DISCLOSURES

• NONE
GUIDELINES

The Society for Vascular Surgery practice guidelines on the care of patients with an abdominal aortic aneurysm

ABSTRACT

Background: Decision-making related to the care of patients with an abdominal aortic aneurysm (AAA) is complex. Assessment with varying risk of rupture, and patient-specific factors influence anticipated life expectancy, operative risk, and need to intervene. Careful attention to the choice of operative strategy along with optimal treatment of medical comorbidities is critical to achieving excellent outcomes. Moreover, appropriate preoperative surveillance is necessary to minimize subsequent aneurysm-related death or morbidity.

Methods: The committee made specific practice recommendations using the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) system. Five systematic reviews were conducted to support this guideline. The focus was on evaluating the best evidence and optimal frequency for surveillance after endovascular aneurysm repair (EVAR). A third focused on identifying the best available evidence on the diagnosis and management of AAA. Specific areas of focus included (1) general approach to the patient, (2) treatment of the patient with an AAA, (3) anatomic considerations and preoperative management, (4) perioperative and long-term management, and (5) cost and economic considerations.

Results: Along with providing guidance regarding the management of patients throughout the continuum of care, we have issued a number of prior recommendations and addressed a number of new areas of significance. New guidelines are provided for the surveillance of patients with an AAA, including recommended surveillance imaging at 2-month intervals for patients with an AAA of 40 to 44 mm in diameter. We recommend endovascular aneurysm repair as the preferred method of treatment for infrarenal necks less than 20 mm and for patients with a ruptured aneurysm. For patients with an AAA of 55 mm or greater in diameter, we recommend open aneurysm repair. We also recommend that elective AAA be limited to hospitals with a documented mortality rate of less than 5% or less than 5%, and that patients with an AAA be treated in hospitals that have a documented mortality rate of less than 5% or less than 5%.

Conclusions: The Society for Vascular Surgery recommends using the GRADE system to guide decision-making in patients with aortic aneurysms. This guideline provides a comprehensive approach to the care of patients with an AAA, including recommendations for surveillance, treatment, and management. It also highlights the importance of cost-effectiveness in the decision-making process.
ABDOMINAL AORTIC ANEURYSM

NOTABLE VICTIMS

- KING GEORGE II*
- AUGUSTE RODIN
- ROY ROGERS
- SENATOR R. DOLE
- JOE DIMAGGIO
- RODNEY DANGERFIELD
- CANDIDO JACUZZI

- DUKE OF WINDSOR
- CHARLES DE GAULLE*
- GEORGE C SCOTT*
- LUCILLE BALL*
- ALBERT EINSTEIN*
- EMILE ZOLA

* CAUSE OF DEATH
DEFINITIONS

A LOCALIZED DILATATION

- ABSOLUTE DIAMETER EXCEEDING 3.0 CM *
- DIAMETER 1.5X ADJACENT NORMAL DIAMETER

*SVS GUIDELINES 2018
ABDOMINAL AORTIC ANEURYSM
ABDOMINAL AORTIC ANEURYSM

MAGNITUDE OF PROBLEM: USA

- PRESENT IN > 1 MILLION MEN / WOMEN >50
- 45-50,000 AAA REPAIRS ANNUALLY
- 9,000-10,000 DEATHS / YEAR FROM RUPTURE
- 2ND MOST FREQUENT CAUSE OF DEATH OF ALL EMERGENCY SURGICAL CONDITIONS
- AAA RUPTURE IS A MAJOR CAUSE OF DEATH IN MEN OVER 65
ABDOMINAL AORTIC ANEURYSM

DEMOGRAPHIC FACTORS

- **MALE : FEMALE :** 3:1 - 6:1
- **AGE :** 7th - 8th DECADES
- **RACE :** 90% CAUCASIAN; 10% BLACK, ASIAN, HISPANIC
- **LOCATION :** 95% INFRARENAL
  - 5-15% SUPRA-RENAL
  - 2.5% THORACO-ABDOMINAL
- **CORONARY ART DISEASE :** 25% SYMPTOMATIC
- **HYPERTENSION :** 40%
- **PERIPH OCCLUSIVE DISEASE :** 20-30%
- **SMOKING:** 90%
ABDOMINAL AORTIC ANEURYSM

ASSOCIATED ANEURYSMS

- THORACIC 12%
- ILIAC 25%
- FEM / POP 14%
  - FEM + AAA 85%
  - POP + AAA 62%
- INTRA-CRANIAL 14%
ANEURYSMOSIS
ABDOMINAL AORTIC ANEURYSMS

PREVALENCE*

- **MEN OVER 55 YEARS**
  - > 3CM: 4.6%
  - > 4CM: 1.4%

- **WOMEN**: 1.0%

- **WITH FAMILY HISTORY**: 5%

- **WESTERN COUNTRIES**: 1.2-7%

*DATA FROM ADAM TRIAL, 1977*
DECLINING PREVALENCE

- USA
- ENGLAND
- WALES
- SCOTLAND
- NORWAY
- SWEDEN
- NEW ZEALAND
- AUSTRALIA
- FINLAND
AORTIC ANEURYSM MORTALITY

FACTORS

- ECONOMIC POLICY
- SCREENING PROGRAMS
- TECHNOLOGIC ADVANCES: EVAR
- CV RISK FACTOR CONTROL
AORTIC ANEURYSM MORTALITY

WORLD-WIDE TRENDS

- DECREASED PREVALENCE
- DECREASED MORTALITY
  - NOT UNIFORM
- LINEAR RELATIONSHIP WITH TRENDS IN
  - SBP
  - CHOLESTEROL
  - SMOKING
  - BMI (NEGATIVE)
- DIABETES: NO OR NEGATIVE IMPACT

SIDLOFF ET AL. CIRCULATION 2014
DECLININGAAA MORTALITY

WHO DATABASE 2001-2015

PNG ET AL, EUR J VASC ENDOVASC SURG 2021, 61:900-907
AAA MORTALITY AND SMOKING

US annual adult per capita cigarette consumption and US age-adjusted AAA mortality per 100,000 white men by year.

Frank A. Lederle Circulation. 2011;124:1097-1099
CIGARETTE SMOKING

- LINEAR RELATIONSHIP WITH ANEURYSM
  - DEVELOPMENT
  - EXPANSION
  - RUPTURE
- SMOKERS 7X MORE LIKELY TO HAVE AAA
- EACH YEAR OF SMOKING INCREASES INCIDENCE 4%
- SMOKING INCREASES AAA GROWTH RATE 35%
SMOKING AND AAA

- AAA RELATIONSHIP TO CIGARETTE SMOKING 2ND ONLY TO LUNG CANCER

- THE ONLY KNOWN MODIFIABLE RISK FACTOR
AAA IN WOMEN

--- INTACT
___ RUPTURE

AFTER CRONNENWETT
AAA IN WOMEN

- LOWER PREVALENCE UNTIL MENOPAUSE
- OLDER WHEN DIAGNOSED
- FASTER GROWTH RATE
- INCREASED RUPTURE RATE
  - RUPTURE RISK 4X THAT OF MALES
  - RUPTURE AT SMALLER SIZE (5-10 MM)
  - LOWER REPAIR RATES
- INCREASED OPERATIVE MORTALITY
  - OLDER AGE
  - MORE & LESS-WELL CONTROLLED RISK FACTORS
  - SMALLER VESSELS; LESS EVAR
- OVER 40% OF ALL AAA-RELATED DEATHS OCCUR IN WOMEN
AAA MORTALITY: MEN VS WOMEN

ANNUAL IN-HOSPITAL MORTALITY

\*P=0.003; P<.0001; ΔP<.0001

McPHEE ET AL. JVS 2007
AAA IN WOMEN

- INCREASED SUPRA-RENAL INVOLVEMENT
- STRONGER RELATIONSHIP WITH SMOKING
  - DECREASED SMOKING WORLD-WIDE
  - DECREASE SMOKING IN WOMEN < DECREASE IN MEN
  - HIGHER INCIDENCE AAA THAN NON-SMOKING MEN
- STRONGER FAMILY HISTORY
- ALMOST NO FEM / POP ANEURYSMS
- ESTROGEN PROTECTIVE EFFECT
  - DECREASED MACROPHAGE PRODUCTION MMP-9
  - DISAPPEARS AFTER MENOPAUSE
DIABETES AND AAA

- DIABETIC PARADOX
  - 15% AAA PATIENTS HAVE DIABETES
  - LOWER RISK TO DEVELOP AAA (OR 0.8)
  - LOWER RATE OF AAA GROWTH
  - LOWER RUPTURE RISK
METFORMIN AND AAA

- DIABETIC PARADOX
  - DECREASED PREVALENCE
  - DECREASED RUPTURE RATE
  - DECREASED GROWTH
    - ADAM (42%)
    - UKSAT (30%)
    - ITOGA (20%)

ITOGA ET AL. JVS 2019;69;710
FUJIMURA ET AL. JVS 2016;64:46
Editor’s Choice — Metformin Prescription is Associated with a Reduction in the Combined Incidence of Surgical Repair and Rupture Related Mortality in Patients with Abdominal Aortic Aneurysm

Jonathan Golledge, Dylan R. Morris, Jenna Pinchbeck, Sophie Rowbotham, Jason Jenkins, Michael Bourke, Bernard Bourke, Paul E. Norman, Rhonda Jones, Joseph V. Moxon

WHAT THIS PAPER ADDS
Currently there is no drug therapy for abdominal aortic aneurysm (AAA). This cohort study examined the association between metformin prescription and the combined incidence of AAA repair or AAA related mortality (AAA events). Patients with diabetes who were prescribed metformin, but not patients with diabetes who were not prescribed metformin, had a lower incidence of AAA events compared with those without diabetes. A randomised controlled trial is needed to definitively test whether metformin reduces AAA related clinical events in patients with small AAAs.
**METFORMIN & AAA**

**Inflammation:**
- Decreased of B cells, macrophages, CD4 and CD8 T cells accumulation in the aneurysmal wall
- Inhibition of CD36, LOX1, proteins level in total lysate aorta
- Inhibition of serum concentration of MCP-1 and TNFa
- Increased of serum concentration of IL-10

**Extacellular matrix remodelling:**
- Preservation of elastin fibres
- Decreased of matrix metalloproteinases expression

**Protective effects of metformin on AAA**

**Neovangiogenesis:**
- Decreased neovessel density

**Vascular smooth muscle cells (VSMC):**
- Preservation of VSMC

**Relationship between metformin and abdominal aortic aneurysm**
Juliette Raffort, MD, PhD, Reda Hassen Khodja, MD, PhD, Élémé Jean Baptiste, MD, PhD, and Fabien Langroy, MD, PhD, Nice, France

JVS 2020;71:1056-61
LIMIT TRIAL

LIMITING AAA WITH METFORMIN
DOES METFORMIN PREVENT AAA FROM ENLARGING?

- RCT
- 480 NON-DIABETIC PTS
- AAA 35 - 49 MM
- HGBA1C <6.5%
- METFORMIN VS PLACEBO

INFO: RONALD DALMAN (RLD@STANDORD.EDU)  WWW.CLINICALTRIALS.GOV
ETIOLOGIC CLASSIFICATION

- Atherosclerotic
- Degenerative
- Inflammatory / Immunologic
- Infectious (Mycotic)
- Traumatic
- Post-Dissection
- Familial
- Congenital
- Genetic / Connective Tissue
ETIOLOGY OF AAA

ASSOCIATIONS

MAJOR
- AGE
- CIGARETTE SMOKING (CUM SMOKING BURDEN)
- ATHEROSCLOROSIS
- MALE GENDER
- FAMILY HISTORY

LESSER
- PULMONARY EMPHYSEMA / ING HERNIA
- HYPERTENSION
- DIVERTICULAR DISEASE
- CADMIUM
ANEURYSMAL VS OCCLUSIVE DISEASE

DISTINGUISHING CHARACTERISTICS

- Transmural vs Intimal Process
- Localized vs Diffuse
- Entire Circumference vs Eccentric
- Not Associated With
  - Diabetes Mellitus (Diabetic Paradox)
  - Hyperlipidemia
  - Hyperhomocystinemia
ETIOLOGY OF AAA

ESTABLISHED PATHOLOGIC FACTS

- THINNING OF AORTIC WALL
- DECREASED MEDIAL SMC
- DEGRADATION OF STRUCTURAL PROTEINS
- CHRONIC INFLAMMATION OF AORTIC WALL
- INCREASED EXPRESSION OF MATRIX METALLOPROTEASE (MMP 2, 9)
AORTIC LAMELLAR STRUCTURE

COLLAGEN FIBERS

ELASTIN MICROFIBRILS

X 21,000
PATHOGENESIS OF AAA

CURRENT CONCEPTS

- ANEURYSM FORMATION
  - ELASTIN FRAGMENTATION
    » HALF-LIFE 40-70 YEARS
    » NOT SYNTHESIZED IN ADULT AORTA

- ANEURYSM GROWTH AND RUPTURE
  - COLLAGEN DEGRADATION
    » DEPOSITION
    » REMODELING
PATHOGENESIS OF AAA

MMP-9

- DEGRADATES COLLAGEN AND ELASTIN
- ABUNDANT IN AAA WALL, INFLAMMATORY CELLS AND MACROPHAGES.
- INCREASED PLASMA LEVELS IN 50% OF PTS WITH AAA
- LEVELS RETURN TO NORMAL AFTER AAA REPAIR
- AORTIC INFUSION WITH RMMP-9 IN MMP-9 KNOCKOUT MICE PRODUCES AAA
PATHOGENESIS OF AAA

INFLAMMATION

- INFLAMMATION IS PROMINENT FEATURE IN ADVENTITIA OF AAA (MACROPHAGES, T AND B LYMPHOCYTES)
- LEUKOCYTES HAVE INCREASED ELASTASE
- INFLAMMATORY CELLS ELABORATE PROTEOLYTIC ENZYMES AND CYTOKINES THAT MODULATE EXPRESSION OF MATRIX PROTEINS AND PROTEOLYTIC ENZYMES BY MESENCHYMAL AND SM CELLS
- INFLAMMATORY MEDIATORS MAY BE ELABORATED SECONDARILY IN RESPONSE TO CHEMICAL, MECHANICAL OR OTHER INJURY
INTRALUMINAL / MURAL THROMBUS
MURAL THROMBUS

- THICKNESS NOT UNIFORM
- CONTAINS INFLAM CELLS AND MMP-9
- AREAS OF THICKER MT ASSOCIATED WITH LOCAL AAA WALL DEGENERATION AND HIGHER MMP-9

DUCAS ET AL. JVS VASCULAR SCIENCE 2020;1:1190-99
INTRALUMINAL THROMBUS

- CORRELATES WITH MORE RAPID AAA GROWTH
- CONTAINS ↑MMP-9, INFLAMM INFILTRATE
- ↓ELASTIN /COLLAGEN CONTENT
- ↑LOCAL PROTEOLYTIC ACTIVITY

AORTIC WALL

Increased matrix metalloproteinase 9 activity correlates with flow-mediated intraluminal thrombus deposition and wall degeneration in human abdominal aortic aneurysm

Annie A. Ducas, MD, MSc, David C. S. Kuhn, PhD, Lauren C. Bath, BS, Med, Richard J. Litzow, PhD, and April J. Boyd, MD, PhD, Manitoba, Canada
INFLAMMATION AND AAA

PERIVASCULAR FAT IN AAA

- PRO-INFLAM GENE EXPRESSION INCREASED
- ANTI-INFLAM GENE EXPRESSION DECREASED
- STIMULATES SAME IN NORMAL AORTIC SM
PATHOGENESIS OF AAA

• INFLAMMATION
• TRAUMA
• SMOKING
• HEMODYNAMIC STRESS
• OXIDANT INJURY
• AUTOIMMUNITY
• CYTOKINES
• ATHEROSCLEROSIS
• GENETICS

MATRIX DEGRADATION

DYSFUNCTIONAL REMODELING

DECREASE TENSILE STRENGTH
GENETICS AND AAA

- Familial clustering of AAA
- 15-25% of patients undergoing AAA repair have 1st degree relative with AAA (only 2-3% in age-matched controls)
- Over trial: 13.7% had family history of AAA
- Increased incidence of affected relatives in women with AAA (12% vs 7% in men)
- Monozygotic twin (24% prob; 71 x)
- Several suspect genes identified
FAMILIAL AAA

- PATIENTS 5-7 YEARS YOUNGER
- MORE OFTEN WOMEN
- RUPTURE MORE FREQUENTLY
- RUPTURE AT SMALLER SIZE
CLINICAL MANIFESTATIONS

- PAIN
- COMPRESSION OF ADJACENT STRUCTURES
- DISTAL EMBOLIZATION
- THROMBOSIS
- PULSATION
- 2/3 DISCOVERED ON TEST PERFORMED FOR ANOTHER REASON
DIAGNOSIS OF AORTIC ANEURYSM

- PHYSICAL EXAMINATION
- PLAIN X-RAY
  - ABDOMINAL
  - SPINE
- ULTRASOUND
- CT SCAN
- MRI
IMAGING OF AAA
CT SCAN FOR AAA

- DETECTS ANATOMIC VARIATIONS
- DETECTS MURAL THROMBUS / PLAQUE
- DETERMINE WALL STRESS

RETROAORTIC LEFT RENAL VEIN
VENOUS ANOMALIES

ZAMMIT S, CASSAR K. EUR J VASC ENDOVASC SURG (2021) 61, 918
AORTO-CAVAL FISTULA

CT

DSA

CTA

PEVEC WC, ET AL. JVS 2010;51:475
UNUSUAL ABDOMINAL ANEURYSMS

- INFLAMMATORY
- MARFAN’S
- SACCULAR
- INFECTED
DIAGNOSIS OF AAA: CT SCAN

RUPTURE
SCREENING FOR AAA

METHODS

● SERUM BIOMARKERS
  - FIBRINOGEN, D-DIMER, IL-6, CRP
  - MMP-9, TIMP-1
  - APOLIPOPROTEIN-A, APO(a)
  - MFAP4
  - MICRO RNAS

● ULTRASOUND
  - IDENTIFIES ANEURYSMS
  - DETERMINES SIZE
SCREENING FOR AAA

JAMA | US Preventive Services Task Force | RECOMMENDATION STATEMENT
Screening for Abdominal Aortic Aneurysm
US Preventive Services Task Force Recommendation Statement

For men aged 65 to 75 years who have ever smoked: Grade B
Perform 1-time screening for abdominal aortic aneurysm (AAA) with ultrasonography in men who have a history of smoking.

For men aged 65 to 75 years who have never smoked: Grade C
Selectively offer screening to men who do not have a history of smoking, rather than routinely screening all men in this group.

For women who have never smoked and have no family history of AAA: Grade D
Do not screen women who have never smoked and do not have a family history of AAA.

For women aged 65 to 75 years who have ever smoked or have a family history of AAA: I statement
Evidence is Insufficient to assess the balance of benefits and harms of screening for AAA with ultrasonography in women aged 65 to 75 years who have ever smoked or have a family history of AAA.

FAMILY HISTORY ADDED AS FACTOR IN WOMEN

JAMA. 2019;322:211-8
AAA-RELATED DEATHS

- 31-42% WOMEN
- 22% NON-SMOKERS
- 9% MEN < 65 YEARS

NONE INCLUDED IN USPSTF GUIDELINES
SCREENING FOR AAA

USPSTF 2019 vs SVS

- MEN 65-75 YEARS
  - EVER SMOKED B
  - NEVER SMOKED C

- WOMEN 65-75
  - EVER SMOKED, + FAM HX I, SVS YES
  - NEVER SMOKED, NO FAM HX D

- MEN OR WOMEN 65-75
  - FIRST-DEGREE RELATIVES SVS YES
  - > 75 IF IN GOOD HEALTH
ULTRASOUND SCREENING

● DECREASE
  – AAA RUPTURE
  – EMERGENCY SURGERY
  – AAA- RELATED MORTALITY 50%
  – DECREASE ALL- CAUSE MORTALITY +/-

● COST EFFECTIVE

● CONCERNS
  – ONLY 40% ELIGIBLE ARE SCREENED
  – ONLY 65% FOLLOWUP OF POSITIVE SCANS
  – SURVEILLANCE GAPS: 6X RUPTURE RISK

● AAA IN 2.3% OF MEN 4-10 YEARS AFTER SCREEN
ABDOMINAL AORTIC ANEURYSMS

EXPANSION RATE

INITIAL DIAMETER (CM)

- 3.5 - 4.0
- 4.0 - 5.0
- 5.0 - 5.9

ENLARGEMENT RATE (CM / YR)

AVE: 2.2-3.0 MM/YEAR
## SURVEILLANCE INTERVALS

### INITIAL AAA SIZE

<table>
<thead>
<tr>
<th>Size Range</th>
<th>Surveillance Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.0-3.9 cm</td>
<td>24 MONTHS</td>
</tr>
<tr>
<td>4.0-4.9 cm</td>
<td>36 MONTHS *</td>
</tr>
<tr>
<td>5.0-5.4 cm</td>
<td>12 MONTHS</td>
</tr>
<tr>
<td></td>
<td>6 MONTHS</td>
</tr>
</tbody>
</table>

*SVS GUIDELINES 2018

THOMPSON ET AL, JAMA 2013:309:806
REPAIR OF AORTIC ANEURYSMS

DECISION TO OPERATE

- AAA RUPTURE RISK
- ELECTIVE OPERATIVE RISK
  - PATIENT RISK FACTORS
  - SURGEON / HOSPITAL VOLUME
- LIFE EXPECTANCY
- PATIENT PREFERENCE
## SIZE AND AAA RUPTURE

### RISK OF RUPTURE

<table>
<thead>
<tr>
<th>DIAMETER</th>
<th>RR / YR (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.0-5.4</td>
<td>1.0</td>
</tr>
<tr>
<td>5.5-5.9</td>
<td>9.4</td>
</tr>
<tr>
<td>6.0-6.9</td>
<td>10.2</td>
</tr>
<tr>
<td>6.5-6.9</td>
<td>19.1</td>
</tr>
<tr>
<td>&gt;7.0</td>
<td>32.5</td>
</tr>
<tr>
<td>&gt;8.0</td>
<td>26.7 (6 MOS)</td>
</tr>
</tbody>
</table>

Lederle ET AL, JAMA, 2002
ELECTIVE AAA REPAIR

THRESHOLD DIAMETER*

- MEN: 5.5 CM
- WOMEN: 5.0 CM

SVS GUIDELINES, 2018
TREATMENT OF AORTIC ANEURYSM

GOALS

- PREVENT RUPTURE
- PROLONG LIFE
- MAINTAIN QOL
MEDICAL TREATMENT OF AAA

REDUCE EXPANSION RATE

- SMOKING CESSION
- STATINS
- BETA BLOCKADE
  - PROPRANOLOL
- MMP INHIBITION
  - DOXYCYCLINE *
  - ROXITHROMYCIN
- RAS INHIBITORS (ARB, ACE)
- ANTI-INFLAMMATORY DRUGS (MAST CELLS)
- EXERCISE
- ANTI-PLATELET AGENTS

*BAXTER, ET AL. JAMA, 2020; 323: 2029
MEDICAL TREATMENT OF AAA

REDUCE EXPANSION RATE

- MEDITERRANEAN DIET

- CHEMICAL STABILIZATION OF ECM

- NECTERO (ENDOVASC ARTERY STABILIZATION TREATMENT)
TREATMENT OF AAA

MEDICAL MANAGEMENT

- GREEN TEA POLYPHENOL
  - EPICALLOJCATECHIN-3-GALLATE (EGCG)
    » ATTENUATES AAA PROGRESSION
      * REGENERATION OF ELASTIN
      * PREVENTION ELASTIN DEGRADATION

SETOZAKI ET AL, JVS 2017
CIPRO, AAD & AAA

- AORTIC DESTRUCTION IN MICE
  - INCREASE AORTIC DISSECTION
  - INCREASE AAA FORMATION
  - INCREASE DEATH
- HUMANS
  - ACHILLES RUPTURE
  - AORTIC ECM DYSREGULATION → DEGRADATION
- OTHER FLUOROQUINOINALONES
- FDA WARNING (DECEMBER 2018)
GENE THERAPY FOR AAA

LNC RNA H19

- LONG NONCODING RNA: MOLECULAR REGULATORS
- UPREGULATING H19 CORRELATES WITH AORTIC SMC CONTENT AND APOPTOSIS IN ANEURYSMS IN MICE, MINI-PIGS AND HUMAN AORTIC TISSUE
- LOWERING LEVELS ASSOCIATED WITH DECREASED AORTIC SMC APOPTOSIS IN HUMANS
- INHIBITION OF H19 MIGHT BE MOLECULAR THERAPEUTIC TARGET TO DECREASE AAA GROWTH

LI DY. CIRC 2018;138:1551
ABDOMINAL AORTIC ANEURYSM

RUPTURE AND SIZE

- SMALL AAA (<5CM) CAN RUPTURE
- LARGE AAA MAY NOT RUPTURE
- AAA OF SAME DIAMETER MAY HAVE DIFFERENT RUPTURE POTENTIAL
SIZE AND AAA RUPTURE

5.3 CM

25 CM

KRIEVES, JVS 61;2015
SYMPTOMATIC AAA

- **INCIDENCE:** 160/1429 (11%)
- **AAA SIZE:** 6.0-6.3CM
- **SYMPTOMS**

<table>
<thead>
<tr>
<th>SYMPTOM</th>
<th>NUMBER</th>
<th>PERCENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABD PAIN</td>
<td>95</td>
<td>59.4</td>
</tr>
<tr>
<td>BACK PAIN</td>
<td>63</td>
<td>39.3</td>
</tr>
<tr>
<td>AAA TENDERNESS</td>
<td>20</td>
<td>12.5</td>
</tr>
<tr>
<td>EMBOLIC EVENT</td>
<td>14</td>
<td>8.7</td>
</tr>
<tr>
<td>INFLAMM AAA</td>
<td>7</td>
<td>4.4</td>
</tr>
</tbody>
</table>

CHANDRA ET AL, JVS 2017
UN-OPERATED AAA

NATURAL HISTORY

- 154 PATIENTS, HELSINKI UNIV HOSP, 2000-2010
- AAA > 5.5CM DIAMETER
- REASONS FOR NO SURGERY
  - CARDIO-RESP 33%
  - OVERALL CONDITION 33%
  - CANCER 8%
  - PATIENT CHOICE 21%
- CAUSE OF DEATH
  - RUPTURE 66 (43%)
  - 5 / 12 (42%) SURVIVED OPERATION FOR RUPTURE

NORENEN ET AL. EUR J VASC ENDOVASC SURG 2013; 45:326
SURVEILLANCE FREQUENCY

- META-ANALYSIS OF 18 STUDIES
- 15,471 PATIENTS
  - 13,728 MEN
  - 1,743 WOMEN
- AAA < 5.5 CM
- TIME TO REACH THRESHOLD OF 5.5 CM
  - 3.0 CM: 7.4 YEARS
  - 4.0 CM: 3.2 YEARS
  - 5.0 CM: 8 MONTHS
- 4X RISK OF RUPTURE FOR WOMEN

MURAL THROMBUS

- CONTAINS INFLAMMATORY CELLS AND ↑MMP-9
  - PROTEOLYSIS
  - ENZYMATIC WALL DEGENERATION
- ? MARKER OF DECREASED WALL STRENGTH
- MAY EXPLAIN RUPTURE OF SMALL AAA

HALLER ET AL. JVS 2018;67:1051
LAW OF LAPLACE

\[ T = PR \]

**T**: TANGENTIAL STRESS  
**P**: TRANSMURAL PRESSURE  
**R**: RADIUS