APPENDIX

Definition and Data Derivation of Co-Primary and Secondary Efficacy Variables [5].

Co-Primary Efficacy Variable: Timely Reperfusion in STEACS

Rationale: Timely reperfusion has been associated with substantially reduced mortality in patients with STEACS. Several initiatives have been promoted to measure reperfusion time in other countries and have produced substantial improvement over time. In Italy, timely reperfusion is not systematically collected.

Definition: Proportion of STEACS patients intended to receive urgent reperfusion (i.e. DB1) with acceptable time delay defined as follows:

Non-transfer patients: Proportion of patients with <60 min from door-to-arterial access for reperfusion with PCI.

Transfer patients: Door-in-door-out time of <30 min.

Fibrinolysis: <30 min from fist medical contact to needle.

Denominator: DB1 patients

Method of reporting: Proportion (standard error).

Dimension of care: Center organization and efficiency

Co-Primary Efficacy Variable: Optimal Medical Therapy at Discharge

Rationale: Optimal medical therapy at discharge has been a cornerstone metric for quality improvement. According to ESC recommendations, 6 we will calculate a composite QI including key discharge prescriptions calculated using the 'all or none' method. This method best reflects the interest of the patient, since even one missing component in the score may influence outcome.

Definition: In patients discharged alive with a final diagnosis of MI/UA and with HF or LVEF≤0.40, the variable will be calculated from *five* individual QIs: 1) low-dose aspirin 2) adequate P2Y12 inhibition 3) highly effective statin regimen 4) ACE-inhibitor (or ARB if intolerant to ACEI) 5) beta-blockers (unless clear

contraindication). For patients discharged alive with a final diagnosis of MI/UA without HF or LVEF≤0.40, the variable is calculated using *three* individual QIs: 1) low-dose aspirin 2) adequate P2Y12 inhibition 3) highly effective statin regimen.

1. Low dose aspirin

Proportion of patients without documented intolerance who receive a daily dose ≤ 160 mg. For patients with documented intolerance, the proportion of patients undergoing aspirin desensitization procedures will be also collected.

2. Adequate P2Y12 inhibition

Proportion of eligible patients (i.e. without high bleeding risk) who receive intense P2Y12 inhibition – i.e. prasugrel 10 mg OD or ticagrelor 90 mg BID. According to label indications for specific agents and validated definitions of high risk of bleeding 17 eligibility for intense P2Y12 inhibition will be derived as follows:

- **Ticagrelor 90 mg BID**: Patients with a diagnosis of MI/UA at discharge and without the following high-bleeding risk criteria: concurrent therapy with oral anticoagulants; history of hemorrhagic stroke; current active bleeding; previous bleeding episode(s) which required hospitalization if the underlying cause has not been definitively treated; documented anemia defined as hemoglobin levels <10.5 g/dl or transfusion within 4 weeks before enrollment; systemic conditions associated with an increased bleeding risk including hematological disorders, history of or current thrombocytopenia defined as a platelet count <100,000/mm3 (<100 x 109/L), or any known coagulation disorder associated with increased bleeding risk, diagnosed malignancy (other than skin) considered at high bleeding risk including gastro-intestinal, genito-urethral/renal and pulmonary. 13
- **Prasugrel 10 mg OD**: age ≥ 75 years old, weight ≤ 60 kg, history of any stroke or transient ischemic attack as well as all ticagrelor–specific high bleeding risk criteria.

3. *Highly effective statin regimen*: This includes atorvastatin 40 mg or 80 mg, rosuvastatin 10 mg, 20 mg or 40 mg. In addition, any of the above statin doses administered in combination with ezetimibe would qualify as highly effective therapy.

4. *ACE-Inhibitor (or ARBs* if intolerant of ACEI) unless contraindicated in patients with clinical evidence of HF or LVEF≤0.40.

5. *Beta-blockers*, unless contraindicated, in patients with clinical evidence of HF or LVEF≤0.40.

For each patient, this variable is rated=1 if all components are present and it is rated=0 if one or more components are missing.
Individual components will be also collected as secondary efficacy variables.
Denominator*:* Patients discharged alive with a diagnosis of myocardial infarction or unstable angina stratified by clinical evidence of HF or LVEF≤0.40 (see above).
Method of reporting: at patient level, can be 0 or 1. At site level, mean value (95% confidence interval).
Dimension of care: Therapy

Secondary Efficacy Variable: Assessment of Left Ventricular Ejection Fraction

Rationale: all patients with LVEF≤0.40 need specific medical treatment.
Definition: proportion of patients with assessment of LVEF before discharge.
Denominator: patients discharged alive with a diagnosis of myocardial infarction or unstable angina.
Method of reporting: proportion (standard error).
Dimension of care: Diagnosis

Secondary Efficacy Variable: Concordant Final Diagnosis at Discharge

Rationale: Reaching a diagnosis is essential for optimal patient management. We will therefore collect the proportion of patients with the disease of interest at discharge (ie MI/UA) among all included ACS patients. We will also collect the proportion of patients with an etiologic diagnosis among all included patients – i.e. excluding patients with descriptive diagnosis such as “non-cardiac symptoms”, “chest pain of uncertain origin”, or similar.

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Definition: proportion of patients with a final diagnosis of MI/UA and with a final etiologic diagnosis.

Denominator: all patients

Method of reporting: proportion (standard error).

Dimension of care: Diagnosis

Secondary Efficacy Variable: Use of Radial Access

Rationale: Radial over femoral access is recommended for coronary angiography and PCI in patients with ACS. 18

Definition: proportion of patients with an initial radial access among all patients invasively managed.

Denominator: all patients managed invasively, i.e. DB1 and DB3.

Method of reporting: proportion (standard error).

Dimension of care: Therapy

Exploratory Efficacy Variables

- 30-day GRACE 2.0-Adjusted Mortality
- Patient satisfaction measured at the site-level. Sites will be considered to implement successfully patient satisfaction if they collect systematically patient-level information on pain control, explanations provided by doctors and nurses (about the coronary disease, the benefit/risk of the discharge treatment, and medical follow-up) and discharge information regarding what to do in case of recurrence of symptoms and recommendation to attend
- use of cardiac rehabilitation program (including smoking cessation and diet counseling).
- One-year incidence of death, (recurrent) MI, stroke, stent thrombosis, or bleeding. Events will be adjudicated according to pre-specified definition (see appendix).
- Proportion of ACS patients (ALL, ie DB1, DB2, DB3, DB4) with no documented intolerance who received aspirin at admission;

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- Proportion of ACS patients, among those discharged with MI/UA and with no documented aspirin intolerance, who received aspirin at discharge;
- Proportion of patients with no documented contraindications who received beta-blockers at admission;
- Proportion of patients with no documented contraindications and with HF during hospitalization or discharge LVEF of 0.40 or less who received beta-blockers at discharge;
- Proportion of ACS patients, among those discharged with MI/UA and with no documented intolerance, who received highly effective statin at discharge
- Proportion of patients discharged with MI or UA with LVEF evaluated
- Proportion of patients with no documented contraindications and with HF during hospitalization or discharge EF of 0.40 or less who received ACE-I or ARB at discharge
- Proportion of STEACS patients (ie DB1) with FMC to balloon of 90 minutes or less
- Proportion of ACS patients, among those discharged with MI/UA and who received PCI, who were discharged with dual antiplatelet therapy (ASA + $P2Y_{12}$ inhibitor)
- Proportion of ACS patients, among those discharged with MI/UA and who received CABG, who were discharged with dual antiplatelet therapy (ASA + $P2Y_{12}$ inhibitor)
- Proportion of ACS patients, among those discharged with MI/UA and who received CABG, who were discharged with highly effective statin therapy
- Proportion of ACS patients, among those discharged with MI/UA and who DID NOT receive PCI or CABG (ie medically managed), who were discharged with dual antiplatelet therapy (ASA + clopidogrel or ticagrelor)

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ENDPOINT DEFINITIONS

The following endpoints – death, myocardial infarction, stroke, bleeding – will be systematically triggered and adjudicated by at least two independent physicians. Other medical events of interest (eg. coronary revascularization unrelated to MI, stent thrombosis without MI, thrombocytopenia), will be recorded as adverse events but will not undergo adjudication.

DEATH

All deaths will be categorized as cardiovascular, non-cardiovascular, or undetermined
based on the definitions below.

Cardiovascular Death

Cardiovascular death is defined as death resulting from an acute myocardial infarction, sudden cardiac death, death due to heart failure, death due to stroke, death due to cardiovascular (CV) procedures, death due to CV hemorrhage, and death due to other cardiovascular causes.

Death due to Acute Myocardial Infarction

Death by any mechanism (arrhythmia, heart failure, low output) within 30 days after a myocardial infarction (MI) related to the immediate consequences of the myocardial infarction, such as progressive congestive heart failure (CHF), inadequate cardiac output, or refractory arrhythmia. If these events occur after a “break” (e.g., a CHF and arrhythmia free period of at least a week), they should be designated by the immediate cause, even though the MI may have increased the risk of that event (e.g., late arrhythmic death becomes more likely after an acute MI). The acute MI should be verified to the extent possible by the diagnostic criteria outlined for acute MI or by autopsy findings showing recent MI or recent coronary thrombus. Sudden cardiac death, if accompanied by symptoms suggestive of myocardial ischemia, new

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ST elevation, new left bundle branch block, or evidence of fresh thrombus by coronary angiography and/or at autopsy should be considered death resulting from an acute MI, even if death occurs before blood samples or 12-lead electrocardiogram (ECG) could be obtained, or at a time before the appearance of cardiac biomarkers in the blood.

Death resulting from a procedure to treat a MI such as percutaneous coronary intervention (PCI), coronary artery bypass graft surgery (CABG), or to treat a complication resulting from MI, should also be considered death due to acute MI.

Death resulting from an elective coronary procedure to treat myocardial ischemia (i.e., chronic stable angina) or death due to a MI that occurs as a direct consequence of a CV investigation/procedure/operation should be considered as a death due to a CV procedure.

Sudden Cardiac Death

Death that occurs unexpectedly, not following an acute AMI, and includes the following deaths:

1. Death witnessed and occurring without new or worsening symptoms.
2. Death witnessed within 60 minutes of the onset of new or worsening cardiac symptoms, unless documented (i.e. by ECG or other objective) to be due to acute MI.
3. Death witnessed and attributed to an identified arrhythmia (e.g., captured on an ECG recording, witnessed on a monitor, or unwitnessed but found on implantable cardioverter-defibrillator review).
4. Death after unsuccessful resuscitation from cardiac arrest.
5. Death after successful resuscitation from cardiac arrest and without identification of a noncardiac
etiology.
6. Unwitnessed death without other cause of death (information regarding the patient's clinical
status preceding death should be provided, if available).

<u>General Considerations</u>

A subject seen alive and clinically stable 24 hours prior to being found dead without

any evidence or information of a specific cause of death should be classified as "sudden cardiac death."

Typical scenarios include:

Subject well the previous day but found dead in bed the next day;

Subject found dead at home on the couch with the television on;

Deaths for which there is no information beyond "Patient found dead at home" may be

classified as "death due to other cardiovascular causes".

Death due to Heart Failure or Cardiogenic Shock

Death due to heart failure refers to a death in association with clinically worsening symptoms and/or signs of heart failure not following an acute MI. Deaths due to heart failure can have various etiologies, including single or recurrent MIs, ischemic or non-ischemic cardiomyopathy, hypertension, or valvular disease.

Cardiogenic shock not occurring in the context of an acute myocardial infarction or as the consequence of an arrhythmia occurring in the absence of worsening heart failure is defined as systolic blood pressure (SBP) < 90 mm Hg for greater than 1 hour, not responsive to fluid resuscitation and/or heart rate correction, and felt to be secondary to cardiac dysfunction and associated with at least one of the following signs of hypoperfusion:

- Cool, clammy skin or
- Oliguria (urine output < 30 mL/hour) or
- Altered sensorium or
- Cardiac index < 2.2 L/min/m^2

Cardiogenic shock can also be defined if systolic blood pressure < 90 mm Hg and increases to ≥ 90 mm Hg in < 1 hour with positive inotropic or vasopressor agents alone and/or with mechanical

support.

Death due to Stroke

Death due to stroke refers to death after a stroke that is either a direct consequence of the stroke or a complication of the stroke. Acute stroke should be verified to the

extent possible by the diagnostic criteria outlined for stroke.

Death due to Cardiovascular procedures refers to death caused by the immediate complications of a cardiac procedure and excludes death resulting from procedures to treat an acute MI or the complications resulting from an acute MI.

Death due to cardiovascular hemorrhage refers to death related to hemorrhage such as
a non-stroke intracranial hemorrhage, non-procedural or non-traumatic vascular rupture (e.g., aortic aneurysm), or hemorrhage causing cardiac tamponade.

Death due to other cardiovascular causes
Death due to other cardiovascular causes refers to a cardiovascular death not included in the above categories (e.g., pulmonary embolism or peripheral arterial disease).

Non-cardiovascular Death

Non-cardiovascular death is defined as any death that is not thought to be due to a cardiovascular cause. The following categories may be collected:

Non-Malignant Causes

- Pulmonary
- Renal
- Gastrointestinal
- Hepatobiliary
- Pancreatic
- Infection (includes sepsis)
- Non-infectious (e.g., systemic inflammatory response syndrome)
- Hemorrhage*, excluding hemorrhagic strokes and bleeding in the setting of coronary
  revascularization
- Non-cardiovascular procedure or surgery

- Accidental (e.g., physical accidents or drug overdose) or trauma
- Suicide
- Prescription Drug Error (e.g., prescribed drug overdose, use of inappropriate drug, or drug-drug interaction)
- Neurological process that is not a stroke or hemorrhage
- Other non-cardiovascular, specify: _____

*Examples: Death due to GI bleeding is not considered a CV death. Death due to retroperitoneal hematoma following PCI is considered CV death. Death due to intracerebral hemorrhage is considered CV death.

Malignant Causes

Death results directly from the cancer;

OR

Death results from a complication of the cancer (e.g. infection, complication of surgery/ chemotherapy /radiotherapy);

OR

Death results from withdrawal of other therapies because of concerns relating to the poor

prognosis associated with the cancer

Cancer deaths may arise from cancers that were present prior to randomization or which

developed subsequently should be further classified (worsening prior malignancy; new

malignancy).

Undetermined Cause of Death

Undetermined cause of death refers to a death not attributable to one of the above categories of cardiovascular death or to a non-cardiovascular cause, due to absence of any information (e.g., the only available information is “patient died”). **The use of this category of death is discouraged** and should apply to a minimal number of cases when no information at all on the circumstances of death are available (i.e. found on

obituary of local newspaper). In all circumstances the reviewer will use all available information to attribute to one of the categories based on best clinical judgment.

2. MYOCARDIAL INFARCTION

The term acute myocardial infarction (MI) refers to evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischemia.
Under these conditions any one of the following criteria meets the diagnosis for MI:

1. Detection of a rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)] with at least one value above the upper reference limit (URL) and with at least one of the following:

- Symptoms suggestive of myocardial ischaemia
- New or presumed new significant ST-segment–T wave (ST–T) changes or new left bundle
- branch block (LBBB)
- Development of pathological Q waves in the ECG
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality
- Identification of an intracoronary thrombus by angiography or autopsy.

2. Cardiac death with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or new LBBB, but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would be increased.

3. Percutaneous coronary intervention (PCI) related MI is defined by elevation of cTn values
(>5 x URL) occurring within 48h of the procedure in patients with normal baseline values
(≤URL) or a rise of cTn values >20% if the baseline values are elevated and are stable or
falling.

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In addition, at least one of the following is required:

- Symptoms suggestive of myocardial ischaemia
- New ischaemic ECG changes
- Angiographic findings consistent with a procedural complication
- Imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality

4. Stent thrombosis associated with MI when detected by coronary angiography or autopsy in the setting of evidence of myocardial ischaemia and with a rise and/or fall of cardiac biomarker values with at least one value above the URL.

5. Coronary artery bypass grafting (CABG) related MI is defined by elevation of troponin values (>10 x URL) occurring within 48h of the procedure in patients with normal baseline cTn values (≤URL). In addition at least one of the following is required new pathological Q waves or new LBBB angiographic documented new graft or new native coronary artery occlusion imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Cardiac troponin is the preferred biomarker for diagnosis of MI. In absence of troponin, CKMB will be used.

Silent Myocardial Infarction

Asymptomatic patients who develop new pathologic Q wave criteria for MI detected during
routine ECG follow-up, or reveal evidence of MI by cardiac imaging, that cannot be directly
attributed to a coronary revascularization procedure, are termed ‘silent MI”. Any one of the
following criteria meets the diagnosis:

• Pathological Q waves with or without symptoms in the absence of non-ischemic causes.

• Imaging evidence of a region of loss of viable myocardium that is thinned and fails to
contract, in the absence of a non-ischaemic cause.
• Pathological findings of a prior MI

All MI events will be classified by Third Universal MI Definition subtypes as follows:

Type 1: Spontaneous myocardial infarction related to atherosclerotic plaque rupture, ulceration,
fissuring, erosion, or dissection with resulting intraluminal thrombus in one or more of the
coronary arteries leading to decreased myocardial blood flow or distal platelet emboli with
ensuing myocyte necrosis. The patient may have underlying severe CAD but on occasion
non-obstructive or no CAD.

Type 2: In instances of myocardial injury with necrosis where a condition other than CAD contributes to an imbalance between myocardial oxygen supply and/or demand, e.g. coronary
endothelial dysfunction, coronary artery spasm, coronary embolism, tachy/bradyarrhythmias,
anemia, respiratory failure, hypotension, and hypertension with or without LVH.

Type 3: Cardiac death with symptoms suggestive of myocardial ischemia and presumed new
ischemic ECG changes or new LBBB, but death occurring before blood samples could be obtained, before cardiac biomarker could rise, or in rare cases cardiac biomarkers were not collected.

Type 4a

Myocardial infarction associated with PCI

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Type 4b

Myocardial infarction associated with stent thrombosis

Type 4c

Myocardial infarction associated with restenosis (restenosis is the only angiographic explanation)

Type 5

Myocardial infarction associated with CABG

All MI events will be sub-classified into STEMI vs. NSTEMI as follows:

STEMI: New ST elevation at the J point in two contiguous leads with the cut-points: ≥0.1 mV in all leads other than leads V2–V3 where the following cut points apply: ≥0.2 mV in men ≥40 years; ≥0.25 mV in men <40 years, or ≥0.15 mV in women. If ECG does not meet STEMI criteria will be classified as NSTEMI. If ECGs are unavailable or uninterpretable the MI will be classified accordingly (ie unavailable or uninterpretable).

All MI events will be sub-classified into Q wave vs. Non Q wave MI as follows:

Criteria for abnormal Q-waves are any one of: Any Q wave in leadsV2–V3 ≥0.02 sec or QS complex in leadsV2 andV3. Q wave ≥0.03 sec and ≥0.1 mV deep or QS complex in leads I, II, aVL, aVF or V4–V6 in any two leads of a contiguous lead grouping (I, aVL; V1–V6; II, III, aVF). a. The same criteria are used for supplemental leads V7-V9. R wave ≥0.04 sec in V1–V2 and R/S ≥1 with a concordant positive T wave in absence of conduction defect. If Q-waves criteria are not met, MI is classified as non-Q-wave MI. If ECGs are unavailable or uninterpretable the MI will be classified accordingly (ie unavailable or uninterpretable).

3. STROKE

Stroke is defined as an acute episode of focal or global neurological dysfunction caused by

central nervous system (CNS) vascular injury as a result of hemorrhage or infarction. CNS

includes brain, spinal cord and retina.

Classification

Ischemic Stroke

Ischemic stroke is defined as an acute episode of focal cerebral, spinal, or retinal dysfunction caused by CNS infarction. Evidence of infarction is defined as pathological, imaging, or other objective evidence of acute cerebral, spinal cord, or retinal focal ischemic injury in a defined vascular distribution; or, in absence of the above (i.e. imaging or autopsy unavailable), clinical evidence of cerebral, spinal cord, or retinal focal ischemic injury is based on symptoms persisting ≥24 hours or until death, and other etiologies excluded.

Note that hemorrhagic infarction, defined as a parenchymal hemorrhage after CNS infarction, is
considered an ischemic stroke.

Ischemic strokes may be further classified according to most likely etiology (example large artery atherosclerosis, cardio-embolic, etc.

Cerebral Hemorrhage

Hemorrhages in the CNS are classified as stroke if they are nontraumatic, caused by a vascular event, and result in injury to the CNS. In contrast, traumatic hemorrhages will not be characterized as stroke. Subdural hematoma will not be classified as a stroke. The diagnoses included in this section are intracerebral hemorrhage (intraparenchymal and intraventricular) and subarachnoid hemorrhage (both aneurysmal and nonaneurysmal).

Stroke caused by intracerebral hemorrhage

Rapidly developing clinical signs of neurological dysfunction (focal or global) attributable to a

focal collection of blood within the brain parenchyma or ventricular system that is not caused
by trauma.

Stroke caused by subarachnoid hemorrhage

Rapidly developing signs of neurological dysfunction (focal or global) and/or headache because of bleeding into the subarachnoid space (the space between the arachnoid membrane and the pia mater of the brain or spinal cord), which is not caused by trauma.

Hemorrhages may be further classified according to location (example, supratentorial, subtentorial, etc.)

Stroke not otherwise specified

An episode of acute neurological dysfunction presumed to be caused by ischemia or hemorrhage, persisting ≥24 hours or until death, but without sufficient evidence to be classified as one of the above.

BLEEDING

All suspected bleeding events will be classified according to the Bleeding Academic Research Consortium Definition for Bleeding as follows:

Type 0: no bleeding

Type 1: bleeding that is not actionable and does not cause the patient to seek unscheduled performance of studies, hospitalization, or treatment by a healthcare professional; may include episodes leading to self-discontinuation of medical therapy by the patient without consulting a healthcare professional

Type 2: any overt, actionable sign of hemorrhage (eg, more bleeding than would be expected for a clinical circumstance, including bleeding found by imaging alone) that does not fit the criteria for type 3, 4, or 5 but does meet at least one of the following criteria: (1) requiring nonsurgical, medical intervention by a healthcare professional, (2) leading to hospitalization or increased level of care, or (3) prompting evaluation

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Type 3

Type 3a

- Overt bleeding plus hemoglobin drop of 3 to <5 g/dL* (provided hemoglobin drop is related to bleed)
- Any transfusion with overt bleeding

Type 3b

- Overt bleeding plus hemoglobin drop ≥5 g/dL* (provided hemoglobin drop is related to bleed)
- Cardiac tamponade
- Bleeding requiring surgical intervention for control (excluding dental/nasal/skin/ hemorrhoid)
- Bleeding requiring intravenous vasoactive agents

Type 3c

- Intracranial hemorrhage (does not include microbleeds or hemorrhagic transformation, does include intraspinal) with subcategories confirmed by autopsy or imaging or lumbar puncture
- Intraocular bleed compromising vision

Type 4: CABG-related bleeding

- Perioperative intracranial bleeding within 48 h
- Reoperation after closure of sternotomy for the purpose of controlling bleeding
- Transfusion of ≥5 U whole blood or packed red blood cells within a 48-h period†

Chest tube output 2L within a 24-h period

Type 5: Fatal bleeding

Type 5a: Probable fatal bleeding; no autopsy or imaging confirmation but clinically suspicious

Type 5b: Definite fatal bleeding; overt bleeding or autopsy or imaging confirmation.

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NOTE: CABG indicates coronary artery bypass graft. Platelet transfusions should be recorded and reported but are not included in these definitions until further information is obtained about the relationship to outcomes. If a CABG-related bleed is not adjudicated as at least a type 3 severity event, it will be classified as not a bleeding event. If a bleeding event occurs with a clear temporal relationship to CABG (ie, within a 48-h time frame) but does not meet type 4 severity criteria, it will be classified as not a bleeding event. *Corrected for transfusion (1 U packed red blood cells or 1 U whole blood=1 g/dL hemoglobin). †Cell saver products are not counted.

Pre-specified Variable for the Multivariables Model

Variables included in univariable and multivariable models to assess their relationship with 30-day mortality

Age, sex, smoking (current, past, never), prior myocardial infarction (MI), prior coronary artery bypass graft, hypertension, diabetes, heart failure, renal disease, heart failure clinical severity (New York Heart Association class), weight, height, systolic blood pressure, heart rate, presentation type (STEACS vs NSTEACS), left ventricular ejection fraction at discharge, baseline serum creatinine, baseline hemoglobin.