Glucose-Insulin-Potassium and Tri-Iodothyronine Individually Improve Hemodynamic Performance and Are Associated With Reduced Troponin I Release After On-Pump Coronary Artery Bypass Grafting

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Background—Both glucose-insulin-potassium (GIK) and triiodothyronine (T3) may improve cardiovascular performance after coronary artery surgery (CABG) but their effects have not been directly compared and the effects of combined treatment are unknown.

Methods and Results—In 2 consecutive randomized double-blind placebo-controlled trials, in patients undergoing first time isolated on-pump CABG between January 2000 and September 2004, 440 patients were recruited and randomized to either placebo (5% dextrose) (n=160), GIK (40% dextrose, K+ 100 mmol·L⁻¹, insulin 70 u·L⁻¹) (0.75 mL·kg⁻¹·h⁻¹) (n=157), T3 (0.8 µg·kg⁻¹ followed by 0.113 µg·kg⁻¹·h⁻¹) (n=63) or GIK+T3 (n=60). GIK/placebo therapy was administered from start of operation until 6 hours after removal of aortic cross-clamp (AXC) and T3/placebo was administered for a 6-hour period from removal of AXC. Serial hemodynamic measurements were taken up to 12 hours after removal of AXC and troponin I (cTnI) levels were assayed to 72 hours. Cardiac index (CI) was significantly increased in both the GIK and GIK/T3 group in the first 6 hours compared with placebo (P<0.001 for both) and T3 therapy (P=0.009 and 0.029, respectively). T3 therapy increased CI versus placebo between 6 and 12 hours after AXC removal (P=0.01) but combination therapy did not. Release of cTnI was lower in all treatment groups at 6 and 12 hours after removal of AXC.

Conclusions—Treatment with GIK, T3, and GIK/T3 improves hemodynamic performance and results in reduced cTnI release in patients undergoing on-pump CABG surgery. Combination therapy does not provide added hemodynamic effect. (Circulation. 2006;114[suppl I]:I-245–I-250.)

Key Words: glucose ■ hemodynamics ■ insulin ■ myocardial injury ■ thyroid ■ troponin I

The possible benefits of glucose-insulin-potassium (GIK) and triiodothyronine (T3) for supporting cardiac function after coronary artery bypass graft surgery (CABG) continue to be debated. Perioperative GIK therapy has been used to improve the efficiency of energy utilization, hemodynamic performance, and outcomes in patients undergoing cardiac surgery.¹² Experimental models have demonstrated a direct positive inotropic effect of insulin on postischemic hearts independent of its metabolic effects as well as reducing apoptotic injury to the myocardium when administered at reperfusion.³⁵ Patients undergoing cardiac surgery may develop the euthyroid sick syndrome (ESS). ESS is characterized by a low circulating level of T3 with normal or low levels of thyroid stimulating hormone and thyroxine (T4), dependent on the severity of the disease process.⁶ CABG is associated with a low T3 ESS that may persist for up to 4 days after surgery. These responses may be adaptive or detrimental and whether to treat ESS remains uncertain. Experimental and clinical evidence has demonstrated that T3 can act as an inotrope and vasodilator⁷ improving hemodynamic performances after CABG.

Whether GIK or T3 should be used as adjuncts to standard myocardial protection against ischemic injury remains unclear. The peri-ischemic and postischemic improvements in myocardial function of GIK and T3 occur via purportedly different mechanisms. We investigated the hemodynamic effects of GIK and T3, singly and as combination therapy, and report combined data from 2 consecutive single-center, prospective, double-blind, randomized controlled trials.
### Methods

#### Study Design

We performed 2 consecutive randomized, double-blind, prospective, placebo controlled trials. Initially 280 patients were randomized (1:1) to receive either placebo or GIK therapy (placebo n=142 and GIK n=138). After an interim safety analysis on the first 80 patients, the second study was powered to investigate the hemodynamic effects of placebo, GIK, T3, and combined GIK/T3 therapy (n =160; placebo, n=18; GIK, n =19; T3, n=63; GIK/T3, n =60). The second study was performed with continuous randomization schedules (1:1:3:3) and identical trial protocols. Here we present the results of the entire study population, comprising 440 patients undergoing CABG between January 2000 and September 2004. Inclusion and exclusion criteria are listed in Table 1. The Local Research Ethics Committee approved the studies and all patients gave written informed consent.

Pretrial, computer-generated randomization schedules were constructed, stratified by surgeon and left ventricular (LV) function. Trial investigators, medical and nursing staff were blinded to allocation.

#### Trial Solutions

Trial solutions placebo, GIK, T3, and GIK/T3 solutions were independently prepared in identical volumes in non-identifiable containers, Placebo therapy was 5% dextrose. The GIK solution comprised 40% dextrose containing 70 IU·L⁻¹ human Actrapid insulin (Novo Nordisk A/S, Bagsvaerd, Denmark) and 80 mmol·L⁻¹ potassium chloride. GIK/placebo therapy was administered after anesthetic induction until 6 hours after removal of aortic cross-clamp at 0.113 mL·kg⁻¹·h⁻¹. T3 solution (Goldshield Pharmaceuticals) was administered as an intravenous bolus (0.8 µg·kg⁻¹) at removal of aortic cross-clamp followed by infusion (0.113 µg·kg⁻¹·h⁻¹) for 6 hours. Syringes of placebo/T3 were made up to identical volumes using a prepared nomogram. GIK/T3 patients received both therapies.

#### Surgery, Anesthesia, Cardiopulmonary Bypass and Myocardial Protection

As previously reported, anesthesia, cardiopulmonary bypass, myocardial protection, and surgical techniques were standardized. Anesthesia was induced using intravenous etomidate, fentanyl, and pancuronium and maintained with enflurane, propofol, and alfentanil. Intermittent antegrade cold blood St. Thomas No. 2 cardioplegia (Martindale Pharmaceuticals, Essex, UK) was used for myocardial protection (12 mL·kg⁻¹ for induction and 6 mL·kg⁻¹ at 20-minute intervals). Distal anastomoses were constructed during a single aortic cross-clamp period and proximal anastomoses during partial aortic occlusion. Intravenous glycopyrrolate, atropine, atrial, or dual chamber epicardial pacing were used to achieve a target heart rate (70 to 110 beats per minute).

### Postoperative Management

Dopamine (3 to 10 µg·kg⁻¹·min⁻¹) was commenced if mean arterial pressure (MAP) was <65 mm Hg with a cardiac index (CI) ≤2.2 L·min⁻¹·m⁻² in the presence of a central venous pressure ≥12 mm Hg and/or pulmonary capillary wedge pressure ≥14 mm Hg and heart rate (70 to 110 bpm). Support was also permitted if the operating surgeon identified poor contractility at separation of cardiopulmonary bypass or if marginal hemodynamics were noted by attending physicians. Intravenous vasoconstrictors were used for mean arterial pressure <65 mm Hg, systemic vascular resistance (SVR) <800 dynes·s·cm⁻² and CI >3 L·min⁻¹·m⁻². Bolus phenylephrine was used until the administration of protamine, after which a norepinephrine infusion was substituted. Extubation, intensive therapy unit, and hospital discharge criteria and atrial fibrillation management were standardized.

### Trial Investigations

Hemodynamic studies were performed before infusion, before and 15 minutes after protamine, and 2, 4, 6, 9, and 12 hours after reperfusion. Serum cardiac troponin I (cTnI) samples were collected at baseline, 6, 12, 24, 48, and 72 hours after reperfusion and analyzed in batches using a commercial assay (Bayer Corporation, Tarrytown, NY). Preoperative and postoperative day 1 and day 4 electrocardiograms were obtained.

### End Points

The primary outcome measure was comparison of CI. Secondary outcomes included indexed systemic vascular resistance (SVR), left ventricular stroke work index, cardiac power output (CPO), incidence of myocardial injury on electrocardiograms and enzymatic criteria, episodes of low cardiac output (LCOE), inotrope, and vasoconstrictor requirement. Perioperative myocardial infarction (PMI), assessed by an independent cardiologist, was defined by the presence of new left bundle branch block or new “Q” waves ≥2-mm depth in ≥2 contiguous leads by postoperative day 4. Myocardial injury was defined as PMI and/or a cTnI ≥13.1 ng·mL⁻¹ 12 hours after reperfusion, LCOE, assessed by a blinded committee, was defined as a CI ≤2.1 L·min⁻¹·m⁻² with a central venous pressure ≥12 mm Hg and pulmonary capillary wedge pressure ≥14 mm Hg in the presence of a native or paced synchronized heart rate >70 min⁻¹. The individual total weight indexed dose of inotrope and vasoconstrictor use were calculated.

### Statistical Analysis

The study was powered to detect a change in CI of ≥0.3 L·min⁻¹·m⁻² between groups. Data were analyzed using SPSS version 12.0 software (SPSS, Inc). Categorical data were compared using χ² testing. Continuous data are presented as mean and standard deviation (SD) or median and interquartile range. Normally distributed data were compared using an independent t test. Skewed data were analyzed non-parametrically (Mann-Whitney U or Kruskal-Wallis test). Serial measurements were compared by repeated measures analysis of variance (RMANOVA). Statistical significance was assigned when P≤0.05.

The authors had full access to the data and take responsibility for their integrity. All authors have read and agree to the manuscript as written.

### Results

Enrollment, exclusion, treatment allocation and analysis are summarized in a CONSORT flow diagram (Figure 1). Preoperative clinical details, operative details, and postoperative data are shown in Table 2. There was no significant difference detected in either the incidence of infection, atrial fibrillation, or in-hospital deaths between groups.
Hemodynamics

Throughout the study, heart rate, central venous pressure, and PAWP did not significantly differ. Baseline CI was not different between the 4 groups. CI was higher in the GIK and GIK/T3 groups throughout during the first 6 hours compared with placebo (RMANOVA \( P < 0.001 \) for both). T3 did not improve CI within the first 6 hours but improved over the 6- to 12-hour period (RMANOVA \( P = 0.01 \)) versus placebo (Figure 2). Baseline SVRI was not different. SVRI was lower in the first 6 hours for GIK and GIK/T3 versus placebo (\( P = 0.001 \)) and for GIK/T3 versus T3 (\( P = 0.028 \)) (Figure 3). There was no significant difference in left ventricular stroke work index between groups. However, CPO was significantly higher in all treatment groups versus placebo in the first 6 hours (GIK, \( P = 0.01 \); T3 and GIK/T3, \( P < 0.001 \)) and remained higher in the 6- to 12-hour period for the T3 and GIK/T3 groups (\( P < 0.001 \) and \( P = 0.002 \) respectively). Baseline CPO was not different for GIK (0.66±0.21 W) versus placebo (0.64±0.30 W) but was significantly greater for both T3 (0.81±0.25 W) and GIK/T3 (0.74±0.24 W) groups (\( P < 0.001 \) and \( P = 0.033 \)). Systemic oxygen delivery index was increased at 6 hours in GIK (\( P = 0.033 \)) and GIK/T3 (\( P = 0.038 \)) versus placebo. Systemic oxygen consumption was not different between the groups.

### TABLE 2. Clinical and Operative Details and Postoperative Data

<table>
<thead>
<tr>
<th></th>
<th>Placebo (n=160)</th>
<th>GIK (n=157)</th>
<th>T3 (n=63)</th>
<th>GIK/T3 (n=60)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoperative demographics</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Age, y</td>
<td>63.9 (8.9)</td>
<td>64.5 (8.7)</td>
<td>62.9 (7.9)</td>
<td>67.2 (7.1)</td>
<td>0.025 for T3 vs GIK/T3</td>
</tr>
<tr>
<td>Gender, male (%)</td>
<td>132 (82.5)</td>
<td>137 (87.3)</td>
<td>54 (85.7)</td>
<td>50 (80.3)</td>
<td>0.675</td>
</tr>
<tr>
<td>Euroscore</td>
<td>2 (1–4)</td>
<td>2 (1–4)</td>
<td>2 (1–4)</td>
<td>3 (2–4)</td>
<td>0.139</td>
</tr>
<tr>
<td>Operative details</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-ischemic infusion time (min)</td>
<td>83 (23.8)</td>
<td>81.5 (23.5)</td>
<td>78.6 (28.3)</td>
<td>85.9 (30.4)</td>
<td>0.563</td>
</tr>
<tr>
<td>Cumulative CPB (min)</td>
<td>87.9 (28.3)</td>
<td>88.6 (24.8)</td>
<td>92.5 (36.1)</td>
<td>90.7 (29.1)</td>
<td>0.87</td>
</tr>
<tr>
<td>Cumulative AXC (min)</td>
<td>47.1 (18.3)</td>
<td>49.1 (15.4)</td>
<td>50.2 (18.3)</td>
<td>50.6 (15.5)</td>
<td>0.333</td>
</tr>
<tr>
<td>Distal anastomoses</td>
<td>2.9 (0.7)</td>
<td>2.8 (0.7)</td>
<td>3 (0.8)</td>
<td>3 (0.8)</td>
<td>0.455</td>
</tr>
<tr>
<td>Postoperative data</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Total ITU stay (hours)</td>
<td>28.1 (23.6–50.2)</td>
<td>28.9 (24.0–50.1)</td>
<td>28.4 (23.2–49.6)</td>
<td>41.3 (24.5–60.1)</td>
<td>0.586</td>
</tr>
<tr>
<td>Postoperative stay (days)</td>
<td>7 (6–11)</td>
<td>7 (6–10)</td>
<td>7 (6–10)</td>
<td>8 (7–13)</td>
<td>0.253</td>
</tr>
<tr>
<td>New postoperative atrial fibrillation N (%)</td>
<td>79 (49.4)</td>
<td>90 (57.3)</td>
<td>32 (50.8)</td>
<td>31 (51.7)</td>
<td>0.537</td>
</tr>
<tr>
<td>DO(_2) index at 6 hours after AXC (mL(^2)·min(^{-1})·m(^{-2}))</td>
<td>309.9 (55.9)</td>
<td>395.3 (87.3)</td>
<td>349.6 (86.3)</td>
<td>373.4 (80.8)</td>
<td>&lt;0.04 for placebo vs GIK and GIK/T3</td>
</tr>
<tr>
<td>VO(_2) index at 6 hours post AXC (mL(^2)·min(^{-1})·m(^{-2}))</td>
<td>90 (32.2)</td>
<td>110.3 (30.1)</td>
<td>103.2 (50.2)</td>
<td>105.0 (39.9)</td>
<td>0.652</td>
</tr>
<tr>
<td>Infective episodes, N (%)</td>
<td>35 (22)</td>
<td>44 (28.6)</td>
<td>13 (23.2)</td>
<td>139 (23.5)</td>
<td>0.582</td>
</tr>
<tr>
<td>In-hospital mortality, N (%)</td>
<td>3 (1.9)</td>
<td>3 (1.9)</td>
<td>1 (1.6)</td>
<td>...</td>
<td>0.764</td>
</tr>
</tbody>
</table>

Data are shown as mean (standard deviation), median (interquartile range), or N (%). Systemic oxygen delivery (\( DO_2 \) calculated using formula \( 0.134 \times \text{cardiac index} \times \text{hemoglobin} \times \text{arterial oxygen saturation} \)).

Systemic oxygen consumption (\( VO_2 \): calculated using formula \( 0.134 \times \text{cardiac index} \times \text{hemoglobin} \times \text{arterial oxygen saturations} \) — \( \text{hemoglobin} \times \text{mixed venous oxygen saturation} \)).
Inotrope and Vasoconstrictor Use

Fewer patients in all treatment groups required inotropic support in the first 6 hours after aortic cross-clamp removal compared with placebo (66/160, 41.3%); GIK (28/157 [17.8%]; \textit{P}<0.001), T3 (14/63 [21.0%]; \textit{P}=0.03), and GIK/T3 (9/60 [15.0%]; \textit{P}<0.001). Total dopamine utilization was also less for GIK (\textit{P}=0.002) and for GIK/T3 (\textit{P}=0.007). Total dopamine use was less for T3 versus placebo but did not reach significance (\textit{P}=0.07) (Figure 4). Vasoconstrictor (norepinephrine) therapy was more frequently required in the first 6 hours for both GIK treatment groups compared with placebo (\textit{P}<0.001 and \textit{P}=0.06 for GIK and GIK/T3) and was reduced (but not significantly) for those patients receiving T3 therapy versus placebo. Total norepinephrine use was greater for patients receiving GIK and GIK/T3 (\textit{P}<0.001) but not for the T3 group (\textit{P}=1) (Figure 5) versus placebo.

Myocardial Injury

New PMI on electrocardiograms was not different (placebo, 11/160 [6.9%]; GIK, 6/157 [5.8%]; T3, 3/63 [4.8%]; GIK/T3, 1/60 [1.7%]). Less patients receiving GIK, T3, or GIK/T3 therapy had a cTnI value >13.0 ng⋅mL\(^{-1}\) versus placebo (GIK, 18/132 [13.6%]; placebo, 34/141 [24.1%]; T3, 7/56 [12.5%]; GIK/T3, [19.2%]); however, this was not statistically significant when adjusted for multiple comparisons. The number of LCOEs was lower in all treatment groups (GIK, 33/157 [21.0%] \textit{P}=0.228; GIK/T3, 10/60 [16.7%] \textit{P}=0.186; T3, 13/63 [20.6%] \textit{P}=0.42; versus placebo 50/160 [31.3%]) but did not reach significance on multiple comparison testing. Troponin I levels were lower at 6 hours in all treatment groups (placebo, 5.8 [3.1 to 11.3] ng⋅mL\(^{-1}\); GIK, 5.1 [2.7 to 7.7] ng⋅mL\(^{-1}\) \textit{P}=0.19; T3, 2.74 [1.0 to 5.9] ng⋅mL\(^{-1}\) \textit{P}<0.001; GIK/T3, 3.3 [1.5 to 7.8] ng⋅mL\(^{-1}\) \textit{P}<0.01). cTnI release at 6 hours was also lower in the T3 group versus GIK (\textit{P}=0.013) We have previously reported a reduction in cumulative area under the curve (CAUC) cTnI release with GIK therapy.\(^1\) CAUC analysis up to 48 hours demonstrated less cTnI release for T3 versus placebo (\textit{P}=0.019) (Figure 6). Combination therapy with GIK/T3 did not reduce cTnI release.

Discussion

The ability of any agent to improve hemodynamic performance and reduce inotrope requirement and myocyte injury is of clear
Importance. Low cardiac output, inotropy, and increased biochemical markers of myocyte damage are all adverse prognostic indices after CABG both early and at 2 years.\textsuperscript{9–11} We have previously reported the first 280 patients of these trials comparing GIK and placebo,\textsuperscript{1} confirming the hemodynamic benefits of GIK. This combined report, allowing comparison of GIK, T3, and combination GIK/T3 demonstrates that GIK and T3 individually improve hemodynamic performance but combination therapy does not add further to this effect. Our data demonstrate an improvement in CI and CPO but not left ventricular stroke work index. This suggests that both GIK and T3 act predominantly as vasodilators or inodilators in this clinical setting.

GIK may induce vasodilatation, improve substrate utilization, have direct inotropic effects, reduce circulating free fatty acids, and be anti-apoptotic.\textsuperscript{12–14} Insulin’s inotropic effect may be related to enhanced β-adrenoreceptor sensitivity as well as improved calcium handling properties of the myocyte.\textsuperscript{15,16} T3 acts on the myocyte directly and indirectly and by genomic and nongenomic mechanisms.\textsuperscript{17,18} It is able to lower vascular resistance\textsuperscript{7,19} and have positive effects on the adrenergic and calcium handling properties of the myocyte.\textsuperscript{20,21} Nongenomic actions of T3 are mainly related to contractile relevant ion-channels or pumps\textsuperscript{18} and take place too quickly to be attributed to protein synthesis. Acute T3 administration can increase cardiac output and lower SVR within 3 minutes in healthy euthyroid human subjects.\textsuperscript{19} In our study, the predominant effect of T3 was seen after infusion was complete (6 to 12 hours after reperfusion) and could reflect both genomic and nongenomic modes of action.

In experimentally induced ischemia, GIK has been demonstrated to reduce infarct size.\textsuperscript{22} Insulin at reperfusion may also attenuate infarct size via an anti-apoptotic mechanism and this attenuation is reduced if administration is delayed until after reperfusion.\textsuperscript{5,23} Because we administered GIK before reperfusion in this study, this phenomenon may account for the observation of reduced cTnl. Although posts ischemic T3 improves cardiac function, little is known of the myocardial-protective effect we observed.

Higher circulating levels of cTnl and LCOE are associated with increased morbidity and mortality after CABG\textsuperscript{9,10} and both these end points can be reduced by administration of GIK and T3. We do not, however, have data of longer-term outcomes. The reduction in cTnl was not sustained beyond 12 hours for GIK therapy\textsuperscript{4} and thus may reflect protection from myocyte injury rather than necrosis. Nevertheless, the mech-

![Systemic vascular resistance index (SVRI) from baseline up to 12 hours after removal of aortic cross clamp (AXC). SVRI lower in the first 6 hours for GIK and GIK/T3 vs placebo (P<0.001).](image)

![Mean dopamine use (μg · kg\(^{-1}\)) in first 6 hours after removal of aortic cross-clamp (AXC). GIK and GIK/T3 vs placebo at 6 hours. \(^*P<0.002\) and \(^{**}P=0.007\). T3 therapy vs placebo (P=0.07).](image)

![Cardiac troponin I (cTnl) release after removal of aortic cross-clamp (AXC). T3 therapy significantly reduces area under the curve cTnl release up to 48 hours (P=0.019).](image)
anisms of myocyte protection for both GIK and T3 require further elucidation.

A number of studies have shown a reduction in the incidence of atrial fibrillation with the use of both GIK and T3 as individual therapies.2,4 We did not observe this effect in any of our treatment groups but we withheld reintroduction of beta-blockers until the fourth postoperative day. Thus, an additive protective effect remains possible.

Both GIK and T3 improve hemodynamic performance after CABG.1,7 However, GIK/T3 in combination does not have an additive effect as we initially hypothesized and combined therapy appears inadvisable for postsurgical circulatory management. However, perioperative GIK management may be appropriate in certain high-risk CABG patients and posts ischemic T3 management may have a role in problematic patients not pretreated with GIK.

Neither GIK nor T3 alone or in combination increased any morbid outcome after CABG. Infection rates were identical despite the fact that GIK is associated with a postoperative hyperglycemia.1 Within our relatively low-risk patient group neither metabolic nor hormonal therapy improved intensive therapy unit or postoperative length of stay. This may be caused in part by the detailed investigation and management required for both treatment and placebo patients. In addition treatment patients required more and longer periods of vasoconstrictor therapy.

Conclusions

Our study demonstrates that perioperative GIK and postischemic T3 therapy can be used safely to improve hemodynamics, minimize inotropic support, and reduce myocardial injury after on-pump CABG with moderate hypothermia without increasing whole-body oxygen consumption. It is unclear whether the effects observed are related to changes in contractility or afterload as we primarily used a load-dependent assessment. Combination GIK/T3 therapy does not provide an additive effect.

Further studies are required to establish whether the GIK and T3 change long-term outcome after CABG and to clarify the mechanisms involved in enhancing myocardial protection.

Study Group

Statistical advisor: Peter Nightingale, PhD. Anesthesiologists: David Green, MB, FRCA; Tariq Hoth, MB, FRCA; Peter Jackson, MB, FRCA; John P. Lilley, MB, FRCA; Peter Townsend, MB, FRCA; Laura Tasker, MB, FRCA; Deborah Turfrey, MB, FRCA; Mark Wilkes, MB, FRCA. Data collection: Alison Walker, MSc, RGN.

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Disclosures

None.

References

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