oxygen saturation was 84% on room air and bibasal crepitations, more at right base posteriorly. Electrocardiogram confirmed atrial fibrillation without ischemic changes. Chest x-ray differed little from previous films aside from some consolidation in the right lower zone. High resolution chest CT showed atelectasis at both bases and some pneumonic consolidation of posterior segment of right lower lobe. There was no evidence of interstitial lung disease or pulmonary embolism. Her arterial blood gas was consistent with type II respiratory failure (pH 7.37, pO_2 6.9, pCO_2 9.1, HCO_3 38.6). We considered the possibility of left sided heart failure and pneumonia but were unsure on the cause of her respiratory failure. Despite treatment with digoxin, frusemide and antibiotics there was no improvement. Over the next few days, she had developed a 'head drop' being unable to hold her head upright. At this point it became clear to us that we were dealing with a case of neuromuscular junction disorder. She denied any previous history of diplopia, dysphagia or muscular weakness and fatigability. Tensilon test was positive with dramatic improvement in the power of her neck extensor muscles and oxygen saturation rising from 88-93% on room air. We were unable to check arterial blood gas as she had high international normalized ratio being on warfarin for atrial fibrillation. Auto antibodies screening was negative. Muscle enzymes, serum calcium, magnesium, electrolytes and thyroid function tests were all normal. Anti acetylcholine receptor antibodies assay titer was positive at 222 x 10^ 10M (normal range <5 x 10^ 10M). She responded very well to prednisolone and pyridostigmine and was discharged home. She lived a normal life until recently when she was admitted under surgical services with severe acute pancreatitis and unfortunately succumbed to death.

Myasthenia gravis classically affects young patients in their fourth and fifth decades. Incidence of myasthenia gravis is increasing and this may be explained by the aging population, improvement in prognosis and higher detection rates of patients with milder symptoms.[1] Due to late onset occurrences, myasthenia gravis is still substantially under diagnosed in older people.[2] The majority of patients initially present with ptosis and diplopia and later on develops weakness of proximal limb muscles and respiratory muscles. Approximately 15% of patients have weakness, remained confined to extraocular muscles.[3] Myasthenia gravis has been reported in the elderly patients presenting with falls and bulbar symptoms[4,5] and children presenting with respiratory failure. However, respiratory failure as the first manifestation is unusual and not reported before in the elderly. We wish to recommend that myasthenia gravis should be considered in elderly with breathlessness and respiratory failure of obscure origin.

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Preliminary results of muscle diseases prevalence in patients from Jordan

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We reported 123 patients affected with a remarkable change in muscle strength and muscle weakness of variable clinical severity. The number of patients seen in all clinics during the months of July 2000 to September 2004 is shown in **Table 1**. The most common disease entities were muscular dystrophies (46 cases, 37.4%) followed by myositis diseases (25 cases, 20.3%) most of them were due to autoimmune diseases. Data demonstrated that motor neuron diseases represented the third common group of muscle diseases in this study (24 cases, 19.5%) whereas, mitochondrial myopathies were the fourth common group of muscle diseases (11 cases, 8.9%). On the other hand, type II atrophy which accounted for the fifth group was 8 cases (6.5%). However, metabolic myopathies (5 cases, 4.1%) included lipid storage diseases (2 cases, 1.6%) and glycogen storage diseases (3 cases, 2.4%). Furthermore, our data showed other rare causes of muscle diseases. Congenital muscular dystrophy (2 cases, 1.6%), end

Table 1 - An analysis of muscle disease types during the months of July 2000 to September 2004.

Muscle disease type	Cases diagnosed n	(%)
Dystrophies	46	(37.4)
a. Duchenne/Beckers	29	
b. Limb girdle dystrophy	11	
c. Fasciscapular humeral dystrophy	2	
d. Myotonic dystrophy	1	
e. SCARMD	3	
Myositis	25	(20.3)
a. Autoimmune	21	
b. Viral	1	
c. Inclusion body	3	
Neurogenic atrophy	24	(19.5)
a. Nerve origin	12	
b. ALS	6	
c. Spinal muscular atrophy	5	
d. Myelitis	1	
Mitochondrial myopathy	11	(8.9)
Type II atrophy	8	(6.5)
a. Disuse	4	
b. II b atrophy	3	
c. Paraneoplastic syndrome	1	
Metabolic myopathy	5	(4.1)
a. Glycogen storage disease	3	
b. Lipid storage disease	2	
Congenital muscular dystrophy	2	(1.6)
End stage muscle disease	1	(0.8)
Stiffman syndrome	1	(0.8)

SCARMD - Severe childhood autosomal recessive muscle dystrophy, ALS - amytrophic lateral sclerosis

stage muscle disease (1 case, 0.8%) and Stiffman syndrome (1 case, 0.8%). The muscle biopsy of congenital muscular dystrophy showed severe dystrophic findings by light microscopy. However, the ultrastructural findings confirmed the diagnosis in metabolic and mitochondrial myopathies. Collectively, these findings showed that the classification of muscle diseases in subtypes with a different clinical presentation is useful in clinical practice.

Muscle samples were obtained from the thigh, at a site in the lateral portion of quadriceps femoris by the needle method for the histochemical and electron microscopical investigations.[1-3] The procedure is carried out under sterile conditions. The skin and subcutaneous tissue at the biopsy site were anesthetized by the local infiltration of 2% lidocaine. After anesthesia, a small scalpel incision, approximately 1 cm long using a size 15 blade, is made in the skin and deep fascia to facilitate the pathway of the needle. Upon removal of the needle from the muscle, the wound is closed over with pressure and skin Elastoplast. Frozen specimens were stored at -70ºC until sectioning. For light microscopy, all sections were taken in series and stained with the following histological and immunocytochemical staining list: hematoxylin and eosin mATPase at alkaline and acidic pH; Nicotinamide adenine dinucleotide; gomori trichrome; phosphorylase; succinate dehydrogenase. However, double fixation with 2.5% glutaraldehyde followed by 2% osmium tetroxide of fresh samples, provides the optimal preservation for ultrastructural investigations of muscle samples from patients with mitochondrial and metabolic myopathies.

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Pregnancy outcome following exposure to orlistat, ramipril, glimepiride in a woman with metabolic syndrome

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Most babies are exposed to drugs in utero. Drugs given to mothers during pregnancy can affect the fetus by acting directly on the embryo and produce lethal, toxic or teratogenic effects such as altering placental function, changing the myometrial activity, altering the biochemical dynamics of the mother and by acting indirectly. The use of drugs during pregnancy is a complicated problem, especially for new drugs.

Orlistat, a new drug, has pancreatic lipase inhibitory property that decreases the absorption of ingested dietary fat.[1] Data for the use of orlistat in treating obesity in pregnant women are not available currently. In case of hypertension and angiotensin converting enzyme (ACE) inhibitors in pregnancy,