Is direct stenting superior to stenting with predilation in patients treated with percutaneous coronary intervention? results from a meta-analysis of 24 randomised controlled trials

Federico Piscione, Raffaele Piccolo, Salvatore Cassese, et al.

Heart 2010 96: 588-594
doi: 10.1136/hrt.2009.183277

Updated information and services can be found at:
http://heart.bmj.com/content/96/8/588.full.html

These include:

References
This article cites 53 articles, 19 of which can be accessed free at:
http://heart.bmj.com/content/96/8/588.full.html#ref-list-1

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Topic collections
Articles on similar topics can be found in the following collections

- Drugs: cardiovascular system (22640 articles)
- Interventional cardiology (6855 articles)
- Acute coronary syndromes (1350 articles)
- Percutaneous intervention (386 articles)
- Epidemiology (4771 articles)

Notes

To order reprints of this article go to:
http://heart.bmj.com/cgi/reprintform

To subscribe to Heart go to:
http://heart.bmj.com/subscriptions
Is direct stenting superior to stenting with predilation in patients treated with percutaneous coronary intervention? results from a meta-analysis of 24 randomised controlled trials

Federico Piscione, Raffaele Piccolo, Salvatore Cassese, Gennaro Galasso, Claudia D’Andrea, Roberta De Rosa, Massimo Chiariello

ABSTRACT

Background In the last decade, direct stenting has been proposed as an alternative strategy to conventional stenting with balloon predilation. The aim of this study was to perform a meta-analysis of randomised trials comparing a direct stenting strategy versus a conventional one.

Methods A literature search was performed using Medline, EMBASE, the Cochrane Central Register of Controlled Trials, scientific session abstracts and relevant websites, from inception of each database to June 2009. Included studies comprised randomised controlled trials evaluating direct versus conventional stenting in patients undergoing percutaneous coronary intervention. Primary endpoint was the composite of death or myocardial infarction and secondary endpoints were myocardial infarction and target-vessel revascularisation occurrence.

Results 24 trials met inclusion criteria, with 6803 patients enrolled (3412 or 50.15% randomised to direct stenting and 3391 or 49.85% randomised to conventional stenting). Up to 6-month follow-up, the composite of death or myocardial infarction was significantly reduced with direct stenting compared with conventional stenting (3.95% versus 5.10% respectively, OR = 0.76 (95% CI 0.60 to 0.96), p = 0.02). This reduction was primarily driven by a lower myocardial infarction occurrence (3.16% versus 4.04%, respectively, OR = 0.77 (0.59 to 0.99), p = 0.04). Furthermore, direct stenting was not associated with a reduction in target-vessel revascularisation (6.50% versus 6.96%, respectively, OR = 0.92 (0.76 to 1.12), p = 0.42).

Conclusion This meta-analysis demonstrates that, in selected coronary lesions, direct stenting improves outcome in patients undergoing percutaneous coronary intervention, primarily reducing myocardial infarction incidence.

INTRODUCTION

In everyday clinical practice, stent implantation occurs in about 95% of patients with coronary artery disease undergoing percutaneous coronary intervention (PCI).1 In the last decade, direct stenting (DS), usually referred to stent deployment without predilation of the target lesion, has become a feasible and safe technique, thanks to dramatic improvements in stent and deliver system design (eg, lower crossing profiles, greater flexibility, trackability and pushability).2 Initially, experimental data supported the concept of a reduced vessel wall damage with DS compared to conventional (with predilation) stenting (CS).3 Afterwards, several clinical studies evaluated DS and CS in patients treated with PCI, reporting conflicting results. Thus, the issue of whether DS can improve clinical outcomes still remains unsolved. Therefore, we performed a meta-analysis of randomised trials to assess the clinical impact of a DS strategy compared with CS in patients undergoing PCI.

METHODS

Search strategy and selection criteria

We searched Medline, EMBASE, the Cochrane Central Register of Controlled Trials (CENTRAL), scientific session abstracts in Circulation, Journal of the American College of Cardiology, European Heart Journal and the American Journal of Cardiology, and relevant websites (www.acc.org, www.americanheart.org, www.europcr.com, www.escardio.org, www.cardiosource.com/clinicaltrials, www.clinicaltrialresults.org, www.tctmd.com and www.theheart.org) for studies in any language (from inception of each database until June 2009). The reference list of relevant studies was additionally scanned. The key words used were: ‘randomised trial’, ‘direct stenting’, ‘predilation’, ‘percutaneous coronary intervention’, ‘angioplasty’, ‘sten’, ‘coronary artery disease’. To be included, the citation had to meet the following criteria: (1) random treatment allocation; (2) availability of complete clinical features. Exclusion criteria were: (1) ongoing studies or irretrievable data, and (2) >10% of patients lost to follow-up.

Data collection and quality assessment

Two investigators (SC and RP) independently assessed reports for eligibility at title and/or at abstract level, with divergences resolved with a third reviewer (FP), and studies that met the inclusion criteria were selected for further analysis. Two investigators (SC and RP) took care of data extraction. Studies were evaluated with respect to the following methodological items: randomisation, adequacy of allocation concealment, performance of the analysis according to the intention-to-treat principle, sample size calculation and specification of loss of patients.

Outcome variables

The primary endpoint was the composite of death or myocardial infarction. Patients with both events
were considered in the same way as those with a single event. Secondary endpoints were myocardial infarction (MI) and target-vessel revascularisation (TVR). If TVR was not available, target-lesion revascularisation was considered instead. All clinical endpoints were evaluated according to per protocol definitions, up to 6-month follow-up.

Statistical analysis

The κ statistic was used to assess agreement between reviewers for study selection, as previously reported. We used the Peto fixed effects model to calculate the pooled ORs and 95% CIs, since this is the most appropriate model when relatively few events occur in individual trials and when there are roughly equal numbers per treatment group. However, to test the robustness of significant results, we performed a secondary analysis by using a fixed-effect model with the Mantel-Haenszel method or DerSimonian and Laird random-effect model in case of significant heterogeneity across studies. The Breslow-Day chi² test was calculated to test the statistical evidence of heterogeneity across the studies. In addition, we used the I² statistic, which describes the percentage variation across studies that is due to heterogeneity rather than chance. A funnel plot and the adjusted rank correlation test, according to the method of Begg and Mazumdar, were used to assess publication bias with respect to each endpoint. Moreover, we performed a sensitivity analysis, in which the meta-analysis estimates are computed omitting one study at time. Finally, we performed a meta-regression in order to evaluate the influence of crossover rates of each trial on the primary endpoint. In all the studies, crossover was decided whenever the lesion could not be crossed by DS, the device was withdrawn and predilation was performed before re-attempting stent deployment. Briefly, we undertook a weighted least-square regression, using a linear regression model, with weighting provided by the number of patients included in each trial; R² and β-coefficients with 95% CI were reported. Crossover rates were considered as an independent variable and the natural logarithm of the primary endpoint as a dependent variable. Statistical analyses were performed with Review Manager 5.0.16 (RevMan, The Nordic Cochrane Centre, The Cochrane Collaboration, 2008), Stata 10.0 statistical software and SPSS 16.0 statistical package.

The study was performed in compliance with the Preferred Reporting Items for Systematic reviews and Meta-analyses (PRISMA) guidelines.

RESULTS

Eligible studies

As showed in figure 1, we screened the title or the abstract of 627 potentially eligible publications and identified initially 27 randomised trials. Two trials were excluded because of prospective single-arm study design. One trial was excluded since investigators randomised to DS or CS coronary lesions and not to patients. Finally, a total of 24 trials were included in the meta-analysis, enrolling 6803 patients (3412 or 50.15% randomised to DS and 3391 or 49.85% randomised to CS). The inter-observer agreement for study selection was very good, with

Figure 1  Flow diagram of trial selection.

627 potentially relevant citations identified and screened for retrieval

600 were excluded as not relevant or duplicated

27 randomized trials identified for more detailed evaluation

2 trials excluded because of prospective single arm study design (39-40)

25 randomized trials selected for further detailed evaluation

1 trial excluded investigators randomized coronary lesions (not patients) (41)

24 randomized trials included in the meta-analysis enrolling 6,803 patients (15-38) 
(3,412 or 50.15% randomized to DS and 3,391 or 49.85% randomized to CS)
κ = 0.97. Table 1 summarises the main characteristics of included studies. All implanted stents were bare-metal stents, with the exception of those in 34 patients in the study by Cuisset et al.\textsuperscript{20} Crossover rates ranged from 0% to 29.7%, probably owing to different inclusion criteria and patient selection. As reported in table 1, routine angiographic follow-up was planned in 11 trials.

### Table 1 Main characteristics of included trials

<table>
<thead>
<tr>
<th>Trial name</th>
<th>Study design</th>
<th>Multicentre</th>
<th>Primary endpoint</th>
<th>Age (years)</th>
<th>Exclusion criteria</th>
<th>Stent type</th>
<th>Crossover (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Airoldi et al\textsuperscript{15}</td>
<td>DS (n=140) vs CS Yes</td>
<td>Procedural outcome</td>
<td>59</td>
<td>No</td>
<td>Left main disease, calcified, tortuous, bifurcation lesions, CTO, IRS</td>
<td>Crossflex (Cordis)</td>
<td>13.2</td>
</tr>
<tr>
<td>BET\textsuperscript{16}</td>
<td>DS (n=173) vs CS Yes (n=165)</td>
<td>Death, MI, UA, CABG, re-PCI</td>
<td>64</td>
<td>No</td>
<td>Ostial lesion, calcified, tortuous lesions, thrombus</td>
<td>Tenax (Biotronik)</td>
<td>13.9</td>
</tr>
<tr>
<td>Brueck et al\textsuperscript{17}</td>
<td>DS (n=171) vs CS No (n=164)</td>
<td>Procedural success</td>
<td>63</td>
<td>Yes</td>
<td>Left main disease, calcified, tortuous, bifurcation lesions, CTO, IRS, LVEF &lt;30%</td>
<td>Bx Velocity (Cordis)</td>
<td>5</td>
</tr>
<tr>
<td>CK TEST\textsuperscript{18}</td>
<td>DS (n=59) vs CS Yes (n=44)</td>
<td>Procedural outcome, death, MI, TVR</td>
<td>64</td>
<td>No</td>
<td>Left main disease, CTO, bifurcation, calcified, tortuous, restenotic lesions</td>
<td>Several</td>
<td>0</td>
</tr>
<tr>
<td>CONVERTIBLE\textsuperscript{19}</td>
<td>DS (n=101) vs CS Yes (n=101)</td>
<td>Mean lumen diameter by QCA after stent placement</td>
<td>63</td>
<td>Yes</td>
<td>Calcified, tortuous, bifurcation lesions, CTO, IRS, LVEF &lt;30%</td>
<td>BeStent2 (Medtronic)</td>
<td>6</td>
</tr>
<tr>
<td>Cuisset et al\textsuperscript{20}</td>
<td>DS (n=25) vs CS No (n=25)</td>
<td>Index of microcirculatory resistance</td>
<td>66</td>
<td>No</td>
<td>LVEF &lt;30%, previous MI, CTO, ISR, calcified, bifurcation, ostial lesions.</td>
<td>Not reported</td>
<td>0</td>
</tr>
<tr>
<td>Danzi et al\textsuperscript{21}</td>
<td>DS (n=61) vs CS No (n=61)</td>
<td>Procedural success</td>
<td>58</td>
<td>Yes</td>
<td>Calcified lesions, CTO</td>
<td>NIR (Medinol), Paragon (Progressive Angioplasty System)</td>
<td>3</td>
</tr>
<tr>
<td>DECIDE\textsuperscript{22}</td>
<td>DS (n=64) vs CS Yes (n=64)</td>
<td>Angiographic restenosis</td>
<td>56</td>
<td>Yes</td>
<td>Ostial, tortuous, calcified lesions, CTO</td>
<td>NirElite (Boston Scientific)</td>
<td>29.7</td>
</tr>
<tr>
<td>DIRAMI\textsuperscript{23}</td>
<td>DS (n=110) vs CS No (n=107)</td>
<td>Procedural success</td>
<td>56</td>
<td>Yes</td>
<td>Cardiogenic shock, pulmonary oedema</td>
<td>Bx Velocity (Cordis), Multilink (Guidant), other</td>
<td>12</td>
</tr>
<tr>
<td>DIRECT\textsuperscript{24}</td>
<td>DS (n=210) vs CS Yes (n=201)</td>
<td>Value MI, TVR</td>
<td>60</td>
<td>No</td>
<td>Left main disease, calcified, tortuous, bifurcation lesions, CTO, AMI</td>
<td>Not reported</td>
<td>2.8</td>
</tr>
<tr>
<td>DIRECTQ\textsuperscript{25}</td>
<td>DS (n=65) vs CS Yes (n=72)</td>
<td>Difference in mean length of stent</td>
<td>59</td>
<td>No</td>
<td>Calcified, tortuous, bifurcation, CTO, long lesions</td>
<td>NIR (Medinol)</td>
<td>11</td>
</tr>
<tr>
<td>DISCO\textsuperscript{26}</td>
<td>DS (n=210) vs CS Yes (n=206)</td>
<td>Feasibility and safety</td>
<td>59</td>
<td>Yes</td>
<td>Left main disease, calcified, tortuous, bifurcation, AMI, CTO, IRS</td>
<td>Several</td>
<td>3</td>
</tr>
<tr>
<td>Hoffman et al\textsuperscript{27}</td>
<td>DS (n=125) vs CS No (n=124)</td>
<td>Endothelin levels after stent implantation</td>
<td>63</td>
<td>Yes</td>
<td>Ostial, calcified, bifurcation, tortuous lesions, CTO, thrombus</td>
<td>Bx Velocity (Cordis)</td>
<td>9</td>
</tr>
<tr>
<td>Isselmuiden et al\textsuperscript{28}</td>
<td>DS (n=200) vs CS No (n=200)</td>
<td>Death, MI, TVR and stent thrombosis</td>
<td>61</td>
<td>Yes</td>
<td>Ostial, bifurcation, calcified lesions and CTO</td>
<td>AVE S670 (Medtronic)</td>
<td>11.7</td>
</tr>
<tr>
<td>ISAR-DIRECT\textsuperscript{29}</td>
<td>DS (n=456) vs CS No (n=454)</td>
<td>Angiographic restenosis</td>
<td>65</td>
<td>Yes</td>
<td>AMI, left main disease, total vessel occlusion</td>
<td>Several</td>
<td>21.7</td>
</tr>
<tr>
<td>Kovar et al\textsuperscript{30}</td>
<td>DS (n=37) vs CS Yes (n=40)</td>
<td>Mean number of utilised device</td>
<td>62</td>
<td>No</td>
<td>CTO, vein graft, AMI</td>
<td>Several</td>
<td>5.4</td>
</tr>
<tr>
<td>Loubeyre et al\textsuperscript{31}</td>
<td>DS (n=102) vs CS No (n=104)</td>
<td>Angiographic and clinical outcomes</td>
<td>59</td>
<td>No</td>
<td>Calcified lesions, left main disease, vein graft</td>
<td>Bx Velocity (Cordis), Multilink (Guidant), AVE (Medtronic), other</td>
<td>7.8</td>
</tr>
<tr>
<td>NIR future\textsuperscript{32}</td>
<td>DS (n=39) vs CS Yes (n=42)</td>
<td>Equipment cost, fluoroscopy time, contrast use</td>
<td>61</td>
<td>No</td>
<td>Ostial, bifurcation, calcified lesions, IRS, thrombus, CTO, LVEF &lt;30%, TIMI grade 0-1</td>
<td>NIR Primo (Boston Scientific)</td>
<td>7.7</td>
</tr>
<tr>
<td>Ozdemir et al\textsuperscript{33}</td>
<td>DS (n=25) vs CS No (n=25)</td>
<td>Angiographic results</td>
<td>57</td>
<td>No</td>
<td>Cardiogenic shock, left main disease</td>
<td>divYsio (Biocompatibles Ltd)</td>
<td>0</td>
</tr>
<tr>
<td>PREDICT\textsuperscript{34}</td>
<td>DS (n=198) vs CS Yes (n=201)</td>
<td>Death, MI, TLR and stent thrombosis</td>
<td>62</td>
<td>Yes</td>
<td>Calcified, tortuous, thrombotic lesions, recent AMI, LVEF &lt;30%</td>
<td>AVE S670 (Medtronic)</td>
<td>8</td>
</tr>
<tr>
<td>Sabatier et al\textsuperscript{35}</td>
<td>DS (n=65) CS No (n=65)</td>
<td>No reflow</td>
<td>61</td>
<td>No</td>
<td>Left main disease, calcified, tortuous lesions, ISR, vein graft</td>
<td>NIR PRIMO (Boston Scimed)</td>
<td>6</td>
</tr>
<tr>
<td>SWIBAP\textsuperscript{36}</td>
<td>DS (n=197) vs CS Yes (n=199)</td>
<td>Angiographic success</td>
<td>60</td>
<td>No</td>
<td>Left main disease, AMI, UA, bifurcation, restenotic, calcified lesions</td>
<td>NIR (Medinol)</td>
<td>2.5</td>
</tr>
<tr>
<td>TRENDS\textsuperscript{37}</td>
<td>DS (n=379) vs CS Yes (n=395)</td>
<td>Death, MI, TLR</td>
<td>60</td>
<td>Yes</td>
<td>Left main disease, calcified, ostial lesions, recent AMI</td>
<td>Multilink (Guidant)</td>
<td>5.7</td>
</tr>
<tr>
<td>VELVET\textsuperscript{38}</td>
<td>DS (n=200) vs CS Yes (n=201)</td>
<td>Death, MI, CABG, TLR stroke</td>
<td>61</td>
<td>No</td>
<td>Left main disease, recent AMI, LVEF &lt;30%, bifurcation, IRS, TIMI 0, SVG</td>
<td>Bx Velocity (Cordis)</td>
<td>9.2</td>
</tr>
</tbody>
</table>

**AMl, acute myocardial infarction; CABG, coronary artery bypass graft; CS, conventional stenting; CTO, chronic total occlusion; DS, direct stenting; ISR, in-stent restenosis; LVEF, left ventricular ejection fraction; MI, myocardial infarction; SVG, saphenous vein graft; TIMI, Thrombolysis in Myocardial Infarction; TLR, target-lesion revascularisation; TVR, target-vessel revascularisation; UA, unstable angina.**
All studies included were randomised and 14 out of 24 provided detailed descriptions of appropriate randomisation methods, mainly based on computer-generated randomisation lists. 

All trials reported the number of patients, if any, lost to follow-up and 15 trials reported sample-size calculations. 

The analysis according to the intention-to-treat principle was performed in all trials.

**Primary endpoint**

The composite endpoint of death or myocardial infarction occurred in a total of 308 patients (4.53%). As reported in figure 2, DS was associated with a significant death/myocardial infarction reduction (3.95% versus 5.10%, DS vs CS, respectively, OR = 0.76 (95% CI 0.60 to 0.96), p = 0.02). No significant heterogeneity was observed across trials (I² = 12%, phet = 0.30). This reduction was primarily driven by a lower MI incidence, since no significant difference in the occurrence of death was found between groups (0.72% vs 1.00%, DS vs CS, respectively, OR = 0.73 (0.42 to 1.26), p = 0.26).

**Secondary endpoints**

Myocardial infarction was experienced by a total of 245 patients (3.60%). As depicted in figure 3, patients treated with DS experienced less MI than CS patients (3.16% versus 4.04%, respectively, OR = 0.77 (95% CI 0.59 to 0.99), p = 0.04). A modest heterogeneity was observed across trials (I² = 16%, phet = 0.24), probably owing to different MI definition across the included trials (see supplementary data). However, even if we excluded trials that did not report MI definition or did not clearly adopt creatine kinase MB isoenzyme (CK-MB) evaluation, MI was still significantly reduced in patients allocated to DS (OR = 0.69 (95% CI 0.49 to 0.98), p = 0.04).

**Bias and sensitivity analysis**

None of the funnel plots showed skewed distributions, suggesting that no publication bias was present. Furthermore, the adjusted rank correlation test did not point out any publication bias. Sensitivity analysis demonstrated that no single study significantly altered the summary ORs. Also with the Mantel-Haenzel method, the primary endpoint remained in favour of DS (OR = 0.76 (95% CI 0.60 to 0.96), p = 0.02) and myocardial infarction (OR = 0.77 (95% CI 0.60 to 1.00), p = 0.05).

**Meta-regression**

Using a weighted least-square regression, no significant relation was found between crossover rates and the natural logarithm of OR for death/MI (R² = 0.007, β-coefficient = 0.08 (95% CI – 0.04 to 0.06), p = 0.70) (see supplementary data).

**DISCUSSION**

The main finding of this meta-analysis is that a DS strategy is associated with a reduction in the occurrence of death or MI, driven primarily by lower rates of MI, up to the 6-month follow-up.

Most of the included trials were originally designed to evaluate a possible role in reducing restenosis associated with DS technique, as suggested from initial experimental data.
According to those data, there would be much more endothelial preservation and less vascular inflammatory response associated with DS compared with CS, leading to lower restenosis rates. However, none of included trials demonstrated a significant benefit in patients treated with DS in terms of TVR incidence, with the exception of Brueck et al. As reported above, the present meta-analysis showed no differences between DS and CS in terms of TVR, consistently with an earlier meta-analysis.

**Figure 3** OR of myocardial infarction associated with direct stenting versus conventional stenting.

**Figure 4** OR of target-vessel revascularisation associated with direct stenting versus conventional stenting.
including only less than half of patients. Unfortunately, initial encouraging experimental data did not translate in a significant clinical benefit in terms of reduced restenosis. Of note, the aforementioned data were performed in healthy animal vessels, using low pressure balloon inflation, which might have positively influenced endothelial response to mechanical injury. In addition, the function of endothelial cells after DS in humans still remains unclear. Furthermore, although no data exist to suggest that MI after revascularisation reduces the likelihood of restenosis, it would be expected that patients who experience myocyte necrosis in the territory of a restenotic vessel would be less likely, according to the presence of non-viable myocardium, to experience angina as a result of recurrent narrowing. In turn, these patients would be less likely to undergo referral for ischaemia-driven TVR. This aspect might deserve additional research to explain how the TVR incidence was not affected by the DS strategy.

Besides reinforcing the safety and the feasibility of DS approach, we also observed a 25% reduction in the odds of MI associated with DS. In this respect, a randomised study showed a benefit of DS compared with CS in patients going elective PCI in terms of lower levels of post-PCI microvascular resistance and lower myocardial injury as mirrored from higher post-PCI troponin T values. Similarly, a prospective study found higher post-PCI troponin I levels in patients treated with a conventional stenting approach, which are associated with a worse prognosis. A further theoretical advantage is that DS could avoid clot and plaque material distal microembolisation, with a possible improvement in coronary and myocardial perfusion, thus minimising myocardial cell injury. Of note, microembolisation of plaque debris and side-branch occlusion has been proposed as the most likely mechanism of troponin release after PCI. Furthermore, we previously demonstrated an improvement in post-procedural TIMI 3 grade flow and corrected TIMI frame count in patients undergoing PCI with DS. As specified above, the vast majority of included trials in this meta-analysis performed PCI with bare-metal stent implantation. For this reason, these results cannot be extended to drug-eluting stents and must be taken only as generating a hypothesis, since some concerns have been raised about the potential damage of polymer coating or a non-uniform drug elution associated with the DS technique. This aspect was confirmed from the exclusion of the DS approach in the preliminary trials evaluating both sirolimus-eluting and paclitaxel-eluting stents. However, recent studies have shown the feasibility and the effectiveness of DS with both drug-eluting stents, possibly reducing angiographic restenosis and TVR.

Despite these considerations, DS has some intrinsic potential disadvantages that need to be pointed out: a higher risk of failure to cross the lesion, stent dislodgment, loss or embolisation, inadequate choice of stent diameter and length. In addition, specific coronary lesion subsets, like chronic total occlusions, calcified, tortuous or angulated lesions, are not really suitable for a direct stenting approach. In fact, these lesions have been excluded in most of the included trials (table 1). Notably, meta-regression did not report a significant relation between crossover rates and the occurrence of the primary endpoint. On the other hand, DS is advocated when PCI is performed in saphenous vein graft lesions since it has been associated with decreased major cardiac events when compared to CS.

This meta-analysis presents several important limitations. First, this is a meta-analysis at study level, and we could not properly assess the role of confounding factors. However, a clear limitation of patient-level data analysis is that patient-level data are not always available from all investigators, introducing several biases. Second, some RCTs were underpowered to detect significant differences between interventions in the main outcomes; however, this reinforces the necessity of the present study. Third, different MI definitions, adopted among the included trials, possibly could have influenced the final results, despite a low and not significant heterogeneity being reported. Fourth, although the observed reduction in MI seems to be related to lower peri-procedural MIs, we were unable to evaluate this issue in detail, since post-PCI incidence was available for only a few studies. However, in-hospital MI data, available for 20 studies (4619 patients), showed a significant reduction in patients allocated to the DS arm (1.42% versus 2.52%, p=0.007, OR=0.56 (95% CI 0.37 to 0.85)).

In conclusion, this meta-analysis demonstrates that, in selected coronary lesions, direct stenting is not only a feasible technique, but also reduces the occurrence of myocardial infarction.

Competing interests None.

Contributors FP had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: FP, RP, MC. Acquisition of data: SC, RDR, C’A. Analysis and interpretation of data: FP, RP, SC. Drafting of the manuscript: RP, SC, GG, C’A. Critical revision of the
manuscript for important intellectual content: FP, MC, GG. Statistical analysis: FP, GG, RDR. Study supervision: FP, MC.

Provenance and peer review
Not commissioned; externally peer reviewed.

REFERENCES