Cardiovascular

1. Test tube: turbid specimen
When a tube of turbid plasma is left overnight in a refrigerator at 4°C, chylomicrons float on top of the plasma (form a supranate), since they have very little protein, while VLDL produces an infranate, since it contains more protein and is more dense than chylomicrons. This tube has both a supranate and an infranate (pinkish staining) indicating the presence of both chylomicrons and VLDL. This is type V hyperlipoproteinemia, which is commonly seen in diabetic ketoacidosis. (CV001)
2. Test tubes
The tube on your left is clear, however the LDL fraction is markedly increased. LDL contains CH, which does not produce turbidity. The patient has a type II hyperlipoproteinemia. The tube on the right has a supranate (arrow) and clear infranate indicating the presence of chylomicrons. The most common cause of this is someone who did not fast and had a breakfast full of saturated fats. If this was a hyperlipoproteinemia, it would be a type I hyperlipoproteinemia. (CV002)

3. Patient: Achilles tendon xanthoma
This type of lipid deposit is pathognomonic for familial hypercholesterolemia, which is an AD disease with absence of LDL receptors. (CV003)

4. Patient with xanthelasmas
Note the yellow, plaque-like lesions on the upper and lower eyelids in both eyes. These lesions are associated with hypercholesterolemia - e.g. familial hypercholesterolemia, obstructive jaundice, or hypothyroidism. (CV204)
5. **Vessel: hyaline arteriolosclerosis**
Arteriosclerosis (hardening of the arteries) involves both small and large vessels. Hyaline arteriolosclerosis is a small vessel arteriosclerosis that is commonly found in diabetics and hypertensives. *(CV004)*

6. **Vessel: hyperplastic arteriolosclerosis:** This is the other type of small vessel arteriosclerosis. It is predominantly seen in malignant hypertension and renal disease associated with polyarteritis nodosa and progressive systemic sclerosis. It is also characteristic of the arterioles in the spleen in patients with SLE. Note the "onion skin" appearance of the vessels. This is secondary to hyperplasia of the smooth muscle in the wall of the vessels. *(CV005)*

7. **Aorta: complicated atheromatous plaques**
Note the raised yellow plaques and the fissures in between the plaques. Dystrophic calcification is likely present as well. *(CV205)*

8. **Aorta: aorta with complicated atheromatous plaques**
Note the fissured-appearing endothelial surface and raised plaque-like structures from the surface. Red clot material is adherent to the plaques in multiple areas. These clots consist of platelets held together by fibrin strands. Aspirin would have prevented these clots from forming. The blotchy white areas represent dystrophic calcification. This is an example of advanced
atherosclerosis. It is likely that this patient had an increase in LDL.
(CV006)

9. Patient: dry gangrene of the foot in a diabetic
Note the blackened big toe and little toe. Ulcers are present on the undersurface of the big toe and the metatarsophalangeal joint. These are called pressure ulcers and are primarily due to peripheral neuropathy and inability to feel any pain in areas of trauma. Ischemia and infection also play a role in their development. Coagulation necrosis is primarily operative in this patient. However, anaerobic infection commonly occurs leading to gas production, further ulceration of the foot, a malodorous exudate, and gas in the subcutaneous tissue. In this case, liquefactive necrosis would be the predominant type of infection, hence the term wet gangrene. (CV007)

10. Aorta: atherosclerotic abdominal aortic aneurysm
Note the fusiform dilatation of the aorta below the renal arteries. The lumen is filled with atheromatous debris. Abdominal aortic aneurysms are the most common overall aneurysm. Weakening of the wall by atherosclerosis occurs owing to no vasa vasorum below the renal artery orifices. (CV008)
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11. **Ascending aorta: dissecting aortic aneurysm**
The tear in the aorta is at the top of the screen. A toothpick extends through the opening. Blood dissected proximally and emptied into the pericardial sac. The small toothpick is where the pericardial reflection is located. *(CV208)*

12. **Aortic arch vessels: dissecting aortic aneurysm**
This is a cross-section of two of the elastic arteries coming off the arch of the aorta. Note how the lumens of both vessels are compressed by clotted blood within the wall of the vessels. This is a type A proximal dissection, where blood entered an intimal tear and extended proximally and up into the arch vessels. *(CV009)*

13. **Aorta: dissecting aortic aneurysm**
Note the two lumens in the aorta (double barreled aorta). The lumen on the top is a false lumen (clot has been removed) created by a more proximally located dissection. The false lumen represents the outer media and adventitia of the aorta. The lumen below the false lumen is the true lumen. This is a type B dissection. *(CV010)*
14. Patient with Marfan's syndrome
Marfan's syndrome is an AD disorder with a defect in the synthesis of fibrillin, hence elastic tissue is weakened. Note the longitudinal scar on the patient's chest, most likely representing repair of a previous dissection. The arm span is greater than the height of the patient and the distance from the pelvic brim to the heels is greater than the distance from the brim to the top of the head. This describes eunuchoid proportions. (CV210)

15. Patient with Marfan's syndrome: dislocated lens and high arched palate
Since the suspensory ligament holding the lens in place is composed of elastic tissue and the elastic tissue is weakened in Marfan's syndrome, the lens commonly dislocates. Note the high arched palate, which is also a characteristic feature. Arachnodactyly (not shown) is yet another physical finding. (CV211)
16. Arch of aorta: syphilitic aneurysm
Note the large depression just below the ruler which represents the aneurysm. The aortic valve ring is stretched, which leads to the murmur of aortic regurgitation. The pathogenesis relates to a vasculitis of the vasa vasorum (endarteritis obliterans), which decreases blood flow to the tissue within the aorta. The weakened tissue begins to outpouch in response to the increased stroke volume. *(CV207)*

17. Patient: varicose veins in the lower extremities
Note the bulging and tortuous veins particularly in the left lower leg. There is some discoloration along the medial portion of the ankle, suggesting that the varicosities may be due to deep venous thrombosis and a backup of blood into the superficial saphenous system. *(CV212)*
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<tr>
<td>18. Superficial saphenous veins: primary varicosities involving the superficial saphenous veins</td>
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<tr>
<td>Note the marked tortuosity and dilatation of the vessels. Usually there is absence of the sentinel valve in the femoral vein. (CV014)</td>
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<td>19. Patient: superior vena caval syndrome:</td>
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<td>This patient has a small cell cancer of the lung that has blocked off the superior vena cava. Note the congestion of the head and neck due to the backup of venous blood. (CV015)</td>
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<tr>
<td>20. Patient: lymphedema of the arm post-modified radical mastectomy and radiation</td>
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<tr>
<td>Note the marked enlargement of the extremity. Lymphedema is non-pitting. (CV016)</td>
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21. X-ray study: Takayasu's arteritis
The arrows point to numerous areas of constriction in the subclavian arteries. The patient most likely had weak to absent pulses in both upper extremities. (CV219)

22. Arteriole: fibrinoid necrosis (leukocytoclastic vasculitis):
Note the pink staining material (fibrinoid necrosis) in multifocal areas of the thickened wall of the venule. The material represents protein derived from the plasma that has deposited in the vessel wall owing to an increase in vessel permeability from the inflammatory process. It is called fibrinoid because it looks like fibrin in a clot but it is really protein. Small vessel vasculitis is usually due to immune complex (IC) disease (type III hypersensitivity). ICs are deposited in the vessel wall and then activate the complement system. C5a, a chemotactic factor, attracts neutrophils (only a few are visible at around 7 o'clock). Neutrophils are responsible for the vessel injury. (CV017)
23. Patient's lower legs: palpable purpura
Note the red-blotchy areas scattered randomly over the skin surface. These lesions would be palpable, hence the term palpable purpura. Palpable purpura is due to a small vessel vasculitis with destruction of the vessel leading to an extravasation of blood into the subcutaneous tissue. The inflammation around the vessel (tumor of inflammation) makes the lesion palpable. This patient had Henoch-Schoenleinöös purpura. (CV018)

24. Patient: temporal arteritis
Note the prominent temporal artery. A biopsy of the artery reveals multinucleated giant cells (granulomatous vasculitis, see arrow). (CV019)

25. Patient: thromboangiitis obliterans (Buergeröös disease)
Note the blackened digits (coagulation necrosis). Autoamputation of the digits is likely to occur in this patient. (CV020)
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<tr>
<td><strong>26. Child: swelling of the hand in Kawasaki's disease</strong></td>
<td>The patient had all the findings of this disease including chest pain (coronary artery vasculitis), erythema of mucous membranes and swelling of the dorsum of the hands. Desquamation of skin has not yet occurred. (CV220)</td>
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<td><strong>27. Patient: saddle nose of Wegener's granulomatosis</strong></td>
<td>Note the flattened nose in this patient owing to destruction of the nasal septum by granulomatous inflammation. Congenital syphilis can also produce the saddle nose deformity. (CV021)</td>
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<td><strong>28. Patient: Raynaud's phenomenon</strong></td>
<td>Note the white discoloration of some of the digits owing to spasm of the digital vessels. (CV022)</td>
</tr>
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29. **Vessel: Mucormycosis**
Vasculitis may be secondary to fungi that invade vessel walls. Note the non-septate hyphae and wide-angled branches of Mucor. Candida and Aspergillus can also invade vessels. *(CV023)*

30. **Stomach: hereditary telangiectasia (Osler-Weber-Rendu disease)**
The stomach close-up reveals a vessel dilatation in the submucosa. A histologic section of the lesion exhibits dilated vascular channels. These lesions bleed causing the loss of blood in the stool. Similar lesions occur on the skin and mucous membranes of the mouth. *(CV024)*
### Cardiovascular

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<tr>
<th>Patient</th>
<th>Lesion Description</th>
<th>Image</th>
<th>Notes</th>
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<tbody>
<tr>
<td>Spider angiomata</td>
<td>Note the tentacle-like appearance of this lesion. Compression of the central portion of this lesion causes the tentacles to disappear, hence this lesion is an arteriovenous fistula. They are secondary to hyperestrinism (pregnancy, cirrhosis of the liver).</td>
<td><img src="image" alt="Spider Angioma" /></td>
<td>(CV025)</td>
</tr>
<tr>
<td>Child</td>
<td>Capillary hemangioma on the nose</td>
<td><img src="image" alt="Capillary Hemangioma" /></td>
<td>These benign lesions that present at birth invariably disappear with increasing age in the patient. Most pediatricians recommend that they be left alone. (CV216)</td>
</tr>
<tr>
<td>Patient</td>
<td>Kaposi's sarcoma:</td>
<td><img src="image" alt="Kaposi's Sarcoma" /></td>
<td>Note the raised, elongated red lesion on the neck. This patient is HIV positive and has Kaposi's sarcoma, which is due to HSV-8. Kaposi's sarcoma is an AIDS-defining lesion. (CV027)</td>
</tr>
</tbody>
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| 34. Renal angiogram: left renal artery stenosis  
Note the narrowing of the lumen of the proximal right renal artery. The narrowing is due to an atherosclerotic plaque. (CV028) |
| 35. Renal angiogram: bilateral fibromuscular hyperplasia of the renal arteries  
Note the "beaded" appearance in the right renal artery. This is due to fibromuscular hyperplasia in the wall of the renal artery (narrow lumen) alternating with a normal sized lumen. (CV029) |
| 36. Kidney: benign nephrosclerosis (BNS)  
Note the cobblestoned surface of this kidney in a patient with long-standing essential hypertension. BNS is the kidney of hypertension. The cortical effect is secondary to hyaline arteriolosclerosis (Slide 4) of the arterioles in the cortex with subsequent tubular atrophy and glomerular fibrosis.  
(CV030) |
|---|
| 37. Retina: grade I hypertensive retinopathy  
Note the narrow caliber of the retinal vessels. There is very mild AV nicking (slightly to your left of the red line going down the slide) where the large vessel at the top of the screen crosses a small venule.  
(CV031) |
| 38. Retina: grade III hypertensive retinopathy  
Note the flame hemorrhage (ruptured microaneurysm) directly superior to the optic disc (pale area at 5 o'clock). The white lesions (arrow) are well demarcated and represent hard exudates (increased vessel permeability). There is no papilledema.  
(CV032) |
39. **Retina: grade IV hypertensive retinopathy**
Note the obliteration of the optic disc (papilledema, arrow to center of the slide), flame hemorrhages, soft exudates (white exudates with a fuzzy margin like cotton [cotton wool exudates, arrow at 9 o'clock] representing microinfarctions, hard exudates (white patch that is well demarcated [arrow along the periphery of the slide at 3 o'clock] indicating increased vessel permeability. *(CV033)*

40. **Hearts: normal heart on the left; concentric hypertrophy on the right**
Recall that concentric hypertrophy is due to an increase in afterload (e.g., essential hypertension, aortic stenosis). *(CV034)*
Cardiovascular

41. Heart: right and left ventricular hypertrophy
The left ventricle is on your left and the right ventricle on your right. Note that both ventricles are thickened. One possible explanation for RV hypertrophy is pulmonary artery hypertension, which imposes an increased afterload against the RV. (CV035)

42. Lungs: microscopic of pulmonary edema
Note the pink staining fluid (transudate) filling the alveoli. Pulmonary edema occurs in left heart failure. (CV232)

43. Liver: congestive hepatomegaly ("nutmeg" liver)
Note the red dots scattered throughout the liver parenchyma. These represent blood backed up into the terminal hepatic venules (central vein) in a patient with right heart failure. Recall the terminal hepatic venules eventually coalesce to form the hepatic vein, which empties into the vena cava, which empties into the right heart. Hence an increase in systemic venous pressure transmits back into the liver. (CV042)
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| **44. Aorta: postductal coarctation of the aorta**  
Note the area of constriction just distal to the ligamentum arteriosum (arrow) extending off the transected pulmonary artery. The proximal aorta is dilated and the posterior wall of the aorta hit by the jet-stream of blood going through the narrow opening is also aneurysmally dilated. You can trace a figure 3 lying on its side. |   |
|   | (CV043) |
| **45. Children: cyanotic congenital heart disease**  
Note the blue discoloration of the lips in these two children, both of which have tetralogy of Fallot. Refer to course notes for why these patients squat to relieve cyanosis. |   |
|   | (CV044) |
| **46. Right coronary artery angiogram**  
RCA stenosis secondary to atherosclerosis. Note the area of constriction (arrow) in the mid-portion of the coronary artery. |   |
|   | (CV045) |
47. Heart: heart with coronary artery bypasses using saphenous vein grafts
Note the 2 veins, one bypassing the LAD coronary artery (arrow) and the other the RCA. (CV046)

48. Coronary artery: coronary artery atherosclerosis and thrombus occluding the lumen
Note the red thrombus overlying an atheromatous plaque (arrow). The slit like spaces are where cholesterol used to be present. (CV047)

49. Heart: anterior myocardial infarction
Note the pale infarct in the anterior portion of the left ventricle (on your left) with extension into the anterior 2/3rds of the interventricular septum. This is the distribution of the left anterior descending coronary artery. (CV240)

50. Heart: coagulation necrosis of cardiac muscle in an AMI
Note the eosinophilic staining cells with no cross striations or nuclei. (CV049)
Cardiovascular

51. Heart: microscopic section of a 3 day old acute myocardial infarction
Unlike the previous slide, which showed the early stages of coagulation necrosis, this slide depicts a heavy neutrophilic infiltrate with destruction of the cardiac fibers. The neutrophils came into the area of the infarct from the periphery, so this should not be confused with liquefactive necrosis. Macrophages will eventually replace the neutrophils and then collagen tissue will be deposited in the area of infarction. (CV241)

52. Heart: cardiac tamponade secondary to rupture of the left ventricle in a patient with an AMI
At the apex of the heart, there is a rupture site in the area of a transmural AMI. Blood fills the pericardial sac and encircles the heart. (CV050)

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| 53. | Heart: rupture of the anterior wall of the left ventricle in a patient with an acute myocardial infarction  
     | The rupture site is at 10 o’clock in the slide. The pericardial sac is filled with blood. (CV255) |
| 54. | Heart: posteromedial papillary muscle rupture in a patient with a right coronary artery thrombosis  
     | A portion of papillary muscle hangs from the chordae tendineae of the mitral valve. A biopsy of the muscle shows coagulation necrosis. Recall that the right coronary artery supplies this muscle. (CV254) |
| 55. | Heart: rupture of the interventricular septum in a patient with an acute myocardial infarction  
<pre><code> | A plastic stick marks the site of rupture in the IVS. Blood flows from left to right and volume overloads the right heart leading to right heart failure. The majority of IVS ruptures are due to thrombosis of the LAD coronary artery. (CV262) |
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<th><strong>56. Heart: fibrinous pericarditis post anterior acute myocardial infarction</strong></th>
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<td>Note the shaggy exudate along the surface of the visceral pericardium. This may be a complication of a transmural infarction during the first week or an autoimmune pericarditis 6-8 weeks later with autoantibodies directed against pericardial tissue. This is called Dressler's syndrome. <em>(CV070)</em></td>
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<th><strong>57. Heart: mural thrombus on anterior wall of the ventricle</strong></th>
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<td>Note the large thrombus occupying most of the lumen of the left ventricle. A pale infarct overlies the thrombus. It is a mixed clot with the part attached to the endocardium representing a platelet thrombus and the part projecting into the lumen a &quot;venous&quot; type clot. <em>(CV261)</em></td>
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<th><strong>58. Heart: ventricular aneurysm</strong></th>
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<tr>
<td>Note the thin aneurysmal sac extending from the anterior portion of the left ventricle. Very little functional muscle tissue is left in the ventricle. These are late complications and present with a precordial bulge of the anterior chest with each systolic contraction. Rupture is uncommon, since the tissue is scar tissue. Most patients die of heart failure. <em>(CV245)</em></td>
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59. Heart: scar tissue in an old anterior AMI
Note the completely calcified LAD coronary artery buried in the epicardial fat. The white patchy area in the anterior wall is fibrous tissue. (CV053)

60. Heart: sterile vegetations on the mitral valve in a patient with acute rheumatic fever
Note the red, wart-like vegetations along the line of closure of the valve (arrow). The vegetations are examples of fibrinoid necrosis. (CV247)
## Cardiovascular

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<tr>
<td><strong>61. Heart: Aschoff nodule</strong>&lt;br&gt;The Aschoff body is the pathognomonic lesion of rheumatic fever. It consists of a collection of Anichkov's myocytes (reactive histiocytes; in the center of the slide) and Aschoff multinucleated giant cells (a few multinucleated cells are visible) within an area of fibrinoid necrosis (pink staining material surrounding the histiocytes) located in the myocardium. <strong>(CV055)</strong></td>
<td><img src="CV055.jpg" alt="Image of Aschoff nodule" /></td>
</tr>
<tr>
<td><strong>62. Patient: erythema marginatum in a patient with acute rheumatic fever</strong>&lt;br&gt;This skin rash is one of the five Jone's criteria for diagnosing acute rheumatic fever. Note the erythematous circular lesions with central areas of pallor. <strong>(CV248)</strong></td>
<td><img src="CV248.jpg" alt="Image of erythema marginatum" /></td>
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63. Heart: severe mitral stenosis with a dilated left atrium containing a thrombus
Note the fixed, stenotic opening of the mitral valve (arrow in low center). The chordae are thickened owing to scar tissue formation. Stasis in the enlarged left atrium predisposes to clot formation (arrow in top center). LA dilatation predisposes to atrial fibrillation, which is responsible for breaking the clot up into small emboli. (CV056)

64. Heart: mitral stenosis
You are looking down from the enlarged left atrium into the orifice of the mitral valve. Note that it has a fish-mouth appearance. This patient probably had a significant component of mitral insufficiency as well as stenosis. (CV057)

65. Heart: mitral valve prolapse
Note the baggy-appearing mitral valve leaflets. When blood collects beneath these voluminous valves, it projects them into the left atrium like a parachute. When the chordae abruptly stop the valves from moving any further, a systolic click is heard followed by a murmur of mitral insufficiency. (CV058)
66. Heart: mitral valve prolapse
Note the parachute-like redundancy of the mitral valve and how it has prolapsed into the left atrium. A close-up of the valve shows that one of the chordae has ruptured causing the patient to develop acute mitral regurgitation, and, in this case, death. (CV249)

67. Aortic valve: bicuspid aortic valve with associated aortic stenosis
Note that there are only 2 cusps when there should be 3. The AV orifice is stenotic due to dystrophic calcification of the valve leaflets (knobby appearing material). (CV060)

68. Heart: aortic stenosis with left ventricular hypertrophy
The left atrium is at the bottom of the screen and the left ventricle lumen is markedly diminished owing to LVH. The aortic valve just above the left atrium is markedly calcified. (CV250)
69. Heart: aortic insufficiency secondary to old rheumatic heart disease
Note that the aortic valve is fibrotic and plastered against the aorta, hence leaving an expanded AV orifice (blood will leak back into the LV during diastole). As expected, the LV was volume overloaded and exhibits both dilatation and hypertrophy. (CV061)

70. Aortic valve: infective endocarditis complicating a ventricular septal defect
A metal probe exits one of the two holes in the membranous portion of the IVS. This represents a VSD. Small vegetations line the valve cusps and the thread extends through a hole in the valve that developed from the infection. The edge of the other valve cusp looks half eaten away. This underscores the increased incidence of infective endocarditis in patients with an underlying congenital defect. (CV264)
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71. Mitral Valve: acute infective endocarditis
The vegetations are on the atrial side of the valve leaflets. Note the bulky vegetations and chordae tendineae that has been ruptured. *Staphylococcus aureus* grew out in the blood culture. (CV259)

72. Patient: splinter hemorrhages on your left and Osler's nodes on your right
This patient has acute infective endocarditis. Recall that these lesions are an example of immunocomplex vasculitis (type III hypersensitivity). (CV064)

73. Retina: Roth's spot in a patient with acute infective endocarditis
Note the red lesion with a pale center (arrow). This represents an immunocomplex vasculitis with hemorrhage. (CV065)
## 74. Heart: Libman-Sack's endocarditis involving the mitral valve in a patient with SLE

The warty vegetations located all over the valve and portions of the chordae are sterile, just like those in acute rheumatic fever. Both are examples of immunologic damage leading to immune deposits in the valve and fibrinoid necrosis. Pericarditis, rather than endocarditis, is the most common cardiac lesion in patients with SLE. *(CV257)*

## 75. Heart: congestive cardiomyopathy

Note the extreme dilatation of both the left (on your right) and right ventricular chambers (on your left). The patient had a previous history of a coxsackie myocarditis. *(CV066)*

## 76. Heart: hypertrophic cardiomyopathy

Note the asymmetric thickening of the interventricular septum when compared to the thickened free LV wall. The anterior leaflet of the MV is stretched tautly and almost touches the bulging interventricular septal muscle. Note the very narrow orifice created by these two closely approximated structures. When blood passes through this narrow opening, the negative pressure behind it sucks the anterior leaflet against the IVS muscle and obstructs blood flow. *(CV067)*

## 77. Heart: restrictive cardiomyopathy secondary to amyloidosis

Note the thickened myocardial tissue and the blotchy-white deposits of amyloid in the muscle. The special stain on the right demonstrates the blue-green staining amyloid encasing and destroying the myocardial fibers. *(CV068)*
78. Heart: left atrial myxoma
Note the huge ball-like structure attached to the wall of the left atrium. One can easily picture it dropping on top of the MV orifice and obstructing blood flow (syncope occurs) or pieces of the tumor embolizing into the systemic circulation. (CV069)

79. Heart: fibrinous pericarditis
This is a classic example of fibrinous inflammation involving the visceral pericardium ("bread and butter" pericarditis). Fibrinous inflammation is due to increased vessel permeability leading to the leakage of a sterile exudate with fibrin on to the surface of the heart. The patient would likely have had a pericardial friction rub. This particular patient had SLE. (CV070)

80. Chest x-ray: pericardial effusion
Note the "water bottle" configuration of the heart owing to a collection of fluid in the pericardial sac. The patient had a coxsackie myocarditis and pericarditis. (CV071)

81. Heart and Pericardium: constrictive pericarditis secondary to TB
Note the caseous necrosis in the hilar lymph nodes directly above the heart. The pericardium is markedly thickened and has chalky white area most likely representing dystrophic calcification. Note what little room the heart has to expand with blood and why a pericardial knock would likely be heard when the ventricle hit the thickened parietal pericardium during diastole. (CV072)