Response from Dr Paul Poirier to P De Groot's: Cardiologists and abdominal obesity: lost in translation?

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Waist circumference: a waste of time? Response to Dr Paul Poirier’s: Cardiologists and abdominal obesity: lost in translation?

To the editor: With great interest, we read the articles of a recent issue of Heart addressing the important role of abdominal obesity in relation to cardiovascular risk and the metabolic syndrome.1 2 The metabolic syndrome, considered the ailment of the 20th century, emerges from clustering and interactions of multiple cardiovascular risk factors affecting a large proportion of the population. The presence of the metabolic syndrome in children is of particular interest given the rapid and alarming increase in prevalence in the past decade. Classification of body weight in children is challenging because body proportions vary considerably during growth. A uniform definition of the metabolic syndrome in the paediatric population would be relevant for early diagnosis and treatment and for scientific and public health purposes. Current metabolic syndrome criteria for children as recommended by the International Diabetes Federation3 define childhood obesity as the 90th percentile of waist circumference, which has important and unacceptable drawbacks.

First, prevalence of obesity among children has increased threefold in the past 30 years.4 Using the 90th percentile of waist circumference to define obesity raises the problem of a moving cutoff point to define obesity, which will potentially mask the impact of the problem. For example, a child who was defined obese 5 years ago may not reach the cutoff value for obesity today but instead falls within the normal range, which could lead to an incorrect conclusion that obesity prevalence is declining. In addition, the prevalence of being overweight and obese differs markedly among children from different countries and populations.

A previously published systemic review in school-aged youth from 34 (mostly European) countries illustrates a large variation in overweight and obesity prevalence rates, that is, the two countries with the highest prevalence of overweight and obesity were Malta and the USA (25.4% and 7.9%; 25.1% and 6.8%, respectively), whereas the lowest prevalence rates were found in Lithuania (5.1% and 0.4%) and Latvia (5.9% and 0.5%).3

These numbers clearly demonstrate that using the 90th percentile of waist circumference to define childhood obesity makes international comparison between populations and studies extremely difficult.

As body composition and proportions change during childhood, we are aware of the highly challenging task to obtain a universal measure to define childhood obesity. However, we strongly believe that efforts should be made to obtain sex- and age-dependent absolute cutoff points to define the degree of obesity in the paediatric population in the future.

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Response from Dr Paul Poirier to P De Groot’s: Cardiologists and abdominal obesity: lost in translation?

The author’s reply: In response to the comments on my recent editorial, I would like to emphasise that I agree with the clarification of de Groot et al.1 Indeed, in youth between 2 and 18 years of age, obesity is defined as a body mass index (BMI) of 95th percentile or BMI of ≥30 kg/m², whichever is lower. For children <2 years of age, BMI normative values are not available.2 Data indicate that extreme obesity in children is increasing in prevalence, and these children are at high risk for multiple cardiovascular disease (CVD) risk factors. A proposed definition of severe childhood obesity is 99th percentile BMI, which corresponds to a BMI of approximately 30–52 kg/m² for youths 10—12 years of age and 45 kg/m² for youths 14—16 years of age.2 Thus, the improvement in risk factor recognition and management that developed through the years in modern cardiology may be counteracted by the rising incidence of obesity. Indeed, it was suggested that the life-shortening effect of obesity could increase as the obese who are now at younger ages carry their elevated risk of death into middle and older ages.3 There is no debate relative to the issue of assessing excess adiposity in children.

As pointed out by de Groot et al, waist circumference (WC) measurements are not recommended routinely in children because reference values for children that identify risk over and above the risk from BMI category are not available.2 Nevertheless, clinicians may add WC to the tools they use to assess risk. If they do, clinicians should use as high, age-specific percentile cutoff point, such as 90th or 95th percentile, to evaluate risk.2 There are numerous indices to evaluate obesity (BMI, WC, waist-to-hip ratio) or body fat content (bioelectrical impedance, hydrostatic weighing, dual energy x-ray absorptiometry, air displacement plethysmography). Accurate diagnosis of obesity may entail more refined assessment of body fat composition/distribution. Through the years, researches have helped refining indices associated with CVD. For example, total cholesterol has been replaced by low- and high-density lipoprotein cholesterol to better evaluate the patient’s risk of CVD. With obesity occurring at younger ages, the children and young adults of today will carry and express obesity-related risks for more of their lifetime than previous generations. Without a doubt, obesity is a risk factor for CVD. Today, we are no longer using total weight to appreciate the presence of obesity.2 Today’s paediatric practice has changed tremendously. Paediatric endocrinologists are no longer treating only type 1 diabetes but are faced with a new challenge; type 2 diabetes-associated obesity at younger age. It will probably be the same with paediatric cardiologists who will have to adapt their practice, which used to be mostly structural cardiac abnormalities and arrhythmias to new comers such as cardiomyopathy due to HIV or management of risk factor-induced obesity. Although BMI has been useful in epidemiological study to assess the presence of obesity, it fails to differentiate between differences in body compositions. BMI does not characterise excess centrally distributed obesity, which is more consistently associated with adverse effects on metabolism, dyslipidaemia and insulin resistance. One index that may be of use in children is waist-to-height ratio.6 However, further researches are needed to clarify the role of this index in clinical practice. While waiting for such studies to be conducted, I do not feel that measuring WC is a waste of time.

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Measuring waist circumference: do not “throw the baby out with the bath water!”

The authors’ reply: Although the objective of my review paper was not to address the relevance of measuring waist circumference in children, I certainly agree with de Groot et al that the proposal to define childhood obesity as the 90th percentile of waist circumference has important limitations. However, in adults, an elevated waist circumference predicts a further increased risk of morbidity and mortality at any body mass index value. Furthermore, it is likely to be a better therapeutic target than weight loss as some individuals may lose abdominal fat without necessarily losing weight when they are involved in a healthy eating/physical activity/exercise lifestyle modification programme. Thus, although I fully heartily endorse their position that further work is needed in children, the catchy title of their letter (“Waist Circumference: A Waste of Time”) could be misleading.

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T2-weighted magnetic resonance imaging to assess myocardial oedema

To the editor: We read with interest the comprehensive review article on T2-weighted magnetic cardiovascular magnetic resonance imaging (CMRI) by Edwards et al. We wish to make one comment for the sake of clarity and to bring to the reader’s attention important new data, which have become available in the meantime, and shed light on some of the issues raised by the authors. The authors cite one of the first reports employing T2-weighted CMRI to assess acute myocardial injury at a very early stage but fail to bring out one key aspect of that study. We used polyvinyl alcohol alcohol foam particles (contour emboli) rather than ethanol injections to induce therapeutic infarction in patients undergoing septal artery embolisation. As a result, these ischaemic insults were fairly comparable with the clinical event of atherothrombotic coronary occlusions. Although an elevated T2 signal was not consistently observable within the first 60 minutes after the onset of ischaemia, it was present on the next day and remained elevated for up to 4 weeks. Whereas this study demonstrated the ability of T2-weighted CMRI to identify acute myocardial injury, the ‘early blind spot’ was possibly related to the experimental design with embolisation of a very small coronary territory. Furthermore, the impact of such particles on the T2 signal was not investigated, but may have been a confounder. Recently, Abdel-Aty et al added a few more pieces to the jigsaw: one of the dogs was instrumented with a snare around the first diagonal artery. Ischaemia duration was deliberately restricted so as to avoid relevant cell destruction (ie, infarction). While contractility was immediately affected (reflecting energetic depletion), the pathological T2 signal (reflecting tissue oedema) did not visually become apparent before 28±4 minutes after the onset of ischaemia and was further increased after reperfusion. The marked increase in T2 signal was parallelled by a relatively small, yet relevant and significant, increase in myocardial water content (~2%), probably reflecting cellular rather than interstitial oedema, with disruption of cell membranes. This experiment elucidates the T2-weighted CMRI profile of non-lethal ischaemia in a dog model with some, albeit inherently limited, transferability into the human scenario. Moreover, the relationships of the T2 signal with conditions such as stunning, repetitive ischaemia–reperfusion and peri-ischaemic inflammation are still awaiting to be unravelled.

Incremental technical advances in T2-weighted CMRI will facilitate that and are expected to enhance our understanding of the ischaemia–reperfusion sequence and improve clinical decision-making.

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The authors’ reply: We thank Bohl et al for their additional comments relating to the timing of T2-weighted imaging to detect acute ischaemia. They reference their own experimental data that has been instrumental in the development of T2-weighted cardiovascular magnetic resonance imaging (CMRI) and emphasise the methodological differences that might explain the failure to detect myocardial oedema within the first 24 h. This paper was important not only in raising the issue of the earliest time point for the detection of myocardial oedema but also for the long-term 180-day follow-up period assessing the duration of the changes detectable on T2-weighted CMRI. Recent data from animal models and clinical human studies have provided more information regarding the ‘time window’ in which the earliest appearances of myocardial oedema occur. The recent paper by Abdel-Aty et al, which became available during revision of our