Washington State Health Care Authority
Health Technology Assessment

Health Technology Assessment

Program Overview

Josh Morse, Program Director
Health Technology Assessment
September 20, 2013

Presentation Overview

Today's Topics:

- Cardiac Nuclear Imaging
- Carotid Artery Stenting

- HTA Program Overview

Washington State Health Care Authority
Health Technology Assessment

HTA Background

- The Health Technology Assessment Program (HTA) is located within the Health Care Authority (HCA)

- 2006 legislation designed HTA program to use <u>evidence reports</u> and a <u>panel of clinicians</u> to make coverage decisions for certain medical procedures and tests based on evidence of:
  - ➢ Safety
  - ➢ Efficacy/ Effectiveness
  - ➢ Cost-Effectiveness

Washington State
Health Care Authority
Health Technology Assessment

HTA Background

- Multiple state agency programs participate to identify topics and implement policy decisions:
  - Health Care Authority
    - Uniform Medical Plan
    - Medicaid
  - Labor and Industries
  - Corrections

- Implementation:
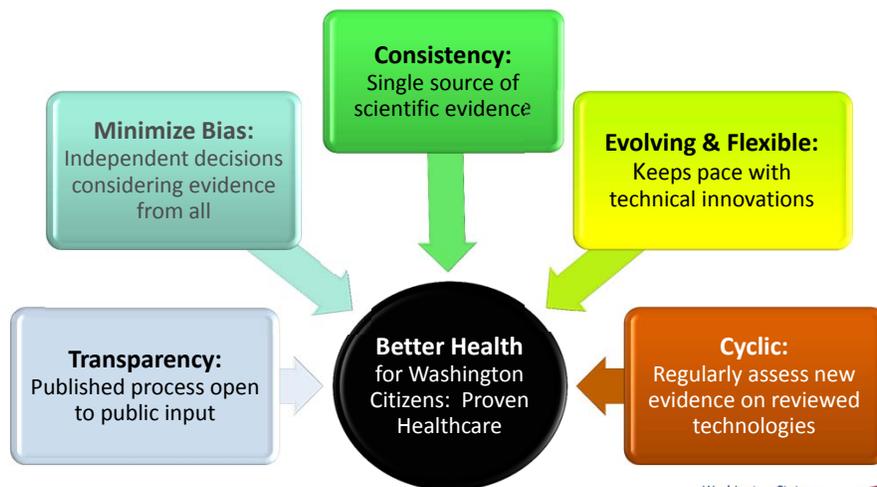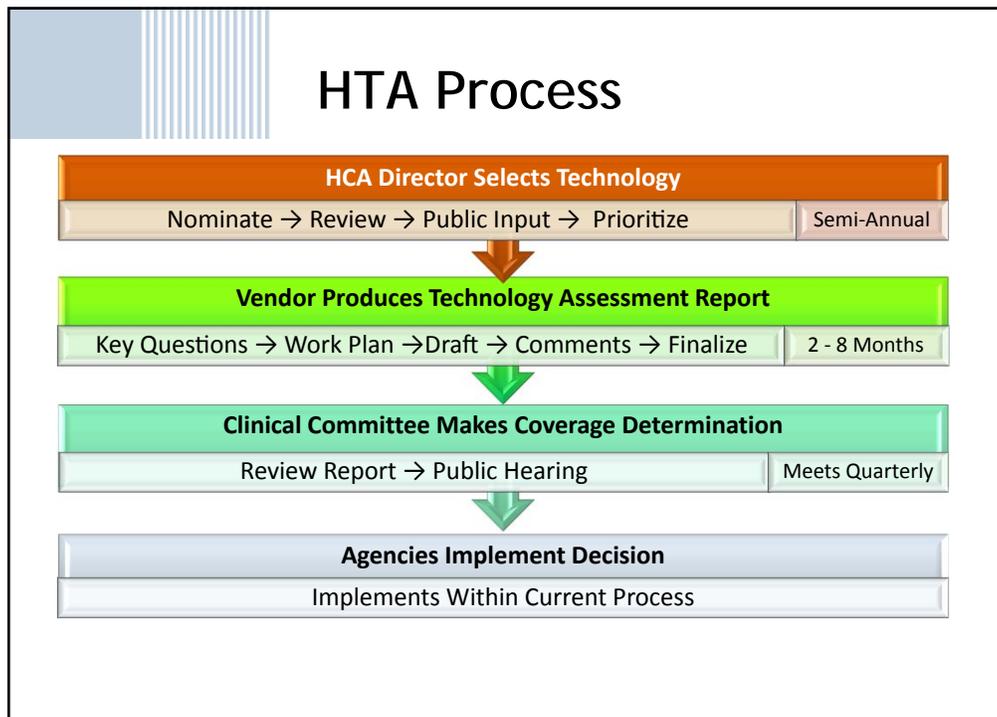 - Agencies implement determinations of the HTA program within their existing statutory framework.

Washington State
Health Care Authority
Health Technology Assessment

Purpose: Pay for What Works

Ensure medical treatments, devices and services paid for with state health care dollars are safe and proven to work.

- Provide resources for state agencies purchasing health care

- Develop scientific, evidence-based reports on medical devices, procedures, and tests.

- Facilitate an independent clinical committee of health care practitioners to determine which medical devices, procedures, or tests meet safety, efficacy, and cost tests.

Washington State Health Care Authority
Health Technology Assessment

Objectives

Consistency: Single source of scientific evidence

Minimize Bias: Independent decisions considering evidence from all

Evolving & Flexible: Keeps pace with technical innovations

Transparency: Published process open to public input

Better Health for Washington Citizens: Proven Healthcare

Cyclic: Regularly assess new evidence on reviewed technologies

Washington State Health Care Authority
Health Technology Assessment

HTA Process

HCA Director Selects Technology	
Nominate → Review → Public Input → Prioritize	Semi-Annual

Vendor Produces Technology Assessment Report	
Key Questions → Work Plan →Draft → Comments → Finalize	2 - 8 Months

Clinical Committee Makes Coverage Determination	
Review Report → Public Hearing	Meets Quarterly

Agencies Implement Decision
Implements Within Current Process

Key Questions

- Is it safe?

- Is it effective?

- Does it provide value (i.e. improve health outcomes)?

Washington State
Health Care Authority
Health Technology Assessment

HTA Values

Transparency: Publish topics, criteria, reports, conduct
open meetings

Best Evidence: Formal, systematic process for review of
selected health care technologies.

Independent Decisions: Committee of practicing clinicians make
decisions that are scientifically based, transparent,
and consistent across state health care purchasing
agencies.

Washington State
Health Care Authority
Health Technology Assessment

HTCC Decision Basis

Clinical Committee decisions must give greatest weight
to most valid and reliable evidence.

- Objective Factors for evidence consideration
  - Nature and source of evidence
  - Empirical characteristics of the studies or trials upon which evidence is
    based
  - Consistency of outcomes with comparable studies

- Additional evaluation factors
  - **Recency** (date of information)
  - **Relevance** (applicability of information to the key questions presented
    or participating agency programs and clients)
  - **Bias** (conflict of interest or political considerations)

Washington State
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Health Technology Assessment

Technology Topics 2013

- Carotid Artery Stenting
- Cardiac Nuclear Imaging
- Hyaluronic Acid/Viscosupplementation (Update)
- Hip Resurfacing (Update)
- Facet Neurotomy
- Non-Pharmacological Treatments for Treatment-Resistant Drepression
- Proton Bean Therapy

Washington State
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Health Technology Assessment

How To Participate

- **Visit** the HTA Web site: www.hta.wa.gov

- **Join** the HTA stakeholder distribution list: shtap@hta.hca.wa.gov
  Stakeholders notified of all program publications and meetings

- **Comment** on:
  - Proposed topics
  - Key questions
  - Draft & final reports
  - Draft decisions

- **Attend** HTCC public meetings
  All meeting materials posted on the web

- **Present** comments at Clinical Committee meetings

- **Nominate** health technologies for review

Washington State
Health Care Authority
Health Technology Assessment

HTA Contact Information

Email: shtap@hca.wa.gov

Website: hta.hca.wa.gov/

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Thank you!

Health Technology Clinical Committee
Date: May 17, 2013
Time: 8:00 am – 5:00 pm
Location: SeaTac Airport Conference Center
Adopted:

Meeting materials and transcript are available on the HTA website at:
http://hta.hca.wa.gov/past_materials.html

HTCC MINUTES

Members Present: C. Craig Blackmore MD, MPH; Marie-Annette Brown PhD, RN; Joann Elmore, MD MPH; Carson E. Odegard DC, MPH; Richard C. Phillips MD, MS, MPH; Seth Schwartz MD, MPH; Michael Souter MB, Ch-B, DA, Christopher Standaert, MD; Kevin Walsh MD

Members Absent: David McCulloch, MD; Michelle Simon PhD, ND

HTCC FORMAL ACTION

1. **Call to Order:** Dr. Blackmore, Chair, called the meeting to order. Sufficient members were present to constitute a quorum.

2. **March 22, 2012, Meeting Minutes:** Chair referred members to the draft minutes; motion to approve and second, and adopted by the committee.

   Action: Nine committee members approved the March 22, 2012 meeting minutes. Two members were absent.

3. **Hyperbaric Oxygen Treatment for Tissue Damage Including Wound Care and Treatment of Central Nervous System Conditions Draft Findings & Decision:** Chair referred members to the draft findings and decision and called for further discussion or objection.

   3. Hyperbaric Oxygen Treatment for Tissue Damage Including Wound Care and Treatment of Central Nervous System Conditions Draft Findings & Decision was approved and adopted by the committee.

   Action: Nine committee members approved the Hyperbaric Oxygen Treatment for Tissue Damage Including Wound Care and Treatment of Central Nervous System Conditions Draft Findings & Decision document. Two members were absent.

4. **Cervical Spinal Fusion Draft Findings & Decision:** Chair referred members to the draft findings and decision and called for further discussion or objection.

   Cervical Spinal Fusion Draft Findings & Decision was approved and adopted by the committee.

   Action: Nine committee members approved the Cervical Spinal Fusion Findings & Decision document. Two members were absent. **Cochlear Implants: Bilateral versus Unilateral**

Scheduled and Open Public Comments:

The Chair called for public comments. Four individuals had scheduled time for public comments:

- Douglas Backous, MD, Swedish Medical Center
- Stacy Watson, MS, CCC-A, Swedish Medical Group
- John K Niparko, MD Chair, American Cochlear Implant Alliance, Tiber Albert Professor Chair, Otolaryngology-Head & Neck Surgery, University of Southern California
- Kathy Sie, MD, Seattle Children's Hospital

Presentation materials and conflict of interest forms are available with May 17 meeting materials.
No open public comments were presented.

Agency Utilization and Outcomes:

Kerilyn Nobuhara MD, MHA, Senior Medical Consultant, Health Care Authority, presented the state agency utilization rates for Cochlear Implants: Bilateral versus Unilateral to the committee. The full presentation is published with May 17 meeting materials.

Vendor Report and HTCC Q & A:

The Chair introduced the clinical expert, Jay Rubinstein, MD, PhD, Virginia Merrill Bloedel Professor and Director, Virginia Merrill Bloedel Hearing Research Center, University of Washington

Teresa Rogstad, MPH of Hayes, Inc, presented the evidence review addressing Cochlear Implants. The full presentation is published with May 17 meeting materials.

Committee Discussion and Decision:

The HTCC reviewed and considered the Cochlear Implants technology assessment report and information provided by the state agencies. They also heard comments from the evidence reviewer, the clinical expert, the public, and agency medical directors. The committee considered all the evidence and gave greatest weight to the evidence it determined, based on objective factors, to be the most valid and reliable.

HTCC Committee Coverage Determination Vote			
	Not Covered	**Covered Unconditionally**	**Covered Under Certain Conditions**
Cochlear Implants: Bilateral versus Unilateral	0	0	9

- *Discussion:* The Chair called for discussion of conditions of coverage for Cochlear Implants following the majority voting for coverage under certain conditions. The following conditions were discussed and approved by a majority of the clinical committee:

- *Limitations of Coverage:*

  Bilateral Cochlear Implants are a covered benefit for patients:
  - Twelve months or older;
  - With bilateral, severe to profound sensorineural hearing loss;
  - Limited or no benefit from hearing aids;

- o Cognitive ability and willingness to participate in an extensive auditory rehabilitation program
- o Freedom from middle ear infection, an accessible cochlear lumen that is structurally suited to implantation, and freedom from lesions in the auditory nerve and acoustic areas of the central nervous system;
- o No other contraindications for surgery;
- o Device used in accordance with the FDA approved labeling.

Non-covered Indications: None.

The committee checked for availability of a Medicare decision. The Centers for Medicare & Medicaid Services (CMS) cover Cochlear Implants for the treatment of bilateral pre- or postlinguistic, sensorineural, moderate-to-profound hearing loss in individuals who demonstrate limited benefit from amplification. For conditions addressed by the CMS National Coverage Determination and the HTA review, the HTCC determination is consistent with the CMS policy.

Chair directed HTA staff to prepare a draft coverage determination document for the topic.

5. **Catheter Ablation Procedures for Supraventricular Tachyarrhythmia (SVTA):**

Scheduled and Open Public Comments: The Chair called for public comments. Two individuals scheduled time for public comments.

- Jeanne Poole, MD, Director, Electrophysiology Division of Cardiology, University of Washington
- Gerhard H. Muelheims, MD, FACC (Did not appear before the committee.)

One individual accompanied Dr Poole and provided open comments:

- Mohan Viswanathan, MD, University of Washington

Presentation materials and conflict of interest forms are available with May 17 meeting materials.

Agency Utilization and Outcomes:

G. Steven Hammond, PhD, MD, MHA, Medical Director, Department of Corrections, presented the state agency utilization rates for Catheter Ablation Procedures for SVTA to the committee. The full presentation is published with May 17 meeting materials.

Vendor Report and HTCC Q & A:

The Chair introduced the clinical expert, Ramakota Reddy, MD, Electrophysiologist, Oregon Cardiology.

Robin Hashimoto, PhD, of Spectrum Research, Inc., presented the evidence review addressing Catheter Ablation Procedures for SVTA. The full presentation is published with May 17 meeting materials.

Committee Discussion and Decision

The HTCC reviewed and considered the Catheter Ablation Procedures for SVTA technology assessment report and information provided by the state agencies. They also heard comments from the evidence reviewer, the clinical expert, the public, and agency medical directors. The committee considered all the evidence and gave greatest weight to the evidence it determined, based on objective factors, to be the most valid and reliable.

HTCC Committee Coverage Determination Vote			
	Not Covered	**Covered Unconditionally**	**Covered Under Certain Conditions**
Catheter Ablation Procedures for SVTA	0	0	9

- *Discussion:* The Chair called for discussion of conditions of coverage for Catheter Ablation Procedures for SVTA following the majority voting for coverage under certain conditions. The following conditions were discussed and approved by a majority of the clinical committee:

- *Limitations of Coverage:*

  For adults with supraventricular tachyarrhythmias, cardiac catheter ablation procedures (radiofrequency or cryoablation) are **covered with conditions**:

  - Reentrant tachycardias (e.g. WPW AVRT AVNRT)
  - Atrial flutter
  - Symptomatic atrial flutter
  - Atrial fibrillation
  - Symptomatic atrial fibrillation
  - Drug therapy is either not tolerated or ineffective

  Catheter Ablation Procedures for SVTA **is not a covered benefit** for other non-reentrant supraventricular tachycardias.

  The committee checked for availability of a Medicare coverage decision. CMS does not have a national coverage determination (NCD) for catheter ablation procedures for supraventricular tachyarrhythmia. The committee determined their decision is similar to several evidence-based guidelines developed by professional Societies and is based on more current evidence.

  The Chair directed HTA staff to prepare a draft coverage determination document for the topic.

  The Chair called for further comments. No further comments on review of Catheter Ablation Procedures for SVTA.

6. **Meeting adjourned.**

Washington State Health Care Authority
Health Technology Assessment

Health Technology Clinical Committee
Draft Findings and Decision

Topic: **Cochlear Implants: Bilateral versus Unilateral**
Meeting Date: **May 17, 2013**
Final Adoption:

Number and Coverage Topic:

20130517A – Cochlear Implants: Bilateral versus Unilateral

HTCC Coverage Determination:

Bilateral Cochlear Implants are a **covered benefit with conditions** consistent with the criteria identified in the reimbursement determination.

HTCC Reimbursement Determination:

Limitations of Coverage

- Age 12 months or older;
- Bilateral severe to profound sensorineural hearing loss;
- Limited or no benefit from hearing aids;
- Cognitive ability and willingness to participate in an extensive auditory rehabilitation program;
- Freedom from middle ear infection, an accessible cochlear lumen that is structurally suited to implantation, and freedom from lesions in the auditory nerve and acoustic areas of the central nervous system;
- No other contraindications for surgery; and
- Device used in accordance with the FDA approved labeling.

Non-Covered Indicators

N/A

Agency Contact Information

Agency	Phone Number
Labor and Industries	1-800-547-8367
Public Employees Health Plan	1-800-200-1004
Washington State Medicaid	1-800-562-3022

Draft

HTCC Coverage Vote And Formal Action:

Committee Decision

Based on the deliberations of key health outcomes, the committee decided that it had the most complete information: a comprehensive and current evidence report, public comments, and agency and state utilization information. The committee concluded that the current evidence on Bilateral Cochlear Implants demonstrates that there is sufficient evidence to cover with conditions.   The committee considered all the evidence and gave greatest weight to the evidence it determined, based on objective factors, to be the most valid and reliable. Based on these findings, the committee voted to cover with conditions Bilateral Cochlear Implants.

Cochlear Implants: Bilateral versus Unilateral Vote

HTCC Committee Coverage Determination Vote			
	Not Covered	**Covered Unconditionally**	**Covered Under Certain Conditions**
Bilateral Cochlear Implants	0	0	9

Discussion

The Chair called for discussion on conditions for use of Bilateral Cochlear Implants due to the majority voting for coverage with conditions. The following conditions were discussed and approved by a majority:

Limitations of Coverage

Covered Conditions

- Age 12 months or older;
- Bilateral severe to profound sensorineural hearing loss;
- Limited or no benefit from hearing aids;
- Cognitive ability and willingness to participate in an extensive auditory rehabilitation program;
- Freedom from middle ear infection, an accessible cochlear lumen that is structurally suited to implantation, and freedom from lesions in the auditory nerve and acoustic areas of the central nervous system;
- No other contraindications for surgery; and
- Device used in accordance with the FDA approved labeling

Non-Covered Indicators

N/A

Action

The committee reviewed the evidence report for existing clinical guidelines and Centers for Medicare & Medicaid Services (CMS) decisions. CMS allows coverage of CI for the treatment of bilateral pre- or postlinguistic, sensorineural, moderate-to-profound hearing loss in individuals who demonstrate limited benefit from amplification.

Draft

The committee Chair directed HTA staff to prepare a Findings and Decision document on Bilateral Cochlear Implants reflective of the majority vote for final approval at the next public meeting.

Health Technology Clinical Committee Authority:

Washington State's legislature believes it is important to use a science-based, clinician-centered approach for difficult and important health care benefit decisions. Pursuant to chapter 70.14 RCW, the legislature has directed the Washington State Health Care Authority (HCA), through its Health Technology Assessment (HTA) program, to engage in an evaluation process that gathers and assesses the quality of the latest medical evidence using a scientific research company and that takes public input at all stages.

Pursuant to RCW 70.14.110 a Health Technology Clinical Committee (HTCC) composed of eleven independent health care professionals reviews all the information and renders a decision at an open public meeting. The Washington State HTCC determines how selected health technologies are covered by several state agencies (RCW 70.14.080-140). These technologies may include medical or surgical devices and procedures, medical equipment, and diagnostic tests. HTCC bases its decisions on evidence of the technology's safety, efficacy, and cost effectiveness. Participating state agencies are required to comply with the decisions of the HTCC. HTCC decisions may be re-reviewed at the determination of the HCA Administrator.

> **Meeting materials and transcript are available on the HTA website at:**
> http://www.hta.hca.wa.gov/past_materials.html

Draft

Cochlear Implants: Bi versus Unilateral

Findings & Decision
Timeline and Overview of Comments

The Health Technology Assessment (HTA) program received comments in response to the posted Health Technology Clinical Committee (HTCC) draft findings and decision on Cochlear Implants: Bi versus Unilateral.

Category	Comment Period June 6 – 21, 2013	Cited Evidence
Patient, relative, and citizen	0	0
Legislator and public official	0	0
Health care professional	0	0
Industry & manufacturer	0	0
Professional society & advocacy organization	0	0
Total	0	0

Comments with Evidence: None.

Comments without Evidence: None.

Technology Assessment Timeline		
Study Stage	**Date**	**Public Comment Days**
Technology recommendations published	November 1, 2011	
Public comments due	**November 15, 2011**	**15**
Selected technologies published	November 29, 2011	
Public comments due	**December 29, 2011**	**30**
Draft Key Questions published	November 15, 2012	
Public comments due	**November 29, 2013**	**30**
Final Key Questions published	January 10, 2013	
Draft report published	March 5, 2013	
Public comments due	**April 5, 2013**	**31**
Final report published	April 17, 2013	
Public meeting date	May 17, 2013	
Findings & decision published	June 6, 2013	
Public comments due	**June 21, 2013**	**16**

Health Technology Clinical Committee
Draft Findings and Decision

Topic: Catheter Ablation Procedures for Supraventricular Tachyarryhthmias (SVTA)
Meeting Date: May 17, 2013
Final Adoption:

Number and Coverage Topic:

20130517B – Catheter Ablation Procedures for Supraventricular Tachyarryhthmias (SVTA)

HTCC Coverage Determination:

Catheter ablation procedures are a **covered benefit with conditions** consistent with the criteria identified in the reimbursement determination.

HTCC Reimbursement Determination:

Limitations of Coverage

For adults with supraventricular tachyarrhythmias , cardiac catheter ablation procedures (radiofrequency or cryoablation) are **covered with conditions**:

- Reentrant tachycardias (e.g. Wolff-Parkinson-White Syndrome (WPW), Atrioventricular reentrant tachycardia (AVRT), Atrioventricular nodal reentrant tachycardia (AVNRT)
- Atrial flutter:
    - Symptomatic atrial flutter
- Atrial fibrillation:
    - Symptomatic atrial fibrillation
    - Drug therapy is either not tolerated, or ineffective.

Non-Covered Indicators

- Other, non-reentrant supraventricular tachycardias

Agency Contact Information

Agency	Phone Number
Labor and Industries	1-800-547-8367
Public Employees Health Plan	1-800-200-1004
Washington State Medicaid	1-800-562-3022

Draft

HTCC Coverage Vote And Formal Action:

Committee Decision

Based on the deliberations of key health outcomes, the committee decided that it had the most complete information: a comprehensive and current evidence report, public comments, and agency and state utilization information. The committee concluded that the current evidence on catheter ablation procedures demonstrates that there is sufficient evidence to cover with conditions. The committee considered all the evidence and gave greatest weight to the evidence it determined, based on objective factors, to be the most valid and reliable. Based on these findings, the committee voted to cover with conditions catheter ablation procedures for supraventricular tachyarryhthmias (SVTA).

Catheter Ablation Procedures for SVTA Coverage Vote

HTCC Committee Coverage Determination Vote			
	Not Covered	**Covered Unconditionally**	**Covered Under Certain Conditions**
Catheter Ablation Procedures	0	0	9

Discussion

The Chair called for discussion on conditions for use of catheter ablation procedures for supraventricular tachyarryhthmias (SVTA) due to the majority voting for coverage with conditions. The following conditions were discussed and approved by a majority:

Limitations of Coverage

Covered Conditions:

For adults with supraventricular tachyarrhythmias , cardiac catheter ablation procedures (radiofrequency or cryoablation) **are covered with conditions**:

- Reentrant tachycardias (e.g. Wolff-Parkinson-White Syndrome (WPW), Atrioventricular reentrant tachycardia (AVRT), Atrioventricular nodal reentrant tachycardia (AVNRT)
- Atrial flutter
   - Symptomatic atrial flutter
- Atrial fibrillation
   - Symptomatic atrial fibrillation
   - Drug therapy is either not tolerated, or ineffective.

Non-Covered Indications:

Other non-reentrant supraventricular tachycardias

Draft

Action

The committee reviewed the evidence report for existing clinical guidelines and Centers for Medicare & Medicaid Services (CMS) decisions. CMS does not have a national coverage determination (NCD) for catheter ablation procedures for supraventricular tachyarryhthmias.

The committee Chair directed HTA staff to prepare a Findings and Decision document on catheter ablation procedures reflective of the majority vote for final approval at the next public meeting.

Health Technology Clinical Committee Authority:

Washington State's legislature believes it is important to use a science-based, clinician-centered approach for difficult and important health care benefit decisions. Pursuant to chapter 70.14 RCW, the legislature has directed the Washington State Health Care Authority (HCA), through its Health Technology Assessment (HTA) program, to engage in an evaluation process that gathers and assesses the quality of the latest medical evidence using a scientific research company and that takes public input at all stages.

Pursuant to RCW 70.14.110 a Health Technology Clinical Committee (HTCC) composed of eleven independent health care professionals reviews all the information and renders a decision at an open public meeting. The Washington State HTCC determines how selected health technologies are covered by several state agencies (RCW 70.14.080-140). These technologies may include medical or surgical devices and procedures, medical equipment, and diagnostic tests. HTCC bases its decisions on evidence of the technology's safety, efficacy, and cost effectiveness. Participating state agencies are required to comply with the decisions of the HTCC. HTCC decisions may be re-reviewed at the determination of the HCA Administrator.

> **Meeting materials and transcript are available on the HTA website at:**
> http://www.hta.hca.wa.gov/past_materials.html

Draft

Catheter Ablation Procedures for Supraventricular Tachyarrhythmia Including Atrial Flutter, Atrial Fibrillation

Findings & Decision
Timeline and Overview of Comments

The Health Technology Assessment (HTA) program received comments in response to the posted Health Technology Clinical Committee (HTCC) draft findings and decision on Catheter Ablation Procedures for Supraventricular Tachyarrhythmia Including Atrial Flutter, Atrial Fibrillation.

Category	Comment Period June 7 – June 21, 2013	Cited Evidence
Patient, relative, and citizen	0	0
Legislator and public official	0	0
Physician & health care professional	13	5
Industry & manufacturer	0	0
Professional society & advocacy organization	0	0
Total	13	5

Comments with Evidence:

Physician & Health Care Professional

Eteri Byazrova, MD, Kootenai Heart Clinics Northwest

Patients with ectopic atrial tachycardias have debilitating symptoms. Medical therapy is ineffective for most of them. Without curative ablation, patients may make more trips to the Emergency Room, be forced to seek disability or develop-tachycardia-induced congestive heart failure.

For over 60% of patients, antiarrhythmic drugs are ineffective, not tolerated, or contraindicated due to safety concerns. Other risks of medications include drug toxicity , proarrhythmia, and sudden death.

Atrial fibrillation was traditionally considered a non-life threatening condition. An AFFIRM study of atrial fibrillation patients randomized to two groups, rate control or rhythm control with antiarrhythmic drugs, showed antiarrhythmic drugs helped maintain sinus rhythm in about 30% of patients. It also determined do difference in mortality rates between the two groups. However, follow-up analysis revealed a 0.54 hazard ratio for patients in the sinus rhythm group and 1.41 for patients on rhythm control drugs. The beneficial effect of sinus rhythm maintenance drugs was offset by the toxic effects of the medications, resulting in no overall mortality difference between the rate control and rhythm control groups.

Two prospective studies showed regression of LV dysfunction and congestive heart failure in atrial fibrillation patients, and the benefit was present in patients with appropriate rate contral at baselaine.

One randomized trial demonstrated superiority of catheter ablation t biventricular pacing/ ablation of the AV node in patients with congestive heart failure and atrial fibrillation. At least one study has addressed the issue of cost. Catheter ablation as a first first-line therapy was compared to antiarrhythmic drug trail as first line, and at two years, cumulative cost was the same in both groups.

Harold Goldberg, MD, FACC, Providence Spokane Heart Institute, Spokane Cardiology

The Committee should not restrict procedures that have well-demonstrated clinical benefit. Atrial tachycardia represents 5% or less of episodes of rapid heart rhythms. In addition to symptoms of rapid beating, patients can occasionally have episodes of tachycardic-dependent cardiomyopathy – weak heart muscle as a result of heart racing.

A study by Dr. Shih-Ann Chen of China demonstrated efficacy-98% cure of atrial tachycardia. In the study of 36 patients, 7 out of 36 had automatic atrial tachycardia, 9 out of 36 had triggered atrial tachycardia. Therefore, 20 patients had reentrant tachycardia and in summary 16 had non-reentrant tachycardia. The study demonstrated efficacy in both groups.   It would not be appropriate to isolate non-reentrant tachycardia patients for non-reimbursement when ablation has been demonstrated to be effective.

Harold A Kwasman, MD, FACC, Providence Spokane Heart Institute, Spokane Cardiology

Non-reentrant arrhythmias are equally amenable to ablative therapy. This is frequently the only treatment to control these arrhythmias, since many are not effectively treated pharmacologically.

A non-reentrant arrhythmia reflects atrial myocardial cells that fire automatically. This is usually a very localized focal event and with appropriate mapping techniques, electrophysiologists are able to identify that focus and ablate the rogue cells, rendering the patient free of their arrhythmia.

Often times, patients with these ectopic atrial tachycardias develop secondary cardiomyopathy. Controlling this arrhythmia often results in normalization of the patient's heart function.

Jordan Prutkin, MD, MHS, FHR, Division of Cardiology/ Electrophysiology, University of Washington

Ablation for atrial tachycardia is considered standard of care for patients who have failed antiarrhythmic drug therapy. Guidelines from the US and European cardiology societies say that catheter ablation for recurrent, symptomatic, focal atrial tachycardia is a Class I recommendation. It is also a class I recommendation for asymptomatic or symptomatic incessant atrial tachycardia. Success rate for this procedure, according to a pooled analysis, was 86%.

Mohan Viswanathan, MD, FACC, Assistant Professor of Medicine, Division of Cardiology/Cardiac Electrophysiology, University of Washington School of Medicine

Agrees with the document in its overall spirit and identifies one omission – focal atrial tachycardia. When a focal atrial tachycardia is left unchecked, this arrhythmia can degenerate into atrial fibrillation. With catheter ablation for the dominant focal atrial tachycardia, risks of morbidity and mortality are reduced and the patient escapes the perils of atrial fibrillation later in life.

Comments without Evidence:

Physician & Health Care Professional

Chris Anderson, MD, Electrophysiologist, Providence Center for Congenital Heart Disease

There are many studies documenting poor response to medical therapy for these tachyarrhythmias as well as a high cure rate with catheter ablation. It would fall well short of the standard of care to try to manage these patients with medication alone. He is also concerned about the lack of inclusion of intraatrial reentry tachycardia (also termed atrial reentry tachycardia) in the covered conditions.

Mark Harwood, MD

Shocked to hear that Committee concluded there is no benefit to the ablation of non-reentrant atrial arrhythmias.   Beyond the example of individuals that receive a potentially life altering treatment, there is plentiful data to support the ongoing practice of ablation. The field of electrophysiology is driven by data. We, as Electrophysiologists, support evidence based medicine backed by clinical trials. Cardiology and its subspecialties have for years led the way for evidence-based medicine based on data. The data is there for review. It is regretful that this conclusion was made.

Alan Heywood, MD, Overlake Medical Clinics, Overlake Hospital Medical Center

Expresses deep concern regarding the proposed decision. Ablation of cardiac arrhythmias provides life-changing and at times, a life-saving, cure for cardiac tachyarrhythmias in many patients. Excluding atrial tachycardia from ablation therapy, means patients will not be able to receive the life-saving, curative treatment that is readily available. This would be an unconscionable restriction on our ability to provide appropriate medical care.

Benjamin John, MD, The Vancouver Clinic, Department of Cardiology

Analysis of data demonstrates a time bias based on when these studies were performed.   Much of the understanding of the validity of randomized trial data and the validity analysis methods of them came to be appreciated and widely applied in the last decade. Many of the sentinel studies addressing the efficacy of catheter ablation for re entrant and non re entrant SVTs were undertaken prior to the modern understanding of an appropriately designed randomized control trial. Thus many articles are not considered as valid evidence.

There is much more accepted trial data on the efficacy of atrial fibrillation ablation, a procedure developed within the last decade. This gives the appearance that atrial fibrillation ablation is more efficacious than SVT ablation which runs contradictory to current understanding of the relative efficacies of these procedures among current electrophysiology practitioners.

Timothy J. Lessmeier, M.D., FACC, Electrophysiologist, Heart Clinics Northwest

Patients who present with SVT who have debilitating symptoms despite medical therapy are often offered EP study and ablation as a curative therapy alternative. We do not know the mechanism of

their SVT before hand and while the majority have reentrant SVT, approximately 5% have automatic types of atrial tachyarrhythmias, also often curable with ablation.

A diagnostic study and ablation are typically performed in the same setting. Under your current proposed criteria, if we found a patient who had an atrial tachycardia and not reentry, we would not be able to pursue curative therapy. Additionally, there are other rhythms such as atrial fibrillation that are not due to reentry which are cured with ablation. These patients would be excluded from curative therapy.

Gerhard H. Muelheims M.D., Director Cardiac Electrophysiology, Providence Sacred Heart Hospital

Surprised by the decision to not cover non-reentrant tachycardias. When an electrophysiology study is performed and a clinical arrhythmia is induced, the ablation almost always occurs at the same time. The exact mechanism of the arrhythmia is not known until the arrhtyhmia is induced and diagnostic pacing maneuvers performed. Inducing a clinical arrhythmia and not ablating it would be a disservice to patients.

The decision to not cover ablation of non-reentrant tachycardias is not standard of care and would lead to increased patient morbidity and higher cost.

Robert D Swenson MD, FACC, The Vancouver Clinic

Consider revising your recent decision to exclude "other, non-reentrant SVTs" from coverage for catheter ablation. These rhythm disorders account for a relatively small proportion of patients (5-15%), but they affect individuals of all ages, are often sustained, highly symptomatic and difficult to treat with antiarrhythmic drug therapy. They have a significant impact on patients and their families. Excluding these patients from catheter ablation therapy seems arbitrary and short sighted.

Robert Wark, MD

Puzzled and disappointed in the decision not to cover ablation for non-reentrant SVT mechanisms. This would specifically preclude ablation for other forms of atrial tachycardia.

Technology Assessment Timeline		
Study Stage	**Date**	**Public Comment Days**
Technology recommendations published	November 1, 2011	
Public comments due	**November 15, 2011**	**15**
Selected technologies published	November 29, 2012	
Public comments due	**December 29, 2012**	**32**
Draft Key Questions published	July 20, 2012	
Public comments due	**August 10, 2012**	**22**
Final Key Questions published	September 25, 2012	
Draft report published	March 4, 2013	
Public comments due	**April 5, 2013**	**32**
Final report published	April 17, 2012	
Public meeting date	May 17, 2013	
Findings & decision published	June 7, 2013	
Public comments due	**June 21, 2013**	**15**

Masters, Christine V. (HCA)

From: Wark, Robert S <Robert.Wark2@providence.org>
Sent: Thursday, June 13, 2013 11:13 AM
To: HCA ST Health Tech Assessment Prog
Subject: Coverage for SVT ablation

Follow Up Flag: Follow up
Flag Status: Completed

It is my understanding that HCA has reached a decision regarding coverage for catheter ablation for SVT. I am puzzled and disappointed in the decision not to cover ablation for non-reentrant SVT mechanisms. This would specifically preclude ablation for other forms of atrial tachycardia. Atrial tachycardia ablation is a less common ablation procedure but when clinically present oftentimes does not respond well at all to attempts at medical therapy. My personal approach in these patients has been to initially attempt suppression with medical therapy but to consider ablation if ineffective or not well tolerated, much the same as approach for atrial fibrillation. From my perspective I do not think that your decision of non-coverage for this type of ablation is in the best interest of our patients and should certainly be reconsidered. To the best of my knowledge, there are no reputable electrophysiology institutions who would argue that ablation for atrial tachycardia should not be done.

Please take these comments into consideration for your final decision.

Sincerely,

Robert Wark MD

Heart Clinics NORTHWEST

A division of
Kootenai Medical Center

William R. Bennett, MD
Eteri S. Byazrova, MD
Donald A. Chilson, MD
Dennis B. Cooke, MD
John P. Everett, MD
Angelo S. Ferraro, MD
Ronald M. Fritz, DO
Carl L. Hanson, MD

R. Dean Hill, MD
Michael D. Hostetler, MD
Marek Janout, MD
Ronald D. Jenkins, MD
Keith A. Kadel, MD
Kevin M. Kavanaugh, MD
Timothy J. Lessmeier, MD
Eric C. Orme, MD

G. Scott Smith, MD
Wolfgang J.T. Spyra, MD
Eric D. Stucky, MD
Stephen T. Thew, MD
L. Douglas Waggoner Jr., MD
Michael P. Williams, MD

June 17, 2013

Washington State Health Care Authority
SHTAP@HCA.wa.gov

Dear Sirs:

I am responding to a public comment regarding catheter ablation procedures for supraventricular tachy arrhythmia (SVTA) including atrial flutter, atrial fibrillation. It has come to my attention that the Washington State Health Care Authority is proposing limiting catheter ablation for SVT to reentry arrhythmias only. I believe this approach is based on flawed logic and will limit appropriate curative therapy to many Washington residents with heart rhythm problems. I am a practicing electrophysiologist in Spokane, Washington. Patients who present with SVT who have debilitating symptoms despite medical therapy are often offered EP study and ablation as a curative therapy alternative. We do not know the mechanism of their SVT before hand and while the majority have reentrant SVT, approximately 5% have automatic types of atrial tachy arrhythmias, also often curable with ablation. A diagnostic study and ablation are typically performed in the same setting. Under your current proposed criteria, if we found a patient who had an atrial tachycardia and not reentry, we would not be able to pursue curative therapy. Additionally, there are other rhythms such as atrial fibrillation that are not due to reentry which are cured with ablation. These patients would be excluded from curative therapy. In both of these two instances I have raised, I believe you will find the literature supports that cost for ongoing medical therapy will be higher than with curative ablation therapy, let alone the quality of life benefits to the affected person. It is my hope that you will strongly reconsider this decision. I do not believe this decision will foster savings and it appears to be directly opposed to evidence-based medicine and good medical practice.

Please don't hesitate to contact me if you wish further comments.

Sincerely,

Timothy J. Lessmeier, M.D., FACC
Electrophysiologist, Heart Clinics Northwest
TJL/ch

cc: Gerhard Muelheims, MD
        Eteri S. Byazrova, MD
        Donald A. Chilson, MD, FACC
        Jeanne Poole, MD- University of Washington

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From:	Ben John MD <bjohn@tvc.org>
Sent:	Monday, June 17, 2013 11:40 PM
To:	HCA ST Health Tech Assessment Prog
Subject:	Noncoverage on non re entrant tachycardias

Follow Up Flag:	Follow up
Flag Status:	Completed

I am writing to voice my concern over the determination Health Technology Clinical Committee preliminary determinations of non coverage for non re entrant tachycardias. I feel the preliminary analysis lacks a fundamental mis understanding of modern management of focal autonomic atrial tachycardias and SVTs in general. My general take on the analysis of data used on assessing the role of catheter ablation in tachyarrhythmias demonstrates a time bias in when these studies were perform. Much of the understanding on the validity of randomized trial data and the validity analysis methods of them have come to be appreciated and widely applied in the last decade. Many of the sentinel studies addressing the efficacy of catheter ablation for re entrant and non re entrant SVTs predate this methodology. The modern understanding of an appropriately designed randomized control trial was not as desseminated in the realm of academic research at the time the original studies addressing the efficacy of SVT ablation were undertaken. Thus many of the sentinel articles addressing the now modern and commonly practiced procedures for treatment of SVT both re entrant and non re entrant having been improperly discarded or not considered as valid evidence. A prime example of this time bias is the fact that there is much more accepted trial data on the efficacy of atrial fibrillation ablation, a procedure developed within the last decade for which trial design reflect current accepted validity standards compared to a relative dearth of trial data on SVTs. This gives the appearance that atrial fibrillation ablation is more efficacious than SVT ablation which runs contradictory to current understanding of the relative efficacies of these procedures among current electrophysiology practitioners. Abandonment of ablation of "non re entrant" tachycardias, more appropriately focal autonomic tachycardias, would be a literal step back in time in the management of these arrhythmias. If applied, I feel the committee would soon discover the error of this analysis when faced with both patient and physician demands for this procedure trying to cope with the highly symptomatic nature of these arrhythmias ineffectively or at times unsafely treated with drug therapy alone.

Thank you for your consideration

Benjamin John, MD

The Vancouver Clinic, Department of Cardiology

700 NE 87th AVE

Vancouver, WA 98664

From:	Alan Heywood, MD <Alan.Heywood@overlakehospital.org>
Sent:	Thursday, June 20, 2013 2:49 PM
To:	HCA ST Health Tech Assessment Prog
Subject:	Public Comment for: Catheter Ablation Procedures for Supraventricular Tachyarrhythmia (SVTA) Including Atrial Flutter, Atrial Fibrillation

Follow Up Flag:	Follow up
Flag Status:	Completed
Categories:	Red Category

Dear Washington State Health Care Authority,

I am writing with deep concern regarding the proposed decision to exclude ablation as an appropriate therapy for atrial tachycardias. Ablation of cardiac arrhythmias provides life changing, and at times life saving, cure of cardiac tachyarrhythmias for many patients.

Atrial tachycardia is a potentially dangerous arrhythmia that is commonly refractory to medical therapy. Uncontrolled atrial tachycardia leads to dilated cardiomyopathy, associated heart failure, and ultimately a high risk of death in some patients.

I would like to share the story of a young native American woman who was pregnant when she developed persistent focal atrial tachycardia. She was hospitalized for a week at another hospital with her heart rate persistently elevated at 160-180 beats per minute. Multiple medications were tried to no avail. The persistently elevated heart rate placed both the young mother to be and her fetus at substantial risk. She was transferred to our hospital by medical air transport for definitive treatment of her atrial tachycardia.

She underwent a successful ablation of her focal atrial tachycardia. She was discharged from the hospital the following day at returned to her home. The remainder of her pregnancy was uncomplicated and she delivered a healthy baby.

If the Health Care Authority proceeds with the proposal to exclude atrial tachycardia from ablation therapy, patients such as this will not be able to receive the life saving, curative treatment that is readily available. This would be an unconscionable restriction on our ability to provide appropriate medical care.

Please reconsider the preliminary decision to restrict coverage of ablation for focal atrial tachycardia. It is clearly in the best interest of the Washington State residents covered by this policy to have access to this curative procedure.

Sincerely,

J. Alan Heywood, MD
Overlake Medical Clinics
Overlake Hospital Medical Center
Bellevue, WA

Masters, Christine V. (HCA)

From:	Anderson, C Chris <Charles.Anderson2@providence.org>
Sent:	Thursday, June 20, 2013 3:58 PM
To:	HCA ST Health Tech Assessment Prog
Cc:	gmuelheims@spokanecardiology.com
Subject:	HTCC reimbursement of SVT ablation

Follow Up Flag:	Follow up
Flag Status:	Completed

Dear Sirs,

I am writing to express deep concern about a recently drafted policy statement regarding reimbursement of cardiac catheter ablation of supraventricular tachyarrhythmias. Specifically, the draft document states that "Other, non-reentrant supraventricular tachycardias" are not covered. In my experience of 11 years in electrophysiology practice, I have done many cardiac catheter ablations on patients who presented with incessant focal atrial tachycardia (also termed atrial ectopic tachycardia or ectopic atrial tachycardia). In virtually every case, the patient had an incessantly elevated heart rate associated with ventricular dysfunction or dilation, both indicators of tachycardia-induced cardiomyopathy, and some of whom had early evidence of congestive heart failure caused by the arrhythmia. There are many studies documenting poor response to medical therapy for these tachyarrhythmias as well as a high cure rate with catheter ablation. It would fall well short of the standard of care to try to manage these patients with medication alone. I am happy to provide specific references in peer-reviewed literature if you require it.

I am also concerned about the lack of inclusion of intraatrial reentry tachycardia (also termed atrial reentry tachycardia) in the covered conditions. Many would term this condition "atrial flutter", but that term is often inaccurate despite being quite similar mechanistically. Please refer to revised EP codes currently in development for reference.

I would appreciate your prompt attention to these matters. Please do not hesitate to contact me with any questions relating to these matters. My contact information is below.

Sincerely yours,

Chris Anderson, MD
Electrophysiologist, Providence Center for Congenital Heart Disease
101 W. 8th Ave, Ste 4300
Spokane, WA 99204
509-474-6707
Email: Charles.anderson2@providence.org

From:	Viswanathan, Mohan <viswanam@cardiology.washington.edu>
Sent:	Friday, June 21, 2013 6:28 AM
To:	HCA ST Health Tech Assessment Prog
Subject:	Comment on: WA HCA Health Technology Assessment Decision on Catheter Ablation of Supraventricular Tachycardia

Follow Up Flag:	Follow up
Flag Status:	Completed

Dear WA HCA, HTA Committee on Catheter Ablation of Supraventricular Tachycardia,

I wanted to take this opportunity to comment on the decision that was made re: Catheter Ablation for Supraventricular Tachycardia. I acknowledge and thank the committee for having the hearing on May 17, 2013, which I attended and provided commentary with my colleague, Jeanne Poole, MD from the University of Washington.

I agree with the document in its overall spirit and I think the committee for developing the document and it's clarity. There is one omission that I wanted to bring to the attention of the committee, that is the form of supraventricular tachycardia (SVT) that we refer to as Atrial Tachycardia, namely "Focal" Atrial Tachycardia. There are three mechanisms of arrhythmias, whether they arise in the atrium or ventricle and these are:
Myocardial reentry, triggered activity, and increased automaticity. The latter two mechanisms, triggered activity and increased automaticity are the mechanisms by which focal arrhythmias develop, i.e., an errant site of myocardial cells that fires off out of control because of local properties of the heart muscle at that site. This type of behavior in certain circumstances leads to what are described as focal arrhythmias. These arrhythmias, namely focal atrial tachycardias and focal ventricular tachycardias, arise from repetitive firing from this site and result in either an SVT coming from that errant site in the atrium, or ventricular tachycardia, VT, from a focal site in the ventricle.

There has been much work published on the mechanisms of arrhythmias, and focal arrhythmias can cause as much morbidity and mortality at times as reentrant arrhythmias. Many times, focal atrial tachycardias arise in young individuals, pregnant women, patients with prior cardiac surgery, patients with congenital heart defects. These individuals cannot be placed on lifelong medications to control these arrhythmias due to these medications' side effects, and furthermore, medications for the most part, are only so good at suppressing these focal sites from firing off, and inevitably, patients breakthrough the medications with incessant and refractory SVT.

Our success rates for catheter ablation of focal SVTs are quite good. With modern-day mapping systems, and with good electrophysiology recording equipment, a focal atrial tachycardia can be targeted and mapped and terminated with success rates on the order of 90%-95%, quite akin to our success rates for reentrant SVTs.

Furthermore, focal atrial tachycardias many times are seen in patients with atrial fibrillation and/or atrial flutter, and in those situations, sometimes these focal atrial tachycardias destabilize and degenerate into atrial fibrillation, the chaotic arrhythmia that the committee has read a lot about. Thus, many times, when a focal atrial tachycardia is identified, we offer catheter ablation because if left unchecked, this arrhythmia can degenerate into atrial fibrillation, and the more this happens, then the patient is suffering from atrial fibrillation, which in some ways is more difficult to treat. Thus, the morbidity and mortality faced by atrial fibrillation patients can sometimes be averted if there is recognition of a focal atrial tachycardia as the trigger of atrial fibrillation, and by performing catheter ablation for the dominant focal atrial tachycardia, the patient escapes the perils of atrial fibrillation later in life.

In addition, incessant focal atrial tachycardias can sometimes lead to an entity called, tachycardia-induced cardiomyopathy. This is a weakening of the heart muscle that leads to congestive heart failure, and all the problems

that accompany that diagnosis. It is the incessant atrial tachycardia with heart rates above 100 bpm, that ultimately leads to a weakening of the heart muscle. This affects more younger patients and once catheter ablation is performed, the cardiomyopathy, or weakening of the heart can in some cases recover, with an encouraging end to the all the congestive heart failure, the numerous hospitalizations, and all the drain on the health care system seen from that one diagnosis, congestive heart failure.

I have included a few articles for your review here. There are many more, and I would encourage you to consider the articles published by the group from Melbourne, Australia led by Dr. Jonathan Kalman and Dr. Peter Kistler. They have done a large amount of work on focal atrial tachycardias, their characteristics, their prevalence, their natural history, course, and also catheter ablation of these arrhythmias and how this is done. The October 2012 Lancet article by Lee, Sanders and Kalman is a great article of the state of the art of catheter ablation and goes into details of focal atrial tachycardias and how they can be ablated.

I would strongly ask the committee to reconsider the coverage omission of non-reentrant SVTs as focal atrial tachycardias are an important entity in this group that is being omitted. This form of SVT has been targeted for ablation for over two decades, with remarkable success and it has had a major impact on the lives of many patients in the US and in the state of Washington over the years. The intolerance to medications, the lack of efficacy of medications for this arrhythmia, and not to mention the tendency of this arrhythmia to affect younger individuals, tends to drive these patients to seek catheter ablation strategies, and these are some of our biggest success stories as an electrophysiologist. To be able to offer a 20 year-old college student, or 25 year-old expectant mother a catheter ablation procedure to rid them of an incessant focal atrial tachycardia so that they don't have to be exposed to the years of potential medical therapies, is a boon for these individuals. When they are able to go on and live a normal life after their SVT is cured by catheter ablation, one sees the benefit of this type of approach for numerous individuals across the state.

Please do not hesitate to contact me for any questions or comments at: viswanam@uw.edu; or by phone at: 206-543-3068.

Thank you for this opportunity to provide commentary on your proceedings.

With best regards,

Mohan

P.S. – Here are the articles that I have included for your review. There are numerous others, but these are potentially helpful in your considerations and decisions re: Catheter ablation of SVT:

Incessant atrial tachycardia: Cause or consequence of heart failure, and the role of radiofrequency ablation. Chahadi FK, Singleton CB, McGavigan AD.

Int J Cardiol. 2013 Jul 1;166(3):e77-9. doi: 10.1016/j.ijcard.2013.01.056. Epub 2013 Feb 13. No abstract available.

Catheter ablation of atrial arrhythmias after cardiac transplantation: findings at EP study utility of 3-D mapping and outcomes. Nof E, Stevenson WG, Epstein LM, Tedrow UB, Koplan BA.

J Cardiovasc Electrophysiol. 2013 May;24(5):498-502. doi: 10.1111/jce.12078. Epub 2013 Feb 6.

Left septal atrial tachycardias: electrocardiographic and electrophysiologic characterization of a paraseptal focus. Wong MC, Kalman JM, Ling LH, Medi C, Teh A, Lee G, Kumar S, Morton JB, Kistler PM.

J Cardiovasc Electrophysiol. 2013 Apr;24(4):413-8. doi: 10.1111/jce.12049. Epub 2012 Dec 17.

Atrial flutter, atrial fibrillation, and other primary atrial tachycardias.

Benditt DG, Benson DW Jr, Dunnigan A, Gornick CC, Anderson RW. Med Clin North Am. *1984 Jul;68(4):895-918.*

Catheter ablation of atrial arrhythmias: state of the art. Lee G, Sanders P, Kalman JM.

Lancet. 2012 Oct 27;380(9852):1509-19. doi: 10.1016/S0140-6736(12)61463-9. Review.

Thank you,

Mohan Viswanathan, MD

_____
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Masters, Christine V. (HCA)

To: Health Technology Clinical Committee

From: Gerhard H. Muelheims M.D.
      Director Cardiac Electrophysiology
      Providence Sacred Heart Hospital
      Spokane, WA 99204

I am writing to address the HTCC Reimbursement Determination in regards to Catheter Ablation of Supraventricular Tachyarrhythmias.

I phoned in and listened to the session on May 17, 2013 and frankly surprised by the decision to not cover non-reentrant tachycardias.

This decision seemed to be based on the fact that the literature regarding ablation of atrial tachycardia's (a non-reentrant tachycardia)

wasn't reviewed or presented.   I have been an electrophysiologist for over 15 years and have done hundreds of ablations. When an

electrophysiology study is performed and a clinical arrhythmia is induced, the ablation almost always occurs at the same time.

The exact mechanism of the arrhythmia is not known until the arrhtyhmia is induced and diagnostic pacing maneuvers performed.

Once the mechanism is known, the ablation follows. To induce a clinical arrhythmia and to not ablate it would be doing a disservice

to our patients. (why would you do an EP study, find an ablatable arrhythmia, and not do a procedure that has a high chance of success)

In effect, making the diagnosis and then treating the patient with potentially toxic long term medications that are likely not to work.

The decision to not cover ablation of non-reentrant tachycardias is not standard of care and would lead to increased patient morbidity and

higher cost. As physicians, we clearly want to provide evidence based medicine that is efficacious and cost effective. I think the committee

has made a mistake and would appreciate a chance to review this with them. If you would like, I would be happy to provide references to

the success and improved patient outcomes in patients that undergo ablation of non-reentrant tachycardias.

I do believe that the clinical committee is charged with a task that is quite difficult. No-one on the committee has experience with cardiac

electrophysiology and the various forms of treatment. It may have been helpful to have an outside expert electrophysiologist available in the

committee meeting at the end of the session to answer questions or give insight.

Please feel free to call me anytime and thank you for allowing my comments.

Sincerely,

Gerhard H. Muelheims M.D.
Providence Spokane Cardiology
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PROVIDENCE
Spokane Heart Institute

Spokane Cardiology

June 19, 2013

Washington State Health Care Authority
Health Technology Assessment
shtap@hca.wa.gov

RE: Catheter Ablation Proc7edures for Supraventricular Tachyarrhythmias

Dear Sir or Madam:

I am a Clinical Electrophysiologist practicing in Spokane, Washington. I have been in practice
with Spokane Cardiology since 1988. I have been involved in ablation at one of the early
centers that performed ablation, University of California San Francisco, in 1986. I was a clinical
assistant professor in electrophysiology at UCSF.

It is understandable that you are attempting to determine what technology is clinically useful in
an attempt to be prudent about the utilization of healthcare dollars. At the same time, it is also
clear that one needs to be responsible in decision making and not restrict procedures that have
clinical benefit that are well demonstrated. I understand that presently it has been the interim
determination of the committee that atrial tachycardia, if not reentrant in nature, will not be
reimbursed. As I believe you also understand, atrial tachycardia represents 5% or less of
episodes of rapid rhythm that significantly impact patient lives. In addition to having a sense of
rapid beating, patients can occasionally have episodes of tachycardic-dependent
cardiomyopathy – weak heart muscle as a result of heart racing.

Studies have been done, one of which is included, to demonstrate ablation for non-reentry
tachycardia is very effective. I am enclosing information from the work of Dr. Shih-Ann Chen of
China published in Circulation 1994.   The study demonstrated efficacy-98% cure of atrial
tachycardia. In the study of 36 patients, 7 out of 36 had automatic atrial tachycardia, 9 out of 36
had triggered atrial tachycardia.   Therefore, 20 patients had reentrant tachycardia and in
summary 16 had non-reentrant tachycardia. The study demonstrated efficacy in both groups.

It would not be appropriate to isolate a particular group for non reimbursement in which efficacy
has been demonstrated in ablation. At this point, certainly this type of cure rate – 98% –
demonstrates cost effectiveness in this subgroup of patients.

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Therefore, it is absolutely appropriate that ablation for symptomatic atrial tachycardia be performed in appropriate circumstances. Young patients who have these often very symptomatic arrhythmias will be faced with lifelong medical therapy. If the procedure is determined to be performed by an electrophysiologists it is appropriate to do so, has a high likelihood of success, and should therefore be covered by the HCA.

If you have any questions I would be happy to respond.

Sincerely,

Harold Goldberg, MD, FACC

Enclosure: Article "Sustained Atrial Tachycardia in Adult Patients"

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Sustained Atrial Tachycardia in Adult Patients

Electrophysiological Characteristics, Pharmacological Response, Possible Mechanisms, and Effects of Radiofrequency Ablation

Shih-Ann Chen, MD; Chern-En Chiang, MD; Chin-Juey Yang, MD; Chen-Chuan Cheng, MD; Tsu-Juey Wu, MD; Shih-Pu Wang, MD; Benjamin N. Chiang, MD; Mau-Song Chang, MD

Background Mechanisms and electropharmacological characteristics in adult patients with atrial tachycardia (AT) are not well described. We proposed that a combination of electropharmacological characteristics, recording of monophasic action potential, and effects of radiofrequency ablation could further determine the mechanisms and achieve a new classification in adults with various types of AT because they were important in regard to the correlation between mechanisms and pathophysiology, clinical syndrome, and responses to specific pharmacological or nonpharmacological therapies.

Methods and Results Thirty-six patients (11 female, 25 male; mean age, 57±13 years) with AT were referred for electropharmacological studies and radiofrequency ablation. Resetting response pattern, entrainment phenomenon, recording of monophasic action potential, serial drug test, response to Valsalva maneuver, endocardial mapping technique, and radiofrequency ablation were performed. Seven patients had automatic AT provocable with isoproterenol; neither initiation nor termination was related to programmed electri-

cal stimulation. The other 29 patients had AT initiated or terminated by electrical stimulation and mechanisms related to triggered activity or reentry; nine of them needed isoproterenol to facilitate initiation of AT, associated with delayed afterdepolarization in monophasic action potential. All responded to adenosine (15 to 60 μg/kg) and Valsalva maneuver. Dipyridamole terminated AT and decreased the slope of afterdepolarization. Afterdepolarization was not found in the patients with automatic or reentrant AT. In 40 of 41 (98%), AT was ablated successfully, with late recurrence in 2 of 40 (5%) (follow-up, 18±4 months).

Conclusions This study demonstrates the diverse mechanisms and electropharmacological characteristics of AT in adults. Furthermore, radiofrequency ablation of various types of AT could achieve high success and low recurrence rates. (***Circulation.*** 1994;90:1262-1278.)

Key Words • tachycardia, atrial • pharmacology • radiofrequency • catheter ablation

Atrial tachycardia (AT) has been well described in pediatric patients.[1-3] However, differences in electrophysiological and pharmacological characteristics between children and adults with AT are not clear. AT does not require atrioventricular (AV) node or ventricular tissue for initiation; it is different from atrial flutter, atrial fibrillation, and sinoatrial reentrant tachycardia.[4-13] The possible mechanisms of AT have been presumed to include reentry, abnormal automaticity, and triggered activity.[1,2,4-13] Reentry is usually confirmed by demonstrating initiation and termination of AT with programmed electrical stimulation and by presence of manifest or concealed entrainment; it is associated with anatomic or functional regions of block as well as areas of slow conduction.[5,7,8-13] Abnormal automaticity cannot be initiated or terminated with programmed electrical stimulation, and it is usually provocable after administration of intravenous isoproterenol.[1,2] Tachycardia related to nonreentrant, nonau

tomatic mechanisms (probably triggered activity) has not been well defined.[14-17] Although the response to programmed stimulation (resetting phenomenon) is not a specific test for mechanisms of arrhythmia, entrainment could be demonstrated only in patients with tachycardia caused by reentry.[1,10,16-18] It seems possible that a monophasic action potential catheter could help record the delayed afterdepolarization from atrial tissues with triggered activity.[19,20]

Pharmacological manipulation (including adenosine, acetylcholine, verapamil, propranolol, and isoproterenol) suggested diverse potential mechanisms of ventricular tachycardia.[21,22] However, the basic electrophysiological characteristics of atrial and ventricular tissues were different, and the serial drug tests had not previously been performed in adult patients with AT. Basic electrophysiological studies from our laboratory on human atrial fibers found that exogenous adenosine and acetylcholine had similar effects[23-25] and exerted their effects on myocardium (inhibition of adenylate cyclase) by binding to their receptors, which are coupled to a pertussis toxin–sensitive inhibitory, guanine nucleotide–binding (G_i) protein.[26,27] However, a complex reflex sequence (both sympathetic and parasympathetic) induced by adenosine and Valsalva maneuver might affect tachycardia.[28-30] It was postulated that the mechanism

of AT could be further elucidated by dipyridamole,[31,32] edrophonium, and competitive adenosine A_1 and muscarinic cholinergic receptor antagonists (aminophylline and atropine, respectively). β-Adrenergic blockade has been shown to be specifically effective in suppressing catecholamine-sensitive tachycardia, and verapamil is effective in AV reentrant tachycardia and ventricular tachycardia related to triggered activity.[1,2,21-23,34,35] The roles of both drugs in the treatment of AT have not been well defined.

Radiofrequency catheter ablation of AT has been reported before.[13,36-38] However, comparative studies of radiofrequency ablation of various ATs in adults have not been performed. Application of radiofrequency energy to a critical area, resulting in termination of AT and prevention of its recurrence, would confirm that a critical myocardial focus is responsible for AT. Furthermore, a successful outcome would help establish the concept of accurate mapping of AT. Using a combination of electrophysiological characteristics, pharmacological response, and effects of radiofrequency ablation to further determine the mechanism and produce a new classification in adult patients with various types of AT is important, because these mechanisms correlate with pathophysiology, clinical syndromes, and response to specific pharmacological or nonpharmacological therapies.

Methods

Patient Population

Thirty-six consecutive patients (11 women and 25 men; mean age, 57 ± 13 years) with AT were referred to this institution for electrophysiological study and treatment of tachycardia by radiofrequency catheter ablation from November 1990 to October 1993. The procedure was performed prospectively under a protocol approved by the Clinical Investigation Committee. None of the 36 patients had previously undergone diagnostic electrophysiological study. The histories of AT ranged from 2 to 18 years (mean, 3 ± 1 years). Tachycardia was incessant in 3 patients and intermittent in the remainder. Five patients had cardiomyopathy, 1 had biatrial enlargement identifiable by echocardiography, 4 had hypertension, 1 had coronary artery disease, 1 had congenital aortic stenosis, 1 had dextrocardia, and 3 had other associated tachycardias. All patients had multiple ECG recordings of tachycardia before referral (Table 1).

Electrophysiological and Pharmacological Study

Baseline Electrophysiological Study

As described previously,[13,39] the patients were studied in the postabsorptive, nonsedated state. Antiarrhythmic drugs were discontinued for at least five half-lives in all patients. Three multipolar, tip-deflectable, closely spaced (2 mm) electrode catheters (Mansfield, Boston Scientific) were positioned in the right atrium, His-bundle area, and right ventricle; and two orthogonal electrode catheters (Mansfield) were positioned in the coronary sinus for recording and/or stimulation. Three surface leads (I, II, and V_1) were recorded simultaneously with intracavitary electrograms using a VR-13 recorder (Electronics for Medicine) at a paper speed of 100 to 150 mm/s and filtered between 30 and 500 Hz. Electrical stimulation was delivered by a programmable stimulator (DTU-215, Bloom Associates, Ltd) with a pulse duration of 2 milliseconds at approximately twice the diastolic threshold. Baseline electrophysiological study consisted of measurement of conduction intervals, followed by determination of atrial and AV node refractory periods (coupling intervals decreasing by 10-millisecond intervals). Rapid right atrial stimulation (paced cycle length from 600 milliseconds to 2:1 capture was noted) and

right atrial extrastimuli (single and/or double) were used for induction and termination of AT, and they were repeated two to four times to ensure reproducibility of the responses. If programmed electrical stimulation failed to induce AT, isoproterenol (at graded dosages from 1 to 4 μg/min) or atropine (0.01 mg/kg) was infused intravenously until AT emerged or the sinus cycle length was accelerated to 400 milliseconds. After the initiation of AT, atrial extrastimuli (single or double) or rapid atrial pacing was delivered to terminate AT. Electrophysiological criteria used for diagnosis of AT included the following: (1) P-wave configuration different from that of sinus rhythm and atrial activation sequence supporting a nonsinus origin for initial depolarization; (2) tachycardia induction and maintenance independent of AV node conduction; and (3) excluding accessory pathways, AV nodal reentry, sinoatrial reentrant tachycardia, and atrial flutter (cycle length <280 milliseconds) as diagnostic possibilities.[1,2,4-13] Detailed attention was given to the 12-lead ECG of any AT to determine similarity to the previously documented clinical tachycardia.

Resetting Response Study

To assess resetting response patterns, a single atrial extrastimulus was synchronized to the atrial activation wave and was delivered at the high lateral right atrium during AT in 10-millisecond decrements (beginning at 10 milliseconds less than the cycle length of AT, until the entire cardiac cycle was scanned, local atrial refractoriness was reached, or AT was terminated). Each coupled single atrial extrastimulus was delivered after every eighth beat with stable cycle length and was repeated two to four times to ensure reproducibility of the response.[13] Resetting of the AT was said to have occurred if (1) a less than compensatory pause occurred after a single extrastimulus as measured at the stimulation site with >20-millisecond advancement of the AT cycle and (2) the same AT (identical cycle length and morphology) resumed after the extrastimulus, suggesting a fixed exit site from the tachycardia circuit. For each AT, the difference between the longest and shortest coupling interval that resulted in resetting was defined as the resetting interval. The return cycle was defined as the interval from the last stimulated electrogram to the next electrogram measured at the stimulation site. Measurements were made from the rapid deflection of the local electrograms recorded from the proximal pair of the quadripolar electrode catheter.[13] Resetting response patterns were characterized by plotting the return cycles versus the coupling intervals of the extrastimuli. The responses observed could be grouped into three patterns. A flat pattern was defined by the presence of a ≤10-millisecond difference in return cycle occurring over a ≥30-millisecond range of coupling intervals. An increasing pattern was defined as an increase in the return cycle as the coupling interval of the extrastimulus was decreased. There were tachycardias meeting the criteria for a flat response at long coupling intervals followed by an increasing response at shorter coupling intervals; such a pattern was defined as mixed or "flat plus increasing" (Fig 1).

Endocardial Mapping and Entrainment Study

Endocardial mapping studies were done at the same time as the baseline electrophysiological study. After the diagnosis of AT was established with a uniform P-wave configuration and stable cycle length, a 7F quadripolar electrode catheter with 2-mm interelectrode spacing (deflectable, 4-mm–tipped; Mansfield, Inc) was inserted into the right atrium via the right femoral vein for mapping and ablation. By use of biplane fluoroscopy (right and left anterior oblique views), the right atrial endocardium was mapped by rotation of the mapping catheter in the anteromedial, anterior, anterolateral, lateral, posterolateral, posterior, posteromedial, and septal positions at several levels, beginning high near the superior vena cava and then withdrawing 1 cm at a time to the inferior vena cava (ie, high, -1, -2, and -3 cm). Recordings from two or three sites near the right atrial appendage, the fossa ovalis, and

TABLE 1.   Clinical Characteristics in Patients With Atrial Tachycardia

Patient	Age, y/Sex	Pattern/ History, y, of AT	Associated Cardiac Arrhythmia	Associated Cardiac Disease
Group 1				
1	56/M	I/5	None	DCM (EF 32%)
2	62/M	P/2	None	None
3	60/F	P/3	None	None
4	69/M	P/2	None	HT, BAE
5	70/M	P/2	None	None
6	48/M	P/4	None	None
7	32/M	P/4	None	None
8	24/F	P/3	AVNRT	AVNRT
9	55/M	P/15	AVRT	CWPW
10	18/M	P/7	None	AS
11	24/F	P/3	None	None
12	55/M	P/38	None	DCM (EF 36%)
13	64/M	P/3	None	None
14	82/M	P/1	None	HT
15	62/F	P/1	None	None
16	68/M	P/2	None	None
17	40/F	P/10	None	CAD+DCM (EF 30%)
18	28/F	P/3	None	None
19	77/M	P/2	None	None
20	38/M	P/22	None	Dextrocardia
Group 2				
1	32/M	P/3	None	None
2	62/M	P/2	None	None
3	30/M	I/18	None	DCM (EF 32%)
4	20/F	P/2	None	None
5	25/F	P/3	None	None
6	36/M	I/5	None	DCM (EF 30%)
7	22/M	P/2	None	None
Group 3				
1	25/F	P/2	None	None
2	25/M	P/8	None	None
3	56/F	P/10	AVNRT	AVNRT
4	60/F	P/20	None	HT
5	72/M	P/2	None	HT
6	65/M	P/3	None	None
7	69/M	P/4	None	None
8	75/M	P/7	None	None
9	54/M	P/10	None	None

AT indicates atrial tachycardia; I, incessant; DCM, dilated cardiomyopathy; P, paroxysmal; HT, hypertension; BAE, biatrial enlargement; AVNRT, atrioventricular nodal reentrant tachycardia; AVRT, atrioventricular reciprocating tachycardia; CWPW, concealed Wolff-Parkinson-White syndrome; AS, congenital aortic stenosis; CAD, coronary artery disease; and EF, ejection fraction of left ventricle.

below the fossa around the coronary sinus ostium were made. The left atrium was recorded via the coronary sinus catheter, often facilitated by the use of long, curved vascular sheaths (Cordis) for transseptal mapping, even in patients with a patent foramen. These sheaths ensured left atrial access during catheter changes and improved torque transmission and overall catheter steering ability. Atriograms were performed to clarify anatomic landmarks. Local activation time,
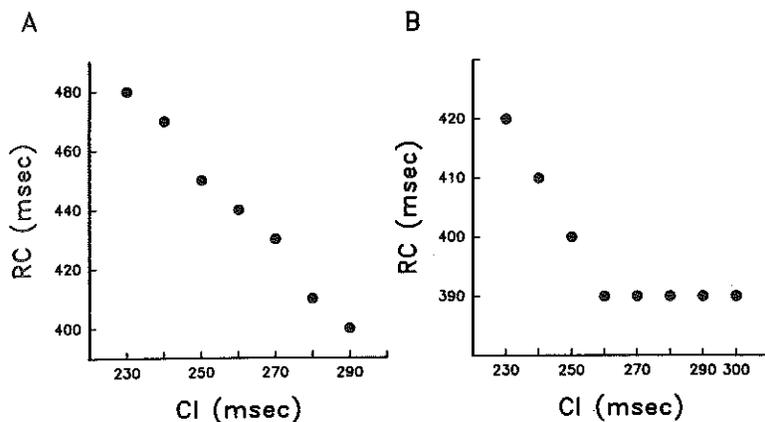
FIG 1. Resetting response curves in reentrant atrial tachycardia (group 1). A, Increasing-type resetting response curve; progressive increases of return cycles (RC) were demonstrated when the premature stimuli coupling intervals (CI) were shortened (patient 10). B, Mixed-type resetting response curve; return cycles were fixed when coupling intervals were shortened from 300 to 260 milliseconds, and return cycles were increased when the coupling intervals were shortened from 250 to 230 milliseconds (patient 18).

defined as the first sharp deflection of the endocardial electrogram, was referenced to the earliest onset of the P wave during AT in surface ECG leads I, II, and V_1. An activation sequence map was constructed manually to determine the earliest atrial activation. If P-wave onset was indistinct or obscured by T-wave activity, a stable intracardiac signal (eg, low septal right atrium in the His bundle area) was initially used as the reference. In patients with reentrant AT, concealed entrainment pace mapping was used to identify the shortest stimulus–to–P-wave interval and exit site of the reentrant circuit.[13] In patients without a demonstrable entrainment phenomenon (eg, AT from triggered activity or enhanced automaticity), pacing mapping (pacing during sinus rhythm) was performed to identify the similarity of P-wave morphology between pacing and AT (recorded in a 12-lead ECG). Furthermore, the earliest atrial activation during AT was found in the mapping procedure to facilitate ablation.

Monophasic Action Potential Study

An electrode catheter for recording monophasic action potential (EP Technology), designed by Franz,[19,20] was used to record monophasic action potential from the area close to the presumed exit site of AT and other atrial sites (similar to the endocardial mapping sites) remote from the exit site during AT. Usually, it was not possible to record monophasic action potentials from the exit site because pressure on the exit site frequently could terminate AT. The recording techniques followed Franz and Koller's[19,20] description, and artifacts were avoided as much as possible. For reproducibility of recording, the presumed exit site of AT and the first recording site were recorded in the cine films, and they were displayed in the monitor during study. The monophasic action potential study included the action potential characteristics in the initiation of AT and termination of AT responsive to adenosine (in different doses), edrophonium, Valsalva maneuvers, and carotid sinus massage. The monophasic action potential duration is usually determined at a repolarization of 90% of monophasic action potential amplitude,[9] defined as the distance from the baseline to the crest of the monophasic action potential plateau.[19] Early afterdepolarizations interrupted or retarded repolarization during phase 2 or 3 or both of the monophasic action potential; delayed afterdepolarization arose after full repolarization of the monophasic action potential. The mechanisms of AT related to afterdepolarizations were determined in the initiation of tachycardia or after administration of drugs. The amplitude of delayed afterdepolarization was defined as the distance from the baseline to the crest of the late potential in the recording of the monophasic action potential. The size of the area in which afterdepolarization could be recorded was as great as 10×10 mm from the monophasic action potential tip electrode.[19] The exit site and recording site of monophasic action potential were recorded in the cine film in every patient, and they were displayed in the monitor during study. This

recording technique had been established in our laboratory before we started this study.

Pharmacological Study

Drugs for terminating AT were administered after sustained AT (duration >4 minutes) was established. Electrophysiological study was performed before and after administration of each drug. Dipyridamole (0.56 mg/kg IV) was infused over a period of 4 minutes, followed by a continuous infusion of 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, and it was infused only when systolic blood pressure during AT was >90 mm Hg. Aminophylline (2.8 mg/kg IV) was selectively evaluated during concurrent infusion of dipyridamole. The total dose of aminophylline was delivered over 3 to 10 minutes. The dosage of intravenous verapamil was 0.15 mg/kg body wt over a period of 2 minutes as a loading dose followed by continuous infusion at 0.005 $mg \cdot kg$ body $wt^{-1} \cdot min^{-1}$. The dosage of intravenous propranolol was chosen to achieve significant β-adrenergic blockade: 0.2 mg/kg body wt at an infusion rate of 1 mg/min. For edrophonium, an initial injection of 2 mg was given over a period of 30 seconds, and an additional 8 mg was administered about 60 seconds later. If the AT was sensitive to edrophonium, the effect of muscarinic cholinergic receptor blockade with atropine (0.04 mg/kg IV) was evaluated. An intravenous bolus of adenosine was given over a period of 2 seconds, followed by saline flush; each dose of 15, 30, 60, 120, and 240 $\mu g/kg$ was separated by at least 4 minutes, and the minimal dose and time required to terminate AT were recorded. Termination of AT by Valsalva maneuver or carotid sinus pressure (left or right side) was also performed. Other antiarrhythmic agents irrelevant to the present study were not used. Briefly, electropharmacological studies in the morning of the first day included baseline electrophysiological study, endocardial mapping, resetting and entrainment study, recording of monophasic action potentials, drug test (adenosine, edrophonium, and atropine), and Valsalva maneuver. After the above procedures, all the electrode catheters were removed and only one sheath with side arm was left in the jugular vein. Four hours later, drug tests with dipyridamole followed by aminophylline (reversal of persantin effect) were again performed in the laboratory. In the afternoon of days 3 and 5, drug tests with verapamil and propranolol, respectively, were performed (the electrode catheter for programmed stimulation was inserted from the sheath left in the jugular vein). Radiofrequency ablation was performed in the afternoon of day 7, and multiple electrode catheters were inserted.

Classification of Possible Mechanisms of AT

Results of basic electrophysiological studies were difficult to extrapolate to explain findings in intact human hearts, and clinical electrophysiological study techniques had practical limitations in definitely differentiating reentry, automaticity, and triggered activity related to afterdepolarization. In this

study, the major criteria of classification were according to whether (1) automatic AT could be initiated only by isoproterenol; (2) AT related to reentry or triggered activity could be initiated or terminated by programmed electrical stimuli; (3) only AT related to triggered activity had early or delayed afterdepolarization recorded in the monophasic action potential; and (4) only AT related to reentry had entrainment phenomenon during atrial pacing. The characteristics of each group are described in the "Results" section.

Radiofrequency Ablation of AT

As described previously,[13,39] radiofrequency ablation was performed with a radiofrequency generator (Radionics-3C, Radionics, Inc) providing 500-kHz, unmodulated sine-wave energy, connected to the distal 4-mm tip of the mapping/ablation catheter (Mansfield, Boston Scientific) via a switch box and grounded to the patient's posterior chest wall with a standard electrosurgical grounding pad. Applied voltage and measured current were displayed continuously, and impedance was monitored by a meter on the radiofrequency generator. Radiofrequency energy was applied in a power range from 20 to 40 W, commencing with constant rhythm monitoring. The presumed ablation site was identified by several methods: concealed entrainment pace mapping and earliest atrial activation in patients with reentrant AT and pace mapping and earliest atrial activation in patients with automatic and triggered AT. If AT was terminated within 15 seconds after energy application (test pulse), a full 60-second application (long pulse) was delivered at that site. Radiofrequency discharge was terminated immediately on the occurrence of impedance rise, displacement of catheter, or a patient complaint of severe chest pain. If an ablation attempt at a particular site was unsuccessful in terminating AT within 15 seconds, radiofrequency discharge was terminated. Endocardial mapping was repeated in an attempt to identify a more accurate ablation site by systematically repositioning the ablation catheter several millimeters around the initial position. If a second AT focus was found, procedures including electropharmacological study, endocardial mapping, and radiofrequency ablation were repeated.

After successful ablation of AT, programmed stimulation was performed immediately and after ≈20 to 30 minutes to determine whether AT was inducible. For the prevention of possible recurrence of tachycardia, a long pulse (60 seconds) of radiofrequency energy was delivered to the successful ablation site. Isoproterenol (2 to 4 μg/min) was routinely used to facilitate induction after initial success. After completion of the study, patients were monitored in the coronary care unit overnight.

All patients had regular follow-up and did not take any antiarrhythmic drugs. They were seen in the outpatient clinic at 1 week, 1 month, and then every 3 months. A history of recent symptoms was taken. Physical examination, 12-lead ECG, and 24-hour Holter monitoring were performed.

Statistical Analysis

Data were analyzed by the t test for paired or unpaired data and, where appropriate, the Wilcoxon rank sum test. Data are expressed as mean±SD. Statistical significance is defined as $P<.05$.

Results

Electropharmacological Characteristics

Group 1: Reentrant AT

Twenty patients had reentrant AT, and 7 (35%) of them had double AT (none of the group 2 and 3 patients had multiple AT) (see Tables 1 and 2). All the clinical ATs were inducible. None of the 7 patients with double AT had clinical documentation of double AT.

Only 1 of the 20 patients (5%) had exercise-induced AT during treadmill exercise test. The mean AT cycle length was 370±48 milliseconds, and exit sites of AT were in the right atria in 24 patients and in the left atria in 3. Exit sites of these double ATs were not located close to one another, suggesting reentry with different exits. The second AT was always found after the first AT was ablated. Thirteen (48%) of the 27 AT exit sites were in the septal area. They were identified by the following characteristics: (1) they could be reproducibly initiated (Fig 2) and terminated with programmed stimulation (patients 10 and 12 needed isoproterenol [1 μg/min] to sustain AT); (2) they fulfilled criteria for manifest and concealed entrainment in all patients (Figs 3 and 4) without afterdepolarization in the recording of monophasic action potential; (3) the interval between the initiating premature beat and the first beat of AT was inversely related to the premature coupling interval of atrial extrastimuli; (4) there were two types of resetting response patterns, with a significant difference in resetting intervals (11 were of the mixed type, with a resetting interval of 81±20 milliseconds, and 16 of the increasing type, with a resetting interval of 49±9 milliseconds, $P<.01$); (5) adenosine terminated 24 of 27 cases of AT (89%), with a minimal effective dose of 37.5±26.5 μg/kg; the time required to terminate AT shortened progressively when the adenosine dose increased ($P<.05$ at 30, 60, 120, and 240 μg/kg compared with 15 μg/kg) (Fig 5), and the minimal shortening of action potential duration at 90% of repolarization (APD$_{90}$) just before termination of AT was 15±5 milliseconds (n=15, $P<.01$) (Fig 6); (6) dipyridamole terminated 21 of 27 cases of AT (78%) (mean, 1.2±0.3 minutes); 19 of the 27 (70%) were still inducible under dipyridamole. Aminophylline reversed the effects of dipyridamole, and AT was reinitiated in 7 of the 7 patients who were responsive to dipyridamole; (7) propranolol terminated 13 of 27 cases of AT (48%), and AT was still inducible under propranolol in 27 cases (9 with nonsustained AT, and 8 of the 18 [44%] with sustained AT had prolongation of AT cycle length) (Fig 3C); clinical information showed that only 4 patients had good response to oral propranolol (80 to 160 mg/d) before ablation, and they were responsive to intravenous propranolol; (8) verapamil terminated AT in 26 of 27 cases (96%), and AT was still inducible under verapamil (nonsustained AT) in 3 of the 27 (11%) (Fig 3D); clinical information showed that 8 patients had good response to oral verapamil before ablation (240 to 320 mg/d), and they were responsive to intravenous verapamil; (9) Valsalva maneuver and carotid sinus pressure terminated AT in only 2 of 27 cases (7.4%), with shortening of action potential duration (from 170 or 160 to 150 or 145 milliseconds, respectively); (10) edrophonium infusion terminated AT in only 2 of 18 cases (11%), and in 17 of the 18 (94%), AT (sustained AT) was still inducible under edrophonium; and (11) aminophylline and atropine could reverse the effects of dipyridamole and edrophonium, respectively.

Group 2: Automatic AT

Seven patients (see Tables 1 and 3) had inducible automatic AT, with minimal AT cycle length ranging from 320 to 400 milliseconds (mean, 356±33 milliseconds); exit sites of AT were in the right atria in 4

TABLE 2. El...
(Group 1)

Pa-tient	Indu Termi
1	SE;
	SE;
2	SE,RP
	SE,RP
3	SE,RP
	SE,RP
4	SE,RP
5	SE,RP
6	SE,RP
7	SE,RP
8	SE,RP
9	SE,RP
10	SE,RP
11	SE,RP
12	SE,RP
	SE,RP
13	SE,RP
	SE,RP
14	SE,RP
	SE,RP
15	SE,RP
16	SE,RP
17	SE;
18	SE,RP
19	SE,RP
	SE,RP
20	SE,RP

AT indicat
L, left; SE, si
I, increasing;
MS, midsept

patients ar
None of tl
exercise-in
were ident
could be in
stimulatior
could be t
(4) propr;
clinical inf
good respc
before abl;
response p
interval of
type, with
(6) adenos
transiently
effective d

TABLE 2. Electrophysiological and Pharmacological Characteristics in Patients With Reentrant Atrial Tachycardia (Group 1)

Patient	Induction/Termination	AT CL, ms/AT Exit Site	Resetting Pattern/Interval, ms	Manifest Entrainment	Adenosine Termination/Dose, μg/kg	Dipyridamole Ter	Ind	Propranolol Ter	Ind	Verapamil Ter	Ind	Valsalva/CSP (R/L)	Edrophonium Ter	Ind
1	SE; SE,RP	440,R-PS	M, 60	+	+, 15	+	–	–	+	+	–	–	ND	ND
	SE; SE,RP	400,R-LPL	M, 50	+	+, 15	+	–	–	+	+	–	–	ND	ND
2	SE,RP;SE,RP	360,R-LP	I, 50	+	+, 60	–	NS	–	+	+	–	–	ND	ND
	SE,RP;SE,RP	330,R-ML	I, 50	+	+, 30	+	NS	–	+	+	–	–	ND	ND
3	SE,RP;SE,RP	370,R-HM	M, 80	+	+, 60	–	NS	+	+(P-CL)	+	NS	–	ND	ND
	SE,RP;SE,RP	410,R-AS	M, 80	+	+, 60	+	NS	+	+(P-CL)	+	NS	–	ND	ND
4	SE,RP;SE,RP	430,R-PS	I, 50	+	+, 15	+	–	+	+(P-CL)	+	NS	–	ND	ND
5	SE,RP;SE,RP	420,R-MS	I, 40	+	+, 15	+	–	+	+(P-CL)	+	–	–	ND	ND
6	SE,RP;SE,RP	460,L-MP	I, 50	+	+, 15	+	–	+	NS	+	–	–	–	+
7	SE,RP;SE,RP	440,R-LPL	M, 60	+	+, 15	+	–	+	NS	+	–	–	–	+
8	SE,RP;SE,RP	330,R-PS	I, 50	+	+, 60	+	NS	–	+	+	–	–	–	+
9	SE,RP;SE,RP	370,R-AS	I, 40	+	+, 60	+	NS	–	+	+	–	–	–	+
10	SE,RP;SE,RP	360,R-LP	I, 60	+	+, 30	+	NS	+	NS	+	–	–	–	+
11	SE,RP;SE,RP	330,R-PS	I, 40	+	+, 30	+	NS	+	NS	+	–	–	–	+
12	SE,RP;DE,RP	360,L-LP	M,110	+	+, 15	+	–	+	NS	+	–	+(R)	+	–
	SE,RP;DE,RP	290,R-PS	I, 40	+	+, 60	+	NS	+	NS	+	–	–	–	+
13	SE,RP;DE,RP	330,R-A	I, 70	+	–	–	+	–	+(P-CL)	+	–	–	–	+
	SE,RP;DE,RP	280,R-AS	I, 40	+	–	–	+	–	+(P-CL)	+	–	–	–	+
14	SE,RP;SE,RP	400,R-MS	M,100	+	+, 60	+	NS	–	+	+	–	–	–	+
	SE,RP;SE,RP	420,R-MA	M,110	+	+, 60	+	NS	–	+	+	–	–	–	+
15	SE,RP; RP	370,R-MA	I, 50	+	+, 30	+	NS	+	NS	+	–	ND	ND	ND
16	SE,RP;DE,RP	300,R-ML	I, 40	+	–	–	+	–	+	–	+	–	–	+
17	SE; SE,RP	320,R-HL	I, 50	+	+, 30	+	NS	–	+(P-CL)	+	–	–	–	+
18	SE,RP;SE,RP	350,R-MS	M, 70	+	+, 15	+	–	–	+(P-CL)	+	–	–	–	+
19	SE,RP;SE,RP	400,L-LP	M, 80	+	+, 15	+	NS	+	NS	+	–	+(L)	+	NS
	SE,RP;SE,RP	380,R-AS	I, 60	+	+, 15	+	NS	+	NS	+	–	–	–	+
20	SE,RP;SE,RP	350,R-AS	M, 90	+	+,120	–	+	–	+	+	–	–	–	+

AT indicates atrial tachycardia; CL, cycle length; Ter, termination of AT; Ind, inducibility of AT; CSP, carotid sinus pressure; R, right; L, left; SE, single extrastimuli; RP, rapid pacing; PS, posteroseptum; M, mixed; ND, not done; LPL, low posterolateral; LP, low posterior; I, increasing; NS, nonsustained; ML, middle lateral; HM, high medial; P-CL, prolongation of tachycardia cycle length; AS, anteroseptum; MS, midseptum; MP, midposterior; DE, double extrastimuli; A, atrial appendage; MA, middle anterior; and HL, high lateral.

patients and in the left atria in 3 (all in the free wall). None of them had multiple AT. All the patients had exercise-induced AT during treadmill exercise test; they were identified by the following characteristics: (1) AT could be initiated only with isoproterenol; (2) electrical stimulation could not initiate or terminate AT; (3) AT could be transiently suppressed with overdrive pacing; (4) propranolol terminated all (100%) of the AT; clinical information showed that 5 of the 7 patients had good response to oral propranolol (120 to 240 mg/d) before ablation; (5) there were two types of resetting response patterns (4 of the mixed type, with a resetting interval of 136±26 milliseconds; 3 of the increasing type, with a resetting interval of 110±20 milliseconds); (6) adenosine could not terminate any AT, and AT was transiently suppressed in only 4 of 7 cases (minimal effective dose, 240 μg/kg); (7) dipyridamole, verapamil,

Valsalva maneuver, carotid sinus pressure, and edrophonium could not terminate any AT; (8) recordings of monophasic action potential from all the recording sites did not find afterdepolarization, but recordings from the area near the exit site showed increasing phase 4 diastolic slope just before the onset of AT; minimal shortening of APD_{90} just before the transient suppression of AT by adenosine was 14±5 milliseconds (n=4, $P<.01$) (Fig 7); and (9) sinus node electrograms from the presumed ablation sites in the 4 patients with AT exit sites in the high lateral aspect of the right atrium did not show sinus node activity.

Group 3: Nonreentrant, Nonautomatic AT; Probably Triggered Activity

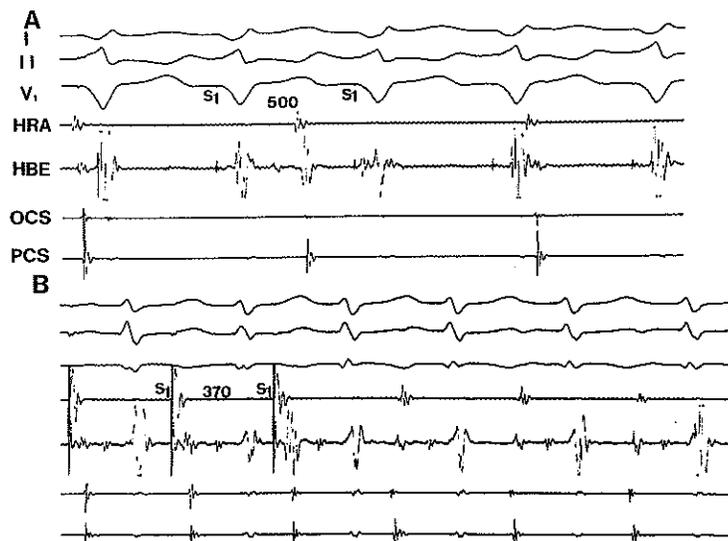Nine patients (see Tables 1 and 4) had AT related to triggered activity, and 4 of them could be initiated by

FiG 2.   Tracings showing induction of atrial tachycardia (group 1, patient 11). Ventricular pacing (S_1S_1) with a cycle length of 500 milliseconds showed no ventriculoatrial conduction (A). Rapid atrial pacing (cycle length, 370 milliseconds) initiated atrial tachycardia. HRA indicates high right atrium; HBE, His bundle area; OCS, coronary sinus orifice; and PCS, proximal coronary sinus.

treadmill exercise test. All cases of clinical AT were inducible. The exit sites of AT were in the right atria in 6 patients and in the left atria in 3 (4 [44%] of the 9 in the septal area). None of the 9 patients had multiple AT, and the mean AT cycle length was 396±66 milliseconds. They were identified by the following characteristics: (1) initiation of AT was reproducible with rapid atrial pacing in 9 patients, and AT could also be initiated by atrial extrastimuli under isoproterenol in only 1 of the 9 patients. Isoproterenol alone initiated AT in 1 patient. In addition, AT usually demonstrated cycle length dependence, and its initiation was dependent on achieving a critical range of atrial pacing cycle lengths; (2) delayed afterdepolarization was more prominent in the recordings of monophasic action potential just before onset of AT (Fig 8), and recordings from the areas remote from the exit site did not show any afterdepolarization; (3) termination of AT was reproducible with programmed stimulation; (4) entrainment was not found, but overdrive suppression and overdrive termination were demonstrated; (5) there were two types of resetting response patterns (6 of the mixed type of AT, with a resetting interval of 160±56 milliseconds, and 2 of the increasing type, with a resetting interval of 75±7 milliseconds); (6) adenosine terminated AT in all cases; the minimal effective dose was 23.3±15.2 μg/kg (lower than group 1, but without statistical significance, $P>.05$), the time required to terminate AT shortened progressively when the adenosine dose increased ($P<.05$ at 60, 120, and 240 μg/kg compared with 15 μg/kg) (Fig 6), and the minimal shortening of APD_{90} just before termination of AT was 16±4 milliseconds (n=8, $P<.01$); (7) dipyridamole (termination time, 0.7±0.2 minutes), propranolol, verapamil, Valsalva maneuvers, carotid sinus pressure (termination time, 5±2 seconds; shortening of APD_{90}, 15±5 milliseconds, n=8, $P<.01$), and edrophonium terminated AT in all group 3 patients; (8) under dipyridamole, propranolol, verapamil, and edrophonium infusion, AT (nonsustained) was inducible in 4 (44%), 6 (66%), 0 (0%), and 4 (44%) of the 9 patients with AT, respectively; clinical information showed that 2 patients had good response to oral verapamil (240 to 320

mg/d), 2 patients had good response to oral propranolol (80 to 160 mg/d) before ablation, and AT was not inducible after intravenous verapamil and propranolol, respectively; and (9) aminophylline and atropine could reverse the effects of dipyridamole and edrophonium in all patients. In 4 patients, the amplitude of delayed afterdepolarization on the last beat of nonsustained AT decreased significantly after dipyridamole ($-2.5±0.2$ mV, $P<.05$ compared with the first initiating afterdepolarization) (Fig 8).

Radiofrequency Ablation of Various ATs

Thirty-four patients with 41 cases of AT received radiofrequency ablation therapy. The endocardial mapping data, biophysical characteristics of radiofrequency energy, and results of radiofrequency ablation are shown in Table 5. The earliest atrial activation time preceding surface ECG P waves and number of test pulses for successful ablation were similar in the three groups. Radiofrequency ablation failed in 2 patients with AT related to triggered activity, and 1 of them had successful ablation in the secondary ablation session. During the follow-up period (2 to 37 months; mean, 18±4 months), 2 patients had recurrent AT and received successful secondary ablation sessions without later recurrence. None of the patients had any procedure-related complication.

Discussion

In this study, the major findings of electropharmacological characteristics are summarized in Table 6: (1) programmed stimulation could not initiate or terminate AT in patients with automatic AT; (2) induction of AT related to triggered activity is usually dependent on achievement of critical pacing cycle length with increasing amplitude of delayed afterdepolarization; (3) entrainment phenomenon and slow conduction were demonstrable only in patients with reentrant AT; (4) overdrive suppression after atrial pacing was demonstrable in AT related to automaticity and triggered activity; (5) resetting response patterns were similar in the three groups; (6) verapamil, adenosine, and acetylcholine (endogenous and exogenous) were not effective in terminating automatic AT; (7) propranolol was spe-
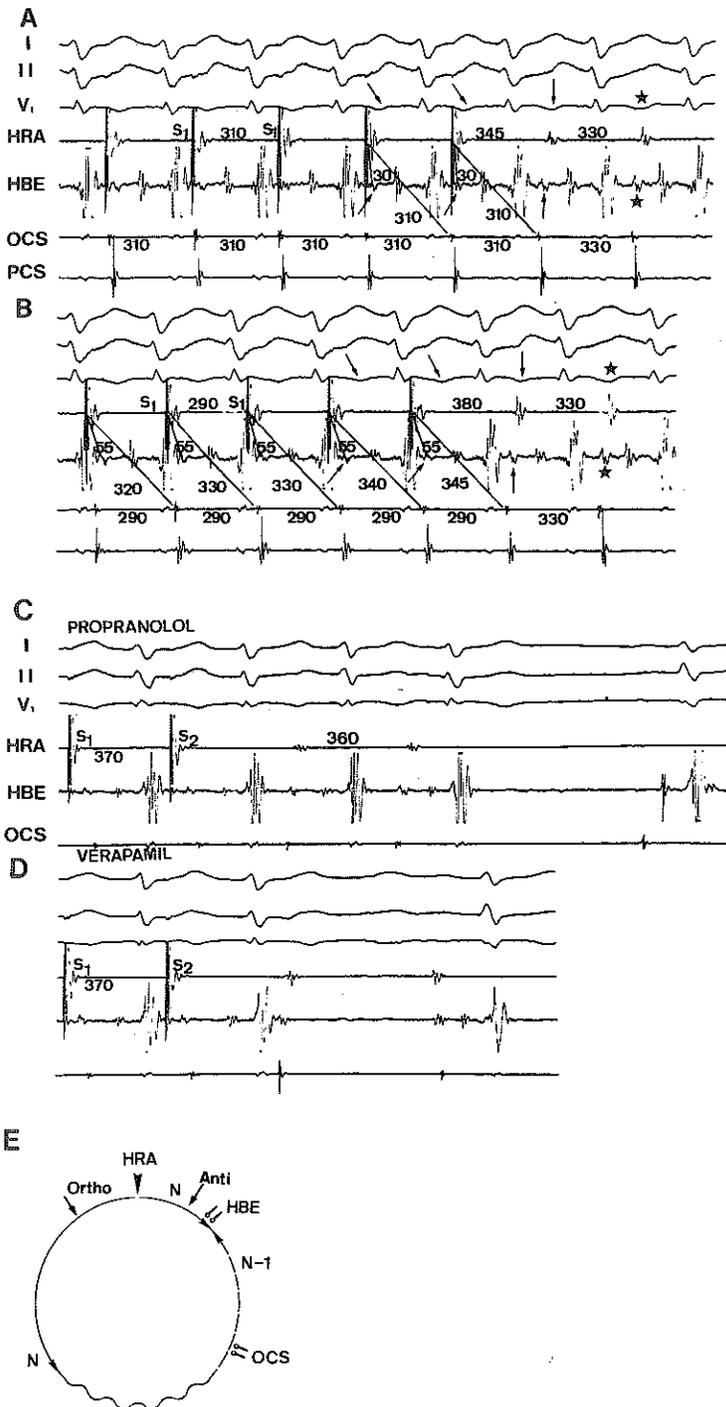
FIG 3. Tracings showing manifest entrainment of reentrant atrial tachycardia (AT) (group 1, patient 11). The earliest atrial activation was near the coronary sinus orifice (OCS). PCS indicates proximal coronary sinus. A and B, AT cycle length of 330 milliseconds; rapid pacing (S_1S_1) was performed from the high right atrium (HRA) with a cycle length of 310 and 290 milliseconds, respectively. Constant fusions in different pacing rates were demonstrated (oblique arrows). The morphology of the last paced beat (vertical arrow) was similar to the spontaneous AT beat (star). Please note the local electrogram in the His bundle area (HBE). The atrial activation electrograms were different between pacing (oblique arrow) and AT (vertical arrow and star). It is possible that the atrial activation in the HBE during pacing was from antidromic activation and the last entrained beat (vertical arrow) was from orthodromic activation. Furthermore, conduction time from the HRA to OCS was progressively prolonged. A decremental conduction area (slow conduction area) would be present in the orthodromic activation direction (E). C, Atrial extrastimuli (coupling interval, 370 milliseconds) initiate two atrial reentrant echoes with a longer cycle length (360 milliseconds) after intravenous propranolol. D, Initiation of one atrial reentrant echo after intravenous verapamil. E, The antidromic (Anti) wavefront (N) collided with the orthodromic (Ortho) wavefront from the previous beat (N-1). The curved line was the presumed slow conduction area.

cific for terminating catecholamine-sensitive AT; and (8) neither verapamil nor adenosine was specific for AT related to reentry or triggered activity.

Electrophysiological Characteristics of AT

Response to programmed stimulation and electropharmacological study suggested that the mechanisms of tachycardia included reentry, triggered activity, and catecholamine-mediated automaticity. Tachycardia resulting from triggered activity related to delayed afterdepolarizations has been identified and characterized in both animal and human studies.[14-18] Within a critical range of cycle lengths, delayed afterdepolar-

izations may attain threshold and elicit an action potential that, if perpetuated, can lead to a sustained rhythmic activity.[14-17] In this study, AT suggestive of triggered activity conformed to some of the electrophysiological features described in isolated tissue preparations. However, differentiation between reentry and triggered activity remains difficult, because both reentry and triggered activity may have similar clinical electrophysiological manifestations despite differences in their basic electrophysiological mechanisms. As demonstrated in this study, reentry and triggered activity could be initiated and terminated by programmed electrical stimulation. The initiation of
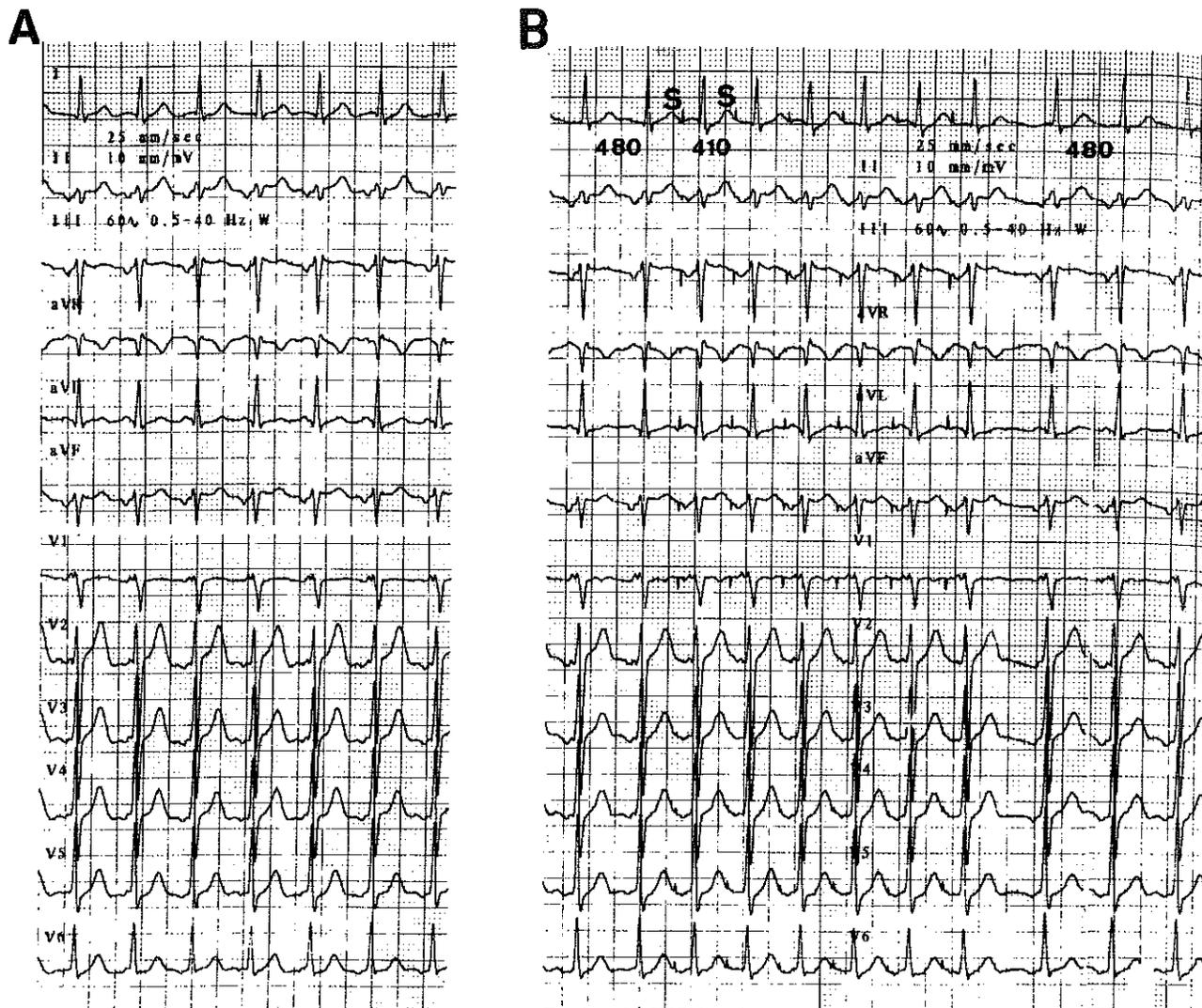
A

B

FIG 4. A, Tracings showing atrial tachycardia (AT) exit site in the midposterior area of the left atrium with a cycle length of 480 milliseconds (patient 6, group 1). B, Rapid atrial pacing with a cycle length of 410 milliseconds from the area near the exit site of AT. Concealed entrainment pacing mapping was performed; the morphologies of 12-lead P waves were identical during both entrainment pacing and spontaneous AT, and the stimulus–P-wave interval was short (about 30 milliseconds).

triggered activity may be a cycle length–dependent phenomenon.[14-17] Nevertheless, shortening of the pacing cycle length may induce unidirectional block in one pathway and slow conduction in the other pathway, setting up the conditions for reentry. Overdrive pacing can cause enhancement of electrogenic sodium extrusion, which hyperpolarizes the membrane potentials and may thereby abolish triggered activity.[40] Furthermore, overdrive pacing may penetrate and alter refractoriness of reentrant pathways, thereby terminating a reentrant arrhythmia. Basic electrophysiological study usually showed that the interval between the last paced beat and the first beat of arrhythmia tended to increase in reentry (reverse relation), whereas it tended to decrease in triggered activity (concordant relation) as the paced cycle lengths or extrastimuli coupling intervals progressively decreased.[15,16] However, studies in different cardiac tissues had different results, and exceptions to the rule in basic and clinical studies may occur.[15,16] In reentry, this interval presumably reflects total conduction time in both the ante-
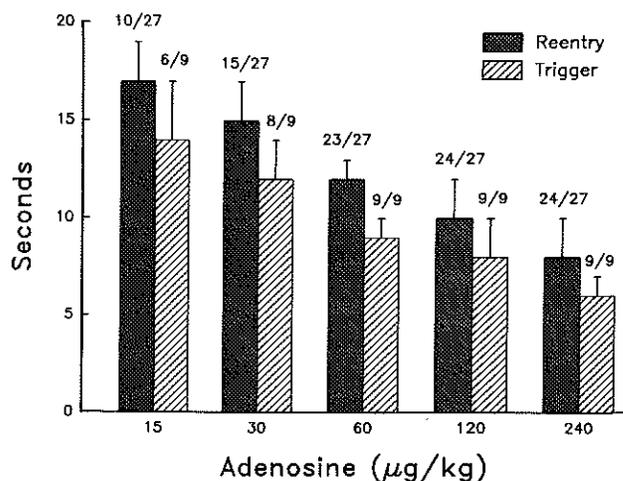
FIG 5. Bar graph showing effects of intravenous adenosine on atrial tachycardia (AT) related to reentry or triggered activity. AT was terminated within a shorter time interval when the adenosine dose was higher, and more ATs were responsive to adenosine at a higher dose.
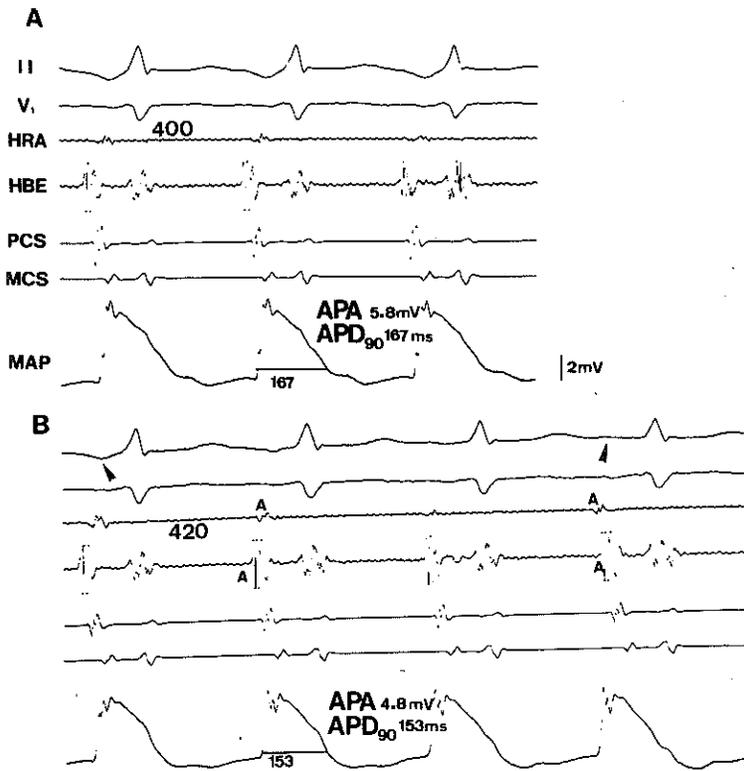
FIG 6. Tracings showing termination of reentrant atrial tachycardia (AT) after intravenous bolus adenosine (15 μg/kg) in patient 1 (group 1). A, AT with cycle length of 400 milliseconds, surface ECGs from leads II and V_1, and intracardiac electrograms from the high right atrium (HRA), His bundle area (HBE), and proximal and middle portions of coronary sinus (PCS, MCS). Monophasic action potential (MAP) was recorded from the atrium near the exit site of AT; action potential amplitude (APA) was 5.8 mV; action potential duration at 90% repolarization (APD_{90}) was 167 milliseconds. B, Prolongation of AT cycle length (420 milliseconds) and decrease of APA (4.8 mV) and APD_{90} (153 milliseconds) just before termination of AT. Left arrow, AT with negative P wave in lead II; right arrow, positive P wave of sinus rhythm.

grade and retrograde limbs of the reentrant circuit. If the retrograde limb conducts rapidly because of early recovery of excitability at shorter cycle lengths, the total conduction time may remain unchanged or even decrease rather than increase. Therefore, the interval between the initiating beat and first beat of arrhythmia alone may not be a strong indicator for either reentry or triggered activity.[21] As described previously,[13] this study showed that the reverse relation was found in AT related to reentry. In AT related to triggered activity, nine patients had AT initiated during one to three pacing cycle lengths, and only one patient had AT initiated during extrastimuli. Therefore, demonstration of a concordant relation was difficult to achieve. The findings in ventricular tachycardia related to reentry were similar to those of the present study, with increasing or mixed resetting response

pattern in AT related to reentry.[41,42] Although basic studies usually showed a flat or decreasing resetting response pattern in tachycardias related to triggered activity, a full compensatory pause with mixed or increasing resetting response pattern was possible.[16,43] However, the clinical studies of resetting response pattern in tachycardia related to triggered activity were limited. This study also found both increasing and mixed response patterns in automatic AT. It is possible that resetting of an automatic or triggered rhythm occurs when a premature beat modifies the sloping diastolic depolarization of the rhythmic cells, and resetting of a reentry tachycardia occurs when a premature beat penetrates and traverses the reentry circuit earlier than expected. It should be noted that the stimulation site was always in the high lateral aspect of right atrium, and the results might be a little

TABLE 3. Electrophysiological and Pharmacological Characteristics in Patients With Automatic Atrial Tachycardia (Group 2)

Patient	Induction/ Termination by PES	Minimal AT CL, ms/ AT Exit	Resetting Pattern/ Interval, ms	Entrainment/ Overdrive Suppression/ Termination	Adenosine Termination/ Dose, μg/kg	Dipyridamole		Propranolol		Verapamil		Valsalva/ CSP (R/L)	Edrophonium	
						Ter	Ind	Ter	Ind	Ter	Ind		Ter	Ind
1	ISO,-(S)	380,R-HL	M,140	-,+,-	-,240*	-	+	+	-	-	+	-	-	+
2	ISO,-(S)	320,L-ML	I, 90	-,+,-	-,240	-	+	+	-	-	+	-	-	+
3	ISO,-(S)	360,L-LP	M,120	-,+,-	-,240	-	+	+	-	-	+	-	-	+
4	ISO,-(S)	400,R-HL	M,160	-,+,-	-,240*	-	+	+	-	-	+	-	-	+
5	ISO,-(S)	380,L-MP	M,100	-,+,-	-,240	-	+	+	-	-	+	-	-	+
6	ISO,-(S)	320,R-HL	I,110	-,+,-	-,240*	-	+	+	-	-	+	-	-	+
7	ISO,-(S)	330,R-HL	I,130	-,+,-	-,240*	-	+	+	-	-	+	-	-	+

PES indicates programmed electrical stimulation; ISO, isoproterenol; (S), spontaneous termination after isoproterenol washout; and *, transient suppression of AT followed by AT warm-up. Other abbreviations as in Tables 1 and 2.
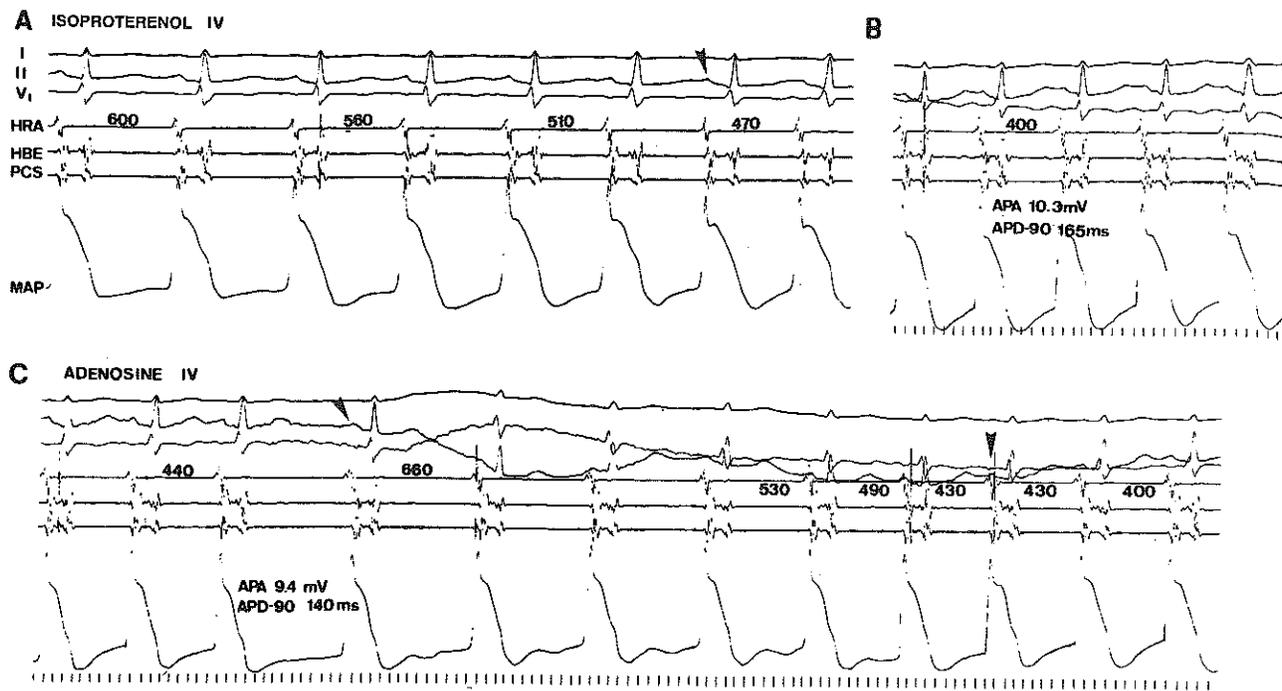
FIG 7. Tracings showing atrial tachycardia (AT) related to automatic mechanism (patient 4, group 2). A, Appearance of automatic AT (arrow) after intravenous isoproterenol with a P-wave morphology (arrow) a little different from the sinus rhythm. Diastolic interval was shortened, and diastolic slope was progressively increased. B, Automatic AT with a minimal cycle length of 400 milliseconds (action potential amplitude [APA], 10.3 mV; action potential duration at 90% repolarization [APD_{90}], 165 milliseconds). C, Transient suppression of automatic AT with appearance of sinus beats (left arrow), and AT (right arrow) followed immediately. The APA and APD_{90} decreased before transient suppression of AT. HRA indicates high right atrium; HBE, His bundle area; PCS, proximal coronary sinus; and MAP, monophasic action potential.

different if the stimulation site were changed. Furthermore, the bidirectional conduction time, entrance sites of extrastimuli, and exit sites of AT have pronounced effects on the resetting response pattern. Therefore, the resetting response patterns could not differentiate the definite mechanisms of AT.

Entrainment, a specific electrophysiological phenomenon in patients with reentrant tachycardia,[44] was demonstrated in all patients with reentrant AT. However, the entrainment phenomenon was not found in cardiac tissues with triggered activity[17,18] nor in

the present study patients with AT related to triggered activity or automaticity. It might reasonably be expected that some triggered activity met criteria 1 and 2 of manifest entrainment (progressive and constant fusion); however, the first beat after cessation of atrial pacing might approximate the pacing cycle length, or the triggered rhythm might gradually or suddenly terminate.[44] Hence, it appears that some distinctions really existed between triggered and reentrant rhythms regarding the entrainment phenomenon.

TABLE 4. Electrophysiological and Pharmacological Characteristics in Patients With Atrial Tachycardia Related to Triggered Activity (Group 3)

Patient	Induction/ Termination		AT CL, ms/AT Exit Site	Resetting Pattern/ Interval, ms	Entrainment/ Overdrive Suppression/ Termination	Adenosine Termina- tion/ Dose, μg/kg	Dipyr- idamole		Pro- pranolol		Verap- amil		Val- salva CSP (R/L)	Edro- phonium	
							Ter	Ind	Ter	Ind	Ter	Ind		Ter	Ind
1	ISO+RP;	SE,RP	400,R-MS	M,120	−,+,+	+,30	+	NS	+	NS	+	−	+(L)	+	NS
2	ISO,ISO+RP;	SE,RP	530,L-LP	M,200	−,+,+	+,60	+	NS	+	−	+	−	+(R)	+	−
3	ISO+ES,+RP;SE,RP		340,R-HL	M,100	−,+,+	+,15	+	−	+	−	+	−	+(R)	+	−
4	ISO+RP;	DE,RP	340,R-PS	I, 70	−,+,+	+,15	+	NS	+	NS	+	−	+(L)	+	−
5	ISO+RP;	SE,RP	320*,R-AS	ND	−,+,+	+,30	+	NS	+	NS	+	−	+(L)	+	NS
6	ISO+RP;	SE,RP	450,R-LP	M,230	−,+,+	+,15	+	−	+	NS	+	−	+(L)	+	NS
7	ISO+RP;	SE,RP	420,R-PS	M,200	−,+,+	+,15	+	−	+	NS	+	−	+(L)	+	NS
8	ISO+RP;	SE,RP	400,R-LP	I, 80	−,+,+	+,15	+	−	+	NS	+	−	+(R)	+	−
9	ISO+RP;	SE,RP	360,R-LP	M,110	−,+,+	+,15	+	−	+	−	+	−	+(R)	+	NS

*Alternans of AT CL (300 to 340 ms; mean, 320 ms). Other abbreviations as in Tables 1 through 3.
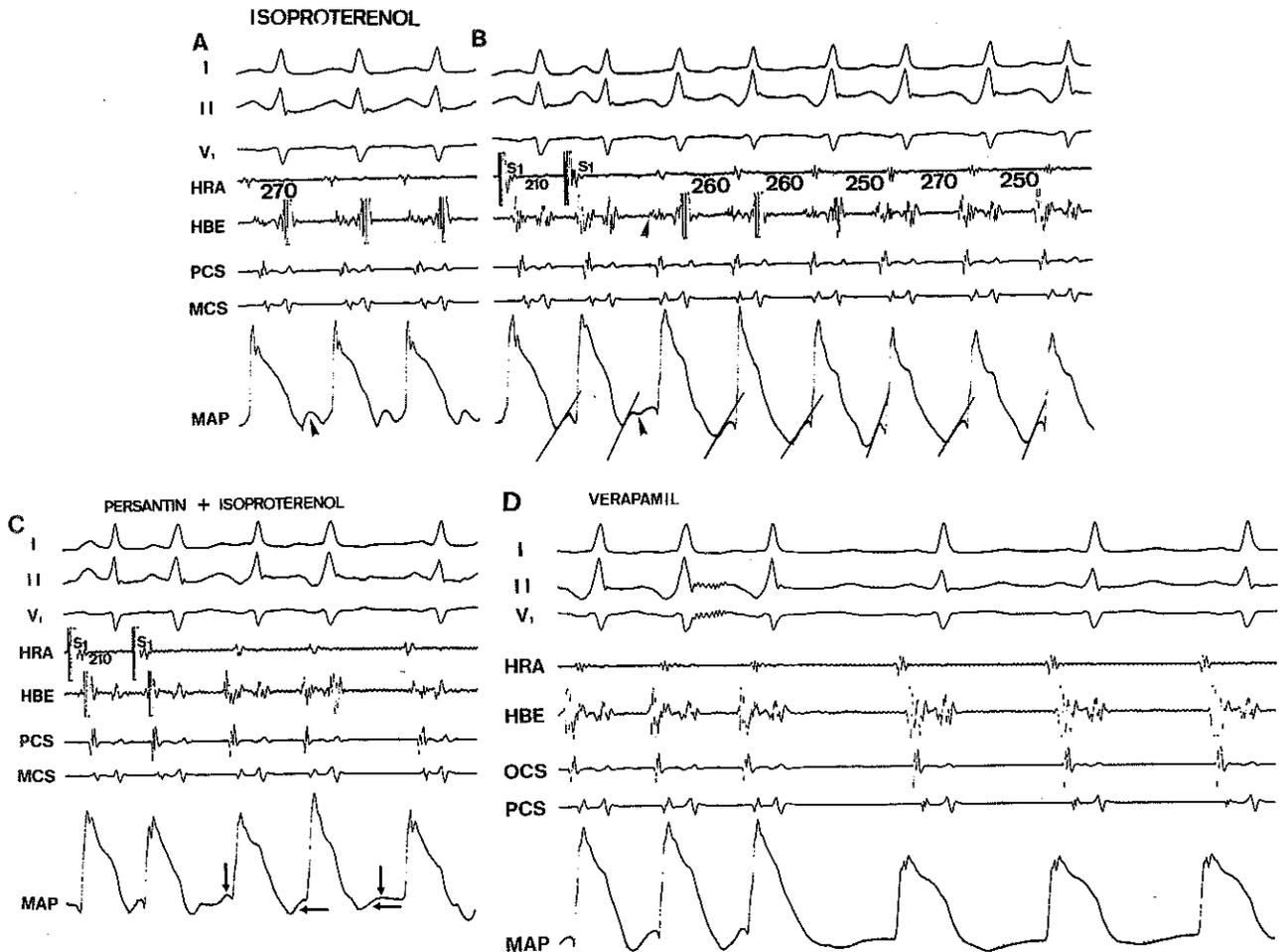
FIG 8. Tracings showing atrial tachycardia (AT) related to triggered activity (patient 4, group 2). A, Appearance of sinus tachycardia (cycle length, 270 milliseconds) with delayed afterdepolarization (arrow) in the monophasic action potential (MAP) recordings after intravenous isoproterenol. B, AT caused by triggered activity was initiated after rapid atrial pacing (cycle length, 210 milliseconds) and isoproterenol infusion. After the last paced beat, delayed afterdepolarization was followed by AT (arrow). The earliest atrial activation was recorded in the His bundle area (HBE). Oscillation of afterdepolarization rising slope was accompanied by cycle length alternans. C, After intravenous persantin and isoproterenol, 2 AT beats were initiated by triggered activity. The third afterdepolarization showed a less steep rising slope (horizontal arrows) than in the second afterdepolarization, and a smaller amplitude (vertical arrows on the crest of afterdepolarization) than in the first afterdepolarization. D, Termination of AT after intravenous verapamil; sinus rhythm with stable monophasic action potential recordings followed. Afterdepolarization was not found (compared with A). HRA indicates high right atrium; PCS and MCS, proximal and middle coronary sinus, respectively; and OCS, coronary sinus orifice.

Usefulness of Monophasic Action Potential Recording

Monophasic action potentials are extracellularly recorded waveforms that can reproduce the repolarization time course of intracellular action potentials, and this recording technique is suitable for studying local repolarization, bridging the gap between basic and clinical electrophysiology.[19,20] During AT, the monophasic action potential showed a regular and repeating wave form with characteristics very similar to that during sinus rhythm. Despite the fast rate, repolarization is always complete between successive depolarizations, and they are separated from each other by a distinct diastolic interval. The possibility that the delayed afterdepolarization and the increasing diastolic slope arose as a result of atrial wall motion or an unstable recording must be excluded, but it seems unlikely. First, the onset of delayed afterdepolarization or increasing diastolic slope in monophasic action potential was earlier than or simultaneous with the onset of the P wave during AT, indicating that the recording site was close to the exit site of AT; second, delayed afterdepolarization and increasing diastolic slope were not recorded from any site remote from the AT focus; third, delayed afterdepolarization and increasing diastolic slope were not found after intravenous verapamil and propranolol, respectively; and fourth, the resting potential from which the first sinus beat after the termination of AT arose was relatively constant, implying a stable recording. These findings demonstrate a close relation between the specific findings from monophasic action potential recording and AT, and they are helpful in differentiating the mechanisms of AT. Study limitations showed that the absolute values of maximal diastolic potential and upstroke velocity of phase 0 (dV/dt) were difficult to obtain in monophasic action potential recording,[19] and the recording sites might not be exactly in the reentrant path or exit sites of AT. Further studies are necessary to learn more about various types of AT.

TABLE 5.   Endocardial Mapping, Biophysical Parameters, and Results of Radiofrequency Ablation in Patients With Atrial Tachycardia

	Group 1 (n=27)	Group 2 (n=6)	Group 3 (n=8)
Activation-P, ms	−36±6	−30±4	−32±5
Stimulus-P, ms*	25±4		
RF pulses, No.			
Test	7±3	8±4	5±1
Long	2±0	2±0	2±0
RF power, W	29±4	27±5	30±2
RF duration, s	98±15	104±19	100±16
Results			
Immediate S,F	27,0	6,0	6,2
Long-term S,F	27,0	6,0	7,1
Recurrence	1/27	0/6	1/7
Complications	0/27	0/6	0/8
Follow-up, mo	18±11	16±11	15±9

P indicates P wave; RF, radiofrequency; No., number of atrial tachycardia foci for RF ablation; S, success; F, failure; and *, the shortest stimulus–P-wave interval measured during concealed entrainment.

Role of Slow Channel Action Potential and β-Receptor Agonist in AT

It has been suggested that slow channel action potentials may become dominant under pathological conditions, especially in the regions (such as atrial tissues) ordinarily dominated by the fast sodium channel response (see Table 6).[17,18,45-48] In patients with atrial arrhythmias, cellular electrophysiological studies in atrial tissues excised in open heart surgery showed that most of the excised atrial tissues had slow response action potential.[47,48] Furthermore, slow channel action potentials have been implicated in the genesis of reentrant arrhythmias because of their propensity for very slow propagation of impulses.[49] Although most of the patients had structurally normal atria, as shown by echocardiography, the ultrastructures of the atrial tissues might be abnormal. As this study showed, slow conduction was evident in rapid atrial pacing, and most of the reentrant AT was responsive to verapamil, with prolonged tachycardia cycle length before termination. It is possible that verapamil probably exerted its antiarrhythmic effect by depressing the slow conduction part of the reentrant circuit.[50,51]

The stimulus-evoked calcium entry might be via voltage- and time-dependent channels activated by depolarization and modulated via agonist and β-adrenergic receptor interaction, and delayed afterdepolarizations might result from a transient inward current carried by sodium and modulated by calcium.[17,18,52] Therefore, verapamil (inhibiting the calcium-dependent slow channel response) and tetrodotoxin (fast sodium channel blocker) may suppress delayed afterdepolarizations.[52] As demonstrated in this study, initiation of triggered activity was facilitated with intravenous isoproterenol. The mechanism by which β-adrenergic stimulation increases the slow inward calcium current has been demonstrated to be mediated through the adenylate cyclase–cAMP system. It was demonstrated that stimulation of β-adrenergic receptors could promote conversion of cytosolic ATP to cAMP, and cAMP activates cAMP-dependent protein kinase A, which phosphorylates protein constituents of the membrane-bound calcium channels; therefore, the availability of functioning slow inward calcium channels is increased.[53,54] Verapamil directly blocks the voltage- and time-dependent slow inward calcium channels and may also act as a competitive β-adrenergic receptor antagonist.[55,56] Conversely, propranolol circumvents the stimulatory effects of β-adrenergic agonists on the slow inward calcium current by competitive inhibition. In patients with AT related to afterdepolarization, AT could be totally terminated and noninducible after verapamil, and propranolol could suppress the inducibility of AT in only three (30%) of nine patients. It is possible that AT related to delayed afterdepolarizations in this study relies principally on the voltage- and time-dependent calcium channels, with less dependence on β-adrenergic receptor stimulation.

Isoproterenol could provoke AT of various mechanisms by exhibiting a variety of electrophysiological effects. It enhances phase 4 diastolic depolarization (automaticity), decreases refractory periods of myocardial tissues and thus enhances the occurrence of reentry, accelerates the sinus rate (shortening of cycle length), increases intracellular cAMP, and thus potentiates triggered activity.[57] If the initiation of a reentrant or triggered rhythm is dependent solely on intravenous infusion of isoproterenol, β-adrenergic blockade with intravenous propranolol may be effective in suppressing the inducibility of arrhythmia in all the patients. However, for nearly complete β-adrenergic blockade, the dose of intravenous propranolol may require up to 0.6 mg/kg body wt.[58] Although the basic study showed that

TABLE 6.   Summary of Electropharmacological Characteristics and Radiofrequency Ablation Responses in Various Types of Atrial Tachycardia

	PES	AT Foci	AT Site	Resetting	Entrain-ment	DAD	Termination by Drugs and VM, %				Successful RFA, %
	Ind/Ter	S/M	R/L	M/I			ADO	VER	PRO	VM	
Group 1 (reentrant)	27/27	20/7	24/3	11/16	+	−	89	96	48	7.4	100
Group 2 (automatic)	0/0	7/0	4/3	4/3	−	−	0	0	100	0	100
Group 3 (triggered)	9/9	9/0	8/1	6/2	−	+	100	100	100	100	88

PES indicates programmed electrical stimulation; AT, atrial tachycardia; DAD, delayed afterdepolarization; VM, vagal maneuver; Ind/Ter, induction/termination; S/M, single/multiple; R/L, right/left; M/I, mixed/increasing; ADO, adenosine; VER, verapamil; PRO, propranolol; and RFA, radiofrequency ablation.

the velocity of upstroke and the overshoot of action potentials were decreased at higher concentrations of propranolol[59] and that high-dose propranolol may suppress either reentry or triggered activity through inhibition of the depolarizing fast inward sodium current,[59] it would be clinically inappropriate. For the automatic AT provocable with isoproterenol, intravenous propranolol suppresses provocability of automatic AT by competitively antagonizing stimulation of catecholamines at β-adrenergic receptors. It is demonstrated that not just a single ionic current is responsible for the automatic depolarization.[60] Decrease of outward background current (such as Na-K pump current), increase of calcium current, funny current (I_f), and inward background current (such as Na-Ca exchange current) must be considered.[60] Therefore, verapamil might be ineffective in patients with automatic AT, and this automatic rhythm was not caused only by the calcium-dependent slow channel response.

Effects of Adenosine and Acetylcholine on AT

Lerman[22] recently described one type of ventricular tachycardia that was responsive to adenosine and vagal maneuvers (increase of acetylcholine level) that antagonized catecholamine-associated increases in cAMP and provided strong evidence for cAMP-mediated triggered automaticity as the mechanism of tachycardia. These observations also suggested that adenosine might be used as a diagnostic tool to identify a unique subset of patients with idiopathic ventricular tachycardia.[22] However, the electrophysiological characteristics, ion currents, and autacoid receptors of atrial tissues were different from those in ventricular tissues. Basic electrophysiological studies from our laboratory on human atrial fibers showed that acetylcholine (0.55 to 5.5 μmol/L) reduced contraction force, shortened the APD_{90}, shifted the plateau of action potential to more negative values, abolished phase 4 depolarization, suppressed the activity of spontaneous fibers, and abolished epinephrine-induced delayed afterdepolarization.[23] An in vitro study on human atrial fibers also showed that adenosine (1 nmol/L to 10 μmol/L) did not induce consistent effects on the characteristics of fast response action potential, but it reduced the upstroke velocity and amplitude of slow response action potential, inhibited rate of spontaneous discharge in a concentration-dependent manner, and suppressed epinephrine-induced delayed afterdepolarization.[25] However, another study of human atrial fibers showed that adenosine had no effect on the action potential characteristics and sarcolemmal Na,K-ATPase activity at physiological concentrations, but adenosine decreased amplitude of action potential and shortened action potential duration (APD_{50}, APD_{75}) at unphysiological concentrations (>100 μmol/L).[24]

Triggered Activity

In this study, all patients with AT related to triggered activity were responsive to adenosine, edrophonium, and vagal maneuvers. Monophasic action potential recordings from triggered AT showed that adenosine, vagal maneuver, and edrophonium shortened APD_{90} and shifted the diastolic potential to more negative values. The findings were similar to the in vitro study from our laboratory.[23,25] The proposed mechanism was

that adenosine and endogenous acetylcholine inhibited adenylate cyclase through receptors coupled to G_i protein and terminated AT related to triggered activity. Exogenous adenosine boluses and the Valsalva maneuver may have multiple other effects in vivo, some of which may also be antiarrhythmic or proarrhythmic. Exogenous adenosine may trigger complex autonomic responses by stimulating carotid chemoreceptors, sympathetic nerve activity, and ventilatory drive.[29,61] Marked increase in ventilatory drive could in turn attenuate sympathetic nerve activity by activating thoracic stretch receptors.[62] Termination of AT with exogenous adenosine could therefore be related to reactive withdrawal of sympathetic nerve traffic. Likewise, the Valsalva maneuver and carotid sinus pressure may terminate AT independent of muscarinic cholinergic receptor activation. These effects might be attributed to a decrease in venous return to the heart with reduction of myocardial stretch.[30] If the antiarrhythmic effects of adenosine and vagal maneuvers on AT are related to these latter effects (and not to activation of their respective myocardial receptors and inhibition of adenylate cyclase), then termination of tachycardia with adenosine and vagal maneuvers may be unrelated to specific cellular mechanisms of AT such as adenosine and acetylcholine-sensitive potassium channel and cAMP-mediated triggered activity. Therefore, the response of AT to endogenous adenosine and acetylcholine (both inhibit adenylate cyclase through binding to their respective receptor) was studied to circumvent both autonomic and noncardiac receptor effects. In this study, intravenous dipyridamole at the dose used elevated endogenous interstitial adenosine levels but probably had minimal clinical effect on reflex autonomic nervous activity. This is supported by findings of minimal effect on systolic or diastolic blood pressure during AT (<10 mm Hg) without acceleration of the rate of AT. Aminophylline, an adenosine receptor antagonist, completely reversed the antiarrhythmic effects of dipyridamole, resulting in reemergence of sustained AT. Although methylxanthines can also inhibit phosphodiesterase and stimulate release of norepinephrine from nerve terminals and the adrenal medulla, these effects occur at levels at least 20-fold greater than that required to antagonize the adenosine A_1 receptor.[63] Therefore, the antiarrhythmic effects of dipyridamole on AT in this study are related to its elevation of endogenous adenosine and its effect at the myocardial adenosine A_1 receptor. Previous studies of ventricular myocytes showed that adenosine had no effect on quinidine-induced early afterdepolarizations or ouabain-induced cAMP-independent delayed afterdepolarization.[64] Catecholamines may also mediate triggered activity through a mechanism that was independent of cAMP and could enhance α-receptor effects.[65] However, adenosine did not antagonize the α-receptor–mediated effects.[66] These data as well as the results of this study suggested that termination of AT related to triggered activity by adenosine is probably a mechanism-specific response related to adenosine. This study showed that the cholinesterase inhibitor edrophonium could terminate AT, and the reversal of edrophonium's effects could be demonstrated by blocking the muscarinic cholinergic receptor with atropine. These data were consistent with a muscarinic cholinergic receptor–mediated

process for vagal termination of AT related to triggered activity.

Reentrant AT

Haines and DiMarco[12] found that adenosine was not effective in 13 of 14 patients with reentrant AT. However, the following findings could not support the proposal that the mechanism of AT in that study was caused by reentry: (1) atrial tachycardia could not be reproducibly initiated in seven patients (50%); (2) five patients needed antiarrhythmic drugs to facilitate termination by atrial pacing; (3) single or double extrastimuli were not effective in terminating any AT; (4) entrainment criteria were not demonstrated in all the AT; and (5) four patients had three AT cycle lengths with different P-wave morphologies, and no detailed study about the mechanism or definite mapping of these AT exit sites was performed to exclude the possibility of multifocal atrial tachycardia caused by abnormal automaticity or other mechanisms. Furthermore, a high incidence of associated atrial flutter-fibrillation (68%) was found in that study.[12] Josephson[11] reported that adenosine was effective in some patients with reentrant AT, and several case reports found that adenosine could terminate reentrant AT and atrial flutter.[67-69] This study found that adenosine had a dose-dependent effect on reentrant AT and could terminate 24 of 27 cases of reentrant AT. It is possible that the electrophysiological properties of atrial tissues might be different among these studies, causing the responses to pharmacological therapy to be different.[11,47,48,50,67-69]

Acetylcholine could induce arrhythmogenic effects in rabbit atrial tissues perfused with acetylcholine.[70,71] It was demonstrated that acetylcholine shortened action potential duration,[23,72] decreased the wavelength, and induced the creation of reentrant circuits by decreasing their size.[70-72] In this study, vagal maneuvers and edrophonium terminated reentrant AT in only two patients, and action potential duration did not show significant changes in the other patients. Josephson[11] also found that some reentrant ATs were responsive to carotid sinus pressure. It was unknown whether higher vagal tone or acetylcholine level could shorten action potential duration with more hyperpolarization of atrial tissues and terminate AT. However, degeneration of reentrant AT to atrial flutter-fibrillation would be possible, and different densities of parasympathetic innervations of the atrial tissues might result in different effects.

Automatic AT

In contrast, AT that was dependent on an automatic mechanism was less sensitive to perturbations that inhibited adenylate cyclase. It has been demonstrated that the L-type calcium current and funny current were modulated by a β-adrenergic agonist, and the two currents were responsible for automatic AT.[60] Acetylcholine could have a profound effect on calcium current and can also inhibit funny current by inhibiting basal adenyl cyclase activity.[73,74] Belardinelli et al[75,76] also found that adenosine (10 μmol/L) could significantly activate the adenosine-sensitive potassium channel without any measurable effect on calcium current; however, adenosine could reduce calcium current and inhibit funny current when the currents were enhanced by

isoproterenol. In isolated preparations, adenosine transiently suppressed but did not terminate enhanced automaticity, and adenosine had no effect on abnormal automaticity whether or not it was dependent on catecholamine stimulation.[77] In this study, a large dose of adenosine only transiently suppressed AT, and a rebound effect with acceleration of AT rate followed. Vagal maneuvers also showed an effect similar to that of adenosine on automatic AT. Because of limitations of the study technique, a detailed and definable mechanism governing normal or abnormal automaticity and the effects of adenosine and vagal maneuvers on automatic AT were not clear.

Radiofrequency Ablation of Atrial Tachycardia

Poor control of AT may ultimately necessitate aggressive therapy. Antiarrhythmic drugs have shown short-term effects, but the long-term outcome has been disappointing as a result of recurrent arrhythmia or drug side effects. In this study, oral medication before ablation varied widely among the study patients, and a definite relation between oral and intravenous drug effects will be obtained after study of a larger group. Direct-current ablation carried a potential risk of perforation through the relatively thin atrial wall. Radiofrequency lesions at the atrial level have been shown to produce transmural necrosis that heals with a well-organized fibrous scar and have not been associated with significant risk for either early or late perforation.[78] Radiofrequency ablation is also free of the barotrauma and catheter fling seen with direct-current ablation, further decreasing the likelihood of acute atrial trauma.[78] In patients with reentrant AT, concealed entrainment with a long stimulus–to–P-wave interval was demonstrated during pacing near the earliest activation site, and a short stimulus–to–P-wave interval was demonstrated during pacing at the exit site, suggesting the existence of an area of slow conduction. Radiofrequency energy delivered to the exit site could eliminate AT. Furthermore, Feld et al[79] reported that a combination of endocardial activation mapping and entrainment pace mapping could determine a critical area for radiofrequency ablation of common-type atrial flutter. In patients with AT not related to reentry, a combination of pace mapping and finding of earliest atrial activation would be suitable. Furthermore, the success rate and radiofrequency pulse number were similar in the three groups.

In the present study, fluoroscopy and procedure times were similar to the times reported for radio frequency ablation of accessory atrioventricular pathways or slow atrioventricular nodal pathways in our laboratory.[39] Innovations in mapping catheter design may permit faster simultaneous mapping of multiple atrial sites; more experience with this ablation technique and laboratory equipment available with the capacity for pulsed fluoroscopy at slow frame rates will shorten procedure time and reduce radiation exposure time for both the patient and operator. Unlike the accessory pathways and slow atrioventricular nodal pathways, accurate localization of an atrial focus must involve three-dimensional mapping and is further confounded by the absence of reliable electrogram markers (such as accessory pathway or slow atrioventricular nodal pathway activation potentials), apart from local activation

time and entrainment pace mapping. Despite these difficulties, radiofrequency ablation was successful in 33 of 34 patients, who remained in sinus rhythm without medications. Radiofrequency ablation of atrial tissue could be recommended as an alternative to medication or surgery in patients with various types of AT.

Limitations

Several study limitations were noted. First, this institution had a predominance of male and older patients in the study population. Second, resetting response patterns from different pacing sites might obtain different results. Third, endogenous interstitial adenosine and acetylcholine levels were not measured, and total autonomic blockade was not performed in this study. It is possible that both autonomic and noncardiac receptor effects were not really circumvented. Fourth, the differential diagnosis between automatic AT with exit site near the sinus node and paroxysmal sinus tachycardia deserves further study.

Conclusions

This study demonstrated the diverse mechanisms of AT in adult patients; the major mechanism was reentry. However, the serial electropharmacological responses were not obtained in the pediatric patients. Although radiofrequency ablation could achieve a high success and low recurrence rate in patients with various types of AT, further detailed study about the electropharmacological mechanism will be necessary.

Acknowledgments

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To Whom It May Concern:

The HTCC Coverage determination concerning catheter ablation procedures for supraventricular tachyarrhythmias (20130517B) in my opinion is flawed and reflects the committee's lack of understanding of the nature of cardiac arrhythmias and the ablative process.

While the committee correctly understands that reentrant arrhythmias are readily ablatable, they fail to understand that non-reentrant arrhythmias are equally amenable to ablative therapy and is frequently the only treatment to control these arrhythmias since many of them are not treatable effectively by pharmacologic means. A non-reentrant arrhythmia reflects atrial myocardial cells that fire automatically. This is usually a very localized focal event and with appropriate mapping techniques, electrophysiologists are able to identify that focus and ablate the rogue cells, rendering the patient free of their arrhythmia. Often times, patients with these ectopic atrial tachycardias develop secondary cardiomyopathy. Controlling this arrhythmia often times results in normalization of the patient's heart function. The decision to not cover ablation of these arrhythmias, in my opinion, reflects the committee's failure to understand the nature of these non-reentrant supraventricular tachycardias.

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If you have any questions I would be happy to respond.

Sincerely,

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From:	Harwood, Mark D <Mark.Harwood@providence.org>
Sent:	Friday, June 21, 2013 1:12 PM
To:	HCA ST Health Tech Assessment Prog
Subject:	SVT ablation

Follow Up Flag:	Follow up
Flag Status:	Completed

Dear Health Care Technology Clinical Committee,

I'm writing in regard to the assessment on ablation of supraventricular tachycardia. I'm shocked to hear that you concluded that there is no benefit to the ablation of non-reentrant atrial arrhythmias. I hope that this was merely an oversight on the part of the committee and not a true belief that this procedure does not benefit the people of Washington. Just within this last month, I have ablated a young father who was so incapacitated by his atrial tachycardia that he submitted for disability. He had failed multiple medications and had no other options for treatment. I was able to safely and successfully ablate the rhythm (which was non-reentrant!) and he is already feeling better with plans to get to work soon. Is that not beneficial?

Beyond the example of individuals that receive a potentially life altering treatment, there is plentiful data to support the ongoing practice of ablation. The field of electrophysiology is driven by data. We, as Electrophysiologists, support evidence based medicine backed by clinical trials. Cardiology and its subspecialties have for years led the way for evidence-based medicine based on data. The data is there for review. It is regretful that this conclusion was made.

The people of Washington deserve better medical care than you are suggesting. Without this procedure, there will be increased Emergency Room visits, increased demand on overwhelmed primary care providers, higher morbidity and poor workplace performance – perhaps none at all. I fear that you are trying to prevent a problem by not paying for the treatment. That is illogical and ultimately more costly.

Thank you for your time,

Mark Harwood, MD

Masters, Christine V. (HCA)

Dear Sir/Madam,

I write as a concerned practitioner about the proposed rules regarding catheter ablation procedure for SVT, specifically regarding the decision not to cover ablation for atrial tachycardia. Ablation for atrial tachycardia is concerned standard of care for patients who have failed antiarrhythmic therapy. In fact, guidelines from the US and European cardiology societies say that catheter ablation for recurrent, symptomatic, focal atrial tachycardia is a Class I recommendation, and is also a class I recommendation for asymptomatic or symptomatic incessant atrial tachycardia (1). Success rate for this procedure according to a pooled analysis was 86%, and this is prior to the current technology that likely increases success rate. Many of my patients have had durable success with ablation for focal atrial tachycardia.

I respectfully ask you to reconsider this decision.

Sincerely,

Jordan Prutkin, MD, MHS, FHRS

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Washington State Health Care Authority
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Dear Ladies and Gentlemen:

I am an electrophysiologist in Spokane, WA.
I am responding to a public comment regarding catheter ablation procedures for supraventricular tachy arrhythmias, as Washington State Health Care Authority is proposing limiting catheter ablation for SVT to reentry arrhythmias only.

This approach will prohibit most appropriate therapy to many patients with heart rhythm problems, specifically, ectopic atrial tachycardias and atrial fibrillation.

Patients with ectopic atrial tachycardias, just like patients with reentrant types of SVT, have debilitating symptoms. Medical therapy is ineffective for most of them. If curative ablation is not an option, most will end up making frequent Emergency Room visits, many will be forced to seek disability, and some will develop tachycardia induced congestive heart failure.

Atrial fibrillation is a highly prevalent arrhythmia, and many affected patients experience severe and disabling symptoms. Antiarrhythmic drugs are ineffective, not tolerated, or contraindicated due to safety concerns for over 60% of patients. Besides, risk of antiarrhythmic drug toxicity / proarrhythmia / sudden death even in appropriately selected patients exists and should not be underestimated.
While we currently recommend catheter ablation only to symptomatic patients with atrial fibrillation, benefit of the intervention probably extends far beyond symptom control.

Up until several years ago, we thought that atrial fibrillation is a non-life threatening condition.
This misconception was based results of AFFIRM study (1), where atrial fibrillation patients were randomized to rate control versus rhythm control with antiarrhythmic drugs, and no mortality difference was seen between the groups.
Antiarrhythmic drugs helped maintain sinus rhythm in about 30% of patients.
Follow up analysis if the study (2) was eye opening and showed the following hazard ratios for mortality:
> 0.54 in patients in sinus rhythm
> 1.41 in patients on rhythm control drugs
The explanation for the findings is that beneficial effect of sinus rhythm maintenance in those 30% of patients was offset by toxic effect of the medications, and, therefore, no overall mortality difference was seen between the rate control and rhythm control groups.

Retrospective studies demonstrated improvement in risk of mortality, stoke, and dementia in patients who underwent catheter ablation compared to medically treated patients (3,4).

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Two prospective studies showed regression of LV dysfunction and congestive heart failure in atrial fibrillation patients, and the benefit was present and pronounced even in patients with appropriate rate control at baseline (5,6). One randomized trial demonstrated superiority of catheter ablation to biventricular pacing / ablation of the AV node in patients with congestive heart failure and atrial fibrillation (7).

We understand that considerations of cost are also important. There is at least one study that addressed the issue (8). Catheter ablation as first line therapy was compared to antiarrhythmic drug trial as first line, and at 2 years, cumulative cost was same in both groups.

I hope that you give this a thoughtful consideration, and we continue to provide our patients with most appropriate care.
I will be happy to discuss this issue further,

Best Regards,
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6. Journal of Cardiovascular Electrophysiology, 2007, vol. 18, p. 9
7. New England Journal of Medicine, 2008, vol. 359, p. 1778
8. Journal of Cardiovascular Electrophysiology, 2009, vol. 20, p. 7

Washington State Health Care Authority   June 21, 2013

Health Technology Assessment

I am writing to request that you consider revising your recent decision to exclude "other, non-reentrant SVTs" from coverage for catheter ablation per your announcement of May 17,2013. As you have previously heard from several of my colleagues this decision comes as a surprise since these rhythm disorders were not specifically addressed in your committee's previous review published April 17, 2013.

The diagnosis of a "non- reentrant" form of SVT is difficult for a cardiac electrophysiologist to make. Surface ECG findings are nonspecific and even findings obtained during extensive electrophysiologic studies can be difficult to interpret and differentiate automatic and triggered activity from micro reentry.

These rhythm disorders account for a relatively small proportion of patients (5-15%) referred for consideration of catheter ablation but they affect individuals of all ages and are often sustained (lasting days at a time), highly symptomatic and difficult to treat with antiarrhythmic drug therapy. They have a significant impact on patients and their families causing days off work, inability to perform normal activities of daily living and repeated Emergency Room visits and hospitalizations. Three dimensional mapping systems as well as improved ablation catheter technologies have resulted in a significant improvement in the outcomes from ablation in this group of patients. Excluding these patients from catheter ablation therapy seems arbitrary and short sighted.

Thank you for your consideration.

Sincerely,

Robert D Swenson MD, FACC

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