Ischemic Postconditioning During Primary Percutaneous Coronary Intervention
Mechanisms and Clinical Application

Jian Liu, MD FACC FESC FSCAI
Chief Physician, Professor of Medicine
Department of Cardiology, Peking University People’s Hospital
Infarct size is a determinant of mortality in Acute Myocardial Infarction
Reperfusion improves outcome

van Domburg et al. JACC 2005:15–20
Current treatment of AMI

- β-blockers
- ACE inhibitors
- statins
- ....

improve post-MI outcome, but *not via a reduction in infarct size*

Action on infarct size

- **Ischemic damage**: YES
  - thrombolysis / PCI ischemia time
  - antiplatelet agents ischemia time

- **Reperfusion damage**: NO
Reperfusion Injury: the two facets

Acute thrombotic occlusion

Thrombolysis/Angioplasty

Myocardial injury: no reflow
Myocyte reperfusion injury

Death or stunning

No recovery
Delayed recovery
Infarction: a two-component damage
Reperfusion injury increase infarct size

- Increasing myocyte cell death, activation of apoptosis and promotion of endothelial dysfunction
Main mechanisms of cardiomyocyte cell death during myocardial reperfusion

Hausenloy D.J et al Eur H J 2017
Ischemic Postconditioning

Zhao et al. were the first to describe a phenomenon known as “post-conditioning” in which a sequence of repetitive interruption of coronary blood flow was applied immediately after reopening of the occluded vessel can reduced infarct size.

Zhao, ZQ et al. AM J Physiol Heart Circ 2003
Ischemic Postconditioning

- Repetitive reversible ischemia during early reperfusion after the prolonged ischemic insult.
- Comparable protective effects to preconditioning in animal studies.

Ischaemic postconditioning: cardiac protection after the event
Does Postconditioning protect the human heart?

A « proof of concept » study

Postconditioning the Human Heart

Patrick Staat, MD; Gilles Rioufol, MD, PhD; Christophe Piot, MD, PhD; Yves Cottin, MD, PhD; Thien Tri Cung, MD; Isabelle L Huillier, MD; Jean-François Aupetit, MD, PhD; Eric Bonnefoy, MD, PhD; Gérard Finet, MD, PhD; Xavier André-Fouët, MD; Michel Ovize, MD, PhD

(Circulation. 2005;112:2143-2148.)
Study population
A First « Human Model » of Postconditioning

Inclusion criteria

1. Age ≥ 18
2. First acute (STE)MI / chest pain onset < 6 hrs
3. Need for emergency PTCA

Exclusion criteria

1. Cardiac arrest
2. Cardiogenic shock
3. Circumflex coronary artery as culprit for AMI
Post-Conditioning algorithm

Occluded coronary artery

Reperfusion

Control

Direct stenting

Postcond

Balloon inflations - deflations

Staat et al. Circulation. 2005;112:2143-2148
Determinants of infarct size

Area at Risk size (ACS)  

Duration of Ischemia

(control  PostC)

(%)  

(min.)

ns  

ns
CK release during reperfusion

- 36 % (p < 0.05)

Staat et al. Circulation. 2005;112:2143-2148
CK release versus ACS
(infarct size versus area at risk)
Estimation of « no reflow »

Staat et al. Circulation. 2005;112:2143-2148
Ischemic Postconditioning

- Postconditioning reduced enzymatic infarct size in patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI).
  - Staat P et al. Circulation 2005, the first report in human

- Inconsistent results of studies using CE-MRI for infarct size.
  - Thuny F et al. J Am Coll Cardiol 2012
  - Sorensson P et al. Heart 2010
  - Freixa X et al. Eur Heart J 2012
  - Tarantini G et al. Int J Cardiol 2012

- No large scale trials

PostC is Protective!

PostC is harmful!
Post-conditioning and injury biomarkers

In a multi-center randomized controlled study, Roubille et al. failed to show any significant decrease in CK and TnI release, even after adjustment for the size of the area at risk.

Roubille, F et al, European Heart Journal 2014
Using contrast-enhanced cardiac-MRI within 3 days after reperfusion, Mewton et al. showed that post-conditioning was associated with smaller, early and late microvascular obstruction size ($p = 0.01$).
Post-conditioning effects

Post-conditioning and left ventricular function

- In the POSTEMI trial, 272 patients were randomized to post-conditioning group \((n = 136)\) and control group \((n = 136)\).
- Primary endpoint was infarct size measured by cardiac MRI.
- After 4 months, no difference was observed between control group and post-conditioning group.

Post-conditioning effects

Post-conditioning and clinical outcome

In a meta-analysis of 15 randomized trials including 1545 patients with a mean follow-up of 4.7 months, Khalili et al. did not note any impact of mechanical post-conditioning on mortality (OR = 1.52; 95% CI 0.77–2.99; $p = 0.23$), recurrent myocardial infarction (OR = 3.04; 95% CI 0.74–12.54; $p = 0.12$), stent thrombosis (OR = 1.24, 95% CI 0.51–3.04; $p = 0.83$), or the composite MACE outcome (OR = 1.53; 95% CI 0.89–2.63; $p = 0.13$).
Effect of Postconditioning on Myocardial Reperfusion during Primary Percutaneous Coronary Intervention: POST Trial Design

A Korean multicenter, prospective, randomized, open-label, blinded endpoint trial

- STEMI patients undergoing primary PCI
- Randomization after diagnostic coronary angiogram (n=700)
- Postconditioning with primary PCI (n=350)
- Conventional primary PCI (n=350)

Assessment of myocardial reperfusion:
- ST-segment resolution
- Myocardial blush grade

Clinical follow-up

ClinicalTrials.gov identifier: NCT00942500
Study Protocol

Postconditioning

- Four episodes of 1-minute balloon occlusion and 1-minute deflation*
- Immediately (within 1 minute) after restoration (TIMI grade ≥2) of coronary flow (without regard to method of achieving reflow)
- Aspirin 300 mg and clopidogrel 600 mg
- Thrombus aspiration, predilation before stenting, or use of glycoprotein IIb/IIIa inhibitors were left to the operators’ discretion.

Endpoints

- **Primary End point**
  - Complete ST-segment resolution (STR >70%) at 30 minutes after the procedure

- **Secondary End Points**
  - TIMI flow grade after PCI
  - Myocardial blush grade
  - Major adverse cardiac events (MACE: a composite of death, reinfarction, severe heart failure*, or stent thrombosis†) at 30 days
  - Each component of MACE at 30 days
  - Target vessel revascularization at 30 days

* Heart failure with documented arterial partial pressure of oxygen less than 60 mmHg or with pulmonary edema documented radiographically or requiring intubation, 100% oxygen, or insertion of a mechanical support device.
†Definite or probable stent thrombosis by the ARC definition
<table>
<thead>
<tr>
<th>Category</th>
<th>Postconditioning</th>
<th>Conventional PCI</th>
<th>Difference (95% CI)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>n / total n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;65</td>
<td>87/211 (41.2%)</td>
<td>88/205 (42.9%)</td>
<td>-1.7 (-11.1 to 7.7)</td>
<td>0.73</td>
</tr>
<tr>
<td>≥65</td>
<td>51/130 (39.2%)</td>
<td>51/130 (39.2%)</td>
<td>0.0 (-11.7 to 11.7)</td>
<td>0.99</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>105/269 (39.0%)</td>
<td>98/249 (39.4%)</td>
<td>-0.3 (-8.7 to 8.0)</td>
<td>0.94</td>
</tr>
<tr>
<td>Female</td>
<td>33/72 (45.8%)</td>
<td>41/86 (47.7%)</td>
<td>-1.8 (-17.0 to 13.5)</td>
<td>0.82</td>
</tr>
<tr>
<td>Infarct-related artery</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAD</td>
<td>32/158 (20.3%)</td>
<td>28/151 (18.5%)</td>
<td>1.7 (-7.2 to 10.5)</td>
<td>0.70</td>
</tr>
<tr>
<td>Non-LAD</td>
<td>106/183 (57.9%)</td>
<td>111/184 (60.3%)</td>
<td>-2.4 (-12.3 to 7.6)</td>
<td>0.64</td>
</tr>
<tr>
<td>Symptom onset-to-reperfusion time</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;3 hours</td>
<td>71/154 (46.1%)</td>
<td>75/153 (49.0%)</td>
<td>-2.9 (-13.9 to 8.2)</td>
<td>0.61</td>
</tr>
<tr>
<td>≥3 hours</td>
<td>67/187 (35.8%)</td>
<td>64/181 (35.4%)</td>
<td>0.5 (-9.3 to 10.2)</td>
<td>0.93</td>
</tr>
<tr>
<td>Thrombus aspiration</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>60/154 (39.0%)</td>
<td>67/170 (39.4%)</td>
<td>-0.5 (-11.0 to 10.1)</td>
<td>0.93</td>
</tr>
<tr>
<td>No</td>
<td>78/187 (41.7%)</td>
<td>72/165 (43.6%)</td>
<td>-1.9 (-12.2 to 8.3)</td>
<td>0.72</td>
</tr>
<tr>
<td>Direct stenting</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>17/41 (41.5%)</td>
<td>19/45 (42.2%)</td>
<td>-0.7 (-20.7 to 19.4)</td>
<td>0.97</td>
</tr>
<tr>
<td>No</td>
<td>121/300 (40.3%)</td>
<td>120/290 (41.4%)</td>
<td>-1.1 (-8.9 to 6.9)</td>
<td>0.80</td>
</tr>
<tr>
<td>Glycoprotein IIb/IIIa inhibitors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>35/79 (44.3%)</td>
<td>36/78 (46.2%)</td>
<td>-1.9 (-17.0 to 13.4)</td>
<td>0.82</td>
</tr>
<tr>
<td>No</td>
<td>103/262 (39.3%)</td>
<td>103/257 (40.1%)</td>
<td>-0.8 (-9.1 to 7.6)</td>
<td>0.86</td>
</tr>
</tbody>
</table>
## Angiographic Outcomes

<table>
<thead>
<tr>
<th></th>
<th>Postconditioning (n=350)</th>
<th>Conventional PCI (n=350)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TIMI flow after PCI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0/1</td>
<td>8/349 (2.3%)</td>
<td>19/348 (5.5%)</td>
<td>0.08</td>
</tr>
<tr>
<td>2</td>
<td>20/349 (5.7%)</td>
<td>23/348 (6.6%)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>321/349 (92.0%)</td>
<td>306/348 (87.9%)</td>
<td></td>
</tr>
<tr>
<td><strong>Myocardial blush grade after PCI</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0/1</td>
<td>60/349 (17.2%)</td>
<td>78/348 (22.4%)</td>
<td>0.20</td>
</tr>
<tr>
<td>2</td>
<td>108/349 (30.9%)</td>
<td>106/348 (30.5%)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>181/349 (51.9)</td>
<td>164/348 (47.1)</td>
<td></td>
</tr>
</tbody>
</table>

TIMI = thrombolysis in myocardial infarction.
# Clinical Outcomes at 1-month Postconditioning

<table>
<thead>
<tr>
<th>Event</th>
<th>Postconditioning (n=350)</th>
<th>Conventional PCI (n=350)</th>
<th>Relative risk (95% CI)*</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>13 (3.7%)</td>
<td>10 (2.9%)</td>
<td>1.30 (0.58-2.92)</td>
<td>0.53</td>
</tr>
<tr>
<td>Cardiac death</td>
<td>10 (2.9%)</td>
<td>9 (2.6%)</td>
<td>1.11 (0.46-2.70)</td>
<td>0.82</td>
</tr>
<tr>
<td>Reinfarction</td>
<td>2 (0.6%)</td>
<td>1 (0.3%)</td>
<td>2.00 (0.18-21.74)</td>
<td>0.99†</td>
</tr>
<tr>
<td>Severe heart failure</td>
<td>2 (0.6%)</td>
<td>5 (1.4%)</td>
<td>0.40 (0.08-2.05)</td>
<td>0.29†</td>
</tr>
<tr>
<td>Stent thrombosis</td>
<td>7 (2.0%)</td>
<td>6 (1.7%)</td>
<td>1.17 (0.40-3.44)</td>
<td>0.78</td>
</tr>
<tr>
<td>Target-vessel revascularization</td>
<td>3 (0.9%)</td>
<td>3 (0.9%)</td>
<td>1.00 (0.20-4.92)</td>
<td>0.99†</td>
</tr>
<tr>
<td>MACE‡</td>
<td>15 (4.3%)</td>
<td>13 (3.7%)</td>
<td>1.15 (0.56-2.39)</td>
<td>0.70</td>
</tr>
</tbody>
</table>

* Relative risk is for the postconditioning group as compared with the conventional PCI group.

† The P value was calculated with the use of Fisher’s exact test.

‡ Major adverse cardiac event was a composite of death, reinfarction, severe heart failure, or stent thrombosis.
Outcomes according to STR, postprocedural MBG and TIMI flow grade

Resolution of ST-Segment Elevation (%)

Myocardial Blush Grade

Postprocedural TIMI flow grade

Death (%)

Major Adverse Cardiac Events (%)

P=0.04

P<0.001

P=0.02

P<0.001
Long-term Effects of Ischemic Postconditioning on Clinical Outcomes

POST trial: 700 STEMI patients randomized to standard primary PCI or PCI plus ischemic postconditioning, July 2009-June 2012.

<table>
<thead>
<tr>
<th>1-Year Outcomes</th>
<th>Postconditioning (n = 350)</th>
<th>Control (n = 350)</th>
<th>HR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major Adverse Events</td>
<td>6.1%</td>
<td>4.6%</td>
<td>1.32 (0.69-2.53)</td>
</tr>
<tr>
<td>Death</td>
<td>4.9%</td>
<td>3.7%</td>
<td>1.32 (0.64-2.71)</td>
</tr>
<tr>
<td>Severe Heart Failure</td>
<td>2.6%</td>
<td>2.3%</td>
<td>1.13 (0.44-2.94)</td>
</tr>
</tbody>
</table>

In addition to failing to improve myocardial reperfusion after primary PCI, ischemic postconditioning does not reduce major adverse events through 1 year.

In this multicenter, prospective, randomized, open-label, blinded endpoint trial,

- Ischemic postconditioning with primary PCI did not improve myocardial reperfusion compared with conventional primary PCI.

- Clinical outcomes at 1-month and 1-year were not significantly different between the randomized groups.

- Cardioprotective effect of ischemic postconditioning was not found in any of prespecified subgroups.
Meta-analysis: Inconsistent Results
More high-quality multicenter RCTs focusing on MACE are warranted.
A meta-analysis of 5 eligible studies on peak CK-MB. Test for overall effect: Z = 7.75 (p < 0.00001)

A meta-analysis of 3 eligible studies on SPECT determining infarct size.

A meta-analysis of 3 eligible studies on complete ST-segment resolution.
Forest plot for clinical outcomes for IPoC vs. conventional PPCI

The cardioprotection of ischemic postconditioning in patients with acute ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention

- IPoC might improve cardiac function and reduce the incidence of heart failure and serious arrhythmia in patients with STEMI undergoing PPCI.

### The effect of IPoC on clinical outcomes in patients with STEMI undergoing PPCI.

<table>
<thead>
<tr>
<th>Outcomes after PPCI</th>
<th>sample size (IPoC/control)</th>
<th>Heterogeneity</th>
<th>WMD or OR</th>
<th>95% CI</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart failure</td>
<td>753/752</td>
<td>0</td>
<td>0.47</td>
<td>0.29 to 0.78</td>
<td>0.003</td>
</tr>
<tr>
<td>Serious arrhythmia</td>
<td>112/113</td>
<td>0</td>
<td>0.34</td>
<td>0.13 to 0.90</td>
<td>0.03</td>
</tr>
<tr>
<td>LVEF(&gt; = 3 months)</td>
<td>307/316</td>
<td>70%</td>
<td>0.03</td>
<td>0.01 to 0.05</td>
<td>0.004</td>
</tr>
<tr>
<td>WMSI</td>
<td>206/219</td>
<td>14%</td>
<td>-0.12</td>
<td>-0.16 to -0.08</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Death</td>
<td>818/815</td>
<td>0</td>
<td>1.36</td>
<td>0.79 to 2.36</td>
<td>0.27</td>
</tr>
<tr>
<td>Stent thrombosis</td>
<td>444/446</td>
<td>0</td>
<td>1.01</td>
<td>0.40 to 2.56</td>
<td>0.99</td>
</tr>
<tr>
<td>Reinfarction</td>
<td>405/406</td>
<td>0</td>
<td>3.11</td>
<td>0.62 to 15.63</td>
<td>0.17</td>
</tr>
<tr>
<td>ACS</td>
<td>486/486</td>
<td>58%</td>
<td>0.53</td>
<td>0.06 to 4.72</td>
<td>0.57</td>
</tr>
<tr>
<td>MACE</td>
<td>605/612</td>
<td>26%</td>
<td>0.96</td>
<td>0.64 to 1.44</td>
<td>0.85</td>
</tr>
<tr>
<td>LVEF in acute phase</td>
<td>670/713</td>
<td>77%</td>
<td>0.02</td>
<td>-0.01 to 0.04</td>
<td>0.19</td>
</tr>
<tr>
<td>CTFC</td>
<td>164/216</td>
<td>58%</td>
<td>-3.96</td>
<td>-6.85 to -1.06</td>
<td>0.007</td>
</tr>
<tr>
<td>CK peak</td>
<td>293/341</td>
<td>93%</td>
<td>-255.66</td>
<td>-745.22 to 231.89</td>
<td>0.30</td>
</tr>
<tr>
<td>CK-MB peak</td>
<td>566/619</td>
<td>95%</td>
<td>-21.29</td>
<td>-65.55 to 22.98</td>
<td>0.29</td>
</tr>
<tr>
<td>Tnl peak</td>
<td>96/100</td>
<td>99%</td>
<td>56.81</td>
<td>-48.24 to 161.85</td>
<td>0.29</td>
</tr>
<tr>
<td>Infarct size</td>
<td>172/171</td>
<td>84%</td>
<td>-0.70</td>
<td>-6.23 to 4.83</td>
<td>0.80</td>
</tr>
<tr>
<td>Area at risk</td>
<td>101/100</td>
<td>0</td>
<td>0.38</td>
<td>-2.54 to 3.30</td>
<td>0.80</td>
</tr>
<tr>
<td>Complete ST segment resolution</td>
<td>664/716</td>
<td>76%</td>
<td>1.58</td>
<td>0.78 to 3.17</td>
<td>0.20</td>
</tr>
<tr>
<td>MBG</td>
<td>725/725</td>
<td>25%</td>
<td>1.18</td>
<td>0.92 to 1.52</td>
<td>0.20</td>
</tr>
<tr>
<td>MSI</td>
<td>107/113</td>
<td>81%</td>
<td>-3.83</td>
<td>-20.12 to 12.48</td>
<td>0.65</td>
</tr>
</tbody>
</table>

LVEF, left ventricular ejection fraction; WMSI, wall motion score index; MACE, major adverse cardiovascular events; CTFC, corrected TIMI frame count; CK, creatine kinase; CK-MB, creatine kinase-MB; Tnl, Troponin I; MBG, myocardial blush grades; MSI, myocardial savage index. WMD, weighted mean difference; OR, odd ratio.
Stenting technique, gender, and age are associated with cardioprotection by ischaemic postconditioning in primary coronary intervention: a systematic review of 10 randomized trials

- Available evidence from the present systematic review and meta-analysis suggests that IPoC may confer cardioprotection for STEMI during primary PCI.

- Cardioprotective effects of IPoC are more pronounced among young and male patients, and those in whom direct-stenting techniques were used.
Strategy

Mechanical post-conditioning

- In the majority of studies, post-conditioning was performed by four 30–60-s cycles of low pressure balloon inflations (4–6 atm) at the site of previous occlusion, each separated by 30–60 s of reflow.
Protocols employed in different trials on post-conditioning in PCI

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Protocol of POC</th>
<th>N POC/controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Staat et al. [6]</td>
<td>2005</td>
<td>60 s x 4</td>
<td>14/16</td>
</tr>
<tr>
<td>Ma et al. [34]</td>
<td>2006</td>
<td>30 s x 3</td>
<td>47/47</td>
</tr>
<tr>
<td>Yang et al. [26]</td>
<td>2007</td>
<td>30 s x 3</td>
<td>23/18</td>
</tr>
<tr>
<td>Thibault et al. [25]</td>
<td>2008</td>
<td>60 s x 4</td>
<td>17/21</td>
</tr>
<tr>
<td>Sorensson et al. [27]</td>
<td>2010</td>
<td>60 s x 4</td>
<td>38/38</td>
</tr>
<tr>
<td>Freixa et al. [28]</td>
<td>2012</td>
<td>60 s x 4</td>
<td>39/40</td>
</tr>
<tr>
<td>Tarantini et al. [29]</td>
<td>2012</td>
<td>60 s x 4</td>
<td>39/39</td>
</tr>
<tr>
<td>Zhao et al. [38]</td>
<td>2012</td>
<td>60 s x 4</td>
<td>32/30</td>
</tr>
<tr>
<td>Hahn et al. [20]</td>
<td>2013</td>
<td>60 s x 4</td>
<td>350/350</td>
</tr>
<tr>
<td>Dwyer et al. [41]</td>
<td>2013</td>
<td>30 s x 4</td>
<td>50/52</td>
</tr>
<tr>
<td>Limalanathan et al. [23]</td>
<td>2014</td>
<td>60 s x 4</td>
<td>136/136</td>
</tr>
</tbody>
</table>
Strategy

Pharmacological post-conditioning alternative

- **Adenosine**
  
  Nicolli et al. showed that the use of adenosine results not only in significant improvement of microvascular obstruction assessed by ST-segment resolution but also in MACE occurrence at 30 days.

- **Natriuretic peptide**
  
  Kitakaze et al. showed patients with AMI who were given atrial natriuretic peptide had lower infarct size of 14.7% (95% CI 3.0–24.9%), and better LVEF at 6–12 months (ratio 1.05, 95% CI 1.01–1.10, p = 0.024).
Remote ischemic conditioning alternative

Remote ischemic conditioning (RIC) is transient non-injurious ischemia of one organ or tissue can protect a distant organ or tissue from ischemic injury.

Several clinical studies have found that RIC using transient arm or leg ischaemia/reperfusion reduced MI size by 20–30% (assessed by cardiac enzymes, SPECT or cardiac MRI) in STEMI patients reperfused by either PPCl or thrombolysis.

Hausenloy D.J et al Eur H J 2017
RIC using transient limb ischaemia/reperfusion holds promise as an adjunct to PPCI in STEMI patients for reducing MI size. Whether it can improve long-term clinical outcomes is not known.
Toward New Clinical Strategies

Ischemic PostC

PCI - thrombolysis

Pharmaco PostC

drug

- adenosine, NO, K$_{ATP}$ openers
- survival kinases
- mPTP inhibitors, .....
Conclusion

- Trials confined to 2003~2015, no large RCTs these two years.
- According to what we have:
  - Ischemic postconditioning during PCI in ST-segment elevation myocardial infarction appears to be superior to PCI alone in reduction of both myocardial injury or damage and improvement in left ventricular function.
  - The effect seems to be more pronounced when a greater myocardial area is at risk, among young and male patients, and those in whom direct-stenting techniques.
  - No detailed operation methods to achieve Ischemic Postconditioning.
Thank you for your attention!