17

Sagittal Abdominal Diameter as the Anthropometric Measure of Cardiovascular Risk

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1. Introduction

Obesity has a profound impact on the cardiovascular disease development, and is associated with a reduced overall survival. There is a strong correlation between the central (abdominal) type of obesity and the cardiovascular and metabolic diseases. Among a variety of anthropometric measurements of the abdominal fat size, sagittal abdominal diameter has been proposed as the valid measurement of the visceral fat mass and cardiometabolic risk level. Many studies have analyzed the relationship between sagittal abdominal diameter (SAD), visceral fat area, and different markers of cardiometabolic disturbances with respect to age, gender and ethnicity. Some of them have offered the cut-off values that could be useful in clinical practice, in identifying individuals who are at higher risk of comorbidities of the obesity. Using the principles of rough set theory, based on producing *If-Then* rules, we have developed a model that allows better applicability of SAD in identifying patients at higher cardiovascular risk. In this chapter, we describe the basic principles of the proposed model. Furthermore, we give a broad overview of the main concerns regarding the significance of SAD and its use in diagnosing the abdominal obesity and predicting the adverse cardiometabolic outcomes.

2. Obesity as a cardiovascular risk factor

The prevalence of obesity has increased dramatically worldwide during the past few decades. Obesity is recognized as an independent factor for the development of the cardiovascular diseases. It also predisposes to the development of other cardiovascular risk factors.

Obesity implies increased body weight due to the enlargement of the adipose tissue to the extent that impairs health. Regional obesity appears to be an important indicator of the risk level. Thus, the diagnosis of obesity depends on three main aspects: relative weight (total body mass with relation to body height), total body fat, and fat distribution.

Body mass index (BMI) has been widely accepted as a simple and the most practical measure of fatness in clinical and epidemiological surveys, eventhough it doesn't distinguish fat from lean body mass. In fact, it is an indicator of the nutritional status, not a measure of body fat mass. It has been shown that BMI≥25 kg/m^2 is associated with increased morbidity, while BMI≥30 kg/m^2 carries increased risk for both morbidity and mortality, primarily from diabetes and cardiovascular diseases (Irribaren et al., 1995). However, recent studies showed that BMI can be a reliable predictor of cardiovascular mortality only in severe obesity (Romero-Corral et al., 2006). The category of overweight people (BMI: 25-29.9 kg/m^2) seems to be the most confusing, especially from the aspect of the therapeutic approach.

BMI doesn't provide sufficient information about fat mass. Therefore, body composition assessment is necessary for the diagnosis of obesity and prediction of its comorbidities. It discriminates individuals with true excess body fat from those with "normal weight obesity", as well as from overweight individuals with normal body fat mass. Using a cut-off value of 30% body fat, Marques-Vidal et al. (2008) reported prevalence of "normal weight obesity" of 10.1% in women, and 3.2% in men, with increasing prevalence with aging.

Specific fat distribution determines the risk level more accurately than the total body fatness *per se*. Excess adipose tissue in the abdominal region is more hazardous than the overall obesity, due to higher visceral fat deposition. Furthermore, it is associated with greater risk of cardiovascular diseases, metabolic disorders and type 2 diabetes mellitus (Després et al., 1990; Molarius & Seidell, 1998). Central or abdominal obesity (firstly assigned as android type of obesity) has been identified as a risk factor for the cardiovascular diseases, as well as a symptom of metabolic syndrome. Normal weight subjects with higher visceral fat mass are at a higher risk (metabolically obese normal weight subjects). In addition, obese subjects with normal visceral fat mass can present with normal metabolic profile (metabolically healthy obese subjects) (Ruderman et al., 1998; Sims, 2001).

2.1 Abdominal obesity

It is well known that the risk of cardiovascular and metabolic abnormalities is determined by specific distribution of the adipose tissue. Abdominal (central) obesity is associated with dyslipidemia, impaired fasting glucose, insulin resistance and hypertension, which result in increased risk of cardio- and cerebrovascular diseases, and consequently premature death (Guzzaloni, 2009).

Adverse effects of the abdominal obesity have been supported by many studies of the metabolism and endocrine activity of adipocytes from different regions of the abdominal adipose tissue. Abdominal fat includes two morphologically and functionally different depots: subcutaneous (superficial) and deep, visceral (intraabdominal). The latter is located in the abdominal cavity and includes intraperitoneal (omental and mesenterial) adipose tissue, which makes 80% of the intraabdominal fat mass, and retroperitoneal adipose tissue, which makes 20% of the intraabdominal fat mass (Misra&Vikram, 2003). Abdominal obesity can reflect expansion of either subcutaneous or visceral depot, or a combination of excess fat in both depots. However, visceral adipose tissue compartment has been considered more important in pathogenesis of the obesity complications. It is responsible for the development of insulin resistance, glucose intolerance and type 2 diabetes mellitus. According to Brochu

et al. (2000), visceral fat depot explains 10% of variability of insulin resistance and 16% of blood glucose. Visceral adipose tissue enlargement is mainly associated with lower values of HDL-cholesterol, elevated tryglicerides, apolipoprotein B (Couillard et al., 1996), as well as with elevated atherogenic lipoprotein subfractions and reduced concentration of HDL particles (Nakata et al., 2010). It positivly correlates with glucose intolerance and hyperinsulinemia (Wajchenberg, 2000), as well as the markers of the proinflammatory and prothrombotic state.

Visceral adipose tissue function plays a crucial role in the development of metabolic abnormalities and insulin resistance, mainly due to the direct access of intraperitoneal adipose tissue to the liver through the portal circulation (Matsuzawa et al. 1995, Bosello & Zamboni, 2000). In comparison to the subcutaneous adipose tissue, visceral adipose tissue contains higher number of adipocytes per unit mass, higher number of endothelial cells in the stromal vascular fraction, higher β_3-adrenoreceptor and α_2-adrenergic receptor sensitivity, and it is better vascularized (Misra & Vikram, 2003; van Harmelen et al., 2004). Visceral adipocytes are more metabolically active, have higher lipolytic activity when stimulated by catecholamines, and are poorly responsive to the antilipolythic action of insulin. In addition, they secrete more proinflammatory (interleukin-6, interleukin-8, interleukin-1β) and prothrombotic (plasminogen-acivator inhibitor-1) adipokines. Obesity is characterized by an increased number of β_3- and decreased number of α_2-adrenergic receptors, decreased insulin activity, increased activity of lipoprotein-lipase and acylation-stimulating protein, with higher upload of triglycerides and lower postprandial suppression of lipolysis (Wajchenberg, 2000; van Herpen & Schrauwen-Hinderling, 2008). All of the above mentioned changes in the visceral adipose tissue provide increased release of free fatty acids and their flux towards the liver, where they induce gluconeogenesis, synthesis of triglycerides and apolipoprotein-B rich lipoproteins, as well as impairment of insulin action (Lonnquist et al., 1995, Freedland, 2004). Free fatty acids also exhibit proarrhythmic properties which explains association between the visceral fat and sudden death (Empana et al. 2004). On the other hand, obese adipose tissue is characterized by impairment of blood flow, development of hypoxia and local inflammation, infiltration by macrophages, and by disturbances in secretion of adipokines, which all together result in insulin resistance and systemic inflammation (Berg & Scherer, 2005; Coppack, 2005; Goossens, 2008).

Computerized tomography (CT) and magnetic resonance imaging (MRI), performed at the L_4-L_5 level, are the most reliable anatomical methods for abdominal fat assessment, since they discriminate between the subcutaneous and the visceral fat depots (van der Kooy et al., 1993). However, these methods are expensive, not feasible and unportable, which makes their use in clinical and epidemiological practice limited. Anthropometric parameters are more suitable as they are inexpensive, non-invasive and simple. Besides, most of them show a strong correlation with visceral abdominal fat size.

2.1.1 Anthropometric parameters of abdominal obesity

Several anthropometric indicators of abdominal obesity have been developed to measure abdominal adipose tissue mass. Some of them are presented in the form of ratios, especially the ones that incorporate body height, which gives more realistic picture of body proportions. On the other hand, it is difficult to interpret them biologically (Bouchard et al., 1990). Many studies have compared them in order to demonstrate advantages of a particular

parameter in predicting the risk and visceral fat mass. As a rule, an ideal anthropometric measure of abdominal adiposity should predict individual cardiometabolic risk and clearly show effects of different preventative and therapeutic approaches.

Waist-to-hip and waist-to-thigh ratios (WHR and WTR, respectively) were originally proposed as the key determinants of android and gynoid obesity (Krotkiewski et al., 1983; Molarius & Seidell, 1998). WHR has been most commonly used in identifying abdominal fat distribution. Waist circumference alone has received more attention in management of obesity since it requires only one measurement. It showed to be a better predictor of visceral fat volume and related cardiovascular risk profile than WHR (Després et al., 1991; Pouilot et al., 1994; Vissher et al., 2001; Wajchenberg, 2000; Logfren et al., 2004). Moreover, changes in waist circumference better reflect changes in cardiovascular risk factors. It is widely accepted as a surrogat marker of abdominal fat. On the other hand, waist circumference has been criticized for measuring both visceral and subcutaneous adipose tissues (Molarius & Seidell, 1998). Other studies suggest waist-to-height ratio (WHTR) as the better marker because it correlates highly with cardiometabolic risk factors (Hsieh & Yoshinaga, 1995; Ashwell, 2005). Conicity index, which is based on cylindrical shape of the body, has also been introduced as a potentially useful measure of abdominal adiposity (Valdez, 1991). However, it is considered to be very complex because it requires calculations from several different anhropometric values (Molarius & Seidell, 1998).

Sagittal abdominal diameter (SAD), or abdominal height was first demonstrated by Kvist et al. (1988) to be a good correlate of visceral adipose tissue volume, observed by CT. Sjöstrom et al. (1994) proposed the use of sagittal abdominal diameter in the assessment of visceral fat mass. Soon after, Richelsen and Pedersen (1995) confirmed its value in assessing the abdominal fatness and prediction of the metabolic risk profile.

3. Sagittal abdominal diameter – Visceral fat measure and cardiovascular risk predictor

First measures of SAD were done on CT images and showed good predictive values in the assessment of visceral adipose tissue volume (Kvist et al., 1988). SAD, thus, may be a reliable represent of the visceral fat size. Moreover, two recent studies confirmed that SAD was a stronger predictor of metabolic syndrome than the whole visceral fat area (Valsamakis et al., 2008; Hoenig, 2010), which could pointed to functionally different adipose tissue in the midline.

External, anthropometric measurement of SAD is usually done using Holtain-Kahn abdominal caliper, at the level of the iliac crest, which approximates to the L_4-L_5 interspace (Figure 1). Since it has been proposed, SAD has been considered as more closely related to visceral fat mass than the other anthropometric measures beacuse it is measured in a supine position, when a subcutaneous fat is moved to the sides of the waist (van der Kooy et al., 1993; Mukunddem-Petersen, 2006; Sampaio et al., 2007). Measuring SAD in that position reflects the width of intraabdominal fat in the antero-posterior plane, like on CT or MRI images. At the same value of SAD, an increase of waist circumference may reflect increase of subcutaneous adipose tissue size.

Many studies confirmed strong association between anthropometrically assessed SAD and visceral adipose tissue area. Anjana et al. (2004), Sampaio et al. (2007) and Yim et al. (2010)

reported stronger correlation between SAD and visceral fat area, comparing to waist circumference. Zamboni et al. (1998) found better association between SAD and visceral fat area in lean and moderately overweight subjects than in the obese. Regarding to gender, some studies have found better correlation in men, ranged between 0.61 and 0.82 (van der Kooy et al., 1993; Zamboni et al., 1998), while others have reported stronger relationship in women, with the range between 0.52 and 0.87 (Pouilot et al., 1994, Sampaio et al., 2007). Some studies demonstrated that SAD is a better predictor of visceral fat than waist circumference in men (Després et al., 1991; van der Kooy et al., 1993), while others gave opposite results in women (Sampaio et al., 2007). In the MRI study, van der Kooy et al. (1993) showed that SAD was superior to waist circumference and WHR in assessing visceral fat mass changes in men, while waist circumference and WHR were better measures in women.

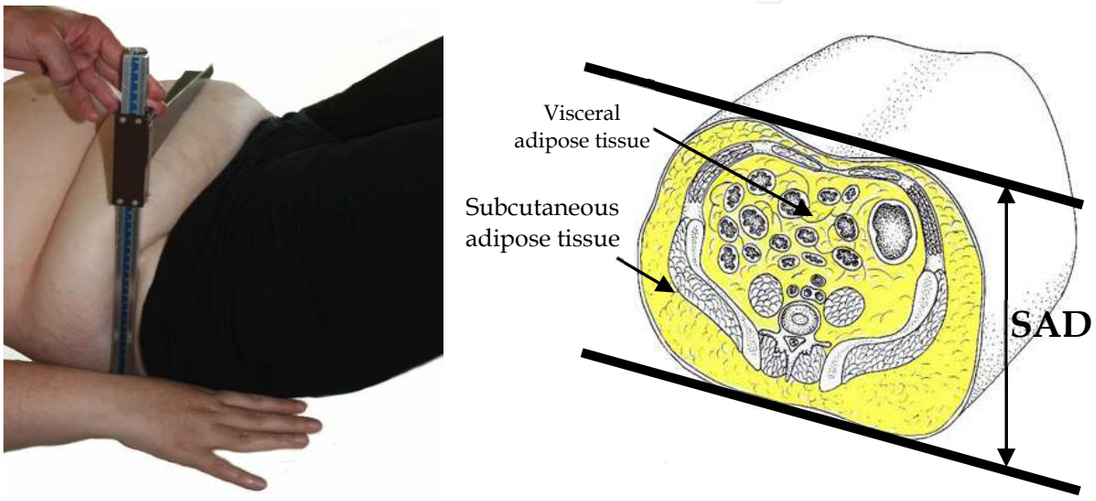


Fig. 1. Measurement of sagittal abdominal diameter (horizontal section of abdomen)

Comparing to other anthropometric measures, like waist circumference or WHR, SAD has been showed to have better correlation with biochemical and hemodynamic parameters associated with cardiovascular diseases and metabolic syndrome in both, lean and obese subjects:

- Higher values of SAD correlate with **atherogenic lipid profile** including elevated triglycerides, reduced HDL-cholesterol, elevated apolipoprotein B levels, and atherogenic lipoprotein subfractions (Pouilot et al., 1994; Sjöstrom, 1994; Richelsen & Pedersen, 1995; Öhrvall et al., 2000; Turcato et al., 2000; Sampaio et al., 2007; Petersson et al., 2007; Nakata et al., 2010).
- SAD is an important factor in prediction of **glucose intolerance and insulin resistance** (Pouliot et al., 1994; Öhrvall et al., 2000; Gustat et al., 2000; Risérus et al., 2004; Mazzali et al., 2006; Vasques et al., 2009a). Risérus et al. (2004) found that SAD was a strong predictor of hyperproinsulinemia, higher values of C-peptide and lower levels of insulin-like growth factor (IGF) binding protein-1.
- SAD highly correlates with **inflammatory and prothrombotic markers**, like CRP (Mazzali et al., 2006; Petersson et al. 2007; Nakata et al., 2010) or PAI-1 (Öhrvall et al., 2000). According to Petersson et al. (2007), every one-centimetre increase in SAD is followed by an increase of C-reactive protein (CRP) by 0.41 mg/L. The same authors

suggested that SAD may carry information concerning inflammatory status and possibly insulin resistance beyond that of other measures of obesity and fat distribution.
- SAD is associated with **blood pressure** and predicts hypertension (Öhrvall et al., 2000; Strazzulo et al., 2001). Gustat et al. (2000) highlight that SAD can predict blood pressure when other measures cannot.
- Among **adipokines**, SAD correlates with leptin and adiponectin blood levels (Mazzali et al., 2006); it also correlates with 11β-hydroxysteroid dehydrogenase-type 1 (11β-HSD-1) mRNA expression in visceral adipose tissue, which is known to be associated with features of metabolic syndrome (Desbriere et al., 2006).

Our previous results showed significantly higher values of SAD in obese women who displayed lipid and lipoprotein disturbances and hyperinsulinemia, comparing to healthy normal-weight women (Stokić et al, 1996, Stokić & Ivković-Lazar, 1996). According to our unpublished results (a group of 1090 men and 1231 women of different BMI-values, aged 18-79 years), SAD showed a significant correlation with sistolic and diastolic blood pressure and glycaemia in both genders, and with total cholesterol and triglycerides in men. In both, men and women, SAD showed best correlation with diastolic blood pressure (men: r=0.340, women:r=0.198). By discriminative analysis we determined range of SAD that correspond with lowest risk (men: 20.12-24.97 cm; women: 19.85-24.75 cm), while extremely high values were in the following ranges: 32.58-34.65 cm (men) and 29.87-31.80 cm (women).

In the large longitudinal study, Iribarren et al. (2006) confirmed utility of SAD in prediction of cardiovascular risk, independently of body mass index. Reed et al. (2003) found association between SAD and carotid artery intima-media thickness.

Empana et al. (2004) established that age-adjusted risk of sudden death increases linearly with SAD increasement in both, normal-weight and overweight men. SAD has been shown to be an independent risk factor for death and morbidity in patients in the intensive care unit (Paolini et al., 2010).

There are also two indexes derived from SAD:

- SAD-to-body height ratio (SAD/H) has been showed as slightly better predictor than SAD alone. Kumlin et al. (1997) reported that SAD/H is a strong predictor of Framingham coronary risk score.
- SAD to mid-thigh circumference ratio, or abdominal diameter index (ADI), has been proposed by Kahn (1993) as even better predictor of cardiovascular risk, which was confirmed by Smith et al. (2005).

3.1 Application of sagittal abdominal diameter in elderly

Aging process is characterized by body composition changes that could not be captured by standard anthropometric measures like BMI. Increasing of total body fat occurs in both genders, which is followed by decreasing of muscle mass (sarcopenic obesity) and body fat redistribution in terms of changes from peripheral to central (abdominal) pattern (Prentice & Jebb, 2001; Greenlund & Nair, 2003; Davidson & Getz, 2004). In women, menopause plays important role in transitioning from a premenopausal gynoid (gluteo-femoral) to a postmenopausal central (visceral) pattern of body fat distribution and increase in total body fat (Movsesyan et al., 2003). Even in early menopause women have a 49% greater visceral fat

mass comparing with premenopausal women (Toth et al., 2000). Together with other physiological and life style changes caused by aging, age-associated central fat distribution contributes to cardiovascular morbidity and mortality. That is why is highly recommended to assess central adiposity in older persons (Dorner & Rieder, 2011).

Some evidences pointed to better predictive value of SAD in younger individuals (Iribarren et al., 2006, Mukuddem-Petersen et al., 2004). However, SAD could be also very useful indicator in the elderly. According to Turcato et al. (2000), SAD and waist circumference are the anthropometric parameters which are the most closely related to cardiovascular risk factors in women and men aged from 67 to 78 years, independently of BMI. Harris et al. (2000) and Snijder et al. (2002) found that SAD was even better predictor of visceral fat area in subjects older than 70 years, comparing to waist circumference, while Mukuddem-Petersen et al. (2004) indicated that SAD had no advantages over simpler and more commonly used anthropometric measures such as waist circumference, regarding to their associations with components of the metabolic syndrome in older subjects.

3.2 Gender and ethnic specific usage of sagittal abdominal diameter

Men and women have different adipose tissue topography. Fat deposition is gluteo-femoral region is more typical for women, while men show preferential abdominal fat accumulation. Within abdominal region, visceral fat compartment is more predominant in men, while women have higher size of subcutaneous fat compartment (Anjana et al., 2004). Men and women also have different dynamics of losing visceral and subcutaneous fat during weight loss - men lose more visceral, and women lose more subcutaneous fat (van der Kooy et al., 1993).

It is assumed that SAD has a stronger capacity to predict visceral fat area, insulin resistance, and cardiometabolic risk in men, because they have higher visceral fat mass (Risérus et al., 2004). Vasques et al. (2009b) confirmed its greater ability to identify insulin resistance in men. However, according to the results given by Mukuddem-Petersen et al. (2004) SAD is a stronger predictor of cardiovascular risk in women, while Duarte-Pimentel et al. (2010) recommend SAD as a marker of central adiposity preferentially in women.

Some ethnic groups show different pattern of body fat distribution and different susceptibility to insulin resistance. For example, Asian Indians have greater abdominal fat mass than Europeans of the same nutrition level (Anjana et al., 2004), while middle-aged and older African-American men and women have lower visceral fat than Hispanic and white men and women (Carroll et al., 2008). There are race and ethnical differences in the relationship between body fat distribution and health risk factors; thus, anthropometric measures could differ regarding the predictive capacity of cardiovascular risk factors. However, according to the results of several studies, SAD seems to be an excellent marker of metabolic and cardiovascular risk factors, irrespectively of national origin or ethnic background (Hwu et al., 2003; Valsamakis et al., 2004; Petersson et al., 2007, Iribarren et al., 2006).

3.3 Treshold values for sagittal abdominal diameter – Reflection of critical visceral fat mass or cardiometabolic risk

In spite of many evidences that SAD is very good in capturing the cardiometabolic risk, its use in clinical practice is limited due to a lack of specific cut-offs. Some authors have

proposed cut-off values for SAD using different criteria, usually its correlation with cardiometabolic parameters and visceral fat area (Table 1). These results were mostly obtained using Receiver Operating Characteristics (ROC) curves, or linear regression analysis, and vary from 19.3 cm (Sampaio et al., 2007) to 27.6 cm (Valsamakis et al., 2004). Our results were obtained using the principles of rough set theory that will be described below. They were derived from evaluation of relationship between SAD and cardiovascular risk factors.

We produced "transparent", semantic model which can be easily analyzed. The most important information which can be extracted from semantic models is concerned with the meaning and importance of its elements, as well as with the relations between them. Data were represented in the form of a table with rows containing the objects and columns containing the attributes, and model was produced in the multiple sets containing *If – Then* rules.

SAD (cut-offs)	Population	Criterion	Author
≥25 cm	Men/Women	Association with multiple metabolic disorders	Pouliot et al., 1994
≥22.8 cm ≥25.2 cm	Men Women	Corresponds to 130 cm^2 of visceral fat area	Lemieux et al., 1996
≥27.6 cm	Men	Predictive value for metabolic syndrome	Valsamakis et al., 2004
≥23 cm	Men	Predictive value for sudden death	Empana et al., 2004
≥20.5 cm ≥19.3 cm	Men Women	Corresponds to 100 cm^2 of visceral area	Sampaio et al., 2007
≥20 cm	Men	Predictive value for insulin resistance	Vasques et al., 2009a
≥22.2 cm ≥20.1 cm	Men Women	Predictive value for an elevated cardiometabolic risk score	Risérus et al., 2010
≥23.1 cm ≥20.1 cm	Men Women	Corresponds to altered waist circumference (>102 cm for men and >88 cm for women)	Duarte Pimentel et al., 2010
≥24.3 cm	Men/Women	Corresponds to increased cardiovascular risk in overweight and obese individuals	Stokić et al., 2010

Table 1. Recommended cut-off values for SAD

4. Model for the better applicability of SAD in identifying patients at higher cardiovascular risk

Modern healthcare and computer science are fields that are interlaced to form the field of medical informatics. As mentioned by Øhrn (1999), Blois and Shortliffe define medical informatics as "the rapidly developing scientific field that deals with the storage, retrieval and optimal use of biomedical information, data, and knowledge for problem solving and decision making." In its broadest sense, medical informatics can be said to concern itself with the management of information in the context of modern healthcare. According to Øhrn (1999) current research in the field of medical informatics covers a wide array of topics, including:

- **Data acquisition:** Capturing and recording of the medical data usually include things that are not easily recorded or precisely defined.
- **Medical vocabularies:** Medical data has to be represented in machine-readable form.
- **Electronic medical records:** An electronic medical record has to be searchable. Its content should be structured internally.
- **Decision support systems:** These are computer programs that help clinicians make clinical decisions.
- **Deployment barriers:** The barriers of deployment may be technical, operational, organizational and legal nature. Often, systems that may prove successful in research settings do not make it into clinical use.
- **Confidentiality issues:** Medical information is often sensitive and with a potential for misuse by third-parties.

Obviously, as a main prerequisite there is a existence of medical dataset or database. Medical datasets are often described as incomplete, sparse, vague, fuzzy, etc. According to Greco et al. (1998), the rough sets theory has often proved to be an excellent mathematical tool for the analysis of a vague description of objects.

On the base of the table-organized data it is possible to produce semantic model which provides information about meaning and importance of its elements, as well as the relationship between them (Brtka et al., 2008; Stokić et al., 2010). In order to investigate relationship between SAD and anthropometric and cardiovascular risk factors, we used methodology based on rough set theory (Pawlak et al, 1995, Pawlak & Skowron, 2007) applied to table–organized data with producing decision rules in the *If -Then* form.

Our study included 1334 subjects (700 women and 634 men), aged 43.49±10.43 years. Following parameters were analyzed: age (years), body mass index (kg/m^2), SAD (cm), body fat mass (%), systolic and diastolic blood pressure (mmHg), total-, LDL- and HDL-cholesterol (mmol/L), triglycerides (mmol/L), fasting plasma glucose (mmol/L), fibrinogen (g/L), uric acid (μmol/L), and 10-year Framingham Risk Score. The experiments were conducted by software system *Rosetta - A Rough Set Toolkit for Analysis of Data*. All numerical attributes were discretized using a simple "equal frequency binning" technique. The attribute SAD was chosen to be the decision attribute while all remaining attributes were chosen to form the set of condition attributes and the set of rules containing rules in the *If – Then* form was generated. The decision attributes (SAD) were classified into three classes:

- 13.00-24.2 cm,
- 24.3-31.6 cm and
- 31.7-36.2 cm.

4.1 Rough-set theory principles

As the mathematical basis of the rough set theory there is the indiscernibility relation. Every object of the universe is described by certain amount of information expressed by means of some attributes used for that object description. The objects characterized by the same information are indiscernible in view of the available information about them.

As in Greco et al. (1998), Øhrn (1999) and Pawlak & Skowron (2007), let U be a universe (finite set of objects), $Q=\{q_1,q_2,...,q_m\}$ is a finite set of attributes, V_q is the domain of attribute q (attribute values) and V is the union of V_q for every $q\in Q$. An information system is the 4-tuple $S=(U,Q,V,f)$ where f is a function such that $f(x,q)\in V_q$ for each $q\in Q$, $x\in U$, called information function.

Let $x,y\in U$ (x and y are two objects e.g. patients), f is an information function and $q\in Q$. Every non–empty subset of attributes P determines an indiscernibility relation on U, denoted by

$$I_P = \{(x,y)\in U\times U : f(x,q) = f(y,q), \forall q\in P\} \quad (1)$$

The I_P is an equivalence relation. The family of all the equivalence classes of the I_P is denoted by U/I_P and the equivalence class containing an element x by $I_P(x)$.

Let us consider a simple example of an information system based on the example from Greco et al. (1998), see Table 2.

Object	Age	Body Mass (BM)	Systolic blood pressure	SAD
x_1	young	good	low	low
x_2	middle-age	medium	low	high
x_3	middle-age	medium	low	low
x_4	old	medium	low	high
x_5	middle-age	good	high	high
x_6	young	medium	high	low

Table 2. Simple example of an information system

In the given table, there is a universe of six objects $U=\{x_1,...,x_6\}$ and each object is described by means of four attributes: *Age*, *Body Mass*, *Systolic Blood Pressure (SBP)* and *SAD*.

If $P=\{Age,BM,SBP\}$ then, by (1), we have: $I_P=\{(x_1,x_1), (x_2,x_2), (x_2,x_3), (x_3,x_2), (x_3,x_3), (x_4,x_4), (x_5,x_5) (x_6,x_6)\}$, $U/I_P=\{\{x_1\}, \{x_2,x_3\}, \{x_4\}, \{x_5\}, \{x_6\}\}$.

4.1.1 The definition of the rough set

The rough set theory proved to be an excellent mathematical tool for the analysis of data in various domains. The information about the real world is given in a form of a decision system. The next definitions are based on Pawlak et al. (1995), Greco et al. (1998), Øhrn (1999) and Pawlak & Skowron (2007).

Let $C \subset Q$ and $D \subset Q$ so that C∩D=∅, where Q is a set of attributes. The attributes from C are called the condition attributes and the attributes from D are called the decision attributes. An information system where the set of condition attributes and the set of decision attributes are defined is called the decision system.

In most cases there is usually one binary decision attribute, while the other attributes are the condition attributes. In the previous example P might be the set of the condition attributes, and the set of decision attributes contains one element: $D=\{SAD\}$.

Let X be a non–empty subset of U and $\varnothing \neq P \subseteq Q$. The set X is approximated by means of P–lower (2) and P–upper (3) approximations of X:

$$\underline{P}(X) = \{x \in U : I_P(x) \subseteq X\} \tag{2}$$

$$\overline{P}(X) = \bigcup_{x \in X} I_P(x) \tag{3}$$

The P–boundary region $Bn(X)$ of X is defined by:

$$Bn(X) = \overline{P}(X) - \underline{P}(X) \tag{4}$$

For example, let us consider a case when the set X contains only those elements where the value of the decision attribute SAD is low: $X=\{x_1,x_3,x_6\}$ (Table 2). Now, we can approximate the set X using only the information contained in P by constructing the P–lower (2) and P–upper (3) approximations of X:

$$\underline{P}(X) = \{x_1, x_6\},$$

$$\overline{P}(X) = \{x_1, x_2, x_3, x_6\}.$$

The P–boundary region (4) of X is:

$$Bn(X) = \{x_2, x_3\}.$$

The reader may notice that the objects x_2 and x_3 have exactly the same values of the condition attributes but different value of the decision attribute. So, they constitute boundary region. We can say that the rough sets can be defined as follows (Pawlak et al., 1995; Pawlak & Skowron, 2007):

The set X is rough (inexact) with respect to I_P, if the boundary region of X is nonempty. The set X is crisp (exact) with respect to I_P, if the boundary region of X is empty.

4.1.2 Data reduction

If we manage to identify equivalence classes then some savings (reductions) are to be made since only one element of the equivalence class is needed to represent the entire equivalence class. An issue of practical importance in reduction is to keep only those attributes that preserve the indiscernibility relation and consequently, the set approximation. The rejected attributes are redundant (superfluous) since their removal cannot worsen the classification (Greco et al., 1998).

Let $\varnothing \neq P \subseteq Q$ and $a \in P$. Attribute a is superfluous in P if $I_P = I_{P-\{a\}}$.

For example, if R= {*Age*, *BM*}, S= {*Age*, *SBP*}, and T= {*BM*, *SBP*} (Table 2), then it is obvious that $I_R = I_P$ and $I_S = I_P$ while $I_T \neq I_P$. This means that R and S are reducts of P, while T is not. The attribute *Age* is indispensable, but the attributes *BM* and *SBP* may be mutually exchanged. This means that it is enough to use reduct {*Age*, *BM*} to estimate the value of the decision attribute *SAD*. In the analog case we can use the reduct {*Age*, *SBP*} to estimate the value of decision attribute *SAD*, but we can not use the reduct {*BM*, *SBP*}.

The calculation of all reducts is very complex but in many practical applications it is not necessary to calculate all the reducts, but only some of them.

4.1.3 Discretization

If we want numerical attributes to be properly incorporated into the classification rules, we should to discretize them. This enables the numerical attributes to be treated as categorical ones, and several algorithms for this purpose are available (Greco et al., 1998; Øhrn, 1999). The goal of the discretization process is to search for intervals or bins, where all cases that fall within the same interval are grouped together. This process can be also seen as the process of classification of the attributes' value set to some classes. The discretization is not specific to the rough set approach but is a pre – required step and is often performed implicitly, behind the scene, using human expert knowledge.

4.1.4 Decision rules

The expression $a = v$, where a is an attribute and v is an attribute value is called the descriptor. Now, it is possible to investigate the rules of the form: *If* α *Then* β. Here α denotes a conjunction (AND logical operator) of descriptors that only involve attributes of some reduct (rule's antecedent) and β (rule's consequent) denote a descriptor $d = v$, where d is a decision attribute and v is the allowed decision value.

For example, if we use the reduct *R={Age, BM}* from Table 2 and *SAD* as a decision attribute, then it is possible to generate the rules with two descriptors in the antecedent part and one descriptor in the consequent part of the rule. It is important to notice that a shorter reduct set means shorter decision rules in the rule set generated from that reduct.

4.2 Application of rough-set theory in identification of patients at higher cardiovascular risk using SAD

Our results showed that SAD could be a clinically useful marker for identification of combination and structure of risk factors by applying different rules in individuals of different BMI-categories.

Lower values of SAD in normal-weight individuals younger than 50 years always corresponded with Framingham risk score <9. However, in normal-weight subjects older than 50 years, SAD couldn't identify those at lower risk. SAD values between 24.3 and 31.6 cm, or even lower, between 13.0 and 24.2 cm, corresponded with Framingham risk score between 9 and 14 (Table 3). It is in agreement with observations which indicate a centralization of adipose tissue with aging, irrespective of BMI. This would represent a category of metabolically obese normal weight individuals.

According to these results, measurement of SAD is not enough for identification of cardiovascular risk in normal-weight individuals. In that regard, it is necessary to include other methods of fat mass assessment, like CT or MRI.

IF												THEN SAD (cm)
Age (years)	Body fat mass (%)	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)	Total cholesterol (mmol/L)	Triglycerides (mmol/L)	LDL-cholesterol (mmol/L)	HDL-cholesterol (mmol/L)	Glycaemia (mmol/L)	Fibrinogen (g/L)	Acidum uricum (μmol/L)	Framingham risk score	
<41	<28.92	<123	>88	<5.37	1.4-2.19	<3.24	1.01-1.21	<4.7	3.06-3.76	273-344	<9	13.0-24.2
	<28.92	123-141	>88	>6.51	<1.4	<3.24	<1.01	4.7-5.4	>3.76	<273	<9	
41-50	<28.92	<123	78-88	<5.37	<1.4	3.24-4.0	<1.01	<4.7	<3.06	<273	<9	
	<28.92	123-141	>88	<5.37	>2.19	<3.24	<1.01	4.7-5.4	>3.76	<273	<9	
	<28.92	<123	78-88	>6.51	1.4-2.19	>4.00	>1.21	>5.4	3.06-3.76	<273	<9	
>50	<28.93	123-141	>88	>6.51	>2.19	3.24-4.0	>1.21	4.7-5.4	3.06-3.76	273-344	9-14	13.0-24.2 OR 24.3-31.6

Table 3. Obtained rules for normal-weight subjects (BMI<26.43 kg/m²)

By examining the decision rules, SAD could point out a group of overweight patients with high level of visceral fat with different combination and composition of cardiovascular risk factors (Table 4). Thus, in overweight individuals aged 41-50 years, with higher fat mass, and SAD between 24.3 and 31.6 cm, we could expect higher values of diastolic blood pressure, total- and LDL-cholesterol, triglycerides, uric acid, as well as Framingham risk score over 14. The same range of SAD in older overweight individuals (>50 years) with higher fat mass, include higher systolic blood pressure, fasting plasma glucose and fibrinogen. On the other side, in younger overweight subjects with lower SAD (<24.3 cm) we could expect lower values of all atherogenic parameters, higher values of HDL-cholesterol and Framingham risk score <9, even if they have higher fat mass.

It means that SAD could identify metabolically healthy overweight individuals.

IF												THEN SAD (cm)
Age (years)	Body fat mass (%)	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)	Total cholesterol (mmol/L)	Triglycerides (mmol/L)	LDL-cholesterol (mmol/L)	HDL-cholesterol (mmol/L)	Glycaemia (mmol/L)	Fibrinogen (g/L)	Acidum uricum (µmol/L)	Framingham risk score	
<41	>39.71	<123	<78	<5.37	<1.4	<3.24	>1.21	4.7-5.4	>3.76	>344	<9	13.0-24.2
41-50	>39.71	<123	>88	>6.51	>2.19	<3.24	1.01-1.21	>5.4	>3.76	>344	9-14	13.0-24.2 OR 24.3-31.6
	28.92-39.71	<123	>88	>6.51	<1.4	>4.0	1.01-1.21	<4.7	>3.76	>344	9-14	
>50	<28.92	123-141	>88	5.37-6.51	>2.19	<3.24	>1.21	4.7-5.4	>3.76	<273	9-14	
	<28.92	<123	78-88	>6.51	1.4-2.19	>4.00	1.01-1.21	>5.4	>3.76	273-344	9-14	
<41	<28.92	123-141	78-88	<5.37	<1.4	<3.24	>1.21	4.7-5.4	3.06-3.76	>344	<9	24.3-31.6
41-50	28.92-39.71	<123	>88	>6.51	>2.19	>4.00	1.01-1.21	4.7-5.4	<3.06	>344	>14	
	>39.71	123-141	>88	>6.51	<1.4	>4.00	>1.21	<4.7	3.06-3.76	<273	9-14	
>50	28.92-39.71	>141	>88	>6.51	>2.19	>3.24	1.01-1.21	>5.4	3.06-3.76	273-344	9-14	
	28.92-39.71	>141	>88	>6.51	>2.19	>4.00	>1.21	<4.7	<3.06	<273	9-14	

Table 4. Obtained rules for overweight subjects (BMI: 26.43-32.52 kg/m^2)

As it is displayed in the Table 5, SAD values above 31.7 cm in obese subjects always correspond to Framingham risk score >14. Younger obese individuals (<41 years) with lower values of SAD usually are at the lower cardiovascular risk (metabolically healthy obese individuals).

IF												THEN SAD (cm)
Age (years)	Body fat mass (%)	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)	Total cholesterol (mmol/L)	Triglycerides (mmol/L)	LDL-cholesterol (mmol/L)	HDL-cholesterol (mmol/L)	Glycaemia (mmol/L)	Fibrinogen (g/L)	Acidum uricum (μmol/L)	Framingham risk score	
<41	>39.71	<123	<78	<5.37	>2.19	<3.24	1.01-1.21	4.7-5.4	3.06-3.76	<273	<9	13.0-24.2
	>39.71	<123	>88	<5.37	<1.4	3.24-4.00	1.01-1.21	<4.7	3.06-3.76	>344		
	28.92-39.71	123-141	>88	>6.51	1.4-2.19	>4.00	>1.21	>5.4	>3.76	>344	>14	
41-50	>39.71	123-141	>88	>6.51	>2.19	3.24-4.00	>1.21	4.7-5.4	<3.06	>344	9-14	
<41	>39.71	<123	78-88	5.37-6.51	<1.4	>4.00	>1.21	4.7-5.4	>3.76	>344	<9	24.3-31.6
	>39.71	<123	<78	<5.37	<1.4	<3.24	<1.01	<4.7	3.06-3.76	<273		
	>39.71	123-141	>88	<5.37	<1.4	<3.24	>1.21	4.7-5.4	3.06-3.76	>344		
	>39.71	<123	<78	<5.37	<1.4	<3.24	<1.01	<4.7	3.06-3.76	<273		
	>39.71	123-141	>88	>6.51	1.4-2.19	3.24-4.00	<1.01	4.7-5.4	3.06-3.76	273-344	9-14	
>50	>39.71	123-141	78-88	5.37-6.51	1.4-2.19	<3.24	1.01-1.21	4.7-5.4	3.06-3.76	>344	>14	
41-50	>39.71	>141	>88	5.37-6.51	>2.19	3.24-4.00	<1.01	>5.4	3.06-3.76	273-344	>14	24.3-31.6 OR 31.7-36.2
>50	28.92-39.71	123-141	>88	>6.51	<1.4	3.24-4.00	>1.21	>5.4	>3.76	273-344	9-14	
<41	>39.71	123-141	78-88	>6.51	>2.19	3.24-4.00	<1.01	>5.4	>3.76	>344	>14	31.7-36.2
>50	>39.71	>141	>88	>6.51	>2.19	>4.00	>1.21	>5.4	>3.76	>344	>14	

Table 5. Obtained rules for obese subjects (BMI>32.52 kg/m^2)

5. Conclusion

Many studies have proved that SAD is a good predictor of abdominal, especially visceral, fat mass, as well as of cardiometabolic risk. Several authors have suggested specific cut-off values for SAD that corresponded with cardiovascular and metabolic risk or with visceral

fat area obtained by CT. Using the concept of a rough set, proved as a formal tool for modeling and processing information systems, we developed a useful model for identification of individuals with multiple cardiovascular risk factors using SAD.

Our results revealed connection between SAD and cardiovascular risk factors which showed dependence on age and nutrition level. We primarily recommend application of SAD in the assessment of the cardiovascular risk in overweight and obese individuals. SAD values ≥24.3 cm in overweight and obese subjects older than 41 years should correspond to increased risk, while values <24.3 cm in overweight subjects younger than 41 years could point to healthy metabolic profile.

6. References

Anjana, M.; Sandeep, S.; Deepa, R.; Vimaleswaran K.S.; Farooq, S. & Mohan, V. (2004). Visceral and central abdominal fat and anthropometry in relation to diabetes in Asian Indians. *Diabetes Care*, Vol.27, No.12, pp. 2948-2953

Ashwell, M. (2005). Waist to height ratio and the Ashwell shape chart could predict the health risks of obesity in adults and children in all ethnic groups. *Nutrition&Food Science*, Vol.35, No.5, pp. 359-64

Berg, A.H. & Scherer, P.E. (2005). Adipose tissue, inflammation, and cardiovascular disease. *Circulation Research*, Vol.96, pp. 939-949

Bosello, O. & Zamboni, M. (2000). Visceral obesity and metabolic syndrome. *Obesity Reviews*, Vol.1, pp. 47-56

Bouchard, C.; Bray, G.A. & Hubbard, V.S. (1990). Basic and clinical aspects of regional fat distribution. *American Journal of Clinical Nutrition*, Vol.52, pp.946-950

Brochu, M.; Starling, R.D.; Tchernof, A.; Matthews, D.E.; Garcia-Rubi, E. & Poehlman, E.T. (2000). Visceral adipose tissue is an independent correlate of glucose disposal in older obese postmenopausal women. *The Journal of Clinical Endocrinology & Metabolism*, Vol.85, pp. 2378-2384

Brtka, V.; Stokić, E. & Srdić, B. (2008). Automated extraction of decision rules for leptin dynamics--a rough sets approach, *Journal of Biomedical Informatics*, Vol.41, No.4, pp. 667-674

Carroll, J.F.; Chiapa, A.L.; Rodriquez, M.; Phelps, D.R.; Cardarelli, K.M.; Vishwanatha, J.K.; Bae, S. & Cardarelli, R. (2008). Visceral fat, waist circumference, and BMI: impact of race/ethnicity. *Obesity*, Vol.16, No.3, pp. 600–607

Coppack, S.W. (2005). Adipose tissue changes in obesity. *Biochemical Society Transactions*, Vol.33, pp. 1049-1052

Couillard, C.; Lamarche, B.; Tchernof, A.; Prud'homme, D.; Tremblay, A.; Bouchard, C.; Moorjani, S.; Nadeau, A.; Lupien, P.J. & Després J-P. (1996). Plasma high-density lipoprotein cholesterol but not apolipoproetin A-I is a good correlate of the visceral obesity-insulin resistance dyslipidemic syndrome. *Metabolism*, Vol.45, pp.882-888

Davidson, J. & Getz, M. (2004). Nutritional risk and body composition in free-living elderly participating in congregate meal-site programs. *Journal of Nutrition for the Elderly*, Vol.24, No.1, pp. 53-68

Desbriere, R.; Vuaroqueaux, V.; Achard, V.; Boullu-Ciocca, S. Labuhn, M.; Dutour, A. & Grino, M. (2006). 11β-Hydroxysteroid Dehydrogenase Type 1 mRNA is Increased

in Both Visceral and Subcutaneous Adipose Tissue of Obese Patients. *Obesity*, Vol.14, pp. 794–798

Després, J-P.; Moorjani, S.; Lupien, P.J.; Tremblay, A.; Nadeau, A. & Bouchard C. (1990). Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. *Arteriosclerosis*, Vol.10, No.4, pp. 497-511

Després, J-P.; Prud'homme, D.; Pouiliot, M.C.; Tremblay, A. & Bouchard C. (1991). Estimation of deep abdominal adipose-tissue accumulation from simple anthropometric measurements in men. *American Journal of Clinical Nutrition*, Vol.54, pp. 471-477

Dorner, T.E. & Rieder, A. (2011). Obesity paradox in elderly patients with cardiovascular diseases. *International Journal of Cardiology*, doi: 10.1016/j.ijcard.2011.01.076

Duarte Pimentel, G.; Portero-McLellan, K.C.; Maestá, N.; Corrente, J.E. & Burini, R.C. (2010). Accuracy of SAD as predictor of abdominal fat among Brazilian adults: a comparation with waist circumference. *Nutrición hospitalaria*, Vol.25, No.4, pp. 656-661

Empana, J.P.; Ducimetiere, P.; Charles, M.A. & Jouven, X. (2004). SAD and risk of sudden death in asymptomatic middle-aged men – the Paris prospective study. *Circulation*, Vol.110, pp. 2781-2785

Freedland, E.S. (2004). Role of a critical visceral adipose tissue threshold (CVATT) in metabolic syndrome: implications for controlling dietary carbohydrates: a review. *Nutrition&Metabolism*, Vol.1, (November 2004), doi: 10.1 186/1743-7075-1-12

Goossens, G.H. (2008). The role of adipose tissue dysfunction in the pathogenesis of obesity-related insulin resistance. *Physiology&Behaviour*, Vol.94, pp. 206-218

Greco, M.; Benedetto, R.; Slowinski, R. (1998). New developments in the rough set approach to multi–attribute decision analysis, *Proceedings of 16th European Conference on Operational Research*, Brussels, July 1998

Greenlund, LJ.S. & Nair K.S. (2003). Sarcopenia – consequences, mechanisms, and potential therapies. *Mechanisms of Aging and Development*, Vol.124, pp. 287-299

Gustat, J., Elkasabany, A., Srinivasan, S. & Berenson, G.S. (2000). Relation of abdominal height to cardiovascular risk factors in young adults. *American Journal of Epidemiology*, Vol.151, No.9, pp. 885-891

Guzzaloni, G.; Minocci, A.; Marzullo, P. & Liuzzi, A. (2009). SAD is more predictive of cardiovascular risk than abdominal fat compartments in severe obesity. *International Journal of Obesity*, Vol.33, pp. 233-238

Harris, T.B.; Visser, M.; Everhart, J.; Cauley, J.; Tylavsky, F.; Fuerst, T.; Zamboni, M.; Taaffe, D.; Resnick, H.E.; Scherzinger, A. & Nevitt, M. (2000). Waist circumference and sagittal diameter reflect total body fat better than visceral fat in older men and women. The Health, Aging and Body Composition Study. *Annals of the New York Academy of Sciences*, Vol.904, pp. 462-473

Hoenig, M.R. (2010). MRI sagittal diameter is a stronger predictor of metabolic syndrome than visceral fat area or waist circumference in a high-risk vascular cohort. *Vascular Health and Risk Management*, Vol.6, pp. 629-633.

Hsieh, S.D. & Yoshinaga, H. (1995). Abdominal fat distribution and coronary heart disease risk factors in men – waist/height ratio as a simple and useful predictor. *International Journal of Obesity and Related Metabolic Disorders*, Vol.19, pp. 585-589

Hwu, C.M.; Hsiao, C.F.; Sheu, W.H.H.; Pei, D.; Tai, T.Y.; Quertermous, T.; Rodriguez, B.; Pratt, R.; Chen, Y.DI, Ho, L.T. (2003). SAD is associated with insulin sensitivity in Chinese hypertensive patients and their siblings. *Journal of Human Hypertension*, Vol.17, pp. 193-198

Iribarren, C.; Darbinian, J.A.; Lo, J.C.; Fireman, B.H. & Go, A.S. (2006). Value of the SAD in coronary heart disease risk assessment: cohort study in a large, multiethnic population. *American Journal of Epidemiology*, Vol.164, No.12, pp. 1150–1159

Iribarren, C.; Sharp, D.S.; Burchfiel, D.M. & Petrovich, H. (1995). Association of weight loss and weight fluctuation with mortality among Japanese men. *New England Journal of Medicine*, Vol.333, pp. 686-692

Kahn, H.S. (1993). Choosing an index for abdominal obesity: an opportunity for epidemilogical clarification. *Journal of Clinical Epidemiology*, Vol.46, pp. 491-494

Krotkiewski, M.; Björntorp, P.; Sjöström, L. & Smith, U. (1983). Impact of obesity on metabolism in men and women. Importance of regional adipose tissue distribution. *Journal of Clinical Investigation*, Vol.72, pp. 1150-1162

Kumlin, L.; Dimberg, L. & Mårin, P. (1997). Ratio of abdominal sagittal diameter to heigh is strong indicator of coronary risk (letter). *British Medical Journal*, Vol.314, p. 830

Kvist, H.; Chowdhury, B.; Grangard, U.; Tylen, U. & Sjostrom L. (1988). Total and visceral-adipose tissue volumes derived from measurements with computed tomography in adults man and women: predictive equations. *American Journal of Clinical Nutrition*, Vol.48, No.6, pp. 1351-1361

Lemieux, S.; Prud'homme, D.; Bouchard, C.; Tremblay, A. & Despres, A. (1996). A single threshold value of waist girth identifies normal-weight and overweight subjects with excess visceral adipose tissue. *American Journal of Clinical Nutrition*, Vol.64, pp.685-693

Lofgren, I.; Herron, K.; Zern, T.; West, K.; Partalay, M.; Shachter, N.S.; Koo, S.I. & Fernandez, M.L. (2004). Waist circumference is a better predictor than body mass index of coronary heart disease risk in overweight premenopausal women. *Journal of Nutrition*, Vol.134, pp. 1071–1076

Lonnqvist, F.; Thorne, A.; Large, V. & Arner, P. (1995). Sex differences in visceral fat lipolysis and metabolic complications of obesity. *Atherosclerosis, Thrombosis, and Vascular Biology*, Vol.17, No.7, pp. 1472-1480

Marques-Vidal, P.; Chiolero, A. & Paccaud, F. (2008). Large differences in the prevalence of normal weight obesity using various cut-offsfor excess body fat. *e-SPAN, the European e-Journal of Clinical Nutrition and Metabolism*, Vol.3, pp. e159-e162

Matsuzawa, Y.; Shimomura, I.; Nakamura, T.; Keno, Y.; Kotani, K. & Tokunaga, K. (1995). Pathophysiology and pathogenesis of visceral fat obesity. *Obesity Research*, Vol.3, No.2, pp.187-194S.

Mazzali, G.; Di Francesco, V.; Zoico, E.; Fantin, F.; Zamboni, G.; Benati, C.; Bambara, V.; Negri, M.; Bosello, O. & Zamboni M. (2006). Interrelations between fat distribution, muscle lipid content, adipocytokines, and insulin resistance: effect of moderate weight loss in older women. *American Journal of Clinical Nutrition*, Vol.84, No.5, pp. 1193-1199

Misra, A. & Vikram, N.K. (2003). Clinical and patophysiological consequences of abdominal adiposity and abdominal adipose tissue depots. *Nutrition*, Vol.19, pp. 457-66

Molarius, A. & Seidell, J.C. (1998). Selection of anthropometric indicators for classification of abdominal fatness: A critical review. *International Journal of Obesity and Related Metabolic Disorders*, Vol.22, No.8, pp. 719-727

Movsesyan, L.; Tanko, L.B.; Larsen, P.J.; Christiansen, C. & Svendsen, O.L. (2003). Variations in percentage of body fat within different BMI groups in young, middle-aged and old women. *Clinical Physiology and Funcional Imaging*, Vol.23, pp.130-133

Mukuddem-Petersen, J.; Snijder, M.B.; van Dam, R.B.; Dekker, J.M.; Bouter, L.M.; Stehouwer, C.D.A.; Heine, R.J.; Nijpels, G. & Seidell, J.C. (2004). SAD: no advantage compared with other anthropometric measures as a correlate of components of the metabolic syndrome in elderly from the Hoorn Study. *American Journal of Clinical Nutrition*, Vol.84, pp.995-1002

Nakata, K.; Choo, J.; Hopson, M.S.J.; Ueshima, H.; Curb, D.; Shin, C.; Evans, R.W.; Kadowaki, T.; Otake, T.; Kadota, A.; Kadowaki, S.; Miura, K.; El-Saed, A.; Edmudowicz, D.; Sutton-Tyrell, K.; Kuller, L.H. & Sekikawa, A. (2010). Stronger associations of SAD with atherogenic lipoprotein subfractions than waist circumference in middle-aged US white and Japanese men. *Metabolism Clinical and Experimental*, Vol.59, pp.1742-1751

Øhrn, A. (1999). Discernibility and Rough Sets in Medicine: Tools and Applications. PhD thesis. Department of Computer and Information Science (Norwegian University of Science and Technology, Trondheim, Norway)

Öhrvall, M.; Berglund, L. & Vessby, B. (2000). SAD compared with other anthropometric measurements in relation to cardiovasular risk. *International Journal of Obesity and Related Metabolic Disorders*, Vol.24, No.4, pp.497-501.

Paolini, J.B.; Mancini, J.; Genestal, M.; Gonzalez, H.; McKay, R.E.; Samii, K. & Fourcade, O.A. (2010). Predictive value of abdominal obesity vs. body mass index for determining risk of intensive care unit mortality. *Critical Care Medicine*, Vol.38, No. 5, pp. 1308-1314

Pawlak, Z. & Skowron, A. (2007). Rudiments of rough sets. *An International Journal of Information Sciences*, Vol.177, pp. 3–27

Pawlak, Z.; Grzymala-Busse, J.; Slowinski, R. & Ziarko, W. (1995). Rough sets. *Communications of the ACM*, Vol.38, No.11, pp. 89–95R.

Petersson, H.; Daryani, A. & Risérus, U. (2007). SAD as a marker of inflammation and insulin resistance among immigrant women from the Middle East and native Swedish women: a cross-sectional study. *Cardiovascular Diabetology*, Vol.6, pp. 10.

Pouliot, M.C.; Despres, J.P.; Lemieux, S.; Moorjani, *S.*; Bouchard, C. & Tremblay, A. (1994). Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *American Journal of Cardiology,* Vol.73, No.7, pp. 460-468

Prentice, A.M. & Jebb, S.A. (2001). Beyond body mass index. Obesity Reviews, *Vol.2, No.3, pp. 141-147*

Reed, D.; Dwyer, K.M. & Dwyer, J.H.; (2003). Abdominal obesity and carotid artery wall thickness. The Los Angeles Atherosclerosis Study. *International Journal of Obesity and Related Metabolic Disorders*, Vol.27, pp. 1546-1551

Richelsen, B. & Pedersen, S.B. (1995). Associations between different anthropometric measurements of fatness and metabolic risk parameters in non-obese, healthy, middle-aged men. *International Journal of Obesity*, Vol.19, No.3, pp. 169-174

Risérus, U.; Ärnlöv, J.; Brismar, K.; Zethelius, B.; Berglund, L. & Vessby, B. (2004). SAD is a strong anthropometric marker of insulin resistance and hyperinsulinemia in obese men. *Diabetes Care*, Vol.27, No.8, pp. 2041-2046

Risérus, U.; de Faire, U.; Berglund, L. & Hellénius, M.L. (2010). SAD as a screening tool in clinical research: cutoffs for cardiometabolic risk. *Journal of Obesity*, doi:10.1155/2010/757939

Romero-Corral, A.; Montori, V.M.; Somers, V.K.; Korinek, J.; Thomas, R.J.; Allison, T.G.; Mookadam, F. & Lopez-Jimenez, F. (2006). Association of bodyweight with total mortality and with cardiovascular events in coronary artery disease: a systematic review of cohort studies. *Lancet*, Vol.368, No.9536, pp.666-678

Ruderman, N.; Chisholm, D., Pi-Sunyer, X. & Schenider, S. (1998). The metabolically obese, normal-weight individual revisited. *Diabetes*, Vol.47, pp.699-713

Sampaio, L.R.; Simões, E.J.; Assis, A.M.O. & Ramos, L.R. (2007). Validity and reliability of the SAD as a predictor of visceral abdominal fat. *Arquivos Brasileiros de Endocrinologia & Metabologia*, Vol.51, No.6, pp. 980-986

Sims, E.A. (2001). Are there persons who are obese, but metabolically healthy? *Metabolism*, Vol.50, pp. 1499-1504

Sjöstrom, L. (1994). The sagittal diameter is valid marker of visceral adipose tissue. *International Journal of Obesity*, Vol.18, No.2, pp. 46-52

Smith, D.A.; Ness, E.M.; Herbert, R.; Schechter, C.B.; Phillips, R.A.; Diamond, J.A. & Landrigan, P.J. (2005). Abdominal diameter index: a more powerful anthropometric measure for prevalent coronary heart disease risk in adult males. *Diabetes, Obesity and Metabolism*, Vol.7, pp.370-380.

Snijder, M.B.; Visser, M.; Dekker, J.M.; Seidell, J.C.; Fuerst, T.; Tylavsky, F.; Cauley, J.; Lang, T.; Nevitt, M. & Harris, T.B. (2002). The prediction of visceral fat by dual-energy X-ray absorptiometry in the elderly: a comparison with computed tomography and anthropometry. *International Journal of Obesity*, Vol.26, pp. 984-993

Stokić, E. & Ivković-Lazar, T. (1996). Relation between the abdominal sagittal diameter, fat tissue distribution and metabolic complications. *Medicinski Pregled*, Vol.49, No. 9-10, pp. 365-368

Stokić, E.; Brtka, V. & Srdić, B. (2010). The synthesis of the rough set model for the better applicability of SAD in identifying high risk patients. *Computers in Biology and Medicine*, Vol. 40, pp. 786-90

Stokić, E.; Đureković-Katona, A. & Ivković-Lazar, T. (1996). The role and significance of SAD in the determination of adipose tissue distribution. *Medicinski Pregled*, Vol.49, No.5-6, pp. 217-220

Strazzullo, P.; Barba, G.; Cappuccio, F.P.; Siani, A.; Trevisan, M.; Farinaro, E.; Pagano, E.; Barbato, A.; Iacone, R. & Galletti, F. (2001). Altered renal sodium handling in men with abdominal adiposity: a link to hypertension. *Journal of Hypertension*, Vol.19, No.12, pp. 2157-2164.

Toth, M.J.; Tchernof, A.; Sites, C.K. & Poehlman, E.T. (2000). Effect of menopausal status on body composition and abdominal fat distribution. *International Journal of Obesity*, Vol.24, pp. 226-231

Turcato, E.; Bosello, O.; Di Francesco, V.; Harris, T.B.; Zoico, E.; Bissolo, L; Fracassi, E. & Zamboni, M. (2000). Waist circumference and abdominal sagittal diameter as surogates of body fat distribution in the elderly: their relation with cardiovascular risk factors. *International Journal of Obesity*, Vol.24, pp. 1005-1010

Valdez, R. (1991). A simple model-based index of abdominal adiposity. *Journal of Clinical Epidemiology*, Vol.44, pp. 955-956

Valsamakis, G.; Chetty, R.; Anwart, A.; Banerjee, A.K.; Barnett, A. & Kumar, S. (2004). Association of simple anthropometric measures of obesity with visceral fat and the metabolic syndrome in male Caucasion and Indo-Asian subjects. *Diabetic Medicine*, Vol.21, pp. 1339-1345

Valsamakis, G.; Jones, A.; Chetty, R.; McTernan,P.G.; Boutsiadis, A.; Barnett, A.H.; Banerjee, A.K. & Kumar, S. (2008). MRI total SAD as a predictor of metabolic syndrome compared to visceral fat at L4-L5 level. *Current Medical Research&Opinion*, Vol.28, pp. 1853-1860

van der Kooy, K.; Leenen, R.; Seidell, J.C.; Deurenberg, P. & Visser M. (1993). Abdominal diameters as indicators of visceral fat: comparison between magnetic resonance imaging and anthropometry. *British Journal of Nutrition*, Vol.70, No.1, pp. 47-58

van Harmelen, V.; Röhrig, K. & Hauner, H. (2004). Comparison of proliferation and differentation capacity of human adipocyte precursor cells from the omental and subcutaneous adipose tissue depot of obese subjects. *Metabolism,* Vol.53, No.5, pp.632-7

van Herpen, N.A, & Schrauwen-Hinderling, V.B. (2008). Lipid accumulation in non-adipose tissue and lipotoxicity. *Physiology&Behaviour*, Vol.94, pp. 231-41

Vasques, A.C.; Rosado, L.E.; Rosado, G.P.; Ribeiro R. de C.; Franceschini S. do C.; Geloneze, B.; Priore, S.E. & Oliveira, D.R. (2009a). Predictive ability of anthropometric and body composition indicators in the identification of insulin resistance. *Arquivos Brasileiros de Endocrinologia & Metabologia*, Vol.53, No.1, pp.72-79

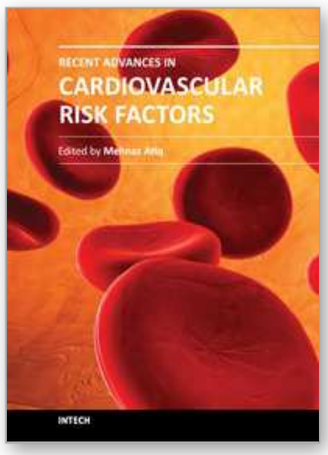
Vasques, A.C.; Rosado, L.E.; Rosado, G.P.; Ribeiro R. de C.; Franceschini, S. do C.; Geloneze, B.; Priore, S.E. & Oliveira, D.R. (2009b). Different measurements of the SAD and waist perimeter in the prediction of HOMA-IR. *Arquivos Brasileiros de Endocrinologia & Metabologia*, Vol.93, No.5, pp. 511-518

Visscher, T.L.; Seidell, J.C.; Molarius, A.; van der Kuip, D.; Hofman, A. & Witteman, J.C. (2001). A comparison of body mass index, waist-hip ratio and waist circumference as predictors of all-cause mortality among the elderly: the Rotterdam study. *International Journal of Obesity and Related Metabolic Disorders*, Vol.25, No.11, pp. 1730–1735

Wajchenberg, B.L. (2000). Subcutaneous and visceral adipose tissue: their relation to the metabolic syndrome. *Endocrine Reviews*, Vol.21, No.6, pp. 697-738

Yim, J.Y.; Kim, D.; Lim, S.H.; Park, M.J.; Choi, S.H.; Lee, C.H.; Kim, S.S. & Cho, S.H. (2010). SAD is a strong anthropometric measure of visceral adipose tissue in the Asian general population. *Diabetes Care*, Vol.33, No.12, pp. 2665-2670

Zamboni, M.; Turcato, E.; Armellini, F.; Kahn, H.S.; Zivelonghi, A.; Santana, H.; Bergamo-Andreis, I.A. & Bosello, O. (1998). SAD as a practical predictor of visceral fat. *Interantional Journal of Obesity*, Vol.22, pp. 655-660



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Among the non-communicable diseases, cardiovascular disorders are the leading cause of morbidity and mortality in both the developed and the developing countries. The spectrum of risk factors is wide and their understanding is imperative to prevent the first and recurrent episodes of myocardial infarction, stroke or peripheral vascular disease which may prove fatal or disabling. This book has tried to present an update on risk factors incorporating new research which has thrown more light on the existing knowledge. It has also tried to highlight regional diversity addressing such issues. It will hopefully be resourceful to the cardiologists, general practitioners, family physicians, researchers, graduate students committed to cardiovascular risk prevention.

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