

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800

Open access books available

122,000

International authors and editors

135M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.

For more information visit www.intechopen.com



Components of Metabolic Syndrome and Their Relation to Tinnitus

Ludovit Gaspar¹, Michal Makovnik¹,
Matej Bendzala¹, Stella Hlinskova¹,
Ivan Ocadlik¹ and Eva Gasparova²

¹Second Department of Internal Medicine, University Hospital Bratislava

²ENT Outpatients Department, Novapharm Bratislava
Slovak Republic

1. Introduction

Tinnitus is defined as a non-specific symptom characterized as buzzing, whistling, fizzing, ringing or sensing of wide frequency range and different intensity sounds in one or both ears. Tinnitus is a sensation of sound perceived by an individual in the absence of an external sound source. It affects approximately 15% of the population worldwide. Tinnitus is usually connected with many kinds of hearing disturbances, but it could be also a symptom of different other health problems. Metabolic syndrome and its components as arterial hypertension, diabetes mellitus, obesity and dyslipoproteinaemia with arteriosclerosis are important and frequent causes of tinnitus. Due to the high number of heterogeneous etiologic factors in subjective and objective tinnitus a complex approach in differential diagnosis is crucial. Tinnitus can be a concomitant symptom to many disorders of peripheral statoacoustic apparatus. It is necessary to distinguish whether tinnitus is an acute symptom in onset, e.g. in Menière's disease, acute trauma, inflammation of the inner ear, or it is a part of chronic disease otological in origin. Various cardiovascular diseases can be the cause of newly arisen or persistent tinnitus. Among the most important causes there is a reduced perfusion with ischemic changes. Vascular changes can be localized either extra- or intracranially. Among them are stenosis of arteries, haemangioma and glomus caroticum tumours. Changes in the rheology of blood in anaemia or polycythemia can be the cause of tinnitus, as well. It is important to think also about vasculitis as a cause of tinnitus especially among younger patients. Diagnosis of hypertension requires serious and careful approach. The use of a 24-hour ABPM should be considered and carried out as soon as possible in patients with presence of tinnitus. Despite our results are from the limited number of patients with tinnitus, the diagnostic of arterial hypertension is very important, as several prospective studies have reported that ABPM give better prediction of clinical outcomes compared with conventional clinic or office blood pressure measurements. The inner ear, like the brain, is totally lacking in energy reserves. Its metabolism depends directly on the supply of oxygen and glucose from the blood supply. Alterations in glucose metabolism therefore have great potential for disturbing the workings of the inner ear.

2. Classification of tinnitus

a. Objective tinnitus

Objective tinnitus is usually of rhythmical character and can be registered. It can arise from muscle spasms or alteration to the vessels. Although there is a wide spectrum of these disorders, objective tinnitus is not very common, only about 1-2 % of all tinnitus cases (Møller, 2007).

b. Subjective tinnitus

Subjective tinnitus is defined as the perception of sound on the absence of an internal or external sound source. Subjective tinnitus is perceived only by a patient. Its origin is in acoustic analyser or in the brain. Tinnitus itself is not a disease. It is a symptom going along with other diseases. But in fact it can be a dominant sign of the whole clinical picture especially when it is persistent, it has increasing intensity and negative effect on work ability and psychosomatic status. The etiology of a majority of cases is not known (Feldmann, et al., 1992).

It has to be distinguished from acoustic hallucinations as a part of some psychiatric disorders. The most frequent causes of tinnitus are shown in table 1.

Disorders of acoustic analyzer and central nervous system
Alternation of the structures of the inner ear and acoustic nerve
Menière's disease
Infection of the inner ear and sinuses
Acoustic trauma
Chronic noise exposure
Otosclerosis
Neurinoma of the auditory nerve
Arterial hypertension
Arterial hypotension
Diabetes mellitus
Arteriosclerosis
Aneurysm
Disorders of metabolism
Kidney diseases
Head and neck trauma
Cervical spine and temporomandibular joint diseases
Hematological diseases with the change in viscosity of blood and changes in microcirculation
Exogenous noxious substances - nicotine, alcohol, caffeine
Medications: antibiotics, NSAIDs, antidepressants, cytostatics, furosemide, oral contraceptives
Industrial toxins - heavy metals, carbon monoxide, aniline, benzol
Artificial sweeteners containing aspartame
Lues
Autoimmune diseases

Table 1. The most frequent causes of tinnitus

Tinnitus is described as an unwelcome symptom. Tinnitus can be an attendant phenomenon in specific diseases, but in some cases his origin and cause is unknown. Through the precise etiology in most cases is unknown, an abnormal interaction between peripheral and central auditory pathways is believed to play a role in tinnitus development. Tinnitus is associated with the most important symptoms in neurootology, besides vertigo, nausea and hearing loss. There are many forms of subjective tinnitus witch can occur with different severity.

The parameters of tinnitus that require identification are intensity, location, duration, quality, maskability and rebound. The patient may report the intensity as mild, moderate or severe. Various grading systems have been used to provide a means of communication between the patient and examiner.

The biological mechanisms leading to the perception of tinnitus are still not completely understood. Increasing agreement, however, posits that different forms of tinnitus may differ in their pathophysiological mechanisms. Also generally accepted is that most forms of subjective chronic tinnitus are the consequence of central nervous system reorganization processes induced by altered peripheral auditory and somatosensory input. Peripheral deafferentation may result in an imbalance between excitatory and inhibitory function, causing maladaptive plastic changes in the structural and functional organization of the auditory system at several levels (dorsal cochlear nucleus, inferior colliculus, medial geniculate body and auditory cortex).

Sudden sensorineural hearing loss is defined as hearing loss equal to or greater than 30 dB at three or more consecutive frequencies, the onset of which takes place over a period 3 days or fewer. In most cases, it is severe, nonfluctuating, unilateral and idiopathic. Approximately one-third of cases are accompanied by complains of tinnitus, dizziness and ear fullness.

3. Obesity and metabolic syndrome

Obesity is a chronic progressive disease characterized by the accumulation of fat tissue with multiple organ-specific pathological consequences which has a significant impact on morbidity, as well as the quality and length of the obese individuals` life.

The prevalence of obesity in many European Union countries has tripled since the year 1980 and further is increasing alarmingly, only during the last 10 years the number of obese has increased by 10 to 40%. Obesity is responsible for 2-8% of the costs and 10 to 13% of deaths in the region of the European Union. Overweight and obesity in the European region has more than 50% of the population. 20% of children and adolescents are overweight and a third of them are obese (Branca et al., 2007). Data from United States show that 65% of the local population has excess weight and 31% are obese. These data highlight the epidemic situation of obesity, and therefore the WHO considers reversing of this trend as one of its priorities (Haslam & James, 2005).

In Table 2 are examples of the connection of obesity with a number of associated diseases and conditions.

An abdominal adiposity as the cardiovascular risk factor is associated with components of metabolic syndrome, namely with insulin resistance, disturbed glucose metabolism, hypertension and dyslipidemia. With these components it is very closely linked to acceleration of the process of atherosclerosis and its serious systemic complications. It is important that the effect of these components (factors) together in mutual combination is not simply added, but multiplied.

Cardiovascular disease: coronary artery disease, myocardial infarction, systemic arterial hypertension, atherogenic dyslipidemia, stroke
Oncologic diseases: endometrial and ovarian cancer, prostate, breast, rectum, kidney, liver, gallbladder cancer
Diabetes mellitus type 2 and insulin resistance
Hepatic steatosis
Osteoarthropathy
Deep vein thrombosis and pulmonary embolism
Chronic renal failure
Polycystic ovary syndrome
Hyperuricemia
Gallstones
Reproductive disorders
Lumboischialgic syndrome
Ventilation pulmonary complications
Sleep apnea syndrome
Psychological and social problems
Complications in pregnancy
Complications in the perioperative period

Table 2. Obesity is connected with a number of associated diseases and conditions

Obesity and overweight are important markers of coronary heart disease and lead to an increase in cardiovascular and cerebrovascular morbidity and mortality (Narkiewicz, 2006). Adipose tissue is an endocrine organ. It synthesizes and releases several specific components, eg. cortisol, estrogen, leptin, adiponectin, resistin, nonesterified free fatty acids, interleukin 6 (IL-6), tumor necrosis factor alpha (TNF-alpha), plasminogen activator inhibitor-1 (PAI-1) and others. These influence and are in relationship to the overall cardiometabolic risk in an individual patient (Bonora, 2006). Many epidemiological studies show that prevalence of hypertension increases progressively with increasing BMI (body mass index) as for men as well as in women. Relationship between BMI and blood pressure (BP) is stronger for systolic (SBP) as the for diastolic (DBP) blood pressure. Data from the NHANES III study (Foley et al., 2005) clearly indicate, that the risk of hypertension developing is closely relate to the waist, as well as waist / hip ratio (WHR). In general, it is estimated that in about 75% of men with hypertension and in 65% of women with hypertension is present overweight or obesity. Data from the Finnish population studies suggest up to 85% prevalence of hypertension in persons with BMI over 25 kgm² (Kastarinen et al., 2000). Obesity and particularly central obesity leads to increased cardiovascular risk. Results of population studies suggest that at least two thirds of the prevalence of hypertension can be directly attributed to the impact of the obesity (Krauss et al., 1998).

3.1 Pathophysiological changes in the cardiovascular system in obesity

In the obesity there is a change in the fundamental characteristics of circulation, as heart stroke volume and cardiac output, peripheral vascular resistance, changes in the flow distribution. It leads, in association with other risk factors, to the development of arterial hypertension and coronary heart disease. The degree of obesity correlates directly with

cardiac output and is inversely associated with systemic vascular resistance. The increase in cardiac output in obese is resulting from an increased stroke volume, while heart rate does not correlate with BMI (Kurtz & Klein, 2009; Messerli et al., 1981). Stroke volume is probably associated with an increased intravascular volume (non-obese 50 ml/kg vs. obese 75 ml/kg). Central type of obesity, however, is associated with lower cardiac output and higher total peripheral vascular resistance, and the deviation of above parameters is even increased by the mental stress. Splanchnic blood flow is increased by 20%, flow through the kidneys and the brain remains unchanged (Jern et al., 1992). Distribution of body fat influences the muscle morphology and density of the muscle capillary network. Persons with a higher waist circumference have less type I muscle fibers and more type IIB fibers. Type IIB has about 20 to 30% lower density of capillary network. In the case of stress response can be expected to lower vasodilative response in the muscles, followed by the less efficient splanchnic vasoconstriction in stress defense response. Abdominal obesity has been associated with increased activity of hypothalamic-pituitary-adrenal axis, which is a major part of the neuroendocrine system that controls reactions to stress and defense reaction (Howell et al., 2004; Rahmouni et al., 2005).

There is a higher cardiac output in obese individuals, higher circulating intravascular volume and increased oxygen consumption, but total blood flow in relation to body weight is reduced. An increased sympathetic activity is also common in obesity, which may in long-term activation cause hypertension by the peripheral vasoconstriction and increased tubular reabsorption. Studying regional sympathetic activity in obese subjects was found an excess of noradrenaline in relation to the kidneys as a key organ of cardiovascular homeostasis. Both obesity and hypertension lead to the development of left ventricular hypertrophy and subsequent diastolic dysfunction (Redon et al., 2008).

Adipose tissue is through several mechanisms, directly integrated into the pathogenesis of hypertension. These mechanisms include alteration of renin-angiotensin-aldosterone system (RAAS), increased activity of the sympathetic nervous system, insulin resistance, leptin resistance, alteration of coagulation and inflammatory factors in the development of endothelial dysfunction. Activation of adipose RAAS is involved particularly in the creation and development of hypertension in the central (visceral) type of obesity. Especially in this type of obesity is increased level of plasma aldosterone (Goodfriend & Calhoun, 2004). The cause of the increase in production can be potent mineralocorticoid-releasing factors, or effect of oxidised linolenic acid compounds on aldosterone synthesis (Goodfriend et al., 2004).

Leptin is a link between adiposity and increased sympathetic activity. In addition to effects on appetite and metabolism, leptin increases blood pressure by the sympathetic activation in hypothalamus (Carlyle et al., 2002). Understanding the role of leptin in the pathophysiology of sympathetic overactivity in obese patients facilitates the concept of selective leptin resistance. Leptin-induced increase in renal sympathetic activity and blood pressure is mediated by the ventromedial and dorsolateral hypothalamus (Marsh et al., 2003). Selectivity refers to the different signaling pathways distal to the leptin receptor. Enzyme phosphoinositol-3 kinase is an important intracellular signaling pathway for the control of renal sympathetic system, because inhibition of this enzyme reduces sympathetic activity. Melanocortin system is also an important regulatory axis, with the effects on renal sympathetic and blood pressure (da Silva et al., 2004).

Hypertension contributes to increased cardiovascular and cerebrovascular morbidity and mortality through effects on atherogenesis by multiple mechanisms: it leads to endothelial

dysfunction with impairment of responses to vasodilating substances, to increased vascular permeability for macromolecules (also for lipoproteins), to the increased production of vasoconstrictor endothelin and to increased adherence of leukocytes, also to the remodeling of smooth muscles in large arteries, whereas in small arteries it causes increased vascular tone. An increase in angiotensin II level and PDGF act as growth factors for smooth muscle cells of the media, their effect is potentiated by the endothelin. Phenotypic change of vascular smooth muscle cells may also occur, thus increasing their proliferative potential and their proliferative response to growth factors (Fábryová, 2008).

Endothelial dysfunction in terms of reduced reactivity to nitric oxide (NO) is a typical abnormality in obesity. Impaired endothelium is a major risk factor for cardiovascular diseases because it leads to structural changes such as the rebuilding of the intima and media of wall of blood vessels. Strong factor for the development of endothelial dysfunction is the increased production of endothelin-1 in patients with hypertension and obesity. Blockade of endothelin receptor-A induces significant vasodilation in subjects with overweight and obesity, but not in the case of normal-weight patients with hypertension (Cardillo et al., 2004).

Obesity is associated with several structural and functional changes in kidneys. RAAS and sympathetic activation increases the levels of plasma aldosterone with consequent increases in renal sodium reabsorption, which leads to impaired sodium excretion and the expansion of circulating blood volume. Obesity can also cause structural changes in the kidney, leading to chronic renal failure with a further increase in blood pressure. Mechanic pressure of abdominal adipose tissue in the visceral type of obesity may have also a role in renal impairment. Besides sympathetic autonomic nervous system also the leptin plays a role, which acts directly by increasing oxidative stress with a demonstrable increase in isoprostane levels in blood and urine, increased lipid peroxidation products in kidney tissue and decreased renal activity of aconitase (Beltowski et al., 2004). Obesity can cause glomerular hyperfiltration, abnormal albuminuria and progressive loss of kidney function due to focal segmental glomerulosclerosis (Kambham et al., 2001). Number of nephrons is reduced in patients with primary hypertension (Keller et al., 2003). In patients with pre-existing renal disease, obesity accelerates the progression of the disease and renal insufficiency (Praga et al., 2000). Structural kidney damage may further increase blood pressure and predispose to cardiovascular events.

3.2 Pathophysiological changes in the respiratory system in obesity

Obesity, especially abdominal type, is associated with deterioration of pulmonary function. Abdominal obesity is limiting diaphragm descent and expansion of lung parenchyma, in comparison with total adiposity, which compresses the chest wall. Compression of the lung parenchyma leads to decrease in expiratory reserve volume and functional residual capacity, which reduces the compliance of respiratory system. In obese patients with normocapnia breath work is at rest increased by 30%, in physical activity may be insufficient breath work limit for further increase in minute output with development of the relative hypoventilation. Products associated with abdominal type of obesity, such as serum leptin, CRP, leukocytes, fibrinogen have an inverse relationship to FEV1, therefore, inflammation may be connection between altered respiratory function and mortality (Schünemann et al., 2000; Sin & Man, 2003).

3.3 Obesity and sleep apnea syndrome

Obesity is associated with sleep apnea syndrome and obesity hypoventilation syndrome (Pickwickian syndrome, alveolar hypoventilation). In sleep apnea syndrome, pharyngeal airway passive collapse during deep stages of sleep, causing snoring and intermittent airway obstruction. The resulting hypoxia and hypercapnia cause disturbance of the normal sleep quality and subsequent daytime sleepiness, development of pulmonary and systemic vasoconstriction, polycythemia and right heart failure. Relative hypoventilation may cause progressive desensitization of respiratory centers to hypercapnia, followed by respiratory insufficiency.

Obesity hypoventilation syndrome is one of several diseases associated with chronic hypercapnia and alveolar hypoventilation. Obesity is associated with increased risk of alveolar hypoventilation and carbon dioxide retention. Hypoxemia and increased alveolar-arterial oxygen gradient in patients with obesity hypoventilation syndrome draws attention not only to alveolar hypoventilation, but also the ventilation - perfusion mismatch.

Patients with chronic obstructive pulmonary disease and sleep apnea, as well as patients with obesity hypoventilation syndrome in addition to systemic arterial hypertension also have high prevalence of pulmonary arterial hypertension (Nowbar et al., 2004; Šimková, 2009).

It is clear that hypertension dependent on obesity is a multifactorial disease. Obese individuals tend to hypertension, but vice-versa, hypertensive individuals tend to weight gain. As the Framingham and Tecumseh studies also showed, that future weight gain is significantly greater in hypertensive than in normotensive persons. This finding indicates that the hypertensive patients, even with normal weight are at increased risk of developing obesity and other complications resulting from it (Julius et al., 2000).

The link between increased BMI and mortality is documented by the analysis of 57 prospective studies (Flegal et al., 2007). For both sexes, the lowest mortality was found in BMI from 22.5 to 25 kg/m². In all age categories 35 to 59 years, 60 to 69 year, 70 to 79 y. and 80 to 89 years was revealed significant increase in mortality in persons with BMI 25 to 50 kg/m². BMI in its entirety from 15 to 50 kg/m² was associated almost linearly with systolic and diastolic blood pressure. Obesity was significantly associated with diabetes. This explains the positive and approximately log-linear relationship of BMI and an increase in the number of deaths due to ischemic heart disease with a 40% increase in CHD mortality for an increase in BMI for every 5 kg/m². The same results were found for stroke. Mortality due to heart failure and hypertension was also strongly associated with higher BMI.

Different groups of antihypertensive agents differ significantly in their metabolic effects. It is important to consider significant differences in this effect between different types of pharmaceuticals within the group itself, which is particularly true for beta-blockers. From the centrally acting antihypertensive drugs, it has been proven beneficial effect for weight reduction in moxonidine. Administration of ACE inhibitors and AT1 blockers is significantly reducing the risk of type 2 diabetes. Their complex beneficial effects affect not only the hypertension itself, but also the whole cascade of metabolic changes; therefore preparations that affect the renin-angiotensin system are essential treatment of hypertension in obesity and metabolic syndrome. From the group of sartans, telmisartan is unique substance in this respect, which significantly improves insulin sensitivity. From the drugs which may adversely affect weight and cause its sharp increase are particularly significant psychiatric medications. This undesirable side effect can be observed mainly in the traditional neuroleptics and tricyclic antidepressants (Svačina, 2002).

4. Metabolic disease

Extensive laboratory tests should be performed at the time of the general medical evaluation in addition to sugar and thyroid metabolism to evaluate the status of serum cholesterol and triglycerides level. Triglyceride levels have been reported to be considered in evaluating patients with inner ear or Menière's disease. There has been evidence of a positive experience in a small but significant number of patients, more male than female, for the identification of these factors in association with secondary endolymphatic hydrops. Significantly, the incidence of subjective idiopathic tinnitus in patients with diabetes mellitus has been infrequent in the series. The subjective idiopathic tinnitus was cochlear and was influenced positively by glucose control.

Nutritional deficiencies and imbalances are considered of significance in relation to ear disease and specifically for complaints of sensorineural hearing loss, vertigo and tinnitus. The clinical history is correlated with laboratory test results, which attempt to identify imbalances, deficiencies, and/or excesses of nutrients that are considered to upset the biochemical balances of the hearing organ resulting in malfunction of hearing and/or balance process. Impaired carbohydrate metabolism and its control have been identified in a significant number of patients with Menière's disease. The identification by clinical history and laboratory tests including determination of insulin levels, followed up by specific treatment to correct such an imbalance, is reported to significantly improve symptoms of endolymphatic hydrops (sensorineural hearing loss, vertigo, tinnitus). Hyperinsulinemia has three different effects: sodium retention within cochlear duct with a resulting endolymphatic hydrops, vasoconstriction caused by the effect of prolonged hyperinsulinemia in long term hypertrophy of arterial smooth muscles cells, and cholesterol formation and atherosclerosis within the arterial walls of the branches internal auditory artery. Impairment of glucose metabolism is of particular significance in the aged population. The chemical attachment of the sugar glucose to proteins without the aid of enzymes may contribute to age-related impairment of both cells and tissues. In this nonenzymatic process, glucose is described to have the capability of attaching itself to several sites along any available peptide chain. This process is called nonenzymatic glycosylation of certain proteins in the body. Glucose may play a role in tissue changes associated with normal aging. Elevated cholesterol and triglycerides levels have been reported with fluctuant hearing loss, vertigo, and tinnitus. A triad of elevated levels of insulin and triglycerides and impaired glucose tolerance has been reported, particularly in obese patients. The identification of these abnormalities combined with dietary control has been reported to result in considerable control of fluctuant sensorineural hearing loss, vertigo, and tinnitus. Association of a triad of hypertension and atherogenic diet and chronic noise exposure acts synergistically for the development of a sensorineural hearing loss (Pillsbury, 1986).

5. Arterial hypertension and tinnitus

Stabilization of the symptoms of hypertension has been found to have a significant positive influence on treatment in general and for the following clinical types of subjective idiopathic tinnitus (cochlear tinnitus, vestibular tinnitus and central tinnitus). Tinnitus has been found to be a "soft" sign of cerebro-vascular disease and secondary endolymphatic hydrops. For these clinical types of tinnitus as well as tinnitus of specific causes, blood pressure control

has afforded appreciable control. Ear and/or head blockage complaints should also be respected as an accompaniment of a possible "symptom less" hypertension. The term essential hypertension has been applied to idiopathic hypertension. Essential hypertension is marked by its heterogeneity and multifactorial nature. There have been identified clinical similarities between essential hypertension and subjective idiopathic tinnitus. Classically, patients have reported problems of tinnitus in association with elevated blood pressure. These are usually anecdotal reports, in the past, control of hypertension resulted in significant control of subjective idiopathic tinnitus. Therefore, the concept of and role of hypertension in the production and control of tinnitus is an old one. What is new is to reverse the thinking of hypertension as resulting in tinnitus to that of the presence of tinnitus as reflecting possible hypertension. In a significant number of tinnitus patients, the early identification of tinnitus has been accompanied by an elevation in blood pressure. Blood pressure control not only has resulted in a reduction in tinnitus intensity, but also has provided hearing conservation as well as the alteration or control of progression of complaints of cerebrovascular disease, cardiovascular complications, e.g. myocardial infarction, congestive heart failure, vertigo, progressive sensorineural hearing loss, and ear and/or head blockage. In a significant number of tinnitus patients, any or all of these symptoms together with that of tinnitus have been found to present as a symptoms complex reflecting an early manifestation of hypertension.

The essential quality of the hypertension requires investigation and attention by the internist. This is particularly evident among aged patients. In a significant number of patients in the aged group have either an acute onset of tinnitus and/or exacerbation of chronically present tinnitus accompanied by an elevation of blood pressure. The clinical correlation of hypertension and the tinnitus onset has also been found in the younger population, i.e., below age 40, but less frequently. Epidemiological studies concerning the relationship of blood pressure elevation with age concluded that this relationship has, as yet, not been determined (Morris, 2007). In some patients, blood pressure does increase with middle age. Some studies (Miall et al., 1967), did show positive correlation with advancing years. Tinnitus may be an early symptom that has wide-reaching consequences in a significant number of the aged population. The heterogeneity and multifactorial elements involved in essential hypertension, particularly in the aged, can explain the great degree of variability of its in tinnitus patients with or without hypertension. What is significant is that tinnitus may be a very early sign of onset of hypertension and/or an inadequate blood pressure control.

Sodium and water retention and a reduction in urinary excretion are of particular significance for those subjective tinnitus patients who may have a vestibular asymmetry with or without subjective complaints of vertigo and who are diagnosed with secondary endolymphatic hydrops. Secondary endolymphatic hydrops may be considered in the subjective tinnitus patient to be a mechanism for gradual progressive sensorineural hearing loss. Its control has, in a significant number of patients, resulted in satisfactory tinnitus control. Significant is that the initial blood pressure increase, which has been shown to originate in an area other than that of the kidney, is, however, maintained by the kidney by complex changes occurring in renal dynamics, as manifested by fall in renal blood flow and renin and increase in total renal vascular resistance.

Psychobiologic factors are significant for both subjective idiopathic tinnitus and essential hypertension. Essential hypertension can serve as can no other disease as model of the

multifactorial nature of all diseases in which complex interactions between inherited, environmental, social, cultural, experiential, physiological and biochemical factors occur. No single cause of essential hypertension appears to exist, no qualitative, discontinuous alteration has been discovered to explain its etiology or pathogenesis. Like essential hypertension, subjective tinnitus demonstrates heterogeneity of its manifestations, both for the mechanism involved in its production and/or its clinical manifestation. Both conditions share common factors including socio-economic, cultural, ethnic, behavioural and psychological factors.

Consideration should be given to the group of tinnitus patients for whom adequate sleep is accompanied by reduction in tinnitus. It has been known for some time, that behavioural states can affect blood pressure levels, which are much lower in sleep, than in the waking state. In both states, psychological factors are significant. In essential hypertension, the psychological factors alone are not considered sufficient to explain the predisposition to or pathogenesis of the disorder. The physiologic heterogeneity that results in hypertension is reflected in the psychological heterogeneity. Future clinical investigations of the relationship between the sensory and affect components in both these health problems may clarify diagnosis, treatment and control of subjective idiopathic tinnitus. In both conditions, social and psychological factors interfere in the clinical results of the prescribed treatment. Hypertensive patients often do not follow treatment and medication regimens. Similarly, tinnitus patients may not accept a treatment method that may provide tinnitus relief, because the recommendations may not fulfil the patient's expectation for cure or provide sufficient control.

When developing treatment and control methods for both tinnitus and essential hypertension, the physician must consider that both are disorders not only of adaptation but also of regulation, which may be reflected in one or more systems. This has already been established for hypertension, and it may apply to tinnitus.

6. Ambulatory Blood Pressure Monitoring (ABPM) in patients with tinnitus

The aim of our study was to assess the prevalence of unknown arterial hypertension in patients with tinnitus.

Patients and methods: We investigated 18 patients (8 female, 10 male) with median age 46 (age spread 34 - 64 years) with tinnitus lasting from 4 months to 6 years. Known history of arterial hypertension and antihypertensive medication was an excluding criterion. After otorhinolaryngologic examination and estimating the tinnitus magnitude and frequency with pure tone audiometry with Interacoustics, Denmark AC40 audiometer (figure 1), we performed ambulatory blood pressure monitoring (ABPM) using CardioSoft - TONOPORT V - General Electric, USA device with oscilometric BP measuring (figure 2).

The general medical-physical and otorhinolaryngologic examination should always precede the neurootologic investigation. The physical examination includes attention to the identification of any deformity or limitation of movement of the head, neck, spinal column, and joints. Inspection of the scalp and skull are supplemented by careful palpation to attempt to identify a localized thickening of the skull, abnormal scalp vessels, depressions of the skull, abnormal contours and asymmetry of the skull, past craniotomy, and/or other operative scars. In the presence of a large head, particularly in an adult, percussion is performed for evidence of possible hydrocephalus. Auscultation with a stethoscope is performed over the skull and neck for bruits. The effect on the bruit of head turning and compression of the carotid artery, right and left, back and forwards is noted.



Fig. 1. Clinical Audiometer AC 40, Interacustics, Denmark used for measurement of thresholds of audibility and specification of tinnitus.



Fig. 2. CardioSoft – Tonoport – V – General Electric, USA device used for 24-hours ABPM

Figure 3 shows audiogram with bilateral symmetric perceptible acoustic disorder. Then we performed internal examination and indicated a 24-hour ambulatory blood pressure monitoring (ABPM). We used for this examination CardioSoft-Tonoport V-General Electric, USA device, which uses oscillometric method of measurement of blood pressure. None of the group member has previous known diagnosis of arterial hypertension, or use of any medication altering blood pressure level. As some medications can evoke or strengthen tinnitus thoroughly, history of patient's medication is very important (Holcát, 2007).

6.1 Results of our study

The average values of blood pressure during a 24-hour ABPM were 140/89 mmHg. In daytime phase and nighttime phase the average values were 143/91 mmHg and 130/81 mmHg, respectively. Diurnal sign was vanished in 5 individuals (28%). 6 persons (33%) had normotension during a 24-hour ABPM. 12 persons (66%) had hypertension with an average blood pressure values more than 130/80 mmHg for 24-hours. All records had a good quality what permits interpretation of ABPM profile including mean daytime, nighttimes, 24-hour measurement and diurnal pattern loss. Based on these results, we followed the patients from the group with abnormal ABPM results further and also with repeat measurement of casual blood pressure; we confirmed the arterial hypertension with necessity of antihypertensive drug treatment.

7. Discussion

Due to the high number of heterogeneous etiologic factors in subjective and objective tinnitus a complex approach in differential diagnosis is crucial. Tinnitus can be a concomitant

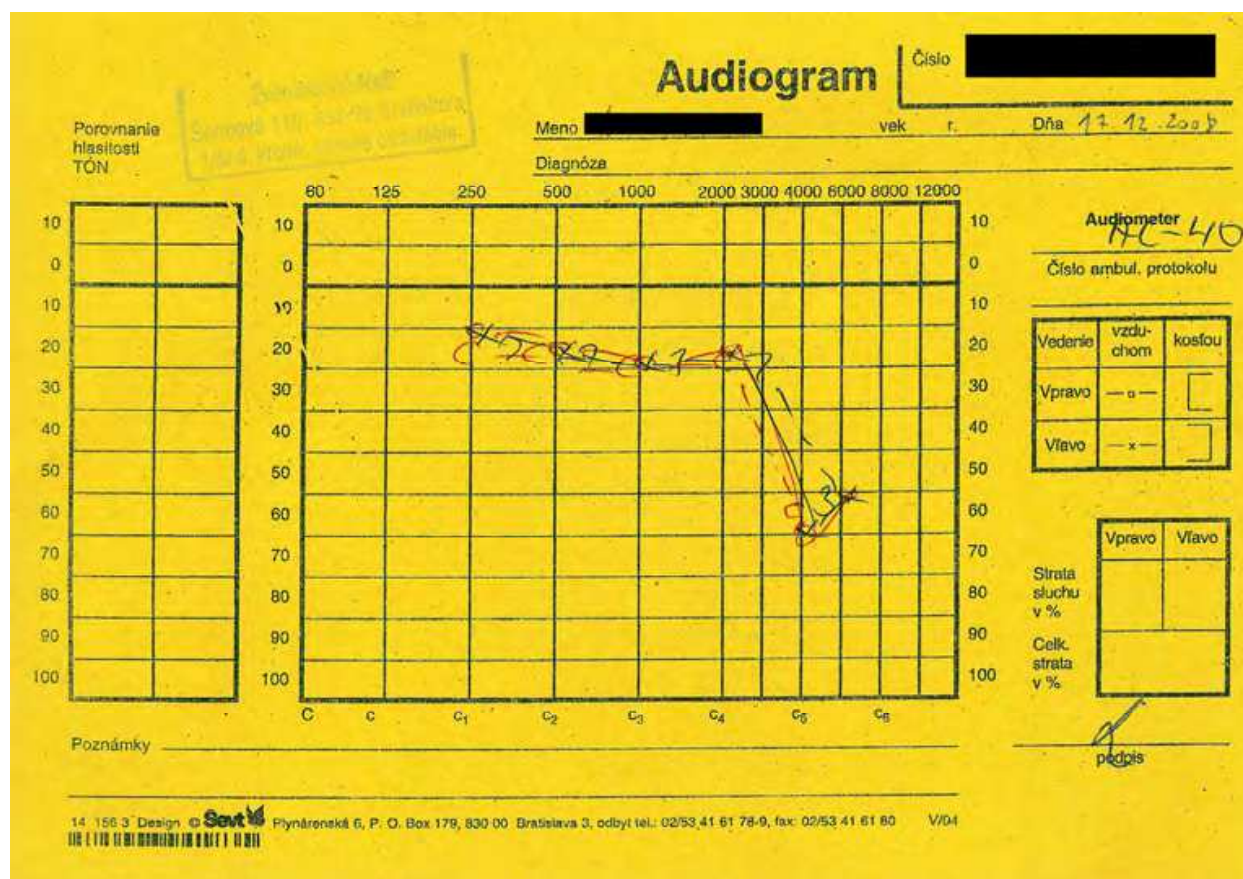


Fig. 3. Audiogram with bilateral symmetric perceptual acoustic disorder, decrease of hearing thresholds bilateral (red line - right ear, blue line - left ear) in frequency 3000-6000 Hz

symptom to many disorders of peripheral statoacoustic apparatus. It is necessary to distinguish whether tinnitus is an acute symptom in onset, e.g. in Menière's disease, acute trauma, inflammation of the inner ear, or it is a part of chronic disease otological in origin (Møller, 2003). Various cardiovascular diseases can be the cause of newly arisen or persistent tinnitus. Among the most important causes there is a reduced perfusion with ischemic changes. Vascular changes can be localized either extra- or intracranially. Among them are stenosis of arteries, haemangioma and glomus caroticum tumours. Changes in the rheology of blood in anaemia or polycythemia can be the cause of tinnitus, as well. It is important to think also about vasculitis as a cause of tinnitus especially among younger patients (König et al., 2006).

The process of differential diagnosis usually starts at the otolaryngologist but interdisciplinary approach to the patient is needed.

Tinnitus is frequent among patients with arterial hypertension, diabetes mellitus, thyroid gland disorders, rheumatologic and neurologic diseases. Two-thirds of our 18 patients with tinnitus had newly diagnosed hypertension during a 24-hour ABPM.

The mean duration of tinnitus in our group was 4 years (range 4 months to 6 years). As none of the patients had formerly diagnosed hypertension, it is likely that hypertension arose after the onset and duration of tinnitus. Diagnosis of hypertension requires serious and careful approach and the use of a 24-hour ABPM should be considered and carried out as soon as possible in patients with presence of tinnitus. Despite that our results are from the

limited number of patients with tinnitus, the diagnostic of arterial hypertension is very important, as several prospective studies have reported that ABPM give better prediction of clinical outcomes compared with conventional clinic or office blood pressure measurements (Staessen et al., 1999).

Tinnitus is a stressor, which can significantly decrease the quality of life, and it can cause serious mental disorders. Some circumstances (stress, acute infection, anaemia) can lead to increase of duration and intensity of tinnitus. Concomitant sleep disorders, anxiety, despair, frustration and depression as well as aggressiveness are related to higher frequency of psychosomatic disorders (Crönlein et al., 2007). Tinnitus then becomes a complex health problem and symptom becomes syndrome (Langguth et al., 2007). Due to the increased incidence of arterial hypertension in this group of patients a 24-hour ABPM is fully indicated. Approximately 17% of the population of USA and Europe and 15% of the population worldwide had manifestation of some form of tinnitus. Only half of them sought for medical help. In 15 - 20% of cases, tinnitus affects quality of life, with repercussions involving sleep habits, concentration, emotional stability and social activities (Axelsson & Ringdahl, 1989; Dobie, 1999). The degree of discomfort caused by this symptom may be mild when perceived by an individual only in particular situations (occurring among approximately 7% of the individuals); moderate when the individual is aware of its existence but does not feel bothered by it; intense when the unpleasant sensation is disturbing and has a negative effect on activities of daily living. It is severe when the symptom becomes unbearable, ever-present and ceaselessly affecting the activities of daily living with concomitant psychic problems. The degree of discomfort, intolerance, or incapacity caused to the individual is often not related to the loudness (sensation of intensity) of the tinnitus (Prestes & Gil, 2009).

8. Conclusions

1. Our result suggests that undetected hypertension is very often in patients with tinnitus.
2. Arterial hypertension is one of many factors involved in tinnitus origin and continuing.
3. ABPM is a suitable tool for hypertension detection and management also in this group of patients.
4. Diagnosing and treating arterial hypertension may not influence tinnitus presence and intensity, but can avoid serious complications of hypertension e.g. stroke, myocardial infarction, heart or renal failure.
5. Diabetes mellitus as one of the components of metabolic syndrome with its complications as micro- and macroangiopathy, cardiovascular autonomic neuropathy is an important and frequent etiologic factor of tinnitus.

9. References

- Axelsson, A. & Ringdahl, A. (1989). Tinnitus--a study of its prevalence and characteristics. *British Journal of Audiology*, Vol.23, No.1, (February 1989), pp. 53-62, ISSN 0300-5364

- Beltowski, J.; Wójcicka, G.; Marciniak, A. & Jamroz, A. (2004). Oxidative stress, nitric oxide production, and renal sodium handling in leptin-induced hypertension. *Life Sciences*, Vol.74, No.24, (April 2004), pp. 2987-3000, ISSN 0024-3205
- Bonora, E. (2006). The metabolic syndrome and cardiovascular disease. *Annals of Medicine*, Vol.38, No.1, (2006), pp. 64-80, ISSN 0785-3890
- Branca, F.; Nikogosian, H. & Lobstein, T. (2007) The Challenge of Obesity in the WHO European Region and the Strategies for Response, In: *World Health Organisation 2007*, 12.09.2010, Available from <http://www.euro.who.int/document/E90711.pdf>
- Cardillo, C.; Campia, U.; Iantorno, M. & Panza, J. (2004). Enhanced vascular activity of endogenous endothelin-1 in obese hypertensive patients. *Hypertension*, Vol.43, No.1, (January 2004), pp. 36-40, ISSN 1524-4563
- Carlyle, M.; Jones, O.; Kuo, J. & Hall, J. (2002). Chronic cardiovascular and renal actions of leptin: role of adrenergic activity. *Hypertension*, Vol.39, No.2, (February 2002), pp. 496-501, ISSN 1524-4563
- Crönlein, T.; Langguth, B.; Geisler, P.; & Hajak, G. (2007). Tinnitus and insomnia *Progress in Brain Research*, Vol.166, (2007), pp. 227-233, ISSN 0079-6123
- da Silva, A.; Kuo, J. & Hall, J. (2004). Role of hypothalamic melanocortin 3/4-receptors in mediating chronic cardiovascular, renal, and metabolic actions of leptin. *Hypertension*, Vol.43, No.6, (June 2004), pp. 1312-1317, ISSN 1524-4563
- Dobie, R. (1999). Indirect A review of randomized clinical trials in tinnitus. *The Laryngoscope*, Vol.109, No.8, (August 1999), pp. 1202-1211, ISSN 0023-852X
- Fábryová, L. (2008). Vzťah viscerálnej obezity ku kardiometabolickým faktorom. *Via practica*, Vol.5, No.3, (2008), pp. 122-125, ISSN 1336-4790
- Feldmann, H.; Lenarz, T. & von Wedel, H. (1992). *Tinnitus*, Georg Thieme Verlag, ISBN 313-770-0027, Stuttgart, Germany
- Flegal, K.; Graubard, B.; Williamson, D. & Gail, M. (2007). Cause-specific excess deaths associated with underweight, overweight, and obesity. *JAMA: The Journal of the American Medical Association*, Vol.298, No.17, (November 2007), pp. 2028-2037, ISSN 1538-3598
- Foley, R.; Wang, C. & Collins A. (2005). Cardiovascular risk factor profile and kidney function stage in the general population. *Mayo Clinic Proceedings*, Vol.80, No.10, (October 2005), pp. 1270-1277, ISSN 0025-6196
- Goodfriend, T. & Calhoun, D. (2004). Resistant hypertension, obesity, sleep apnea, and aldosterone: theory and therapy. *Hypertension*, Vol.43, No.3, (March 2004), pp. 518-524, ISSN 1524-4563
- Goodfriend, T.; Ball, D.; Egan, B.; Campbell, W. & Nithipatikom, K. (2004). Epoxy-keto derivative of linoleic acid stimulates aldosterone secretion. *Hypertension*, Vol.43, No.2, (February 2004), pp. 358-363, ISSN 1524-4563
- Haslam, D. & James, W. (2005). Obesity. *Lancet*, Vol.366, No.9492, (October 2005), pp. 1197-1209, ISSN 0140-6736
- Holcát, M. (2007). Tinnitus and diabetes. *Vnitřní Lékařství*, Vol.53, No.5, (May 2007), pp. 534-536, ISSN 0042-773X

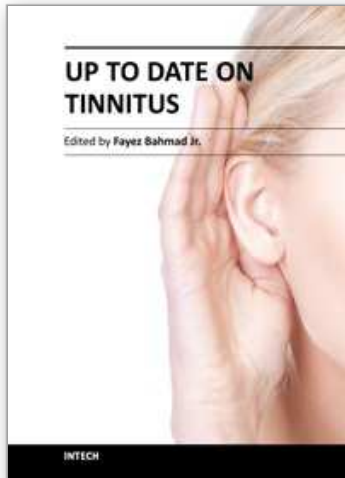
- Howell, S.; Sear, J. & Foëx, P. (2004). Hypertension, hypertensive heart disease and perioperative cardiac risk. *British Journal of Anaesthesia*, Vol.92, No.4, (April 2004), pp. 570-583, ISSN 0007-0912
- Jern, S.; Bergbrant, A.; Björntorp, P. & Hansson, L. (1992). Relation of central hemodynamics to obesity and body fat distribution. *Hypertension*, Vol.19, No.6, (June 1992), pp. 520-527, ISSN 0194-911X
- Julius, S.; Valentini, M. & Palatini, P. (2000). Overweight and hypertension : a 2-way street?. *Hypertension*, Vol.35, No.3, (March 2000), pp. 807-813, ISSN 1524-4563
- Kambham, N.; Markowitz, G; Valeri, A.; Lin, J. & D'Agati, V. (2001). Obesity-related glomerulopathy: an emerging epidemic. *Kidney International*, Vol.59, No.4, (April 2001), pp. 1498-1509, ISSN 0085-2538
- Kastarinen, M.; Nissinen, A.; Vartiainen, E.; Jousilahti, P.; Korhonen, H.; Puska, P. & Tuomilehto. (2000). Blood pressure levels and obesity trends in hypertensive and normotensive Finnish population from 1982 to 1997. *Journal of Hypertension*, Vol.18, No.3, (March 2000), pp. 255-262, ISSN 0263-6352
- Keller, G.; Zimmer, G.; Mall, G.; Ritz, E. & Amann, K. (2003). Nephron number in patients with primary hypertension. *The New England Journal of Medicine*, Vol.348, No.2, (January 2003), pp. 101-108, ISSN 1533-4406
- König, O.; Schaette, R.; Kempfer, R. & Gross, M. (2006). Course of hearing loss and occurrence of tinnitus. *Hearing Research*, Vol.221, No.1-2, (November 2006), pp. 59-64, ISSN 0378-5955
- Krauss, R.; Winston, M. & Fletcher, B. (1998). Obesity : impact on cardiovascular disease. *Circulation*, Vol.98, No.14, (October 1998), pp. 1472-1476, ISSN 1524-4539
- Kurtz, T. & Klein, U. (2009). Next generation multifunctional angiotensin receptor blockers. *Hypertension Research: Official Journal of the Japanese Society of Hypertension*, Vol.32, No.10, (October 2009), pp. 826-834, ISSN 1348-4214
- Langguth, B.; Kleinjung, T.; Fischer, B.; Hajak, G.; Eichhammer, P. & Sand, P. (2007). Tinnitus severity, depression, and the big five personality traits. *Progress in Brain Research*, Vol.166, (2007), pp. 221-225, ISSN 0079-6123
- Marsh, J.; Fontes, M.; Killinger, S.; Pawlak, D.; Polson, J. & Dampney, R. (2003). Cardiovascular responses evoked by leptin acting on neurons in the ventromedial and dorsomedial hypothalamus. *Hypertension*, Vol.42, No.4, (October 2003), pp. 488-493, ISSN 1524-4563
- Messerli, F.; Christie, B.; DeCarvalho, J.; Aristimuno, G.; Suarez, D.; Dreslinski, G. & Frohlich, E. (1981). Obesity and essential hypertension. Hemodynamics, intravascular volume, sodium excretion, and plasma renin activity. *Archives of Internal Medicine*, Vol.141, No.1, (January 1981), pp. 81-85, ISSN 0003-9926
- Miall, W.; Heneage, P.; Khosla, T.; Lovell, H. & Moore, F. (1967). Factors influencing the degree of resemblance in arterial pressure of close relatives. *Clinical Science*, Vol.33, No.2, (1967), pp. 271-283, ISSN 0143-5221
- Møller, A. (2003). Pathophysiology of tinnitus. *Otolaryngologic Clinics of North America*, Vol.36, No.2, (April 2003), pp. 249-266, ISSN 0030-6665

- Møller, A. (2007). Tinnitus: presence and future. *Progress in Brain Research*, Vol.166, (2007), pp. 3-16, ISSN 0079-6123
- Morris, J. (2007). Uses of Epidemiology. *International Journal of Epidemiology*, Vol. 36, No.6, (2007), pp. 1165-1172, ISSN: 0300-5771, 1464-3685
- Narkiewicz, K. (2006). Obesity and hypertension – the issue is more complex than we thought. *Nephrology Dialysis Transplantation*, Vol.21, No.4, (February 2009), pp. 264-267, ISSN 0931-0509
- Nowbar, S.; Burkart, K.; Gonzales, R.; Fedorowicz, A.; Gozansky, W.; Gaudio, J.; Taylor, M. & Zwillich, C. (2004). Obesity-associated hypoventilation in hospitalized patients: prevalence, effects, and outcome. *The American Journal of Medicine*, Vol.116, No.1, (January 2004), pp. 1-7, ISSN 0002-9343
- Pillsbury, H. (1986). Hypertension, hyperlipoproteinemia, chronic noise exposure: is there synergism in cochlear pathology?. *The Laryngoscope*, Vol.96, No.10, (October 1986), pp. 1112-1138, ISSN 0023-852X
- Praga, M.; Hernández, E.; Herrero, J.; Morales, E.; Revilla, Y.; Díaz-González, R. & Rodicio, J. (2000). Influence of obesity on the appearance of proteinuria and renal insufficiency after unilateral nephrectomy. *International Kidney International*, Vol.58, No.5, (November 2000), pp. 2111-2118, ISSN 0085-2538
- Prestes, R. & Gil, D. (2009). Impact of Tinnitus on Quality of Life, Loudness and Pitch Match, and High-Frequency Audiometry. *International Tinnitus Journal*, Vol. 15, No.2, (2009), pp. 134-138, ISSN 0946-5448
- Rahmouni, K.; Correia, M.; Haynes, W. & Mark, A. (2005). Obesity-associated hypertension: new insights into mechanisms. *Hypertension*, Vol.45, No.1, (January 2005), pp. 9-14, ISSN 1524-4563
- Redon, J.; Cifkova, R.; Laurent, S.; Nilsson, P.; Narkiewicz, K.; Erdine, S. & Mancia, G. (2008). The metabolic syndrome in hypertension: European society of hypertension position statement. *Journal of Hypertension*, Vol.26, No.10, (October 2008), pp. 1891-1900, ISSN 0263-6352
- Schünemann, H.; Dorn J.; Grant, B.; Winkelstein, W. & Trevisan, M. (2000). Pulmonary function is a long-term predictor of mortality in the general population: 29-year follow-up of the Buffalo Health Study. *Chest*, Vol.118, No.3, (September 2000), pp. 656-664, ISSN 0012-3692
- Šimková, I. (2009). *Plúcna hypertenzia očami kardiológa*, SAP – Slovak Academic Press s.r.o., ISBN: 978-80-8095-051-4, Bratislava, Slovakia
- Sin, D. & Man, S. (2003). Impaired lung function and serum leptin in men and women with normal body weight: a population based study. *Thorax*, Vol.58, No.8, (August 2003), pp. 695-698, ISSN 0040-6376
- Staessen, J.; Thijs, L.; Fagard, R.; O'Brien, E.; Clement, D.; de Leeuw, P.; Mancia, G.; Nachev, C.; Palatini, P.; Parati, G.; Tuomilehto, J. & Webster, J. (1999). Predicting cardiovascular risk using conventional vs ambulatory blood pressure in older patients with systolic hypertension. Systolic Hypertension in Europe Trial Investigators. *JAMA: The Journal of the American Medical Association*, Vol.286, No.6, (August 1999), pp. 539-546, ISSN 0098-7484

Svačina, Š. (2002). *Obezita a psychofarmaka*, Triton, ISBN: 80-7254-253-2, Prague, Czech Republic

IntechOpen

IntechOpen



Up to Date on Tinnitus

Edited by Prof. Faye Bahmad

ISBN 978-953-307-655-3

Hard cover, 186 pages

Publisher InTech

Published online 22, December, 2011

Published in print edition December, 2011

Up to Date on Tinnitus encompasses both theoretical background on the different forms of tinnitus and a detailed knowledge on state-of-the-art treatment for tinnitus, written for clinicians by clinicians and researchers. Realizing the complexity of tinnitus has highlighted the importance of interdisciplinary research. Therefore, all the authors contributing to the this book were chosen from many specialties of medicine including surgery, psychology, and neuroscience, and came from diverse areas of expertise, such as Neurology, Otolaryngology, Psychiatry, Clinical and Experimental Psychology and Dentistry.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Ludovit Gaspar, Michal Makovnik, Matej Bendzala, Stella Hlinstakova, Ivan Ocadlik and Eva Gasparova (2011). Components of Metabolic Syndrome and Their Relation to Tinnitus, Up to Date on Tinnitus, Prof. Faye Bahmad (Ed.), ISBN: 978-953-307-655-3, InTech, Available from: <http://www.intechopen.com/books/up-to-date-on-tinnitus/components-of-metabolic-syndrome-and-their-relation-to-tinnitus>

INTECH
open science | open minds

InTech Europe

University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China

Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821

© 2011 The Author(s). Licensee IntechOpen. This is an open access article distributed under the terms of the [Creative Commons Attribution 3.0 License](#), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

IntechOpen

IntechOpen