Numerous authors have commented on the particular relevance of impaired explosive power, which depends on both strength and speed of movement, to the development of functional disability. Nevertheless, little is known about how to combine improvements in muscle speed with improvements in muscle strength, in order to optimise the gain in muscle power. Current exercise programmes have mostly targeted muscle strength (i.e. the ability to generate maximal force), balance and gait. However, muscle power is not only more important for daily-life functional mobility, but it is also more severely impaired. Recent findings in healthy and mildly mobility-limited, community-dwelling older women have shown some beneficial effects of high-velocity training on muscle power, but the results are inconclusive as to the best intervention method for improving power and the applicability of the techniques to the frailest individuals. In addition, the relationship between resistance training and the ability to perform functional tasks remains unclear and it is controversial whether improvements in functional abilities may be a carried over effect of power training, or are more likely to occur if the functional task is also practised. Improved knowledge on the mechanisms of power decline is crucial with regard to the development of effective prevention and treatment programmes for restoring mobility and independence in older people. As power is the product of force and velocity, anything that will affect force production or speed of shortening of a muscle will also affect its power output. Possible mechanisms underlying the improvement in peak power may include specific increases in the cross-sectional area of type II muscle fibres and increases in specific force and shortening velocity of single muscle fibres. These changes would occur through a poorly understood series of events that appears to involve the recruitment of satellite cells to support adaptations of mature muscle fibres. Moreover, appropriate training interventions can induce significant increases in stiffness of the tendon-aponeurosis structures, together with changes in muscle architecture such as increases in resting fascicle length and pennation angle. It has been shown that changes in single motor-unit behaviour contribute to the increase in contraction speed after dynamic training in humans, which could justify training induced changes in power. These changes in motor-unit behaviour include earlier motor-unit activation, extra doublets, i.e. brief (2-5 ms) motor-unit interspike intervals, and enhanced maximal firing rate. Increased activation of the prime mover muscles, better coordination of synergistic and antagonist muscles and increased neural drive from the highest levels of the central nervous system, can also explain improvements in power. Also the slowing in nerve conduction velocities due to ageing may be a mechanism to be recovered following power training. However, it has been shown that conduction velocity of the posterior tibial nerve did not change in older male adults following 16 weeks of resistance training of plantar flexor muscles, thus suggesting that decreased nerve conduction velocity may be due to degenerative phenomena rather than disuse.