

# Diabetes, Insulin Resistance, and the Metabolic Syndrome in Patients With Acute Myocardial Infarction Without Previously Known Diabetes

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**OBJECTIVE** — Individuals with diabetes have an increased morbidity from acute myocardial infarction (AMI). Based on an oral glucose tolerance test (OGTT), 40–45% of patients with AMI have diabetes. The objective of this study was to characterize the glucometabolic profile of patients with AMI without known diabetes and to see if sustained glucometabolic perturbations are predictable during the hospital phase of the disease.

**RESEARCH DESIGN AND METHODS** — A total of 145 patients with AMI and no previous diagnosis of diabetes were subjected to an OGTT at hospital discharge and 3 months thereafter. Based on the OGTT after 3 months, they were defined as having normal glucose tolerance (NGT;  $n = 50$ ), impaired glucose tolerance (IGT;  $n = 59$ ), or diabetes ( $n = 36$ ). Components of the metabolic syndrome, including insulin resistance assessed by homeostasis model assessment (HOMA-IR), were recorded.

**RESULTS** — Patients with AMI had no changes in insulin resistance from hospital discharge to follow-up. An OGTT and/or a single blood glucose taken 60 min (BG-60) after ingestion of 75 g glucose at hospital discharge were predictors of the outcome of the OGTT at follow-up. With a cutoff value for BG-60 of 8.6 mmol/l, 70% of the patients were correctly predicted as either belonging to the NGT group or the IGT/diabetes group after 3 months. Age, BMI, anti-hypertensive treatment, HbA<sub>1c</sub>, fasting blood glucose, blood lipids, insulin, proinsulin, HOMA-IR, and plasminogen activator inhibitor 1 did not add predictive power.

**CONCLUSIONS** — Patients with AMI and no previous diagnosis of diabetes have no changes in insulin resistance from hospital discharge to a 3-month follow-up. An OGTT or a single BG-60 performed at hospital discharge predicts the diagnosis of IGT or diabetes 3 months thereafter.

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**Abbreviations:** AMI, acute myocardial infarction; BG-60, blood glucose after 60 min; BG-120, blood glucose after 120 min; FBG, fasting blood glucose; HOMA-IR, homeostasis model assessment of insulin resistance; IGT, impaired glucose tolerance; NGT, normal glucose tolerance; OGTT, oral glucose tolerance test; PAI-1, plasminogen activator inhibitor 1; WHO, World Health Organization.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

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Individuals with diabetes have an increased cardiovascular morbidity and mortality (1,2). This risk is already apparent among people with pre-diabetic conditions, such as impaired glucose tolerance (IGT) (3–5). Indeed, a systematic overview suggested an increased risk already at blood glucose levels well below the diabetic threshold (6). In addition, there is a positive relation between blood glucose at hospital admission for an acute myocardial infarction (AMI) and long-term mortality in patients with and without diabetes (7). The Diabetes Insulin Glucose in Acute Myocardial Infarction study suggested that the glucometabolic state at admission, as indicated by blood glucose and HbA<sub>1c</sub>, is a long-term risk marker in patients with diabetes and AMI (8). Similar findings were made among patients without known diabetes (9,10).

Cardiovascular morbidity and mortality are associated with the metabolic syndrome (11,12). According to World Health Organization (WHO) criteria, the metabolic syndrome comprises patients with IGT and diabetes or patients with normal glucose tolerance (NGT) who are insulin resistant and in addition meet at least two of four criteria: 1) hypertension; 2) raised plasma triglycerides and/or low HDL cholesterol; 3) abdominal obesity and/or BMI >30 kg/m<sup>2</sup>; and 4) microalbuminuria (11,13).

Slightly more than 20% of patients with AMI have previously diagnosed diabetes (2,14). However, if an oral glucose tolerance test (OGTT) is performed as a basis for the diagnosis, the prevalence is higher, presumably as high as 40–45% (15). It is also notable that HbA<sub>1c</sub> and fasting blood glucose (FBG) at hospital discharge are independent predictors of abnormal glucose tolerance diagnosed 3 months after the acute event (15). Even taking the limited reproducibility of an OGTT into account, it remains the most valuable tool for early recognition of individuals with diabetes or at increased risk

for diabetes and heart disease (16). The well-known relationship among impaired glucose metabolism, insulin resistance, cardiovascular disease, and the novel finding of an unexpectedly high prevalence of abnormal glucose metabolism in unselected patients with AMI strengthened our interest in further exploring the metabolic profile of this patient group.

The objective of this study was to characterize the glucometabolic profile of patients with AMI without any history of diabetes and to see if sustained glucometabolic perturbations are predictable during the acute phase of the disease.

## RESEARCH DESIGN AND METHODS

### Patients

Patients admitted to the coronary care units of the Karolinska and Västerås Hospitals, Sweden, for AMI between 1 November 1998 and 15 December 2000 were eligible for the study. Patients were included if capillary blood glucose at admission was  $<11.1$  mmol/l, serum creatinine  $<200$   $\mu$ mol/l, and age  $\leq 80$  years. Exclusion criteria were known diabetes and residence outside the catchment areas. Based on an OGTT performed 3 months after hospital discharge, the patients were classified, according to WHO definitions from 1998, as having NGT (FBG  $<6.1$  mmol/l, 120-min blood glucose [BG-120]  $<7.8$  mmol/l), IGT ( $<6.1$  mmol/l, 7.8–11.0 mmol/l), or diabetes ( $\geq 6.1$  mmol/l and/or  $\geq 11.1$  mmol/l) (13). A detailed description of patient recruitment, clinical characteristics of the patients, and the classification of glucose tolerance have been given elsewhere (15). In brief, 181 patients were included, of whom 145 were classified as having either NGT (34%,  $n = 50$ ), IGT (41%,  $n = 59$ ), or manifest diabetes (25%,  $n = 36$ ) (15). The reasons for the withdrawal of 36 patients were death ( $n = 7$ ), cancer ( $n = 3$ ), depression ( $n = 1$ ), coronary artery bypass surgery ( $n = 1$ ), complicated coronary angiography ( $n = 2$ ), alcohol abuse ( $n = 1$ ), stroke ( $n = 1$ ), unstable angina pectoris ( $n = 1$ ), and back problems leading to surgery ( $n = 2$ ). A total of 16 patients refused to undergo the OGTT, and in 1 patient, there were logistic problems. Withdrawn patients ( $n = 36$ ) had a mean age of  $64.6 \pm 10$  years, 25% had a history of previous myocardial infarction, and 28% were on antihypertensive therapy,

and in these respects, they did not differ from the present study population, which is characterized in Table 1.

### Study protocol

Blood glucose was analyzed as soon as possible after admission to the coronary care unit. During hospitalization, HbA<sub>1c</sub> and FBG were measured on the first morning after admission. An OGTT with ingestion of 75 g glucose dissolved in 200 ml water flavored with citric acid was performed according to WHO standards, including an FBG and a blood glucose measurement after 120 min (BG-120) (13), and in addition, a blood glucose value was obtained after 60 min (BG-60). The OGTT was performed immediately before hospital discharge (usually on day 5) and repeated 3 months after hospital discharge, when an HbA<sub>1c</sub> measurement was also obtained.

Plasma insulin and plasma proinsulin were analyzed in fasting samples taken on the first morning after admission and in samples obtained before and 120 min after the ingestion of glucose for the OGTT on day 5 and after 3 months. Plasma concentrations of plasminogen activator inhibitor 1 (PAI-1) were analyzed in fasting samples drawn on day 5 and after 3 months. Triglycerides, total cholesterol, and HDL cholesterol were analyzed in fasting samples taken the first morning after admission. Urine specimens collected in the morning after 3 months were analyzed for albumin and creatinine. BMI was measured at admission and after 3 months.

### Laboratory tests

Blood glucose was analyzed immediately in capillary whole blood by means of the HemoCue procedure (photometer; HemoCue, Ångelholm, Sweden). HbA<sub>1c</sub> was analyzed by high-performance liquid chromatography on capillary blood applied on filter paper with an upper normal limit of 5.3% (Boehringer Mannheim Scandinavian, Bromma, Sweden) (17).

Insulin and intact proinsulin were quantified with enzyme immunoassays from Dako Diagnostics (Cambridgeshire, U.K.). Intra- and interassay coefficients of variation (CVs) for these analyses were 6 and 7% for insulin and 5 and 6% for proinsulin, respectively. Mean  $\pm$  SD and interquartile range values established in our laboratory by analysis of samples from population-based, healthy middle-aged

subjects (40–60 years old, mean age  $53.0 \pm 4.9$  years,  $n = 379$ ) were  $43 \pm 24$  (28–50 pmol/l) for insulin and  $4.5 \pm 3.4$  (2.6–5.4 pmol/l) for proinsulin (for conversion to milliunits per liter, divide by 6.0) (18).

PAI-1 activity was determined with an immunoactivity assay using the Chromolize PAI-1 kit (Biopool, Umeå, Sweden). Intra- and interassay CVs were 5 and 5%, respectively.

Triglycerides, total cholesterol, and urine creatinine analyses were performed on a Victor 950 (Johnson & Johnson Clinical Diagnostics, Rochester, NY). HDL cholesterol was determined enzymatically (Genzyme liquid *N*-geneous HDL-c Assay) on a Mitachi 911 instrument (Boehringer Mannheim, Mannheim, Germany).

Urine albumin was measured on the Image Immunochemistry systems (Beckman Instrument, Fullerton, CA). Results were considered positive if the albumin-to-creatinine ratio was  $\geq 20$  mg/g (13).

Insulin resistance expressed as homeostasis model assessment for insulin resistance (HOMA-IR) was calculated in fasting conditions as plasma insulin (pmol/l)  $\times$  blood glucose (mmol/l)  $\times$  1.13 (correction for plasma glucose)/135 (19). HOMA-IR was used to compare the three groups (NGT, IGT, and diabetes) and to evaluate changes over time. The mean  $\pm$  SD and interquartile range values for HOMA-IR in the reference group were  $1.60 \pm 1.03$  and 0.94–1.87, respectively.

To estimate the prevalence of insulin resistance in our population, we used the WHO cutoff defining insulin resistance as a value of HOMA-IR over the 75th percentile of the general population (in our case, the laboratory reference group). The prevalence of the metabolic syndrome was calculated according to the WHO criteria from 1998 (13) as abnormal glucose tolerance or insulin resistance at 3 months in combination with at least two of the following four criteria: 1) hypertension (treatment at admission or high blood pressure at 3 months); 2) dyslipidemia (treatment at admission or elevated plasma triglycerides and/or low HDL cholesterol in day 2); 3) obesity (BMI  $>30$  kg/m<sup>2</sup>); or 4) microalbuminuria at 3 months.

### Statistical analysis

Values are presented as mean  $\pm$  SD or median (interquartile range). Jonckheere-Terpstra's test was used to test for or-

Table 1—Pertinent clinical characteristics and glucometabolic data of patients with NGT, IGT, and diabetes as seen during the hospital phase and follow-up

Variable	NGT	IGT	Diabetes	P value
<i>n</i>	50	59	36	
Age (years)	61.4 ± 9.3	63.9 ± 9.3	64.7 ± 9.1	0.214
BMI (kg/m <sup>2</sup> )	26 ± 4.5	26 ± 3.9	28 ± 3.4	0.199
Male (%)	74	70	72	0.871
Current smokers (%)	38	34	33	0.802
Family history of type 2 diabetes (%)	18	24	20	0.746
Previous disorders (%)				
Myocardial infarction	10	20	28	0.102
Angina pectoris	26	41	31	0.250
Hypertension (treated)	26	46	19	0.014
Hyperlipidemia (treated)	12	19	14	0.610
Treatment during hospital stay and at discharge (%)				
Thrombolysis	36	43	42	0.740
Aspirin	92	95	94	0.816
β Blockers	86	97	94	0.104
ACE inhibitors	36	41	44	0.716
Statins	62	67	69	0.746
Metabolic parameters				
Blood glucose (mmol/l)				
At admission	6.0 (1.4)	6.2 (1.6)	7.1 (2.2)	0.047
At discharge	5.0 (0.65)	5.1 (0.93)	5.6 (0.83)	0.001
HbA <sub>1c</sub> day 2 (%)	4.7 (0.55)	4.9 (0.65)	5.2 (0.90)	0.001
Insulin (pmol/l)				
Day 2	60.5 (57.5)	55.0 (60.0)	70.0 (67.3)	0.052
Day 5	52.0 (51.0)	52.0 (47.0)	71.6 (47.3)	0.087
3 months	47.0 (42.3)	50.5 (39.3)	70.5 (54.0)	0.027
2-h insulin (pmol/l)				
Day 5	341 (368)	390 (424)	534 (574)	0.026
3 months	178 (190)	360 (323)	560 (543)	0.000
Proinsulin (pmol/l)				
Day 5	5.4 (3.5)	5.8 (3.9)	7.8 (6.4)	0.015
3 months	5.5 (4.5)	5.2 (4.6)	8.4 (6.5)	0.002
Lipids				
Triglycerides day 2 (mmol/l)	1.8 (1.3)	2.0 (1.6)	2.5 (1.4)	0.027
Total cholesterol (mmol/l)	6.1 (1.5)	5.4 (1.2)	6.1 (2.2)	0.735
HDL cholesterol day 2 (mmol/l)	1.2 (0.4)	1.2 (0.4)	1.1 (0.3)	0.05

Data are mean ± SD or median (interquartile range). P values based on Jonckheere-Terpstra test for trend or  $\chi^2$  test.

dered differences between the three groups (NGT, IGT, and diabetes). Changes in metabolic profile over time for all patients were tested with Wilcoxon's signed-rank test after taking the logarithm and patients were excluded list wise. To assess the agreement between OGTT at discharge and after 3 months, we used Cohen's  $\kappa$ . Prediction of abnormal glucose tolerance at 3 months after discharge was obtained by means of a logistic regression model with forward stepwise selection with inclusion of variables at the 5% level and removal at the 10% level at each step. Backward stepwise elimination was also

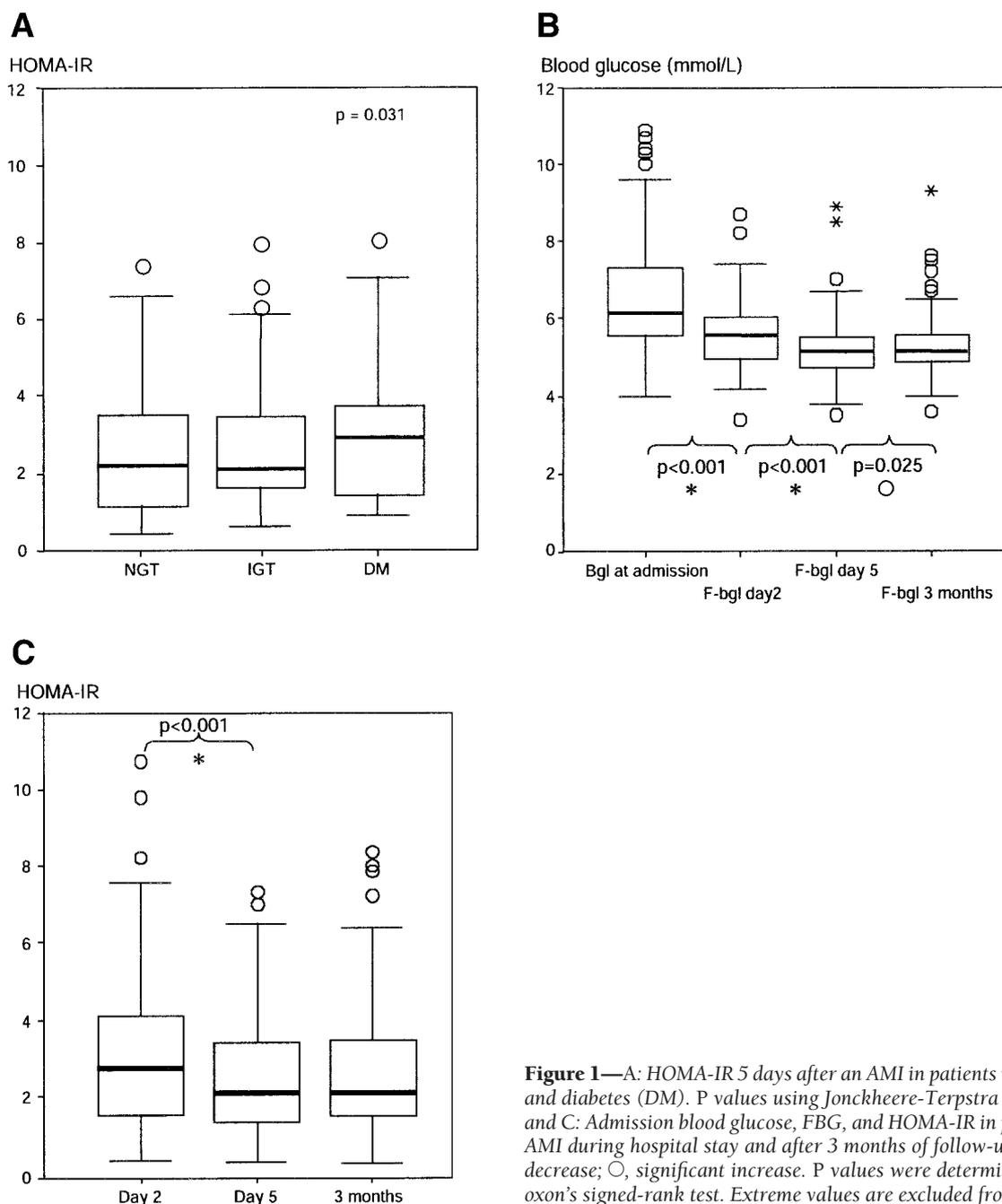
run using the same levels for inclusion/exclusion of variables to ensure that the models were robust. The following variables were tested: age, BMI, and treatment for hypertension; HbA<sub>1c</sub> at admission; classification based on FBG and BG-120 into NGT, IGT, and diabetes with an OGTT performed at discharge; a single FBG and BG-60 at discharge; fasting triglycerides and HDL cholesterol day 2; and fasting insulin, fasting proinsulin, HOMA-IR, and PAI-1 at discharge. The result of the logistic regression was used to classify the patients into NGT and IGT or diabetes. Cross-validation (leaving one out) was

used to correct for the overfit in original estimate when the whole dataset was used.

SPSS version 11.0 was used for all statistical analyses except the logistic regression with cross-validation, which was run in Minitab version 13.32. A two-tailed  $P < 0.05$  was considered statistically significant.

#### Ethical considerations

The regional Ethics Committee of the Karolinska Institute approved the study. All patients gave their written informed consent to participate.



**Figure 1**—A: HOMA-IR 5 days after an AMI in patients with NGT, IGT, and diabetes (DM). P values using Jonckheere-Terpstra test for trend. B and C: Admission blood glucose, FBG, and HOMA-IR in patients with an AMI during hospital stay and after 3 months of follow-up. \*, significant decrease; ○, significant increase. P values were determined using Wilcoxon's signed-rank test. Extreme values are excluded from the box plots.

## RESULTS

### Baseline characteristics

Pertinent clinical characteristics of the three groups of patients (NGT, IGT, and diabetes), as seen during the hospital phase, are presented in Table 1. Treatment for hypertension was most common among those with IGT.

### Metabolic profiles

As could be expected, admission blood glucose, FBG, and HbA<sub>1c</sub> were lowest

among patients with NGT, whereas diabetic patients had the highest values (Table 1). Fasting proinsulin and insulin 2 h after ingestion of 75 g glucose were lowest among patients with NGT, whereas diabetic patients had the highest values at all time points. Triglycerides increased, and HDL cholesterol decreased with the degree of glucose intolerance. Before hospital discharge, 56, 71, and 69% of patients with NGT, IGT, and diabetes, respectively, had an HOMA-IR above the 75th percentile of the reference group defining

insulin resistance. Figure 1 shows that according to the P value ( $P = 0.031$ ) for trend, HOMA-IR (median [interquartile range]) obtained before hospital discharge was lowest in patients with NGT (2.3 [2.5]), medium in IGT (2.1 [2.0]), and highest in patients with diabetes (3.1 [3.2]) (Fig. 1A) and that blood glucose (Fig. 1B) and HOMA-IR (Fig. 1C) for all patients taken together decreased during hospital stay with no further decrease until follow-up.

Plasma PAI-1 activity levels (median

**Table 2—Prevalence of the metabolic syndrome and its different components at follow-up in patients with NGT, IGT, and diabetes according to WHO definitions**

Variable	NGT	IGT	Diabetes	P value
n	50	59	36	
Metabolic syndrome (%)	26	53*	42	0.019
Obesity (BMI >30 kg/m <sup>2</sup> ) (%)	16	19	25	0.578
Dyslipidemia (%)	66	66	86	0.071
Hypertension (%)	26	49*	22	0.008
Microalbuminuria (%)	15	11	9	0.742
Insulin resistance (%)	52	65	86*	0.004

P values based on  $\chi^2$  test; \*indicates differences compared with other groups.

[interquartile range]) were higher among patients with diabetes (11.7 [17.3 IU/l]) compared with patients categorized as having IGT (6.7 [14.0]) and NGT (6.8 [17.4]) on day 5 ( $P = 0.053$ ) and after 3 months (12.0 [21.3], 9.5 [16.5], and 8.2 [13.7], respectively) ( $P = 0.066$ ).

There was a decrease in 2-h insulin from day 5 to 3 months among patients with NGT ( $P < 0.001$ ) but not in patients with IGT or diabetes (Table 1). The corresponding values for mean BG-120 day 5 to 3 months showed a decrease in patients with NGT (7.8 vs. 6.3 mmol/l;  $P < 0.001$ ), no change in patients with IGT (9.2 vs. 9.1 mmol/l;  $P = 0.676$ ), and an increase in patients with diabetes (11.1 vs. 12.4 mmol/l;  $P = 0.013$ ). Mean BG-60 was unchanged from day 5 to 3 months after discharge in all patient groups: NGT (9.8 vs. 9.5 mmol/l;  $P = 0.395$ ), IGT (10.7 vs. 10.2 mmol/l;  $P = 0.079$ ), and diabetes (12.7 vs. 13.5 mmol/l;  $P = 0.145$ ). HbA<sub>1c</sub> was stable from admission to 3 months (90% CI for the difference 0.0–0.15).

**Metabolic syndrome**

As can be seen in Table 2, the metabolic syndrome was most common among patients with IGT, seemingly driven by the high prevalence of hypertension. Insulin resistance was most common in patients with diabetes.

**Prediction of abnormal glucose tolerance**

Forty-nine percent of the OGTT performed at discharge and after 3 months allocated the patients into the same glucose tolerance category (NGT, IGT, or diabetes) on both occasions (Table 3). The agreement between the OGTT classification at discharge and after 3 months could be expressed as  $\kappa = 0.23$  ( $P < 0.001$ ).

As single predictor to classify diabetes and IGT or diabetes after 3 months, BG-60 and BG-120 at day 5 were very similar, and since the correlation between them were satisfactory ( $r = 0.613$ ,  $P < 0.001$ ), it was decided to use only BG-60 in subsequent computations.

The hospital-derived variables that predicted diabetes 3 months thereafter were OGTT ( $P = 0.001$ ) and a single BG-60 ( $P = 0.008$ ). Adding age, BMI, antihypertensive treatment, and HbA<sub>1c</sub> at admission, fasting triglycerides or HDL cholesterol on day 2, and a single FBG, fasting insulin, fasting proinsulin, HOMA-IR, and PAI-1 on day 5 to the logistical regression model did not further improve the predictive value. BG-60 was the only predictive variable ( $P < 0.001$ ) when a similar analysis was performed aiming at the prediction of IGT or diabetes after 3 months. The odds ratio for a 1-mmol/l increase in BG-60 was 1.38 with a 95% CI given by 1.16–1.64. With a cutoff value for BG-60 equal to 8.6 mmol/l, 70% of the patients were correctly predicted as either belonging to the NGT group or the IGT/diabetes group after 3 months, using cross-validation.

**CONCLUSIONS**— This study reveals that patients with an AMI and no previous diagnosis of diabetes have a high

prevalence of insulin resistance both during the hospital stay and 3 months thereafter and that the insulin resistance is unchanged from hospital discharge and during 3 months of follow-up. The 2-h insulin level after 75 g glucose did not change from discharge to follow-up in patients with IGT and diabetes, but there was a decrease in patients with NGT, indicating that these patients had a more adequate insulin response during the acute phase (20).

Accordingly, it should be possible to evaluate the glucometabolic state before hospital discharge and to derive algorithms suitable for this purpose. A BG-60 and classification with an OGTT performed just before hospital discharge were the best ways to predict the diagnosis of diabetes 3 months after hospital discharge, and BG-60 was the only predictor of abnormal glucose tolerance, i.e., IGT or diabetes. We used BG-60 because there was a good correlation to BG-120, as has been reported before (21). An OGTT performed before discharge from hospital after an AMI provides a reliable estimate of diabetes classified at 3 months. Nevertheless, the intraindividual tracking of oral glucose tolerance from discharge to follow-up was fairly poor in our study, like in other studies performing repeated OGTTs (22,23). This is the background for recommending repeated test procedures for the identification of true-risk individuals (22). According to WHO recommendations, a single OGTT is sufficient for epidemiological and screening purposes, but to establish a diagnosis, the test should be repeated (13). Following these recommendations, it would be reasonable to perform repeated OGTTs to settle the diagnosis of IGT or diabetes after an AMI. Still, a single BG-60 taken before hospital discharge is superior to an OGTT to predict IGT or diabetes diagnosed with an OGTT 3 months after discharge.

**Table 3—Results of an OGTT in patients with AMI at discharge from hospital and 3 months thereafter (n = 142)**

	OGTT at discharge	OGTT at 3 months		
		NGT	IGT	Diabetes
NGT	48 (100)	23 (48)	23 (48)	2 (4)
IGT	47 (100)	18 (38)	21 (45)	8 (17)
Diabetes	47 (100)	7 (15)	15 (32)	25 (53)

Data are n (%).

A blood glucose value  $>8.6$  mmol/l 1 h after ingestion of 75 g glucose predicts abnormal glucose tolerance with good sensitivity but lower specificity. This value may, however, serve as a practical, cheap, and reasonably discriminative screening tool in the selection of dysglycemic patients with unstable coronary artery disease for potential and metabolically oriented secondary preventive measures. Interestingly, in our study, more sophisticated and at the same time complex and expensive analyses, such as plasma insulin, proinsulin, or HOMA-IR and PAI-1, did not add any further predictive information. The most likely explanation is the high prevalence of insulin resistance observed across glucose-tolerance categories.

Insulin resistance was calculated from the HOMA-IR index (19). Of note, HOMA-IR might be an imperfect index of insulin resistance in older individuals with abnormal glucose tolerance (24), but because insulin resistance is not included in the definition of the metabolic syndrome in patients with IGT and diabetes (13), this does not affect the prevalence. We used the WHO definition from 1998 for the metabolic syndrome (13) because this was the only one available when the study was designed and because it is a definition commonly used in other studies (11,12), thus enabling comparisons between the present and previous reports. In the Botnia study, in recruiting subjects aged 35–70 years, the prevalence of insulin resistance was 25% in individuals with NGT, ~60% among those with IGT, and almost 90% in those with type 2 diabetes (11). It is notable that the prevalence of insulin resistance was ~29% in male and 35% in female subjects aged 60–69 years with NGT compared with 52% in our postinfarction patients with NGT. It seems that patients with NGT and an acute coronary event are more insulin resistant than individuals with NGT in the general population. Almost 90% of the oldest patients with diabetes in the Botnia study were insulin resistant, a proportion that may be compared with 86% of our patients with newly diagnosed diabetes. Although this comparison indicates a high prevalence of insulin resistance among our patients, an age- and sex-matched control group without known diabetes and coronary heart disease would be required to finally assess the significance of the present observation.

Insulin resistance and dyslipidemia were common irrespective of glucose tolerance, whereas hypertension was less common among patients with diabetes. The latter finding decreased the prevalence of the metabolic syndrome in the diabetic group. Furthermore, in AMI, changes of therapy (i.e., lipid-lowering agents,  $\beta$  blockers, and ACE inhibitors) may influence factors of crucial importance for the metabolic syndrome. Compared with the outcome of the Botnia study, the present prevalence of the metabolic syndrome is twice as high in patients with NGT, similar in those with IGT, and 50% lower in patients with diabetes (11).

In a recent report on patients with AMI without any diagnosis of diabetes, hyperglycemia was an independent predictor of the prognosis after 1 year, whereas BMI or blood lipids were not. It was assumed that blood glucose per se, rather than hyperglycemia as part of a metabolic syndrome, is the link to an adverse prognosis (25). The present observations add further support to this assumption, underline the need for aggressive glucose management, and support the value of a more vigorous screening strategy for the early detection of dysglycemia in patients with acute coronary syndromes.

Patients with AMI and no previous diagnosis of diabetes have a high prevalence of insulin resistance with no change from hospital discharge to 3 months thereafter. Readily available routine tests such as an OGTT or a single blood glucose value taken 60 min after ingestion of 75 g glucose at discharge predict the diagnosis of abnormal glucose tolerance after 3 months. Other components of the metabolic syndrome do not add further predictive value. We recommend improved screening for dysglycemia in patients with AMI during hospital stay as a tool to initiate more aggressive management of dysglycemia, a frequent and presumably major risk factor in this category of patients.

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