Adopted: 6/05

Abdominal Aortic Aneurysm (AAA): Detection and Referral

Abdominal aortic aneurysms are probably the most common, potentially life-threatening condition routinely detected by a chiropractor.

Main Clinical Points

- For men, incidence starts at age 50, increases rapidly after 55, with peak prevalence (5.9%) at age 80-85.
- For women, incidence starts at age 60, increases rapidly after 70, with peak prevalence (4.5%) after age 90.
- The most common presentation of symptomatic AAAs is pain felt in the back, abdomen, flank, groin, or testicles (generally more on the left side) unaffected in quality or intensity by changes in position. Sometimes a pulsing sensation in the abdomen.
- A pulsatile abdominal mass, slightly to the left of the spine between the xiphoid and umbilicus, may be the first clinical sign.
- Abdominal palpation is the only physical exam procedure with proven value in the detection of AAAs.
- Signs of rupture include the classic triad (hypotension, back pain, pulsatile abdominal mass), but may be limited to severe or "piercing" pain of recent onset or recently progressive; abdominal palpation may be very tender.
- Manipulation is absolutely contraindicated in an active AAA and referral is either emergent or urgent.
- The imaging modality of choice for suspected aneurysm is ultrasound of the abdominal aorta, (not an abdominal ultrasound).
- X-rays have limited sensitivity and limited accuracy in measurement of aneurysms. In high risk individuals (e.g. elderly males) opening the collimation on both frontal and lateral radiographs may aid in detection.
- U.S. men between 65 and 70 years old who have ever smoked should be screened one time with ultrasound.

BACKGROUND

An aneurysm is defined as a permanent focal dilatation of an artery with an increase of 1.5 times its normal lumen diameter (Ebaugh 2001, Pearce 2005, Santilli 1997, USPSTF 1996). For the infrarenal abdominal aorta, the normal measurement is considered to be on average 2.0 cm in diameter (21.4 mm in men and 18.7 mm in women) (Santilli 1997). Increases in aortic diameter naturally occur with increasing age, in males, in African Americans, and with increasing body dimensions (Pearce 2005). Aortas measuring >2.0 and <3.0 cm are considered to exhibit *dilatation* or *ectasia.* An abdominal aortic aneurysm (AAA) is generally considered to be a lumen diameter of 3.0 cm or greater (Ebaugh 2001, USPSTF 1996). AAAs may exhibit a wide variation in size, shape, and extent of aorta involved (Gorski 1999, Pearce 2005).

Most abdominal aortic aneurysms (AAA) occur between the branches of the renal and common iliac arteries. This portion of the aorta withstands high pulsatile stress due to its tapering shape, the absence of major branches, and high resistance from the lower extremities. Also, there are fewer elastic lamellae present in the walls here compared with the thoracic aorta. (Gorski 1999)

Incidence and Prevalence

Most abdominal aortic aneurysms occur in white males over the age of 60 years (Pearce 2005, USPSTF 1996) with the incidence increasing with age (Geraghty 2003, USPSTF 1996). They are rare in patients under 50 years old. Most authors place the incidence in the 5-8% range (Santilli 1997, Sparks 2002, USPSTF 1996). However, the prevalence of abdominal aortic aneurysms in the general population aged 60 years or greater is considered to be from 0.5-10.7% depending on the source and type of study (i.e., screening study, mixed gender studies, autopsy, etc.) (Ebaugh 2001,

Pearce 2005, Rigatelli 2003, Santilli 1997, Sparks 2002, USPSTF 1996).

- For men, incidence starts at age 50, increases rapidly after 55, with peak prevalence (5.9%) at age 80-85.
- For women, incidence starts at age 60, increases rapidly after 70, with peak prevalence (4.5%) after age 90.

Abdominal aortic aneurysms occur more frequently in men and occur in men at an earlier age than they do in women (Ebaugh 2001). The male-to-female incidence ratio in patients younger than 80 years of age is 2:1. For patients over the age of 80 years, the male-to-female ratio is 1:1 (Pearce 2005). However, the male-to-female ratio for *death* from AAA is significantly higher at 11:1 in patients aged 60-64 years and 3:1 for patients aged 85-90 years (USPSTF 1996). Men over the age of 60 and women over the age of 70 account for 95% of all AAA-related deaths (USPSTF 1996).

Ruptures of abdominal aortic aneurysms are the 13th leading cause of death in the United States (Gorski 1999) and the 9th leading cause of death for men over the age of 65 in Canada (Blanchard 2000). In developed countries, the rate of incidence has been increasing significantly over the past several decades (Blanchard 2000). Between 1950 and 1980, the incidence of AAA in the U.S. increased from 8.7 per 100,000 persons to 36.5 per 100,000 persons, representing a four-fold increase (Geraghty 2003, Santilli 1997, Sparks 2002). The increase in prevalence of AAA may be explained in part by growth in numbers of the elderly population and advancements in diagnostic imaging that allow for better detection rates (Bush 2003, Geraghty 2003, Santilli 1997, Sparks 2002).

It is estimated that 1.5 million people in the United States alone have abdominal aortic aneurysms and there are approximately 200,000 new cases diagnosed each year (Bush 2003). Approximately 15,000-16,000 rupture-related AAA deaths occur each year (Bush 2003, Geraghty 2003, Santilli 1997). There are an estimated 45,000 elective AAA repair surgeries performed annually in the United States (Bush 2003, Geraghty 2003, Santilli 1997). These statistics will increase drastically over the coming decades as the U.S. Census Bureau estimates that the population aged 85 and older will increase from 4 million in 2000 to 18 million by 2050 (Geraghty 2003).

Etiology

It has long been thought that atherosclerotic disease was the primary factor responsible for the development of aortic aneurysms, but a sole causal relationship has not been confirmed (Blanchard 2000, Gorski 1999, Pearce 2005). Additionally, the observation that atherosclerotic occlusive disease is associated with AAAs in only 25% of patients (Santilli 1997) has prompted reevaluation of aneurysm etiology. Greater than 90% of aortic aneurysms are considered to be primarily due to degeneration of the aortic walls. (Gorski 1999, Pearce 2005, Santilli 1997).

It is theorized that aneurysms develop in two steps. The first step is the destruction of the elastic media. In vitro studies have shown that disruption of elastin results in arterial dilatation of 25%-65% (Blanchard 2000). Atherosclerotic disease and cigarette smoking (Gorski 1999) may contribute in part to this first step. The second step is failure of the collagen within the arterial wall, resulting in the dilatation progres-sing to aneurysm expansion and rupture. Several factors may contribute to this second step. A genetic predisposition of collagenolysis (Blanchard 2000, Pearce 2005, Santilli 1997) may be associated with both X-chromosome linked and autosomal dominant patterns of inheritance (Santilli 1997). Autoimmune inflammatory reactions within the artery walls, among other processes, may weaken the aorta (Blanchard 2000).

Other potential mechanisms of aneurysm formation include the following: infection due to direct extension, septic embolism, penetrating trauma, or mycosis; specific forms of arteritis like Takayasu's arteritis; inherited connective tissue disorders like Marfan's disease or Ehlers-Danlos syndrome; cystic medial necrosis; trauma; and aterial anastomoses. (Gorski 1999, Pearce 2005, Santilli 1997)

Clinical Presentation

Only 20-30% of patients with AAA have symptoms. Symptoms result from either expansion, pressure on adjacent structures, embolization, dissection, small tears and leaks, or a full rupture. The clinical suspicion of AAAs is dependent on patient history and the presence of risk factors, and/or associated diseases or conditions (Blanchard 2000). When symptoms are present, they are non-specific and vary considerably depending on location, adjacent structures, and the nature of change in morphology of the aneurysm.

- The most common presentation of symptomatic AAAs is pain. The pain may be located in the back (Gorski 1999, Pearce 2005, Santilli 1997), abdomen (Gorski 1999, Pearce 2005, Santilli 1997), flank (Gorski 1999, Santilli 1997), groin (Gorski 1999, Pearce 2005), or testicles (Gorski 1999) and is generally felt more on the left side. Sometimes patients report feeling pulsations in their abdomen. The onset is usually acute and is unaffected in quality or intensity by changes in position (Santilli 1997).
- The most common presentation of a rupture is shock, a pulsatile abdominal mass, and acute onset of excruciating back or abdomen pain (Gorski 1999, Pearce 2005, Santilli 1997).
- The pulsatile abdominal mass, located slightly to the left to the spine between the xiphoid and umbilicus (Gorski 1999, Pearce 2005, Santilli 1997) may be the first clinical sign.
- Transient hypotension (Gorski 1999), syncope (Gorski 1999, Pearce 2005, Santilli 1997), and/or a temporary loss of consciousness (Pearce 2005) may precede other symptoms attributable to aneurysm rupture.

Abdominal aortic aneurysms may produce symptoms due to pressure on adjacent structures or viscera. The diffuse and nonspecific nature of these symptoms often may cause errors or delays in the diagnosis (Santilli 1997). Symptomatic AAAs may be mis-diagnosed as renal calculus, diverticulitis, incarcerated hernia, and/or lumbar spine disease among other entities (Pearce 2005). Isolated groin pain may result from pressure on the right or left femoral nerve (Santilli 1997).

Symptom Variety

Specific Pathology	Symptoms
Intestinal compression (most commonly the duodenum)	early satiety, nausea, weight loss (Gorski 1999, Santilli 1997), and vomiting (Pearce 2005, Santilli 1997)
Ureteral obstruction	urinary urgency, hydronephrosis and presenting as flank pain, pyelonephritis (Gorski 1999), and/or a flank mass (Pearce 2005)
Dissection of the urinary artery	flank pain and hematuria mimicking a renal stone (a common misdiagnosis)
Free intraperitoneal rupture	hemodynamic instability resulting in prompt cardiovascular collapse and death (Santilli 1997)
Contained rupture into the retroperitoneal space from a small posterolateral tear (may stay relatively contained for an indeterminate amount of time before progressing to intraperitoneal rupture)	This type of aortic compromise often presents as acute pain with or without initial hemodynamic instability preceded by syncope and/or tachycardia (Santilli 1997); then cardiovascular collapse, and death (Santilli 1997)
Rupture into the vena cava creating aortocaval fistulae	tachycardia, congestive heart failure, renal failure, and lower extremity ischemia and swelling (Pearce 2005)
Rupture into the duodenum creating aortoduodenal fistulae	upper gastrointestinal bleeding preceding hemorrhage and death (Pearce 2005)
Thrombosis of small aneurysms may result in acute aortic occlusion	symptoms of acute claudication (Pearce 2005)
Emboli from small AAAs may produce lower extremity ischemia (Gorski 1999, Pearce 2005)	livedo reticularis of the feet (AKA Blue Toe Syndrome) (Pearce 2005)

PHYSICAL EXAMINATION

Summary of examination procedures

- Blood pressure & pulse; temperature (optional)
- Inspection and palpation of the feet
- Abdominal palpation

Vital Signs

The patient may be normotensive or present with either hypertension or hypotension. The presence of hypertension, especially diastolic hypertension, is a risk factor for getting an aneurysm and for rupture of one. Hypotension suggests that the AAA may be already leaking or rupturing. Hypotension may be accompanied by the signs of frank hypovolemic shock (tachycardia, altered mental status, cyanosis and mottling of the skin). Syncope can be the presenting symptom with the pain being less significant. Fever may also be present (Pearce 2005).

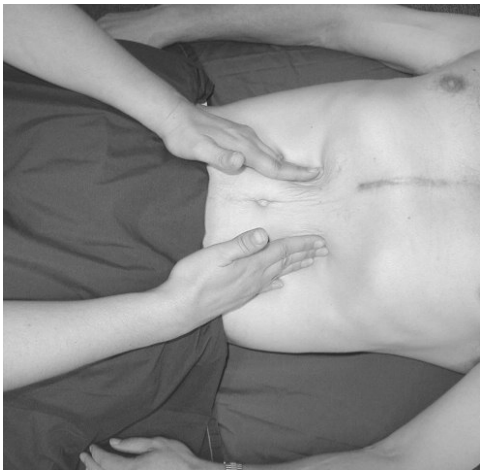
Lower Extremity Examination

Lower extremity symptoms include venous thrombosis, lower extremity ischemia (Gorski 1999, Pearce 2005), or even paralysis (Pearce 2005). An embolic phenomenon of the toes, livedo reticularis (AKA, Blue Toe Syndrome or "trash foot"), presents as tissue breakdown and color change from infarcts in the toes despite good pulses. In other cases, lower extremity pulses may be decreased.

Abdominal Examination

Abdominal palpation is the only physical exam procedure that has shown to have demonstrated value in the detection of AAAs (Venkatasubramaniam 2004). 90% of AAAs are between the renal arteries and common iliac bifurcation. The presence of a pulsatile abdominal mass or an exaggerated abdominal pulsation (Rigatelli 2003) should raise the suspicion of an abdominal aortic aneurysm and the diagnosis should be assumed until proven otherwise (Gorski 1999). It has been suggested that abdominal palpation be included in the pre-manipulaiton examination. (Gatterman 2004, Winterstein 1984)

Abdominal bruit, femoral bruit, absent femoral pulses, pulsations located more than 3 cm caudal to the umbilicus (Lederle 1999), and a transmitted epigastric pulse (Fink 2000) were all found to have no predictive value for the detection of AAAs.



Most clinically significant (i.e., large) AAAs are palpable during the physical examination (Pearce 2005). The patient should be relaxed with hips and knees bent. If possible, raise the head of the examination/adjusting table. Flat hand palpation may be used in an effort to palpate the lateral aspects of the aorta as the patient exhales.

The sensitivity of the procedure increases with increasing aneurysm size (Pearce 2005, Venkatasubramaniam 2004). The sensitivity of palpation for aneurysms measuring 3.0-3.9 cm is estimated at 29% (Lederle 1999)-61% (Fink 2000); for aneurysms measuring 4.0-4.9 cm sensitivity is 50% (Lederle 1999)-72% (Fink 2000); and for aneurysms measuring 5.0 cm or greater the sensitivity is 76% (Lederle 1999)-82% (Fink 2000).

The identification of a widened aorta greatly increases the odds that an AAA is present, with a positive likelihood ratio of 7.6. However, the absence of increased aortic diameter is only moderately effective in ruling out an AAA (Lederle 1999) with a negative likelihood ratio of 0.6.

In addition to aneurysm size, the effectiveness of abdominal palpation depends on examiner skill (Pearce 2005), patient cooperation, the presence of ascites, the tortuosity of the aorta, excessive lumbar lordosis (Santilli 1997), and the size of the patient (Ebaugh 2001, Lederle 1999, Pearce 2005, Venkatasubramaniam 2004). A 1.0 cm increase in the size of the aneurysm doubles the odds of AAA detection whereas a 1.0 cm increase in abdominal girth decreases the odds of detection by palpation by approximately 10% (Fink 2000). *In one study, the sensitivity for AAA detection became 100% when the aneurysm measured greater than 5.0 cm and the patient's*

abdominal girth measured less than 100 cm (40 inches) (Fink 2000).

However, due to its limited sensitivity when averaged over a variety of aneurysm sizes, palpation cannot stand alone and should be used as a complimentary procedure to imaging studies (Venkatasubramaniam 2004). The role of abdominal palpation is to help identify those patients who should undergo imaging to confirm the presence of AAAs (Lederle 1999). In cases where there is clinical suspicion, physical examination should not be relied upon to rule out the presence of an AAA (Lederle 1999).

Risk Factors for Abdominal Aortic Aneurysm

An important part of gauging the likelihood of an AAA is to take into consideration the patient's risk factors. Risk factors were generally stronger for women than for men. This is particularly true for pack-years of smoking. The following table is based on Pearce 2005 unless otherwise noted.

Association	Risk Factor	Comment
Strong	Increasing age	For men: incidence starts at age 50, incidence increases rapidly after 55, peak prevalence (5.9%) at age 80-85. For women: incidence starts at age 60, increases rapidly after 70, peak prevalence (4.5%) after age 90.
Strong	Cigarette smoking	Has been identified as the most closely associated risk factor (Lederle 1997). Strongly associated in a dose-dependent fashion. Long-term smoking increases individual's risk 5x over baseline.
Strong	Male gender	Positively associated. AAAs 4-5x more common in men than in women.
Strong	Family history of AAA	Familial incidence of 15-25%. Increases individual's risk 2x—especially if first degree male relative.
Strong	Caucasian	AAAs are uncommon in African Americans, Asians, and Hispanics.
Strong	Increased diastolic pressure	Strongly associated with AAA.
Moderate	Diabetes mellitus	Significant correlation.
Moderate	Systemic atherosclerotic occlusive disease	Associated in 25% of AAA cases. Modest correlation; more so with large AAAs than with smaller ones.
Moderate	Peripheral vascular disease	Modest correlation.
Moderate	Cerebrovascular disease	Modest correlation.
Moderate	Coronary artery disease/history of coronary bypass (Ebaugh 2001)	Modest correlation. Coronary artery disease present in 70% of patients with infrarenal AAA.
Moderate	History of myocardial infarction	Modest correlation.
Moderate	Presence of peripheral arterial aneurysm	Patients with popliteal artery aneurysm have a 25%-50% incidence of AAAs. Out of patients with AAAs, 41% have iliac aneurysms and 15% have femoropopliteal aneurysms.
Moderate	Carotid artery disease	10% of patients with carotid artery disease also have an AAA.
Moderate	Thoracic aortic aneurysm	20%-30% of patients with thoracic aortic aneurysm also have abdominal aortic aneurysm.
Weak	Hypertriglyceridemia	General hypercholesterolemia appears to have no correlation.
Weak	Hypertension	General hypertension has a weak correlation.
Weak	Chronic obstructive pulmonary disease	Difficult to establish as an independent risk factor.

ANCILLARY STUDIES

Imaging AAA

The vast majority of abdominal aortic aneurysms are found incidentally on imaging studies, usually plain films of the lumbar spine or computed tomography (CT) exams of the abdomen, performed for unrelated reasons. (Santilli 1997) The imaging modality of choice for suspected aneurysm is ultrasound. (Gorski 1999, Santilli 1997) Other advanced imaging modalities such as magnetic resonance imaging (MRI), computed tomography, and MRI- or CT-angiography, etc. may be employed as determined on a case-specific basis. Advanced imaging should be performed in cases of suspected leak or rupture regardless of other findings. (Lederle 1999)

Plain Film

The majority of AAAs is seen on plain film between the levels of L2 and L4. The aorta and its branches will only be visualized on x-rays when calcification is present. Calcium deposits lining the walls of abdominal or pelvic arteries often give a rough indication of the size of vessels specifically of the abdominal aorta and its terminal branches. In aneurysmal blood vessels, the walls become divergent at the site of focal outpouching rather than the normal parallel tracks seen in atherosclerotic, nonaneurysmal vessels. For the abdominal aorta, the radiographic determination of aneurysm versus dilatation occurs at the cut-off measurement of 3.8 cm (Yochum 2005).

Identification of aneurysms of the abdominal aorta and its terminal branches by means of x-ray has a low sensitivity with calcific plaquing exhibited in only 67-75% of AAAs (Santilli 1997); however, since the vast majority of these aneurysms are asymptomatic, this incidental finding by simple and inexpensive (Santilli 1997) means is of immeasurable value.

Plain film should not be used as a definitive imaging modality. The detection of AAAs on plain film merits further evaluation by more sensitive imaging modalities, such as ultrasound or CT, to determine more accurate measure-ments for both size and location. X-rays are very limited in accuracy of the measurement of aneurysms, as only calcified portions are visualized and those may not coincide with the area of largest dimension. Additionally, x-rays are a two-dimensional representation of three-dimensional vessels and the planar nature may underestimate the size of dilatation. Other limitations of x-ray include superimposition of structures, specifically the aorta and the lumbar spine. Often, the right side of the aorta is not visualized on frontal radiographs (Yochum 2005). The right anterior oblique view may help reduce superimposition of structures and lead to better definition of the aorta. Tight collimation on lateral lumbar x-rays may cut-off the anterior margin of the aorta making identification of enlargement difficult to discern (Yochum 2005). ***Note: In high risk individuals (e.g. elderly males) opening the collimation on both frontal and lateral radiographs may aid in the diagnosis of aneurysms***.

The determination of which imaging modality to employ is case-specific and various factors are taken into account, such as co-existing disease processes, the patient's ability to undergo the procedure, and the need to visualize additional structures or organs, etc. The usual and customary follow-up imaging is ultrasound (Gorski 1999, Pearce 2005).

As well as having a prominent role in the detection of AAAs, x-ray is useful as a follow-up modality in imaging post-surgical status of endovascular arterial repair. X-ray films are taken at regular post-surgical intervals (i.e., one month, six months, 12 months, and then annually) and are essential in the detection of stent fractures, separations, graft limb kinking, compression, and device migration (Tanquilut 2003).

Ultrasound

B-mode ultrasound is the method of choice for confirmation of the presence of AAAs (Gorski 1999, Pearce 2005), for the initial evaluation, and for follow-up surveillance (Santilli 1997). Ultrasound has a reported sensitivity of anywhere from 82% to near 100% (Ebaugh 2001, Lederle 1999, Santilli 1997, USPSTF 1996) and a specificity of 100% (Ebaugh 2001, Santilli 1997). Ultrasound is readily available, safe, noninvasive, relatively inexpensive, can be performed in 99% of screened patients and delivers no ionizing radiation (Ebaugh 2001, Santilli 1997). Ultrasound provides information regarding the amount of plaquing and measurements in both the transverse and longitudinal dimensions (Santilli 1997). The margin of error according to the USPSTF is within 2 to 5 mm (Pearce 2005, Santilli 1997, USPSTF 1996). Ultrasound measurements are consistently smaller than those provided by CT by 2.7 to 4.4 mm.

The limitations of ultrasound are few. In a small percentage of patients the aorta may be obscured by large amounts of bowel gas, by periaortic

disease, or due to obesity (Santilli 1997, USPSTF 1996). Additionally, ultrasound is limited in delineating the proximal end of the AAA in relationship to the renal arteries (Santilli 1997).

***Note: When ordering an ultrasound exam for a patient suspected of having an abdominal aortic aneurysm, the physician should specify an ultrasound of the abdominal aorta rather than merely an abdomen ultrasound* (Santilli 1997).**

Computed Tomography (CT)

Computed tomography is the imaging modality that best delineates size, location, longitudinal boundaries, and arterial wall characteristics including breaches (Gorski 1999, Santilli 1997). CT is the imaging modality of choice for ruptured and/or leaking abdominal aneurysms (Santilli 1997). One of the primary benefits of CT over other imaging types is the ability to detect additional aneurysms in other locations as well as the simultaneous assessment of other organs and of the venous system (Gorski 1999). Anywhere from 21-66% of patients with abdom-inal aortic aneurysms also have aneurysms in the common iliac arteries either as separate entities or by extension from the aorta; coexisting aneurysms may also exist elsewhere in the body (Yochum 2005). Computed tomography has essentially exhibited 100% both sensitivity and specificity (Lederle 1999). CT is also considered the imaging modality of choice for anatomical assessment and preoperative planning (Pearce 2005). Some of the drawbacks of this imaging procedure are the exposure of the patient to ionizing radiation, the relative expense, and the potential for motion artifact (Gorski 1999, Santilli 1997). Enhanced or three-dimensional spiral CT can provide further information about anatomical structures essential to preoperative assessment and surgical planning for both open repair and endovascular arterial repair (EVAR) (Pearce 2005).

Computed tomography is also an essential component of the long-term follow-up of patients who have undergone EVAR. CT, with and without contrast, is performed on these patients prior to discharge and at specific intervals (i.e., one month, six months, 12 months, and then annually) to evaluate for endoleaks (flow of blood outside the endograft but inside the aneurysm wall). Endoleaks are a major complication of this method of repair and necessitate secondary procedures, therefore, regular monitoring is vital for detection and for patient survival (Tanquilut 2003).

Magnetic Resonance Imaging (MRI)

In recent years, the utilization of magnetic resonance imaging (MRI) has increased for a wide range of conditions including AAA. MRI provides similar results as CT in visualization of abdominal aneurysms while providing three-dimensional images. With magnetic resonance imaging, no intravenous contrast material is needed and no ionizing radiation is involved (Bush 2003). However, MRI examinations are relatively expensive, are generally less available, and have several patient contraindications, such as claustrophobia and ferromagnetic implants (Santilli 1997), and have a reportedly lower patient satisfaction rate (Bush 2003).

Angiography

Traditionally, conventional contrast angiography was the most sophisticated imaging modality for detailed evaluation of AAAs. Today, with the advent of other methods it is the least useful modality and is comparatively expensive and invasive (Santilli 1997). Traditional angiography is useful in providing details about renal artery and visceral artery involvement and occlusive disease (Gorski 1999, Pearce 2005, Santilli 1997) and should be performed in cases where renal abnormalities exist (i.e., horseshoe kidney) (Pearce 2005). This method is also useful for imaging the tortuosity of the the iliac vessels and for determining the appropriate length of stent-grafts in endovascular arterial repair (Bush 2003).

Traditional contrast angiography is now being surpassed by CT-angiography and MRI-angiography. Both of these methods provide excellent anatomical assessments of the aorta and surrounding structures while being less invasive (Pearce 2005, Santilli 1997).

Additional Modalities

Other diagnostic modalities, such as echocardiography, are useful in the evaluation and monitoring of co-existing diseases and risk factors; their use is determined on a case-by-case basis (Pearce 2005).

MANAGMENT

The discovery of an AAA requires referral. The timing of the referral depends on the circum-stances of the patient and situation. The presence of symptoms attributable to abdominal aortic aneurysm is considered indicative of leak, expansion, rupture (Gorski 1999, Santilli 1997, Sparks 2002,), dissection, embolization, and/or

thrombosis (Gorski 1999, Santilli 1997) and constitutes an immediate medical emergency.

Acute rupture is the manner of presentation in up to 20% of cases (Santilli 1997). When an abdominal aortic aneurysm ruptures, the result is fatal massive intra-abdominal hemorrhage unless successful emergency surgery can be performed (USPSTF 1996).

Red Flags for Rupture

- Of those with ruptures only about 50% have the classic triad of
  - Hypotension
  - Back pain
  - Pulsatile abdominal mass (Ernst 1993)
- Pain of recent onset or recent progression in intensity
- Pain is severe and "piercing"
- Abdominal palpation is very tender

Factors predisposing to rupture

- Increased diameter (especially over 6 cm)
- History of smoking
- Diastolic hypertension
- Family history of rupture
- Chronic obstructive artery disease
- Rate of expansion
- Inflammatory aneurysm

Timing of Referrals

The following recommendations are derived from the WSCC consensus-based protocol "Emergent Referrals."

- **Emergent referral**
  Immediately and directly to an emergency room setting: Patients with back or abdominal pain suspected to have an AAA who additionally have any of the following findings:
  - tenderness with abdominal palpation
  - palpable pulsating mass
  - hypotension/signs of shock
  - rapidly increasing severity of acute back or abdominal pain
  - sudden change or increase in symptoms
  - acute back pain that is not improved or reproduced by position or joint loading
  - the presence of nausea or vomiting
  - x-ray evidence of AAA 7 cm or greater

  Patients with an established AAA who display the above signs or symptoms not attributable to other likely causes (e.g., nausea and vomiting secondary to intestinal flu) should receive emergent referral.

- **Urgent referral**
  The patient should seek further evaluation and care the same day: Large (6 cm or greater) AAA found on an x-ray in a patient with acute back pain, but none of the additional symptoms or signs cited in the emergent referral section.

- **Semi-urgent referral**
  The patient should seek further assessment and care within the next 48 hours: AAA over 6 cm on radiograph found incidentally. If there are incomplete signs of calcification of the aorta and size cannot be well visualized, the case should be treated as if it is over 6 cm.

- **Non-emergent/urgent referral**
  Patients who have an abdominal aortic aneurism under 6 cm, presenting with acute low back pain which appears to be mechanical in nature and who have none of the red flags cited in the emergent referral section should also be investigated but this referral is not as time sensitive.

The detection of AAAs is usually the result of incidental findings seen during an unrelated imaging or surgical procedure or from physical examination (Santilli 1997). Some authors recommend investigating anything over 3.8 cm (2 cm is the typical size of the abdominal aorta, 2-3 cm suggests some possible dilatation).

Manipulation

Patients who are discovered to have an AAA should be referred for further assessment. **Manipulation is absolutely contraindicated in an active AAA.** However, it must be acknowledged that based on the prevalence of the condition, patients with a small, asymptomatic aneurysm undergo manipulative therapy frequently. It is not known whether side posture or other positioning or the adjustment itself generates sufficient force to affect a small aneurysm (Crawford 2003). No reports of manipulation leading to the rupture of an AAA were found for this protocol.

Cases in which the patient and practitioner are aware of a small stable aneurysm, manipulation should be approached with great care. Ebrall suggests the suspicion of an abdominal aortic aneurysm indicates "a need for careful consideration of patient positioning and selection of appropriate techniques." For example, techniques with lower force might be selected or manipulation can be withheld entirely. The presence of aortic calcification elevates the cautions "to the next level." (Ebrall 2004)

The Course of Abdominal Aortic Aneurysms

The natural course of most aneurysms is one of gradual enlargement (Lederle 1999, Pearce 2005, Santilli 1997, Sparks 2002). The rate of growth of AAAs is directly related to aneurysm size and is estimated to be 0.2-0.8 cm per year (Lederle 1999, Pearce 2005, Santilli 1997). In one study the rates of expansion for AAAs detected in men aged 65 years were dependent on aneurysm size at initial diagnosis. The expansion rates were as follows:

Initial AAA size	Annual Expansion Rate
2.6-2.9 cm	0.09 cm/year
3.0-3.4 cm	0.16 cm/year
3.5-3.9 cm	0.32 cm/year

(Pearce 2005)

It is estimated that at least 60% of small AAAs will grow to require treatment within 5 years of initial detection (Ebaugh 2001).

Approximately 1/3 of all AAAs will eventually proceed to rupture (Gorski 1999, Lederle 1999). The incidence rate of rupture is 1-21 cases per 100,000 person-years (USPSTF 1996), though an accurate count is difficult to determine, as the cause for many rupture-related deaths may go undetermined. As with the rates of expansion, the rates of rupture are directly related to initial diameter at the time of diagnosis (Pearce 2005, Santilli 1997, USPSTF 1996); the risk increases with increasing aneurysm size (Lederle 1999). There is relatively low risk of rupture for small, asymptomatic, slow-growing abdominal aortic aneurysms less than 6.0 cm in diameter (USPSTF 1996).

The rupture rates at five years after detection increase significantly with increasing diameter: 25% of aneurysms measuring 5.0 cm diameter, 35% of aneurysms measuring 6.0 cm, and greater than 75% of aneurysms measuring 7.0 cm will rupture within 5 years (Gorski 1999). In addition to increased size, other factors contributing to rupture include the presence of hypertension, concomitant chronic obstructive pulmonary disease (Gorski 1999, Pearce 2005), and continued smoking (Pearce 2005).

Aneurysm Size	Annual Rupture Rate
4.0-4.9 cm	1% (USPSTF 1996, Venkatasubramaniam 2004)
5.0-5.9 cm	3%-6.6 % (Santilli 1997, USPSTF 1996)
6.0-6.9 cm	9% (USPSTF 1996)
≥7.0 cm	19%-25% (Santilli 1997, USPSTF 1996)

Surveillance

For small aneurysms and poor surgical candidates, management consists of periodic surveillance by imaging studies and attempts to reduce the rate of expansion and decrease the risk of rupture by controlling the risk factors (e.g., aggressive hypertension control by use of beta-blocker therapy, cessation of smoking, etc.) (Ebaugh 2001, Lederle 1999, Pearce 2005). The recommendations cited below for reevaluation by imaging exams are relative guidelines only.

Surveillance Intervals (based on Pearce 2005 & Ebaugh 2001)	
Aneurysm Diameter	**Time interval for Imaging**
2.6-2.9 cm	Rescan at 3-5 years
3.0-3.4 cm	Rescan at 2-3 years
3.5-3.9 cm	Rescan at 1 year
4.0-4.9 cm	Rescan at 6 months
5.0-5.4 cm	Rescan at 3 months
≥ 5.5 cm	Evaluate for elective surgery

Surgical Repair

The only treatment for AAA is surgical repair (Gorski 1999, Santilli 1997), either by open major abdominal surgery or endovascular repair techniques (Tanquilut 2003, Hinchliffe 2003, Geraghty 2003). When surgical repair is undertaken as an elective procedure, the outcome is significantly better than with emergency repairs. Elective surgery has a perioperative mortality rate of 1.4%-8.4% (depending on the source) (Bush 2003, Geraghty 2003, Hinchliffe 2003, Pearce 2005, Santilli 1997, Tanquilut 2003). Patients who undergo elective surgery have a 5-year survival rate of 46% (Hinchliffe 2003)-61% (Santilli 1997). In comparison, only 40-50% of patients with ruptured AAAs will survive to receive treatment and the perioperative mortality rate of those

emergency procedures is approximately 50% (Gorski 1999). The overall mortality rate for ruptured of AAAs is 75-90% (Ebaugh 2001, Gorski 1999, Lederle 1999, Santilli 1997).

Elective surgery is considered if the aneurysm is growing quickly (>0.5 cm in 6 months (Santilli 1997) or >1.0 cm in one year (Ebaugh 2001)), is symptomatic (Ebaugh 2001, Gorski 1999), or has reached sufficient size (Ebaugh 2001). The accepted size for surgical consideration is 4.0-6.0 cm, depending upon the facility and/or surgeon (Ebaugh 2001, Gorski 1999, Lederle 1999, Pearce 2005, Santilli 1997, USPSTF 1996), though most commonly 5.0 cm is the threshold used (Gorski 1999, Lederle 1999, Santilli 1997). Surgical repair is considered for anything over 5 to 5.5 cm.

Risk of Death (untreated)	
6 cm	25% risk within 1 year
	50% risk within 5 years
> 6 cm	50% risk within 1 year
	75% risk within 2 years
	90% risk within 5 years
Risk of Death (surgical repair)	
Surgical repair at 1 year	1.4-8.4% mortality rate
After rupture	75-90% mortality rate

Two large studies, the USAT with 1,090 subjects (Powel 1998) and ADAM with 1,136 subjects found that early surgical intervention on AAA that measured 4-5cm offered no advantage over a watch and wait strategy. There was no difference in survival between treatment groups at 2, 4 or 6 years.

The size requirements for surgical intervention are based on males of average height (170 cm) and therefore may not be applicable to women or to men of smaller stature. Smaller patients may be suitable for surgery for aneurysms of proportionally smaller diameter (Pearce 2005).

For each individual patient, the risks of surgical intervention must be weighed against the risks of aneurysm rupture (Lederle 1999, Pearce 2005, USPSTF 1996), taking into account the patient's age, overall health condition, psychological state, the presence of comorbid conditions, and life expectancy (Gorski 1999, Pearce 2005). Contraindications to surgical intervention for aneurysms that meet the criteria (i.e., greater than 5.5 cm diameter, fast growth, and/or symptomatic) include severe chronic obstructive pulmonary disease (COPD), severe cardiac disease, active infection, advanced cancer, medical conditions that preclude surgical procedures (Pearce 2005), as well as a life expectancy of less than two years (Gorski 1999) among others.

Comparison of Open Surgical Repair vs. Endovascular Repair

Survival rates at one year are reported to be approximately 92% for open repair and 95% for endoscopic repair. At two years the survival rate rates are about equal at 89.6% for open repair and 89.7% for endoscopic repair. (Blankensteijn 2005)

	Open Surgical Repair	Endovascular Repair
Invasiveness	High	Low
Perioperative Mortality Rate (Bush 2003, Geraghty 2003, Hinchliffe 2003, Pearce 2005, Santilli 1997, Tanquilut 2003)	1.4-8.4%	1.7%
Length of Hospitalization (Bush 2003)	5-10 days	1-3 days
Hospital stay location (Bush 2003)	Intensive care unit	General vascular ward

Prognosis of AAAs

Long-term prognosis of patients with abdominal aortic aneurysms is related to associated comorbidities (Pearce 2005). As most patients with abdominal aortic aneurysms are elderly and have serious coexisting medical conditions, the majority of these people will die of other causes rather than from those related to the AAA (Pearce 2005, USPSTF 1996). The majority of these patients will die primarily due to coronary artery disease or stroke before they meet the surgical criteria and before rupture (Santilli 1997, USPSTF 1996).

Mass screening of AAA in asymptomatic subjects

The value of mass screening programs aimed at early detection is a matter of debate. Screening studies generally are performed by means of ultrasound. It is well documented that most screening studies identify small (<5.5 cm) aneurysms (Ebaugh 2001, Pearce 2005, USPSTF 1996, Venkata-subramaniam 2004); in one study nearly 80% of AAAs detected were smaller than 4.0 cm (Pearce 2005). The results of several mass screening studies showed that only a very small percentage (0.5%-2.1%) of identified AAAs underwent surgical intervention (Ebaugh 2001).

Concerns about universal screening include accessibility issues (Venkatasubramaniam 2004), patient compliance, high costs, increased surgical workload, and induced anxiety in the population screened (Ebaugh 2001), among others. It is estimated that the clinical benefits are minimal and result in an average increase in life expectancy of only 0.002 year (Norman 2004, USPSTF 1996). However, a Danish study (Lindholdt 2005) reflected a 67% reduction in AAA specific mortality rate as a result of mass screening of 64-73 year males. So the debate continues.

Selective screening of asymptomatic subjects

Patients who are considered high risk due to age or identified risk factors should be evaluated for the presence of an AAA. Although palpation is very useful in symptomatic patients, it is not sensitive enough to rely on as a general screening tool due to its ineffectiveness for detection of small AAAs. In a chiropractic setting, AAAs are most commonly detected with plain film radiography; however, plain film radiography lacks sufficient sensitivity to be relied upon for screening. CT and MRI examinations are too expensive to be practical (USPSTF 1996). Ultrasound remains the imaging modality of choice, even though it, too, has limitations as a screening tool as obesity, excessive bowel gas, periaortic disease may all compromise the examiner's ability to clearly visualize and measure the aorta (USPSTF 1996).

Current Recommendations

In 2005, the US Preventive Services Task Force made the following recommendations:

- U.S. men between 65 and 70 years old who have ever smoked should be screened one time with abdominal ultrasound.
- Routine screening of women is not recommended.

Selective screening may be reasonable for other target populations, such as men over the age of 60 who have other risk factors of AAA including vascular disease, diastolic hypertension (USPSTF 1996), and younger men who have a first-degree male relative with an AAA (Ebaugh 2001, USPSTF 1996). Screening is not appropriate for patients who are poor candidates for surgical intervention such as those with severe cardiac and/or severe pulmonary disease (USPSTF 1996).

Primary author: Shannon Grant, DC

Contributing author: Ronald LeFebvre, DC

Reviewed by: Peter Shull, DC

Radiology Department
- Beverly Harger, DC, DACBR
- Lisa Hoffman, DC, DACBR
- Tim Stecher, DC, DACBR
- Tamara Lovelace, DC
- Adrienne Sciberras, DC
- Eve E. Bonic, DC, RT(R)
- Dane Lockhart-Borman, DC
- Ann Ehrlich, RT
- Hank Hirsh, RT

Revised and reviewed by CSPE Committee
- Daniel DeLapp, DC, DABCO, ND, LAc
- Elizabeth Dunlop, DC
- Lorraine Ginter, DC
- Sean Herrin, DC
- Ronald LeFebvre, DC
- Owen T. Lynch, DC
- Karen E. Petzing, DC
- Ravid Raphael, DC, DABCO
- Anita Roberts, DC
- Steven Taliaferro, DC

Editorial assistant: Anne Byrer

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