I. Lipids: Definition, Classification and Structures:
A. Neutral lipids
   1. Saturated and Unsaturated Fatty Acids
   2. Triglycerides, diglycerides and monoglycerides
   3. Cholesterol (sources of cholesterol) and cholesterol esters
B. Polar lipids (Phospholipids and Sphingolipids)

II. Regulation of triglyceride digestion and storage:
A. Overview of Fat Absorption: In general, fats are not inherently watersoluble and require specific macromolecular complexes that mediate their transport from the intestinal lumen to the blood. The generalized absorption of triglycerides is summarized in Figure 32.8.

B. Bile Salts and Pancreatic Lipase

C. Synthesis, Secretion and Fate of Chylomicrons

Fig. 32.8. Digestion of triglycerols in the intestinal lumen. TG = triacylglycerol; bs = bile salts; FA = fatty acid. 2-MG = 2-monooacylglycerol.
III. Regulation of cholesterol and triglyceride transport by lipoproteins

Figures 56-1 and 34.8 review the process of triglyceride and cholesterol absorption and transport.

(Fig. 56-1) Chylomicron pathway. Dietary triglycerides are hydrolyzed in the intestine by pancreatic lipase to fatty acids (FFA) and monoglycerides (MG), which are reesterified to form triglycerides (TG) in intestinal mucosal cells. Triglycerides are assembled in the ER with other lipids and proteins to form the core of nascent chylomicrons (shaded areas). After completion of assembly in the Golgi apparatus, nascent chylomicrons are secreted into the interstitium of intestinal villi and enter enterocytes. There, and after entry into the blood, the proteins synthesized in the absorptive cells (apo B-48 and the A apoproteins) are augmented by transfer of apo E and C apoproteins from HDL. The first step in chylomicron catabolism takes place in extrahepatic tissues, where most of the triglycerides are rapidly hydrolyzed by lipoprotein lipase to yield chylomicron remnants (see text). The remnants, which retain their component cholesteryl esters, are released from the enzyme and are then taken up by receptors on the surface of hepatocytes that recognize a binding domain on apo E. The remnants are endocytosed and catabolized in lysosomes, from which cholesterol can enter metabolic pathways in hepatocytes, including excretion into the bile. (From Havel RJ: Approach to the patient with hyperlipidemia. Med Clin North Am 66:319, 1982. Used with the permission of the publisher.)

Fig. 34.8. Fate of VLDL. VLDL triacylglycerol (TG) is degraded by LPL, forming IDL. IDL can either be endocytosed by the liver via a receptor-mediated process or further digested, mainly by hepatic triacylglycerol lipase (HTGL) to form LDL. LDL may be endocytosed by receptor-mediated processes in the liver or in peripheral cells, also be oxidized and taken up by "scavenger" macrophages. The scavenger pathway plays a role in ather (see Figs. 34.12 and 34.13). FA = fatty acids; P = inorganic p.
HDL's role in "The Reverse Cholesterol Transport Mechanism"

IV. Regulation of cholesterol uptake by LDL receptors:
A. The LDL Receptor

B. Macrophage Scavenger Receptor: The uptake of oxidized LDL by the macrophage is an initiating step in the genesis of an atherosclerotic plaque.

V. ATHEROSCLEROSIS