# Coronary Perforation Complicating Percutaneous Coronary Intervention in Patients With a History of Coronary Artery Bypass Surgery

## An Analysis of 309 Perforation Cases From the British Cardiovascular Intervention Society Database

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***Background***—The evidence base for coronary perforation (CP) occurring during percutaneous coronary intervention in patients with a history of coronary artery bypass surgery (PCI-CABG) is limited and the long-term effects unclear. Using a national PCI database, the incidence, predictors, and outcomes of CP during PCI-CABG were defined.

***Methods and Results***—Data were analyzed on all PCI-CABG procedures performed in England and Wales between 2005 and 2013. Multivariate logistic regressions and propensity scores were used to identify predictors of CP and its association with outcomes. During the study period, 309 CPs were recorded during 59 644 PCI-CABG procedures with the incidence rising from 0.32% in 2005 to 0.68% in 2013 ($P$<0.001 for trend). Independent associates of perforation in native vessels included age, chronic occlusive disease intervention, rotational atherectomy use, number of stents, hypertension, and female sex. In graft PCI, predictors of perforation were history of stroke, New York Heart Association class, and number of stents used. In-hospital clinical complications including Q-wave myocardial infarction (2.9% versus 0.2%; $P$<0.001), major bleeding (14.0% versus 0.9%; $P$<0.001), blood transfusion (3.7% versus 0.2%; $P$<0.001), and death (10.0% versus 1.1%; $P$<0.001) were more frequent in patients with CP. A continued excess mortality occurred after perforation, with an odds ratio for 12-month mortality of 1.35 for perforation survivors compared with matched nonperforation survivors without a CP ($P$<0.0001).

***Conclusions***—CP is an infrequent event during PCI-CABG but is closely associated with adverse clinical outcomes. A legacy effect of perforation on 12-month mortality was observed. **(*Circ Cardiovasc Interv*. 2017;10:e005581. DOI: 10.1161/CIRCINTERVENTIONS.117.005581.)**

**Key Words:** atherectomy ■ coronary artery bypass ■ myocardial infarction ■ percutaneous coronary intervention ■ stents

Coronary perforation (CP) is a rare but serious complication of percutaneous coronary intervention (PCI) with a previously estimated incidence of ≈0.4%.[1] Although there are several published series of CP, the rarity of the complication has previously been limited to small numbers of cases derived from single-center experience.[2–8] Most recently, we have published a national analysis of temporal trends, predictors, and outcomes of CPs in the United Kingdom and reported that previous coronary artery bypass surgery (CABG) was an independent associate of CP during PCI.[9] Given the association between CP and PCI in patients with a history of CABG surgery (PCI-CABG), a

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## WHAT IS KNOWN

- It is known that coronary perforation is a rare but devastating complication of percutaneous coronary intervention with certain groups being at greater risk.
- Patients undergoing percutaneous coronary intervention with a previous history of coronary artery bypass surgery are said to be at lower risk of tamponade, but this is very poorly defined in the literature and the overall perforation rate uncertain.

## WHAT THE STUDY ADDS

- With 309 events (and the incidence rising from 0.32% in 2005 to 0.68% in 2013), we were able to identify the main predictors of perforation during percutaneous coronary intervention in patients with a history of coronary artery bypass surgery, and these included age, chronic total occlusion intervention, use of rotational atherectomy, number of stents, hypertension, and female sex.
- Tamponade occurred with a similar frequency between native and graft vessel perforation and also with a similar frequency to the overall percutaneous coronary intervention cohort dispelling the myth that tamponade does not occur after perforation in patients with a previous coronary artery bypass surgery history.
- We identified not only a very significant impact on early morbidity and mortality of a perforation but also a longer-term effect on 12-month mortality in 30-day survivors propensity matched to 30-day nonperforation survivors.



greater understanding of CP in this patient cohort is, therefore, warranted.

In patients with prior CABG surgery, it has been assumed that CPs might be less likely to cause cardiac tamponade because of a potential protective role of pericardial adhesions and frequent removal of part of the pericardium. However, several small case series illustrate a range of complications associated with CP and demonstrate that it is not always a benign event. Rapid blood extravasation has been reported to be associated with loculated collections that may be difficult to drain and compression a variety of cardiac structures.[10] Other complications of a perforation in such patients include rupture of blood into lung tissue with hemoptysis or into the pleural space with a massive hemothorax.[11] Indeed, in 1 literature review, the 30-day mortality was as high as 22%.[12] Finally, previous publications have not stratified their analyses according to the site of perforation and whether outcomes are different in cases where a perforation occurs in the native vessel or the bypass graft.

Therefore, the primary objective of the present study was to define the incidence and temporal trends of CP associated with PCI-CABG through analysis of a national PCI database, with stratification according to whether the site of perforation was a native vessel or a bypass graft. Secondary objectives were to describe factors independently predictive of CP in this cohort and to define the impact of this complication on short- and long-term clinical outcomes.

# Methods

## Study Design, Setting, and Participants

We retrospectively analyzed prospectively gathered national data from all patients with history of CABG surgery who underwent PCI-CABG in England and Wales between January 2005 and December 2013. During the study period, 64 117 were recorded as undergoing PCI with a prior CABG history. Patients with a missing field for CP in the database (n=4473) were excluded from the analysis leaving a final study population of 59 644 patients. The study was approved by review board of the National Institute of Clinical Outcomes Research and by the Healthcare Quality Improvement Partnership.

## Setting, Data Source, and Study Size

Data on PCI practice in the United Kingdom were obtained from the British Cardiovascular Intervention Society (BCIS) data set that records this information prospectively and publishes this information in the public domain as part of the national transparency agenda.[13] The data collection process is overseen by the National Institute of Cardiovascular Outcomes Research (http://www.ucl.ac.uk/nicor/) with high levels of case ascertainment. In 2013, 98.6% of all PCI procedures performed in the National Health Service hospitals in England and Wales (www.bcis.org.uk/) were recorded on the database. The BCIS-National Institute of Cardiovascular Outcomes Research database contains 113 clinical, procedural, and outcomes variables with ≈80 000 new records added each year. The participants of the database are tracked by the Medical Research Information Services for subsequent mortality using the patients National Health Service number (a unique identifier for any person registered within the National Health Service in England and Wales). Although the BCIS data set is United Kingdom wide, only patients from England and Wales have mortality tracked by the Office of National Statistics, and so the current analysis is restricted to patients from these 2 countries.

**Table 1. Baseline Demographics of Patients With and Without Coronary Artery Bypass Surgery History**

| Variable | No. of CABG History (n=527 121) | CABG History (n=59 644) | *P* Value |
|---|---|---|---|
| Age, y±SD | 64.8±11.8 | 69.1±9.5 | <0.001 |
| Male, n (%) | 390 036 (74) | 49 231 (83) | <0.001 |
| Hypertension, n (%) | 267 540 (52) | 37 682 (68) | <0.001 |
| Diabetes mellitus, n (%) | 97 251 (19) | 18 114 (32) | <0.001 |
| Previous MI, n (%) | 139 558 (28) | 33 203 (62) | <0.001 |
| Previous stroke, n (%) | 19 318 (4) | 3511 (7) | <0.001 |
| Peripheral vascular disease, n (%) | 23 666 (5) | 5572 (10) | <0.001 |
| Valvular heart disease, n (%) | 6475 (1) | 1629 (3) | <0.001 |
| Renal disease, n (%) | 13 252 (3) | 2614 (5) | <0.001 |
| Previous PCI, n (%) | 114 311 (23) | 23 679 (41) | <0.001 |
| Anticoagulant treatment, n (%) | 5166 (1) | 1224 (2) | <0.001 |
| Ejection fraction<30%, n (%) | 15 337 (6) | 2798 (9) | <0.001 |

CABG indicates coronary artery bypass surgery; MI, myocardial infarction; and PCI, percutaneous coronary intervention.

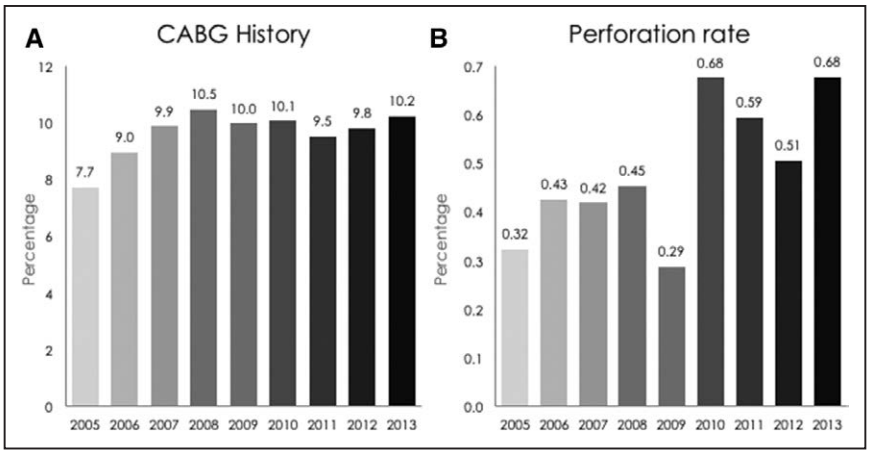


**Figure 1. A**, Annualized percentage of patients undergoing percutaneous coronary intervention in England and Wales with a history of coronary artery bypass surgery (CABG), $P<0.001$ for trend; (**B**) annualized rates of coronary perforation rates in patients with a history of CABG, $P=0.001$ for trend.

## Study Definitions

We analyzed all recorded PCI-CABG procedures that were undertaken in England and Wales between January 1, 2005, and December 31, 2013. CP was defined as evidence of extravasation of dye or blood from the coronary artery during or after the interventional procedure. This was determined either by angiographic appearances consistent with dye outside of the vessel lumen or by echocardiographic evidence of a pericardial effusion. Patients were categorized according to whether they sustained a CP during PCI-CABG or not. Study definitions were used as in the BCIS-National Institute of Cardiovascular Outcomes Research database. Specifically, preprocedural renal failure is defined as any one of the following: creatinine>200 μmol/L, renal transplant history, or dialysis. The outcomes examined were in-hospital mortality, 30-day mortality, 1-year mortality, 5-year mortality, in-hospital reinfarction, in-hospital emergency cardiac surgery, in-hospital cardiac tamponade, in-hospital stroke, and in-hospital major bleeding (defined as gastrointestinal bleed, intracerebral bleed, retroperitoneal hematoma, blood or platelet transfusion, or an arterial access site complication requiring surgery). Complex chronic total occlusion (CTO) techniques were defined as dual arterial access, rotational/laser atherectomy, penetration catheter, microcatheter, CrossBoss/Stingray balloon, and intravascular ultrasound use.

**Table 2. Baseline Participant Characteristics Variables for Patients Undergoing Percutaneous Coronary Intervention With a Prior History of Coronary Artery Bypass Surgery Tabulated by Coronary Perforation Status and Target Vessel Type**

| | Native Vessel PCI | | | Graft PCI | | | |
|---|---|---|---|---|---|---|---|
| Variable | No. of Perforation (n=40 988) | Perforation (n=210) | *P* Value | No. of Perforation (n=16 921) | Perforation (n=83) | *P* Value | *P* Value* |
| Age, y | 68.6±9.6 | 72.0±8.4 | <0.001 | 70.2±9.1 | 72.1±8.5 | 0.029 | 0.500 |
| Female, n (%) | 7472 (18.3) | 52 (24.8) | 0.020 | 2454 (14.6) | 17 (20.5) | 0.172 | 0.449 |
| Smoking history, n (%) | 22 106 (62.4) | 116 (61.7) | 0.903 | 9197 (63.4) | 49 (65.3) | 0.920 | 0.602 |
| BMI, kg/m$^2$ | 28.7±4.9 | 28.0±3.9 | 0.076 | 28.3±4.7 | 27.4±3.9 | 0.102 | 0.271 |
| Hypertension, n (%) | 25 660 (67.2) | 146 (73.7) | 0.059 | 10 603 (68.2) | 53 (67.9) | 0.966 | 0.405 |
| Diabetes mellitus, n (%) | 12 223 (31.5) | 51 (25.4) | 0.072 | 5224 (33.1) | 28 (34.1) | 0.929 | 0.110 |
| Previous MI, n (%) | 21 856 (58.9) | 133 (66.8) | 0.028 | 9977 (66.3) | 53 (68.8) | 0.725 | 0.995 |
| Previous stroke, n (%) | 2233 (5.8) | 13 (6.6) | 0.781 | 1079 (6.9) | 14 (17.9) | <0.001 | 0.016 |
| PVD, n (%) | 3537 (9.3) | 19 (9.6) | 0.968 | 1782 (11.5) | 11 (14.1) | 0.580 | 0.290 |
| Valve disease, n (%) | 1180 (3.1) | 3 (1.5) | 0.284 | 409 (2.6) | 4 (5.1) | 0.309 | 0.103 |
| Renal disease, n (%) | 1731 (4.6) | 11 (5.5) | 0.662 | 795 (5.2) | 4 (5.1) | 0.955 | 1.000 |
| Stable indication, n (%) | 24 707 (60.3) | 146 (69.5) | 0.008 | 7184 (42.5) | 25 (30.1) | 0.031 | <0.001 |
| Shock, n (%) | 515 (1.4) | 5 (2.8) | 0.207 | 231 (1.5) | 2 (2.8) | 0.693 | 1.000 |
| Q-wave on ECG, n (%) | 5901 (18.3) | 31 (18.5) | 0.958 | 2767 (22.3) | 15 (22.4) | 0.435 | 0.481 |
| Previous PCI, n (%) | 16 120 (40.0) | 82 (39.6) | 0.870 | 6896 (42.4) | 32 (40.0) | 0.775 | 1.000 |
| Anticoagulation, n (%) | 870 (2.3) | 7 (3.5) | 0.375 | 313 (2.1) | 2 (2.5) | 0.795 | 1.000 |
| CCS class | 2.48±0.95 | 2.67±0.80 | 0.003 | 2.61±0.84 | 2.78±0.83 | 0.105 | 0.311 |
| NYHA class | 2.04±0.86 | 2.12±0.92 | 0.124 | 2.08±0.82 | 2.32±0.86 | 0.043 | 0.190 |

BMI indicates body mass index; CCS, Canadian Cardiovascular Society; MI, myocardial infarction; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; and PVD, peripheral vascular disease.

*Comparison between perforation in native vessel and graft.

## Data Analyses

We examined the baseline and procedural characteristics of participants by CP status and native vessel or graft PCI. We tested for associations between each categorical variable and CP using a $\chi^2$ test, and for continuous variables, we used 1-way ANOVA. We then performed separate multivariate analyses of the predictors of perforation by vessel type using multivariate logistic regression to investigate the influence of variables that have the potential for being included in the linear component of a proportional hazard model. We selected a final model for each outcome by using forward stepwise variable selection and an inclusion criterion of $P<0.1$. Variables included in these analyses were age, sex, body mass index, Canadian Cardiovascular Society angina class, New York Heart Association class, previous myocardial infarction (MI), history of hypertension, history of stroke, glycoprotein inhibitor use, dual arterial access, use of rotational atherectomy, microcatheter use, CTO presence, CTO attempted, right coronary intervention, and trainee operator. Final model selection was done as follows: we first imputed missing data on baseline covariate using multiple imputations with chained equation (missing data points are presented in Table I in the Data Supplement). To examine the associates of perforation and test if those associates show a trend over time (from 2006 to 2013), a Cochran–Armitage test for trend was used. Individual logistic regressions were done on the imputed data set for each of the major adverse cardiovascular events according to the perforation status of the patients to quantify the independent association between perforation and outcomes.

Finally, from the subset of patients who survived up to 30 days, we explored the association between perforation at procedure and mortality at 12 months a time to event analysis was performed using Kaplan–Meier curves, log-rank tests, and Cox proportional hazard model to estimate the corresponding hazard ratio. To adjust for baseline imbalances, we performed a propensity score analysis to balance for important covariates that might bias estimates for causal inferences. The following variables that were associated with 12-month mortality in the 30-day survivors were used in the propensity score analysis: sex, age at procedure, New York Heart Association status, body mass index previous MI, previous CABG, diabetes mellitus, baseline disease severity, hypertension, smoking status, renal insufficiency, and Q-wave on ECG. The propensity scores for each patient were derived using the inverse probability of treatment weight. More precisely, one estimates the probability that a particular patient is assigned to 1 of the 2 groups as a function of that individuals covariates (the propensity score). Each individual observation was then given a weight equal to the inverse of this propensity score to create 2 pseudopopulations of exposed and unexposed patients who now represent what would have happened to the entire population under those 2 treatment conditions. The advantage of this method is that it is inclusive as it uses all patients in a study; therefore, no loss of sample occurs as in other conditioning methods such as matching or stratification. We also normalized the weights by dividing them by the mean weight. Those weights were then used to derive weighted Kaplan–Meier curves and weighted hazard ratios.

# Results

## Incidence and Baseline Demographics During PCI-CABG by Perforation Status and Vessel Type

Patients with a history of CABG had significantly greater comorbidity than patients without previous CABG (Table 1). The proportion of patients undergoing PCI with a history of CABG surgery increased progressively during the study period from 7.7% in 2005 to 10.2% in 2013 (Figure 1A; $P<0.001$ for trend). A total of 309 CPs were recorded during 59 644 PCI-CABG procedures (overall incidence 0.52%). There was no significant difference in perforation incidence between native vessel PCI and graft PCI (0.51% versus 0.49%, respectively; $P=0.787$). Additionally, the annualized rate of CP rose progressively from 0.32% in 2005 to 0.68% in 2013 (Figure 1B; $P=0.001$ for trend). Baseline characteristics of patients with and without perforation categorized by native vessel or graft PCI are presented in Table 2. Although CP was significantly more likely in stable angina PCI to native vessels (69.5% versus 60.3%; $P=0.008$), it was less likely in graft PCI (30.1% versus 42.5%; $P=0.031$). The annualized

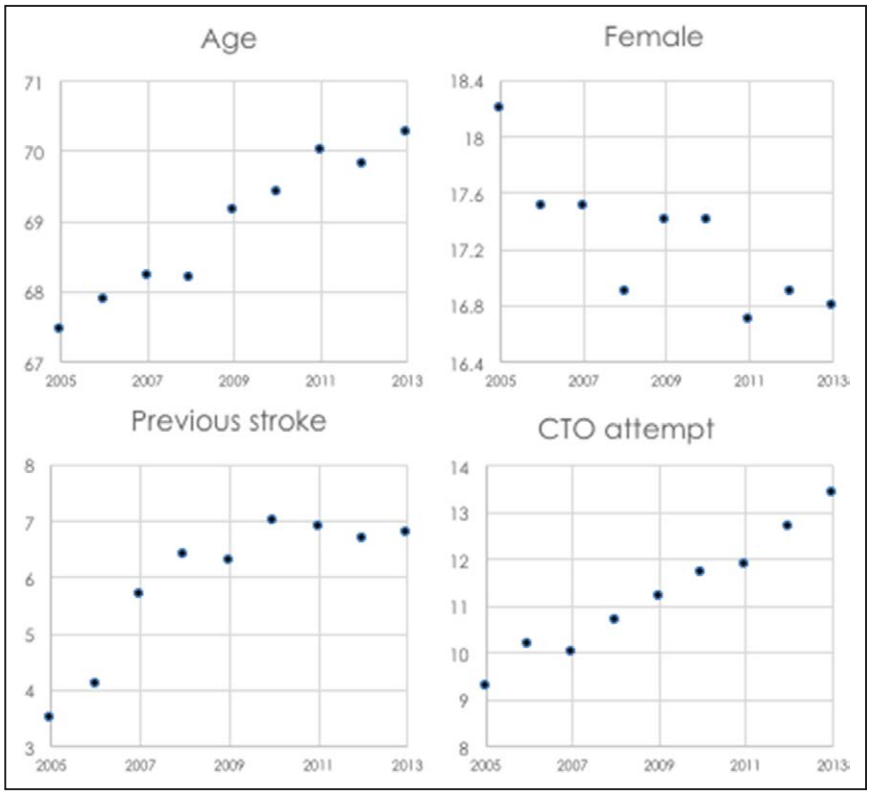


**Figure 2.** Baseline and procedural characteristics associated with perforation in patients undergoing percutaneous coronary intervention with a prior coronary artery bypass surgery plotted over time (2005–2013); $P<0.001$ for all trends. CTO indicates chronic total occlusion.

patient demographics associated with CP are displayed in Figure 2 and illustrates an increase in case complexity during the study period.

## Procedural Variables During PCI-CABG by Perforation Status and Vessel Type

The procedural variables for patients with and without CP by vessel type are presented in Table 3. Baseline disease complexity was associated with CP in native vessels with a nontrainee primary operator, dual arterial access, CTO intervention rotational atherectomy, and microcatheter use all more frequently observed when perforation occurred. In graft intervention, the only use of rotational atherectomy and number of stents were associated with an increased risk of perforation. Potent dual antiplatelet therapy and glycoprotein inhibitor use were not associated with an increased risk of CP. Perforation was significantly more likely to occur as complex PCI strategies (dual access, rotational or laser atherectomy, and microcatheters) were used individually (Figure 3, left and center) or in combination with a significant trend between 0 and 3 or more strategies (Figure 3, right).

## Predictors of CP During PCI-CABG

Using multivariate analyses, covariates found to be associated with native vessel CP are presented by vessel in Table 4. The patient-related factors associated with an increased risk of perforation were age per year (odds ratio [OR], 1.04; 95% confidence intervals [CIs], 1.02–1.06; $P<0.001$), hypertension history (OR, 2.31; 95% CI, 1.40–3.80; $P=0.001$), and female sex (OR, 1.63; 95% CI, 1.07–2.50; $P=0.024$). Several procedural factors were strongly associated with an increased

**Table 3. Procedural Variables by Coronary Perforation Status and Target Vessel Type**

| Variable | Native Vessel PCI | | | Graft PCI | | | |
|---|---|---|---|---|---|---|---|
| | No. of Perforation (n=40 988) | Perforation (n=210) | *P* Value | No. of Perforation (n=16 921) | Perforation (n=83) | *P* Value | *P* Value* |
| Femoral access, n (%) | 27 790 (70.0) | 139 (67.8) | 0.552 | 12 462 (76.4) | 61 (74.4) | 0.767 | 0.266 |
| Dual access, n (%) | 1718 (4.3) | 28 (13.7) | <0.001 | 418 (2.6) | 4 (4.9) | 0.331 | 0.038 |
| No. of grafts present±SD | 2.54±0.94 | 2.66±0.82 | 0.063 | 2.94±0.92 | 3.01±0.77 | 0.329 | 0.033 |
| No. of grafts patent±SD | 1.52±0.92 | 1.52±1.04 | 0.497 | 2.01±1.05 | 2.03±1.0 | 0.442 | 0.002 |
| CTO present, n (%) | 18 655 (53.6) | 141 (75.0) | <0.001 | 8881 (72.2) | 49 (68.1) | 0.594 | 0.222 |
| No. of CTO present±SD | 0.82±0.90 | 1.21±0.96 | <0.001 | 1.44±1.15 | 1.24±1.09 | 0.063 | 0.081 |
| Trainee operator, n (%) | 8865 (23.8) | 28 (14.1) | 0.002 | 3841 (25.5) | 18 (22.8) | 0.668 | 0.108 |
| CTO attempted, n (%) | 5080 (13.4) | 83 (40.5) | <0.001 | 808 (5.9) | 6 (7.8) | 0.492 | <0.001 |
| Vessel attempted, n (%) | | | | | | | |
| Graft | 0 (0) | 0 (0) | … | 16 921 (100) | 83 (100) | … | … |
| Left main | 7455 (19.2) | 42 (20.7) | 0.654 | 0 (0) | 0 (0) | … | … |
| LAD | 10 110 (26.0) | 49 (24.1) | 0.593 | 0 (0) | 0 (0) | … | … |
| Circumflex | 14 803 (38.1) | 65 (32.1) | 0.087 | 0 (0) | 0 (0) | … | … |
| Right coronary | 13 124 (33.8) | 91 (44.8) | 0.001 | 0 (0) | 0 (0) | … | … |
| Rotablation, n (%) | 1595 (4.3) | 24 (12.1) | <0.001 | 46 (0.3) | 2 (2.5) | 0.014 | 0.012 |
| Laser atherectomy, n (%) | 165 (0.4) | 2 (1.0) | 0.516 | 175 (1.2) | 2 (2.5) | 0.564 | 0.319 |
| Penetration catheter, n (%) | 327 (0.8) | 2 (1.0) | 0.851 | 47 (0.3) | 0 (0) | 0.615 | 1.000 |
| Microcatheter, n (%) | 666 (1.8) | 16 (8.0) | <0.001 | 56 (3.8) | 0 (0) | 0.583 | 0.008 |
| Thrombectomy, n (%) | 1269 (3.4) | 8 (4.1) | 0.740 | 1458 (9.8) | 12 (14.2) | 0.184 | 0.003 |
| Distal protection, n (%) | 392 (1.0) | 1 (0.5) | 0.681 | 2843 (18.9) | 15 (18.8) | 0.967 | <0.001 |
| Cutting balloon use, n (%) | 1575 (4.2) | 4 (2.2) | 0.167 | 510 (3.4) | 1 (1.3) | 0.448 | 0.495 |
| Bivalirudin use, n (%) | 507 (1.3) | 1 (0.5) | 0.464 | 281 (1.9) | 1 (1.3) | 0.683 | 0.629 |
| GPI use, n (%) | 5317 (14.2) | 15 (7.7) | 0.013 | 3641 (24.3) | 16 (21.6) | 0.692 | 0.005 |
| No. of stents used±SD | 1.49±1.14 | 1.88±1.80 | <0.001 | 1.34±0.98 | 2.15±1.49 | <0.001 | 0.158 |
| No. of successful lesions±SD | 1.29±0.81 | 1.07±0.99 | <0.001 | 1.13±0.64 | 1.28±10.3 | 0.017 | 0.231 |
| Largest stent/balloon±SD | 3.27±0.69 | 3.36±0.83 | 0.055 | 3.60±0.78 | 3.76±0.86 | 0.046 | 0.001 |
| Longest stent used±SD | 25.5±16.8 | 30.7±26.1 | <0.001 | 24.1±14.9 | 31.1±18.2 | <0.001 | 0.384 |

CTO indicates chronic total occlusion; GPI, glycoprotein inhibitor; LAD, let anterior descending; and PCI, percutaneous coronary intervention.
*Comparison between perforation in native vessel and graft.

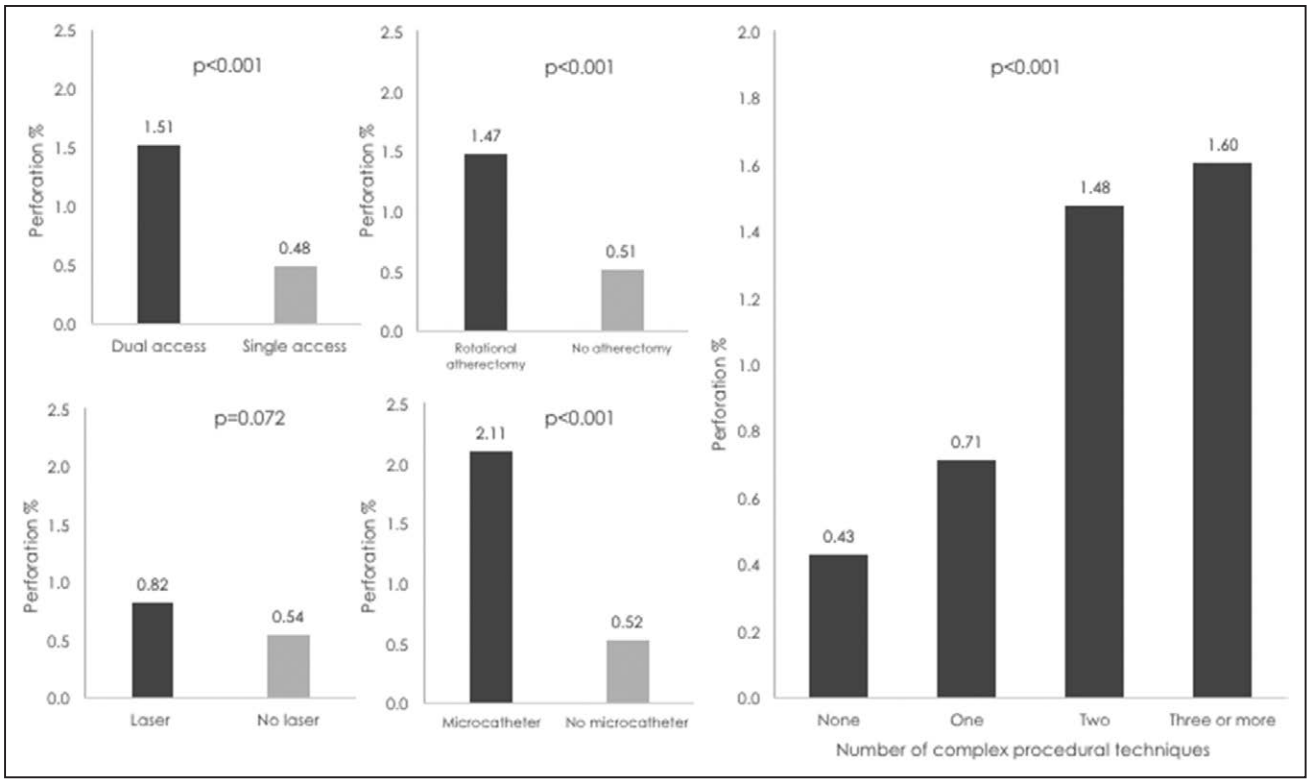


**Figure 3. Left** and **center**, Incidence of coronary perforation indexed for complex strategy (dual vs single arterial access, use or not of rotational atherectomy, use or not of laser atherectomy, and use or not of a microcatheter), *P* value for comparison within each strategy. **Right**, Incidence of coronary perforation with number of complex percutaneous coronary intervention strategies used (dual access, microcatheter, laser or rotational atherectomy, penetration catheter, intravascular ultrasound, or CrossBoss), *P* value for trend.

risk of native vessel perforation including CTO intervention (OR, 3.48; 95% CI, 2.30–5.27; *P*<0.001) and rotational atherectomy use (OR, 2.25; 95% CI, 1.29–3.93; *P*=0.004). In contrast, graft perforation appeared to be more unpredictable with only history of stroke (OR, 1.56; 95% CI, 1.17–2.10; *P*=0.003), New York Heart Association class (OR, 2.14; 95% CI, 1.18–3.92; *P*=0.014), and number of stents (OR, 1.49; 95% CI, 1.01–1.36; *P*<0.001) associated with an increased risk (Table 4).

## Clinical Outcomes by Perforation Status

All immediate procedural complications including cardiogenic shock induction, heart block, coronary dissection, and major side-branch occlusion were more likely when a CP occurred (Table 5) and were similar between native vessel and graft PCI (Table II in the Data Supplement). Tamponade leading to hemodynamic compromise occurred in 10.0% of patients (9.8% for native vessel PCI and 12.5% for graft PCI; *P*=0.576) although emergency reparative cardiac surgery was undertaken rarely (1.5%). The occurrence of tamponade after a perforation seems unpredictable with only femoral artery access (OR, 4.117; 95% CI, 1.062–15.957; *P*=0.041) and consultant operator (OR, 4.27; 95% CI, 1.17–15.6; *P*=0.028) associated with a higher risk.

Overall, coronary complications occurred in 14.2% of PCI-CABG procedures with a perforation and 3.6% without (*P*<0.001). In-hospital clinical complications including Q-wave MI (2.9% versus 0.2%; *P*<0.001), major bleeding (14.0% versus 0.9%; *P*<0.001), blood transfusion (3.7% versus 0.2%; *P*<0.001), renal failure (1.1% versus 0.1%; *P*<0.001), and death (10.0% versus 1.1%; *P*<0.001) were more frequent in patients with CP.

Mortality at all time points was greater in those patients with CP (Table 5) and was similar between native vessel

**Table 4. Multivariate Model of the Significant Associations Between Covariates and Coronary Perforation**

| Variable | Adjusted Odds Ratio (95% Confidence Interval) | *P* Value |
|---|---|---|
| Native Vessel PCI | | |
| Age per year | 1.04 (1.02–1.06) | <0.001 |
| Chronic total occlusion attempted | 3.48 (2.30–5.27) | <0.001 |
| No. of stents used | 1.31 (1.15–1.48) | <0.001 |
| Hypertension | 2.31 (1.40–3.80) | 0.001 |
| Rotational atherectomy use | 2.25 (1.29–3.93) | 0.004 |
| Female sex | 1.63 (1.07–2.50) | 0.024 |
| Diabetes mellitus | 0.55 (0.35–0.86) | 0.008 |
| Graft PCI | | |
| No. of stents used | 1.49 (1.01–1.36) | <0.001 |
| History of stroke | 1.56 (1.17–2.10) | 0.003 |
| NYHA class | 2.14 (1.18–3.92) | 0.014 |

NYHA indicates New York Heart Association; and PCI, percutaneous coronary intervention.

**Table 5. Outcomes by Coronary Perforation Status**

| Variable | No. of Perforation (n=59 335) | Perforation (n=309) | *P* Value |
|---|---|---|---|
| Immediate procedural complications | | | |
| Tamponade, n (%) | 0 (0) | 27 (10.0) | <0.001 |
| Shock induced by procedure, n (%) | 89 (0.2) | 11 (3.6) | <0.001 |
| Heart block, n (%) | 103 (0.2) | 6 (1.9) | <0.001 |
| Emergency cardiac surgery, n (%) | 63 (0.1) | 4 (1.5) | <0.001 |
| Coronary dissection, n (%) | 1076 (2.0) | 35 (11.3) | <0.001 |
| Major side-branch loss, n (%) | 269 (0.5) | 10 (3.2) | <0.001 |
| Slow flow, n (%) | 664 (1.2) | 10 (3.2) | 0.004 |
| Access site complication, n (%) | 418 (0.7) | 6 (2.0) | 0.017 |
| Clinical outcomes | | | |
| Transfusion, n (%) | 101 (0.2) | 10 (3.7) | <0.001 |
| Q-wave MI, n (%) | 96 (0.2) | 8 (2.9) | <0.001 |
| Non Q-wave MI, n (%) | 246 (0.4) | 15 (5.6) | <0.001 |
| Renal failure, n (%) | 67 (0.1) | 3 (1.1) | <0.001 |
| In-hospital major bleed, n (%) | 513 (0.9) | 38 (14.0) | <0.001 |
| In-hospital death, n (%) | 644 (1.1) | 30 (10.0) | <0.001 |
| Mortality at 30 d, n (%) | 855 (1.7) | 28 (10.8) | <0.001 |
| Mortality at 1 y, n (%) | 2936 (6.3) | 41 (17.7) | <0.001 |

MI indicates myocardial infarction.

and graft PCI (Table II in the Data Supplement). The 30-day mortality in patients experiencing a CP during graft PCI was similar to that observed in patients experiencing a perforation during native vessel PCI (10.8% versus 10.7%; *P*=0.926). Temporal changes in mortality are displayed in Figure 4 and

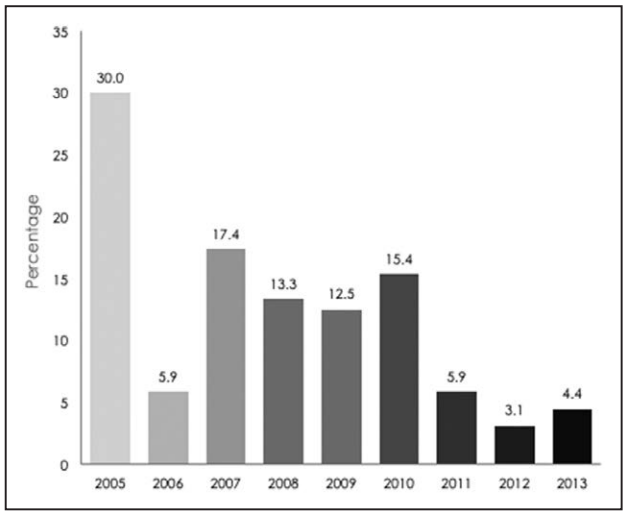


**Figure 4.** Annualized mortality for patients with coronary perforation during percutaneous coronary intervention in patients with a history of coronary artery bypass surgery 2005–2013 (*P*=0.005 for trend).

**Table 6. Univariate Odds of Adverse Outcomes by Coronary Perforation Status**

| Adverse Outcome | Odds Ratio | 95% Confidence Interval | *P* Value |
|---|---|---|---|
| Emergency cardiac surgery | 13.43 | 4.85–37.15 | <0.001 |
| Access site complication | 2.95 | 1.31–6.65 | <0.001 |
| Transfusion | 21.40 | 11.05–41.46 | <0.001 |
| Q-wave MI | 17.88 | 8.60–37.15 | <0.001 |
| Non Q-wave MI | 13.41 | 7.85–22.90 | <0.001 |
| Renal failure | 9.44 | 2.95–30.18 | <0.001 |
| In-hospital major bleed | 17.73 | 12.45–25.26 | <0.001 |
| In-hospital death | 9.78 | 6.67–14.37 | <0.001 |
| Death 30 d | 7.13 | 4.79–10.61 | <0.001 |
| Death 1 y | 3.18 | 2.26–4.46 | <0.001 |

MI indicates myocardial infarction.

demonstrate a significant reduction during the study period (*P*=0.005). Using multiple logistic regression analyses, the adjusted odds of clinical outcomes are presented in Table 6 and indicate a significant impact of CP during PCI-CABG on all short- and long-term outcomes.

An excess of 12-month mortality in 30-day survivors for patients with and without perforation was associated with increased age and baseline comorbidity (Table III in the Data Supplement). Figure 5A illustrates the Kaplan–Meier plots for mortality by perforation status to 12 months confirming the significant impact of a perforation on patient survival. Figure 5B illustrates Kaplan–Meier plots for mortality using a landmark analysis for perforation survivors from 30 days to 12 months compared with propensity matched nonperforation survivors. Using propensity scores for each patient derived from the inverse probability of treatment weight, a legacy effect (ie, a continued impact of perforation on mortality) was evident with a hazards ratio for 12-month mortality of 1.35 compared with those matched patients without a CP (*P*<0.001).

## Discussion

This analysis revealed an overall incidence of CP in patients undergoing PCI with prior CABG surgery of 0.52%, a rate higher than observed in a more general PCI population.[9] First, this likely reflects the complexity of the case mix with advanced patient age and high rates of CTO intervention in patients with a history of CABG. Second, the presence of perforation in such patients was associated with higher morbidity and mortality, irrespective of whether the perforation occurred in a native vessel or a bypass graft. Third, the annual incidence of perforation increased significantly during the study period with perforation being associated with increasing age, history of hypertension, and previous MI or stroke.

Procedure complexity (likely driven by coronary disease complexity) appeared to predict perforation in native vessels with rotational atherectomy use and CTO intervention significantly more likely to be present when a perforation

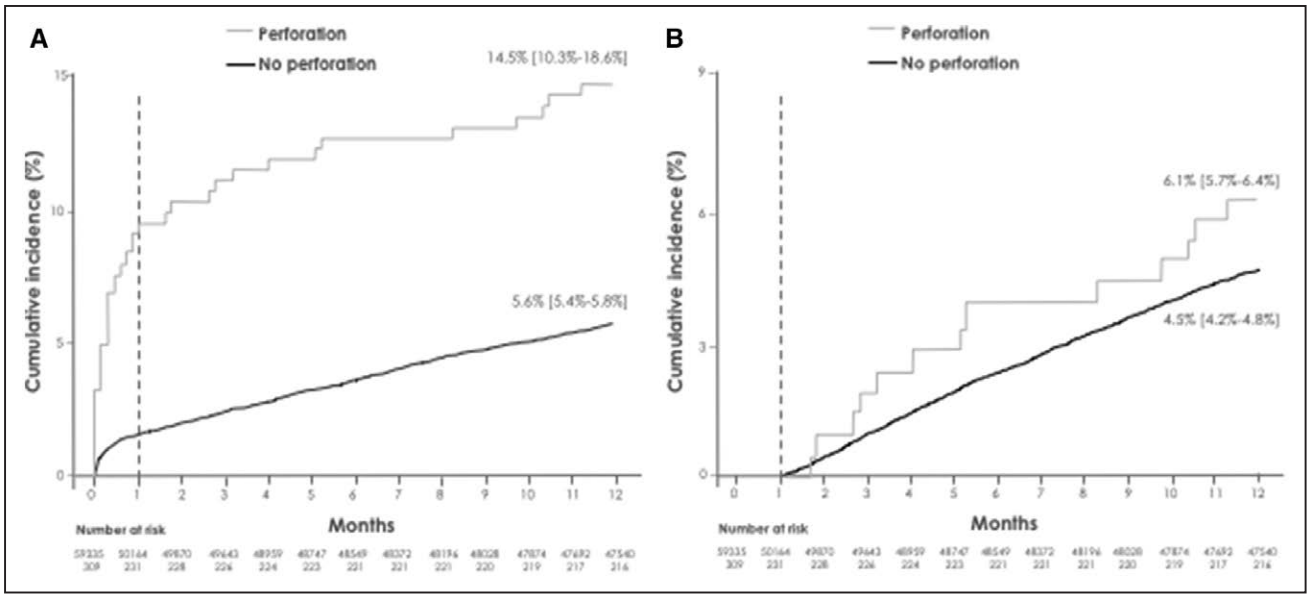


**Figure 5. A**, Unadjusted Kaplan–Meier plots for mortality by perforation status to 12 months; (**B**) inverse probability of treatment weight adjusted Kaplan–Meier plots for mortality by perforation status for perforation survivors from 30 days to 12 months compared with non-perforation survivors from 30 days to 12 months.

occurred. In contrast, perforation in graft vessels appeared unpredictable and less closely associated with procedural complexity, a finding which is important in itself. The association between cerebrovascular accident and perforation in vein grafts is a novel finding, and it is conceivable that a previous stroke is a predictor of more advanced vascular disease and calcification, and this might lead to a greater chance of perforation during PCI. However, these associations are a novel finding and are thus hypothesis generating and worthy of further study.

These data dispel the myth that perforation in patients with previous CABG is a benign event because of the presence of pericardial adhesions and the absence of a closed pericardial sac. However, in the current study, although tamponade was lower than reported in a previous generalized PCI population (16.1%), it still occurred in >10% of the population without a significant difference between graft and native vessel PCI.[9] There are several possible explanations for this observation. First, although the pericardium is often not repaired after CABG, this is not a universal approach. Particularly in younger patients, the pericardium may be closed to facilitate easier future redo surgery if required.[14] Also, a closed pericardium has been reported to paradoxically reduce postoperative tamponade after CABG surgery by protecting the heart from extrapericardial bleeding meaning that some surgeons choose to repair the pericardium.[15,16] There are also several case reports of localized effusions compressing a variety of cardiac structures. Loculated collections of blood after CP have been reported to compress the atria, the right ventricle, or the pulmonary artery with rapid hemodynamic collapse.[10,17] In practice, although pericardiocentesis of a global effusion in a closed pericardial sac is far from straightforward, percutaneous drainage of a loculated effusion is extremely challenging and high risk. Therefore, the management of pericardial tamponade in post-CABG surgery patient is difficult and associated with poor outcomes. Additionally, unrestrained major hemorrhage into other body cavities such as the pleural cavity can occur with catastrophic outcomes.[11,18] Finally, the observation that tamponade was an unpredictable event is relevant and reiterates the importance of early and proactive management of patients who experience a perforation.

All clinical end points were significantly more likely to occur in patients experiencing a CP. There was a 10-fold increase in in-hospital mortality and a 7-fold increase in 30-day mortality. For both end points, the magnitude of the effect of a perforation was greater than observed in a previous cohort of all patients (with or without CABG history) with a perforation.[9] This may reflect the high frequency of comorbidity observed in patients with a history of CABG, which includes advanced age, high rates diabetes mellitus, and vascular and renal disease. However, despite increases in patient and procedural complexity, it is encouraging to observe that mortality declined steadily during the study period. The avoidance of surgical repair is highly desirable, and the utilization of covered stents, as well as, a wider appreciation of techniques such as ping-pong guides, embolization coils, distal fat, or thrombus embolization to treat wire tip perforations, has facilitated improved acute management of perforations and improved survival.[19–25] However, despite this, the incidence of major adverse events in patients with CP was high and these data are a reminder that while CP during PCI is a relatively rare event, its occurrence is associated with poor outcomes.

A continued excess mortality occurred in patients with a perforation after 30 days in patients matched with patients without a perforation. There are several plausible reasons that might explain this observation. First, the number of successful lesions was significantly less in cases complicated by perforation. Residual disease burden has been closely

correlated with adverse outcomes after PCI, and thus 1 explanation for the excess mortality after perforation may be the presence of ongoing ischemia because of untreated coronary stenosis.[26,27] Other mechanistic possibilities for the observed effect include side-branch loss with periprocedural MI,[28] access site complications,[29] major bleeding,[30,31] and transfusion,[32] all of which were significantly more common with perforation and individually each are associated with excess 12-month mortality. Finally, covered stent use may also be associated with poor 12-month outcomes. Small series have reported high rates of stent thrombosis, restenosis, and mortality with their use.[33]

## Limitations

The BCIS database does not differentiate between CPs resulting from guidewire and perforations because of balloon or stent inflation. Additionally, the database does not record the Ellis classification of CP. Therefore, a substratification by perforation severity was not possible. In addition, the BCIS database does not record use of other treatment strategies such covered stents, pericardial drains or embolization techniques. Although operator-level data are now available in the central cardiac audit database, it has only been available for the most recent 2 years. Therefore, it was not included in the analysis. However, it seems unlikely that there is a center-specific effect that is independent of other procedural characteristics. Finally, although we have attempted to adjust for differences in baseline covariates/clinical characteristics, there remains the possibility of residual confounding, which might in part contribute to the findings of the study.

## Conclusions

CP is an infrequent event during PCI in patients with prior CABG but has a significant impact on morbidity and mortality. A legacy effect of perforation on 12-month mortality was observed.

## Disclosures

None.

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# Coronary Perforation Complicating Percutaneous Coronary Intervention in Patients With a History of Coronary Artery Bypass Surgery: An Analysis of 309 Perforation Cases From the British Cardiovascular Intervention Society Database

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