Infective endocarditis: An atlas of disease progression for describing, staging, coding, and understanding the pathology

Gösta B. Pettersson, MD, PhD,^a Syed T. Hussain, MD,^a Nabin K. Shrestha, MD,^b Steven Gordon, MD,^b Thomas G. Fraser, MD,^b Khalid S. Ibrahim, MD, PhD,^a and Eugene H. Blackstone, MD^{a,c}

Supplemental material is available online.

The clinical picture of infectious endocarditis (IE)—its microorganisms, diagnostic criteria (Duke and modified Duke criteria),^{1,2} involved valve, native versus prosthetic valve, and complications—has been well described. Less well described is the pathologic stage. Although recognized as prognostically important, the pathologic description has often been limited to the presence or absence of vegetations and peri- and paravalvular abscess.^{3–5} From our large experience^{6,7} and long-standing interest, we have developed a deep understanding of the pathologic features of IE. Thus, we wished to share this knowledge and try to systematize and standardize the description and staging of IE. Therefore, we have described our understanding of the pathophysiology and pathologic features of IE, translated this into an instrument for recording and coding, and illustrated the main pathologic concepts in an atlas.

DOCUMENTATION

The basis of the present review was the observations and photographic documentation of IE pathologic features in the operating room. Photographs were selected strategically according to the concept illustrated and at different phases of the operation to illustrate essential points. Relevant frames from echocardiographic examinations have been added. Because obtaining good images of aortic valve pathologic features is easier than obtaining good images

of mitral valve pathologic features, photographs of aortic valve IE dominate in the atlas. This also reflects the prevalence of the disease and the diversity of the aortic pathology. Only a limited selection of illustrations has been provided in print. The material available online is more comprehensive and includes portions of echocardiographic videos; it is essential for a better understanding of this paper.

PATHOPHYSIOLOGY

The key to understanding IE is appreciating the pathologic progression.^{8–10} Circulating organisms, bacteria or other— injected, absorbed from the mouth or gastrointestinal tract, or entering the circulation from an infection focus—adhere to damaged areas of the endocardium (valves, ventricular septal defect), endothelium (patent ductus), or foreign material in the bloodstream (prosthetic heart valves, pacemaker leads).

Such damaged areas often have depositions of platelets, fibrin, or clots (sometimes termed “nonbacterial endocarditis”),⁸ facilitating adherence, protection, and growth of the organisms. The organisms multiply and develop colonies, attracting fibrin deposits and cells to form vegetations. Vegetations are the main source of embolization. The risk of embolism is related to the vegetation location, size, and mobility¹¹; however, embolic material can also originate from other sources, such as abscess cavities. Aortic valve vegetations can flip back and forth and cause secondary lesions on the anterior mitral valve leaflet (common) or aortic wall (uncommon), termed “kissing lesions.” The organisms produce and release enzymes that disintegrate tissue, primarily valve cusps and leaflets, and result in regurgitation. The enzymes are organism specific with regard to their tissue specificity and efficiency.

When tissue disintegration involves the valve annulus, the infection invades extravascular areas; this has been termed “invasive disease.” Invasive disease develops in stages: cellulitis, abscess, abscess cavity, and, finally, pseudoaneurysm formation. Invasion is defined by its location and extent of spread outside the valve annulus, described by the percentage of the circumference of the annulus involved and the depth and size of the cavities. Internal fistulas, perforations, and heart block constitute the specific consequences of invasion.

IE pathologic features can be further characterized by the involved valve: whether the valve is native (ie, normal, diseased, or diseased and repaired) or prosthetic, and by

From the Department of Thoracic and Cardiovascular Surgery,^a Heart and Vascular Institute, Department of Infectious Disease,^b Medicine Institute, Department of Quantitative Health Sciences,^c Research Institute, Cleveland Clinic, Cleveland, Ohio.

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Address for reprints: Gösta B. Pettersson, MD, PhD, Department of Thoracic and Cardiovascular Surgery, Heart and Vascular Institute, Cleveland Clinic, 9500 Euclid Ave, Mail stop J4-1, Cleveland, OH 44195 (E-mail: petterg@ccf.org).

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Abbreviations and Acronyms

IE = infectious endocarditis

PVE = prosthetic valve endocarditis

activity (ie, active or remote—active unless sterile after treatment or spontaneously).

With this understanding of IE, we have developed a form for coding the pathologic features ([Appendix E1](#)), with the definitions provided in [Appendix E2](#). The coding was determined using all available information but mainly the operative findings and pre- and intraoperative transesophageal

echocardiographic findings. We have proposed an extensive coding schema, in part to obtain detailed information that might in time be consolidated into a simpler form. In the present atlas, we have proposed broad concepts based on the consolidation of the data obtained. We have also proposed this schema as a starting point for the future development of consensus around the pathologic features of IE.

ATLAS

The concepts in the atlas are generally self-explanatory. Only some salient factors have been explained here.

Concept 1

Vegetations on endothelial defects or injuries are the primary manifestation of IE ([Figure 1](#) and [Figure series E1](#)).

FIGURE 1. Vegetations on endothelial defects or injuries are the primary manifestation of infective endocarditis. A, View of active aortic valve endocarditis (native valve; NVE) showing excised aortic valve cusps with vegetations and cusp disintegration. B, View of remote NVE showing excised aortic valve cusps with healed vegetations but residual cusp defects (*arrows*). L, Left; NC, noncoronary; R, right. C, Infected bioprosthesis with large vegetations, which confers a high embolic risk. D, Infected mechanical valve with vegetations (*arrows*) above and below prosthesis attached to junction of valve and tissue covering of the sewing ring at multiple sites. E, Kissing lesion with perforation of anterior mitral valve leaflet (*arrow*). These lesions are often amenable to autologous pericardial patch repair after debridement, as shown. F, Uncommon kissing lesion showing mycotic aneurysm in posterior wall of ascending aorta (*arrow*) above aortic valve in a patient who had a large mobile vegetation.

FIGURE 2. Extra-aortic invasion of native valve endocarditis is localized (see also [Figure E2](#)). A, Before invasion. Superficially damaged endocardium present in a triangle beneath the right–noncoronary cusp commissure but still not breaking the aortic wall. B1, Invasion with cellulitis outside the aortic wall. The invasion might not be obvious because of the presence of an infected aortic valve with rheumatic disease displaying small vegetations on the right–left commissure. The immediate appearance did not suggest invasion or extra-aortic extension. B2, Same aortic valve after peeling off the aortic wall disclosed the true extent of the extra-aortic invasion. Incomplete debridement is associated with a high probability of recurrent infection. C, Invasive disease healed by antibiotics, leaving an extra-aortic horseshoe-shaped pseudoaneurysm. The aortic root has been laid open by complete debridement and pulmonary autograft harvest. An initial stitch was placed to begin suturing the autograft to the base of the mitral valve. *LCA*, Left coronary artery; *MV*, mitral valve; *NCS*, noncoronary sinus; *RCA*, right coronary artery; *RVOT*, right ventricular outflow tract.

Vegetations on native valves almost invariably involve the cusps or leaflets. Valve salvage requires arresting the destructive bacterial activity before integrity is lost; antibiotic treatment can only prevent additional bacterial growth, but cannot improve valve function. Although prosthetic valve endocarditis (PVE) usually involves the sewing ring, and the infection is most often invasive, the visible manifestation will still be vegetations. In the case of mechanical valve PVE, the vegetations will typically attach to the sewing ring in the interface between the valve housing and sewing ring or to clots in a low-flow area of the prosthesis. Bioprosthetic PVE may be limited to vegetations on the cusps; such cases can be cured by antibiotics. Bioprosthetic cusps also seem to be more resistant to disintegration by bacterial enzymes than native valve cusps.

Concept 2

Extra-aortic invasion of native valve endocarditis is localized ([Figure 2](#) and [Figure series E2](#)).

Although invasion can occur anywhere along the aortic valve annulus, subcommissural invasion occurs most frequently. Often the site of annulus penetration is small, hiding a widely spread extra-aortic infection that requires unroofing of the entire infected area for adequate

debridement. Enzymatic degradation has a preference for connective tissue and fat while respecting muscle; thus, proximal loss of substance is unusual. This important fact is illustrated in several of our picture series. If an invasive infection has been successfully treated, the infected extra-aortic area will eventually be transformed into a pseudoaneurysm, which, from a small entry, can extend all around the aorta—a horseshoe abscess. (The concept “abscess” has often been misused to describe a residual cavity after an abscess has ruptured and drained; once a cavity has formed and has been irrigated by blood, there is no longer any pus.) During surgery, it is unusual to find large collections of pus; however, when operated on in the acute phase, infected areas are characterized by necrotic tissue with small pockets of pus. Because invasion—with extracardiac cellulitis, abscess, and cavity formation—almost exclusively occurs on the left side of the heart, the higher blood pressure and presence of oxygenated blood might be related to the development and progression of invasive pathologic features.

Concept 3

Extra-aortic invasion of PVE is often circumferential ([Figure 3](#) and [Figure series E3](#)).

FIGURE 3. Extra-aortic invasion of prosthetic valve endocarditis is often circumferential. A, In a patient with an infected prosthetic sewing ring, the infected prosthesis can often be easily removed because the tissue holding the sutures has been disintegrated by bacterial enzymes. B1, Infected mechanical aortic valve prosthesis showing circumferential involvement of the sewing ring, with some vegetations attached to both sides of the prosthesis. B2, Although the aortic annulus has disintegrated with aortoventricular discontinuity, the left ventricular outflow tract (LVOT) appears well preserved after debridement. Proximal destruction of the muscle is uncommon, even in cases infected by aggressive organisms such as *Staphylococcus aureus*. C, Composite graft prosthetic valve endocarditis. Although the graft is circumferentially infected and surrounded by pus, the LVOT is well preserved and intact after radical debridement. LCA, Left coronary artery; RCA, right coronary artery.

PVE usually involves the sewing ring and, in contrast to native valve endocarditis, the invasion is often circumferential. Deep penetration with cellulitis and formation of abscesses and abscess cavities can occur anywhere along the annulus; however, large root abscesses will preferentially develop posteriorly and to the left, under the pulmonary trunk. Although eradicating the infection with antibiotics has occasionally been successful, periprosthetic regurgitation and pseudoaneurysm formation will be the end result. Severe regurgitation, hemolysis, and the risk of thromboembolic complications may still require surgery. With less aggressive organisms (eg, enterococci), tissue disintegration causing valve dehiscence could be slow and valve infection difficult to diagnose and verify.

Concept 4

Heart block is caused by bacterial destruction of the atrioventricular node and bundle of His (Figure 4 and Figure series E4).

The most common cause of heart block is bacterial invasion from the aortic root working its way from posterior aortic root invasion into the right atrium and triangle of Koch, destroying the atrioventricular node and upper end of the bundle of His. This mechanism is true for both native valve endocarditis and PVE, including allografts and

pulmonary autografts. Destruction of the conduction bundle at a lower level is uncommon, but has occasionally occurred in PVE.

Concept 5

The organisms are not equally destructive (Figure 5 and Figure series E5).

An enterococcal infection is sometimes minimally invasive, causing only formation of small vegetations on a prosthesis within 1 year, with recurrent episodes of sepsis. In contrast, *Staphylococcus aureus* can dissolve annular tissue within a couple of weeks, causing dehiscence of a prosthesis. Fungal infections are characterized by large vegetations but with minimal invasion and tissue destruction.

Concept 6

Congenital heart defects, repaired or not, are associated with increased risk of endocarditis (Figure series E6).

The turbulence and jets associated with abnormal flow patterns in patients with congenital heart disease damage the endocardium, facilitating bacterial adherence.

Concept 7

Mitral valve endocarditis has some specific features related to its anatomy and degenerative pathologic features (Figure 7 and Figure series E7).

FIGURE 4. Heart block is caused by bacterial destruction of the atrioventricular (AV) node and bundle of His. A1, Native valve endocarditis with sepsis and heart block. Looking into the right atrium reveals vegetations close to the Koch triangle and AV node. A2, After complete debridement of the infected and necrotic tissue, the proximity of infection to the AV node can be seen. Also note that a portion of the right coronary artery (RCA) was laid bare; we have not seen destruction or thrombosis of a coronary artery even when surrounded by infection. B1, Prosthetic valve endocarditis with sepsis and heart block, with perforation visible in the right atrium. B2, After debridement, destruction in the location of the AV node can be seen. This infection has worked its way around the aorta counterclockwise over an extended period, displaying a pseudoaneurysm stage anteriorly and an active cellulitis stage posteriorly and into the right atrium. The left ventricular outflow tract (LVOT) is intact and ready for reconstruction. CFB, Central fibrous body; CS, coronary sinus; LCA, left coronary artery; RA, right atrium; TV, tricuspid valve.

FIGURE 5. Not all organisms were equally destructive. A, Explanted bioprosthesis infected with an *Enterococcus* species. This patient had a history of 4 recurrent episodes of sepsis in the previous year. Multiple small vegetations and no valvular dehiscence explain why echocardiography had failed to provide convincing evidence of endocarditis. B, View of an explanted, partially dehiscid valve prosthesis infected with *Staphylococcus aureus*. The patient had been ill for 2 weeks. C1, Fungal endocarditis with large vegetations on the aortic valve. C2, After complete debridement, with no destruction and no invasion observed.

FIGURE 7. Mitral valve endocarditis has some specific features related to its anatomy and degenerative pathologic features. A, Mitral valve endocarditis with large vegetations on posterior mitral valve leaflet, with leaflet perforation. The patient had a history of an embolic stroke. B1, Mitral valve endocarditis associated with severe hemorrhagic pericarditis. The patient was septic and not responding to adequate antibiotic therapy. B2, After peeling the fibrinous capsule off the heart, these multiple necrotic spots, suggesting abscesses along the atrioventricular groove, became apparent. B3, Vegetations were found on the base of the P3 scallops of posterior leaflet. B4, After debridement, multiple abscess cavities in the atrioventricular groove were seen to communicate with both the left atrium and the pericardium. Intact coronary vessels bridged the infected groove. B5, Annulus defect and communication were closed from inside the left atrium using an autologous pericardial patch. The groove was allowed to drain to the pericardium.

Vegetations can form on both leaflets and chordae. Regurgitation can result from a loss of leaflet integrity or chordal rupture, or both. Mitral annular calcification is frequently the starting site of both infection and invasion. However, invasive disease is less common with mitral than with aortic valve IE and, when it occurs, will often be shallow. Anterior annulus invasion leads to destruction of the subaortic curtain; invasion posteriorly leads to entry into the atrioventricular groove and separation of the atrium from the ventricle. Complete debridement and drainage of the infected area to the atrium or pericardium is difficult to accomplish. Surgical reconstruction instead often means sealing off the infected and debrided cavity, with a resulting increased risk of recurrent infection.

Concept 8

Right-sided IE is characterized by vegetations and disintegration of valve leaflets or cusps, but almost never by invasion ([Figure 8](#) and [Figure series E8](#)).

Infection of the tricuspid valve is more common than infection of the pulmonic valve. Infected pacemaker and defibrillator leads and intravenous drug abuse play important roles in the development of right-sided infections.

Concept 9

Complete debridement is the basic principle of successful reconstructive surgery for invasive IE ([Figure 9](#) and [Figure series E9](#)).

FIGURE 8. Right-sided infective endocarditis is characterized by vegetations and disintegration of valve leaflets or cusps but almost never invasion. A, Large vegetations on tricuspid valve in an intravenous drug abuser. Invasive disease with extravalvular destruction is unlikely for right-sided endocarditis. B, Infected pacemaker lead in right atrium.

The first condition for success is complete debridement of all infected and necrotic tissue and foreign material, as illustrated by many of the surgical pictures. To reconstruct the aortic root, an aortic allograft is preferred, with minimal use of additional foreign material. Mitral valves should be repaired whenever possible.

Study Limitations and Conclusions

The present atlas is a personal collection and selection of views illustrating concepts we believe are essential to understanding IE—its pathologic features, complications,

and surgical treatment. We have not claimed that it is complete or indisputable. Rather, we have provided a starting point for developing a standardized reporting system of the extent and pathologic features of IE to better understand and compare the results of IE surgery. Combining this information with available tools such as the Society of Thoracic Surgeons Adult Cardiac Surgery Database, as reported by Gaca et al,¹² would allow for uniformity of reporting, facilitate better correlation of the pathologic findings with the clinical presentation, and, possibly, lead to the optimal choice of operation for the best patient outcomes.

FIGURE 9. Complete debridement is the basic principle of successful reconstructive surgery for invasive infective endocarditis. A1, After complete debridement of all infected tissue, the right atrium is reconstructed with autologous pericardium. A2, An aortic allograft is sutured to the left ventricular outflow tract (LVOT) with running monofilament suture. A3, Allograft is tied down and well seated, allowing the debrided infected areas to communicate with and drain to the pericardium. B, Aggressive debridement of the pathologic features is key to curing the infection. In some patients with double prosthetic valve endocarditis, the aorto-mitral curtain will be disintegrated or can be divided by choice to achieve perfect exposure. Dividing the superior vena cava will increase the possible exposure further. LCA, Left coronary artery; RA, right atrium; RCA, right coronary artery.

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APPENDIX E1. CODING FORM FOR INFECTIVE ENDOCARDITIS

The following form should be completed using all available information, mainly the operative findings and pre- and

intraoperative transesophageal echocardiographic results. The definitions used are presented in [Appendix E2](#).

	Aortic			Mitral			Tricuspid			Pulmonic		
	Native	Native Rep.	Prosthetic	Native	Native Rep.	Prosthetic	Native	Native Rep.	Prosthetic	Native	Native Rep.	Prosthetic
Affected Valve:												
Disease Activity:	Active	Remote		Active	Remote		Active	Remote		Active	Remote	
Anuloplasty Ring:				Y	N		Y	N				
Vegs present:	Y	N		Y	N		Y	N		Y	N	
Largest Size (mm)												
Location (check all locations):												
Rt. cusp												
Lft. cusp												
Non-cor. cusp												
Ant. Leaflet												
Post leaflet												
Septal leaflet												
Ant. Chord												
Post Chord												
Septal Chord												
Valve Integrity												
Abnormality:	Y	N		Y	N		Y	N		Y	N	
Rt. cusp	None	Perf	Tear									
Lft. cusp	None	Perf	Tear									
Non-cor. cusp	None	Perf	Tear									
Ant. Leaflet				None	Perf	Tear	None	Perf	Tear			
Post leaflet				None	Perf	Tear	None	Perf	Tear			
Septal leaflet							None	Perf	Tear			
Ant. Chord				None	Rupture		None	Rupture				
Post Chord				None	Rupture		None	Rupture				
Septal Chord							None	Rupture				
AML Jet Lesion:	Y	N										
Invasion/Adv.Disease:	Y	N		Y	N							
Invasion location:	Ant.	Left	Post.	Ant.Lat.	Post.med.							
Invasion/Abscess Stage												
(note all that apply):	Cellulitis	Abscess		Cellulitis	Abscess							
	Abscess Cavity	Pseudoaneurysm		Abscess Cavity	Pseudoaneurysm							
Circumferential Extent	<1/3	1/3-1/2	full circumference	<1/3	1/3-1/2	full circumference						
Intervalvular fibrosa destruction:				Y	N							
AV node/bundle destruction (if known AV block):				Y	N							
Cavity depth (if appl.):	Shallow	Deep (>1cm)		Shallow	AV-groove involvement							
Fistula destination (if appl.):	to RA	to LA	to RV	to RA	to RV	perf. to pericardium						
Prosthetic Valve (check all that apply):												
Restriction of disc/ball												
Paraprosthetic leak												
Unstable/Rocking												
Asc. Aortic graft infection												
Infected Pacer Leads (check all that apply):												
Infected:												
Rt. Atrium:												
Rt. Ventricle:												
Tricuspid Valve:												
Pocket:												
Epicardial:												
Other site (vegetations):	Y/N			Largest Size (mm):								

APPENDIX E2. DEFINITIONS FOR INFECTIVE ENDOCARDITIS CODING FORM

Affected valve: note whether the affected valves are native (including Ross/autograft), native previous repair, or prosthetic (mechanical prosthesis, bio-prosthesis, or allograft)

Disease activity

Active: vegetations, pus, or necrotic tissue

Remote: no signs of active infection

Annuloplasty ring present: annuloplasty ring present on either mitral or tricuspid valves

Vegetations present: any vegetation seen on affected valves

Largest vegetation size: largest size in millimeters of any vegetation for each affected valve

Location of vegetations: check all locations that apply for each valve

Integrity abnormality (relevant for cusps, leaflets, and chord): for each location affected, note the integrity of the cusp, leaflet, and chord

Cusps/leaflets

None: no integrity abnormality as a result of infection

Perforation: preserved free margin as a result of infection

Tear: torn or stretched margin as a result of infection

Anterior mitral leaflet jet or kissing lesion: infectious lesion on anterior mitral valve leaflet caused by regurgitant jet ("jet" or mobile vegetation ["kissing"])

Chords

None: infection has not affected the chordae

Rupture: chord is ruptured as a result of infection

Invasive disease: infectious process extends beyond cusps or leaflets into annulus or surrounding structures

Invasion location: if invasive disease has been documented, note the location

Aortic valve

Posterior: below commissure between left and non-coronary cusps, mid-left to mid-noncoronary cusp

Left: below commissure between left and right, mid-left to mid-right cusp

Anterior: between right and noncoronary cusp, mid-right to mid-noncoronary cusp

Mitral valve

Anterior lateral: anterolateral side of posterior mitral valve leaflet

Posterior medial: posteromedial side of posterior mitral valve leaflet

Invasion/abscess stage: if invasive disease is present, note all stages; >1 stage can be present

Cellulitis: preabscess cellulitis, invasion without collections of pus or microabscesses

Abscess: macroscopic collection of pus

Abscess cavity: cavity with debris and clots suggesting active endocarditis

Pseudoaneurysm: endothelialized cavity without pus

Circumferential extent of invasion: if invasive disease, determine the extent of infection or abscess in regard to the circumference of the annulus (include any and all abscess stages)

Intervallular fibrosa (subaortic curtain) destruction: involvement of subaortic curtain (contiguous aortic and mitral valve annuli)

Atrioventricular (AV) node/bundle destruction: if patient has AV block and infectious pathologic destruction of AV node/bundle is present, note whether cellulitis or tissue necrosis is in the area

Invasive cavity depth: if a cavity is noted, note the depth, defined as shallow or deep (> 1 cm)

Fistula destination: if a fistula is noted under the involved valve, note the destination of the fistula

Prosthetic valve: if the patient has an infected prosthetic valve, note all abnormalities

Restriction of disc/ball: restriction of mechanical valve motion by vegetation, infected thrombus, or pannus

Para- or periprosthetic leak

Unstable/rocking: excessive mobility caused by an undermined annulus

Infected pacer leads

For endocardial pacing leads, note sites of infection

For epicardium leads, note whether epicardium is infected

For the pacer pocket, note whether the pocket is infected

Other sites: note whether any vegetations have been found at other sites (patent foramen ovale, atrial septum, right atrial wall, right ventricular outflow tract, left atrium), and the largest size (in millimeters)