Quick Reference Guide to Urine Dipstick Analysis and Functional Urinalysis



Dicken Weatherby, N.D.

"The Perfect Companion to My In-Office Lab Testing System Reference Manual"

Urine Dipstick Analysis and Microscopy

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Bear Mountain Publishing • Ashland, OR

Urine Dipstick Analysis and Microscopy

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Urine Specific Gravity

Clinical implications

HIGH

Clinical Implication	Additional information	
Abnormal solutes in urine	An ↑ S.G. with ↑ or normal urine volume. Need to check dipstick to confirm	
	presence of protein or glucose.	
Adrenel in sufficiency	A high urinary chloride and a high specific gravity is an indication of adrenal	
Adrenal insufficiency	insufficiency.	
Increased mineral loss	A high specific gravity may be due to increased mineral solutes in the urine.	
Diabetes mellitus	Large amounts of glucose or protein ↑ the S.G. to > 1.050.	
	Note: Every 1% of glucose in the urine will 1 the S.G. 0.004	
Dehydration	Excess water loss from sweating, fever, vomiting	
Other causes of S.G. increase	Hepatic disease, Congestive heart failure, Protein malnutrition, collagen vascular	
	disease	

LOW

Clinical Implication	Additional information	
Congested lymphatic system		
Early chronic renal disease		
Diabetes insipidus	✓ S.G. and ↑↑ volume	
Kidney inflammation and infection	 ✓ S.G. and ✓ volume Glomerulonephritis (inflammation without infection) Pyelonephritis (inflammation with infection) 	

Urine Bilirubin

Normal values: Zero

Clinical implications

Even trace amounts of urinary bilirubin are abnormal and therefore further testing is indicated.

Positive reading

	V	
Clinical Implication	Additional information	
Gall bladder dysfunction	Biliary stasis or gallstones. Further testing should be performed to assess this situation.	
Protein maldigestion	This can interfere with the transport of bilirubin into the small intestine.	
Oxidative stress	Excess red blood cell destruction, leading to increased bilirubin levels, may be caused by increased oxidative stress	
Liver detox stress	Consider phase II liver detoxification problems	
Liver dysfunction (Inflammation or infection causing conjugation problems)	 Infectious hepatitis Cirrhosis of the liver Metastatic disease of the liver Congestive heart failure 	 Gilbert's disease Jaundice Other liver diseases caused by toxic or infectious agents

Note: Urine bilirubin is negative in hemolytic diseases

More comprehensive diagnostic information can be obtained by comparing urine bilirubin with urine urobilinogen levels:

Bilirubin	Urobilinogen	Clinical Implication	
↑	↑	Liver dysfunction, hepatocellular or partial obstruction	
↑	Normal	Biliary stasis or gall stones	
Negative	^	Hemolytic	
Negative	Normal	Negative	

Urine Blood or Hemoglobin

Normal levels: None

<u>Clinical implications</u> Hematuria Non-Hemolyzed

Clinical Implication	Additional information	
Conditions associated with hematuria	 Lower urinary tract infections Kidney stones Hypertension Allergies Urinary tract or kidney cancer Glomerular infection or inflammation Lupus Heavy smokers Trauma 	

Hemolyzed

Clinical Implication	Additional information	
Oxidative stress	Oxidation and breakdown of red blood cells causes an increase in hemolysed blood. Check Oxidata test.	
Other conditions	Liver pathologyAllergies	

Urine Color

Normal values: The color of the urine is straw to amber

Color of Urine	Clinical Implications	
Colorless	 Large fluid intake Diabetes insipidus Untreated diabetes mellitus 	 Alcohol ingestion Severe iron deficiency Chronic interstitial nephritis
Orange-colored	 Concentrated urine (inadequate fluid intake, excessive fluid loss, fever) Bile Drugs (pyridium, rifampin, aco- gantrisin, furoxone, dilantin) 	 Diet (carrot juice, carotenes, riboflavin, food dyes) Uric acid crystals
Brownish color or greenish yellow	 Bilirubin in urine Biliverdin (oxidation of bilirubin on stan indican, pseudomonas infection 	ding), drugs (methylene blue, elavil),
Red (straw to port wine)	 Blood, hemoglobin, or myoglobin, Porphyria (port wine color), Drugs: phenophthaleins, dorbane (laxative), 	 Diet (beets, blackberries), Herbs: cascara, senna, Aniline dyes
Brown	 Blood (acid hematin), Bilirubin and other bile pigments (yellow-brown to yellow green). Urobilinogen, Melanin (melanogin conversion by exposure to light in multiple myeloma, melanotic tumor, addison's disease), 	 Indican, Phenols, Drugs (flagyl, nitrofurantoin, I-dopa, methyldopa, metronidazole, sulfonamides), lysol poisoning (brown-black), Rhubarb
Blue hue	Food dyesMedication	Pseudomonas infectionSome porphyries
Green	Pseudomonas infection	

Urine Glucose

Normal value: Negative

Cli	nical implications	HIGH
Clinical Implication	Additional information	
	 Diabetes mellitus (also ↑ S.G.) 	4. Extreme emotional stress
Glycosuria with high blood	2. Endocrine diseases	5. Obesity
sugars	3. Infections	6. Diabetes insipidus
	1. Renal tubule disease (lowered renal	4. Fanconi's syndrome (amino acid
Glycosuria without a high blood	threshold)	reabsorption defect)
sugar	2. Pregnancy	5. Inflammatory renal disease
	3. Heavy metal poisoning	-

Urine Ketones

Normal value: Negative

Clinic	cal implications	HIGH (Ketosis)
Clinical Implication	Additional information	
Low carbohydrate, & high	Ketones often get produced in these types	of diets due to the lack of carbohydrate
fat/protein diets	consumed (Zone and Atkins type diets)	
Liver dysfunction	Ketosis often occurs with a decreased liver glycogen. There may also be adrenal hypofunction, as cortisol is needed to stimulate the liver to release glycogen.	
Dietary conditions	 Increased fat intake or inability to metabolize fats Starvation and fasting Prolonged vomiting 	 Anorexia Increased protein intake
Carbohydrate maldigestion	This is especially true if the patient is eating carbohydrates and there are ketones in the urine	
Kidney disease or kidney failure	Renal glycosuria	
Blood sugar abnormalities	1. Diabetic acidosis	2. Severe hypoglycemia
Dehydration	Kidneys are unable to eliminate ketones efficiently	
Increased metabolic states	1. Hyperthyroidism 2. Fever	2. Pregnancy or lactation

Urine Leukocyte Esterase

Normal values: Zero.	A color change occurs with >	5 WBCs/high powered field
	5	5 1

Cli	nical implications	Positive reading
Clinical Implication	Additional information	
	 Intestinal inflammation 	Prostatitis
Infection or inflammation	Pyelonephritis (acute or chronic)	Kidney stones
	Cystitis or Urethritis	Acute glomerulonephritis
Other causes for the presence	 retained foreign body 	Fever
of leukocyte esterase	Dehydration	Stress

Urine Nitrites

Normal value: Negative for bacteria

CI	inical implications	Positive reading
Clinical Implication		Additional information
Bacteriauria	urinary tract infection. This te	es the presence of bacteria in the urine, suggesting a est does not confirm an infection, so further testing in the on of urine and urine culture needs to be performed.

 \clubsuit Nitrites along with an \clubsuit Leukocyte esterase = infection

Urine Odor

Normal values: Urine is normally odorless

Ammonia/fetid	•	Presence of bacterial overgrowth	•	Loss of alkaline buffers in the body
Sweetish, brown, frothy	•	Presence of bile (bile duct obstruction)		
Sweet	٠	Look for sugar problems e.g. diabetes	•	Biliary problems
Fruity and sweet	٠	Ketoneuria		
Foul	٠	Fecal contamination, recto-urethral fistula		
Mousy, musty	•	Phenylketonuria		
Maple syrup	•	Maple syrup urine disease		
Any strong, unusual, persistent	•	Maybe herbs or medications		
odor	•	Metabolic disorders		

Urine Protein

Normal Ranges: Negative or trace

Proteinuria

Glomerular damage	Proteinuria is usually the result of an i	increased glomerular filtration rate	
Renal diseases	 Nephritis/glomerulonephritis, Nephrosis, Malignant hypertension, 	Polycystic kidneys,Chronic urinary tract obstruction	
Non-renal diseases	 Allergies Fever, Acute infection, Leukemia/multiple myeloma 	 Toxemia Diabetes mellitus SLE 	
▲ Protein and ▲ Leukocytes	Usually an infection at some level in the urinary tract		

Urine Turbidity or Appearance Normal values: Fresh urine is clear to slightly hazy

Hazy	 Cooling of the sample, Ph change, 	3. RBC's
Cloudy urine- unable to see through the sample	 Amorphous sediment or amorphous crystals, depending on urine ph (phosphates with alkaline urine, urates with acidic urine) Pus, with WBC count > 200 cells / mm3 Blood, with RBC count > 500 cells / mm3 Epithelial cells Bacteria Fat - milky appearance 	 Chylomicrons - creamy color - obstruction of lymph vessels by parasites, thoracic duct obstruction, trauma, or tumor Conjugated bilirubin - parenchymal liver disease, biliary tract obstruction Urobilinogen - parenchymal liver disease, hemolytic disease Oxalic or glycolic acids Mucus

Urine Urobilinogen

Normal Ranges: Trace

HIGH

Clinical Implication	Additio	onal information
Increased destruction of blood cells	Hemolytic anemiaPernicious anemiaMalaria	 ↑ Xenotoxins Infections ↑ Oxidative stress
Hemorrhage into the tissues	Pulmonary infarct	Excessive bruising
Reduced conjugation of bilirubin by the liver ↑ Toxins in the body	↑ Urobilinogen is a sign that the liver	is not functioning very well
Hepatic damage as a result of:	 Gall bladder disease- biliary obstruction Cirrhosis 	Acute hepatitis
Check all conditions that affect blood break down		

LOW

		EOT		
Clinical Implication		Additional information		
Anything that prevents bilirubin		Gall stones	Severe inflammation of biliary ducts	
excretion into th	ne intestines	Biliary stasis	Cancer of the head of the pancreas	
Antibiotic therapy		Antibiotics wipe out the normal digestive flora which may prevent the formation of urobilinogen from bilirubin		
Interfering Facto	Interfering Factors: Diurnal variation: Peak excretion occurs from noon to 4:00 PM			
More comprehen	sive diagnostic info	ormation can be obtained by comparing urine	e urobilinogen with urine bilirubin levels:	
Bilirubin Urobilinogen Clinical Implic		Clinical Implication		
↑	↑	Liver dysfunction, hepatocellular or partial	obstruction	
↑	Normal	Biliary stasis or gall stones		
Negative	^	Hemolytic		
Negative	Normal	Negative		

Urine Volume

Ranges for a 24-hour sample:

Normal volume:	Polyuria:	Oliguria:
800-2000ml	> 2400ml	<800ml
Abnormal solutes:	Poor kidney conc.:	The average value:
>1800ml with S.G.>1.020	<1400ml with S.G.<1.020	1500 ml.

HIGH (>2400ml)

Clinical Implication	Additional information		
Eating a junk food diet	Junk food diets or Standard American Diets can have a diuretic effect on then body causing a mild polyuria		
Ingested diuretics	Taking of diuretic medications and the consumption of tea, coffee, soda, alcohol etc. can cause polyuria		
Other functional problems	1. Allergies 2. Underactive adrenals		
Polyuria - with ↑ BUN and creatinine levels	1. Diabetic ketoacidosis,	2. Partial obstruction of urinary tract	
Polyuria with normal BUN and creatinine levels	1. Diabetes mellitus3. Certain tumors of brain and spin cord2. Diabetes insipiduscord		

LOW (<800ml)

Clinical Implication	Additional information		
Renal causes	1. Renal ischemia	3. Renal disease caused by toxic agent	
Relial Causes	2. Glomerulonephritis and nephritis		
Dehydration	Cause by prolonged vomiting, diarrhea or excess sweating		
Other equade of eliquitie	Over active adrenals, edema, recovering fr	om fever, urinary tract obstruction, cardiac	
Other causes of oliguria	insufficiency		

Urinary Microscopy

Discussion

Urine microscopy is performed on the sediment of urine that has been centrifuged. The sediment is evaluated for cellular elements (red and white blood cells and epithelial cells), casts, crystals and bacteria which might originate from anywhere in the genitourinary tract.

When would you run this test?

1. To investigate and further evaluate positive findings from the Urine reagent dipstick testing

	Discussion	Normal	Clinical implications	Interfering factors
RBCs	RBCs occasionally can be found in the urine. Persistent findings of even small amounts of erythrocytes should be investigated because they come from the kidney and may signal serious renal dysfunction. They are usually diagnostic for glomerular diseases.	0-2/HPF normal >2 is abnormal and needs to be investigated	 Renal or systemic disease Trauma to kidneys Kidney stones Pyelonephritis Cystitis Prostatitis 	Alkaline urine hemolyzes red blood cells Heavy smokers have small amounts of RBCs in urine Menstruation Strenuous exercise
Red cell casts	Red cell casts indicate acute inflammatory or vascular disorders in the glomerulus. Their presence in the urine may be the only manifestation of certain diseases.	Zero casts	Acute glomerulonephritis (GN)Associated with SLE	May appear after strenuous physical activity or contact sports Alkaline urine dissolves RBC casts
WBCs	WBCs may originate from anywhere in the genitourinary tract	0-4/HPF	 >50/HPF indicates acute bacterial infection within urinary tract (perform urine culture) All renal diseases Cystitis or prostatitis Chronic pyelonephritis (PN) 	Strenuous exercise Vaginal discharge- need clean catch
WBC casts	Always come from the kidney tubules Indicates renal parenchymal infection	Zero casts	PN (most common cause)Occasionally acute GN	
Epithelial cells	Cells from the kidneys, bladder or urethra and vagina (squamous)	0-2/HPF (renal) Squamous are common	Acute tubular damageAcute GN	

	Discussion	Normal	Clinical implications	Interfering factors
Epithelial cell casts	Caused by the cast-off tubule cells in the kidney that slowly degenerates. Will appear in large numbers when there is damage to tubule epithelium	Zero	NephrosisGN	
Bacteria	Increased amounts are seen with renal and urinary tract infections	Small amounts in non-clean catch	20 or more bacteria per high powered microscope field may indicate a UTI (do urine culture)	Non-clean catch
Yeast	Usually indicates vaginal contamination	Zero	In males: immunosupression	Non-clean catch
Hyaline casts	Formed from precipitation of protein within the tubules. Their presence depends on flow of urine, urine pH and if present degree of proteinuria. Usually non pathological	0-2/LPF	non-pathological, form after exercise or in concentrated or highly acidic urine With proteinuria Indicates possible damage to glomerular membrane, which permits leakage of proteins: Nephritis Malignant HTN Chronic renal disease	

Urine Crystals

May present with no symptoms or are associated with kidney stone formation. The type of crystal formed varies with urine pH.

Type of Crystal	Ph of urine	Clinical implication
Uric acid	5.0-6.5	gout, acute febrile conditions, chronic nephritis
Amorphous urates, sodium urate	5.0-6.5	salts of Na+, K+, Mg++, Ca++; normal
Calcium oxalate	Up to pH 7.5	Fat digestion problems, ethylene glycol poisoning, DM, liver disease, severe renal disease, ingestion of oxalate-rich foods
Cystine	5.0-6.5	pathological ; indicates an inherited metabolic condition
Leucine	5.0-6.5	pathological ; maple syrup or oathouse urine disease, liver disease
Tyrosine	5.0-6.5	pathological; tyrosinosis, Oathouse urine disease, liver disease
Hippuric acid	5.0-6.5	no significance
Cholesterol	5.0-6.5	indicates excessive tissue breakdown - nephrotic syndrome, chyluria (fat in urine), filariasis, tumors
Triple phosphates	7.5-9.0	ammonium-magnesium-phosphate - with urinary calculi, chronic pyelitis, chronic cystitis, BPH with urinary retention
Amorphous phosphates	7.5-9.0	similar to amorphous urates ; no significance
Calcium carbonate	7.5-9.0	no significance
Calcium phosphate	7.5-9.0	may form calculi
Ammonium urate	7.5-9.0	found with bacterial infection if in freshly voided urine

Urine Dipstick Results form

Client's Name:_____

Practitioner:_____

Pathology Screening With Reagent Test Strip Date:						
TEST	NORMAL		AB	NORMAL	FINDINGS	;
Color	Straw to amber	Colorless	red gr	een/yellow	orange	brown
Turbidity	Clear to hazy	Cloudy	very cloudy	/	mucous	
Volume	1500 ml	< 800ml (oligi	uria)		> 2400ml	(polyuria)
Glucose	Negative	+1	+2		+3	+4
Bilirubin	Negative	+1	+2		+3	
Ketones	Negative	+1	+2		+3	
Blood	Negative	Hemolyzed:	+1 (5-10)	+2 (10-25)	+3 (25-50)	+4 (>50)
BIUUU	Negalive	Non-heme.:	+1 (5-10)	+2 (10-25)	+3 (25-50)	+4 (>50)
Protein	Negative	Trace (5-20m	g) +1 (30mg)) +2 (100mg	ı) +3 (300mg) +4
Urobilinogen	Trace	+1	+2		+3	+4
Nitrites	Negative	Positive				
Leukocytes	Negative	+1 (10-25)	+2 (25-7	75)	+3 (>75)	

Pathology Screening With Reagent Test Strip Date:					
TEST	NORMAL		ABNORMA	L FINDINGS	3
Color	Straw to amber	Colorless	red green/yellow	/ orange	brown
Turbidity	Clear to hazy	Cloudy	very cloudy	mucous	
Volume	1500 ml	< 800ml (oligu	uria)	> 2400m	l (polyuria)
Glucose	Negative	+1	+2	+3	+4
Bilirubin	Negative	+1	+2	+3	
Ketones	Negative	+1	+2	+3	
Blood	Negative	Hemolyzed:	+1 (5-10) +2 (10-25	5) +3 (25-50)	+4 (>50)
Вюба	Negalive	Non-heme.:	+1 (5-10) +2 (10-25	6) +3 (25-50)	+4 (>50)
Protein	Negative	Trace (5-20m	g) +1 (30mg) +2 (100r	ng) +3 (300mg	g) +4
Urobilinogen	Trace	+1	+2	+3	+4
Nitrites	Negative	Positive			
Leukocytes	Negative	+1 (10-25)	+2 (25-75)	+3 (>75)	

Pathology Screening With Reagent Test Strip Date:					
TEST	NORMAL		ABNORMA	L FINDINGS	6
Color	Straw to amber	Colorless	red green/yellow	orange	brown
Turbidity	Clear to hazy	Cloudy	very cloudy	mucous	
Volume	1500 ml	< 800ml (oligi	uria)	> 2400ml	(polyuria)
Glucose	Negative	+1	+2	+3	+4
Bilirubin	Negative	+1	+2	+3	
Ketones	Negative	+1	+2	+3	
Blood	Magativa	Hemolyzed:	+1 (5-10) +2 (10-25)) +3 (25-50)	+4 (>50)
Вюби	Negative	Non-heme.:	+1 (5-10) +2 (10-25)	+3 (25-50)	+4 (>50)
Protein	Negative	Trace (5-20m	g) +1 (30mg) +2 (100m	ng) +3 (300mg) +4
Urobilinogen	Trace	+1	+2	+3	+4
Nitrites	Negative	Positive			
Leukocytes	Negative	+1 (10-25)	+2 (25-75)	+3 (>75)	

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And Other In-Office Tests

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In-Office Lab Testing Assessment Patterns

Introduction

This section focuses on the patterns or combinations that exist between 2 or more elements and the diagnostic information that can be found with such an analysis.

When analyzing the patterns it might be useful to look back at each of the individual component.

The following is a glossary of terms that are used in describing some of these patterns:

Digestion: The breakdown of food particles in the GI tract **Absorption**: Passage of food particles across the intestinal mucosa **Assimilation**: Nutrients are assimilated into the blood stream **Utilization**: Passage of nutrients from the blood through the cell membrane

- 1. Assimilation and digestion
- 2. Acid/Alkaline Assessment
- 3. Electrolyte assessment
- 4. Calcium and mineralization
- 5. Macronutrient Maldigestion Patterns
- 6. Urine bilirubin with urine urobilinogen levels

Assimilation and digestion

PATTERN	INTERPRETATION	CLINICAL IMPLICATIONS
↑ Indican ↑ Sediment	Hypochlorhydria Pancreatic Insufficiency Leaky Gut Syndrome	 High indican levels are a reflection of protein mal-digestion and an excess of undigested food particles. Both of these are signs of hypochlorhydria. High sediment reflects poor breakdown of the absorbed nutrients due to leaky gut syndrome or pancreatic insufficiency (lack or decreased activity of digestive enzymes). Patients with this pattern may inform you that their appetite is extremely high and that they eat even when they are not hungry.
↑ Indican ♦ Sediment	Maldigestion Malabsorption	This pattern indicates poor digestion and absorption of nutrients across the gut wall into the blood and cell. There may be damage to the small intestine mucosa, as a result of a bacterial overgrowth or other infection, causing decreased permeability or a reduced intestinal mucosal surface area. One of the symptoms of this might be an excessive appetite. The maldigestion may be from hypochlorhydria or pancreatic insufficiency.
N indican ✔ Sediment	Malabsorption Deficient Dietary intake	This pattern indicates malabsorption without maldigestion. There may also be a relatively deficient dietary intake as a result of poor diet or a relative reduction in food intake. There may be damage to the small intestine mucosa.
N indican ∱ Sediment	Leaky Gut Syndrome Vitamin/mineral deficiencies	This pattern indicates good digestion but an increased permeability. With increased sediment there is evidence of abnormal metabolites being absorbed through a leaky gut. The increase in abnormal metabolites may be due to a deficiency in minerals and vitamins that act as co-enzymes to the enzymatic processes of digestion. This is a pattern often seen in people who are eating large amounts of one food group
↑ Indican ↑ Calcium	Hypochlorhydria	This pattern is associated with poor digestion, especially proteins, due to an inability to produce enough acidity in the stomach i.e. Hypochlorhydria. Since half of the circulating calcium is bound to protein, a protein deficiency resulting from an HCL deficiency could increase the ionized (diffusible) calcium, which is readily excreted in the urine.
↑ Indican ↓ Calcium	Lowered systemic pH Bicarbonate deficiency ↑ Phosphorous loss	This pattern may suggest a high loss of phosphorous due to increased systemic acidity. This may be result from a deficiency in bicarbonate buffers. There is decreased calcium because it is being used to buffer excess hydrogen ions in the extracellular fluid.

Acid/Alkaline Assessment

PATTERN	INTERPRETATION	CLINICAL IMPLICATIONS
 ▲ Resp. rate ▲ Breath hold ↓ Urine pH ▲ Saliva pH 	Metabolic Acidosis	 Alkaline saliva- the respiratory system kicks in by increasing the rate and depth of breathing to blow off as much CO2 as possible. This will lower the carbonic acid levels in the body leading to alkaline saliva. Acidic urine- this represents the kidney excreting H+ Increased respiratory rate- The body is attempting to blow off CO2 to decrease carbonic acid levels Decreased breath holding time- acidosis causes a decreased oxygen transport and uptake, thus leading to a decreased ability to hold ones breath
 ↑/ ♥ Resp. rate ♥ Breath hold ♥ Urine pH ♥ Saliva pH 	Respiratory Acidosis	 Acid saliva- due to the increased levels of CO2 and carbonic acid Acidic urine- due to the kidney excretion of H+ Increased respiratory rate- The body is attempting to blow off CO2 to decrease carbonic acid levels that have built up as a result of the hypoventilation, which is a hallmark of respiratory acidosis Decreased breath holding time- acidosis causes a decreased oxygen transport and uptake, thus leading to a decreased ability to hold ones breath
 ↑/♥ Resp. rate ↑ Breath hold ↑ Urine pH ↑ Saliva pH 	Respiratory Alkalosis (Also known as stress or anxiety alkalosis)	 Alkaline saliva- due to the increased loss of CO2 and carbonic acid Alkaline urine- due to the kidney retention of H+ The respiratory rate may be increased or decreased- The body is attempting to blow off CO2 to decrease carbonic acid levels but the respiration patterns are often irregular Increased breath holding time- alkalosis causes an increased oxygen transport and uptake, thus leading to an increased ability to hold ones breath
 ✔ Resp. rate ↑ Breath hold ↑ Urine pH ✔ Saliva pH 	Metabolic alkalosis	 Acidic saliva- a slowing of the respiration rate will cause more carbonic acid in the extracellular fluids leading to an acidic saliva Alkaline urine- due to kidney excretion of bicarbonate and retention H+ Decreased respiratory rate- due to the suppression of the respiratory centers (the body is attempting to lessen the blow off CO2 to increase carbonic acid levels) Increased breath holding time- alkalosis causes an increased oxygen transport and uptake, thus leading to an increased ability to hold ones breath

Electrolyte assessment

PATTERN	INTERPRETATION	CLINICAL IMPLICATIONS
 ↓ Urine chloride ↑ Urine pH 	Excess alkaline reserves	The extracellular fluid is alkaline. Large amounts of chloride are reabsorbed resulting in a decreased urine chloride. The renal tubules release bicarbonate and hold onto H+ in order to buffer the excess alkalinity. The urine becomes alkaline. This is a normal variation.
♣ Urine chloride ♥ Urine pH	Excess acid reserves Electrolyte insufficiency	The extracellular fluid is acidic. The body copes by causing the renal tubules to reabsorb bicarbonate in order to buffer the acidity. Urine becomes more acidic. Chloride ion reabsorption is decreased resulting in a high urine chloride. This is a normal variation.
 ♥ Urine chloride ♥ Urine pH 	Potassium deficiency Salt deficiency	The blood is deficient in potassium, from eating the standard American diet, too much refined sugar or diuretic use, produces this pattern. The body is excreting H+ and retaining chloride, which leads to an acidic urine. Because of the low pH the body excretes more potassium. If patient has this pattern and reports that their urine output is low consider sodium deficiency because the body is retaining chloride and excreting H+.
 ↑ Urine chloride ↑ Urine pH ↑ Calcium 	Excess salt	In this pattern the body is excreting bicarbonate and chloride as well as calcium. This pattern is seen in people who consume excess amounts of salt.
 ↑ Urine chloride ↑ Urine pH ↓ Calcium 	Excess potassium	This pattern is similar but different from the one above. In this pattern the body is excreting bicarbonate and chloride, but retaining calcium. This pattern is seen in salt deficient diets or people who are taking too much potassium.

Calcium and mineralization

PATTERN	INTERPRETATION	CLINICAL IMPLICATIONS
 ↓ Urine pH ↓ Calcium 	Excess stomach acid	 Excess stomach acid- possible causes often associated with this pattern are: Very high protein diet Magnesium deficiency, because magnesium neutralizes HCI in the stomach. Medications Taking Betaine HCI Acid retention due to kidney disease Ketosis from fasting or diabetes
⊎ Urine pH ↑ Calcium	Complex carbohydrate deficiency Alkaline mineral deficiency	Complex carbohydrate deficiency associated with the standard American Diet i.e. fast food diet high in sugar and protein (↑ sugar can cause ↑ calcium in the urine) Alkaline minerals are being depleted in order to alkalinize the cell. A pattern seen in respiratory acidosis and respiratory conditions such as asthma and emphysema. You may see this pattern after an acute asthma attack.
↑ Urine pH↓ Calcium	Hypochlorhydria	 Hypochlorhydria can cause poor protein digestion leading to low calcium levels since half of the calcium is bound to protein. It is also suggestive of the following: Poor protein and calcium digestion and transportation due to Hypochlorhydria Poor reserve levels of calcium in the bones Fatty acid deficiency.
 ↑ Urine pH ↑ Calcium 	Protein deficiency	This pattern can be due to protein deficiency due to low protein diet or poor protein absorption. Use of protease to increase absorption may be useful. The increase in calcium may be due to the intake of a non-ionizing form of calcium
N Urine pH ✔ Calcium	Low calcium levels in body	May be caused by insufficient intake of calcium or other factors that affect calcium digestion, absorption and utilization. Most of the unabsorbed calcium will be excreted in the stool.

Macronutrient Maldigestion Patterns

PATTERN	INTERPRETATION	CLINICAL IMPLICATIONS
 ♥ Urine chloride ↑ S.G. 	Protein maldigestion	This pattern indicates a difficulty in digesting protein either from a deficiency in protease enzyme or hypochlorhydria. This is associated with a loss of muscle mass, poor recovery time after exercise, hypoglycemia/blood sugar dysregulation, and poor utilization of calcium and magnesium, which must bind with amino acids to be fully assimilated. People with this pattern may also have intestinal mucosal integrity problems causing ileocecal valve problems, constipation and other lower bowel problems. This may be due to glutamine deficiencies.
 ↓ Urine chloride ↓ S.G. 	Fat maldigestion	This pattern indicates a difficulty in dealing with fats either from a deficiency in lipase enzymes or poor bile emulsification. Your patients may talk about having a fat intolerance. This is associated with a deficiency in essential fatty acids, fat soluble nutrient deficiencies and liver and/or gallbladder problems.
 ↑ Urine chloride ↑ S.G. 	Fiber and carbohydrate maldigestion	This pattern indicates fiber and carbohydrate maldigestion and metabolism, which may result from a deficiency in amylase or cellulase, or a high carbohydrate, low protein, low sodium and low fat diet. This pattern is associated with irritable bowel like symptoms, such as diarrhea. With this combination the pituitary increases the stimulation of ADH and GH to retain electrolytes. The patient may suffer from poor circulation, cold hands and feet, and a low sex drive.
↑ Urine chloride ↓ S.G.	Sugar maldigestion	 This pattern is common in people who have problem digesting and handling sugar. Patients may consume large amounts of carbohydrates and say that they are sugar intolerant. This pattern is associated with the following conditions: Sugar handling difficulties Malabsorption, Decreased cell permeability Sugar intolerance may also lead to depression, insomnia, emotional instability, and panic attacks.

Urine bilirubin with urine urobilinogen levels

PATTERN	INTERPRETATION	CLINICAL IMPLICATIONS
↑ bilirubin ↑ Urobilinogen	Liver dysfunction	This pattern has its origin in the liver with possible hepatocellular dysfunction or partial obstruction
↑ Bilirubin N Urobilinogen	Biliary Stasis	This pattern is associated with more of a gallbladder origin either biliary stasis with congested bile or gall stones
Neg Bilirubin ↑ Urobilinogen	Hemolytic in origin	This pattern is more hemolytic in origin. There is an increase in red blood cell destruction due to hemolytic anemia, oxidative stress, ↑ xenotoxins.

Other patterns:

Increased Oxidative Stress	 ↑ Oxidata test ↑ Urinary urobilinogen ↑ Hemolysed blood in urine
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CONDITIONS AND TERRAIN ASSESSMENT TESTS

CONDITION	HIGH	LOW
Adrenal		✓ Urine chloride
hyperfunctioning		
Adrenal	↑ Urine chloride	
hypofunctioning		
Alkaline mineral	↑ Saliva pH	✓ Saliva pH
insufficiency	↑ Calcium oxalate sediment	
	↑ Urine chloride	
Antioxidant	↑ Oxidata test	
insufficiency		
Bowel toxemia	↑ Indican	
	↑ Calcium phos. sediment	
Carbohydrate	↑ Urine chloride	l ✔ Saliva pH
maldigestion	↑ Specific gravity	
	↑ Urine ketones	
Complex	↑ Urine Calcium	✓ Urine pH
carbohydrate		
deficiency		
Deficient dietary	Normal Indican	
intake		
Dysbiosis	↑ Indican	
Electrolyte	↑ Urine chloride	
insufficiency		
Electrolyte stress	↑ Urine pH	Urine chloride
Essential fatty acid		✓ Saliva pH
deficiency		_
	↑ Indican	↓ Urine calcium
Excess protein intake	↑ Uric acid sediment	
	↑ Urine ketones	

CONDITION	HIGH	LOW
Fat maldigestion	 ↑ Indican ↑ Calcium oxalate sediment 	 ↓ Urine pH ↓ Saliva pH
		 ↓ Urine chloride ↓ Specific gravity
Gallbladder	↑ Calcium oxalate sediment	
insufficiency	↑ Urine Bilirubin	
	↑ Saliva pH	↓ Urine calcium
	↑ Indican	
Hypochlorhydria	↑ Uric acid sediment	
	↑ Urine chloride	
	↑ Urine pH	
Hypothyroidism,		Basal body temp
Subclinical		↓ Iodine
Cubennear		
Immune dysfunction	↑ Urine pH	
Iodine insufficiency		
	↑ 1 st AM Urine pH	
Kidney stress	↑ Urine chloride	
	↑ Oxidata test	
Leaky gut syndrome	↑ Total sediment	
Leaky gut syndronie	↑ Indican	
	↑ 1 st AM Urine pH	
Liver stress	↑ Urine bilirubin	
	↑ Urine ketones	
	↑ Urine urobilinogen	
Low calcium levels		
Low redox potential		✓ Oxidata test
	↑ Indican	🖌 Saliva pH
Malabsorption		Total urine sediment

CONDITION	HIGH	LOW
Maldination	↑ Saliva pH	✓ Urine pH
Maldigestion	 ↑ Indican ↑ Oxidata test 	
	♠ Respiration rate	Breath holding time
Metabolic acidosis	↑ Saliva pH	 ↓ Urine pH ↓ Calcium
	↑ Breath holding time	Respiration rate
Metabolic alkalosis	 ↑ Urine pH ↑ Calcium 	✓ Saliva pH
	▲ Oxidata test	
Oxidative stress	↑ Urine chloride ↑ Urine bilirubin	
Oxidative stress	↑ Onne billrubin ↑ Urine urobilinogen	
	▲ Urine blood- hemolysed	
Pancreatic	↑ Total sediment	✓ Urine pH
insufficiency		✓ Saliva pH
Protein deficiency	↑ Urine pH	
	↑ Urine calcium	
	↑ Urine pH	
	↑ Indican	
Protein maldigestion	↑ Uric acid sediment	
	↑ Specific gravity	
	↑ Urine bilirubin	At Despiration rate
	↑ Respiration rate	✓ Respiration rate
Respiratory acidosis	↑ Urine calcium	 Breath holding time Urine pH
		♥ Onne pH ♥ Saliva pH
	▲ Respiration rate	✓ Saliva pri ✓ Respiration rate
	▲ Breath holding time	Ψ calcium
Respiratory alkalosis	↑ Saliva pH	
	↑Urine pH	

INDIVIDUAL TESTS

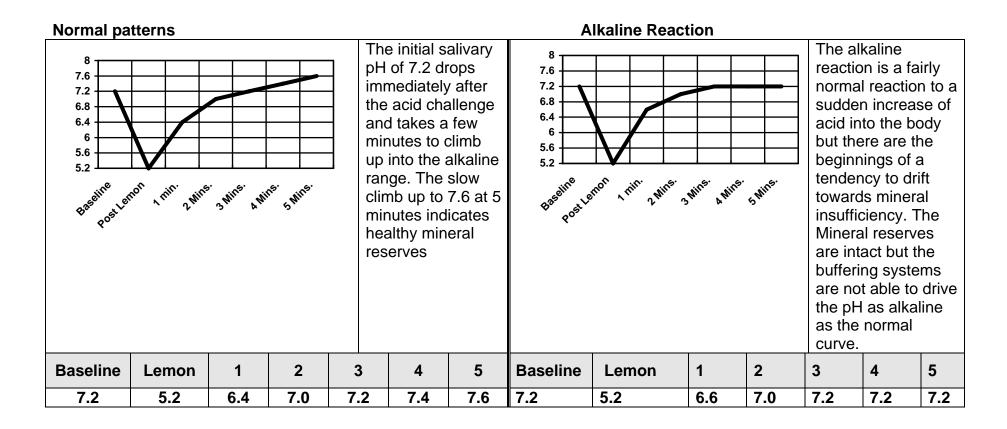
Acid-base Terrain

Tests used to identify patterns of acid/alkaline imbalance

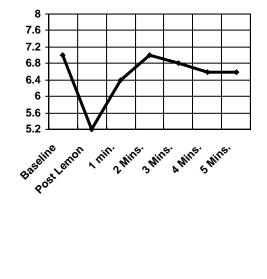
↑ Breath hold	↑ Resp. Rate	↑ Urine pH	∱ Saliva pH
 Metabolic alkalosis Respiratory alkalosis 	 Metabolic acidosis Respiratory acidosis (compensation) Respiratory alkalosis (acute) Sympathetic stress 	 Bacterial infection Susceptibility to yeast and viruses Protein maldigestion Alkalosis (respiratory and metabolic) Calcium metabolism problems 	 Metabolic acidosis Respiratory alkalosis Maldigestion Hypochlorhydria Sympathetic dominance Alkaline mineral insufficiency Dental tartar

		↓ Urine pH	↓ Saliva pH
 Metabolic acidosis Respiratory acidosis Anemia Antioxidant insufficiency Anxiety Stress 	 Metabolic alkalosis Respiratory acidosis (acute/primary cause) Respiratory alkalosis (Compensation) 	 Maldigestion Carbohydrate and fat maldigestion Phase III detoxification issues Pancreatic insufficiency Acidosis (respiratory and metabolic) Inflammation Arthritis 	 Metabolic alkalosis Respiratory acidosis Malabsorption Carbohydrate maldigestion Pancreatic insufficiency EFA deficiency Fat digestion problems Alkaline mineral insufficiency Dental caries

Dr. Bieler's salivary pH acid challenge-Identifying Imbalances in Secondary buffering systems



2. Mineral insufficiency

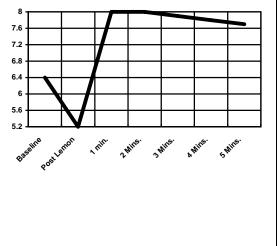


In the mineral insufficiency pattern the initial salivary pH of 7.2 drops immediately with the acid challenge and takes a few minutes to climb up to the alkaline range. The slow climb up to a pH of 6.8 at 2 minutes starts to look like the normal curve, but it fails to completely alkalinize the saliva. This is an indication of mineral insufficiency. There are mineral reserves present but they are not replete enough to fully buffer the acidity.

The more the curve begins to drop the weaker the reserves are.

Baseline	Lemon	1	2	3	4	5
7.0	5.2	6.4	7.0	6.8	6.6	6.6

3. Hypersympathetic overload with mineral insufficiency



Starting point is acidic at 6.4. This pattern is already displaying signs of buffering problems before the test has started.

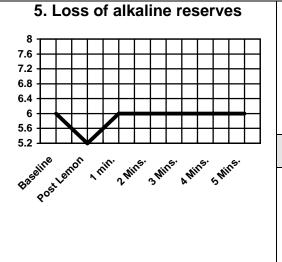
The alkaline spike after 1 minute indicates that ammonia is being used as a buffer. Ammonia, and not minerals, is being released. You may notice the ammonia response in the urine, which may have an ammonia smell.

This patient will complain of being wiped out and fatigued. They probably do not sleep well, are stressed and complain of feeling depleted. Any types of stress reduction techniques are essential for these people along with adrenal restoration. They often complain of not being able to relax. Notice also that the curve does not come down very quickly. The ammonia is quite a long term buffer.

Baseline	Lemon	1	2	3	4	5
6.4	5.2	8	8	7.9	7.8	7.7

4. Hypersympathetic overload with signs of mineral sufficiency

8 7.6 7.2 6.8 6.4 6 5.6 5.2 8 8 6.4 6 5.6 5.2 8 8 8 9 05 1,2 00 1,10 1,10 1,10 1,10 1,10 1,10 1,	IT. 2 MINS. 3 MINS. 4 MINS. 5 MINS.	still the ar coming of	mmonia spike but	after 2 minutes the		netic patient. There i eral reserve activity nal range.	is
Baseline	Lemon	1	2	3	4	5	
6.8	5.2	8.0	7.6	7.4	7.4	7.4	



This pattern is an indication of a loss of buffering capacity, at least in the short term. There is probably cell rigidity and the kidneys are probably no longer reclaiming acidity. The first morning urine pH may be alkaline. Check the urine dipstick for any abnormalities and run a blood chemistry screen and CBC

Baseline	Lemon	1	2	3	4	5
6.0	5.2	6.0	6.0	6.0	6.0	6.0

Gastrointestinal Terrain

▲ Bowel Toxicity Test	↑ Sediment	Alkaline Gastro-test	▲ Urine Calcium
 Bowel toxemia Dysbiosis Hypochlorhydria Maldigestion Malabsorption High protein intake 	Total: • Poor assimilation • Pancreatic insufficiency • Leaky Gut Syndrome Calcium phosphate: • Carbohydrate, sugar and starch maldigestion Uric acid: • Protease deficiency • Hypochlorhydria • Protein maldigestion • Excess protein intake Calcium oxalate: • Fat maldigestion • Lipase deficiency • Poor fat emulsification • Calcium and magnesium deficiency • Malabsorption	 Hypochlorhydria Achlorhydria (>5.0) Use bicarbonate challenge to test acid reserves 	 Excess calcium supplementation Calcium mobilized from bone High refined sugars in diet Hyperparathyroidism Urine calcium Low calcium in body Excess protein intake Malabsorption Hypoparathyroidism

Hormonal Terrain

High Urine Chloride	Low Urine Chloride
Adrenal hypofunctioning	Adrenal hyperfunction
Hypochlorhydria	Electrolyte stress/increased toxicity
Kidney stress	Malabsorption syndrome
Alkaline mineral insufficiency	Diarrhea/excess vomiting
Oxidative stress	

Oxidative Stress Terrain

Low Redox	+2 Oxidative stress	+3 Oxidative stress
 Loss of high energy electron intermediates Low electron potential Susceptible to degenerative diseases Premature tissue aging 	 Liver stress Kidney stress Pancreas stress Blood sugar problems Adrenal stress Lymphatic congestion Fatigue 	 Lymphatic stress Xenotoxins Greatly reduced ATP production Maldigestion Blood sugar dysregulation

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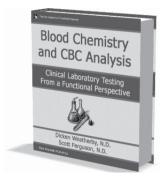
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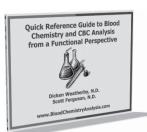
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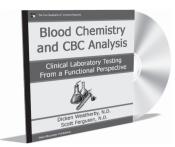
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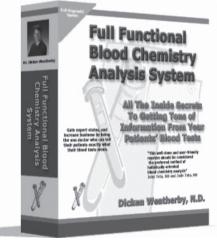
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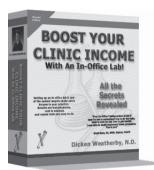
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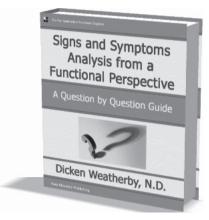


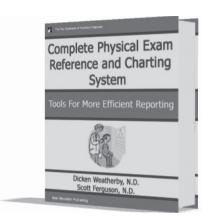
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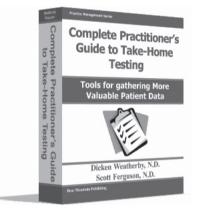
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