Cardiovascular Disease (CVD)

Heart Failure and Non-ischemic Cardiomyopathies
CARDIOVASCULAR DISEASE (CAD):
Heart Failure and Non-ischemic Cardiomyopathies

INTRODUCTION

Law enforcement officers (LEOs) with known cardiovascular disease (CVD) may be capable of safe and effective job performance. However, LEOs with CVD (or CVD risk factors) are at increased risk for sudden incapacitation, jeopardizing their ability to perform critical job functions (see also LEO chapter on Essential Job Functions). Therefore, an individualized assessment of the LEO is needed to ensure safe and effective job performance.

The person conducting the evaluation must be familiar with the following:

- the physiologic demands of the critical job functions;
- CVD and its management; and
- the risk for sudden incapacitation associated with CVD conditions and CVD risk factors.

OVERVIEW OF MEDICAL EVALUATION

CVD represents several distinct medical conditions. Therefore, the evaluative criteria will be discussed in individual sections devoted to these conditions. The treating physician (preferably a cardiologist) must provide the police physician with two documents:

1) a written statement regarding whether the LEO meets the relevant criteria in the sections below regarding hypertension; and
2) relevant medical records.

HEART FAILURE (HF)

Law enforcement officers (LEOs) with mild heart failure (HF) may be capable of safe and effective job performance. However, HF increases the risk of sudden incapacitation and is associated with reduced exercise capacity, thus jeopardizing the LEO’s ability to perform essential job functions (see LEO Essential Job Functions chapter). Therefore, an individualized assessment of the LEO with HF (see Appendix A) is needed to ensure safe and effective job performance. Any underlying structural heart disease should be assessed according to the appropriate section of this document.

Two common classification systems group/stage HF patients based on severity and prognosis: 1) the New York Heart Association (NYHA); and 2) the American College of Cardiology Foundation/American Heart Association (ACCF/AHA) (see Table 1). LEOs with HF grouped as a NYHA I or staged as an ACCF/AHA A or B can return to full duty if all of the following criteria are met:

- asymptomatic;
- normal 12-lead electrocardiogram (ECG);
- normal 24-hour ECG;
- normal left ventricular function defined by a normal ejection fraction (EF) as measured by a gated blood pool scan, 2-dimensional echocardiogram with Doppler, or ventriculogram;
- normal imaging exercise stress test (EST) to at least 12 METs (see EST in Coronary Artery Disease section);
- stable regimen of cardiovascular medications over the past month;
- no disqualifying side effects from medications (see LEO Medications chapter);

Normal results on 24-hour ECG monitoring can include episodic supraventricular premature complexes, and occasional premature ventricular complexes (PVCs) – less than 30 PVCs per hour or less than 200 PVCs per 24 hours.

EF values are dependent on the imaging technique used, method of analysis, and operator. While individual testing facilities have determined their own measures of “normal,” most use a LVEF value of greater than or equal to 50%.
The above criteria should be reassessed on an annual basis unless the LEO’s HF is due to a reversible condition as described in the next paragraph.

If the LEO has HF due to a reversible condition (e.g., hyperthyroidism, obesity, inflammation, etc.), the LEO should be provided restrictions. Once the underlying condition and HF have resolved and all the above criteria are met, the LEO can return to work without restrictions. No ongoing cardiac testing is indicated for these reversible HF conditions.

LEOs with a NYHA classification II, III, or IV or an ACCF/AHA Stage C or D should be given job restrictions due to: a) the risk of sudden incapacitation, and b) the lack aerobic capacity to perform all essential job functions.

**HYPERTROPHIC CARDIOMYOPATHY (HCM)**

LEOs with HCM and a normal left ventricle size may be capable of safe and effective job performance. However, HCM is strongly associated with an increased risk of sudden cardiac death, thus jeopardizing the LEO’s ability to perform essential job functions. Therefore, an individualized assessment of the LEO with HCM is needed to ensure safe and effective job performance.

LEOs with known or suspected HCM, should be restricted unless a normal left ventricle is documented by both echo-cardiography and cardiac magnetic resonance imaging (MRI) on an annual basis (see HCM in Appendix A).

**DILATED CARDIOMYOPATHY (DCM)**

LEOs with mild DCM may be capable of safe and effective job performance. However, DCM increases the risk of sudden cardiac events. DCM is also frequently associated with reduced exercise capacity, thus jeopardizing the LEO’s ability to perform essential job functions. Therefore, an individualized assessment of the LEO with DCM is needed to ensure safe and effective job performance.

LEOs diagnosed with DCM should be restricted until a thorough medical history and work-up is completed. LEOs with DCM can be cleared for full duty if all the following criteria are met on an annual basis:

- asymptomatic;
- normal 12-lead ECG;
- normal 24-hour ECG;
- normal left ventricular function defined by a normal EF as measured by a gated blood pool scan, 2-dimensional echocardiogram with Doppler, or ventriculogram;
- normal imaging EST to at least 12 METs (see LEOs with Known CAD in Coronary Artery Disease section);
- stable regimen of cardiovascular medications for the past month;
- no disqualifying side effects from medications (see LEO Medications chapter);

**ARRHYTHMOGENIC CARDIOMYOPATHY**

Arrhythmogenic cardiomyopathy, previously known as “arrhythmogenic right ventricular cardiomyopathy” is a primary disease of heart muscle that results in fibro-fatty replacement of the right ventricle and the sub-epicardial region of the left ventricle. Patients are at high risk for ventricular tachyarrhythmias and sudden death, particularly during intensive exercise. LEOs with arrhythmogenic cardiomyopathy should be provided with restrictions.

**MYOCARDITIS**

Myocarditis is one type of cardiomyopathy due to infection, inflammation, or immune reaction. LEOs diagnosed with acute myocarditis should be restricted until ALL the following criteria are met:

- asymptomatic;
- normal 12-lead ECG;
- normal 24-hour ECG;
HEART TRANSPLANT

Most heart transplant patients will be unable to perform the essential job functions of a LEO due to their risk of sudden cardiac death, rejection, and inability to increase their heart rate due the lack of sympathetic nerve stimulation. However, a few heart transplant patients may be able to perform the essential job functions of a LEO. Therefore, an individualized assessment of the LEO who has undergone a heart transplant is needed to ensure safe and effective job performance.

LEOs having undergone a heart transplant should be restricted for 1-year post transplant surgery and can return to full duty when all the following criteria are met on an annual basis:

- normal 24-hour Holter ECG;
- normal graft function (normal LVEF) by echocardiogram;
- imaging EST to at least 12 METs (see EST in Coronary Artery Disease section);
- no implantable cardioverter defibrillator;
- if a pacemaker placed, not pacemaker dependent;
- evaluate for co-morbidities (e.g., diabetes mellitus, renal failure, cognitive impairment, etc.). If found, refer to the appropriate section of this LEO document;
- stable regimen of cardiovascular medications for the past month;
- no disqualifying side effects from medications (see LEO Medications chapter);
- under the ongoing care (at least annually) by a transplant cardiologist or cardiac transplant surgeon.

If vasculopathy or rejection is found by angiogram or endocardial biopsy, the LEO should be restricted until the issue is resolved. The LEO would still need to meet the bulleted criteria above.
APPENDIX A. HEART FAILURE AND NON-ISCHEMIC CARDIOMYOPATHIES

Heart Failure (HF)

HF is a complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood. The structural or functional impairments can result from disorders of the pericardium, myocardium, endocardium, heart valves, great vessels, or metabolic abnormalities. The disease can have a wide spectrum of left ventricle functional abnormalities ranging from normal left ventricle size and function – i.e., normal ejection fraction (EF) – to severe dilatation with markedly reduced EF.\(^1\) Any underlying structural heart disease should be assessed according to the appropriate section of this document.

The cardinal manifestations of HF are dyspnea, fatigue, and fluid retention all of which can limit exercise tolerance. Because some patients can present without signs or symptoms of volume overload, the term “heart failure” is preferred over “congestive heart failure.” HF is not synonymous with either cardiomyopathy or left ventricular dysfunction; these latter terms describe possible structural or functional reasons for the development of HF.\(^1\)

HF is diagnosed by history and physical examination. While there is no single diagnostic test, ejection fraction (EF) is a useful diagnostic and prognostic tool. Two common classification systems group/stage HF patients based on severity and prognosis: The New York Heart Association (NYHA),\(^3\) and American College of Cardiology Foundation/American Heart Association (ACCF/AHA).\(^4\)

NYHA Grouping\(^1\)

The NYHA grouping of HF patients is based on exercise capacity and symptoms (see Table 1). It is a subjective assessment by a clinician and can change over short periods of time, particularly with treatment. Although reproducibility and validity may be problematic, it is an independent predictor of mortality.\(^1,14,15\)

ACCF/AHA Staging\(^4\)

ACCF/AHA stages HF patients into four groups (A-D) according to the development and progression of the disease (see Table 1). The ACCF/AHA stages are progressive; once a patient moves to a higher stage, regression to an earlier stage of HF is not observed.\(^1\) Progression in HF stages is associated with reduced 5-year survival and increased plasma natriuretic peptide (BNP) concentrations.\(^16\)

Table 1. ACCF/AHA Stages of Heart Failure and the New York Heart Association Functional Classifications*

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<thead>
<tr>
<th>ACCF/AHA Stages of HF</th>
<th>NYHA Functional Classification</th>
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<tbody>
<tr>
<td>A At high risk for HF, but without structural heart disease or symptoms of HF.</td>
<td>I No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF.</td>
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<tr>
<td>B Structural heart disease, but without signs or symptoms of HF.</td>
<td>I No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF.</td>
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<tr>
<td>C Structural heart disease with prior or current symptoms of HF.</td>
<td>I No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF.</td>
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<td></td>
<td>II Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in symptoms of HF.</td>
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<td></td>
<td>III Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes symptoms of HF.</td>
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<td></td>
<td>IV Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest.</td>
</tr>
<tr>
<td>D Refractory HF requiring specialized interventions.</td>
<td>IV Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest.</td>
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*Adapted from Yancy, et al. 2013\(^1\)
Clinicians should routinely assess the HF patient’s potential for adverse outcome. Numerous methods objectively assess risk, including biomarker testing (e.g., BNP), and a variety of multivariable clinical risk scores. One well-validated risk score, the Seattle Heart Failure Model, is available in an interactive application that provides information about risk of mortality in ambulatory patients with HF.17

Hypertrophic Cardiomyopathy (HCM)

HCM is a relatively common genetic disorder with varying phenotypic expression.12 Mutations in more than a dozen genes encoding sarcomere-associated proteins cause HCM. MYH7 and MYBPC3, encoding β-myosin heavy chain and myosin-binding protein C, respectively, are the two most common genes involved, together accounting for about 50% of the HCM families. In about 40% of HCM patients, the causal genes remain to be identified. The histological features of HCM include myocyte hypertrophy and disarray, as well as interstitial fibrosis. The hypertrophy is also frequently associated with left ventricular diastolic dysfunction.

HCM is characterized by left ventricle that is hypertrophied, but not dilated, with preserved or increased ejection fraction.18 HCM is associated with exertional dyspnea, impaired exercise performance, pre-syncope, syncope, and sudden cardiac death. Most authoritative bodies restrict HCM patients working in safety sensitive positions due to the risk of sudden incapacitating cardiac events.19,20,21

Recent studies, however, suggest that HCM patients with normal left ventricular size are not at increased risk of incapacitating sudden cardiac events.12,22,23 Therefore, it is reasonable to return LEOs with HCM to work if both an echocardiogram and cardiac MRI reveal a normal left ventricular size. Echocardiography may miss left ventricular abnormalities in the anterolateral free wall and apex, therefore both echocardiograms and cardiac MRIs should be performed on an annual basis.12,23 An end-diastolic wall thickness of less than 15 mm is considered normal size for any portion of the left ventricle wall.19 LEOs who have undergone surgical correction for IHSS (e.g., Morrow procedure, etc.) would still need restrictions due to absence of studies demonstrating a long-term reduction in the risk of sudden incapacitating cardiac events.12

Dilated Cardiomyopathy (DCM)

DCM refers to a large group of heterogeneous myocardial disorders characterized by ventricular dilation and depressed myocardial contractility in the absence of hypertension, valvular, congenital, or ischemic heart disease.13 DCM can be subdivided by pathogenesis, such as secondary to a systemic disorder (e.g., Lupus), toxins (e.g., alcohol), infections (e.g., viral), inflammation, or an inherited disorder (typically autosomal dominant).5 In patients where no pathogenesis could be identified are termed “idiopathic” cardiomyopathy. There are some rare reversible causes of DCM, and if these are present, the LEO should be re-evaluated after the underlying condition has been treated.5

DCM is characterized by left or right ventricular enlargement with ventricular wall of approximately normal thickness and varying extent of fibrosis. Patients develop progressive heart failure (e.g., exertional dyspnea, impaired exercise performance), reduced ejection fraction, and increased risk of sudden death.5

Myocarditis

Myocarditis consists of three overlapping phases: 1) acute injury (often caused by a virus); 2) the host innate and acquired immunologic response; and 3) recovery or a transition to scar formation and DCM. The transition from acute myocarditis to chronic DCM probably occurs over months with substantial individual variation.24 Myocarditis is associated with sudden death, particularly in young athletes and those undergoing strenuous physical activity.22,23 Unlike heart failure, the risk of sudden death caused by myocarditis does not appear to correlate with the severity of the myocardial inflammation.12
REFERENCES


