

viewed while held up to a light source. This operates on the principle that the refractive index of a urine specimen will vary directly with the total amount of dissolved solids in the sample. This instrument measures the refractive index of the urine as compared with water on a scale that is calibrated directly into the ocular.

Urinary tract infections and bacterial contamination also will alkalinize pH. Medications such as potassium citrate and sodium bicarbonate will reduce urine pH. Alkaline urine is also found in Fanconi syndrome, a congenital generalized aminoaciduria resulting from defective proximal tubular function. This generally occurs after an initial period of anuria because the damaged tubules are unable to concentrate or dilute the glomerular filtrate.

**pH Determinations** of urinary pH must be performed on fresh specimens because of the significant tendency of urine to alkalinize on standing. In alkaline urine, suspended precipitates of amorphous phosphates and carbonates may be responsible for turbidity, whereas in acidic urine, amorphous urates may be the cause.

**Volume** The volume of urine excreted indicates the balance between fluid ingestion and water lost from the lungs, sweat, and intestine. Polyuria is observed in diabetes mellitus and insipidus (in insipidus, as a result of lack of ADH), as well as in chronic renal disease, acromegaly (overproduction of the growth hormone somatotropin), and myxedema (hypothyroid edema). Alkaline urine (pH 7.0) is observed postprandially as a normal reaction to the acidity of gastric HCl dumped into the duodenum and then into the circulation or following ingestion of alkaline food or medications. Acidity in urine (pH 7.0) is primarily caused by phosphates, which are excreted as salts conjugated to  $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{Ca}^{2+}$ , and  $\text{NH}_4^+$ . Pathologic states, in which increased acidity is observed, include systemic acidosis, as seen in diabetes mellitus, and renal tubular acidosis (RTA). Brownish-black after standing is seen in alkaptonuria (a result of excreted homogentisic acid) and in malignant melanoma (in which the precursor melanogen oxidizes in the air to melanin). Urinary tract infections impart a noxious, fecal smell to urine, whereas the urine of diabetics often smells fruity as a result of ketones.

**Low SG** can occur in diabetes insipidus, pyelonephritis, and glomerulonephritis, in which the renal concentrating ability has become dysfunctional. Fixed SG (isosthenuria) around 1.010 is observed in severe renal damage, in which the kidney excretes urine that is iso-osmotic with the plasma. Owing to the  $\text{Na}^+/\text{H}^+$  exchange pump mechanism of the renal tubules, pH ( $\text{H}^+$  concentration) increases as sodium is retained. The characteristic pungent odor of fresh urine is due to volatile aromatic acids, in contrast to the typical ammonia odor of urine that has been allowed to stand. SG varies with the solute load to be excreted (consisting primarily of NaCl and urea), as well as with the urine volume. High SG can be seen in diabetes mellitus, congestive heart failure, dehydration, adrenal insufficiency, liver disease, and nephrosis. Acidity also reflects the excretion of the nonvolatile metabolic acids pyruvate, lactate, and citrate. In RTA, the tubules are unable to excrete excess  $\text{H}^+$  even though the body is in metabolic acidosis, and urinary pH remains around 6.

**Chemical Analyses** Routine urine chemical analysis is rapid and easily performed with commercially available reagent strips or dipsticks. Anuria or oliguria (