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Q1 Position Statement/Recommendations

Practical management of diabetes patients before, during and after surgery: A joint French diabetology and anaesthesiology position statement

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BNP	brain natriuretic peptide	32
CSII	continuous subcutaneous insulin infusion	33
GDM	gestational diabetes mellitus	34
GLP-1	glucagon-like peptide 1	35
GP	general practitioner	36
HF	heart failure	37
ICU	intensive care unit	38
ISPAD	International Society for Pediatric and Adolescent Diabetes	39
IU	international unit(s)	40
IV	intravenous	41
IVII	intravenous insulin infusion	42
SFAR	French Society of Anaesthesia and Resuscitation	43
OAD	oral antidiabetic drug	44
PACU	post-anaesthesia care unit	45
SC	subcutaneous	46
SFAR	French Society of Anaesthesia and Intensive Care	47
SFD	French-Speaking Society of Diabetes	48
SMI	silent myocardial ischaemia	49
T1D	type 1 diabetes	50
T2D	type 2 diabetes	51

Abbreviations

ACR albumin-to-creatinine ratio
CAN cardiac autonomic neuropathy

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53 Introduction

54 Diabetes is a worldwide disorder. Despite huge advances in care
55 management, diabetes patients may nevertheless suffer from
56 cardiovascular and microvascular complications, which may explain
57 their need for surgery more often than non-diabetes patients.
58 Likewise, known or unknown diabetes increases perioperative risks
59 for, for example, the surgical procedures themselves, anaesthetic
60 drugs, fasting, infusions, associated treatment and stress.

61 Therefore, diabetes patients should be given special attention
62 before, during and after surgery. In this context, experts from both
63 the French-Speaking Society of Diabetes (SFD; Société francophone
64 du diabète) and French Society of Anaesthesia and Resuscitation
65 (SFAR; Société française d'anesthésie et de réanimation) met to
66 devise the following guidelines. The full text is published in French
67 and English, with 20 pages created by the Working Group to
68 illustrate how the guidelines apply in daily practice ([http://sfar.
69 org/gestion-du-patient-diabetique/](http://sfar.org/gestion-du-patient-diabetique/)). The present report is a
70 comprehensive support text particularly dedicated to diabetolo-
71 gists not only for their own uses, but also as an educational tool to
72 communicate with anaesthesiologists.

73 Materials and methods

74 A group of experts (six diabetologists: E.C., B.C., S.J., A.M.L., I.T.,
75 P.V.; and four anaesthesiologists: D.B., C.G., C.I., A.O.) met twice a
76 year between November 2014 and April 2017 in Paris, France, to
77 review the literature, and discuss and write the text of the
78 following expert opinion. Due to the limited number of validated
79 studies, recommendations were not graded and the text should be
80 considered expert advice.

81 Results

82 Preoperative management of adult patients with diabetes

83 Identification of patients with diabetes and at risk for stress 84 hyperglycaemia

85 *Known diabetes: different types, main acute complications
86 and treatment (what should anaesthesiologists know?)*
87 (*Appendix A*). Type 2 diabetes (T2D), the most common form of
88 diabetes, is often discovered as an insidious disease because it is
89 asymptomatic at the time of screening high-risk patients [1]. Thus,
90 T2D may be detected when a patient attends hospital for surgery,
91 when chronic complications are already present. The main risk is a
92 hyperosmolar hyperglycaemic state when polyuria/glycosuria and
93 hyperglycaemia (> 1.8 g/L or 10 mmol/L) are not compensated for
94 by polydipsia, or parenteral hydration in an unconscious patient.
95 Patients with T2D may require oral antidiabetic drugs (OADs) such
96 as sulphonylureas, which enhance endogenous insulin secretion
97 and may be responsible for hypoglycaemias. Injectable glucagon-
98 like peptide (GLP)-1 receptor agonists, including those that can be
99 injected weekly, reduce the speed at which the stomach empties
100 after a meal, thereby leading to gastroparesis (*Appendix B*). Insulin
101 may be combined with these drugs.

102 Type 1 diabetes (T1D) is linked to the autoimmune destruction
103 of pancreatic β cells, which synthesize insulin. The two compo-
104 nents for physiological secretion of insulin are then no longer
105 active, namely:

- 107 • 'basal' secretion or 'insulin for daily living', which is continuous
108 over the nycthemeral period and represents approximately 50%
109 of daily requirements;
- 110 • prandial secretion or 'mealtime insulin'.

Substitution of basal insulin should never be stopped, not even
111 in subjects with euglycaemia, due to the major risk of hyper-
112 glycaemia followed by ketosis and diabetic ketoacidosis. In
113 general, patients with T1D are familiar with this rule of survival.
114 Continuous subcutaneous (SC) insulin infusion (CSII) is often used
115 in T1D, as it reproduces basal-bolus delivery through discontinu-
116 ous SC injections of slow-acting and ultrarapid insulin analogues
117 (*Appendix C*). Thus, continuous infusion of a small amount of
118 ultrarapid insulin reproduces basal insulin and is the basal output.
119 'Pancreatic' diabetes secondary to pancreatic disorders is less
120 common, but also presents with severe insulinopenia, with an
121 increased risk of hypoglycaemia because of a simultaneous
122 decrease in glucagon secretion. Other types of diabetes are
123 extremely rare. 124

125 *Stress hyperglycaemia and undiagnosed preexisting dysglycaemia.*

126 *Stress hyperglycaemia.* Surgical procedures and their inherent
127 metabolic effects can induce a stressed state causing perioperative
128 hyperglycaemia, known as 'stress hyperglycaemia'. According to
129 the American Diabetes Association (ADA), this is defined as
130 transient hyperglycaemia in a previously non-diabetic patient
131 during an acute illness or an invasive procedure [1]. It is
132 characterized by plasma glucose levels ≥ 1.8 g/L (10 mmol/L),
133 with levels returning to normal (< 1.26 g/L or 7.0 mmol/L) after
134 removal of the stressor and withdrawal of glucose-lowering
135 treatment in patients previously with an HbA1c < 6.5%. The
136 severity of stress hyperglycaemia depends on the type of surgery,
137 invasiveness of the procedure and its duration [2], with the highest
138 prevalence noted during cardiac surgery. Other risk factors include
139 catecholamine infusion, corticosteroid use, obesity, age, hypother-
140 mia, hypoxia, cirrhosis, trauma, extensive burns and sepsis [2].

141 The main mechanism responsible for perioperative stress
142 hyperglycaemia is peripheral insulin resistance with an increase
143 in endogenous glucose production [3]. In addition, renal reabsorp-
144 tion of glucose is increased and/or glucose clearance decreased.
145 Stress hormones (glucagon, cortisol, catecholamines) and media-
146 tors of inflammation [interleukin (IL)-1, IL-6] released during
147 surgical stress can lead to perioperative insulin resistance. This
148 affects lipid metabolism with increased release of free fatty acids
149 (FFAs), thus further aggravating insulin resistance [3]. Periopera-
150 tive insulin resistance may last for several days after an invasive
151 procedure and initially involves insulin-dependent peripheral
152 tissues [3]. Perioperative blood loss as well as prolonged
153 immobilization both affect glucose metabolism in skeletal muscles
154 and accentuate perioperative insulin resistance. In addition,
155 prolonged perioperative fasting induces a decrease in hepatic
156 glycogen supply, and an increase in neoglucogenesis, and lipid and
157 protein metabolism [2].

158 Hyperglycaemia abolishes ischaemic preconditioning and
159 results in endothelial dysfunction and decreased phagocytic
160 activity of polymorphonuclear neutrophils, while increasing the
161 formation of lesions in a murine blood-brain barrier model of
162 cerebral ischaemia. These deleterious effects of hyperglycaemia
163 are caused by mitochondrial abnormalities in non-insulin-depen-
164 dent cells, where glucose transporters are overexpressed during
165 stress [2]. The increased release of FFAs is potentially harmful to
166 myocardium as they modify protein metabolism, leading to
167 increased protein catabolism and delayed healing. Insulin therapy
168 mitigates the consequences of insulin resistance, such as the
169 postoperative neurohormonal response to stress and perioperative
170 release of FFAs from peripheral tissues during surgery [2].

171 *Undiagnosed preexisting dysglycaemia.* The prevalence of undi-
172 agnosed T2D is high among hospitalized patients due to age and
173 comorbidities. In a study of 40,836 in-hospital patients (19% with
174 known diabetes), 47% underwent perioperative screening of

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