Corrigenda to first print run of WHO Classification of Tumours of Haematopoietic and Lymphoid Tissues, revised 4th edition

In addition to corrections of minor typographical errors, corrections were made in this print run to improve the text. The chromosomal localization of genes was improved, and Human Genome Variation Society (HGVS) notation was used throughout for translocations, insertions, and other gene alterations. Gene symbols are given in italics as is common usage.

The following acknowledgement of funding bodies was added to page 5:

This volume was produced with support from the following organizations:
American Society of Hematology
Fondazione Italiana Linfomi ONLUS
Fondation José Carreras pour la lutte contre la leucémie, Genève
University of Chicago Medicine Comprehensive Cancer Center
Leukemia Clinical Research Foundation

Summary of corrections:

**Chapter 2: Myeloproliferative neoplasms**

Appel eosinophilic leukaemia, NOS > Table 2.15

1. A referral to p. 57 has been added to the table title, to refer the reader to the Myeloproliferative neoplasm, unclassifiable section, to which this table relates.
2. The word “either” has been removed as shown below. All criteria should be met.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>Table 2.15 Diagnosis criteria for myeloproliferative neoplasm (MPN), unclassifiable</td>
<td>Table 2.15 Diagnosis criteria for myeloproliferative neoplasm (MPN), unclassifiable (see p. 57)</td>
</tr>
<tr>
<td>The diagnosis of myeloproliferative neoplasm (MPN), unclassifiable, requires that either all 3 criteria are met.</td>
<td>The diagnosis of myeloproliferative neoplasm (MPN), unclassifiable, requires that all 3 criteria are met.</td>
</tr>
</tbody>
</table>

**Chapter 6: Myelodysplastic syndromes**

Myelodysplastic syndrome with single lineage dysplasia > Genetic profile

The spelling of the word “cohesin” has been corrected.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatic driver mutations have been identified in 60–70% of cases of MDS-SLD. The underlying mutations affect a haematopoietic stem cell and are present in all lineages despite the limitation of dysplastic findings to one lineage [4354]. TET2 and ASXL1 appear to be the most commonly mutated genes in MDS-SLD [1513]. However, mutations in other DNA methylation genes, splicing factors, RAS pathway genes, cohesin complex genes and RUNX1 are...</td>
<td>Somatic driver mutations have been identified in 60–70% of cases of MDS-SLD. The underlying mutations affect a haematopoietic stem cell and are present in all lineages despite the limitation of dysplastic findings to one lineage [4354]. TET2 and ASXL1 appear to be the most commonly mutated genes in MDS-SLD [1513]. However, mutations in other DNA methylation genes, splicing factors, RAS pathway genes, cohesin complex genes and RUNX1 are...</td>
</tr>
</tbody>
</table>
### Chapter 6: Myelodysplastic syndromes

*Myelodysplastic syndrome with multilineage dysplasia > Genetic profile*

The spelling of the word “cohesin” has been corrected.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>monosomy 5, del(5q) and del(20q), as well as complex karyotypes, are found in as many as 50% of patients with MDS-MLD {2423}. Whole-genome sequencing has shown that more than half of all cases of MDS-MLD carry mutations in genes that are also mutated in MDS with excess blasts and acute myeloid leukaemia. These include genes from the cohesin family (STAG2), chromatin modifiers …</td>
<td>del(5q) or t(5q), and del(20q), as well as complex karyotypes, are found in as many as 50% of patients with MDS-MLD {2423}. Whole-genome sequencing has shown that more than half of all cases of MDS-MLD carry mutations in genes that are also mutated in MDS with excess blasts and acute myeloid leukaemia. These include genes from the cohesin family (STAG2), chromatin modifiers …</td>
</tr>
</tbody>
</table>

### Chapter 8: Acute myeloid leukaemia and related precursor neoplasms

*Acute myeloid leukaemia with mutated NPM1 > Genetic profile*

The spelling of the term “cohesin complex” has been corrected.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>and most frequently involve FLT3 and DNMT3A, but mutations of IDH1, KRAS, NRAS, and cohesin-complex genes are also relatively common {545, 1149}. Although…</td>
<td>and most frequently involve FLT3 and DNMT3A, but mutations of IDH1, KRAS, NRAS, and cohesin complex genes are also relatively common {545, 1149}. Although…</td>
</tr>
</tbody>
</table>

### Chapter 12: Precursor lymphoid neoplasms

*T-lymphoblastic leukaemia/lymphoma > Microscopy*

The word “hyperplasia” was changed to “neoplasia”.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases with histological findings of T-LBL with a significant infiltrate of eosinophils among the lymphoma cells may be associated with eosinophilia, myeloid hyperplasia, and an 8p11.2 cytogenetic …</td>
<td>Cases with histological findings of T-LBL with a significant infiltrate of eosinophils among the lymphoma cells may be associated with eosinophilia, myeloid neoplasia, and an 8p11.2 cytogenetic …</td>
</tr>
</tbody>
</table>
The word “atretic” was changed to “regressed”.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>Like in HCL and splenic diffuse red pulp small B-cell lymphoma, the red pulp of the spleen is diffusely involved and expanded in HCL-v, with atretic or absent...</td>
<td>Like in HCL and splenic diffuse red pulp small B-cell lymphoma, the red pulp of the spleen is diffusely involved and expanded in HCL-v, with regressed or absent...</td>
</tr>
</tbody>
</table>

The diagnostic criterion was changed as shown below.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>There may be as much as 10% bone marrow infiltration by an IgM+ clonal lymphoplasmacytic population. Cases with...</td>
<td>There may be bone marrow infiltration by an IgM+ clonal lymphoplasmacytic population, but it must be &lt; 10%. Cases with...</td>
</tr>
</tbody>
</table>

Table 13.04 has been reformatted as shown below.

<table>
<thead>
<tr>
<th>Table 13.04 Plasma cell neoplasms</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Non-IgM (plasma cell) monoclonal gammopathy of undetermined significance (precursor lesion)</strong></td>
</tr>
<tr>
<td><strong>Plasma cell myeloma</strong></td>
</tr>
<tr>
<td>Clinical variants:</td>
</tr>
<tr>
<td>Smouldering (asymptomatic) plasma cell myeloma</td>
</tr>
<tr>
<td>Non-secretory myeloma</td>
</tr>
<tr>
<td>Plasma cell Leukemia</td>
</tr>
<tr>
<td><strong>Plasmacytoma</strong></td>
</tr>
<tr>
<td>Solitary plasmacytoma of bone</td>
</tr>
<tr>
<td>Extraosseous (extramedullary) plasmacytoma</td>
</tr>
<tr>
<td><strong>Monoclonal immunoglobulin deposition diseases</strong></td>
</tr>
<tr>
<td>Primary amyloidosis</td>
</tr>
<tr>
<td>Systemic light and heavy chain deposition diseases</td>
</tr>
<tr>
<td><strong>Plasma cell neoplasms with associated paraneoplastic syndrome</strong></td>
</tr>
<tr>
<td>POEMS syndrome</td>
</tr>
<tr>
<td>TEMPI syndrome (provisional)</td>
</tr>
</tbody>
</table>
One of the diagnostic criteria for light-chain monoclonal gammopathy of undetermined significance (MGUS) was changed as shown below.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal free light chain ratio (&lt; 0.26 or &gt; 1.65) Increased level of the involved free light chain No immunoglobulin heavy chain expression on immunofixation electrophoresis Urinary M protein &lt; 500 mg/24 hours Clonal plasma cells &lt; 10% Absence of end-organ damage (CRAB) and amyloidosis</td>
<td>Abnormal free light chain ratio (&lt; 0.26 or &gt; 1.65) Increased level of the involved free light chain No abnormal immunoglobulin heavy chain expression on immunofixation electrophoresis Urinary M protein &lt; 500 mg/24 hours Clonal plasma cells &lt; 10% Absence of end-organ damage (CRAB) and amyloidosis</td>
</tr>
</tbody>
</table>

A sentence has been deleted as shown below.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>The postulated normal counterparts are post–germinal centre long-lived plasma cells in which the IG genes have undergone class switch and somatic hypermutation. The cell of origin has not been established.</td>
<td>The postulated normal counterparts are post–germinal centre long-lived plasma cells in which the IG genes have undergone class switch and somatic hypermutation.</td>
</tr>
</tbody>
</table>

Table 13.09 has been reformatted as shown below.

Table 13.09  The International Myeloma Working Group (IMWG) consensus recommendations on genetic testing. Adapted from Forseca R, et al. [1232]

**FISH (on cell-sorted samples or cytoplasmic Immunoglobulin FISH)**

- Minimal panel:
  - t(4;14)(p16;q32),
  - t(14;16)(q32;q23),
  - del(17p)/13.1

- More comprehensive panel:
  - t(11;14)(q13;32),
  - del 13,
  - ploidy category,
  - chromosome 1 abnormalities

Clinical trials should incorporate gene expression profiling
The page number for Table 14.06 and a referral to Table 14.07 have been added.

**Chapter 14: Mature T- and NK-cell neoplasms**  
*Mycosis fungoides > Staging*  

The page number for Table 14.06 and a referral to Table 14.07 have been added.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>See Table 14.06.</td>
<td>See Table 14.06 (p. 387) and Table 14.07 (p. 390).</td>
</tr>
</tbody>
</table>

**Chapter 14: Mature T- and NK-cell neoplasms**  
*Anaplastic large cell lymphoma, ALK-negative > Fig. 14.171*  

The caption for panel C of Fig. 14.171 has been modified as shown below.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>C Typical morphological and phenotypic features of ALCL, including a hallmark cell (arrow), that arose in the gastrointestinal tract (glandular epithelium is visible in the upper left).</td>
<td>C Typical morphological and phenotypic features of ALCL, including a hallmark cell (arrow), that arose in the gastrointestinal tract.</td>
</tr>
</tbody>
</table>

**Chapter 15: Hodgkin lymphomas**  
*Introduction > Fig. 15.08*  

The caption for panel A of Fig. 15.08 has been modified as shown below.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Radioactive in situ hybridization for Igμ mRNA is negative in the Hodgkin/Reed–Sternberg cells (arrows), whereas the two non-neoplastic plasma cells in the upper edges are strongly positive, and the non-neoplastic bystander small B cells are moderately strongly positive.</td>
<td>A Radioactive in situ hybridization for Igμ mRNA is negative in the Hodgkin/Reed–Sternberg cells (arrows), and the non-neoplastic bystander small B cells are moderately strongly positive.</td>
</tr>
</tbody>
</table>

**Chapter 15: Hodgkin lymphomas**  
*Introduction > Fig. 15.09*  

The word “non-radioactive” has been removed from the caption for panel B of Fig. 15.09.

<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>B EBV-infected Hodgkin/Reed–Sternberg cells consistently show a strong expression of EBV-encoded small RNA (EBER) in their nuclei as revealed by non-radioactive in situ hybridization.</td>
<td>B EBV-infected Hodgkin/Reed–Sternberg cells consistently show a strong expression of EBV-encoded small RNA (EBER) in their nuclei as revealed by in situ hybridization.</td>
</tr>
</tbody>
</table>
A cited PMID has been replaced with the corresponding reference number (2832A), and the reference (as follows) has been added to the Reference list at the back of the book (on p. 549):


<table>
<thead>
<tr>
<th>Original Text</th>
<th>Corrected Text</th>
</tr>
</thead>
<tbody>
<tr>
<td>The clinical presentation of monomorphic B-PTLDs is not distinctive and is, in general, similar to the presentation of the lymphomas or plasma cell neoplasms that they resemble. The EBV+ MALT lymphoma M-PTLDs are distinctive, with a frequently cutaneous/subcutaneous presentation. They occur late after transplantation and are solitary, and the patients do well {23773403}.</td>
<td>The clinical presentation of monomorphic B-PTLDs is not distinctive and is, in general, similar to the presentation of the lymphomas or plasma cell neoplasms that they resemble. The EBV+ MALT lymphoma M-PTLDs are distinctive, with a frequently cutaneous/subcutaneous presentation. They occur late after transplantation and are solitary, and the patients do well {2832A}.</td>
</tr>
</tbody>
</table>

List of abbreviations

The following entries have been added to the list of abbreviations at the back of the book:

- **HAART**: highly active antiretroviral therapy
- **IG gene**: immunoglobulin gene
- **KSHV**: Kaposi sarcoma–associated herpesvirus – an alternative name for human herpesvirus 8 (HHV8)
- **LMP1**: latent membrane protein 1 (of Epstein–Barr virus)
- **MALT**: mucosa-associated lymphoid tissue
- **R-CHOP**: the CHOP chemotherapy regimen plus rituximab
- **TR gene**: T-cell receptor gene

Back cover

The barcode is printed incorrectly on the back cover.