Please read the paragraphs adapted from Model-based estimation of muscle forces exerted during movements. (by Erdemir A, McLean S, Herzog W, van den Bogert AJ. *Clin Biomech (Bristol, Avon).* 2007 Feb;22(2):131-54.) carefully and answer the following questions **in Chinese**. **(30%)**

Paragraph 1. The force output of cardiac muscle can be quantified by simply recording arterial pressure. It is far more difficult to obtain clinically relevant information on the function of skeletal muscles. Imagine what could be done with such information. In the treatment of cerebral palsy, the clinician could "see" which muscle is responsible for an abnormal gait pattern, and that muscle could then be directly targeted for surgery. In an athlete with a recurrent overuse injury, we could "see" the loads being placed upon bones and joints during movement and how these loads are altered during rehabilitation. There are many other neurological and orthopaedic problems where knowledge of muscle forces could enhance clinical decision making.

Paragraph 2. Direct measurement of muscle forces in vivo is usually limited to minimally invasive measurements in superficial tendons such as the Achilles (Finni et al., 1998; Komi et al., 1992). Otherwise, in vivo measurements can be conducted in the operation room where a force transducer can be placed on a tendon, following data collection and the removal of the device before the completion of the surgery, e.g. flexor tendons of fingers during surgeries of carpal tunnel (Dennerlein et al., 1998; Dennerlein et al., 1999; Dennerlein, 2005; Schuind et al., 1992). Such approaches may not necessarily be feasible in a clinical setting; therefore such tendon force measurement techniques have been utilized mostly in research laboratories (Ravary et al., 2004; Fleming and Beynnon, 2004). Non-invasive methods rely on the basic principle that muscles produce skeletal movement and ground reaction forces. Clearly, none of these observable variables provides information on any single muscle. Instead, a technique known as inverse dynamic analysis has been developed, based on computational modeling of the dynamics of linked body segments. The analysis produces estimates of joint torques, each of which represents the resultant action of all muscles crossing a joint. While inverse dynamic analysis has become a routine tool in clinical gait analysis (Vaughan et al., 1992; Winter, 2005), muscles are not represented and the approach provides no information on muscular load sharing, agonist–antagonist activity, energy transfer between joints via biarticular muscles, and dynamic coupling (van den Bogert, 1994; Zajac et al., 2002). Electromyograpy (EMG) data can support a clinical inverse dynamic analysis to more effectively interpret joint torques, but there are no estimates of individual muscle forces (Zajac et al., 2003).

Question 1. What is the argument that the authors are trying to build up here in paragraph 1? **(15%)**
Question 2. What do you think is the limitation of "inverse dynamic analysis" stated in paragraph 2, line 10, after reading the second paragraph? **(15%)**

Please read the paragraphs adapted from Gait disturbances in old age: classification, diagnosis, and treatment from a neurological perspective. (by Jahn K, Zwergal A, Schniepp R. *Dtsch Arztebl Int.* 2010 Apr;107(17):306-15.) carefully and answer the following questions **in Chinese**. **(40%)**

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Paragraph 1. Gait disturbances are a common medical problem in old age. Among the patients of a hospital department of acute neurology, old age is the most important risk factor for a gait disturbance (1). A variety of diseases can cause gait disturbances; some, like Parkinson's disease, have well-established treatments according to the principles of evidence-based medicine, while for others, like cerebrovascular gait disturbance, too little evidence is available to support any particular form of treatment. A population-based study has shown a 35% prevalence of gait disorders among persons over age 70 (2). 85% of 60-year-olds still walk normally, but only 20% of 85-year-olds do (3). The latter fact also implies, however, that gait disturbances are not an inevitable accompaniment of old age. Problems of balance and gait are associated with immobility and falls, which markedly impair the quality of life (4). About 30% of persons over age 65 living at home fall at least once per year; among nursing home residents, the corresponding figure is about 50% (5). Mobility is often restricted still further by the fear of falling (6). Patients visiting their family physician because of gait disturbances complain most often of pain, joint stiffness, numbness, weakness, and an abnormal pattern of gait (7).

Paragraph 2. Walking is one of the more frequently performed sensorimotor tasks in everyday life. It relies on a complex, simultaneous interaction of the motor system, sensory control, and cognitive functions. The diagnostic assessment of gait disturbances in old age requires a clear distinction of pathological findings from the normal, physiological changes of aging. Spontaneous walking speed normally decreases by about 1% per year from age 60 onward (8), and the observed decline of maximum walking speed is even greater. On the other hand, a gait disturbance in old age is said to be present when the patient walks even more slowly than expected for age, or when there are qualitative abnormalities of locomotion, such as disturbances of the initiation of gait or of balance while walking (9). The patient's gait should be observed in standardized fashion, and the findings should be compared to age specific norms. Gait disturbances in old age should be clinically classified in purely descriptive terms at first; deficits should be recorded as deviations of the main quantitative parameters of gait—speed, step size, and breadth of stance—from the corresponding age-specific norms. Blanket terms such as "senile gait disturbance" should be avoided, because specific treatment cannot be provided if the relevant deficits underlying the gait disturbance have not been identified.

Paragraph 3. The rhythmic movement pattern of human gait is established at the level of the spinal cord, where so-called central pattern generators, i.e., coordinated groups of interneurons, control the alternating activation of agonist and antagonist muscles during the gait cycle (e1, e2). The existence of autonomous spinal rhythm generators was postulated about 100 years ago, when Thomas Graham Brown demonstrated the persistence of locomotor movements in cats after decerebration, i.e., complete transection of the brainstem. The existence of rhythm generators in man as well is supported by the presence of spinal locomotor movements in paraplegic patients, and by the presence of coordinated movements in all four limbs (arms as well as legs) during bipedal gait (e3, e4). It remains unknown, however, how autonomously the spinal generators control normal human gait; supraspinal control is presumably more important for human, bipedal gait than it is for the quadrupedal gait of the cat. The centers in the spinal cord interact with sensory systems (in particular, the somatosensory afferent pathways) and are under the control of locomotor regions in the brain that enable the initiation of gait, standing still, changes of speed and direction, and reactions to interference with gait. Gait is mainly controlled by the premotor and motor areas of the frontal

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cortex; these areas project fibers to the basal ganglia and onward to the locomotor centers of the brainstem and cerebellum, which, in turn, control the spinal generators. Functional imaging studies have revealed the regions in the human brain that are important for the control of gait (10, 11).

Question 3. Please translate paragraph 1 into Chinese. **(20%)**

Question 4. What are the 2 key concerns that authors stated in paragraph 2 about observing and comparing a patient's gait and why? **(10%)**

Question 5. Briefly explain what is "central pattern generators" in paragraph 3, line 2 **(5%)**, and how human gait is controlled in paragraph 3, line 13. **(5%)**

Please read the paragraphs adapted from Resistance training for obese, type 2 diabetic adults: a review of the evidence. (by Hills AP, Shultz SP, Soares MJ, Byrne NM, Hunter GR, King NA, Misra A. *Obes Rev.* 2010 Oct;11(10):740-9.) carefully and answer the following questions **in Chinese. (30%)**

Paragraph 1. Traditionally, aerobic training (AT) has been promoted as the most effective mode of exercise for treating type 2 diabetes mellitus (T2DM) with improvements in lipid profiles and insulin sensitivity (19). AT, especially high-intensity AT, is metabolically demanding on skeletal muscle, which is a primary target for insulin transport. Therefore, it is suggested that AT may be a useful tool in the prevention and treatment of T2DM (20). Mechanisms for the AT-induced improvement in insulin sensitivity and lipid profile are unknown, although a number of potential metabolic factors have been identified for affecting insulin signaling and insulin-responsive glucose transporter 4 up-regulation and transport. Some of these include decreased cytokines, decreased energy surplus in the skeletal muscle, up-regulation of uncoupling protein 3, more efficient fatty acid metabolism and increased skeletal muscle mitochondrial function (21). More recently, published guidelines have recognized the importance of strength or resistance training (RT) (22,23) and acknowledge the potential role for progressive RT to increase muscle mass, and in turn, 24-h energy expenditure (15). RT normally involves lifting weights (machines or free weights) typically at loads greater than 65% of the one repetition maximum (1 RM). The 2007 (22) and 2009 (24) statements by the American Heart Association and ACSM underline the role of RT to enhance muscular strength and endurance, functional capacity and independence, and quality of life, while the recent ACSM statement highlights the importance of progression in RT with particular detail devoted to individuals with different levels of training (15). RT has also received considerable attention in recent research literature because of its ability to improve glycaemic control and enhance muscular strength and endurance in T2DM patients (25–27).

Paragraph 2. Obesity is characterized by an excess accumulation of total body fat that influences metabolic processes and predisposes an individual to chronic non-communicable diseases. While total body fat is important, the distribution of excess fat in the abdominal or gluteal regions modifies the health risk profile. Abdominal obesity is particularly relevant and can be further subdivided into subcutaneous and intra-abdominal (visceral) fat regions. Visceral adiposity is 'traditionally' implicated in the aetiology of a number of metabolic complications and is expected to decrease the sensitivity of target tissues to insulin (28). There is some evidence that subcutaneous fat may also contribute to insulin resistance and the metabolic syndrome (28). Concomitant decreases in insulin resistance and abdominal fat, specifically in the subcutaneous compartment, would support the relationship between this anatomical compartment and insulin resistance (29,30).

Paragraph 3. The few studies that have specifically addressed the influence of RT on abdominal fat consistently show a decrease in visceral fat (31–34). Recent unpublished research has found that following a 15% (12 kg) diet-induced weight loss, individuals who participated in RT lost 37% of their visceral fat and increased insulin sensitivity 49%. The results of the RT group were 29% higher than women who lost 15%of their weight but did not exercise (GR Hunter et al., unpublished data). In addition, during the year following the weight loss those who participated in RT did not regain any of their visceral fat while participants who did not train regained over 70% of the visceral fat lost (35). Moreover, abdominally obese participants who completed a diet plus RT weight loss programme showed a significantly lower total body fat regain and a lower android to gynoid fat ratio after 12 weeks of free-living compared with similar participants who underwent diet alone weight loss (36). Hence RT has the potential to reduce metabolically active fat deposits through both immediate (during weight loss or weight maintenance) and delayed effects (on weight regain). While the visceral compartment has received the most attention, there is a need to examine the effect of RT on the contribution of the subcutaneous deposit to whole body insulin sensitivity.

Question 6. Please summarize how does aerobic training (AT) and resistance training (RT) benefit type 2 diabetes mellitus (T2DM) patients from paragraph 1. **(15%)**

Question 7. Please explain what is the relation between obesity and body fat **(5%)**, and summarize the resistance training (RT) effects on body fat from reading paragraph 2 and 3. **(10%)**

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