

# Hyperinsulinemia and Tinnitus: A Historical Cohort

Luiz Lavinsky,<sup>1,2</sup> Marcelo W. Oliveira,<sup>2</sup> Humberto J.C. Bassanesi,<sup>2</sup>  
Cíntia D'Avila,<sup>3</sup> and Michelle Lavinsky<sup>1</sup>

<sup>1</sup>Research Center, Hospital de Clínicas de Porto Alegre, <sup>2</sup>School of Medicine, Universidade Federal do Rio Grande do Sul, and <sup>3</sup>Clínica Lavinsky, Porto Alegre, Brazil

**Abstract:** Tinnitus affects millions of people worldwide, and it signals the presence of several underlying diseases, including hyperinsulinemia. The aim of this study was to evaluate the response to dietary treatment in 80 patients with associated tinnitus and hyperinsulinemia. On the basis of data obtained by a questionnaire, two groups were established: One included patients who followed the prescribed diet; the other group included patients who did not comply with the treatment. The likelihood of improving tinnitus symptoms was five-fold higher in hyperinsulinemic patients who followed the diet than in those who did not (relative risk [RR], 5.34; 95% confidence interval [CI], 1.85–15.37,  $p < .05$ ). In addition, resolution of tinnitus was reported by 15% of the patients who followed the diet, as compared to 0% of those who did not. These findings underscore the importance of including hyperinsulinemia in the routine diagnostic investigation of patients with tinnitus regardless of whether associated with neurosensory, dysacusis or vertigo (or both).

**Key Words:** glycemic disorders; hyperinsulinemia; metabolic disorders; tinnitus

Tinnitus affects millions of individuals around the world. Statistics from North America show that 10–15% of the population presents with tinnitus; the direct and indirect social and economic consequences of tinnitus render it a public health issue [1–3]. Tinnitus is a symptom, not a disease, although it reflects an underlying abnormality. A range of clinical entities may be associated with tinnitus, including diseases of an otological, neurological, endocrinological-metabolic, vascular, dental, and even psychological nature [4,5]. As a result, the determination of its etiology requires detailed and careful investigation.

The inner ear is virtually without energy reserves. Its metabolism depends directly on its supply of oxygen and glucose from perfusion. Alterations in blood flow or metabolism, therefore, have great potential for disturbing the homeostasis of the inner ear [6]. Several clinical studies show a relationship between metabolic disorders—especially those involving carbohydrates,

lipids, and thyroid hormones—and potential involvement of the inner ear [7–9]. In fact, Mangabeira Albernaz and Fukuda [10] have demonstrated that 82% of patients with tinnitus and a clinical history suggestive of dysglycemia presented abnormal 5-hour glycemic or insulinemic curves. Spencer showed that patients with tinnitus and metabolic disorders experienced improvement of symptoms after a high-fiber diet, a finding also described by Kraft [11].

Hyperinsulinemia is defined in the presence of fasting insulin levels greater than 30  $\mu\text{U/ml}$  or when the sum of insulinemic values at the second and third hours of the glycemic curve is greater than 60  $\mu\text{U/ml}$ . It is one of the most prevalent etiologies for cochleovestibular disorders: Between 84 and 92% of patients with idiopathic tinnitus present with hyperinsulinemia. Type II and IIIA insulinemic curves are the most frequent curves observed in these patients [10,12]. A logical result of this close, clinically demonstrated relationship between tinnitus and hyperinsulinemia is that the dietary treatment results in decrease of tinnitus symptoms in a significant percentage of patients [12].

The development of hyperinsulinemia is a direct consequence of a metabolic disorder known as *insulin*

Reprint requests: Dr. Luiz Lavinsky, Rua Quintino Bocaiúva, 673, 90440-051 Porto Alegre, RS Brazil. Fax: +55-51-3330-2444; E-mail: lavinsky.ez@terra.com.br

resistance, characterized by a reduced biological response to insulin at the cellular level. Patients with non-insulin-dependent diabetes mellitus (NIDDM) show reductions in whole-body functional insulin levels greater than 35–40% [13].

Demonstrably, the earlier the identification of a metabolic disorder, the better the clinical response, not merely as regards tinnitus but in relation to vertigo and prevention of hearing loss. The presence of underlying metabolic disease should thus be examined in every patient presenting with cochlear or vestibular disorders (or both), by means of the 5-hour insulinemic and glycemic curves. These curves allow the diagnosis of abnormal carbohydrate metabolism much earlier than do the tests traditionally used for this purpose (fasting glycemia and 2-hour glucose tolerance).

The 5-hour insulinemic and glycemic curves, using 100 mg glucose, are the most sensitive tests for the diagnosis of patients with abnormal carbohydrate metabolism. Combined, these curves are able to detect abnormal carbohydrate metabolism before the development of reduced tolerance to glucose or glucose intolerance. The isolated evaluation of fasting insulinemia, using the criterion of a value greater than 30  $\mu$ U/ml, has been shown to have a sensitivity of only 10% for diagnosing hyperinsulinemia, which demonstrates the need for the systematic calculation of the insulinemic curve in case of suspected hyperinsulinemia. A finding of insulin levels greater than 40 mg/dl in the second hour of the insulinemic curve has a sensitivity and specificity of 89% for diagnosing hyperinsulinemia. A sum of insulin levels at the second and third hours greater than 60 mg/dl has a sensitivity and specificity of 99% for diagnosing hyperinsulinemia.

Hyperinsulinemia is believed to precede the development of NIDDM by some years; this concept suggests that hyperinsulinemia and NIDDM are extremes in the continuum of abnormal carbohydrate homeostasis. The development of abnormal carbohydrate metabolism homeostasis seems to occur in three stages: (1) isolated hyperinsulinemia or hyperinsulinemia with euglycemia (normal glucose tolerance), known as *diabetes mellitus* (DM) in situ or occult DM [14,15]; (2) hyperinsulinemia with reduced glucose tolerance; and (3) hyperinsulinemia with hyperglycemia (NIDDM). This sequence shows that hyperinsulinemia undoubtedly precedes hyperglycemia [13]. Hyperglycemia, especially in fasting, is thus a late marker of the complex process of metabolic disorder that has hyperinsulinemia as its earliest marker, and its occurrence is suggestive of pancreatic  $\beta$ -cell dysfunction [16]. Hyperinsulinemia is thus an essential condition for the development of NIDDM [13,15].

Hyperinsulinemic patients in whom the inner ear is

compromised frequently present with insulin-reactive hypoglycemia. In these patients, hypoglycemia almost always results from excessive production of insulin and is not, in general, a primary metabolic disorder [13]. Hyperinsulinemia has been shown to represent an earlier, more consistent change in the diagnosis of this type of metabolic disorder than does hypoglycemia, because of the greater sensitivity of the insulinemic curve in comparison with the glycemic curve. The main limitation of the glycemic curve in isolation is the possible occurrence of hypoglycemic peaks in the intervals between sample collection, yielding false-negative results [8]. Also known is that most hypoglycemic episodes occur more than 3 hours after the administration of oral glucose. This explains the low sensitivity of the traditional glucose tolerance test, consisting of only two samples (fasting and 2 hours after the administration of 75 mg glucose), in diagnosing this condition.

For insulin-reactive hypoglycemia, the diagnostic criterion has been the occurrence of at least one value equal to or below 55 mg/dl. As has been shown, however, in patients presenting with significant hyperglycemic peaks that exceed the 175-mg/dl renal threshold, both the individual glycemic values and the rate of change should be observed. In these patients, a rate of fall in glycemic values of more than 1 mg/dl/min, manifesting as a difference greater than 60 mg/dl between two consecutive samples, should be regarded as indicating a diagnosis of insulin-reactive hypoglycemia.

The finding that occult DM precedes the development of reduced glucose tolerance and of NIDDM has led to the progressive recognition of the importance of hyperinsulinemia as the key metabolic change to be examined when NIDDM is suspected. In fact, the progressive increase in insulinemic levels is known to occur long before any demonstrable change to glycemic levels, whether in fasting, in the 2-hour test, or even in the glycemic curve. Thus, some have suggested that the diabetic state—whether it be occult, reduced glucose tolerance, or NIDDM—be defined on the basis of insulinemic status, with a secondary glycemic designation given by fasting glycemia and 2-hour glucose tolerance. This represents a change from the criteria used today, centered almost exclusively on glycemic levels. This presumably results from the fact that euglycemic hyperinsulinemia (occult DM) is not yet recognized as a clinical entity forming part of a spectrum of abnormalities that reaches its other extreme in NIDDM. For this reason, especially outside the otorhinolaryngological community, early diagnoses of this type of carbohydrate metabolic disorder will continue to be ignored, despite the fact that the early identification of NIDDM would allow the prevention not only of the cochlear and vestibular damage associated with this dysmetabolic

stage but of the progress of the disorder to the stages of reduced glucose tolerance and hyperglycemia, with their known potential for damage. What should also be emphasized is that hyperinsulinemia is now understood to be a risk factor for other comorbidities, further reinforcing the need for early recognition and correction of the disorder.

The aim of this study was to evaluate efficacy of dietary treatment to improve tinnitus in patients with hyperinsulinemia.

## PATIENTS AND METHODS

### Patient Selection

All patients from the Lavinsky Clinic in Porto Alegre, Brazil, presenting with complaints of tinnitus were assessed by means of 5-hour glycemic and insulinemic curves, using 100 mg glucose. Hyperinsulinemia was defined in the presence of one or more of the following [13]: (1) fasting insulinemia greater than 30  $\mu\text{U/ml}$ ; (2) insulinemia greater than 50  $\mu\text{U/ml}$  at 120 minutes; and (3) sum of insulinemic values greater than 60  $\mu\text{U/ml}$  at 120 and 180 minutes.

The diagnosis of insulin-reactive hypoglycemia was based on the occurrence of at least one value equal to or below 55 mg/dl. In patients presenting significant hyperglycemic peaks in the glycemic curve (exceeding the 175-mg/dl renal threshold), both the individual glycemic values and the rate of fall were considered. Therefore, in these patients, a rate of fall of more than 1 mg/dl/min, manifesting as a difference greater than 60 mg/dl between two consecutive samples, was also regarded as a diagnostic criterion for insulin-reactive hypoglycemia.

Thus, according to the aforementioned criteria, patients with diagnosed hyperinsulinemia, regardless of association with insulin-reactive hypoglycemia, were submitted to dietary therapy based on the Updegraff diet for a minimum of 2 years and under the supervision of a nutritionist. Patients were asked to eat every 3 hours to prevent hypoglycemia, even if transient; to avoid refined sugar (or to use artificial sweeteners if necessary); to restrict their intake of fatty foods; to take no more than two cups of coffee a day; to limit their intake of alcoholic beverages; and to drink four to six glasses of water a day.

### Study Methods

On presentation, the patients underwent a diagnostic protocol, including completion of a multiple-choice history examination, otorhinolaryngological examination, examination of the temporomandibular joint and

cervical column, and auscultation of the large vessels of the neck. Tonal and vocal audiometry, impedance testing, and tinnitus parameter testing were also performed, as were the following laboratory examinations: antinuclear factor (ANF) levels; VDRL test; fluorescent treponemal absorption antibody test; cholesterol (total and fractions) and triglyceride levels; toxoplasmosis serological screen; urea and creatinine levels; hemogram; variant surface glycoprotein levels; fasting glycemia; 5-hour glycemic and insulinemic curves; and thyroxine and thyroid-stimulating hormone levels. Depending on the specific indications pertinent to each case, the following tests were also performed: otoacoustic emissions, evoked potential audiometry, electronystagmography, computed tomography, and nuclear magnetic resonance imaging.

Patients were questioned about the type, intensity, frequency, and masking of tinnitus and about the presence of associated symptoms before, during, and after dietary treatment. Our questionnaire included the following inquiries:

- How would you describe the sound?
- Does the sound interfere with your regular activities? If so, in what way?
- Is the sound always present or does it come and go?
- Do you still hear the sound in noisy places?
- Do you have any symptoms other than the sound?
- Was a diet prescribed to control the sound? If so, did you follow the diet? Did you have any improvement during the diet?

The same questions were asked about symptoms during and after the diet.

After a minimum of 4 years, 80 patients were contacted by telephone. The patients were divided into two groups: the study group, consisting of those patients who followed the diet, and the control group, consisting of those who did not follow it.

The following criteria were used to classify the results: patients who described no improvement in symptoms, classified as "no improvement"; those who described improvement but were still bothered by the tinnitus, classified as "some improvement"; those who stated that the tinnitus continued but no longer bothered them or interfered with regular activities, classified as "significant improvement"; and those who stated that the tinnitus had disappeared, classified as "disappeared." *Low intensity* was defined as tinnitus that did not interfere with regular activities; *high intensity* was defined as tinnitus that interfered with regular activities, including sleep; and *medium intensity* was defined as tinnitus that interfered with activities on some occasions, irrespective of details.

**Table 1.** Types of Tinnitus

Symptom	No. of Patients (%)
Hissing	29 (36.2)
Whistling	16 (20)
Insect chirping	16 (20)
Ocean roaring	9 (11.3)
Waterfall rushing	3 (3.7)
Others	6 (7.5)

On the basis of 5-hour insulinemic curves, patients were classified using Kraft's criteria [13] and analyzed according to decrease of symptoms using residual analysis and the chi-square test. The same analysis was performed with Kraft classifications and dyslipidemic changes in patients. Both analyses were performed using the SPSS software with the assistance of a statistician not otherwise involved in the study.

All patients were interviewed by two interviewers using the same script. The interviewers were not involved in the diagnosis or treatment of the patients. Statistical analysis of other data was performed using Fisher's exact test and the EPI-INFO 6.0 software, with a significance level set at 95%.

## RESULTS

Of the 80 patients interviewed, 59 (73.7%) had followed the diet. The mean interval between the start of the diet (consultation) and the interview was 5.65 years. Type and frequency of tinnitus are described in Tables 1 and 2.

Table 3 describes the lessening of symptoms in terms of tinnitus intensity. No relation was found between decrease of symptoms and intensity category ( $p = 0.3$ ; i.e., the abatement of tinnitus symptoms was not associated with the intensity of tinnitus). Of the patients included in the study, 46 (57.5%) presented paroxysmal tinnitus, 33 (41.2%) reported constant tinnitus, and only 1 reported sporadic tinnitus. Most patients (41.2%) reported medium-intensity tinnitus, with high-intensity tinnitus being the second most frequently reported type. Variable-intensity tinnitus was reported by 10 (12.5%) of the patients. Other symptoms, including headache, fainting sensation, and nausea, were reported by 52

**Table 2.** Frequency of Tinnitus

Duration	No. of Patients (%)
Constant	33 (41.2)
Paroxysmal	46 (57.5)
Sporadic	1 (1.3)

**Table 3.** Reduction of Symptoms Considering Tinnitus Intensity

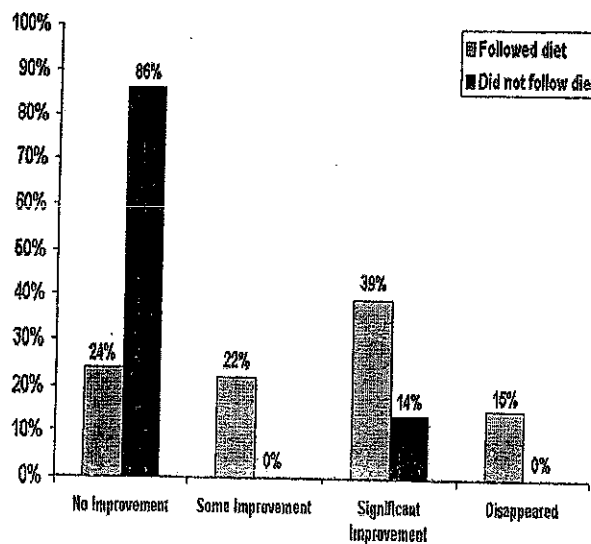
Intensity	Reduction No. of Patients (%)	No Reduction No. of Patients (%)	Total No. of Patients (%)
High	8 (42)	11 (58)	19 (100)
Medium	22 (67)	11 (34)	33 (100)
Low	11 (61)	7 (39)	18 (100)
Variable	7 (70)	3 (30)	10 (100)

$p = .3$  (chi-square test).

(65%) of the patients. Figure 1 shows the results concerning decrease in tinnitus symptoms during the diet.

Among the patients ( $n = 59$ ) who followed the diet (study group), 14 (24%) reported no lessening of tinnitus symptoms with the diet. Among the patients ( $n = 21$ ) who did not follow the diet (control group), 18 (86%) did not present any decrease of tinnitus symptoms (Table 4). The likelihood of presenting with decreased tinnitus symptoms was five times greater in patients who had tinnitus and abnormal carbohydrate metabolism and adequately followed the diet than in those who did not follow the diet (RR, 5.34; 95% CI, 1.85–15.37;  $p = .000003$  [Fisher's exact test]). After control for tinnitus intensity, the likelihood of presenting improvement remained very similar (RR, 5.6; 95% CI, 1.92–16.30), which means that tinnitus intensity does not interfere with the response to the diet.

After classification according to Kraft's criteria and evaluation in terms of decreased symptoms during the diet, no significant difference was found for any specific type of tinnitus ( $p = .56$ ). On evaluation in terms

**Figure 1.** Reduction of tinnitus symptoms during the diet.

**Table 4.** Reduction of Tinnitus Symptoms After Dietary Intervention

Patient Compliance	Reduction	No Reduction	Total
Followed diet	45	14	59
Did not follow diet	3	18	21

Notes: Decrease of tinnitus with diet:  $p = .000003$  (Fisher's exact  $t$ -test); RR, 5.34; 95% CI, 1.85–15.37. Controlled for tinnitus intensity:  $p < .001$ ; RR, 5.6; 95% CI, 1.92–16.30.

of dyslipidemic changes, no difference was found between the types of tinnitus ( $p = .47$ ).

## DISCUSSION

Abnormal carbohydrate metabolism appears to be the most prevalent metabolic disorder associated with tinnitus with or without dysacusis and vertigo. The investigation of abnormal carbohydrate metabolism should be an obligatory step in the diagnosis and therapy of tinnitus patients. Carbohydrate metabolism disorders include a wide range of manifestations, each of which has its peculiarities of presentation, diagnosis, and management, although they may represent different stages in the same physiopathogenic process.

Hyperglycemic states are characterized by fasting hyperglycemia and glucose intolerance resulting from deficient insulin action, with or without the presence of hyperinsulinemia. These states have the potential to compromise the inner ear owing to its close association with carbohydrate metabolism. Hyperglycemic states include DM and reduced glucose tolerance, conditions that should be understood within a continuum of abnormalities in the mechanisms of production, use, and regulation of the metabolism of carbohydrates. The diseases in the hyperglycemic group are prevalent pathologies associated with high morbidity but are largely preventable.

Hyperglycemic patients may present with cochleovestibular disorders through three main mechanisms, which may act in isolation or in association: neuropathy of the eighth cranial pair, vasculopathy of the smaller vessels, and interference in the action of the sodium-potassium ATPase pump at the level of the inner ear (and especially at the vascular stria). The first two mechanisms are more significant for diabetic patients, especially those with severe, difficult-to-manage insulin-dependent diabetes mellitus (IDDM) or NIDDM, which are the main sources of cochleovestibular lesion.

The basic etiopathogenic feature of the less severe hyperglycemic states not defined as DM—including reduced glucose tolerance—is the interference with the sodium-potassium ATPase pump. The physiopatho-

genic mechanism for cochleovestibular change in patients thus affected is complex, with insulin playing an important part, as occurs in patients with occult DM.

The metabolism of the inner ear is intense, depending on oxygen supply and showing no accumulated energy reserves [17–19]. The double function of insulin at the cellular level is well known: it carries glucose into the cell and regulates ion transport through the cell membrane. Situations associated with hyperinsulinemia tamper not only with glycemic levels but, more important, with the sodium-potassium ATPase pump. This pump is responsible for maintaining high potassium concentrations and a low concentration of sodium in the endolymph, as occurs in the intracellular space. Much energy is expended to maintain this ionic balance, because the transport of sodium through the cellular membrane toward the extracellular space goes against a concentration gradient. Patients with hyperinsulinemia, as those with NIDDM or with hypoinsulinemia (IDDM), have an increased concentration of sodium and reduced concentration of potassium at the endolymph level, which therefore alters the endocochlear potential and, consequently, the cochlear microphonic response. The persistence of this metabolic disorder results in injury to the external ciliated cells and to the efferent pathways of the auditory system.

This type of disorder is clinically expressed by the occurrence of tinnitus, which, not rarely, is associated with bilateral, often symmetrical, progressive neurosensory dysacusis. The administration of insulin probably reduces the oxygen level in the inner ear. The increase in the osmotic pressure of the endolymph, as a result of the increased concentration of sodium, is another predictable consequence of these ionic disorders, causing endolymphatic hydrops. Notably, carbohydrate metabolism disorders are one of the possible etiologies of Ménière's syndrome.

In patients with Ménière's disease, tinnitus may occur either alone or in association with dysacusis, aural fullness, or vertigo. Unlike other DM complications, it may present as acute and usually reversible (at least in part) or as a chronic situation, in which at least some level of involvement is irreversible. The acute development of one or more of these symptoms usually occurs in the presence of an important hypoglycemic or hyperglycemic episode, resulting in temporary imbalance of the inner ear. As diabetic patients present with persistent or recurrent disorders that can hinder cochleovestibular function, their sensory organs are gradually injured, producing irreversible disorders [20,21].

DM *in situ* may result in involvement of the inner ear by the same physiopathogenic mechanisms of glucose intolerance and DM. This is perfectly understandable, since DM and glucose intolerance are different degrees

of the same group of metabolic disorders. Although the disorders of the inner ear are a weaker expression of carbohydrate metabolism disorders, they are the same as those presented by patients with frank DM or reduced glucose tolerance. This may be explained by the fact that the sodium-potassium ATPase pump, the function of which is altered in these patients, has one of the highest levels of activity at the level of vascular stria, in such a way that cochleovestibular involvement may be observed even at incipient stages of metabolic disorders involving carbohydrates and insulin.

In the treatment of patients with NIDDM and cochleovestibular disease, special attention should be paid to some peculiarities. The presence of other metabolic disorders, especially those involving lipids, should be ruled out, as their presence requires changes in treatment and represents an additional cochleovestibular and cardiovascular risk factor.

Two treatment approaches are recommended: one approach that focuses on cochlear or vestibular disease (or both) and another that concerns DM. The management of cochleovestibular disease consists of the management of the underlying disease, because eliminating the functional deterioration of the inner ear is possible only by controlling the underlying cause. Neurosensory dysacusis may exhibit different levels of improvement after control of the metabolic problem. Tinnitus, as occurs with hearing loss, may have various levels of attenuation and is usually persistent even after the stabilization of blood glucose levels [22]. Dizziness, often rotational and paroxysmal, is the symptom that diminishes the most with metabolic control. Initially, the patients require vertigo treatment combined with the treatment of metabolic disorders. After DM is stabilized, most patients are free of vestibular symptoms without vertigo treatment [20]. Patients with Ménière's syndrome associated with DM may require continued treatment of the syndrome for a longer period, as a way to control DM-induced endolymphatic hydrops.

The reduction in glycemic supply to the inner ear at levels below a critical threshold results in the involvement (initially transient) of the sodium-potassium ATPase pump in charge of maintaining the ionic-osmotic balance of the membranous labyrinth. A critical level of glycemic supply should be understood as that below which dysfunction of the sodium-potassium ATPase pump and reduction of the cochlear microphonic response occur. The intense metabolic activity of the inner ear, without accumulated energy reserves, renders it susceptible in case of reduced glucose supply. The mechanism of inner-ear injury is the same, regardless of the etiology of the hypoglycemic state, although in some hypoglycemia-related diseases, such as insulin-producing tumors, an additional mechanism might be

involved [23]. The severity of injury to the inner ear is directly related to the intensity, duration, and frequency of hypoglycemic bouts, which are closely related to the etiology of hypoglycemia.

Patients with cochleovestibular disease associated with insulin-reaction hypoglycemia, with or without association of hyperinsulinemia, should be asked to follow a specific diet to control metabolic disorders (characterized by the reduction of carbohydrates, especially those that are quickly absorbed), increase the frequency of meals (ideally at 3-hour intervals), and reduce the food intake in each of these meals. In the presence of obesity, which is commonly associated with late insulin-reaction hypoglycemia, patients should be asked to reduce their calorie intake, with the aim of achieving the ideal weight.

The management of hyperinsulinemic patients with metabolic disorders, in its euglycemic form and with reduced glucose tolerance, focuses on diet and regular physical exercise, which implies a change in lifestyle. An appropriate diet can delay or interrupt the progression of euglycemia to hypoglycemia, in addition to improving or normalizing hyperinsulinemia, regarded as the key disorder in this group of patients. Practicing physical exercise regularly also has a direct impact on the improvement of insulin resistance [13].

Those patients in whom management based on diet and physical exercise allows the maintenance of euglycemia, albeit without reversion of hyperinsulinemia, are at increased risk of developing some disorders that are probably associated with hyperinsulinemia (Table 5). The dietary changes recommended in the clinical treatment of patients with metabolic disorders as far as tinnitus is concerned are believed to attenuate potentially the importance of tinnitus, which results in increased tolerance. As observed in our study, improvement is observed after dietary treatment, regardless of the intensity of tinnitus. The reduction of tinnitus intensity

**Table 5.** Disorders Associated with Hyperinsulinemia

Essential hypertension
Atherosclerosis
Coronary artery disease
Generalized
Primary dysfunction of ovarian follicles
Neurootological conditions
Ménière's syndrome
Migraine
Tinnitus
Secondary endolymphatic hydrops
Vertigo
Dysacusis

Source: Adapted from Kraft [11].

allows its limbic expression to stand out partially, with consequently lower cortical repercussion.

## CONCLUSIONS

The results of our study show the great potential of dietary management to improve tinnitus in patients with carbohydrate metabolism disorder, independently of tinnitus intensity. Dietary management alone was associated with a fivefold increase in the probability of significant reduction in tinnitus. The fact that 76% of the patients who followed a specific diet achieved at least partial decrease of tinnitus symptoms reinforces the need for considering this metabolic disorder as a possible etiological diagnosis in the presence of tinnitus. As far as we know, the significant lessening of tinnitus symptoms observed in this study (with resolution in 15% of cases) has not been observed with any other types of treatment (drug therapy, for example).

The difficulty of treating tinnitus patients (who frequently present with associated depressive or anxiety disorders) is well-known. Therefore, a thorough investigation of associated metabolic alterations using the 5-hour glycemic and insulinemic curves, as performed in this study, should be part of the routine evaluation of patients with tinnitus and may result in significant easing in symptoms and quality of life. Only with the optimization of the diagnosis of metabolic alterations will we be able to correct or at least attenuate those disorders, which may have an extremely relevant impact on tinnitus.

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