

mortality, non-fatal myocardial infarction, referral/admission to hospital with CP) up to a minimum of 6 months after the index assessment were analysed. Typical, atypical and NACP were determined from CP characteristics. 622 patients without confirmed CAD were diagnosed with NACP. Following the 1st visit, 70 patients were recommended coronary angiograms (27 significant CAD, 26 revascularised, 1 treated medically), 7 refused) and 66 had myocardial perfusion scans (14 reversible/2 fixed defects). 62 (10%) patients reached an end-point (2 cancer deaths, 11 non-fatal MI, 40 readmitted and 8 rereferred with CP, 1 non-haemorrhagic stroke). Of these patients, 29 were previously investigated: 21 angiograms (10 significant CAD), 14 MPS (3 reversible/1 fixed defect). Investigations after reaching end-point showed 3 significant CAD (14 angiograms) and 1 reversible defect (5 myocardial perfusion scan). 14 others were not investigated further due to unequivocally negative exercise ECG. Only the presence of diabetes predicted the end-point (OR 5.21, 95% CI 2.67 to 10.15, $p < 0.0001$) in a multiple logistic regression analysis using age, gender, current smoking, total cholesterol > 6.47 mmol/l and diabetes as covariates.

Conclusion 47 (7.6%) patient that are to be discharged without investigation, had significant coronary artery disease. Even with a short follow-up, 10% of these patients needed medical attention for suspected cardiovascular morbidity. It may be unreasonable to discharge patients describing NACP especially diabetics.

139 CLINICAL OUTCOMES OF PATIENTS EXCLUDED FROM CARDIAC INVESTIGATION IN THE NICE GUIDELINES FOR CHEST PAIN OF RECENT ONSET

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Introduction NICE guidance for the investigation of chest pain of recent onset¹ recommends clinical assessment and risk stratification within a diagnostic algorithm. Patients with pain considered "non-anginal", and those with atypical/typical anginal pain but a likelihood of coronary artery disease (CAD) $< 10\%$ are not routinely recommended for cardiac investigation. This study sought to assess whether clinical outcomes support these patients being considered at low risk of CAD.

Methods 557 consecutive patients (50.4% male; median age 55 yrs) attending rapid access chest pain clinics (RACPC) at two hospitals were risk stratified using NICE criteria. Frequency of admission with suspected angina, diagnosis of CAD and incidence of major adverse cardiac events (MACE: myocardial infarction (MI), cerebrovascular accident (CVA), emergency revascularisation or cardiac-related death) were compared for all risk categories at 6 months.

Results Of 360/557 patients with "non-anginal" pain, 14 (3.9%) were subsequently admitted with angina, 34 (9.4%) were diagnosed with CAD, 3 (0.8%) with MI and 2 (0.6%) with CVA. This group accounted for 36.9% of all patients diagnosed with CAD and 38.5% of all patients with MACE. Of 10/557 patients with atypical/typical anginal pain and a likelihood of CAD $< 10\%$, 1 (10%) was diagnosed with CAD. None were admitted with angina or diagnosed with MACE. This group accounted for 1.1% of all patients diagnosed with CAD.

Conclusions This study suggests one in ten patients routinely excluded from cardiac investigation by the NICE algorithm have CAD and just over one in a hundred of them have a MACE episode. Although these patients are considered low risk, they account for one third of adverse cardiac events in patients attending RACPC.

REFERENCE

1. National Institute for Health and Clinical Excellence. *CG95 Chest pain of recent onset: assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin*. NICE, 2010.

140 ASSESSMENT OF PRE-TEST LIKELIHOOD OF CORONARY ARTERY DISEASE IN PATIENTS WITH CHEST PAIN OF RECENT ONSET

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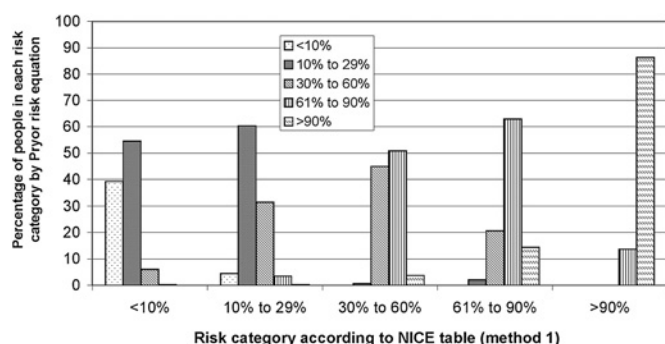
Background The NICE guideline for chest pain of recent onset recommends diagnosis of angina based on clinical assessment and estimated likelihood of coronary artery disease (CAD). Pre-test likelihood (PTL) estimates are provided in a simplified table based on the Pryor risk equation. If the PTL of CAD is $< 10\%$ or $> 90\%$, further diagnostic testing is not required. If $10\% - 29\%$ cardiac CT should be offered, if $30\% - 60\%$ functional imaging, and if $61\% - 90\%$ coronary angiography. We compared risk estimation methods to determine how much use of the table in an uncritical manner vs use of the full risk equation led to a different referral pattern. We also investigated whether interpolation for risk factor profiles would improve assessment.

Methods Data were collected prospectively for all patients referred to the Rapid Access Chest Pain Clinic, Newcastle upon Tyne, between February 2002 and August 2011. For each patient, PTL of CAD was assessed by three methods: (1) Reference to the NICE table based on chest pain (typical or atypical), age in deciles, sex and risk factors—smoking, hyperlipidaemia and diabetes dichotomised into high and low risk. For high risk, all three risk factors needed to be present; otherwise the patient was assumed low risk. (2) As per method one, but risk estimates were interpolated between low and high risk values in the NICE table according to the number of risk factors. (3) Calculation by the Pryor equation which includes, in addition, age in years, prior MI, ECG Q waves, and ST/T changes.

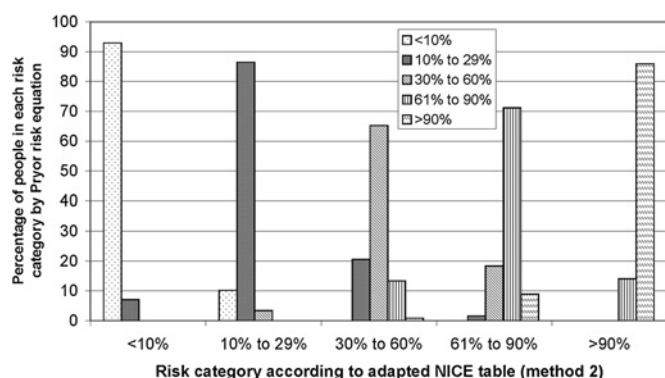
Results Out of an initial 7022 patients, 1820 were excluded as they had non-anginal chest pain. This left 5202 patients, 2889 with atypical angina and 2313 with typical angina. The number (%) of patients in the five different risk groups by the three risk assessment methods is given in Abstract 140 table 1. Use of the risk equation compared to the table led to half as many people in the category $< 10\%$. Fewer patients would be referred for cardiac CT, more for functional imaging and more for invasive coronary angiography. Use of the adapted table correlated with the risk equation better, but there were still discrepancies, and the percentage of patients for coronary angiography would increase overall (Abstract 140 table 1). The NICE table would classify only 39% of patients with PTL $< 10\%$ correctly, 60.3% of those with PTL $10\% - 29\%$, 44.9% of those with PTL $30\% - 60\%$, 62.9% of those with PTL $61\% - 90\%$, and 86.3% of those with PTL $> 90\%$ (Abstract 140 figure 1). The corresponding figures correctly classifying people in the five risk categories using the adapted table were 92.9%, 86.4%, 65.2%, 71.2% and 85.9% respectively (Abstract 140 figure 2).

Abstract 140 Table 1

Pre-test likelihood CAD	$< 10\%$	$10\% - 29\%$	$30\% - 60\%$	$61\% - 90\%$	$> 90\%$
NICE table—n (%)	365 (7.0)	1247 (24.0)	1069 (20.5)	1424 (27.4)	1097 (21.1)
Modified table—n (%)	127 (2.4)	795 (15.3)	1271 (24.4)	1821 (35.0)	1188 (22.8)
Pryor risk equation—n (%)	183 (3.5)	1002 (19.3)	1190 (22.9)	1633 (31.4)	1194 (23.0)



Abstract 140 Figure 1 Percentage of people in each risk category using the Pryor risk equation when initially categorised by the NICE table.



Abstract 140 Figure 2 Percentage of people in each risk category using the Pryor risk equation when initially categorised by the adapted NICE table.

Conclusion Use of the NICE table uncritically for assessing PTL of CAD misclassifies a significant proportion of people for further investigation. It is important to include ECG data, interpolate risk factor information, and take into account actual age when using the table rather than the Pryor equation, use of which takes these factors into account automatically.

141 CARDIAC TROPONIN REFLECTS SILENT MYOCARDIAL ISCHAEMIA IN PATIENTS WITH STABLE CORONARY ARTERY DISEASE

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Background Cardiac troponin is an independent predictor of cardiovascular mortality in patients with stable coronary artery disease, yet the factors that determine circulating troponin concentration in these patients are not known. We hypothesise that circulating cardiac troponin in patients with stable coronary heart disease is due to recurrent silent myocardial ischaemia.

Methods 98 patients with stable coronary artery disease (62 ± 7 years) were assessed on two occasions at least 2 weeks apart. Ambulatory blood pressure and 12-lead Holter monitoring were recorded for 24-h and symptom severity was assessed using the Seattle Angina Questionnaire. Significant silent myocardial ischaemia was defined as horizontal or down sloping ST-segment depression of at least 1 mm lasting at least 60 s. Cardiac troponin I

was measured in serum using the ultra-sensitive Singulex Erenna System (Singulex Inc., Berkeley, California, USA) following each visit.

Results Cardiac troponin I concentrations were quantifiable above the limit of detection (LOD, 0.4 pg/ml) in all patients with a mean concentration of 6.1 pg/ml (95% CI 4.6 to 7.6 pg/ml). The inter-assay coefficient of variation (CV) was 11% at the LOD and was 17% between visits. Concentrations were above the 99th percentile of a healthy reference population (10.1 pg/ml) in 15% of patients. Few patients ($n=2$) reported angina during either visit, but troponin concentrations were significantly higher in patients with silent myocardial ischaemia ($n=17$) compared to those without (16.1 ± 23.0 vs 5.1 ± 7.9 pg/ml; $p < 0.0001$). Troponin concentrations were associated with both maximum ST-segment depression ($r = -0.15$, $p = 0.03$) and total ischaemic burden over the 24-h period ($r = -0.19$, $p = 0.009$).

Conclusion Cardiac troponin concentrations above the recommended threshold for the diagnosis of myocardial infarction are present in as many as one in six patients with stable coronary artery disease and reflect in part reversible silent myocardial ischaemia. These findings have major implications for the diagnosis of myocardial infarction.

142 ONSET OF PREECLAMPSIA IS PRECEDED BY STRUCTURAL CAPILLARY RAREFACTION

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Introduction Microvascular rarefaction, defined as reduced vascular density, is a consistent finding in hypertension. Functional and structural capillary rarefaction occurs in individuals with sustained and borderline essential hypertension, and in their normotensive offspring. Women who develop preeclampsia are at increased risk of hypertension in later life. We hypothesised that capillary rarefaction precedes the onset of preeclampsia and could play a role in its pathogenesis.

Methods In this longitudinal cohort study we recruited 322 Caucasian women, of which 305 subjects completed the study. We used intravital video-microscopy to measure basal (ie, functional) and maximal (ie, structural) skin capillary densities according to a well-validated protocol and measured plasma angiogenic and anti-angiogenic factors. Subjects were studied at five consecutive visits.

Results Preeclampsia occurred in 16 women (mean onset at 35.6 ± 4.8 weeks) and 272 women had normal pregnancy. In women with normal pregnancy significant structural reduction in capillary density occurred at 27–32 weeks, which had resolved by the puerperium (mean change: -2.2 capillaries/field, 95% CI -3.6 to -0.7). In contrast, in women who developed preeclampsia, more significant structural rarefaction was observed earlier at 20–24 weeks (mean change: -6.1 capillaries/field, 95% CI -9.2 to -2.9), which persisted into the puerperium. We also found that the change in soluble Endoglin from 11–16 weeks to 27–32 weeks was significantly correlated with the change in structural capillary density.

Conclusions This is the first study to show that significant structural capillary rarefaction precedes the onset of preeclampsia. Capillary rarefaction could play a role in the pathogenesis of this disease.