



openheart Implementation of the European Society of Cardiology 0/3-hour accelerated diagnostic protocol, using high sensitive troponin T: a clinical practice evaluation of safety and effectiveness involving 3003 patients with suspected acute coronary syndrome

James Daniel Hatherley ^{1,2}, Thomas Salmon,³ Paul O Collinson ⁴, Aleem Khand^{1,3,5}

► Additional supplemental material is published online only. To view, please visit the journal online (<http://dx.doi.org/10.1136/openhrt-2023-002366>).

To cite: Hatherley JD, Salmon T, Collinson PO, *et al.* Implementation of the European Society of Cardiology 0/3-hour accelerated diagnostic protocol, using high sensitive troponin T: a clinical practice evaluation of safety and effectiveness involving 3003 patients with suspected acute coronary syndrome. *Open Heart* 2023;**10**:e002366. doi:10.1136/openhrt-2023-002366

Received 24 May 2023
Accepted 10 November 2023



© Author(s) (or their employer(s)) 2023. Re-use permitted under CC BY. Published by BMJ.

For numbered affiliations see end of article.

Correspondence to
Dr Aleem Khand; aleem.khand@liverpoolft.nhs.uk

ABSTRACT

Background There have been relatively few studies detailing the real-world effectiveness and safety of accelerated diagnostic protocols (ADP), using high sensitivity cardiac troponin (hs-cTn).

Objective To analyse the safety and effectiveness of early emergency department (ED) discharge following implementation of the European Society of Cardiology (ESC) 0/3-hour ADP for suspected acute coronary syndromes (ACS).

Method We prospectively studied 2 cohorts of consecutive suspected ACS presentations to ED before (n=1642) and after (n=1376, 2 centres) implementation of the ESC 0/3-hour ADP incorporating limit of detection rule out. Safety was defined by MACE (major adverse cardiac events) inclusive of type 1 myocardial infarction (MI) in patients discharged from ED, and clinical effectiveness by percentage ED discharge. Continuous variables and categorical data were evaluated by independent t-test and χ^2 test, respectively. Time-to-event data were analysed as survival data and converted to Kaplan-Meier curves for interpretation.

Results In the preimplementation period, there was a higher prevalence of MI. Discharge from ED increased by >100% (from 27.1% to 56.5% of the cohort) with no safety signal (MACE rate 4/444 (0.9%) vs 4/769 (0.52%), p=0.430 for the 2011 and 2018 cohort, respectively). This correlated with a marked reduction in length of stay overall but a more modest reduction for those discharged from ED (6 hours 10 min vs 5 hours 25 min, p<0.001) for the 2011 and 2018 cohort, respectively. There were improvements in presentation to blood draw (163–90 min, p<0.001). Time from presentation to first ECG actually increased (16.2 vs 31.2 min, p<0.001). Analysis of hs-cTn values and ECGs revealed a maximum ED discharge rate of 69%, by applying the 0/3-hour protocol, implying potential for increasing safe ED discharge.

Conclusions Implementation of an ADP with hs-cTn is safe and effective for early rule-out and discharge of

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ High sensitivity cardiac troponin assays have led to the development of multiple accelerated diagnostic pathways (ADPs), including the European Society of Cardiology's (ESC) 0/3-hour protocol, for the rule out of myocardial infarction. The safety of these pathways is well established but there are less data looking into the effect on patient flow in busy emergency departments (EDs).

WHAT THIS STUDY ADDS

⇒ Patients on a local ADP had significantly reduced length of stay when compared with the pre-ADP group, with no compromise on patient safety. Implementation of the ADP into a busy ED made further logistical improvements, such as reduction in time to first blood draw. There was, however, a lengthened time to first ECG.
⇒ The study acknowledges that the troponin result is only part of the patient's journey and focussing on one aspect may lead to delays in other critical areas. As further reductions in the troponin sampling interval are advocated, ED clinicians must reflect on the patient journey in their department to ensure that these improvements impact on patient care.

suspected ACS but require considerable resources and education to optimise maximal patient flow.

INTRODUCTION

Chest pain suggestive of acute coronary syndromes (ACS) is common presentations to the emergency department (ED), accounting for up to 10% of all ED consultations.^{1–3} Although chest pain is common, less than

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ Clinicians can be confident in the safety of the ESC 0/3-hour ADP. Adoption of this pathway is well established and has clear benefits in reducing length of stay.
- ⇒ This study reinforces the need for robust, real world, randomised data comparing rapid diagnostic algorithms. It not only assesses their safety but ensures the reduction in troponin sampling time is reflected in the length of stay in ED. The shorter the sampling interval, the more likely competing factors other than troponin will delay decision-making and discharge. The extent of this is currently unknown.

10% of patients are diagnosed with myocardial infarction (MI).⁴⁻⁶ Excluding MI or other serious pathology early has clear advantages for both the patient and ED. The patient is reassured earlier, reducing exposure to unnecessary interventions and time in ED. The institution gains by improving patient flow and thereby reducing ED overcrowding, a known cause of increased morbidity and mortality especially in suspected ACS. For those with MI, earlier diagnosis leads to earlier monitoring, treatment and risk stratification.^{7,8}

The measurement of cardiac troponin (cTn) is central to the diagnosis and universal definition of MI.^{8,9} The development of high sensitivity cTn (hs-cTn) has led to improved diagnostic sensitivity and earlier exclusion of MI via rule-out algorithms.¹⁰ Improved sensitivity has, however, come at the cost of reduced specificity with detection of myocardial injury in a range of other clinical conditions than MI. Due to the improved analytical performance of hs-cTn assays, concentrations can now be measured in most healthy individuals. To be designated a high sensitivity assay, the 10% coefficient of variation must be below the 99th percentile with the ability to detect at

least 50% of a healthy population.¹¹ The Roche highly-sensitive Troponin-T (hs-TnT) assay has been shown to fulfil these criteria.^{6,12} The development of hs-cTn assays has enabled the European Society of Cardiology (ESC) to recommend an accelerated diagnostic protocol (ADP) for patients with suspected non-ST elevation ACS.⁷ This rule-out algorithm uses hs-cTn samples taken at 0 and 3 hours (figure 1). This pathway was based on multiple prospective studies.¹²⁻¹⁴

Maximising the potential of hs-cTns has been slow with patchy uptake of ADPs.¹⁵ Consequently, there is wide variation in discharge rates for suspected ACS. Reducing this variation in discharge was one of the key National Health Service (NHS) priorities manifested by advanced access collaboratives.¹⁶ We sought to study the impact and challenges of the ADP when implemented in clinical practice. We benchmarked metrics against a previous cohort of patients, who despite the use of hs-cTns still underwent 6-hour and 12-hour repeat troponin testing. We wished to understand the impact of implementing an ADP, particularly focusing on patient flow and discharge rates. We studied the patient journey in suspected ACS, preimplementation and postimplementation of the ADP.

The Liverpool Acute Chest Pain Pathway (LACPP) (figure 2) is an adaptation of the ESC 3-hour rule-out pathway. This approach is based on evidence from multiple perspective studies and a meta-analysis.^{3-6,17,18} Prior to the introduction of the LACPP, patients had troponin sampled at 6 hours, and repeated at 12 hours if greater than the 99th percentile. If blood was sampled before 6 hours of the chest pain onset, the samples were repeated at 6 hours, regardless of the initial result. Rather than an agreed proportional increase or decrease in troponin level over 6 hours, clinical judgement was used to rule-in

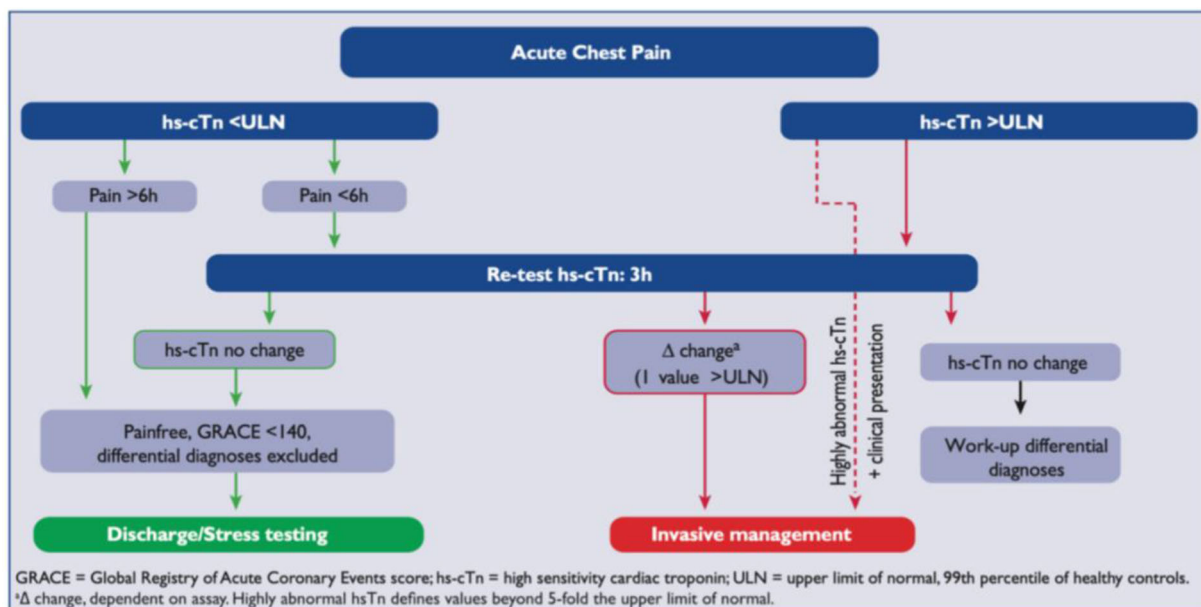


Figure 1 ESC 0/3-hour protocol for suspected acute coronary syndrome. ESC, European Society of Cardiology.

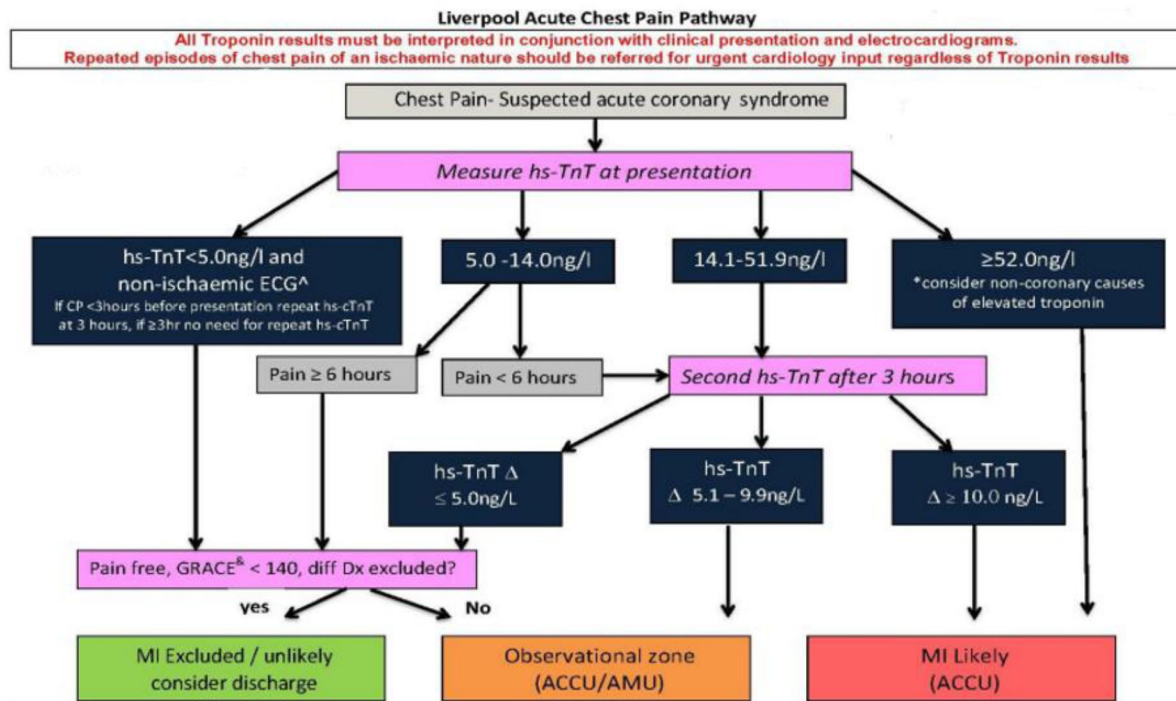


Figure 2 Liverpool 0–3 hours pathway for suspected acute coronary syndrome. ^BGRACE score predicts six month mortality following acute coronary syndrome. This cohort of patients are potentially suitable for single sample rule out if CP onset over three hours from blood sampling. ACCU, Acute Cardiac Care Unit; AMU, Acute Medical Unit; CP, chest pain; GRACE, Global Registry of Acute Coronary Events; hs-TnT, high sensitivity troponin T; MI, myocardial infarction.

MI. There was no single sample rule-out based on limit of detection (LOD).

Aim

To assess the safety of the LACPP when implemented in clinical practice.

Furthermore, to evaluate the impact on discharge from ED and the length of stay (LOS) in ED. Safety is defined using the composite primary endpoint of major adverse cardiac events (MACE), in those discharged from the ED. A direct comparison was made with the preimplementation phase.

METHOD

We undertook a two-centre quality improvement project (QIP) with audit approximately 1 year after implementation of the new ADP in 2018. All consecutive patients presenting with suspected ACS were prospectively enrolled in the study. Suspected ACS was defined as symptoms of chest pain, with a sample taken for troponin measurement and an ECG performed at presentation. This 2018 cohort was compared with a historical prospective cohort from 2011 enrolled using the same criteria. The principle aim of the historical cohort was to evaluate and compare discharge strategies before and after introduction of the ADP using hs-cTnT.⁶

We established in the 2011 cohort, with patients tracked nationally, that no subsequent adjudicated MI, to 1 year, presented outside the local region. We tracked the 2018 validation cohort regionally, rather than nationally.

For both cohorts, we collected data on time of troponin sampling relative to time of presentation, the results of the ECG and the troponin results and kinetics. In addition, length of hospital stay was documented.

Efficiency of process

We collected specifically time to first ECG after presentation, time to first blood sample and second sample, discharge time relative to presentation as metrics of efficiency of the process.

The 2011 preimplementation cohort consisted of 1637 consecutive patients in a single trust (Aintree University Hospital (AUH)). The data in this group were collected retrospectively. The 2018 postimplementation cohort was 1366 consecutive patients across 2 hospital trusts (AUH and the Royal Liverpool University Hospital). Prospective data collection started 1 year after implementation of the ADP. This allowed time for staff education and training across both sites.

Adjudication of MI

All patients with elevated hs-TnT at the index event, or any readmission up to 1 year in any hospital in England (2011) or the NW England (2018) underwent two physician adjudication for type 1 or type 2 MI using all available data. To ensure consistency, patients for both cohorts were adjudicated according to the third universal definition of MI, using hs-TnT as a biomarker of myocardial necrosis.¹⁹ This included results of investigations (eg, coronary angiography, echocardiography) up to 6 weeks

following presentation. A third cardiologist acted as a tie-breaker in case of disagreement over diagnosis.

Follow-up

NHS digital was employed using the UK unique linked hospital database to track any patients for subsequent admission using a range of International Classification of Disease 10th Revision (ICD-10) codes for cardiac or cardiac related conditions including chest pain, MI, angina. Online supplemental table S1 details codes used to input subsequent presentations, admissions, coronary revascularisation procedures to any English hospital.

Outcome

The primary outcome data were MACE and/or type 1 MI at 6 weeks. MACE is a composite endpoint comprising of MI, urgent revascularisation (via percutaneous coronary intervention or coronary artery bypass grafting) or all cause death. Readmissions for unstable angina were not included in the composite endpoint. As a secondary endpoint, we looked at discharge rates and time to discharge from ED. We also looked at time from presentation to first ECG and first hs-TnT blood draw as performance metrics.

Statistics

Continuous variables are presented as medians (IQR) and categorical variables are presented as n (%). Continuous variables were evaluated using independent t-test and Categorical data by χ^2 test. All time-to-event data were

analysed as survival data. These data were converted into Kaplan-Meier curves for interpretation. Log rank analysis was undertaken between the two curves to demonstrate statistical significance.

RESULTS

Demographics

Table 1 details the demographics and epidemiology of the two cohorts. The 2018 cohort was younger and had less cardiovascular risk factors. There was a higher proportion of patients with a history of MI in the 2011 cohort (19.6% vs 13.6% $p=0.001$).

Measures of efficiency

Time from presentation to first ECG was longer with the LACPP (figure 3A). However, time from presentation to first hs-TnT draw was significantly shorter following the introduction of the LACPP (figure 3B). Time between first and second hs-TnT draw was significantly shorter after the introduction of the LACPP (7 hours 19 min, (IQR 5 hours 46 min to 11 hours 39 min) vs 3 hours 54 min, (IQR 3 hours 10 min to 5 hours 24 min) $p<0.001$).

The LACPP significantly reduced LOS in hospital if discharged from the ED, when limiting patients included to those who were discharged within 6 hours of their last troponin being taken (figure 4A). If a patient was not discharged within 6 hours of the troponin, then it was deemed unlikely the result alone hindered their discharge. Figure 4B indicates unsurprisingly discharged

Table 1 Demographics and efficiency

Cohort	2011 (n=1637)	2018 (n=1366)	P value
Demographics			
Male, n (%)	858/1637 (52.4%)	708/1366 (51.8%)	0.750
Age (median, IQR, full range)	(n=1640) 58, (46–72), (18–102)	(n=1376) 55, (42–70), (16–101)	<0.001
≥3 risk factors, n (%)	572/1637 (34.9%)	393/1257 (31.3%)	0.038
With previous MI, n (%)	321/1637 (19.6%)	186/1365 (13.6%)	0.001
With previous PCI or CABG, n (%)	91/1637 (5.6%)	162/1361 (11.9%)	<0.001
With previous CVA, n (%)	118/1637 (7.2%)	67/1356 (4.9%)	<0.001
Timings			
CP to presentation	(n=1621) 9 hours 57 min, (IQR: 2 hours 35 min to 48 hours)	(n=1339) 8 hours 50 min, (IQR: 2 hours 56 min to 24 hours 37 min)	<0.001
Presentation to first ECG (min)	(n=1585) 16.2, (IQR: 7.2–33.0)	(n=1287) 31.2, (IQR: 16.2–60.0)	<0.001
Presentation to first hs-TnT blood draw (min)	(n=1636) 163, (IQR: 89–291)	(n=1364) 90, (IQR: 52–142)	<0.001
At least two hs-TnT, n (%)	676/1637 (41.3%)	529/1366 (38.7%)	0.152
Time between first and second hs-TnT	(n=676) 7 hour 19 min, (IQR: 5 hours 46 min to 11 hours 39 min)	(n=529) 3 hours 54 min, (IQR: 3 hours 10 min to 5 hours 24 min)	<0.001
LOS	(n=1637) 16 hour 19 min (IQR: 6 hours 30 min to 66 hour 51 min)	(n=1354) 7 hours 7 min (IQR: 4 hours 13 min to 25 hours 33 min)	0.046
Time in hospital if discharged without admission	(n=484) 6 hours 10 min (IQR: 4 hours 31 min to 8 hours 15 min)	(n=768) 5 hours 25 min (IQR: 3 hours 42 min to 8 hours 15 min)	<0.001
CABG, coronary artery bypass graft; CP, chest pain; CVA, cerebrovascular accident; ECG, electrocardiogram; hs-TnT, high-sensitivity troponin T; IQR, interquartile range; LOS, length of stay; MI, myocardial infarction; PCI, percutaneous coronary intervention.			

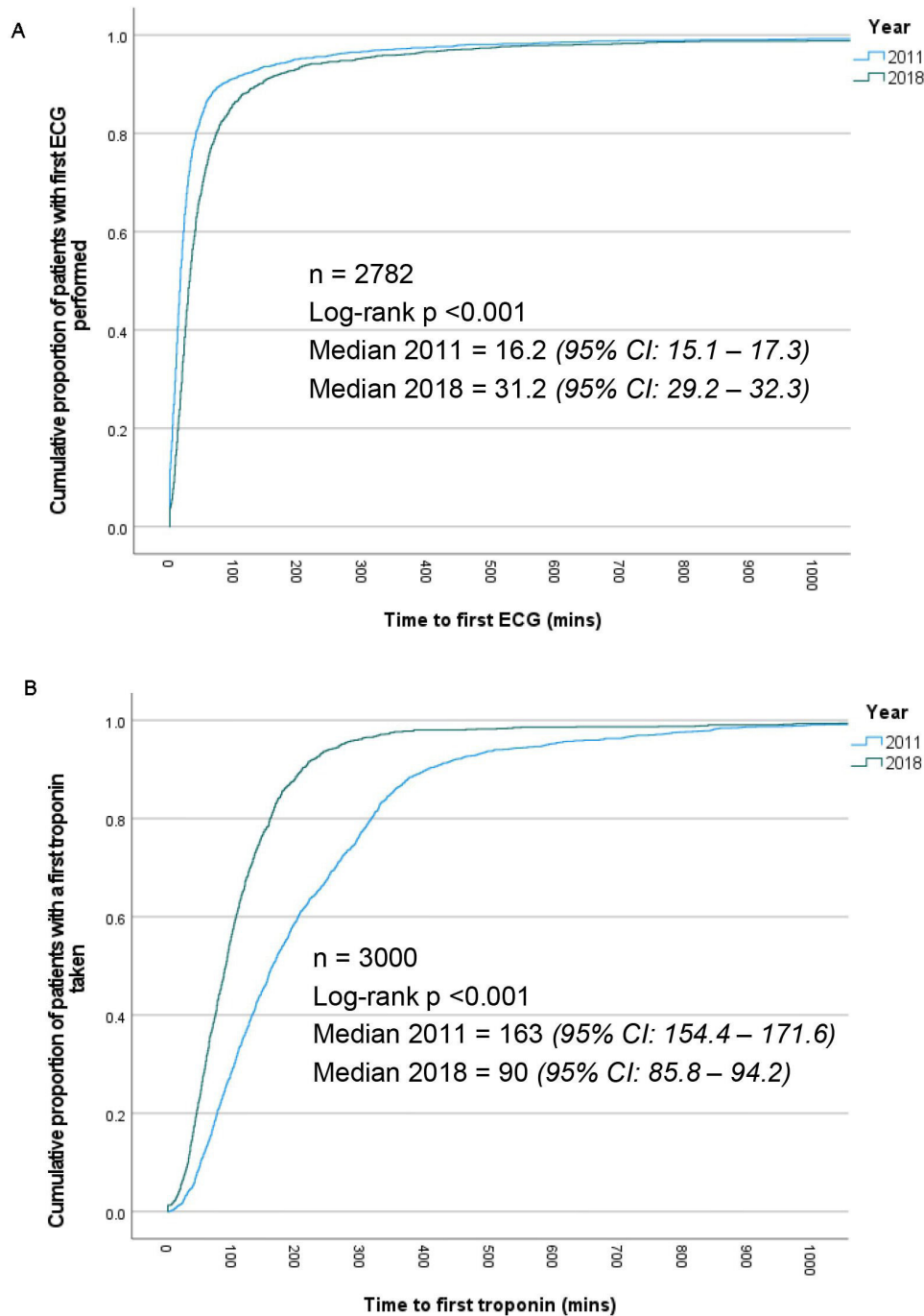


Figure 3 (A) Kaplan-Meier curve comparing time from admission to ECG between the two cohorts. (B) Kaplan-Meier curve comparing the time from admission to first troponin being taken in minutes between the two cohorts.

times were not improved in this cohort. The strong suspicion is that the troponin result was not hindering their discharge, but a range of other factors.

Safety of accelerated diagnostic protocol

Table 2 outlines safety and outcome data. The 2011 preimplementation cohort was a sicker population in terms of cardiovascular disease with a higher index MI rate. Overall MACE at 30 days was significantly greater in 2011 (preimplementation) to 2018 postimplementation (13.9% vs 7.1%: $p < 0.001$). Patients in the 2018 cohort had a significantly higher rate of discharge from

ED (27% vs 57%: $p < 0.001$). For those discharged direct from ED, MACE at 30 days was similar between cohorts (0.9% vs 0.5%: $p = 0.430$). All-cause mortality at 30 days was higher in the 2011 cohort but not significant (1.5% vs 0.7%: $p = 0.064$).

DISCUSSION

The high proportion of patients with chest pain in ED means that prompt assessment is essential to flow within the department. The LACPP has improved LOS in the ED without compromising patient safety.

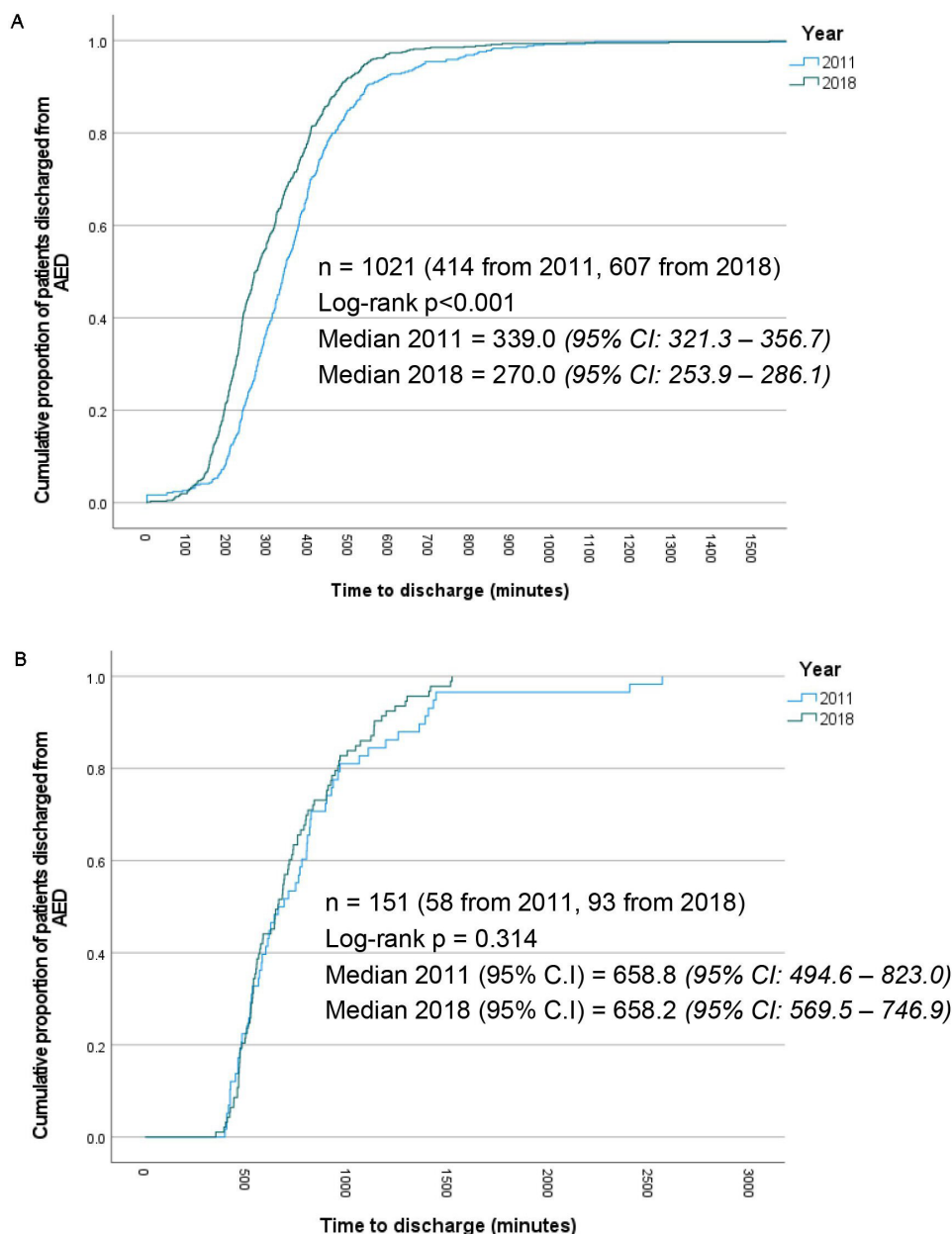


Figure 4 (A) Kaplan-Meier curve showing time from presentation to discharge, in patients discharged directly from the AED within 6 hours of their last troponin (minutes). (B) Kaplan-Meier curve showing time to discharge from AED for all patients not admitted to hospital, discharged between 6 and 24 hours after their last troponin (minutes). AED, Accident and Emergency Department.

The LACPP was adapted from the ESC 3-hour rule-out pathway. Patients in the 2011 cohort were a higher-risk population than the 2018 cohort. Unsurprisingly, there were more type 1 MIs at index presentation, higher rates of MACE and all-cause mortality at 30 days. Despite this, in patients who were discharged from ED, rates of MACE, type 1 or type 2 MI at 30 days were similar between groups. This suggests non-inferiority with regard to safety of rule out between the two cohorts. In a similar study Sandeman *et al*⁴ analysed 10 315 consecutive patients presenting with symptoms suggestive of ACS. Patients who presented prior to June 2016 were managed using the standard protocol. This involved hs-TnT sampling on presentation, with a repeat at 6 hours and potentially a

further repeat at 12 hours. The earliest point of rule-out was 6 hours. Patients presenting after June 2016 were managed using a rapid 3 hour rule-out pathway. In these two cohorts they found that there was no difference in all-cause mortality or cardiovascular mortality at 30 days or 1 year.

Use of the LACPP significantly reduced patients' total LOS. The most obvious explanation is that blood sampling is done at a shorter interval. It also has the capacity to rule out MI with one hs-TnT measurement. It is widely appreciated that LOS is also influenced by how busy ED departments can be, impacting on time to assessment, treatment and discharge. Despite this, improvements were still seen with the LACPP.

Table 2 Outcome and safety

Cohort	2011 (n=1637)	2018 (n=1366)	P value
Final diagnosis and outcome			
Index diagnosis of T1MI, n (%)	172/1637 (10.5%)	61/1366 (4.5%)	<0.001
Index diagnosis of T1 or T2MI, n (%)	192/1637 (11.7%)	82/1366 (6.0%)	<0.001
Index diagnosis of UA, n (%)	500/1637 (30.5%)	236/1366 (17.3%)	<0.001
Admission to hospital, n (%)	1185/1637 (72.4%)	592/1361 (43.5%)	<0.001
Discharged from ED, n (%)	444/1637 (27.1%)	769/1361 (56.5%)	<0.001
Safety			
Death 30 days, n (%)	22/1637 (1.5%)	9/1366 (0.7%)	0.064
MACE at 30 days, n (%)	219/1637 (13.9%)	97/1366 (7.1%)	<0.001
MACE at 30 days for those discharged without admission to hospital, n (%)	4/444 (0.9%)	4/769 (0.5%)	0.430
T1 or T2MI at 30 days for those discharged without admission to hospital, n (%)	3/444 (0.7%)	3/769 (0.4%)	0.495
Adjudicated T1 or T2MI on index admission for those discharged without admission to hospital, n(%)	3/444 (0.7%)	2/769 (0.3%)	0.276
No of patients discharged without admission to hospital, who were adjudicated T1 or T2MI on index admission, n (%)	3/192 (1.6%)	2/82 (2.4%)	0.620

MI, revascularisation or all cause death. P values calculated using χ^2 test of independence.

ED, emergency department; MACE, major adverse cardiac events; NA, not available; T1MI, type 1 myocardial infarction; UA, unstable angina.

Similar results were found by Sandeman *et al.*⁴ They found a significantly lower LOS for patients on a 3-hour rule-out pathway as opposed to the previous 6-hour pathway. In a stepped-wedge cluster RCT in Scotland,²⁰ 31 492 consecutive patients were analysed; 14 700 in the standard care arm and 16 792 in the intervention arm. The standard care involved hs-TnI (Abbot Architect troponin I) sampling at presentation and then repeated at 6–12 hours if required. The intervention arm had a single sample rule out if initial hs-TnI was <5 ng/L and symptom onset was >2 hours prior. Samples were repeated at 3 hours with the potential to rule out MI if the delta values were met. In this trial, the intervention arm reduced LOS from 10.1±4.1 hours to 6.8±3.9 hours (p<0.001). Discharge rates from the ED were increased from 50% to 71% (OR 1.59 (95% CI 1.45 to 1.75%). There was no significant difference in MI or cardiac mortality between the groups at 30 days and 1 year.

The LACPP was introduced following endorsement in the 2015 ESC guidelines for management of patients without persistent ST-elevation on ECG.⁷ This project provides an insight into the practice of the hospital trusts it was undertaken in. However, it must be recognised that these recommendations are somewhat historic. The two most recent guidelines in 2020 and 2023 have endorsed and now recommend 0/1-hour and 0/2-hour rule out algorithms.^{21 22} At the time of data collection, these more rapid rule out algorithms were in their infancy.

The ability for the LACPP to reduce LOS and burden on EDs is dependent on reduced intervals to repeat sampling. With the newly endorsed 0/1-and 0/2-hour algorithms, this interval is further reduced. This is an exciting prospect but not exempt from wider ED

pressures. It relies on timely sampling and prompt action once the result is available. There are various points in the process where there could be delays.

An observational implementation study by Twerenbold *et al.*²³ assessed the effect of the 0/1-hour rule out algorithm on ED LOS. In 2296 patients with suspected ACS, the median LOS in the ED from admission to either transfer or discharge was 150 min. There was no comparator group in this study but given that average time to discharge was less than 3 hours, it is likely that this would be superior to a 0/3-hour algorithm. This study, however, was conducted in private healthcare systems so these results are not directly applicable to public healthcare systems like in the UK. Further research is required to assess feasibility of the 0/1-hour algorithm in this setting.

A randomised controlled trial of 3378 patients compared the 0/1-hour rule out algorithm with a masked, non-high sensitive 0/3-hour algorithm.²⁴ Patients managed with the 0/1-hour and the 0/3-hour algorithm had a median ED LOS of 4.6 hours (IQR 3.4–6.4) and 5.6 hours (IQR 4.0–7.1) respectively (p=0.001). Interestingly, the median LOS of patients managed in this trial's 0/3-hour arm was very similar to those in our QIP, 5.6 hours and 5.5 hours, respectively. This indicates that a similar improvement may be possible with a 0/1-hour algorithm.

Time to blood sampling improved following LACPP implementation, the median time was still 90 min. This could be improved with staff training in triage of suspected ACS patients, including timely troponin sampling at the front door. In addition, phlebotomists present in ED may ease the pressure on other clinical staff. The time to initial ECG was longer following the introduction of the LACPP and was much longer than the recommended 10

min by the ESC.⁷ Dedicated ECG technicians based in ED will lead to improvements here.

The introduction of the LACPP reduced the number of patients having multiple troponins. This was in part due to the inclusion the single sample rule out pathway for patients with pain >3 hours and an initial hs-TnT of <5 ng/L, the LOD of the assay.

A meta-analysis of 11 cohorts with a pooled study of population of 9241 patients, by Pickering *et al*⁶ revealed that an initial hs-TnT <5 ng/L (LOD) and a non-ischaemic ECG gave a pooled sensitivity of 98.7% (95% CI 96.6% to 99.5%) and negative predictive value of 99.3% (95% CI 97.3% to 99.8%). These were favourable results but below the consensus goal of 99.5% for negative predictive value. In this study, of the 14 false negatives in the population, 7 were in patients whose chest pain onset was <3 hours to troponin draw. This highlights the challenges of early presenters with rapid rule-out strategies. Careful consideration is needed to discharge based on a single sample, particularly if the onset is <2 hours before blood draw. Therefore, the LACPP advocates a repeat troponin sample at 3 hours even if the initial is below the LOD.

Reduced need for repeat sampling has economic benefits. Not only does it reduce ED LOS, but it reduces work load in the biochemistry laboratory. It avoids unnecessary testing, which may in turn reduce burden and have a positive effect on lab result turnaround time.

Limitations

The data collected are not randomised. There were differences in patient complexity and demographics between the two cohorts. The 2011 cohort was higher risk, and therefore, comparing discharge rates will be confounded by these differences. Clinical practice changes over time, including discharge decision-making. Given the difference in patient population risk factors, the ADP may not be the only factor influencing discharge. Discharge from ED did almost double, however.

We do not have data regarding rates of angiography and intervention in either cohort. Therefore, we cannot draw conclusions regarding the appropriateness of invasive intervention in those ruled-in based on troponin results.

The 2011 data for this QIP were collected retrospectively. Therefore, not all the recorded information was available and documented for each patient and not all patients were included in analysis for every variable.

The study analysed data up to 30 days post admission. Therefore, we are unable to comment on the safety of the LACPP beyond this period.

Strengths

Data from 3003 patients across two cohorts constitutes a large sample of real-world practice. It is an accurate representation of a busy ED. Patient inclusion was also consecutive with a 100% follow-up rate, nationally in the 2011 cohort and regionally in 2018 cohort. The size of this sample and the unselected, consecutive nature of

data collection indicates generalisability of these findings. Patients diagnosed with MI were adjudicated by two physicians, to ensure accuracy of diagnosis. This level of quality control is something rarely seen in QIPs.

CONCLUSION

In patients presenting to ED with symptoms suggestive of ACS, our data suggest that, early discharge is significantly enhanced with implementation of the ADP without any significant harm. However, maximal potential of the ADP can only be achieved by continued improvements in healthcare provision, staff education and adequate resources.

Author affiliations

¹Liverpool Centre for Cardiovascular Sciences, University of Liverpool, Liverpool, UK

²Department of Cardiology, Royal Liverpool and Broadgreen Hospitals NHS Trust, Liverpool, UK

³Department of Cardiology, Aintree University Hospitals NHS Foundation Trust, Liverpool, UK

⁴Clinical Blood Sciences, St George's University Hospitals NHS Foundation Trust, London, UK

⁵Department of Cardiology, Liverpool Heart and Chest NHS Foundation Trust, Liverpool, UK

Acknowledgements Cheshire and Mersey primary care trust.

Contributors AK conceived the idea for the project, developed conversations with emergency department clinicians, designed the database and Liverpool Acute Chest Pain Pathway. AK undertook data collection for the study with a host of other contributors. AK was a consultant adjudicator for type 1 and type 2 myocardial infarction. JDH and TS undertook statistical analysis of the raw data from the two cohorts. TS created the figures used for the manuscript. JDH wrote the manuscript and is acting as guarantor for the manuscript and work. AK and POC gave intellectual input and provided critical appraisal of the manuscript.

Funding Bayer medical, Dragons den innovation award (Liverpool University Hospital NHS Foundation Trust).

Competing interests AK received a grant of £8000 for this work from Bayer medical. No conflict of interest from all other authors.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and the 2011 cohort involved reporting of any representation with possible MI nationwide. The project was registered with the hospital's research department and the North-West England Regional Ethics Board, which granted full consent to undertake this study. To allow for follow-up special permission was granted, in the absence of individual consent, via a confidential advisory board (UK Government Home Office appointed) for the recruitment of consecutive chest pain population and collection of data from any hospital nationwide. This facilitated the retrieval of clinical records and blood results for patients with possible ACS (15/CAG/0171) (<http://www.hra.nhs.uk/>). The 2018 UK validation cohort was undertaken as a quality improvement programme, and therefore, the requirement of consent for each patient identified was waived. This allowed extraction of data from regional hospitals (circa population 2.6 million). This manuscript conforms to the International Committee of Medical Journal Editors recommendations for the conduct, reporting, editing and publication of work in medical journals. It complies with the declaration of Helsinki.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available on reasonable request. Data used for statistical analysis is available for review on request.

Supplemental material This content has been supplied by the author(s). It has not been vetted by BMJ Publishing Group Limited (BMJ) and may not have been peer-reviewed. Any opinions or recommendations discussed are solely those of the author(s) and are not endorsed by BMJ. BMJ disclaims all liability and responsibility arising from any reliance placed on the content. Where the content includes any translated material, BMJ does not warrant the accuracy and reliability of the translations (including but not limited to local regulations, clinical guidelines,

terminology, drug names and drug dosages), and is not responsible for any error and/or omissions arising from translation and adaptation or otherwise.

Open access This is an open access article distributed in accordance with the Creative Commons Attribution 4.0 Unported (CC BY 4.0) license, which permits others to copy, redistribute, remix, transform and build upon this work for any purpose, provided the original work is properly cited, a link to the licence is given, and indication of whether changes were made. See: <https://creativecommons.org/licenses/by/4.0/>.

ORCID iDs

James Daniel Hatherley <http://orcid.org/0009-0001-3262-434X>

Paul O Collinson <http://orcid.org/0000-0002-7000-5996>

REFERENCES

- Reichlin T, Schindler C, Drexler B, *et al.* One-hour Rule- out and rule-in of acute myocardial infarction using high- sensitivity cardiac troponin T. *Arch Intern Med* 2012;172:1211–8.
- Badertscher P, Boeddinghaus J, Twerenbold R, *et al.* Direct comparison of the 0/1H and 0/3H Algorithms for early rule-out of acute myocardial infarction. *Circulation* 2018;137:2536–8.
- Westwood ME, Armstrong N, Worthy G, *et al.* Optimizing the use of high-sensitivity troponin assays for the early rule-out of myocardial infarction in patients presenting with chest pain: A systematic review. *Clin Chem* 2021;67:237–44.
- Sandeman D, Syed MJB, Kimenai DM, *et al.* Implementation of an early rule-out pathway for myocardial infarction using a high-sensitivity cardiac troponin T assay. *Open Heart* 2021;8:e001769.
- Pickering JW, Than MP, Cullen L, *et al.* Rapid rule-out of acute myocardial infarction with a single high-sensitivity cardiac troponin T measurement below the limit of detection. A collaborative meta-analysis. *Ann Intern Med* 2017;166:715–24.
- Chew PG, Frost F, Mullen L, *et al.* A direct comparison of decision rules for early discharge of suspected acute coronary syndromes in the era of high sensitivity troponin. *Eur Heart J Acute Cardiovasc Care* 2019;8:421–31.
- Roffi M, Patrono C, Collet J-P, *et al.* ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: task force for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. *Eur Heart J* 2016;37:267–315.
- Weber M, Bazzino O, Navarro Estrada JL, *et al.* Improved diagnostic and Prognostic performance of a new high-sensitive troponin T assay in patients with acute coronary syndrome. *Am Heart J* 2011;162:81–8.
- Thygesen K, Alpert JS, Jaffe AS, *et al.* Third universal definition of myocardial infarction. *J Am Coll Cardiol* 2012;60:1581–98.
- Thygesen K, Mair J, Giannitsis E, *et al.* How to use high-sensitivity cardiac Troponins in acute cardiac care. *Eur Heart J* 2012;33:2252–7.
- Mair J, Lindahl B, Müller C, *et al.* What to do when you question cardiac troponin values. *Eur Heart J Acute Cardiovasc Care* 2018;7:577–86.
- Giannitsis E, Becker M, Kurz K, *et al.* High-sensitivity cardiac troponin t for early prediction of evolving non–st-segment elevation myocardial infarction in patients with suspected acute coronary syndrome and negative troponin results on admission. *Clin Chem* 2010;56:642–50.
- Keller T, Zeller T, Ojeda F, *et al.* Serial changes in highly sensitive troponin I assays and early diagnosis of myocardial infarction. *JAMA* 2011;306:2684.
- Mueller C, Twerenbold R, Reichlin T. Early diagnosis of myocardial infarction with sensitive cardiac troponin assays. *Clin Chem* 2019;65:490–1.
- Collinson P, Suvisaari J, Aakre KM, *et al.* How well do Laboratories adhere to recommended guidelines for cardiac biomarkers management in Europe? the cardiac marker guideline uptake in Europe (CAMARGUE) study of the European Federation of laboratory medicine task group on cardiac markers. *Clin Chem* 2021;67:1144–52.
- Gov.UK (Website); UK government Website; accelerated access review. n.d. Available: <https://www.gov.uk/government/publications/accelerated-access-review-final-report>
- Chapman AR, Anand A, Boeddinghaus J, *et al.* Comparison of the efficacy and safety of early rule-out pathways for acute myocardial infarction. *Circulation* 2017;135:1586–96.
- Boeddinghaus J, Nestelberger T, Twerenbold R, *et al.* Direct comparison of 4 very early rule-out strategies for acute myocardial infarction using high-sensitivity cardiac troponin I. *Circulation* 2017;135:1597–611.
- Thygesen K, Alpert JS, Jaffe AS, *et al.* n.d. Third universal definition of myocardial infarction. *Gh*;7:275.
- Anand A, Lee KK, Chapman AR, *et al.* High-sensitivity cardiac troponin on presentation to rule-out myocardial infarction: A stepped-wedge cluster randomized controlled trial. *Circulation* 2021;143:2214–24.
- Collet JP, Thiele H, Barbato E. ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. *Eur Heart J* 2020;41:3495–7.
- Byrne RA, Rossello X, Coughlan JJ, *et al.* ESC guidelines for the management of acute coronary syndromes: developed by the task force on the management of acute coronary syndromes of the European society of cardiology (ESC). *Eur Heart J* 2023;44:3720–826.
- Twerenbold R, Costabel JP, Nestelberger T, *et al.* Outcome of applying the ESC 0/1-hour algorithm in patients with suspected myocardial infarction. *J Am Coll Cardiol* 2019;74:483–94.
- Chew DP, Lambrakis K, Blyth A, *et al.* A randomized trial of a 1-hour troponin T protocol in suspected acute coronary syndromes: the rapid assessment of possible acute coronary syndrome in the emergency Department with high-sensitivity troponin T study (RAPID-TNT). *Circulation* 2019;140:1543–56.

Table s1

ICD-10 codes for chest pain, coronary artery disease and revascularisation used to extract follow-up data.

CLINICAL_CODE	CODE_SET_ID	VERSION	SHORT_DESC	LONG_DESC	OPCS_VERSION
K40	OPCS	4	SAPHENOUS VEIN GRAFT REPLACEMENT	SAPHENOUS VEIN GRAFT REPLACEMENT OF CORONARY ARTERY	4.2
K401	OPCS	4	Saphenous vein graft replaceme	Saphenous vein graft replacement of one coronary artery	4.2
K4010	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B11	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B14	OPCS	4	CORONARY BYPASS GRAFTS - ENDO	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B16	OPCS	4	REPAIR ASC. AORTA & COR. BYPAS	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B17	OPCS	4	REPAIR AORTIC ANEURYSM & BYPAS	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B18	OPCS	4	CABG & OTHER MAJOR CARDIAC (EG	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B1A	OPCS	4	TMR & CABG	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B1B	OPCS	4	CABG & CAROTID ENDARTERECTOMY	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B1C	OPCS	4	CABG & ABDOMINAL AORTIC ANEURY	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B21	OPCS	4	REPLACEMENT/REPAIR OF VALVE(S)	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B22	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K4010B23	OPCS	4	COMPOSITE REPL AORTA, VALVE + C	SAPHENOUS VEIN GRAFT REPLACEMENT OF ONE CORONARY ARTERY	4.2
K402	OPCS	4	Saphenous vein graft replaceme	Saphenous vein graft replacement of two coronary arteries	4.2
K4020	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B11	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B14	OPCS	4	CORONARY BYPASS GRAFTS - ENDO	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B16	OPCS	4	REPAIR ASC. AORTA & COR. BYPAS	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B17	OPCS	4	REPAIR AORTIC ANEURYSM & BYPAS	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B18	OPCS	4	CABG & OTHER MAJOR CARDIAC (EG	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B1A	OPCS	4	TMR & CABG	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B1B	OPCS	4	CABG & CAROTID ENDARTERECTOMY	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B1C	OPCS	4	CABG & ABDOMINAL AORTIC ANEURY	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B21	OPCS	4	REPLACEMENT/REPAIR OF VALVE(S)	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B22	OPCS	4	CABG + AVR + Carotid Endertere	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B23	OPCS	4	COMPOSITE REPL AORTA, VALVE + C	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K4020B32	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF TWO CORONARY ARTERIES	4.2
K403	OPCS	4	Saphenous vein graft replaceme	Saphenous vein graft replacement of three coronary arteries	4.2
K4030	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B11	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B14	OPCS	4	CORONARY BYPASS GRAFTS - ENDO	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B16	OPCS	4	REPAIR ASC. AORTA & COR. BYPAS	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2

K4030B18	OPCS	4	CABG & OTHER MAJOR CARDIAC (EG	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B1A	OPCS	4	TMR & CABG	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B1B	OPCS	4	CABG & CAROTID ENDARTERECTOMY	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B1C	OPCS	4	CABG & ABDOMINAL AORTIC ANEURY	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B21	OPCS	4	REPLACEMENT/REPAIR OF VALVE(S)	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B22	OPCS	4	CABG + AVR + Carotid Endertere	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B23	OPCS	4	COMPOSITE REPL AORTA, VALVE + C	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K4030B42	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF THREE CORONARY ARTERIES	4.2
K404	OPCS	4	Saphenous vein graft replaceme	Saphenous vein graft replacement of four or more coronary ar	4.2
K4040	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF FOUR OR MORE CORONARY AR	4.2
K4040B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	SAPHENOUS VEIN GRAFT REPLACE FOUR OR MORE CORONARY ARTERIES	4.2
K4040B11	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	SAPHENOUS VEIN GRAFT REPLACE FOUR OR MORE CORONARY ARTERIES	4.2
K4040B14	OPCS	4	CORONARY BYPASS GRAFTS - ENDO	SAPHENOUS VEIN GRAFT REPLACE FOUR OR MORE CORONARY ARTERIES	4.2
K4040B16	OPCS	4	REPAIR ASC, AORTA & COR. BYPAS	SAPHENOUS VEIN GRAFT REPLACE FOUR OR MORE CORONARY ARTERIES	4.2
K4040B18	OPCS	4	CABG & OTHER MAJOR CARDIAC (EG	SAPHENOUS VEIN GRAFT REPLACE FOUR OR MORE CORONARY ARTERIES	4.2
K4040B1A	OPCS	4	TMR & CABG	SAPHENOUS VEIN GRAFT REPLACE FOUR OR MORE CORONARY ARTERIES	4.2
K4040B1B	OPCS	4	CABG & CAROTID ENDARTERECTOMY	SAPHENOUS VEIN GRAFT REPLACE FOUR OR MORE CORONARY ARTERIES	4.2
K4040B21	OPCS	4	REPLACEMENT/REPAIR OF VALVE(S)	SAPHENOUS VEIN GRAFT REPLACE FOUR OR MORE CORONARY ARTERIES	4.2
K4040B22	OPCS	4	CABG + AVR + CAROTID ENDARTERE	SAPHENOUS VEIN GRAFT REPLACE FOUR OR MORE CORONARY ARTERIES	4.2
K4040B23	OPCS	4	COMPOSITE REPL AORTA, VALVE + C	SAPHENOUS VEIN GRAFT REPLACE FOUR OR MORE CORONARY ARTERIES	4.2
K4040B52	OPCS	4	Coronary Artery Bypass Grafts	SAPHENOUS VEIN GRAFT REPLACEMENT OF FOUR OR MORE CORONARY AR	4.2
K408	OPCS	4	Other specified saphenous vein	Other specified saphenous vein graft replacement of coronary	4.2
K409	OPCS	4	Unspecified saphenous vein gra	Unspecified saphenous vein graft replacement of coronary art	4.2
K41	OPCS	4	OTHER AUTOGRAFT REPLACEMENT OF	OTHER AUTOGRAFT REPLACEMENT OF CORONARY ARTERY	4.2
K411	OPCS	4	Autograft replacement of one c	Autograft replacement of one coronary artery NEC	4.2
K4110	OPCS	4	CABG + AVR + Carotid Endertere	AUTOGRAFT REPLACEMENT OF ONE CORONARY ARTERY NEC	4.2
K4110B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	AUTOGRAFT REPLACEMENT OF ONE CORONARY ARTERY NEC	4.2
K4110B11	OPCS	4	Coronary Artery Bypass Grafts	AUTOGRAFT REPLACEMENT OF ONE CORONARY ARTERY NEC	4.2
K4110B1A	OPCS	4	TMR & CABG	AUTOGRAFT REPLACEMENT OF ONE CORONARY ARTERY NEC	4.2
K4110B21	OPCS	4	AUTO REPLACE ONE CON ART	AUTOGRAFT REPLACEMENT OF ONE CORONARY ARTERY NEC	4.2
K4110B22	OPCS	4	CABG + AVR + Carotid Endertere	AUTOGRAFT REPLACEMENT OF ONE CORONARY ARTERY NEC	4.2
K412	OPCS	4	Autograft replacement of two c	Autograft replacement of two coronary arteries NEC	4.2
K4120	OPCS	4	CABG + AVR + Carotid Endertere	AUTOGRAFT REPLACEMENT OF TWO CORONARY ARTERIES NEC	4.2
K4120B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	AUTOGRAFT REPLACEMENT OF TWO CORONARY ARTERIES NEC	4.2
K4120B11	OPCS	4	Coronary Artery Bypass Grafts	AUTOGRAFT REPLACEMENT OF TWO CORONARY ARTERIES NEC	4.2
K4120B1A	OPCS	4	TMR & CABG	AUTOGRAFT REPLACEMENT OF TWO CORONARY ARTERIES NEC	4.2
K4120B21	OPCS	4	AUTO REPLACE TWO CON ART	AUTOGRAFT REPLACEMENT OF TWO CORONARY ARTERIES NEC	4.2
K4120B22	OPCS	4	CABG + AVR + Carotid Endertere	AUTOGRAFT REPLACEMENT OF TWO CORONARY ARTERIES NEC	4.2
K413	OPCS	4	Autograft replacement of three	Autograft replacement of three coronary arteries NEC	4.2
K4130	OPCS	4	CABG + AVR + Carotid Endertere	AUTOGRAFT REPLACEMENT OF THREE CORONARY ARTERIES NEC	4.2
K4130B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	AUTOGRAFT REPLACEMENT OF THREE CORONARY ARTERIES NEC	4.2

K4130B11	OPCS	4	Coronary Artery Bypass Grafts	AUTOGRAFT REPLACEMENT OF THREE CORONARY ARTERIES NEC	4.2
K4130B1A	OPCS	4	TMR & CABG	AUTOGRAFT REPLACEMENT OF THREE CORONARY ARTERIES NEC	4.2
K4130B21	OPCS	4	AUTO REPLACE THREE CON ART	AUTOGRAFT REPLACEMENT OF THREE CORONARY ARTERIES NEC	4.2
K4130B22	OPCS	4	CABG + AVR + Carotid Endertere	AUTOGRAFT REPLACEMENT OF THREE CORONARY ARTERIES NEC	4.2
K414	OPCS	4	Autograft replacement of four	Autograft replacement of four or more coronary arteries NEC	4.2
K4140	OPCS	4	CABG + AVR + Carotid Endertere	AUTOGRAFT REPLACEMENT OF FOUR OR MORE CORONARY ARTERIES NEC	4.2
K4140B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	AUTOGRAFT REPLACEMENT OF FOUR OR MORE CORONARY ARTERIES NEC	4.2
K4140B11	OPCS	4	Coronary Artery Bypass Grafts	AUTOGRAFT REPLACEMENT OF FOUR OR MORE CORONARY ARTERIES NEC	4.2
K4140B1A	OPCS	4	TMR & CABG	AUTOGRAFT REPLACEMENT OF FOUR OR MORE CORONARY ARTERIES NEC	4.2
K4140B21	OPCS	4	AUTO REPLACE FOUR > CON ART	AUTOGRAFT REPLACEMENT OF FOUR OR MORE CORONARY ARTERIES NEC	4.2
K4140B22	OPCS	4	CABG + AVR + Carotid Endertere	AUTOGRAFT REPLACEMENT OF FOUR OR MORE CORONARY ARTERIES NEC	4.2
K418	OPCS	4	Other specified other autograf	Other specified other autograft replacement of coronary arte	4.2
K4180	OPCS	4	TMR & CABG	OTHER SPECIFIED	4.2
K4180B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	OTHER AUTOGRAFT REPLACEMENT OF CORONARY ARTERY OS	4.2
K4180B11	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	OTHER AUTOGRAFT REPLACEMENT OF CORONARY ARTERY OS	4.2
K4180B1A	OPCS	4	TMR & CABG	OTHER AUTOGRAFT REPLACEMENT OF CORONARY ARTERY OS	4.2
K419	OPCS	4	Unspecified other autograft re	Unspecified other autograft replacement of coronary artery	4.2
K4190	OPCS	4	TMR & CABG	UNSPECIFIED	4.2
K4190B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	OTHER AUTOGRAFT REPLACEMENT OF CORONARY ARTERY UNSPECIFIED	4.2
K4190B11	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	OTHER AUTOGRAFT REPLACEMENT OF CORONARY ARTERY UNSPECIFIED	4.2
K4190B1A	OPCS	4	TMR & CABG	OTHER AUTOGRAFT REPLACEMENT OF CORONARY ARTERY UNSPECIFIED	4.2
K42	OPCS	4	ALLOGRAFT REPLACEMENT OF CORON	ALLOGRAFT REPLACEMENT OF CORONARY ARTERY	4.2
K421	OPCS	4	Allograft replacement of one c	Allograft replacement of one coronary artery	4.2
K422	OPCS	4	Allograft replacement of two c	Allograft replacement of two coronary arteries	4.2
K423	OPCS	4	Allograft replacement of three	Allograft replacement of three coronary arteries	4.2
K424	OPCS	4	Allograft replacement of four	Allograft replacement of four or more coronary arteries	4.2
K428	OPCS	4	Other specified allograft repl	Other specified allograft replacement of coronary artery	4.2
K429	OPCS	4	Unspecified allograft replacem	Unspecified allograft replacement of coronary artery	4.2
K43	OPCS	4	PROSTHETIC REPLACEMENT OF CORO	PROSTHETIC REPLACEMENT OF CORONARY ARTERY	4.2
K431	OPCS	4	Prosthetic replacement of one	Prosthetic replacement of one coronary artery	4.2
K432	OPCS	4	Prosthetic replacement of two	Prosthetic replacement of two coronary arteries	4.2
K433	OPCS	4	Prosthetic replacement of thre	Prosthetic replacement of three coronary arteries	4.2
K434	OPCS	4	Prosthetic replacement of four	Prosthetic replacement of four or more coronary arteries	4.2
K438	OPCS	4	Other specified prosthetic rep	Other specified prosthetic replacement of coronary artery	4.2
K439	OPCS	4	Unspecified prosthetic replac	Unspecified prosthetic replacement of coronary artery	4.2
K44	OPCS	4	OTHER REPLACEMENT OF CORONARY	OTHER REPLACEMENT OF CORONARY ARTERY	4.2
K441	OPCS	4	Replacement of coronary arteri	Replacement of coronary arteries using multiple methods	4.2
K442	OPCS	4	Revision of replacement of cor	Revision of replacement of coronary artery	4.2
K4420	OPCS	4	POST OP ATTENTION TO GRAFTS -	REVISION OF REPLACEMENT OF CORONARY ARTERY	4.2
K4420B15	OPCS	4	POST OP ATTENTION TO GRAFTS -	REVISION OF REPLACEMENT OF CORONARY ARTERY	4.2
K448	OPCS	4	Other specified other replacem	Other specified other replacement of coronary artery	4.2
K449	OPCS	4	Unspecified other replacement	Unspecified other replacement of coronary artery	4.2

K451	OPCS	4	Double anastomosis of mammary	Double anastomosis of mammary arteries to coronary arteries	4.2
K4510B10	OPCS	4	DOUBLE ANASTOMOSIS OF MAMMARY	DOUBLE ANASTOMOSIS OF MAMMARY ARTERIES TO CORONARY ARTERIES	4.2
K4510B11	OPCS	4	DOUBLE ANASTOMOSIS OF MAMMARY	DOUBLE ANASTOMOSIS OF MAMMARY ARTERIES TO CORONARY ARTERIES	4.2
K4510B21	OPCS	4	CABG WITH VALVE REPAIR	DOUBLE ANASTOMOSIS OF MAMMARY ARTERIES TO CORONARY ARTERIES	4.2
K452	OPCS	4	Double anastomosis of thoracic	Double anastomosis of thoracic arteries to coronary arteries	4.2
K453	OPCS	4	Anastomosis of mammary artery	Anastomosis of mammary artery to left anterior descending co	4.2
K4530	OPCS	4	Coronary Artery Bypass Grafts	ANASTOMOSIS OF MAMMARY ARTERY TO LEFT ANTERIOR DESCENDING CO	4.2
K4530B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B11	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B13	OPCS	4	MIDCAB-MIN INVASIVE BYPASS GRA	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B14	OPCS	4	CORONARY BYPASS GRAFTS - ENDO	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B16	OPCS	4	REPAIR ASC. AORTA & COR. BYPAS	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B18	OPCS	4	CABG & OTHER MAJOR CARDIAC (EG	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B1A	OPCS	4	TMR & CABG	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B1B	OPCS	4	CABG & CAROTID ENDARTERECTOMY	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B21	OPCS	4	REPLACEMENT/REPAIR OF VALVE(S)	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B22	OPCS	4	CABG + AVR + CAROTID ENDARTERE	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B23	OPCS	4	COMPOSITE REPL AORTA, VALVE + C	ANAST MAMMARY ARTERY TO LT ANTERIOR DESCENDING CORON ARTERY	4.2
K4530B62	OPCS	4	Coronary Artery Bypass Grafts	ANASTOMOSIS OF MAMMARY ARTERY TO LEFT ANTERIOR DESCENDING CO	4.2
K454	OPCS	4	Anastomosis of mammary artery	Anastomosis of mammary artery to coronary artery NEC	4.2
K4540	OPCS	4	Coronary Artery Bypass Grafts	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B10	OPCS	4	CORONARY ARTERY BYPASS GRAFTS	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B11	OPCS	4	Coronary Artery Bypass Grafts	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B12	OPCS	4	Coronary Artery Bypass Grafts	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B14	OPCS	4	CORONARY BYPASS GRAFTS - ENDO	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B16	OPCS	4	REPAIR ASC. AORTA & COR. BYPAS	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B18	OPCS	4	CABG & OTHER MAJOR CARDIAC (EG	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B1A	OPCS	4	TMR & CABG	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B1B	OPCS	4	CABG & CAROTID ENDARTERECTOMY	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B21	OPCS	4	REPLACE/REPAIR VALVE(S) & CABG	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B22	OPCS	4	CABG + AVR + Carotid Endertere	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K4540B23	OPCS	4	COMPOSITE REPL AORTA, VALVE + C	ANASTOMOSIS OF MAMMARY ARTERY TO CORONARY ARTERY NEC	4.2
K455	OPCS	4	Anastomosis of thoracic artery	Anastomosis of thoracic artery to coronary artery NEC	4.2
K456	OPCS	4	Revision of connection of thor	Revision of connection of thoracic artery to coronary artery	4.2
K458	OPCS	4	Other specified connection of	Other specified connection of thoracic artery to coronary ar	4.2
K459	OPCS	4	Unspecified connection of thor	Unspecified connection of thoracic artery to coronary artery	4.2
K46	OPCS	4	OTHER BYPASS OF CORONARY ARTER	OTHER BYPASS OF CORONARY ARTERY	4.2
K463	OPCS	4	Implantation of mammary artery	Implantation of mammary artery into heart NEC	4.2
K468	OPCS	4	Other specified other bypass o	Other specified other bypass of coronary artery	4.2
K469	OPCS	4	Unspecified other bypass of co	Unspecified other bypass of coronary artery	4.2
K49	OPCS	4	TRANSLUMINAL BALLOON ANGIOPLAS	TRANSLUMINAL BALLOON ANGIOPLASTY OF CORONARY ARTERY	4.2

K491	OPCS	4	Percutaneous transluminal ball	Percutaneous transluminal balloon angioplasty of one coronar	4.2
K4910	OPCS	4	LASER ANGIOPLASTY - ONE BALLOO	PERCUTANEOUS TRANSLUMINAL BALLOON ANGIOPLASTY OF ONE CORONAR	4.2
K4910D11	OPCS	4	ABANDONED PTCA	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D12	OPCS	4	SIMPLE PTCA - ONE OR TWO BALLO	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D21	OPCS	4	SIMPLE PTCA-MORE THAN 2 BALLOO	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D34	OPCS	4	PTCA - ONE STENT & ONE BALLOON	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D35	OPCS	4	PTCA - 1 STENT, NO BALLOON	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D37	OPCS	4	LASER ANGIOPLASTY - ONE BALLOO	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D41	OPCS	4	PTCA - 2 OR MORE STENTS	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D42	OPCS	4	PTCA - 2 OR MORE STENTS, NO BA	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D44	OPCS	4	PTCA WITH STENT & ROTABLATOR	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D45	OPCS	4	PTCA WITH STENT - 2+ BALLOONS	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D49	OPCS	4	PTCA WITH 2+STENTS (2+ BALLOON	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D4B	OPCS	4	LASER ANGIOPLASTY, MORE THAN O	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K4910D4C	OPCS	4	LASER ANGIOPLASTY WITH STENT(S	PERCUT TRANSLUMINAL BALLOON ANGIOPLASTY ONE CORONARY ARTERY	4.2
K492	OPCS	4	Percutaneous transluminal ball	Percutaneous transluminal balloon angioplasty of multiple co	4.2
K4920	OPCS	4	PTCA with Stent & Rotablator	PERCUTANEOUS TRANSLUMINAL BALLOON ANGIOPLASTY OF MULTIPLE CO	4.2
K4920D11	OPCS	4	ABANDONED PTCA	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D12	OPCS	4	SIMPLE PTCA - ONE OR TWO BALLO	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D21	OPCS	4	SIMPLE PTCA-MORE THAN 2 BALLOO	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D34	OPCS	4	PTCA - ONE STENT & ONE BALLOON	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D35	OPCS	4	PTCA - 1 STENT, NO BALLOON	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D37	OPCS	4	LASER ANGIOPLASTY - 1 BALLOON	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D41	OPCS	4	PTCA - 2 OR MORE STENTS	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D42	OPCS	4	PTCA - 2 STENTS, NO BALLOON	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D44	OPCS	4	PTCA WITH STENT & ROTABLATOR	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D45	OPCS	4	PTCA WITH STENT - 2+ BALLOONS	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D49	OPCS	4	PTCA WITH 2+STENTS (2+ BALLOON	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D4B	OPCS	4	LASER ANGIOPLASTY - 1 BALLOON	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K4920D4C	OPCS	4	LASER ANGIOPLASTY WITH STENT(S	PERCUT TRANSLUM BALLOON ANGIOPLASTY MULTIPLE CORON ARTERIES	4.2
K493	OPCS	4	Percutaneous transluminal ball	Percutaneous transluminal balloon angioplasty of bypass graf	4.2
K4930	OPCS	4	PTCA with Stent & Rotablator	PERCUTANEOUS TRANSLUMINAL BALLOON ANGIOPLASTY OF BYPASS GRAF	4.2
K4930D12	OPCS	4	SIMPLE PTCA - ONE OR TWO BALLO	PERCUT TRANSLUM BALLOON ANGIOPLASTY BYPASS GRAFT CORON ART	4.2
K4930D21	OPCS	4	SIMPLE PTCA-MORE THAN 2 BALLOO	PERCUT TRANSLUM BALLOON ANGIOPLASTY BYPASS GRAFT CORON ART	4.2
K4930D34	OPCS	4	PTCA - ONE STENT & ONE BALLOON	PERCUT TRANSLUM BALLOON ANGIOPLASTY BYPASS GRAFT CORON ART	4.2
K4930D35	OPCS	4	PTCA - 1 STENT, NO BALLOON	PERCUT TRANSLUM BALLOON ANGIOPLASTY BYPASS GRAFT CORON ART	4.2
K4930D41	OPCS	4	PTCA - 2 OR MORE STENTS	PERCUT TRANSLUM BALLOON ANGIOPLASTY BYPASS GRAFT CORON ART	4.2
K4930D44	OPCS	4	PTCA WITH STENT & ROTABLATOR	PERCUT TRANSLUM BALLOON ANGIOPLASTY BYPASS GRAFT CORON ART	4.2
K4930D45	OPCS	4	PTCA WITH STENT - 2+ BALLOONS	PERCUT TRANSLUM BALLOON ANGIOPLASTY BYPASS GRAFT CORON ART	4.2
K4930D49	OPCS	4	PTCA WITH 2+STENTS (2+ BALLOON	PERCUT TRANSLUM BALLOON ANGIOPLASTY BYPASS GRAFT CORON ART	4.2
K494	OPCS	4	Percutaneous transluminal cutt	Percutaneous transluminal cutting balloon angioplasty of cor	4.2

K498	OPCS	4	Other specified transluminal b	Other specified transluminal balloon angioplasty of coronary	4.2
K4980	OPCS	4	PTCA with Stent & Rotablator	OTHER SPECIFIED	4.2
K4980D34	OPCS	4	PTCA - ONE STENT & ONE BALLOON	TRANSLUMINAL BALLOON ANGIOPLASTY OF CORONARY ARTERY OS	4.2
K4980D41	OPCS	4	PTCA - 2 OR MORE STENTS	TRANSLUMINAL BALLOON ANGIOPLASTY OF CORONARY ARTERY OS	4.2
K4980D44	OPCS	4	PTCA WITH STENT & ROTABLATOR	TRANSLUMINAL BALLOON ANGIOPLASTY OF CORONARY ARTERY OS	4.2
K4980D45	OPCS	4	PTCA WITH STENT - 2+ BALLOONS	TRANSLUMINAL BALLOON ANGIOPLASTY OF CORONARY ARTERY OS	4.2
K4980D49	OPCS	4	PTCA WITH 2+STENTS (2+ BALLOON	TRANSLUMINAL BALLOON ANGIOPLASTY OF CORONARY ARTERY OS	4.2
K499	OPCS	4	Unspecified transluminal ballo	Unspecified transluminal balloon angioplasty of coronary art	4.2
K50	OPCS	4	OTHER THERAPEUTIC TRANSLUMINAL	OTHER THERAPEUTIC TRANSLUMINAL OPERATIONS ON CORONARY ARTERY	4.2
K501	OPCS	4	Percutaneous transluminal lase	Percutaneous transluminal laser coronary angioplasty	4.2
K5010	OPCS	4	LASER ANGIOPLASTY - NO BALLOON	PERCUTANEOUS TRANSLUMINAL LASER CORONARY ANGIOPLASTY	4.2
K5010D36	OPCS	4	LASER ANGIOPLASTY - NO BALLOON	PERCUTANEOUS TRANSLUMINAL LASER CORONARY ANGIOPLASTY	4.2
K502	OPCS	4	Percutaneous transluminal coro	Percutaneous transluminal coronary thrombolysis using strept	4.2
K503	OPCS	4	Percutaneous transluminal inje	Percutaneous transluminal injection of therapeutic substance	4.2
K504	OPCS	4	Percutaneous transluminal athe	Percutaneous transluminal atherectomy of coronary artery	4.2
K508	OPCS	4	Other specified other therapeu	Other specified other therapeutic transluminal operations on	4.2
K5080	OPCS	4	PTCA with Rota -1 balloon,1 or	PTDA	4.2
K5080D31	OPCS	4	PTCA WITH ROTA -1 BALLOON,1 OR	OTHER THERAP TRANSLUMINAL OPERATIONS ON CORONARY ARTERY OS	4.2
K5080D32	OPCS	4	CORONARY TEC - ONE BALLOON	OTHER THERAP TRANSLUMINAL OPERATIONS ON CORONARY ARTERY OS	4.2
K5080D33	OPCS	4	DCA - ONE BALLOON	OTHER THERAP TRANSLUMINAL OPERATIONS ON CORONARY ARTERY OS	4.2
K5080D43	OPCS	4	PTCA-2 OR MORE ROTABLATORS	OTHER THERAP TRANSLUMINAL OPERATIONS ON CORONARY ARTERY OS	4.2
K5080D44	OPCS	4	PTCA WITH STENT & ROTABLATOR	OTHER THERAP TRANSLUMINAL OPERATIONS ON CORONARY ARTERY OS	4.2
K5080D46	OPCS	4	PTCA WITH ROTABLATOR - 2+ BALL	OTHER THERAP TRANSLUMINAL OPERATIONS ON CORONARY ARTERY OS	4.2
K5080D47	OPCS	4	CORONARY TEC - 2+ BALLOONS	OTHER THERAP TRANSLUMINAL OPERATIONS ON CORONARY ARTERY OS	4.2
K5080D48	OPCS	4	DCA - 2+BALLOONS	OTHER THERAP TRANSLUMINAL OPERATIONS ON CORONARY ARTERY OS	4.2
K5080D4A	OPCS	4	CORONARY TEC WITH STENT(S) INC	OTHER THERAP TRANSLUMINAL OPERATIONS ON CORONARY ARTERY OS	4.2
K509	OPCS	4	Unspecified other therapeutic	Unspecified other therapeutic transluminal operations on coronary artery	4.2
K75	OPCS	4	PERCUTANEOUS TRANSLUMINAL BALL	PERCUTANEOUS TRANSLUMINAL BALLOON ANGIOPLASTY AND STENTING O	4.2
K751	OPCS	4	Percutaneous transluminal ball	Percutaneous transluminal balloon angioplasty and insertion	4.2
K752	OPCS	4	Percutaneous transluminal ball	Percutaneous transluminal balloon angioplasty and insertion	4.2
K753	OPCS	4	Percutaneous transluminal ball	Percutaneous transluminal balloon angioplasty and insertion	4.2
K754	OPCS	4	Percutaneous transluminal ball	Percutaneous transluminal balloon angioplasty and insertion	4.2
K758	OPCS	4	Other specified percutaneous t	Other specified percutaneous transluminal balloon angioplasty	4.2
K759	OPCS	4	Unspecified percutaneous trans	Unspecified percutaneous transluminal balloon angioplasty an	4.2