A lengthy 17 years after its distinguished predecessor, a second edition of *Pathology of Skeletal Muscle* has now been published. For most of those years I, like many other pathologists, have relied heavily on the first edition for advice on the interpretation of muscle biopsies. To my mind, the descriptions of normal and abnormal structures and their significance, and the clarity and comprehensiveness of the illustrations have not been bettered in any other myopathology text. It was therefore with high expectation that I ordered the second edition, without waiting to see the reviews or even having had a chance to look through it.

The page format is larger and the arrangement of the references different (they are now placed at the end of each topic rather than grouped together at the end of the chapter), but most of the original illustrations are still there and the basic framework has altered very little since the first edition. The majority of changes in the new edition take the form of supplements to the text. These reflect the substantial advances that have occurred over the past decade or so in our understanding of the molecular processes involved in the development of muscle and in the aetiology and pathogenesis of muscular diseases, genetic diseases in particular.

As previously, the book is subdivided into two main sections. The first of these is entitled ‘Structures and reactions’. This starts with an introductory chapter that explains how the book is organized and outlines the authors’ general approach to the examination of muscle biopsies and the classification of muscular diseases. Subsequent chapters cover the techniques involved in taking a muscle biopsy and preparing it for pathological assessment; the normal development, histological, histochemical, immunohistochemical and ultrastructural features of skeletal muscle; the major pathological processes that can affect it; and the range of abnormal reactions and structures that the pathologist might encounter on examining the biopsy. The following are some of the more substantial additions to these chapters. The description of myogenesis has been expanded since the first edition, and includes a summary of the many regulatory factors and other key proteins involved in this process. The chapter on pathological reactions in muscle now considers the roles of apoptosis and necrosis in muscular disease, has an expanded description of muscle regeneration and includes a description of myosin depletion in acute quadriplegic myopathy. The chapter on normal organelles and other constituents in muscle has more detailed information on the cytoskeleton, the composition and organization of myofibrils, excitation–contraction coupling and the structure and function of mitochondria. Finally, the chapter on cells and structures other than skeletal muscle fibres now includes brief information on the composition of the various types of amyloid that can affect muscle, and on the immunophenotype of infiltrating inflammatory cells and the nature of other molecules involved in inflammatory processes within muscle.

The first section of the book includes a few, very minor errors and inconsistencies: I could find no clear description of the histochemical features of type 2C fibres, although my expectations had been raised by the *vide infra* on page 41 to such a description, and plate 9.21, referred to on page 290, seems not to exist. A few diagrams would have helped to explain some aspects of the structure of myofilaments, and excitation–contraction coupling. However, overall this section remains a thorough, very readable and well-illustrated introduction to the main business of myopathology, namely the diagnosis of disease.

The second section has been reorganized, so that diseases of muscle are grouped together in a more logical manner than was previously the case: Chapters 8, 9 and 10 deal respectively with genetic diseases of muscle, sporadic diseases of muscle and denervation. Within Chapter 8, there is further grouping of some of the genetic diseases so that the channelopathies are considered together, as are the congenital muscular dystrophies, diseases due to defects of mitochondrial DNA, lysosomal storage diseases and non-lysosomal storage diseases. This is a more logical approach than the largely alphabetical arrangement of diseases in the first edition and makes it easier for the pathologist to extract comparative information on diseases with similar clinical or pathological features. I think that this approach could usefully have been taken quite a lot further.

Much of the expansion of this section of the book reflects the enormous progress that has been made in our understanding of the aetiology and pathogenesis of most of the genetic disorders of muscle and, aside from a few very recent advances, these topics are mostly well-covered and up-to-date. There are, in addition, completely new entries on some of the congenital muscular dystrophies and on X-linked vacuolar myopathy. In Chapter 9, which covers sporadic diseases of skeletal muscle, there are new entries on HIV- and HTLV-1-related myopathies and on the effects on muscle of zidovudine, and expanded entries on inclusion body myositis and ischaemic myopathy.

In general, the descriptions of diseases of muscle are clear and comprehensive. However, I do have some quibbles. I was slightly disappointed by the brevity of the entry on mitochondrial myopathies and encephalopathies. A few other disorders might have been covered in this book but are not: including macrophagic myofascitis, myoadenylate deficiency (which had an entry in the first edition and about which recent molecular genetic studies have been quite revealing) and the toxic myopathy that occasionally results from the administration of statins. There are some confusing references to plates and figures that have either been renumbered or omitted, e.g. on page 374 to plates 5.10 and 5.11, on pages 496 and 497 to Figs 8.123 and 8.124 (in the legends to Figs 8.112 and 8.113, respectively), on page 505 to Fig. 8.104 (in the legend to Fig. 8.120) and on page 517 to Fig. 8.113 (in the legend to Fig. 8.129). I would have
appreciated a bit more practical advice on differential
diagnosis, and more cross-referencing of some topics (e.g.
it might have been helpful to mention within the entry on
polymyositis the potential confusion with inflammatory
myopathy that could occur when examining biopsies from
patients with facioscapulohumeral dystrophy type 1A or, less
frequently, limb-girdle muscular dystrophy type 2B). 
Although the monochrome illustrations are of high quality
and the additional colour plates well-chosen, it would have
been easier to navigate the book had the colour plates been
incorporated within the relevant text rather than all grouped
together. However, I suppose that this was a deliberate
compromise that was made in order to keep the price of the
book down to a very reasonable level.

I would not want to end this review on a negative
note, for the second edition, like the first, is a well-
written, comprehensively illustrated, competitively priced
book, distilling many years of wisdom from two very
experienced diagnosticians. It should and will, I am sure,
be widely used by neurologists and pathologists with an
interest in myopathology. In the preface, the authors note
that they ‘are not able to provide a quick fix to install an
in-depth knowledge of myopathology’. This book is,
however, a fine place to start.

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Homocysteine in Health and Disease.
Edited by Ralph Carmel and Donald Jacobsen.

This multi-authored book presents 46 chapters written by
more than 50 experts in the area of homocysteine
research, who describe historical aspects, current opinion
and future perspectives of homocysteine in health and
disease.

Homocysteine is a sulphur-containing amino acid that was
first discovered in 1932 and was identified as a product of the
essential amino acid, methionine. Its clinical significance
remained unknown until 1962, when homocysteine was
found to be grossly elevated in the urine of children with
learning disabilities. Other clinical features were seizures,
lens dislocation, skeletal deformities and premature vascular
disease. These children had an inborn error of metabolism in
the homocysteine-metabolising enzyme cystathionine
β-synthase and, since this initial discovery, several other even
rarer metabolic defects of methionine metabolism have been
identified that also cause homocystinuria. In these patients,
vascular disease is the major cause of morbidity, but all are