DGAFMS MED MEMO NO 185

ISCHAEMIC HEART DISEASE (CORONARY ARTERY DISEASE)

INTRODUCTION

1. Coronary Artery Disease (CAD) is a significant cause of morbidity and mortality among Armed Forces personnel. Disconcertingly, there is a noticeable increase in the incidence of coronary artery disease, particularly among the younger population. This paper will outline various aspects including clinical aspects, diagnosis and management of this modern day epidemic.

CLINICAL SPECTRUM

2. CAD can manifest as the following clinical syndromes:

(a) Sudden cardiac death

(b) Acute ST segment elevation myocardial infarction (STEMI).

(c) Unstable angina and Non ST Elevation MI (NSTEMI)

(d) Chronic stable angina.

ACUTE MYOCARDIAL INFARCTION (AMI)

3. This is a commonly encountered, life threatening medical emergency, which often presents as prolonged chest pain lasting more than 30 minutes and frequently for a number of hours. The pain may radiate to left arm, epigastrium, interscapular region and lower jaw. Associated symptoms include diaphoresis, dyspnea, palpitations, profound weakness, nausea, vomiting and dizziness. While chest pain is the commonest presenting symptom of this condition, some patients including the elderly and those with diabetes mellitus can present with acute left ventricular failure (LVF), syncope or hypotension. Myocardial dysfunction, electrical instability and ventricular remodeling underlie most complications of this disorder.

4. **Evaluation of patients with AMI**.

Evaluation of patients with AMI should proceed as follows:

a. History, clinical examination and investigations including serum cardiac markers as per standard protocol.

b. Immediate ECG: - Early recognition of ST elevation MI on ECG is vital for early reperfusion therapy. If the initial ECG is non-diagnostic but the history is suggestive of ischemic pain, ECG should be repeated. Serial ECGs are extremely helpful for detection and localizing the site of AMI.

c. Portable echocardiography would be useful in patients with chest pain compatible with AMI and a non-diagnostic ECG. It can also help in excluding aortic dissection, a condition that mimics AMI but is an absolute contraindication for thrombolytic therapy.

5. **Management**.

Management of AMI consists of measures to limit infarct size and reperfusion of infarct related artery by thrombolysis or angioplasty. All patients with AMI will be admitted to ICU/CCU and treated as follows:

a. **Bed rest**: All patients suspected to have AMI must be given complete bed rest. Patients should be reassured, their vital parameters checked and an intravenous line started with a large bore needle or cannula.

b. **Emergency ECG**: On clinical suspicion of AMI perform ECG as soon as possible- preferably within 10 min of arrival. A continuous ECG monitoring must be carried out where possible.

c. **Supplemental oxygen:** Oxygen should be administered at a flow rate of 4 – 6 liters/minutes by nasal prongs or mask for the first three hours must be administered to all patients even in the absence of dyspnea or arterial desaturation. The inhalation must be continued further in patients who have dyspnea or Sp02 less than 90%.

d. **Aspirin:** Aspirin (160- 325 mg) should be administered to the patient with the instruction that it should be chewed and swallowed as soon as possible. Alternative anti-platelet drugs such as Clopidogrel/Ticlopidine can be administered if the patient is allergic to aspirin.

e. **Pain relief**: Combination of nitrates, analgesics, beta blockers and oxygen are effective in relieving pain. Morphine remains the drug of choice.

f. **Nitrates**: Sublingual nitrates are indicated for most patients because of their ability to enhance coronary blood flow by coronary vasodilation and decreasing preload. They are contra indicated in RV infarction or patients with hypotension.

g. **Beta-blockers**: Injmetoprolol 5 mg, slow intravenous can be given to achieve a heart rate of 60 – 80 / min. This should be avoided in patients with underlying chronic airway disease or patients with LVF. It can be repeated to a maximum of 3 doses given 2-5 minutes apart till appropriate response is achieved in the absence of contraindications, especially in patients with continuing or recurrent pain and those with tachyarrhythmias.

h. **Non – dihydropyridine Calcium channel antagonists**: Diltiazem or varapamil can be administered for patients with continuing ischemia or tachyarrhythmias in whom beta-blockers are contraindicated or ineffective and there is no evidence of cardiac failure, LV dysfunction or heart block.

j. **Angoiotension converting Enzyme (ACE) inhibitors:** These drugs should be administered to patients with large myocardial infarcts in the absence of hypotension, especially if they have left ventricular dysfunction. These drugs should be continued indefinitely.

k. **Anticoagulation**: Unfractionated Heparin 60 Units/kg bolus IV bolous followed by 12 Units/kg/hour (max 1000 Units/hour) under aPTT monitoring or low Molecular weight Heparin subcutaneously in appropriate doses (eg Enoxaparin /Fondaparinux) is indicated in patients with high risk for embolisation (large anterior MI, atrial fibrillation or LV clot on echocardiography) and those patients of ST elevation MI who have not received thrombolysis. Glycoprotein IIb-IIIa receptor antagonists (Abciximab/Eptifibatide/Tirofiban) are indicated in high-risk patients particularly during angioplasty provided they do not have major contraindications such as bleeding risk.

l. **Thrombolysis:**

Thrombolytic therapy – which aims at limiting the size of the infarct by opening the infarct related artery - has been shown to reduce complications and mortality due to infarct related complications. The following fibrinolytic agents are approved for clinical use:

i. rt-PA (preferable)

ii. Tenectaplase

iii. Streptokinase

iv. Urokinase

Although these drugs can produce a few side effects such as hypotension, allergic reactions, minor bleeding etc, the only serious complication of this therapy i.e. intra- cerebral bleeding is rare (0.70%). All patients with history suggestive of AMI who present within 12 hours of onset of chest pain and have ST elevation (of at least 1 mm) in two or more contiguous leads, or a new onset left bundle branch block must be administered thrombolytic therapy. Contraindications to this therapy are listed below.

Contra-indications to thrombolysis

Absolute - Previous hemorrhagic stroke (any time in the past)

- Ischaemic stroke within the last one year
- Known intra- cranial neoplasm
- Active internal bleeding
- Suspected aortic dissection

Relative - Uncontrolled severe hypertension (BP > 180/110 mm Hg)

- Known bleeding diathesis
- Recent trauma/major surgery (in the last 3 weeks)
- Prior exposure to streptokinase (in past 5 days to 2 years)
- Pregnancy
- Active peptic ulcer

Relief of pain normalization of elevated ST, appearance of reperfusion arrhythmias and early peaking of CK- MB indicate successful reperfusion.

m. **Primary Angioplasty in AMI:** Primary angioplasty has emerged as the most effective strategy for establishing rapid blood flow in an occluded infarct related coronary artery. Several large trials have established the superiority of early invasive With increasing expertise, this strategy is also safer and should now be considered in all patients with AMI, wherever it is feasible. The procedure is even more useful in patients with the following:

i. High risk STEMI- Cardiogenic shock, Killip class>3
ii. Contraindications to fibrinolysis
iii. Late presentation > 3 hrs
iv. Doubtful STEMI diagnosis
v. In patients with failed thrombolysis (rescue PCI)

n. **Emergency Coronary Artery Bypass Graft (CABG) Surgery:** Emergency CABG is rarely needed in a patient with AMI. The only indication for emergency CABG surgery in this subset is in patients with: -

(i) Failed primary angioplasty

(ii) AMI refractory to medical therapy with coronary anatomy suitable for surgery

(iii) Patients with mechanical complications of AMI (postinfarction ventricular septal defect or Mitral valve insufficiency).

o. **Others Measures:** Patient must be reassured, given mild sedation (Diazepam), if required and kept nil orally till pain free. Thereafter a low cholesterol and low Calorie diet, rich in fiber is given divided into multiple small feeds and a mild laxative is prescribed to prevent straining at stools. Patients remain in bed for 2 -3 days but can use a bedside commode or bedpan after 12 hours. Gradual ambulation with walks within the ward should be introduced after 48 – 72 hours.

6. **Complications following AMI**:

Various complications following myocardial infarction can be classified as given below:

a. Hemodynamic complications: These include acute left ventricular failure, cardiogenic shock and right ventricular myocardial infarction. Management for these complications includes early revasuclarization (mechanical or pharmacological) along with aggressive pharmacotherapy with afterload reducing agents (nitroglycerine infusion), pre-load reducing agents (intravenous diuretics) and positive inotropes (often beta stimulant adrenergic agents). Additionally, mechanical devices such as intra-aortic balloon pump may be needed to support and augment circulation.

b. Arrhythmic and conduction system complications: These complications are responsible for mortality in the first 72 hours. Prompt recognition and treatment are essential. Various arrhythmic complications and their brief management is listed below:

Ventricular Tachycardia Sustained and polymorphic VT/VF	DC shock 200J, 300J and then 360J
Monomorphic VT with hemodynamic compromise	DC shock 100J
VT with no hemodynamic compromise	IV Lidocaine 1-1.5 mg/kg bolus, followed by : Infusion 2-4mg/min, IV Procainamide, Amiadarone, Synchronished DC shock 50J
Atrial Fibrillation With hemodynamic compromise	Cardioversion
With no hemodynamic compromise	Digoxin/Beta – blockers
High grade AV block	Temporary pacing

c. Mechanical complications: These include pappilary muscle rupture – leading to acute mitral regurgitation, post MI ventricular spetal defect (VSD) and rarely cardiac rupture. These complications are often fatal (unless detected in time) and surgery offers the only treatment for them.

7. **Risk Stratification following AMI**

Survival after AMI depends on three factors viz left ventricular function, residual viable myocardium and susceptibility to ventricular arrhythmias. These three factors are assessed by echocardiography, stress tests and Holter monitoring respectively.

(a) Those patients of acute MI with Post-MI angina, LV dysfunction sustained VT/VF 48 hours after MI are referred for coronary arteriography and revascularization.

(b) In the absence of high risk clinical indicators mentioned above patients undergo a sub-maximal stress test (5-6 METS/80% maximum predicted heart rate) at 2 weeks after MI or Nuclear Scan/ Dobutamine Echo/ Exercise Echo. If this is abnormal, patients undergo coronary arteriography.

(c) Medical therapy is continued in the remainder, in whom a symptom limited exercise test is done subsequently at 6 weeks after MI. If there is evidence of inducible ischemia, patients undergo coronary arteriography. If the test is negative for inducible ischemia preventive measures like Aspirin, Beta-blockers, lipid lowering drugs (to keep LDL < 100 mg/dl) and ACE inhibitors where indicated (LV dysfunction or large regional wall motion abnormality on echocardiography).

UNSTABLE ANGINA AND NON ST ELEVATION MI (NSTEMI)

8. Unstable angina is defined as angina pectoris with at least one of three features.

(a) Angina at rest lasting for more than 20 minutes.

(b) New onset (within one month), severe and described as frank pain.

(c) Crescendo angina (more severe prolonged or frequent than previously).

9. Some of these patients, especially those with prolonged rest pain develop evidence of myocardial necrosis (elevated cardiac markers) without ST elevation or new Q waves in ECG and thus are described to have Non ST elevation MI (NSTEMI).

10. Evaluation and treatment

All patients of unstable angina and NSTEMI are admitted to hospital. History, clinical examination and investigations are carried out on the same lines for chronic CAD. A 12 lead ECG (at admission and during pain) and serial measurement of cardiac markers like Creatine Kinase – MB fraction (CK-MB 12 hrly for 3 days) or cardiac Troponins (twice in 12 hrs after onset of chest pain) are done.

11. Risk Stratification.

Once the patient is stabilized and is free of angina at rest, heart failure or complications for at least 48 hrs, further evaluation with echocardiography and stress test is indicated. Early coronary angiography and revascularization are indicated if the following features of high risk are present.

(a) Continuing ischaemia/angina despite medical therapy.

(b) Dynamic ST-T changes

(c) Hemodynamic instability/sustained VT

(d) Evidence of CCF/LVEF < 40 % on echo

(e) Increased cardiac enzymes (Serum Troponins and CK (MB)

Those who do not require early coronary angiography are put on long-term medical therapy as for chronic coronary artery disease.

12. **Treatment of Unstable angina**:

a. **Antiplatelets**: These drugs form the corner stone of therapy of unstable angina. Since platelets represent one of the principal participants in thrombus formation after plaque disruption, antiplatelet therapy forms the foundation for the treatment of ACS. Drugs included in this group are aspirin and clopidogrel which should be administered as soon as the patient presents to the medical facility.

b. **Glycoprotein IIbIIIa inhibitors**: The use of IIbIIIa inhibitors is limited to patients who are high risk and/or an early invasive stratergy has been planned. These patients include those with recurrent symptoms/ischemia, HF, serious arrhythmias, elevated troponin levels, diabetes, or significant ST-segment depression, and who are not otherwise at high risk for bleeding. Abxicimab is started at a loading dose of 0.25 mg per kg IV bolus and then a maintenance dose of 0.125 mcg per kg per min (max 10 mcg per min) for 12 hours. Eptifibatide is started at a loading dose of 180 mcg per kg and a maintenance dose of 2.0 mcg per kg per min for 18 to 24 hours. (Reduce infusion by 50% in patients with estimated creatinine clearance less than 50 mL per min). Tirofiban is started with a loading dose of 0.4 mcg per kg per min for 30 min, followed by an infusion of 0.1 mcg per kg per min(reduce rate of infusion by 50% in patients with estimated creatinine clearance less than 30 mL per min) for 18 to 24 h.

c. **Anticoagulant therapy**: Anticoagulation should be added to antiplatelet therapy in UA/NSTEMI patients as soon as possible after presentation. Anti-thrombins prevent thrombus propagation by specifically inhibiting thrombin but do not lyse existing thrombi.

d. **Indications for Early Invasive Therapy:** This strategy triages patients to undergo an invasive diagnostic evaluation without getting a noninvasive stress test or without failing medical treatment. Patients treated with an invasive strategy generally will undergo coronary angiography within 4 to 24 h of admission; however, these patients also are treated with the usual UA/NSTEMI medications, including appropriate anti-ischemic, antiplatelet, and anticoagulant therapy, as outlined earlier.

i. High Risk Group: eg Diabetics, past H/O of CAD, intervention for CA D in past.
ii. Continuing ischemia/angina despite medical therapy (Refractory Angina)

iii. Dynamic ST-T changes

iv. Hemodynamic instability: Hypotension

v. Electrical instability: sustained VT

vi. Evidence of CCF/LVF

vii. LVEF less than 40%

viii Elevated cardiac enzymes – such as CPK MB, Troponins etc. (These patients actually form the subset of non ST elevation myocardial infarction).

e. **Initial Conservative Therapy:** When the conservative strategy is chosen, a plan for noninvasive evaluation is required to detect severe ischemia that occurs spontaneously or at a low threshold of stress and to promptly refer these patients for coronary angiography and revascularization when possible. A stress test (e.g., exercise or pharmacological stress) for the assessment of ischemia is recommended before discharge or shortly thereafter to identify patients who may also benefit from revascularization.

Patients in whom **all** of the following are present, an angiography may be deferred or not done during the initial presentation:

- Low risk: No modifiable risk factors
- Normal LVEF
- Stress test negative

CHRONIC CORONARY ARTERY DISEASE (CAD)

13. Chronic CAD can be asymptomatic or may present as sudden death but more often presents as angina pectoris. Angina is typically described as a retrosternal discomfort (strangling, constricting, burning, suffocating) brought on by physical exertion or emotion, (usually radiating to left arm) and relieved with rest or nitroglycerine. Anginal "Equivalents" such as dyspnea, faintness, fatigue and eructations are common in the elderly who may not complain of angina. History of diabetes mellitus, hypertension, smoking and dyslipidemia, and of CAD in other family members must be taken in such patients.

14. **Evaluation.**

Clinical examination should focus on modifiable coronary risk factors (overweight xanthelesma, hypertension) evidence of atherosclerosis elsewhere (carotid or renal bruit, weak peripheral pulses and retinopathy on examination (to look for evidence of cardiac decompensation especially during an episode of angina viz, third or fourth sounds, systolic murmur at cardiac apex).

15. **Laboratory investigations**.

These include routine blood counts, urine for sugar and proteins, blood sugar, lipid profile, uric acid resting ECG, chest radiograph and echocardiographic studies.

A resting ECG may be normal in upto 50% patients : in other it may show depression of ST segment or T waves, and intraventricular conduction abnormalities. Stress test (TMT) or myocardial perfusion imaging in case of a non- diagnostic basal ECG is an important tool for stratification of risk indicative of high risk as detailed in Table 1, and patients with cardiac symptoms despite optimal medical therapy are referred for coronary arteriography (CART). High risk markers on a stress test are outlined below:

a. Abnormal horizontal or downsloping ST segment depression (more than 2mm) at low work load onset at heart rate < 120 min or <6.5 METS) in multiple leads and persisting 6 minutes or beyond into recovery.
b. Flat systolic BP response (< 130 mm Hg) or fall in systolic BP more than 10 mm Hg.
c. Exercise induced Ventricular Tachycardia (VT) elevation of ST segment.

16. **Treatment**

Medical therapy of chronic CAD consists of identifying and treating precipitating factors and control of modifiable coronary risk factors by achieving and maintaining ideal body weight, regular aerobic exercise, stopping smoking, early detection and treatment of hypertension, hyperlipidemia and diabetes mellitus. Drug therapy must include daily aspirin, lipid lowering drugs (to reduce LDL < 100 mg/dl) and antianginal medication. Sublingual nitrogycerin or nitroglycerin spray is useful for immediate relief of angina. Beta-blockers are the first choice thereafter for stable angina. Calcium channel blockers (avoid short acting Nifedipine) or long acting nitrates can be combined with Beta-blockers if angina is not controlled. Prinzmetal (variant) angina due to coronary vasospasm responds well to nitrates and calcium channels blockers.

ISCHAEMIC CARDIOMYOPATHY

17. **Ischaemic Cardiomyopathy** In some patients, CAD results in severe myocardial dysfunction clinically indistinguishable from dilated cardiomyopathy. This ischaemic cardiomyopathy may be due ischaemic dysfunction, hibernation, diffuse fibrosis or multiple infarctions of the myocardium. It is important to assess the extent of residual viable myocardium (by myocardial perfusion imaging or stress echocardiography) with a view to revascularization. Ischaemic cardiomyopathy, treated medically has a poor prognosis.

DISPOSAL OF PATIENTS WITH CAD

18. The following instructions are applicable to Officers, JCOs and OR of the Army and their equivalent in the Navy and Air Force.

19. **Disposal afterAcute MI, Unstable Angina and NSTEMI.**

a. All patients who are stabilized with treatment and have an uncomplicated course will undergo a sub-maximal stress test (TMT) 2 weeks after the onset. If the stress test does not show high- risk features the

individual is sent on 2-6 weeks sick leave (P4-T2-6). All CAD cases to be opined by a Cardiologist before proceeding on sick leave.

b. Those who have a complicated course (LV dysfunction, post-MI angina conduction disturbances) should be referred for coronary angiography and revascularization if indicated and further disposal will follow thereafter.

c. After expiry of sick leave, all patients will be reviewed by a cardiologist, who will consider subjecting them to a symptom limited stress test. Those who are asymptomatic with no inducible ischemia will be placed in P3 (T-24). Subsequently on review, they will be upgraded to P2 (T-24) if he fulfils the following criteria:

i. Asymptomatic clinical status with good effort tolerance.

ii. Good control of all risk factors.

iii. Normal LV function on echocardiography.

iv. No evidence of cardiac arrhythmia.

v. No evidence of inducible ischemia on stress test.

d. If the individual continues to fulfill the above listed criteria at the end of this period, he can be placed in permanent medical category P2. A Cardiologist or a Classified Specialist in Medicine will review such patients every year.

e. Those officers who are only on medical therapy and no PCI/CABG and who have been observed for at least one year in medical category permanent P2 may be considered for upgradation to P1 (B) subject to following criteria :-

i. Patient is asymptomatic with good effort tolerance.

ii. Modifiable risk factors are under control.

iii. TMT (done up to 100%predicted maximal heart rate and at least stage III of Bruce protocol) shows no inducible ischemia/arrhythmias.

iv. Echocardiography shows normal LV functions, No LV clot and no significant MR.

v. Coronary angiogram should show single vessel disease not involving left main or proximal/ostial coronary artery with normal LV function, normal LV end diastolic pressure and no significant MR.

f. All officers considered fit for upgradation to PI(B) will be referred to Senior Consultant(Medicine) at Army Hospital Delhi Cantt. If recommended by Senior Consultant (Medicine) the medical board proceedings will be forwarded

directly to the DGMS concerned for approval. All such cases upgraded to PI(B) will be thereafter reviewed by Senior Adviser (Cardiology) every year.

20. **Disposal of cases of Chronic CAD (Stable angina/asymptomatic CAD)**

All cases will be transferred to the nearest Cardiology Centre for complete evaluation. After the diagnosis is confirmed, all cases with TMT evidence of high-risk features will undergo coronary arteriography. Depending on vessel anatomy, further therapy is indicated.Those cases with no high risk features and those who are on medical therapy after coronary angiography (No PCI/CABG) disposal will be as per preceding paras .

21. **Disposal of cases who have undergone PCI**

(a) All patients after successful PCI may be sent on sick leave for 2-6 weeks (P4-T2-6).

(b) They will undergo stress test on review after sick leave. If they have no evidence of significant inducible ischemia they will be placed in medical category P3(T-12).

(c) During the second review (6 months post PCI) all patient even if asymptomatic, will be evaluated with TMT/myocardial perfusion scans will be upgraded to P2(T-24) and subsequently placed in P2 permanent. Those showing significant redistribution should undergo repeat CART and PCI/CABG surgery if indicated.

(d) Upgradation to P1(B) is applicable to only officers . Period of observation prior to upgradation to P1 (B) shall be for a minimum of 12 months (52weeks), commencing from the time of revascularization (PCI or CABG), will require a concurrence of Senior Advisor/Senior Consultant as mentioned earlier. It may be considered if individual satisfies following criteria:-

(i) There are no major modifiable risk factors.

(ii) Individual has been observed in low medical category after PCI for at least one year.

(iii) There is no objective evidence of myocardial ischemia on myocardial perfusion imaging/stress echocardiography.

(iv) Echocardiography shows normal LV function, no LV clot and no significant dyskinetic segment.

(v) Repeat coronary angiography confirms patency of the culprit vessel.

22. **Disposal of cases who have undergone CABG Surgery**

(a) All patient after uneventful CABG surgery should be sent on sick leave for 6-8 weeks.

(b) After convalescence patient should be evaluated in a Cardiology Centre and placed in Cat P3(T-24). On review after 6 months, if the patient remains asymptomatic with normal LV function on echocardiography and no inducible ischemia on TMT/myocardial perfusion imaging, he should be placed in Cat P2(T-24) and subsequently in P2 permanent, if indicated.

(c) After a period of observation of one year in low medical category after CABG the individual may be considered for upgradation to P1(B) based on the criteria listed for post-PCI patients. A graft angiography may be done if there is abnormality on myocardial imaging/stress echocardiography.

(d) Procedure for such upgradation will be as listed above.

23. **Disposal of patients with Ischaemic Cardiomyopathy**

Patient with ischaemic cardiomyopathy will undergo a detailed evaluation (including myocardial perfusion imaging) to look for viable myocardium. Revascularisation may be offered, depending on coronary anatomy. Those who remain symptomatic despite optimum therapy, with cardiac failure, no significant viable myocardium and LVEF < 30% may be invalided out of service. All cases of Cardiac Resynchronization therapy shall be boarded out of service.

24. **Disposal of cases with non-specific ECG abnormality**

Non-specific ST-T abnormality or other ECG changes may be detected in asymptomatic individual on routine ECG checks. If a detailed cardiac evaluation including TMT and Echocardiography is normal the case will be diagnosed as " Non specific ECG abnormality". All such patients will be evaluated and disposed off by cardiologist on a case –to-case basis.

CHANGES PROPOSED IN THE MEMORANDUM:

PARA	EXISTING PROVISIONS	PROPOSED CHANGES
2.	Old nomenclature and classification of CAD	New classification of CAD
3 to	Chronic CAD, Ischemic Cardiomyopathy, Unstable Angina evaluation	Replaced by investigational and management strategies of STEMI
14 to	Old Management strategies of Acute Myocardial Infarction	New Management strategies of Unstable Angina/NSTEMI
37(a)	Sick leave for uncomplicated CAD 4-6 weeks	Sick leave for uncomplicated CAD 2-6 weeks
37	All CAD cases need not be seen by Cardiologist before proceeding on sick leave	All CAD cases to be seen by Cardiologist before proceeding on sick leave
	Upgradation to P1 (B) to be considered 18 months after PCI	Upgradation to P1 (B) to be considered 12 months after PCI
	Upgradation to P1 (B) to be considered 18 months after CABG	Upgradation to P1 (B) to be considered 12 months after CABG
39	Silent on disposal of Ischemic Cardiomyopathy who undergo CRT	All cases of Cardiac Resynchronization therapy shall be boarded out of service.