Endokrynologia Polska/Polish Journal of Endocrinology
Tom/Volume 62; Numer/Number 3/2011
ISSN 0423–104X

The dawn phenomenon and the Somogyi effect — two phenomena of morning hyperglycaemia

Zjawisko brzasku i efekt Somogyi — dwa zjawiska porannej hiperglikemii

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Abstract

Morning hyperglycaemia in diabetic subjects may be caused by the dawn phenomenon, or the Somogyi effect, or poor glycaemic control. The dawn phenomenon occurs when endogenous insulin secretion decreases or when the effect of the exogenous insulin administered to the patient the day before disappears, together with a physiological increase in insulin-antagonistic hormones. The Somogyi effect is present in the case of excessive amounts of exogenous insulin. The dawn phenomenon is more common than the Somogyi effect. To diagnose these phenomena, it is useful to measure plasma glucose levels for several nights between 3 a.m. and 5 a.m. or use a continuous glucose monitoring system. Although their treatment differs, the best way of preventing both the dawn phenomenon and the Somogyi effect is an optimal diabetes control with insulin therapy. **(Pol J Endocrinol 2011; 62 (3): 276–283)**

Key words: *morning hyperglycaemia, dawn phenomenon, Somogyi effect*

Streszczenie

Poranna hiperglikemia wśród pacjentów z cukrzycą może być spowodowana zjawiskiem brzasku, efektem Somogyi lub złą kontrolą glikemii. Zjawisko brzasku pojawia się, gdy zmniejsza się wydzielanie endogennej insuliny lub gdy skończy się działanie podanej pacjentowi egzogennej insuliny łącznie z fizjologicznym wzrostem sekrecji hormonów antagonistycznych. Natomiast efekt Somogyi obecny jest w przypadku nadmiernej dawki insuliny egzogennej. Zjawisko brzasku występuje częściej niż efekt Somogyi. Do rozpoznania obu tych zjawisk użyteczny jest pomiar stężenia glukozy we krwi między godziną 3. a 5. rano przez kilka kolejnych nocy lub użycie systemu ciągłego monitorowania stężenia glukozy. Mimo że leczenie obu zjawisk różni się, najlepszym sposobem zapobiegania im jest właściwe kontrolowanie cukrzycy insulinoterapią. **(Endokrynol Pol 2011; 62 (3): 276–283)**

Słowa kluczowe: *poranna hiperglikemia, zjawisko brzasku, efekt Somogyi*

Introduction

Diabetes mellitus has been called the epidemic of the 21st century. The number of people suffering from diabetes is systematically growing. International Diabetes Federation data presents the current scale of the diabetes problem. It is estimated that approximately 285 million people worldwide had diabetes in 2010, and by 2030 some 438 million people are projected to do so [1].

Diabetes mellitus is divided into several types. Type 1 diabetes, formerly called insulin-dependent diabetes mellitus, is related to inadequate function of the beta cells of pancreatic islets resulting from the lack of endogenous insulin as a consequence of autoimmune process. Type 2 diabetes, formerly called noninsulin-dependent diabetes mellitus, is related to diminished insulin sensitivity in peripheral tissues, mainly in the muscles and adipocytes. In this type of diabetes, beta cells secrete insulin, but the amount of endogenous insulin is insufficient for tissues' demands. Other types of diabetes are: gestational diabetes and specific types of diabetes resulting from genetic syndromes, surgery, drugs, pancreatic diseases, endocrinopathies, infections, and other illnesses.

The main symptom of all types of diabetes is hyperglycaemia, defined as fasting plasma glucose over 125 mg/dl, casual plasma glucose over 200 mg/dl (on at least two occasions) or 2-h post-glucose load plasma glucose above 200 mg/dl in a standard oral glucose tolerance test (OGTT) [2].

The aim of diabetes treatment is to maintain pre-prandial capillary plasma glucose between 70 and 130 mg/dl and the post-prandial peak under 180 mg/dl for non-pregnant diabetic adults, and ≤ 95 mg/dl (fasting), ≤ 140 mg/dl (1-h post-meal) and ≤ 120 mg/dl (2-h

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post-meal) plasma glucose for pregnant women with diabetes [2].

In some patients, the plasma glucose level in the morning is higher than recommended. The problem of morning hyperglycaemia in a population of diabetic patients is clinically relevant. Therefore, in this paper we decided to focus our attention on this question, reviewing two phenomena of morning hyperglycaemia on the basis of the research.

Physiology and pathophysiology of insulin secretion and glucose metabolism

Glucose is the main energetic substrate in a human organism. Plasma glucose concentration, which in a healthy population ranges between 70 and 130 mg/dl [2], is regulated by many hormones (Table I) [3]. The only hormone which decreases the plasma glucose level is the pancreatic beta cells product, insulin.

Insulin secretion is constant at low level, with post-prandial peaks, and it has its own daily secretion rhythm, with the peak in the early morning and the trough in the evening [4]. Also, hormones with antagonistic action to insulin have their own characteristic circadian rhythms (Figure 1) [5]. The peak of cortisol secretion occurs between 4 a.m. and 5 a.m. and again between 6 a.m. and 9 a.m. [6], while the lowest levels are observed around midnight [7]. Although growth hormone (GH) is secreted in a pulsatile manner throughout the day, nearly 50% of GH secretion takes place during the third and fourth phase of the NREM sleep [8], while the largest secretion surges of this hormone occur with the onset of deep sleep [5]. Daily insulin secretion rhythm is shown in Figure 2 [9]. Insulin synthesis and release is influenced by high plasma glucose levels, which are the strongest stimulus for insulin secretion. The basic glucose concentration which causes the beginning of insulin secretion is 80–100 mg/dl [10]. Insulin deficiency is divided into two types: absolute, related to the absence of insulin release, and relative, associated with insulin resistance. The first leads to rebound hyperglycaemia after hypoglycaemia, induced by excessive doses of exogenous insulin used in the treatment of type 1 diabetes. Hyperglycaemia may also result from insulin resistance, despite hyperinsulinaemia and increased levels of insulin-antagonistic hormones, normally counteracted by proper insulin secretion in type 2 diabetes. Bowen and Moorhouse [11] measured fasting plasma glucose level in the morning and in the afternoon in a group of people without diabetes and in untreated subjects with type 2 diabetes. The results show that in non-diabetic patients, fasting plasma glucose concentration was within normal limits, and higher in the afternoon than in the morning. In turn, in diabetic individuals, fasting plasma glucose concentration was not only abnormally high, but was significantly higher in the morning than in the afternoon. This shows the association between diabetes and morning hyperglycaemia, often observed in a physician's practice. This means it is well worth becoming acquainted with phenomena which are responsible for morning hyperglycaemia so as to properly diagnose and treat such patients.

Fasting hyperglycaemia is a phenomenon observed in almost all individuals with diabetes, and may be caused by a dysregulation of the normal circadian hormonal patterns resulting in increased hepatic glucose output [12]. Morning hyperglycaemia can have three causes the dawn phenomenon, the Somogyi effect, and insufficient insulin supply [13]. Moreover, morning hyperglycaemia can cause insulin resistance, as has been proved by Fowelin et al. [14]. Increased insulin resistance may lead to a further deterioration of glucose tolerance, causing the progression of diabetes and finally resulting in micro- and macroangiopathic diabetic complications.

Table 1. *Blood glucose level regulating hormones*
Tabela 1. *Hormony regulujące stężenie glukozy we krwi*

Hormones increasing the blood glucose level	Hormones decreasing the blood glucose level
Growth hormone	Insulin
Catecholamines	Glucose dependent insulinotropic polypeptide (GIP)*
Glucagon	Glucagon like peptide-1 (GLP-1)*
Glucocorticosteroids	
Thyroid hormones (T_3, T_4)	
Pancreatic peptide (PP)	

*These two hormones act by triggering insulin release immediately after food ingestion, inhibiting glucagon secretion, delaying stomach emptying, and suppressing hunger sensation [3]

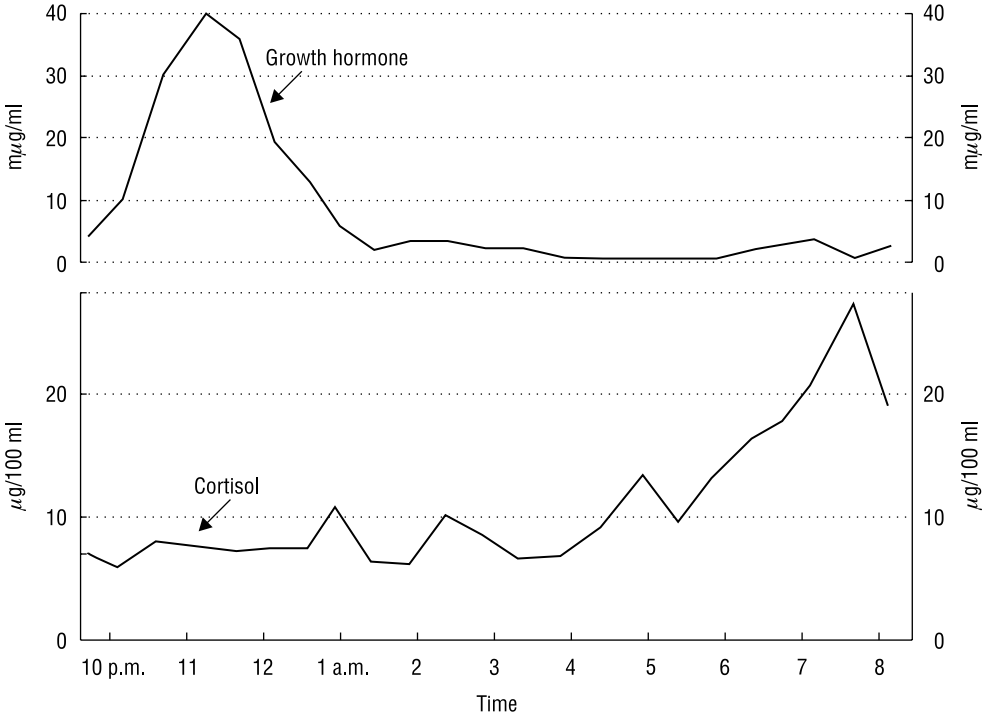

Figure 1. *Growth hormone and cortisol daily biorhythm*
Rycina 1. *Rytm dobowy hormonu wzrostu i kotryzolu*

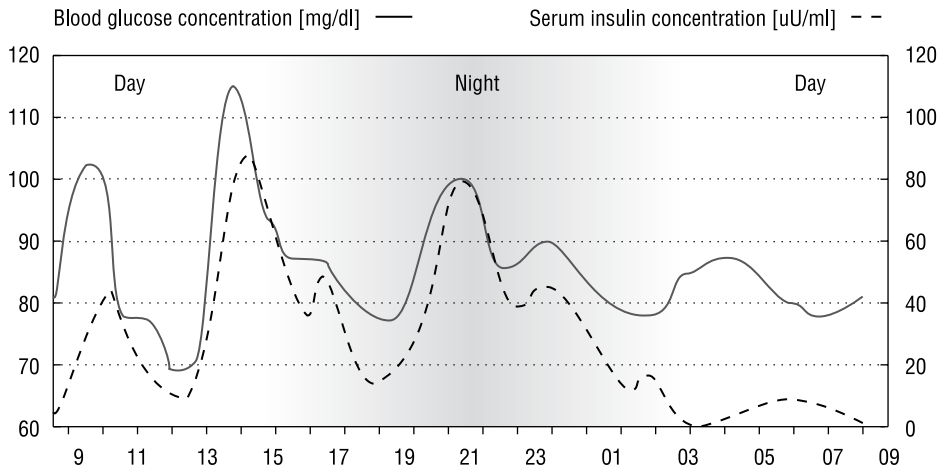

Figure 2. *Insulin and glucose daily biorhythm*
Rycina 2. *Rytm dobowy insuliny i glukozy*

Dawn phenomenon

Definition and pathogenesis

Recurring abnormally high plasma glucose levels in the morning before breakfast are commonly called the dawn phenomenon. According to the daily insulin secretion profile set out in Figure 2 [9], the dawn phenomenon can be divided into two types: physiological and pathological. Both types occur at the same time of the day i.e. between 3 a.m. and 5 a.m., but differ in the value of plasma glucose levels. The physiological dawn phenomenon is associated with a natural decrease of insulin secretion between 3 a.m. and 5 a.m. combined with elevation of blood glucose level remaining up to standard. This decrease of insulin secretion unblocks the secretion of insulin-antagonistic hormones with hyperglycaemic properties, particularly GH. The morning plasma glucose level growth in non-diabetic people

with undisturbed insulin secretion is compensated by an additional burst of insulin. In turn, diabetic patients may experience the pathological dawn phenomenon, where the morning plasma glucose level is abnormally high due to insulin secretion disturbances plus the effects of nocturnal GH secretion [15].

The dawn phenomenon is a combination of an initial decrease in insulin requirements between midnight and 3 a.m., followed by an increase in the insulin needs between approximately 5 a.m. and 8 a.m. [16]. Therefore, the dawn phenomenon can occur among both people with type 1 and type 2 diabetes mellitus with deterioration of beta cells function and without insulin therapy [17–19]. The decrease of endogenous insulin causes the lack of sufficient repression of insulin-antagonistic hormones secretion, mainly GH [20], cortisol and catecholamines [21] and leads to hyperglycaemia. Because of the impaired function of pancreas beta cells, there is also an insufficient insulin secretion in response to hyperglycaemia which causes long-acting hyperglycaemia, detected by patients after awakening as the dawn phenomenon. Likewise, the dawn phenomenon occurs when the action of exogenous insulin administered to the patient the previous day is running out, and at the same time overlapping physiological growth of insulin-antagonistic hormones is observed. There is also the phenomenon called 'extended dawn phenomenon' [22]. This is seen when the high morning glucose level remains high until mid-morning. The cause of extended dawn phenomenon can be too many carbohydrates in the breakfast meal, or the pathologically extended stage of growth hormone secretion which is not repressed by hyperglycaemia, seen more often among diabetic patients [14].

Incidence and diagnosis

The dawn phenomenon occurs in both types of diabetes [23]. However, the incidence is different depending on the type of diabetes and the age of the patient. The dawn phenomenon is rarest among adult type 2 diabetic patients, affecting nearly 3% of this population [19]. Among adults with type 1 diabetes, and especially among children and adolescents, the dawn phenomenon is significantly more frequent, affecting 24.1% of adult type 1 [13] and 27.4% of young type 1 [24] diabetic patients. According to Carroll et al. [25], the dawn phenomenon is experienced by approximately 54% of patients with type 1 diabetes, and by 55% of patients with type 2 diabetes. There is a need for more research into this issue. In type 2 diabetic patients, the three different treatment regimens (glipizide, bedtime NPH insulin, intensive insulin therapy with multiple injections of regular insulin) do not affect the frequency of occurrence of the dawn phenomenon [19].

It is necessary to diagnose the dawn phenomenon correctly so as to be able to counteract and treat this phenomenon. There are two methods of diagnosis: the first is to measure, for the following several nights, plasma glucose concentration between 3 a.m. and 5 a.m. [13, 17, 21]. The virtue of this method is that it helps to distinguish between the dawn phenomenon and the Somogyi effect. The confirmation of the dawn phenomenon is the normal or high plasma glucose level during the nocturnal plasma glucose measurement. In turn, low plasma glucose concentration at this time suggests the presence of the Somogyi effect.

The second method of diagnosing the dawn phenomenon is checking plasma glucose level by a Continuous Glucose Monitoring System (CGMS). This is a small device, which throughout the whole day detects the glucose concentration in the intercellular compartment through a small sensor implanted subcutaneously. After detection, the data is processed by a computer. Thanks to CGMS data, all glucose concentration abnormalities can be found more easily, such as the dawn phenomenon, and proper therapy can be introduced [26].

Prevention and treatment

There are plenty of ways to minimise the influence of the dawn phenomenon on plasma glucose concentration. The dawn phenomenon may be prevented by increasing evening physical activity, increasing the protein-to-carbohydrate ratio in the last meal of the day, and by breakfast consumption even though the dawn phenomenon is suspected or found (this regimen decreases insulin-antagonistic hormones secretion) [17, 21, 27]. In some cases, individual diet modification and/or oral anti-diabetic agent therapy may be enough to reduce fasting hyperglycaemia [12]. Although an increase in the bedtime doses of hypoglycaemic agents with night-time peaks of action may correct early morning hyperglycaemia, it is sometimes associated with undesirable nocturnal hypoglycaemia [25]. On the other hand, for patients who are obliged to replace oral anti-diabetic drugs with exogenous insulin, a solution may be using an insulin pump instead of multiple insulin injections, helping to control plasma glucose level at night. Moreover, the continuous subcutaneous insulin infusion (CSII) by insulin pump imitates the natural daily insulin secretion rhythm [28], decreases the incidence of dawn phenomenon [28, 29] and maintains better plasma glucose profile [30, 31]. Among patients treated with conventional optimised insulin therapy (OCT) it is better to replace long-acting insulin NPH with long-acting insulin analogues e.g. glargine [32]. According to Pesić et al. [33], fasting plasma glucose, HbA1c and frequency of hypoglycaemic events are lower during glargine therapy than conventional inten-

sive insulin therapy with NPH insulin. Furthermore, the dawn phenomenon is an indication for using glargine in standard anti-diabetic therapy [34]. Successful insulinisation appears to minimise the effects of the dawn phenomenon [25].

There is a correlation between the HbA1c value and the degree of dawn phenomenon control. According to Monnier et al. [22], the dawn phenomenon is controlled by oral anti-diabetic drugs, including sulfonylurea and metformin, and by a diet, provided the level of HbA_{1c} is lower than 7%. When HbA_{1c} exceeds 7%, diet and oral anti-diabetic drugs no longer normalise the morning plasma glucose concentration. As the dawn phenomenon is more frequent among patients with poor glycaemic control, [35], intensification of the anti-diabetic treatment allows a reduction in the incidence of the dawn phenomenon.

The Somogyi effect

Definition and pathogenesis

Although the existence and pathogenesis of the dawn phenomenon are indisputable in scientific society, the Somogyi effect is still a matter of debate. The name of the phenomenon derives from the surname of Austro-Hungarian scientist Michael Somogyi. In 1949, during the ACS meeting in Atlantic City, he made a speech about insulin's influence on diabetic patients. On the basis of his investigations, he concluded that people who had been given too large doses of insulin became "actually victims of chronic insulin poisoning" [36]. In his paper entitled: "Exacerbation of diabetes by excess insulin action", Somogyi developed his initial statement by adding a conclusion that too much insulin led through hypoglycaemia to hyperglycaemia [37]. Moreover, Somogyi created a hypothesis that hyperglycaemia after hypoglycaemia is a result of the insulin-antagonistic action of some hormones, especially those belonging to the hypothalamic-pituitary-adrenal axis [37]. The risk of occurrence of the Somogyi effect is also increased by using NPH insulin in diabetes therapy, which can be connected with the evident peak of its concentration taking place 4-5 hours after evening injection and its intermediate duration of action (10–16 hours) [38]. According to Raskin [39], asymptomatic nocturnal hypoglycaemia is common, but subsequent fasting hyperglycaemia is not necessarily the result of "rebound", because the present therapeutic regimens of NPH/Lente insulin given at suppertime cause overnight hyperinsulinaemia. Excessive fasting hyperglycaemia rarely follows nocturnal hypoglycaemia, except when excessive glucose is ingested to correct hypoglycaemia [16]. Moreover, nocturnal hypoglycaemia correlates with falling plasma insulin levels rather than with increasing concentrations of counterregulatory hormones, whose secretion is often disturbed [39]. Such deterioration in insulin-antagonistic hormone levels during asymptomatic nocturnal hypoglycaemia was proved by Jones et al. [40] in a group of patients with type 1 diabetes and without diabetes. Among patients with diabetes, plasma epinephrine and norepinephrine responses to hypoglycaemia were blunted or reduced when they were asleep. The patients' plasma cortisol concentrations did not increase, while GH concentrations increased slightly. This defective glucose counterregulation is associated with substantially increased rates of severe iatrogenic hypoglycaemia in people with type 1 diabetes [41].

Somogyi's hypothesis has been tested by numerous scientists, who have accepted or rejected its existence. Research supporting the existence of the Somogyi effect includes the experiment carried out by Matyka et al. [42]. Their study involved two groups of 29 type 1 diabetic and non-diabetic children. The aim of the study was to determine the response of insulin-antagonistic hormones to hypoglycaemia. The results revealed a small increase of plasma GH and a rise of plasma epinephrine during nightly hypoglycaemia compared to a night without hypoglycaemia. The levels of norepinephrine, cortisol and glucagon were the same after a night with or without hypoglycaemia. Furthermore, the above mentioned study found a significant increase in plasma insulin concentration between 11 p.m. and 3 a.m. among type 1 diabetic children, but not in non-diabetic children [42]. Perriello et al. [43] showed that fasting and post-breakfast plasma glucose levels were significantly higher after nocturnal hypoglycaemia than when hypoglycaemia was prevented. Moreover, fasting levels of plasma glucose in their study correlated directly with overnight plasma levels of epinephrine, GH and cortisol. Bolli et al. [44] drew similar conclusions, and indicated that hypoglycaemia can cause rebound hyperglycaemia in the absence of insulin waning in patients with type 1 diabetes, and that this results primarily from an excessive increase in glucose production due to activation of glucose counterregulatory systems. In another study [45], the authors observed the presence of the relationship between the Somogyi effect and the exuberant counterregulatory release of GH caused by nocturnal hypoglycaemia among patients with type 1 diabetes.

Tordjman et al. [46] assessed whether nocturnal hypoglycaemia actually caused morning hyperglycaemia among type 1 diabetic patients. Their results contradict the existence of the Somogyi effect, as the presence of nightly hypoglycaemia was not followed by the development of morning hyperglycaemia. Moreover, the morning plasma glucose positively correlated with

the nocturnal plasma glucose levels. Similar studies, but using CGMS, conducted by Guillod et al. [47] and Høi-Hansen et al. [48], also proved that morning hyperglycaemia was not related to nightly hypoglycaemia. Furthermore, Monnier et al. [49] demonstrated that nocturnal hypoglycaemia resulted in hyperglycaemia the next day, if it was observed before noon, but not in the early morning, which is called 'mid-morning hyperglycaemia'.

Somogyi's next hypothesis investigated by scientists was the relationship between morning hyperglycaemia and the levels of hormones with antagonistic action to insulin as a result of nightly hypoglycaemia. Gale et al. [50] examined two groups of 15 patients with and without episodes of nocturnal hypoglycaemia for increased plasma level of GH, cortisol and glucagon during the morning hyperglycaemia. The results showed no increase in insulin-antagonistic hormone levels among patients with hyperglycaemia and a negative correlation between the plasma glucose concentration and the plasma free insulin concentration. This proves that during morning hyperglycaemia, a decrease in plasma insulin levels is observed. This suggests that hyperglycaemia may be the result of insulin dropping late at night, rather than an increase of insulin-antagonistic hormones. Similar conclusions were drawn also by Fowelin et al. [14] and Hirsch et al. [51].

According to above-cited data, the existence of the Somogyi effect has not been definitively proven. However, science supposes it to exist, and it is supposed to be present in clinical practice among large number of patients with morning hyperglycaemia. With regard to the impact of the excessive dose of insulin on the Somogyi effect, it is highly probable that this phenomenon can occur not only among patients with type 1 diabetes, but also among patients with type 2 and secondary types of diabetes, provided patients have been intensively treated with insulin.

Incidence and diagnosis

The population's prevalence of the Somogyi effect is assessed to be 12.6% according to Mozersky et al. [13]. The study performed by Winter [45] showed that asymptomatic nocturnal hypoglycaemia combined with rebound hyperglycaemia was present in 18% of investigated patients with type 1 diabetes. However, the incidence of the Somogyi effect according to Cohen et al. [52] is higher, as it is present in about 67% of the population of diabetic patients.

The diagnosis of the Somogyi effect is based on plasma glucose concentration measurement. It is conducted between 3 a.m. and 5 a.m., particularly among patients with type 1 diabetes [13]. If the plasma glucose level is low, it suggests the Somogyi effect. However, a high or normal level of plasma glucose indicates instead the dawn phenomenon [17, 21]. Another method of diagnosing this phenomenon is using CGMS to monitor plasma glucose levels [26, 27, 35, 47]. The Somogyi effect's glycaemic threshold value can be established at the arterialized venous glucose level between 3.8 mmol/l (when glucose counterregulatory hormone glucagon, epinephrine, growth hormone and cortisol secretion increases) and 3.0 mmol/l (when the first symptoms of hypoglycaemia are observed) [41]. This glycaemic threshold value depends on increased rates of severe iatrogenic hypoglycaemia [41] and relates to patients with type 1 diabetes as well as ones with type 2 diabetes on intensive insulin therapy.

Prevention and treatment

Given that the main cause of the Somogyi effect is an excessive dose of insulin, the first step to prevent it should be to modify insulin dosage and, in the case of a patient treated with NPH, replace insulin with a long-acting peakless analogue, e.g. glargine or detemir [33, 53, 54]. According to Tone et al. [55], the frequency of hypoglycaemia was decreased in type 1 diabetes and there was no change in type 2 diabetes after switching insulin glargine to insulin detemir. NPH insulin is an insulin with an intermediate duration of action. It has an onset of action about 36–60 minutes following injection, the peak effect being observed 4–5 hours after injection, and the action lasting for 10–16 hours [38]. Insulin glargine has a slower onset of action (1–2 hours) than NPH insulin [38] and a comparable duration of action (24 hours) to detemir (24 hours) [56]. The absence of the visible peak of insulin glargine [38] and detemir action markedly reduced the risk of hypoglycaemia and should be a standard of dawn phenomenon and Somogyi effect treatment.

A good solution for patients with the Somogyi effect is to administer insulin using continuous subcutaneous insulin infusion (CSII) by insulin pump which minimises the risk of nocturnal hypoglycaemia [28, 30, 54]. To prevent the occurrence of this phenomenon, the proportion of proteins to carbohydrates in the last meal of the day should be increased [17, 27] and a patient should go to bed with a higher level of plasma glucose than usual [27].

Intermediate-acting insulin administered at suppertime easily results in hypoglycaemia in the early evening hours and hyperglycaemia in the fasting state [16]. Therefore, in our opinion the best way to prevent and treat the Somogyi effect and the dawn phenomenon would be modification of the insulin administration hours, and replacement of the NPH by glargine or another long-acting insulin. They can be injected

Table II. *Comparison of the dawn phenomenon and the Somogyi effect*
Tabela II. *Porównanie zjawiska brzasku i efektu Somogyi*

Feature comparing	The dawn phenomenon	The Somogyi effect
Definition	Recurring early morning hyperglycaemia	Early morning hyperglycaemia due to treatment with excessive amount of exogenous insulin
Cause	Decrease of insulin secretion between 3a.m. and 5a.m. and increase of insulin-antagonistic hormones	Nocturnal hypoglycemia due to excessive dose of insulin and the next early morning hyperglycemia due to increase of insulin-antagonistic hormones
Occurrence	Type 1 diabetic patients Type 2 diabetic patients with no insulin therapy	Type 1 diabetic patients Type 2 diabetic patients with insulin therapy
Incidence	Type 1 diabetic children — 27.4% Type 1 diabetic adults — 24.1% Type 2 diabetic adults — 3% Type 1 diabetes generally — 54% Type 2 diabetes generally — 55%	Type 1 and 2 diabetic patients — 12.6–67% Type 1 diabetic patients — 18%
Diagnosis	Measurement of the plasma glucose concentration between 3 a.m. and 5 a.m. during next several nights CGMS The confirmative result: high/normal plasma glucose level	Measurement of the plasma glucose concentration between 3 a.m. and 5 a.m. during next several nights CGMS The confirmative result: low plasma glucose level
Prevention/ /treatment	Increase evening physical activity Increase amount of protein to carbohydrates in the last meal of the day Eat breakfast even though the dawn phenomenon is presented Individual diet modification only if HbA_{1c} is lower than 7% Antidiabetic oral agent therapy only if HbA_{1c} is lower than 7% Use an insulin pump Long-acting insulin analogues like glargine instead of NPH insulin	Modify insulin dosage Long-acting insulin analogues like glargine instead of NPH insulin Use an insulin pump More protein than carbohydrates in the last meal of the day Go to bed with higher level of plasma glucose than usual

once daily between 6 a.m. and 9 a.m. to minimise the incidence of nocturnal hypoglycaemia in the Somogyi effect, and between 6 p.m. and 9 p.m. to prevent the incidence of the dawn phenomenon.

Conclusion

Both the dawn phenomenon and the Somogyi effect are associated with the development of morning hyperglycaemia, but the pathological mechanisms responsible for these effects are different (Table II). The dawn phenomenon is more common than the Somogyi effect. The former occurs when endogenous insulin secretion decreases or when the dose of exogenous insulin administered the previous day is either too small or disappears too soon, especially if this decrease is associated with an increase of insulin-antagonistic hormones.

The latter, the Somogyi effect, is present when the patient is treated with an excessive amount of exogenous insulin. According to research, the dawn phenomenon is more frequent among type 1 diabetic children, even though it is also presented in type 1 and 2 diabetic adults. On the other hand, the existence of the Somogyi effect is not completely proven and therefore its incidence is difficult to assess.

Probably, when early methods of insulin therapy were administered to patients, the Somogyi effect was commonly seen due to excessive doses of insulin. But these days, when insulin therapy has become more advanced and sophisticated, the Somogyi effect appears rarely.

Both phenomena are easy diagnosed and there are some methods of treatment and prevention.

The best way of preventing these phenomena is good diabetes mellitus control with appropriate insulin therapy. As the pathogenesis of these phenomena is still uncertain, further research is warranted, which may help to reduce, or at least seriously limit, the risk of the dawn phenomenon and/or the Somogyi effect.

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