Resistance exercise and control of cerebral blood flow in type 2 diabetes
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Published in:
Diabetologia

DOI:
10.1007/s00125-008-1068-y

Citation for published version (APA):
Resistance exercise and control of cerebral blood flow in type 2 diabetes

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Keywords Blood pressure · Cerebral autoregulation · Cerebral circulation · Exercise · Transcranial Doppler

To the Editor: The challenge in diabetes care is to optimise metabolic control to slow progression to vascular disease. In individuals with type 2 diabetes this may be achieved at least in part by behavioural modification including regular physical activity. In their recent article in Diabetologia, Praet and van Loon [1] comprehensibly and concisely address the need for revision of the current guidelines regarding the prescription of exercise for individuals with type 2 diabetes, given the lack of information provided by current exercise programmes on the preferred type and intensity of exercise.

The combination of endurance and resistance exercise has the potential to increase adherence and compliance rates, as it renders programmes more varied [1]. Loss of skeletal muscle mass is one of the main factors responsible for the increase in the incidence of type 2 diabetes with age. Deconditioning (as a result of physical inactivity) and resistance exercise are associated with opposing adaptations. Resistance exercise provides better metabolic control [2], mitigates disuse-associated tendon stiffness and increases skeletal muscle mass, which improves whole body glucose disposal [3]. Not surprisingly, greater focus on resistance exercise has been recommended for type 2 diabetes, specifically for the subgroup of sarcopenic or severely deconditioned older patients [4].

Resistance and endurance exercise have different cardiovascular effects. Resistance-type activities produce a considerably larger increase in arterial pressure, because of the mechanical compression of blood vessels together with repeated Valsalva-like manoeuvres [5]. Unlike aerobic exercise, resistance training affects central arterial compliance in healthy men [6]. Although acute changes in arterial blood pressure during physiological challenges are transmitted to the cerebral circulation, under normal conditions, cerebral blood flow tends to return to its baseline value within a few seconds [7]. Cerebral vasoconstriction constantly plays a protective role during exercise of moderate to heavy intensity, in particular when pulse pressure exceeds the autoregulatory range [8]. When autoregulatory mechanisms are failing or overwhelmed by acute blood pressure upsurges beyond the autoregulatory range (e.g. in serious hypertension), brain blood flow...
becomes directly related to its perfusion pressure, resulting in cerebral hyperperfusion manifested by retinal oedema and encephalopathy [9].

We consider the arterial pressure surges associated with resistance exercise to be substantial, heavily taxing cerebral autoregulatory mechanisms [10]. The integrity of the cerebral autoregulatory mechanisms protecting the brain is therefore important under these conditions. To date, there is no direct evidence indicating that resistance exercise is disadvantageous for individuals with type 2 diabetes. However, in these individuals, dynamic cerebral autoregulatory capacity is impaired in the early stages of disease development, i.e. prior to the clinical recognition of microvascular complications [11]. This means that arterial pressure surges are buffered less efficiently, with more passive transmission of blood pressure to the cerebral tissue occurring (Y. S. Kim, J. J. van Lieshout, unpublished results, Fig. 1), resulting in increased exposure to cerebral hyperperfusion. Given that repetitive arterial pressure surges may contribute to long-term cerebral vasculopathy, detailed studies are needed to establish the cerebrovascular effects of different types of exercise in type 2 diabetic patients.

We agree with Praet and van Loon that an individually tailored exercise programme providing a more specific framework is important to optimise exercise therapy. However, when devising such programmes, any potential unfavourable effects of blood pressure surges on the brain should be taken into account.

Duality of interest The authors declare that there is no duality of interest associated with this manuscript.

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