A Comprehensive Review of Vascular Disease: Part 1 - Pathophysiology and Early Detection

Abstract
Evidence shows an association between oral disease and systemic vascular disease. Physicians need our dental colleagues’ help if we strive to optimally reduce our patients’ risk of suffering a heart attack or stroke. This four-part series will give dental professionals an understanding of the pathology of cardiovascular disease and describe how you can intervene to reduce risk in your personal life and your patients’ lives. Incorporating a cardiovascular health program in your practice will elevate your credibility as a true health professional, improve your ability to cure dental disease, and drive the much-needed collaboration between physicians and dentists. Part 1 of the series describes the epidemiology of cardiovascular disease and the anatomy, physiology, and pathology of plaque formation. You will also learn the best ways to detect disease at its earliest, most treatable stage.

Learning Objectives:
At the conclusion of this course the attendees will be able to understand:
1. Epidemiology of vascular disease
2. Anatomy and physiology of plaque development
3. Testing available to detect early disease

Author Profile
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In the United States, a heart attack occurs every 25 seconds and a stroke every 40 seconds. In about 25% of people who suffer sudden death from a heart attack, sudden death is their first symptom, usually caused by an arrhythmia.

Advancing medical science has significantly improved the treatment of known vascular disease, reducing mortality and allowing people to live longer following an event. However, we are ineffectively preventing the first event. The incidence of heart attacks was estimated to be 1.5 million in 1986. That only modestly reduced to 1.2 million by 2006.¹

The CDC reports that heart disease and stroke are the #1 and #3 causes of death in the United States, accounting for 31% of all deaths.² A 2010 United Nations report only ranks the United States as 36th in life expectancy.³ Life expectancy in the United States is 78 years, equivalent to citizens of Cuba, Chile, and Czechoslovakia. We would have a longer life expectancy if we lived in Slovenia, Portugal, Malta, or Iceland.⁴

If we are to improve, pathology must be detected earlier in the course of disease development, before an event occurs or a procedure is needed to open obstructed arteries. Necessary lifestyle changes, medications, and supplements can then be implemented to heal and stabilize existing plaque to halt and even reverse the disease process.

Dentists are well positioned to participate in the war against heart attacks and strokes. Many middle-aged Americans rarely visit their Physician, but regularly see their Dentist. By understanding the pathophysiology of vascular disease and red flags for disease, Dentists can identify patients at risk, intelligently discuss needed screenings, and even do some of the testing and health promotion in their office.

Dentists and physicians need to collaborate. Sleep apnea causes heart attacks and Dentists will have a hard time optimally treating their sleep apnea patients without weight loss. Periodontal breakdown may not heal until insulin resistance is identified and treated. Physicians often can’t optimally improve vascular inflammation without identifying and treating existing high risk oral bacteria.

All Health Professionals who care about their patients need to be involved in promoting total health. It’s about the person, not the body part! During this 4 part series you will learn the pathophysiology of vascular disease, state-of-the-art testing available to detect early treatable disease, direct causes of disease, root causes of disease, genetic testing available, and most importantly, what dentists can do to help stabilize or reverse disease in your patients, and perhaps yourself.

Anatomy of Disease- making of a volcano
The heart is a muscle. It receives oxygenated blood from the lungs and delivers it to be used for cellular respiration at the tissue level. After the oxygen is extracted by a tissue, veins transport the blood back to the heart to be reoxygenated.

It is estimated that there are about 7000 miles of arteries in the human body ranging in size from the aorta to tiny arterioles that feed capillary beds. The coronary arteries exit immediately after oxygenated blood leaves the heart. Coronary arteries supply oxygen to the heart’s myocardium.

Figure 1 depicts the anatomy of an artery. The section at the far left is a normal arterial wall. Think of it as a garden hose. Its anatomy includes:
1. Endothelium- the endothelium is the thin layer of cells that separates flowing blood from the rest of the wall.
2. Intima layer- The intima layer within the arterial wall is composed primarily of smooth muscle cells.
3. Outer capsule

![Figure 1. Anatomy of an artery](https://example.com/image-url)
Within the blood, red blood cells carry oxygen and white blood cells modulate inflammatory reactions intended to fight pathologic processes such as infection, toxic exposures, and malignant transformation of cells. Cholesterol products, lipids, are delivered to tissues for cell membrane production. A normal arterial wall does not accumulate a significant amount of lipids. The endothelial lining is intended to block the flow of lipids from the blood into the wall.

**Arterial plaque forms from the buildup of lipids within the intima layer of the arterial wall.** Plaque does not simply stick to the inner surface of the endothelial lining, it actually penetrates the endothelial barrier to accumulate within the arterial wall. That is why a cardiologist is not able to just “scrape out” the plaque.

There is actually a bidirectional flow of lipid particles across the endothelial lining. Think of it as a military battle. Enemy troops (pathogenic lipids) are trying to work their way behind enemy lines (the endothelial barrier) in an effort to kill their target, us! There are also friendly troops (High Density Lipoproteins, HDL) that are trying to protect the military theater. They bind pathogenic lipids and transport these enemy troops out of the wall, back into the bloodstream where they are washed away. This elimination of pathogenic lipids from the wall of the artery by HDL particles is called reverse cholesterol transport.

In a healthy artery, the endothelium is impervious to the influx of pathogenic lipids. Any excess is effectively eliminated by reverse cholesterol transport. Unfortunately, in most Americans, the enemy troops are winning the war, leading to a slow buildup of lipids within the intima layer of our arteries.

Inflammation and endothelial dysfunction drive the pathologic flow of lipids into the arterial wall. Inflammation is actually a normal process designed to eliminate invading organisms and toxins, heal damaged tissue, and repair mutated cells that could lead to cancer. Inflammation is necessary for survival. When a developing problem is recognized by our immune system, macrophages migrate to the injured site and release cytokines. Cytokines are immune system chemicals that kill the invading infection, repair the injured tissue, and eliminate a cancerous cell. Cytokines do so by producing inflammation.

The problem in vascular disease is that the inflammatory processes are never allowed to turn off, which leads to chronic inflammation. Chronic inflammation causes an increased flow of pathogenic lipids across the endothelium, and leaves it fragile, prone to rupture.

Early plaque formation grows deep into the arterial wall, like an iceberg (figure 1, panels 2-4). Eventually, the endothelial lining weakens and small volcanoes begin to bubble up from the surface of the artery. These volcanoes will continue to grow into large plaques that eventually block the flow of blood within the artery as shown on the far right of the diagram.

There are two pathologic outcomes that result from this disease process. In the first, a slow steady build of enemy troops in the arterial wall continues until we can no longer deliver adequate oxygen past the obstruction to adequately supply the distal tissues. At this point, symptoms will begin. Symptoms usually begin during activity since exercising muscles require more oxygen.

Symptoms experienced depend on the location of the diseased artery. If the obstruction occurs in the artery of our legs as occurs with peripheral artery disease, we get cramping in our calves called claudication. If the blocked artery is in our heart we get a symptom called angina, typically experienced as chest pressure, left arm discomfort, nausea, and breaking out in a cold sweat. Think of this as the Bill Clinton version of heart disease. President Clinton began to experience symptoms of angina. Testing showed that three arteries in his heart were 90% obstructed, and bypass surgery was required.

The other pathologic outcome that occurs within the arterial wall is much more dangerous. It’s called plaque rupture as is shown in panel 5 of figure 1. White blood cells from the circulating blood penetrate the arterial wall. Oxidation occurs within the plaque in the arterial wall, which activates the inflammatory cascade to soften the plaque. The reactions weaken the endothelial barrier leaving it prone to crack. The lining may break open, causing the contents from within the plaque to erupt like a volcano into the bloodstream.

This rupture activates the clotting cascade. If the event occurs in a high pressure artery like the carotid artery in our neck, the clot breaks loose, flows to the brain, and causes a stroke. If rupture occurs in a low pressure vessel like the coronary arteries in our heart, the clot continues to grow until it completely blocks blood flow through the artery. If the clot is not recanalized to allow oxygenation of distal tissue, all tissue beyond the event will die. This is a heart attack.

Asymptomatic events can occur in the tiny arteries of our kidneys and brain. These events lead to chronic conditions such as chronic kidney disease and “multi infarct” dementia. Multi-infarct dementia is the second leading cause of dementia, accounting for up to 20% of the cases. It is second to Alzheimer’s dementia.

Rupture can occur in very small plaques. It is estimated that up to 85% of coronary plaque ruptures that cause heart attacks occur in plaques not large enough to make a stress test abnormal. Think of this as the Tim Russert type of vascular disease. TV anchorman Tim Russert had a normal stress test April 2008 and died of a heart attack two months later. He suffered a plaque rupture in a small plaque not picked up on the stress test. Sudden death occurs when the event occurs in a coronary artery that supplies the electrical center of the heart. It’s like tripping a circuit breaker in your house. All electrical activity ceases when this artery thromboses.
Early detection of vascular disease
Discovered early, vascular disease can be stabilized and even reversed. Vascular risk factors like blood pressure, cholesterol, smoking, etc. simply predict the likelihood a person has disease. More important is to determine whether disease actually exists, and if so, how bad it is. Knowledge is power. An individual who discovers they have disease often has a stronger motivation to make the changes necessary to improve their health.

Discovered plaque needs to be protected. Even a very small plaque can rupture to cause a heart attack, stroke, or transient ischemic attack (TIA). Ruptures within tiny arteries in the brain may not cause symptoms, but when they occur hundreds or thousands of times, it can lead to vascular dementia. On a brain MRI the radiologist will sometimes make a side comment of, “volume loss due to chronic ischemic changes.” Translated, that says: “volume loss” = death of brain. “Chronic ischemic changes” = arterial disease. The brain is dying from multiple small imperceptible events! Frequently, these patients have no history of heart attack, stroke, or other obvious vascular event.

We don’t want to wait until there is death of brain or some other event occurs to start treating existing disease. Early detection of disease allows aggressive treatment with lifestyle changes, medications, and supplements to halt, or even reverse the process. Most importantly, we want to prevent a major event.

Sometimes evidence of disease is found in testing already done. A chest x-ray report may comment, “coronary calcifications present” or “calcifications in the aortic arch.” Calcifications are not seen in healthy vessels, they are only seen along with arterial plaque. Similarly, vascular calcifications may be seen in CT scans, Panorex, or mammograms.

Historically, it has been difficult to identify disease early, and impossible to track whether therapeutic interventions have been effective. However, advancing technology has changed that. The following is a review of cardiovascular testing currently available.

EKG
Contrary to popular belief, an electrocardiogram is NOT a screening test for vascular disease. All an EKG does is measure the flow of electricity through your heart. If the flow of electricity is disrupted because of death of heart muscle as occurs in a heart attack, changes will be seen on the EKG. That is how we often find “silent heart attacks.”

Occasionally we will find evidence of cardiomyopathy (heart muscle thickening), electrical disturbances, electrolyte abnormalities, and drug toxicities, but that is the exception rather than the rule.

A routine EKG is most valuable as a baseline study. The flow of electricity can vary between individuals because of anatomic variations. Having it as a comparison when they present with symptoms can be invaluable.

Stress test
Stress tests have historically been the standard of care in screening for vascular disease. Unfortunately, they still are. As many as 70-86 percent of heart attacks occur in people who would have had a normal stress test as occurred with Tim Russert. Why?

The heart is a large muscle whose job is to receive oxygenated blood from the lungs, then contract to deliver oxygenated blood to distant tissue. Just like every muscle, the heart needs oxygen. The coronary arteries branch off just as the aorta exits the heart. Blood flows through these coronary arteries to supply oxygenated blood to the heart muscle.

When a slow buildup of plaque reaches a critical size, the obstruction prevents an adequate supply of oxygen. The person begins to experience symptoms of angina, which include pressure in their chest, shortness of breath, pain in their left arm, diaphoresis (cold sweat), and nausea. This is what happened to Bill Clinton. Symptoms usually first occur with exertion because of the higher oxygen demand.

A stress test is an extension of this concept. It is a functional study, not an anatomical study. A plaque must block at least 60-70 percent of the blood flow before a stress test becomes abnormal.

There are two components to every stress test. The first is exercise to force an increased oxygen requirement. The second is to watch for changes that suggest poor blood flow through the coronary arteries.

The most common method to increase the heart’s oxygen requirement is to follow one of several protocols that use a treadmill. The patient starts by walking slowly on the treadmill with minimal elevation. Every 2-3 minutes the speed and tilt are increased slightly to force a greater workload. The patient’s heart rate, rhythm, and EKG are monitored throughout the test. The workload continues to increase until the patient fatigues, or reaches a target heart rate to qualify as an adequate test. This is called an exercise stress test.

If a person has physical limitations that do not allow them to walk on a treadmill, a pharmacologic stress test is performed. A medication, either Adenosine or Dobutamine are infused through an IV to mimic the effect of exercise.

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used. A second scan is performed shortly after the treadmill
is finished to measure uptake of the tracer in the heart. Poor
tracer flow suggests inadequate oxygen supply.

The use of the stress echocardiogram and nuclear stress
test has significantly improved the sensitivity of picking up
coronary artery blockages. However, our heart only needs
about 60% of normal blood flow to function normally, so any
smaller blockage will not be detected.

Therefore, stress tests are an inadequate method of find-
ing arterial disease at a reasonably early stage. They are very
helpful in evaluating a patient who is having atypical chest
pain that we don’t think is cardiac in origin. A normal test
in that circumstance confirms that their symptoms are from
a different source. It is also a good tool to evaluate arrhyth-
mas, estimate exercise capacity, and to screen a patient
who is sedentary, but wants to initiate a vigorous exercise
program.

Carotid artery Doppler
Carotid Doppler testing has been used for decades. It is one
component of Lifeline screening frequently available in the
community. A small ultrasound probe is placed on your neck
and the flow of blood through the carotid artery is observed.

As blood flows past a significant blockage, it speeds up,
similar to water flowing from a garden hose as you place your
thumb over the opening. This change in speed of blood flow
is observed as color changes on the ultrasound monitor sug-
gesting a partial blockage in that section of the artery. The
size can be estimated based on the speed of flow as indicated
by the color.

Like a stress test, this is a functional study looking for
flow obstruction. It does not look at the specific anatomy of
the arterial wall.

Coronary artery calcium score (CACS)
In the 1990s a new technology was developed to detect ar-
terial disease at a much earlier stage. It was called Electron
Beam Computed Tomography (EBCT). It was able to take
very rapid pictures of the heart through the use of a CT scan.
Like a fast shutter speed on a camera, it was able to take stop
action photos of the heart. CT scans are able to detect cal-
cium within tissue. Normal coronary arteries do not contain
any calcium, but diseased ones often do. EBCT was able to
measure the total amount of calcium contained within the
arteries of the heart and provide a score. A higher score sug-
gested more severe disease, but any disease is significant.

Now most radiology facilities are utilizing ultrafast CT
scanners, not EBCT, to do their coronary artery calcium
testing. Technology has improved and allows for 64 pictures
per second, so the quality of image rivals that of EBCT.
Coronary calcium score by ultrafast CT scan is typically
more readily available and is usually less expensive.

Although CACS is a significant improvement to screen
for early vascular disease, it still has its limitations. A plaque
must be calcified to be detected by CACS. Early plaque and
soft plaque, the most dangerous type, are not typically calci-
{fied. CACS will miss them.

Carotid Intima-Media Thickness ultrasound
(CIMT)
CIMT ultrasound is the best method to detect early dis-
{ease. It is much different than carotid Doppler ultrasound
screening described above. CIMT uses modern B-mode
ultrasound technology together with advanced software
capability to measure the thickness of arterial walls. It
actually looks at the anatomy of the arterial wall.

An ultrasound probe is placed on your neck and many
pictures are taken of the carotid arteries. It is inexpensive
and many insurance companies, including Medicare, cover
its use.

A task force of the American College of Cardiology/Amer-
ocan Heart Association presented a report in Novem-er 2010 reviewing 25 preventive cardiology tests. They
gave CIMT ultrasound a very favorable Class IIa recom-
{mendation based on level B evidence. It was one of their
most highly recommended screenings. Their only caution
was, “Published recommendations on required equipment,
technical approach, and operator training and experience
for performance of the test must be carefully followed to
achieve high-quality results.”

Their caution is completely valid. Although CIMT ultrasound has been used in research for about 15 years,
it has only recently been available to the public. National
standards ensuring adequate equipment, technician cer-
{tification, and quality assurance of results have not been
established.

Often, Physicians purchase equipment and perform
testing in their office. Another option is thru a company
that specializes in CIMT ultrasound. In either case, quality
assurance is critical.

There are 2 measurements taken during analysis. The
first is the mean arterial thickness. The distance between
the outer edge of the intima, and the inner edge of the
media is measured. An average of multiple images at the
posterior wall of the common carotid artery is calculated
to give the mean thickness. This intima-media thickness is
approximately 0.50 mm in a disease free artery. As enemy
troops penetrate the lining and build up within the intima,
the artery wall begins to thicken. There is a normal rate that
the carotid artery wall thickens. However, this process is
anything but normal, it is developing disease!

The mean thickness is a very precise and consistent
measurement to within 0.05mm accuracy. It is quantita-
tive and trackable. The test can be a repeated over time to
look for worsening or improvement. CIMT is the tech-
ology used in many studies over the past 15 years which
have shown that plaque regression occurs with adequate
treatment.
Figure 2 shows a graph the median thickness progression from 16 to 85 years old. The pink curve is women and blue is men. A person’s vascular age is the age at which their thickness falls on the curve. A person with a mean arterial thickness of 0.81 mm has a three-fold increased risk of stroke. Therefore, in a 67 year old man and a 74 year old woman, it’s normal to be abnormal!

The heart attack risk is similar to stroke risk, suggesting that the presence of carotid artery disease predicts the existence of coronary artery disease. This is not surprising since the disease process will likely be similar regardless of where the artery is located.

A vascular age more than five years greater than the actual age is considered advanced plaque deposition. Plaque is accumulating faster than it should. The causes should be identified and treated.

Lastly, a mean thickness measurement is also very helpful to assess people with known disease, including those who...
have undergone stents or bypass surgery. Some of the best mean arterial thickness measurements are in people who have had a heart attack, stroke, or undergone a cardiac procedure. It shows that their lifestyle changes, medication, and supplements are working!

CIMT ultrasound also measures individual plaques, the volcanoes bubbling up from the surface of your arterial wall. Once a volcano of plaque forms, the endpoints of measurement get fuzzy, so this is a qualitative measurement not a quantitative measurement as mean thickness is. We cannot compare measurements between tests looking for improvement.

However, it can be observed whether the plaque is soft, heterogeneous, or calcified. Soft plaque is very dangerous plaque. Think of soft plaque like a blister ready to burst. Its liquefied center is often inflamed. An actively inflamed plaque that cools off and is given a chance to heal becomes fibrous and often calcified. Calcified plaque is the least dangerous type of plaque. It is like an aged blister that has wrinkled and become hard. Heterogeneous plaque is between these two.

An individual plaque larger than 1.9 mm seen on CIMT ultrasound would never be seen on carotid Doppler ultrasound. However, there is up to a 50% increased risk of event. A plaque this size needs to be protected from rupture by extinguishing any inflammation, and optimally controlling all other vascular risk factors.

CIMT ultrasound is able to identify disease earlier than coronary artery calcium score. A study published in the Mayo Clinic Proceedings in 2009 performed CIMT ultrasound on 36-59 year olds with one traditional cardiovascular risk factor but no known disease. 34% were found to have plaque. CACS were also performed in those tested and half with an abnormal CIMT ultrasound had a calcium score of zero. Their plaque was not calcified.

An approach to discover disease
What is a reasonable approach to determine whether a person is at risk of suffering an event, or has advanced plaque deposition?

Start by reviewing the results of previous testing performed like a Panorex, a chest x-ray, CT scans, mammogram, lifeline screening, and cardiac catheterization. If plaque is seen on one of these, the search is over. Arteries are diseased and need to be protected.

The next step is to have CIMT ultrasound performed. CIMT ultrasound may be done even if a patient is known to have existing disease. The mean thickness measurement will tell whether current risk factor treatment is working.

If plaque is seen on CIMT ultrasound, the search is over. If no plaque is seen on CIMT, a coronary artery calcium score should be done to look for evidence of disease in the heart. If no plaque is seen on any of the above tests and their mean arterial thickness is favorable, congratulations! It is time for that person to create optimal health in their life to prevent plaque from ever forming.

What processes allow the flow of lipid particles behind enemy lines to cause disease? There is not one simple answer to that very important question. It is not just about cholesterol level and blood pressure. There are many other contributors to the disease process, all of which must be assessed if developing disease is to be reversed, or at least stabilized.

The next course will begin to answer this question regarding the origin of disease. It will describe the causes of disease that directly attack the arterial wall, including dyslipidemia, hypertension, vascular inflammation, endothelial dysfunction, and oxidative stress.

References

2. Center for Disease Control – Causes of Death Natural Vital Statistics report, volume 58 number 19, 5-20-2010

Author Profile

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Questions

1. Which of the following countries has a longer life expectancy than the United States?
   a. Slovenia
   b. Malta
   c. Portugal.
   d. Iceland
   e. All of the above

2. Which of the following is NOT currently a component of optimal cardiovascular assessment and treatment?
   a. Identify and treat sleep apnea.
   b. Identify and treat sinusitis.
   c. Identify and treat high risk oral bacteria.
   d. Assess and individual’s genetic makeup.

3. Which of the following statements is true about reverse cholesterol (RCT) transport?
   a. It is the process of cholesterol deposition in arterial walls.
   b. It is driven by LDL cholesterol.
   c. High levels of Myeloperoxidase (MPO) amplify RCT.
   d. It is the process of removal of cholesterol buildup in arterial walls.

4. Arterial plaque
   a. Sticks to the lining of arteries
   b. Accumulates within the endothelial lining
   c. Collects within the intima
   d. Collect between the intima and outer capsule
   e. All of the above

5. Reverse cholesterol transport
   a. Is the process by which lipids enter the arterial wall.
   b. Is accomplished by HDL cholesterol.
   c. Accelerates with endothelial dysfunction.
   d. Accelerates with inflammation.
   e. All of the above

6. Acute Inflammation
   a. Is a process where macrophages release cytokines.
   b. Is a normal physiologic process.
   c. Leaves vulnerable plaque prone to rupture.
   d. Is used to kill and invading infection, heal damaged tissue, and repair mutated cells.
   e. All of the above

7. All of the following are potential outcomes from systemic vascular disease EXCEPT
   a. Dementia
   b. Renal disease
   c. Multiple sclerosis
   d. Heart attack
   e. Stroke

8. All of the following are true statements about arterial plaque rupture EXCEPT
   a. Most events occur in non-obstructing plaque.
   b. Sudden death may occur from plaque rupture in a coronary artery.
   c. Small asymptomatic events in the brain are the second leading cause of dementia.
   d. Most people who suffer plaque rupture have warning signs prior to the event.
   e. All statements are true

9. Which of the following is NOT a potential resource for discovering vascular disease
   a. EKG
   b. Mammogram
   c. Panorex.
   d. Chest x-ray.
   e. All are potential resources

10. Which of the following screening tests can detect abnormalities before blood flow is obstructed?
    a. CIMT ultrasound
    b. Coronary artery calcium score
    c. Carotid Doppler screening
    d. Both A and B
    e. All of the above

11. Which of the following is NOT a reason to detect vascular disease at an early stage?
    a. Early detection motivates patients to make necessary lifestyle changes.
    b. Early detection helps a physician determine how aggressively to treat risk factors.
    c. Existing plaques can be protected from plaque rupture.
    d. Early disease may be reversed.
    e. All are reasons for early detection

12. An electrocardiogram
    a. Is a valuable tool to screen for vascular disease.
    b. Cannot detect existing vascular disease.
    c. May detect cardiomyopathy.
    d. May detect electrolyte imbalance.
    e. All of the above

13. Which of the following statements is true about stress tests?
    a. Stress tests usually detect asymptomatic vascular disease.
    b. The patient must be able to walk on a treadmill to be tested.
    c. In a pharmacologic stress test, amiodarone is used to mimic exercise.
    d. In a nuclear stress test, the patient is exposed to ionizing radiation for a C.T. scan.
    e. A stress echocardiogram is more sensitive to pick up disease than a regular stress test.

14. Valuable uses of a cardiac stress test include all of the following EXCEPT
    a. Detect early vascular disease.
    b. Evaluate atypical chest pain.
    c. Estimate exercise capacity.
    d. Evaluate certain arrhythmias.
    e. Screen a patient prior to beginning an exercise program.

15. Which of the following is NOT just flow study that only detects plaques that are obstructing flow, so have a high false negative rate in finding early vascular disease?
    a. Stress echocardiogram
    b. Nuclear stress test
    c. Carotid Doppler ultrasound
    d. Coronary artery calcium score
    e. Can D

16. All of the following are true statements about coronary artery calcium scoring EXCEPT
    a. Detects early, nonobstructing plaque.
    b. Helps guide how aggressive to treat existing vascular risk factors.
    c. 64 slice CT scanners rival the quality of EBCT.
    d. Plaque must be calcified to be detected with calcium scoring.
    e. All statements are true

17. All of the following are true statements about carotid intima-media thickness (CIMT) ultrasound EXCEPT
    a. The American Heart Association does not recommend it for screening.
    b. It is an invasive test.
    c. It is only valuable to assess stroke risk.
    d. Physicians may perform the test in their office.
    e. National standards for quality control have been established.

18. Which of the following is a true statement about carotid intima-media thickness (CIMT) ultrasound?
    a. Carotid intima-media (CIMT) thickness ultrasound
    b. Coronary artery calcium score
    c. Carotid Doppler ultrasound
    d. Nuclear stress test
    e. Stress echocardiogram

19. Vascular disease regression or progression is best seen through repeating which of the following tests?
    a. Carotid intima-media (CIMT) thickness ultrasound
    b. Coronary artery calcium score
    c. Carotid Doppler ultrasound
    d. Nuclear stress test
    e. Stress echocardiogram

20. Which of the following is a true statement about finding early vascular disease?
    a. Disease can be found on chest x-ray, panorex, or mammogram.
    b. A thorough assessment for early vascular disease requires both coronary artery calcium scoring and carotid intima-media thickness (CIMT) ultrasound testing.
    c. Exercise stress tests have a high false negative rate.
    d. A and C
    e. All of the above
A Comprehensive Review of Vascular Disease: Part 1 - Pathophysiology and Early Detection

Name: ___________________________ Title: ___________________________ Specialty: ___________________________

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2. Anatomy and physiology of plaque development
3. Testing available to detect early disease
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