The following 5 questions have to be answered in English (number of potential points is indicated between brackets, 10 points in total for each question: thus 50/5=10)

1. You have to (must, there is no alternative) construct the torque-velocity relationship of the contractile elements of the knee extensor muscles using a dynamometer. (The reason for doing this is to compare your results with the force-velocity relationships obtained previously during experiments on isolated single fibres without tendon)
a. Design the experiment in the dynamometer. Describe as exactly as possible the measurements you want to do and carefully explain the choices you make.(5)

Voluntary (electrically induced are too painful) short activated contractions (to prevent shortening induced force deficit and sec shortening as much as possible) at different imposed velocities, using a broad knee angle range (allowing the motor to accelerate and decelerate, each time measuring torque at 60º knee angle(torque knee angle relationship)

b. List and explain the limitations of your experiment and the problems (both practical and with respect to data interpretation) you anticipate when comparing your results with those of the single fibre results . (5)

-uncertainties about muscle activation especially at the fast velocities
-no reliable measurements possible at high speeds
-you measure torques, which has to be converted into muscle force (internal moment arms)
-the imposed velocities are angular velocities (which have to be converted into contraction speed)
-torque output is net torque output (several synergistic and antagonist muscles)
-fibre pennation angles: no direct alignment with force transducer (as with single fibres)
-muscle temperature probably different
-difficult to assess the correct duration of isometric torque development prior to the shortening

2. Subjects A and B perform an isometric knee extension (60 º knee angle) at 30 % MVC until the point of torque failure. The result of this experiment is: contraction duration of subject A is 80 sec, while that of subject B is 120 sec.
a. List and explain the possible reasons for the better performance (longer duration) of subject B. (6)

-MVC, therefore subjects may have performed at different percentage MTC
-blood flow may be hampered to a different extent in both subjects (e.g. differences in anatomy (fibre angle /curvature) or bloodpressure response
-B has more type I fibre that have higher economy
-differences in the ability to overcome inhibiting effects of pain and metabolite sensitive group III IV afferents (higher central drive)
-less antagonist activity in B

b. How may the sampling of single motor unit EMG during the contractions help to identify the reasons for the longer contraction duration in B. Assume that in each subject the discharges of several motor units could be followed from the beginning to the end of the contraction. (4)

you expect firing rates to decrease during the first part of the contractions and only in well motivated subjects firing rates will(tend to) increase and variability in firing rate will increase towards the end, if maximal VA is approach

3. A subject has his hand in a dynamometer and we electrically stimulate (50 Hz) the m. adductor pollicis at a thumb angle of 57 °. Following 2 seconds isometric torque development we allow the muscle to shorten at a constant velocity (50 °/s, which is a rather slow speed) until a thumb angle of 35 ° is reached. Following the shortening phase we continue stimulation for 4 seconds.
We repeat this experiment exactly except for one difference: now we allow the muscle to shorten at a high speed of 500 °/s.

a. For both experiments draw the torque (force) as a function of time in a single (clear) graph. Indicate the stimulation and thumb angle in the graph (label axis, don't forget units). (2)

key point: similar isometric torque before shortening: steeper force decrease at 500°/s during shortening without linear phase, higher isometric torque following force redevelopment following high speed shortening

b. For the first experiment describe as carefully as possible what happens with the length of the SEC (series elastic component) during the entire contraction: thus from the beginning of the stimulation until after complete relaxation of force at the end. (4)

-SEC lengthens with decreasing speed until isometric plateau then there is a rapid phase of SEC shortening (consequently CE shorten at lower than imposed velocity) then there is a linear phase of force decline (and consequently SEC shortening continuous at slower speed) due to shortening induced force deficit, then again followed by SEC lengthening during torque redevelopment, phase of constant length (second plateau), followed by shortening during relaxation

c. Explain (give reasons and/or potential mechanisms) the differences between both force traces. (4)

–imposed speed higher thus according to the force velocity relationship the force that CE can sustain at high speeds is lower than at slow speeds, consequently SEC shortening is greater at high shortening speeds and may dominated the entire phase of shortening during contraction- force deficit is higher during and following (it recovers a bit during

torque redevelopment) slow shortening , because shortening induced force deficit is work dependent (with higher forces at 50°/sec)
-most likely mechanisms sarcomere in homogeneity or stress induced inhibition of cb formation in the new overlap zone

4. The dorsal flexors of a patient with a drop foot are electrically stimulated for 1 sec during every walking step (using a switch implemented in his shoe). For these 1 sec contractions you have the option of using either constant 10 Hz stimulation or 10 Hz stimulation with two additional pulses at the start (first 15 ms) of the contraction.
a. Draw the isometric torque time traces, when measured in a dynamometer, in response to both stimulation trains as exactly as possible in a single graph. (3)

-10 clear peaks (fusion is far from complete)
-higher first peaks with gradual decrease towards the end with HFIP

b. Also draw in a second graph the intracellular calcium concentration in response to both stimulation trains as exactly as possible (assume that you are able to monitor intracellular calcium concentration) (4)

-10 clear peaks on baseline [Ca2+] (Ca-ATPase is very active)
-higher [Ca2+] at the beginning (first peak only), thus elevation of much shorter duration than elevation of force (the key element of HFIP)

c. List three potential mechanisms which may account for the differences in force responses between both stimulation trains and discuss the likelihood of each of the potential mechanisms.(3)

-MLC phosphorylation leading to increased CB force (unlikely because many more pulses are needed for MLC phosphorylation to occur)
-stretching of SEC by the first pulses leading to better force transmission for the following pulses (accounts for 20 % of the effect)
-after initial cb binding the binding of the subsequent cbs is facilitated by the shift of tropomyosin, which increases the availability of CB binding places on the actin and therefore leads to increased force production during subsequent pulses (hypothesis)
-increased intra cellular Ca2+ , may explain higher peak force at the start, but not during the subsequent pulses when [CA2+]is very comparable that during the constant frequency train

5. A subject performs two isometric knee extensions of several minutes duration (90º knee angle) at 10 % maximal torque capacity (MTC) once with a cuff inflated around his thigh (450 mmHg) and after 30 min rest once again but now without a cuff.
a. Draw in one figure the signals sampled with a NIRS optode-set from the vastus lateralis muscles during both contractions. Explain your figure and the course of the

signals (and label the axis and signals) (4)

-with cuff, tHb stays constant: there is no in or outflow of blood; following force development, after a short delay (ATP/ADP ratio has to decreases first before mitochondrial oxygen consumption increases) [HbO2] starts to decrease (with mirror image increase of HHb), because oxygen leaves HB and Mb, then there is a linear part of of changes is in HHb and HBO2(steepness depends on the rate of oxygen consumption), followed by a leveling of the traces:maximal deoxygenation (all oxygen consumed) Without cuff: 10 % MVC : probably no complete occlusion of blood flow(inflow of oxygen rich blood possible) thus tHb may vary more, and so will the other traces: the HHb and HBO2 signals vary considerable over tine and maxdeox will not be reached

b. How would you determine muscle VO2? (2)

-mVO2, the rate of change: from the slope of the linear part of the HHb and/or HbO2 traces, which have to be normalized to maxdeox

c. Draw in a single graph the relationship between isometric torque (%MTC) and mVO2 for VL and RF muscle operating at the 60 º knee angle and explain your graphs. (2)

-mVO2 of RF is lower at lower contraction intensities because this biarticular muscle is an inefficient knee extensor (its activation would necessitate compensating recruitment of hip extensors (e.g hamstring muscles which would decrease net knee extension torque output) however during maximal effort also the RF will be maximally activated and have a similar mVO2 as the VL (detail: at zero force production, there should be the resting mVO2)

d. Draw, in a another graph, the relationship between isometric torque (%MTC) and mVO2 for VL muscle operating at the 30º and 60 º knee angle and explain your graphs. (2)

For the entire relationship mVO2 at 30º is about 66 % of that of 60º, possibly due to the lower number of cycling cb at 30º: due to the knee angle torque relations (length force relation) the absolute number of force generating cbs will be lower at the 30 º knee angle (shorter muscle length)