
2. Suspected iodine deficiency can be followed by using the Iodine Patch Test
Iodine Patch Test

1. The iodine patch test is a functional assessment for iodine status in the body.
2. By painting the skin with a 2% solution of iodine we can see how quickly the body absorbs the available iodine.
3. The quicker the iodine fades, the greater the insufficiency.

Selenium Deficiency-Pattern

1. Selenium is an essential nutrient for 5'-deiodinase activity, the enzyme involved in the peripheral conversion of T4 into T3.
2. Low selenium levels are associated with a diminished deiodination of T4 into T3.
3. Selenium is also necessary to degrade rT3
4. Consider selenium deficiency if the total T-3 is reduced (<1.84) and free T3 is decreased (<3.0) or T-3 uptake is reduced (<0.27) along with a normal TSH and T-4 level.

Low Body temperature-Wilson’s Thyroid Syndrome

1. In this condition patients have all of the symptoms of thyroid disease but their thyroid labs are normal.
2. The thyroid gland in Wilson’s Thyroid syndrome is usually functioning normally.
3. In most cases the thyroid hormone tests, such as TSH and T4 are normal.
4. There may be an associated low normal or decreased total T-3 (<1.84), free T3 (<3.0) and T-4 level (<96.53) and an increased reverse T3 level.

Low Body temperature-Wilson’s Thyroid Syndrome

1. There is a frequent history of high stress, fasting, dieting which causes impaired conversion of T4 into T3.
2. This leads to a low T3:rT3 ratio.
3. There is often a low average daytime oral temperature (<97.8)
4. In Wilson’s thyroid syndrome there is often a low average daytime oral temperature (<97.8)
5. Low daytime oral temperatures are associated with increased reverse T3 levels.
**rT3 and Basal Body Temperature Testing**

1. Reverse T3 levels tend to increase overnight, which causes a slowing down of the metabolism.
2. The body enters a hibernation state: decreased cortisol, increased melatonin and HGH.
3. To determine ability of the body to produce T3 as opposed to rT3 and increase metabolism you want to look at the difference between early AM temperatures and mean daytime temperatures.
4. If the temperatures are similar there is a likelihood that the body is producing higher than optimal levels of rT3.
5. You want to be able to stimulate metabolism and tell body to get out of hibernation state.
6. Eat a good breakfast to give the body the message that it needs to increase T3 and decrease rT3.
7. Skipping breakfast will cause a decrease in T3 and maintain the hibernation state.

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**Thyroid Hormone Replacement and Thyroid Tests**

1. Thyroid hormone values will be changed by replacement thyroid hormone.
2. Total T-4 will usually be increased (>154.4) along with an increased T-3 uptake (>0.37) with the use of synthetic thyroxine (Synthroid, Eltroxin, Levothroid, Levoxin, Levoxyl and Levo-T) and desiccated thyroid preparations (Armour thyroid, Westhroid, Thyroid strong, S-P-T, Thyrar.)

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**Hyperthyroidism- Pattern**

1. Although less common than hypothyroidism, the following pattern may help elucidate a developing or existent hyperthyroid state.
2. Hyperthyroidism is possible if:
   - TSH is low (<1.3)
   - The likelihood increases when there is also an increased T3 (>3.53), Free T3 (>6.56), T3 uptake (>0.37), FTI (>11.0) and/or T4 (>154.4).
3. Consider running thyroid antibody studies to rule out Hashimoto’s thyroiditis and Grave’s disease.

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**Cardiovascular System**
**Cardiovascular System-Markers**

1. The Cardiovascular system can be assessed by looking at the following elements:

<table>
<thead>
<tr>
<th></th>
<th>Lab Range</th>
<th>Optimal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triglycerides</td>
<td>0.34 – 1.7 mmol/L</td>
<td>0.79 – 1.24</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>3.36 – 5.20 mmol/L</td>
<td>3.9 – 5.69</td>
</tr>
<tr>
<td>HDL</td>
<td>1.03 – 2.32</td>
<td>&gt; 1.42</td>
</tr>
<tr>
<td>LDL</td>
<td>&lt; 3.36</td>
<td>&lt; 3.10</td>
</tr>
<tr>
<td>SGOT</td>
<td>0 – 40</td>
<td>10 – 30</td>
</tr>
<tr>
<td>Uric Acid</td>
<td>131 – 458</td>
<td>♂: 208 – 351</td>
</tr>
<tr>
<td></td>
<td></td>
<td>♀: 178 – 327</td>
</tr>
</tbody>
</table>

**Serum Fats- Background**

1. Serum triglycerides, and cholesterol, are sensitive to dietary intake of fat prior to testing.
2. Post-prandial levels of triglycerides and cholesterol begin to rise about 2 hours after a meal, peaking at 4-6 hours.
3. A 12-hour fast is recommended to prevent the influence of dietary intake of fat on the sample.

**Diet and Serum Triglycerides**

1. Elevated dietary fat is almost never the sole cause of elevated serum triglycerides.
2. Elevated serum triglycerides reflect a breakdown in the body’s regulatory capacity and are more associated with blood sugar dysregulation.
3. Look at some of the fundamental issues of fatty acid metabolism (liver, thyroid, blood sugar regulation, fat digestion etc.) before rushing in with “green” supplements to lower cholesterol and triglycerides (niacin, garlic, gugulipid etc.).

**Lowering Serum Triglycerides**

1. It is important to review dietary patterns with patients who present with elevated triglycerides and cholesterol.
2. Restrict and monitor their intake of grains (especially refined) and other foods high in starch, refined carbohydrates, fruit juices, alcohol, high glycemic fruits and vegetables, hydrogenated fats and oils.
3. Serum triglycerides clearance is accomplished by both the liver and by muscle tissue. An effective method of reducing elevated triglyceride levels is to increase muscle demand for energy with vigorous cardiovascular exercise and/or resistance training.
**Triglycerides – Clinical Implications**

**HIGH**

- Cardiovascular disease
- Atherosclerosis
- Hyperlipidemia
- Hyperlipoproteinemia
- Syndrome X/Metabolic Syndrome
- Fatty liver
- Liver congestion
- Insulin resistance
- Poor metabolism and utilization of fats
- Early stage hyperglycemia/Diabetes
- Primary hypothyroidism
- Adrenal cortical dysfunction
- Secondary hypothyroidism-anterior pituitary dysfunction
- Alcoholism

**Cholesterol - Clinical Implications**

**HIGH**

- Cardiovascular disease
- Atherosclerosis
- Hyperlipoproteinemia
- Poor metabolism and utilization of fats
- Primary hypothyroidism
- Adrenal cortical dysfunction
- Secondary hypothyroidism-anterior pituitary dysfunction
- Alcoholism

**Diet and Cholesterol**

1. Focusing on just lowering dietary cholesterol and fat consumption usually has little to no effect on serum cholesterol levels.
2. A diet high in carbohydrates, especially refined carbohydrates, starches and other fast acting sugars and hydrogenated fats is more likely to cause an increase in total cholesterol.
3. The greatest effect on lowering serum cholesterol levels will be made by addressing other factors such as excessive carbohydrate consumption, sedentary lifestyle, smoking, endocrine dysfunction and liver/biliary congestion.

**Cholesterol - Discussion**

1. The liver, the intestines and the skin produce between 60-80% of the body’s cholesterol. The reminder comes from the diet.
2. It is important to remember that cholesterol is not “bad”.
3. It has the following vital functions:
   - Controls cell membrane fluidity.
   - Provides the structural backbone for every steroid hormone in the body
   - The myelin sheaths of nerve fibers are derived from cholesterol.
   - Bile salt synthesis
Decreased Cholesterol Levels-as Bad as Increased?

1. It is well known that elevated cholesterol levels pose an increased risk for developing atherosclerotic coronary artery disease.
2. What is not so well known is that a decreased cholesterol level can be just as harmful.

The problems of Decreased Cholesterol Levels

1. Cholesterol is essential for human physiology (cellular membrane fluidity, myelin sheaths, steroid hormone synthesis, the immune system).
2. Do not presume that a decreased cholesterol is a "good thing".
3. Low levels of cholesterol pose as much, if not more, of a risk to health than high levels.
4. A cholesterol <3.9 should be an indication for further investigation (oxidative stress, lipid peroxidation, decreasing cell to cell communication)

LDL Functions

1. LDL functions to transport cholesterol and other fatty acids from the liver to the peripheral tissues for uptake and metabolism by the cells.
2. It is known as “bad cholesterol” because it is thought that this process of bringing cholesterol from the liver to the peripheral tissue increases the risk for atherosclerosis.
3. LDL levels are inversely related to HDL in terms of their ratios. As LDL increases, HDL levels decrease and vise versa.

LDL - Clinical Implications

<table>
<thead>
<tr>
<th>HIGH</th>
<th>LOW</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atherosclerosis</td>
<td>n/a</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td></td>
</tr>
<tr>
<td>Oxidative stress</td>
<td></td>
</tr>
<tr>
<td>Syndrome X</td>
<td></td>
</tr>
<tr>
<td>Fatty liver</td>
<td></td>
</tr>
<tr>
<td>Diet- high in refined carbohydrates</td>
<td></td>
</tr>
</tbody>
</table>
**HDL Functions**

1. HDL functions to transport cholesterol from the peripheral tissues and vessel walls to the liver for processing and metabolism into bile salts.
2. It is known as “good cholesterol” because it is thought that this process of bringing cholesterol from the peripheral tissue to the liver is protective against atherosclerosis.

**HDL - Discussion**

1. HDL must be viewed in relation to the total cholesterol and the LDL cholesterol levels.
2. If the total cholesterol is low, a decreased HDL level is not considered a cardiovascular risk.
3. If the total cholesterol is elevated, HDL can be used as a strong independent diagnostic indicator to determine the risk for atherosclerotic coronary artery disease.

**HDL and Cardiovascular Disease**

1. Decreased HDL (<1.42) is considered atherogenic.
2. Increased HDL is considered protective.
3. Both HDL and total cholesterol are independent risk factors.
4. It is quite common for one value to be normal and the other value to be elevated; a value that has a favorable effect does not entirely cancel the unfavorable effect of the other.

**HDL - Clinical Implications**

<table>
<thead>
<tr>
<th>LOW</th>
<th>HIGH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperlipidemia</td>
<td>Autoimmune processes</td>
</tr>
<tr>
<td>Atherosclerosis</td>
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<td>Syndrome X</td>
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<td>Oxidative stress</td>
<td></td>
</tr>
<tr>
<td>Heavy metals</td>
<td></td>
</tr>
<tr>
<td>Fatty liver</td>
<td></td>
</tr>
<tr>
<td>Hyperthyroidism</td>
<td></td>
</tr>
<tr>
<td>Lack of exercise/sedentary lifestyle</td>
<td></td>
</tr>
</tbody>
</table>
SGOT/AST - Background

1. SGOT/AST is found in highest quantities in the skeletal muscle, heart, liver, kidney, and lungs.
2. Levels will be increased when liver cells and/or heart muscle cells and/or skeletal muscle cells are damaged.
3. The cause of the damage must be investigated.
4. If SGOT/AST is increased above GGTP and SGPT/ALT consider that the problem or area of involvement is possible outside the liver and biliary tree (i.e. the heart, muscle, kidneys).

SGOT/AST – Clinical Implications

HIGH
- Dysfunction located outside of the liver and Biliary tree
- Developing Congestive Heart Failure
- Acute MI
- Cardiovascular dysfunction: Coronary artery insufficiency
- Liver cell damage
- Liver dysfunction
- Excess muscle breakdown or turnover
- Infectious mononucleosis, EBV, CMV

Cardiovascular Dysfunction

1. The following are some of the functional problems associated with the cardiovascular system that can be assessed using Blood chemistry and CBC analysis
   - Cardiovascular disease
   - Hyperlipidemia
   - Atherosclerosis
   - Oxidative stress
   - Circulatory disorders
   - Early stage CHF

Cardiovascular disease

1. Patients with a triglyceride level that is higher than the total cholesterol level and a decreased HDL (<1.42) have a higher risk for developing cardiovascular disease.
2. High blood fats or hyperlipidemia are associated with developing coronary artery disease due to atherosclerosis.
**Hyperlipidemia**

1. If HDL is less than 25% of the total cholesterol, then there is a strong clinical indication that hyperlipidemia is present.
2. If the serum triglycerides (>1.24) and LDL (>3.1) are also increased, hyperlipidemia is likely present and atherosclerosis should be investigated.
3. Consider testing homocysteine levels.

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**Atherosclerosis**

1. Increased triglyceride (>1.24) and cholesterol levels (>5.69) are associated with hyperlipidemia, which has been shown to indicate a potential risk of developing atherosclerotic coronary artery disease.
2. Although this may be true, it is important to look at many of the other risks for this disease before jumping to conclusion that elevated triglyceride and cholesterol levels are the culprit.
3. Other risks for atherosclerosis and cardiovascular disease include: smoking, elevated homocysteine levels, B6, B12 and folate deficiency, ingestion of chlorine, blood sugar dysregulation and hypertension.

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**Oxidative Stress and Atherosclerosis**

1. Increased LDL levels are associated with increasing oxidative stress and free radical activity.
2. The peroxidation of LDL may promote the accumulations of cholesterol in the smooth muscle cells of arteries, which can lead to the development of atherosclerotic plaque.
3. The platelet count may be high in atherosclerosis due to the platelet involvement in the plaque formation.
4. C-reactive protein may also be elevated.

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Dr. Dicken Weatherby

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Atherosclerosis- Pattern

1. If there is an increased triglyceride level (>2.26) in relation to total cholesterol (>5.69) with an increased uric acid level (>351), a decreased HDL (<1.16) and an increased LDL (>3.1) atherosclerosis is probable.
2. Platelet levels may also be increased (>450).
3. Homocysteine levels are frequently increased with atherosclerosis and should therefore be checked.

Circulatory Disorders

1. Patients with increased uric acid levels (♀ >327, ♂ >351) should be evaluated for circulatory disorders.
2. The enzyme Xanthine oxidase, which is essential for the formation of uric acid, is activated with poor circulation producing elevated uric acid levels as well as a super-oxide radical.
3. Conditions such as Hypertension, Raynaud’s, Atherosclerosis, and Polycythemia… should be considered and treated appropriately.

Developing Congestive Heart Failure (CHF)

1. Increased SGOT/AST indicates a problem or area of involvement is possible outside the liver and biliary tree.
2. One of the main areas is the cardiovascular system
3. Acute damage to the heart muscle will cause cellular damage and the leaking of SGOT/AST from the cell, leading to increased SGOT/AST levels.
4. From a more functional perspective, a rise in SGOT/AST can help elucidate a developing cardiovascular problem especially a developing congestive heart failure

Developing Congestive Heart Failure

1. An increased SGOT/AST level can be used to assess for the development of a weak heart muscle and CHF.

Some of the other indicators may include:
- Yawn frequently from poor oxygenation/air hunger
- Shortness of breath with moderate exertion
- Edema/ankles swell, especially at end of the day
- Cough when prostrate, especially at night
- Poor Cardiac reserve calculation
**Developing CHF- pattern**

1. Consider the possibility of a developing congestive heart failure if SGOT/AST (> 30) is increased higher than an accompanying SGPT/ALT increase.
2. A normal to increased GGTP (>30).
3. Increased alkaline phosphatase (>100).
4. Decreased CO₂ (<30).
5. Other factors to consider are an increased ESR, normal to increased globulin (> 28) and LDH (>200) and an increased uric acid (♂ > 327, ♀ >351).

**Inflammatory Markers**

1. The following elements are helpful in evaluating the effects of inflammation and tissue damage:

<table>
<thead>
<tr>
<th></th>
<th>Lab Range</th>
<th>Optimal Range</th>
</tr>
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<tbody>
<tr>
<td>Uric acid</td>
<td>131 – 458</td>
<td>Males 208 – 351</td>
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<tr>
<td></td>
<td></td>
<td>Females 178 – 327</td>
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<tr>
<td>LDH</td>
<td>1 – 240</td>
<td>140 – 200</td>
</tr>
<tr>
<td>Globulin</td>
<td>20 – 39</td>
<td>24 – 28</td>
</tr>
</tbody>
</table>

**Uric Acid- Background**

1. Uric acid is produced as an end product of purine, nucleic acid and nucleoprotein metabolism.
2. It is produced primarily in the liver.
3. 2/3rds of the uric acid produced daily is stored and excreted by the kidney. The remaining 1/3rd is excreted in the stool.
4. Uric acid is the end-product of Xanthine oxidase activation, an enzyme that is up-regulated with reduced tissue oxygenation, which will increase uric acid levels.
**Increased Uric acid levels**

1. Increased uric acid levels (>535) are seen conventionally as a marker for gout, renal failure and leukemia.
2. Increased uric acid levels may also be due to:
   1. An over-production of uric acid that occurs in conditions of excessive breakdown and catabolism of nucleic acids
   2. Increased destruction of cells
   3. An inability to adequately excrete uric acid.

**Increased Uric acid and inflammation**

1. Increased Uric Acid (♀ >327, ♂ >351) can be used as a very strong indicator for:
   - Potential inflammation
   - Metabolic disturbance
   - Circulatory and oxidative stress
2. When inflammation is present the body starts to lay down protective tissue (bone spurs, atherosclerosis, fibrosis, advanced liver disease etc…), which activates the Xanthine oxidase pathways causing increased uric acid levels.

**Uric acid and diet**

1. Dietary purines (pork, organ meats, shellfish, legumes etc…) may increase uric acid levels, and should therefore be limited when treating a patient with elevated uric acid levels.
2. However, chemical and physical stressors are more common causes of increased uric acid levels than dietary purines.
3. Consider eliminating refined carbohydrates, alcohol, fried foods, processed or hydrogenated fats and caffeine, which may elevate uric acid levels
4. Encourage your patients to remain well hydrated

**Uric Acid - Clinical Implications**

- Gout
- Pre-Clinical gout
- Atherosclerosis
- Oxidative stress
- Arthralgias
- Renal insufficiency
- Renal disease
- Circulatory disorders
- Leaky gut syndrome
- Dietary purines

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Dr. Dicken Weatherby
**LDH - Discussion**

1. LDH is found in many of the tissues of the body, including:
   - The heart
   - Liver
   - Kidney
   - Skeletal muscle
   - Brain, red blood cells and lungs.
2. LDH is especially useful for determining the presence of tissue damage because damage to any of these tissues will cause an elevated serum LDH level.
3. Anyone with a significantly increased total LDH level should have an LDH isoenzyme study performed to provide a more complete differential diagnosis.

**LDH and Tissue destruction**

1. Any type of tissue destruction in the body will cause the rupture of the cell cytoplasm and the release of LDH.
2. It is our recommendation that any patient who has an increase of more than 10 U/L above the reference range should have an LDH isoenzyme fraction study to determine the exact location of the tissue damage.
3. Other values that are associated with tissue destruction are potassium, which is increased when cellular damage occurs, and ESR, which is a general marker for non-specific inflammation.

**LDH - Clinical Implications**

- High
  - Non-specific tissue inflammation
  - Tissue destruction
  - Liver/biliary dysfunction
  - Cardiovascular disease
  - Anemia- B12/folate deficiency
  - Anemia- hemolytic
  - Viral infection

**Immune System Abnormalities**

1. The following are some of the metabolic abnormalities that can be assessed using Blood chemistry and CBC analysis
   - Arthralgias
   - Gout and Pre-clinical gout
   - Auto-immune processes
   - Immune activation
   - Acidic Neuritis
Arthralgias – Clinical pattern

1. Increased Uric Acid levels are associated with chronic inflammation and arthralgias.
2. An increased Uric Acid (♀ >327, ♂ >351) may be a sign of a developing arthralgia especially if seen with an increased ESR (♀ >5, ♂ >10), a decreased (<40) or normal albumin, alkaline phosphatase (<70) and an increased or normal serum calcium (>2.50).
3. Consider the pattern above if there are clinical indications of joint pain and/or inflammation.

Arthralgias- contributing factors

1. The following are some of the contributing factors for the development of arthralgias and should be investigated and treated appropriately:
   - Viral, yeast and bacterial infection
   - Chronic hypochlorhydria
   - Intestinal and systemic parasites (consider amoebic origin)
   - Intestinal dysbiosis
   - Food and environmental sensitivity
   - Heavy metals

Gout- Pattern

1. Gout is a condition of uric acid crystals precipitating in the tissue, especially the big toe (tophi).
2. If there is an increased Uric Acid (♀ >327, ♂ >351), Gout is possible. The likelihood increases as the uric acid level increases and if there is also a decreased phosphorus (<1.29), an increased cholesterol (>5.69), BUN (>5.71) and a normal or increased creatinine (>97.2).

Pre-Clinical Gout

1. Arthralgias or joint pain may actually be a form of pre-clinical gout.
2. Consider this metabolic problem in patients who are unresponsive to treatment for an assumed arthritis, fibromyalgia, or any migrating joint pain or stiffness/inflammation.
3. A patient may in fact be suffering from a pre-clinical gout even when the uric acid is not elevated above the reference range (>535) and the big toe is not swollen.
4. Pre-clinical gout is actually quite common and can be the cause of a uric acid above the optimal range (♀ >327, ♂ >351).
Autoimmune Processes
1. A potential developing auto-immune processes may be associated with a low triglyceride level
2. Low triglycerides have been associated with an increased risk of tissue damage.
3. The problem may be inflammatory or destructive in nature.
4. By no means diagnostic for an autoimmune disease, consider a low triglyceride, along with other tests, as a marker for further investigation.

Autoimmune Processes-Pattern
1. If triglycerides are decreased (<0.45) with low or normal cholesterol (3.9 – 5.69) and an increased HDL (>1.81), then some kind of autoimmune process may be occurring somewhere in the body.
2. Check for increases in LDH levels (>200) and be sure to run an ESR, which will be elevated as a non-specific marker for inflammation.
3. Consider further testing to rule-out tissue inflammation or destruction (C-reactive protein, ANA, rheumatoid factor, serum protein electrophoresis etc.).

Immune Activation
1. The total globulin level constitutes the body’s antibody system.
2. An increased level of total globulins (>28) indicates an increase in one or more of its fractions (alpha 1, alpha 2, beta and gamma globulin).
3. One or more of these fractions has been activated due to an infectious or inflammatory process.

Acidic Neuritis
1. Acidity in the body due to metabolic acidosis can lead to an acidic neuritis to develop.
2. This has been called “chemical sciatica” because of the build-up of lactic and pyruvic acid on major nerve distributions.
3. Consider an acidic neuritis when structural causes have been ruled-out, particularly if the patient is obese, on a highly refined diet or deficient in Kreb’s cycle nutrients (thiamine, magnesium, potassium).
**Acidic Neuritis- Pattern**

1. CO₂ will be decreased (<25)
2. Calcium will be decreased (<2.30)
3. Chloride will be increased (>106)
4. The anion gap will be increased (>12)
5. Eliminate all refined carbohydrates, alcohol and high glycemic index foods and provide appropriate mineral and B-vitamin support, especially thiamine.

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**Impaired cell membrane health**

1. Increased serum calcium (>2.50) along with an increased LDH (>200) can be suggestive of cellular membrane disruption and/or destruction. Potassium may also be elevated (>4.5)
2. Calcium is a vital component of the interstitial matrix facilitating cell to cell adhesion and communication.
3. It will be released into the serum if this matrix is disrupted.
4. Space-occupying lesions should be considered and ruled out with appropriate examination and testing.

---

**SUMMARY**

**Integrating Functional Blood Chemistry Analysis into your Practice**

1. We recommend a full chemistry panel and CBC on every patient:
   - To establish a working baseline if they have not had recent testing
   - To assess current health status
   - To identify and/or confirm patterns or trends from other findings
   - To support findings found in history, physical assessment and in-office labs
   - To use as a prognostic indicator for potential nutritional and/or metabolic imbalances
**Procedure of Integrating Blood Chemistries**

1. Prior to first visit acquire previous labs with other medical records and initial intake forms
2. First visit: history and physical assessment. Order lab work
3. Follow-up visit at 30 days- review progress, repeat physical exam and pertinent history
4. Follow-up labs after 60 days to monitor program and adjust plan
5. Repeat labs at 4-6 month intervals during Health Recovery
6. Check annually for established patients on Health Maintenance plans
# CHEMSCREEN and CBC RESULTS TRACKING FORM STANDARD U.S. UNITS

<table>
<thead>
<tr>
<th>Test</th>
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<th>Result</th>
<th>Optimal</th>
<th>↓/↑</th>
</tr>
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<td>4.1 – 5.7%</td>
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<td>BUN</td>
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<td>10 – 16</td>
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<tr>
<td>Creatinine</td>
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<td>0.8 – 1.1</td>
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<td>Triglycerides</td>
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<td>33 – 236 male</td>
<td>33 – 236 male</td>
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## COMPLETE BLOOD COUNT

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## CONVERSION CHART FOR CONVERTING STANDARD US UNITS INTO STANDARD INTERNATIONAL UNITS

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<td>Potassium</td>
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<td>mmol/L</td>
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<td>Chloride</td>
<td>mEq/L</td>
<td>1</td>
<td>mmol/L</td>
</tr>
<tr>
<td>CO₂</td>
<td>mEq/L</td>
<td>1</td>
<td>mmol/L</td>
</tr>
<tr>
<td>Anion Gap</td>
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<td>g/L</td>
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<tr>
<td>Albumin</td>
<td>g/dL</td>
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<td>g/L</td>
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<td>Phosphorous</td>
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<td>U/L</td>
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<tr>
<td>SGPT(ALT)</td>
<td>U/L</td>
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<td>U/L</td>
</tr>
<tr>
<td>LDH</td>
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<td>U/L</td>
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<td>U/L</td>
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<td>Globulin</td>
<td>g/dL</td>
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<td>g/L</td>
</tr>
<tr>
<td>A/G ratio</td>
<td>Ratio</td>
<td>1</td>
<td>Ratio</td>
</tr>
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<td>BUN/Creat. Ratio</td>
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<td>Ratio</td>
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<td>mmol/L</td>
</tr>
<tr>
<td>HDL</td>
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<td>mmol/L</td>
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<tr>
<td>Chol/HDL</td>
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<tr>
<td>Ferritin</td>
<td>ng/mL</td>
<td>1</td>
<td>µg/L</td>
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<tr>
<td>TIBC</td>
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<tr>
<td>TSH</td>
<td>µIU/mL</td>
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<td>mIU/L</td>
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<tr>
<td>T-3 uptake</td>
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<td>%</td>
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<tr>
<td>T-3</td>
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<td>nmol/L</td>
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<tr>
<td>Free T-3</td>
<td>Pmol/L</td>
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<td>nmol/L</td>
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<td>Free T-4</td>
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<td>12.87</td>
<td>pmol/L</td>
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<td>FTI/ T-7</td>
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<tr>
<td>WBC</td>
<td>x 10^9/mm³</td>
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<td>10^9/L</td>
</tr>
<tr>
<td>RBC</td>
<td>x 10^12/mm³</td>
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<td>10^12/L</td>
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<tr>
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<td>10</td>
<td>g/L</td>
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<tr>
<td>Hematocrit</td>
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<tr>
<td>MCV</td>
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</tr>
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<td>pg</td>
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<td>pg</td>
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<tr>
<td>MCHC</td>
<td>g/dL</td>
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</tr>
<tr>
<td>Platelets</td>
<td>x 10^9/mm³</td>
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<td>10^9/L</td>
</tr>
<tr>
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<td>Calculated</td>
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<tr>
<td>Neutrophils</td>
<td>%</td>
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<td></td>
</tr>
<tr>
<td>Lymphs</td>
<td>%</td>
<td>1</td>
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</tr>
<tr>
<td>Monocytes</td>
<td>%</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Eosinophils</td>
<td>%</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Basophils</td>
<td>%</td>
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<td>TEST</td>
<td>REF. RANGE</td>
<td>RESULT</td>
<td>OPTIMAL</td>
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<tr>
<td>Glucose</td>
<td>3.61 – 6.38</td>
<td>4.44 – 5.55</td>
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<tr>
<td>Hgb A1C</td>
<td>&lt;0.07</td>
<td>0.041 – 0.057</td>
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<tr>
<td>BUN</td>
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<td>3.57 – 5.71</td>
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<tr>
<td>Creatinine</td>
<td>53.0 – 132.6</td>
<td>70.7 – 97.2</td>
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<tr>
<td>Sodium</td>
<td>135 – 147</td>
<td>135 – 142</td>
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<tr>
<td>Potassium</td>
<td>3.5 – 5.3</td>
<td>4.0 – 4.5</td>
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<tr>
<td>Chloride</td>
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<td>100 – 106</td>
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<td>CO₂</td>
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<td>25 – 30</td>
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<td>Anion Gap</td>
<td>6 – 16</td>
<td>7 – 12</td>
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<td>Uric Acid</td>
<td>131 – 458</td>
<td>208 – 351 male</td>
<td>178 – 327 female</td>
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<tr>
<td>Total Protein</td>
<td>60 – 85</td>
<td>69 – 74</td>
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<tr>
<td>Albumin</td>
<td>35 – 55</td>
<td>40 – 50</td>
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<tr>
<td>Calcium</td>
<td>2.13 – 2.70</td>
<td>2.30 – 2.50</td>
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<tr>
<td>Phosphorous</td>
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<td>0.97 – 1.29</td>
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<tr>
<td>Alk Phos</td>
<td>25 – 140</td>
<td>70 – 100</td>
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<tr>
<td>SGOT(ALT)</td>
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<td>10 – 30</td>
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<tr>
<td>SGPT(ALT)</td>
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<tr>
<td>LDH</td>
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<td>140 – 200</td>
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<tr>
<td>total Bilirubin direct</td>
<td>1.7 – 20.5</td>
<td>1.7 – 20.5 (&gt;44.5)</td>
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<tr>
<td>indirect</td>
<td>0 – 3.4</td>
<td>0 – 3.4 (&gt;13.7)</td>
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<td>GGTP</td>
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<td>10 – 30</td>
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<td>Globulin</td>
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<td>24 – 28</td>
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<td>A/G ratio</td>
<td>1.1 – 2.5</td>
<td>1.5 – 2.0</td>
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<tr>
<td>BUN/Creat.</td>
<td>7 – 14</td>
<td>13 – 17</td>
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<tr>
<td>Total iron</td>
<td>5.37 – 30.45</td>
<td>8.96 – 17.91</td>
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<tr>
<td>Cholesterol</td>
<td>3.36 – 5.20</td>
<td>3.9 – 5.69</td>
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<td>Triglycerides</td>
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<td>0.79 – 1.24</td>
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<tr>
<td>LDL</td>
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<td>&lt;3.1</td>
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<tr>
<td>HDL</td>
<td>1.03 – 2.32</td>
<td>&gt;1.42</td>
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<tr>
<td>Chol/HDL</td>
<td>Ratio</td>
<td>&lt;4</td>
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<tr>
<td>Ferritin</td>
<td>33 - 236</td>
<td>33 – 236 male</td>
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</tr>
<tr>
<td>10 - 122</td>
<td>10 – 122 female</td>
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<tr>
<td>TIBC</td>
<td>44.8 – 62.7</td>
<td>44.8 – 62.7</td>
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</tr>
<tr>
<td>TSH</td>
<td>0.35 – 5.50</td>
<td>1.3 – 2.0</td>
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<tr>
<td>Free T-3</td>
<td>3.59 – 6.56</td>
<td>3.0 – 3.25</td>
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<td>T-3</td>
<td>1.23 – 3.53</td>
<td>1.84 – 1.91</td>
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<td>Free T4</td>
<td>9.1 – 31.0</td>
<td>12.9 – 19.31</td>
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<tr>
<td>T-4 thyroxine</td>
<td>61.8 – 169.9</td>
<td>96.53 – 104.25</td>
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</tbody>
</table>

**COMPLETE BLOOD COUNT**

<table>
<thead>
<tr>
<th>TEST</th>
<th>REF. RANGE</th>
<th>RESULT</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>3.7 – 10.5</td>
<td>5.0 – 7.5</td>
</tr>
<tr>
<td>RBC</td>
<td>4.1 – 5.6</td>
<td>4.2 – 4.9 male</td>
</tr>
<tr>
<td></td>
<td>3.8 – 5.1</td>
<td>3.9 – 4.5 fem</td>
</tr>
<tr>
<td>Reticulocyte</td>
<td>0.5 – 1</td>
<td>0.5 – 1</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>125 – 170</td>
<td>140 – 150 male</td>
</tr>
<tr>
<td></td>
<td>115 – 150</td>
<td>135 – 145 fem</td>
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<tr>
<td>Hematocrit</td>
<td>0.36 – 0.50</td>
<td>0.40 – 0.48 male</td>
</tr>
<tr>
<td></td>
<td>0.34 – 0.44</td>
<td>0.37 – 0.44 fem</td>
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<tr>
<td>MCV</td>
<td>80 – 98</td>
<td>82 – 99.9</td>
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<tr>
<td>MCH</td>
<td>27 – 34</td>
<td>28 – 31.9</td>
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<tr>
<td>MCHC</td>
<td>32 – 36</td>
<td>32 – 35</td>
</tr>
<tr>
<td>Platelets</td>
<td>155 – 385</td>
<td>150 – 385 x 1000</td>
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<tr>
<td>RDW</td>
<td>11.7 – 15.0</td>
<td>&lt;13</td>
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<tr>
<td>Neutrophils</td>
<td>40 – 74%</td>
<td>40 – 60%</td>
</tr>
<tr>
<td>Lymphs</td>
<td>14 – 46%</td>
<td>24 – 44%</td>
</tr>
<tr>
<td>Monocytes</td>
<td>4 – 13%</td>
<td>0 – 7%</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0 – 7%</td>
<td>0 – 3%</td>
</tr>
<tr>
<td>Basophils</td>
<td>0 – 3%</td>
<td>0 – 1%</td>
</tr>
</tbody>
</table>