

THE UNIVERSITY OF
NEW SOUTH WALES



School of Medical Sciences
Department of Pathology
Student Manual
PATH3207
Musculoskeletal Diseases
2013

Musculoskeletal Diseases Manual

PATH3207

2013

Preface

This is the tenth edition of the manual for Musculoskeletal Diseases produced by the staff of the Department of Pathology at the University of New South Wales. It contains a large amount of relevant information regarding the course PATH3207 Musculoskeletal Diseases.

We recognise that this manual might contain some errors and may need further improvements in the future. Therefore, we welcome comments from staff and students and seek your co-operation in identifying errors of content or style, so that they may be corrected in subsequent editions.

Editors:

A/Prof Nicodemus Tedla

A/Prof Gary Velan

Department of Pathology Student Manual: Musculoskeletal Diseases

ISBN 0-7334221-9-5

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Introduction

We would like to warmly welcome third year science students to the **Musculoskeletal Diseases** course, offered in Session 2, 2013, by the Department of Pathology. The course covers bone and joint disease, neuromuscular disease, musculoskeletal trauma and orthopaedics.

This course will be beneficial to students wishing to pursue careers in the health sciences, especially rehabilitation medicine. A sound understanding of musculoskeletal pathology should provide an effective framework from which to approach diagnosis and management of common clinical scenarios that you may well encounter in your future careers.

Staff of the Department of Pathology joins me in wishing you an interesting and enjoyable session.

Nicodemus Tedla

A/Professor in Pathology – PATH3207 Course Convenor

Official Communication by email

All students in course PATH3207 are advised that email is now the official means by which the School of Medical Sciences at UNSW will communicate with you. All email messages will be sent to your official UNSW email address (e.g., z1234567@student.unsw.edu.au) and, if you do not wish to use the University email system, you **MUST** arrange for your official mail to be forwarded to your chosen address. The University recommends that you check your mail at least every other day. Facilities for checking email are available in the School of Medical Sciences and in the University library. Further information and assistance is available from DIS-Connect, Tel 9385 1777. The UNSW Library runs free email courses.

Course Outline

Campus Based Course staff

A/Professor N Tedla (Course Convenor), Dr P Polly, A/Professor G Velan (Head of Teaching in Pathology), Professor N Hawkins (Head of School), Professor R Kumar, Dr M Dziegielewski, Dr S Van Es, Dr C Van Vliet Dr T Grassi and Dr M Meerkin.

Course Administration

Administrative and general problems related to your attendance, or the content and conduct of the course, can in the first instance be addressed by consulting A/Prof Nicodemus Tedla by e-mail (n.tedla@unsw.edu.au) and in the second instance be addressed by consulting A/Prof Gary Velan (g.velan@unsw.edu.au). Students wishing to see their tutors or other members of staff should call in at the School office (ground floor) and make an appointment with the assistance of the staff.

Attendance is **mandatory** at the lectures and practical classes in this course. Students that fail to attend >80% of the lectures tutorials and practical classes may not be allowed to complete the course.

Guidelines on extra-curricular activities affecting attendance are found at:

<http://medicallsciences.med.unsw.edu.au/sites/soms.cms.med.unsw.edu.au/files/Extra-curricularActivitiesSOMS.pdf>

If students have difficulties of a personal nature, they should contact the School's Grievance Officer, Dr P. Pandey (p.pandey@unsw.edu.au) or Professor Nick Hawkins, the Head of School.

If a student(s) want to have a result reviewed (checking of marks and/or reassessment), they should formally apply through <https://my.unsw.edu.au/student/academiclife/assessment/Results.html>

To appeal academic standing or ability to progress visit

<https://my.unsw.edu.au/student/academiclife/assessment/academicstandinghowtoappeal.html>

Should you feel that there are particular circumstances that have affected your performance in the course; you should lodge an application for special consideration. Special considerations sought outside the 3 day rule will not be accepted except under TRULY exceptional circumstances. The procedures involved in this are outlined in the UNSW Student Guide, and special forms are widely available on campus e.g. Student Health Centre, Student Centre or at <https://my.unsw.edu.au/student/atoz/SpecialConsideration.html>

Information on the different research units in the Department of Pathology and the research interests of each staff member is available at Department of Pathology's home page at <http://medicallsciences.med.unsw.edu.au/>

Student Support Service

Those students who have a disability that requires some adjustment in their teaching or learning environment are encouraged to discuss their study needs with the course convenor prior to, or at the commencement of, their course, or with the Equity Officer (Disability) in the Equity and Diversity Unit (9385 4734 or www.equity.unsw.edu.au/disabil.html). Issues to be discussed may include access to materials, note-takers, the provision of services and additional exam and assessment arrangements. Early notification is essential to enable any necessary adjustments to be made. Information on designing courses and course outlines that take into account the needs of students with disabilities can be found at:

www.secretariat.unsw.edu.au/acboardcom/minutes/coe/disabilityguidelines.pdf

Any student experiencing difficulty with the course should discuss this either with the Convenor of PATH3207 A/Prof Tedla, or the Head of Department A/Prof G. Velan.

Course Details

This course is offered during Session 2 and carries six units of credit. Successful completion of an introduction to basic diseases processes in second year (PATH 2201 or PATH 2202) and in basic Histology (ANAT 2511) and Anatomy (ANAT 2111, ANAT 1521 or ANAT 2241) are prerequisites for enrolment in the course. Molecular basis of inflammation and infection in third year (PATH 3205) is highly recommended. Attendance at all tutorials, practical classes and to more than 80% of the lectures is mandatory.

Course Objectives

PATH 3207 comprises teaching current concepts of musculoskeletal diseases including arthritis, metabolic bone diseases, neoplasms in bone, causes of musculoskeletal pain and limitations of movement and neuromuscular

diseases as well as detailed coverage of fracture healing and its complications, multiple traumas and of biomaterial and prosthetic devices relevant to orthopaedic applications.

Student Learning Outcomes

At the completion of this course you should be able to:

1. Describe and explain the molecular and cellular pathogenic mechanisms of musculoskeletal and neuromuscular diseases;
2. Describe the macroscopic and microscopic appearances of musculoskeletal and neuromuscular diseases;
3. Correlate the clinical features of musculoskeletal and neuromuscular diseases with the underlying pathological processes and mechanisms;
4. Describe the sensitivity, specificity, cost effectiveness and availability of laboratory and imaging investigations for the diagnosis of musculoskeletal diseases;
5. Discuss recent advances in biomedical, bioengineering, molecular and biological research related to the treatment of musculoskeletal and neuromuscular diseases;
6. Develop written and oral skills in scientific communication;
7. Develop skills in peer review and assessment of scientific research.

Graduate Attributes

The students will be encouraged to develop the following Graduate Attributes by undertaking the selected activities and knowledge content. These attributes will be assessed within the prescribed assessment tasks. Please see the Assessment section for more details:

1. An in-depth engagement with the relevant disciplinary knowledge in its interdisciplinary context.
2. The capacity for analytical and critical thinking, as well as for creative problem solving
3. The ability to engage in independent, team-based and reflective learning
4. The skills of effective communication

Rationale for the Inclusion of Content and Teaching Approach

The intended learning outcomes are achieved through study of the common patterns of response to injury, which are often referred to as pathological processes. In depth study of mechanisms and causes unique to the musculoskeletal system are highlighted in context of the general pathological processes. To understand these processes, you will draw on your knowledge of normal anatomy, histology, biochemistry, physiology, general pathology and biomedical engineering.

This course will be beneficial to students wishing to pursue careers in the health sciences, especially in clinical rehabilitation medicine, biomedical research or hospital-based laboratory work. A sound understanding of musculoskeletal pathology should provide an effective framework from which to approach diagnosis and management of common clinical scenarios that you may well encounter in your future careers.

Teaching Strategies

The course employs a variety of teaching modes in order to facilitate your learning:

1. A series of lectures introduce you to pathological processes, as well as specific examples of those processes affecting the musculoskeletal system. These lectures are given by invited and campus based discipline experts.
2. Tutorials that are designed in a form of team-based collaborative learning that incorporate small group tutorials and a series of topical quizzes to be completed individually and as a team. It is anticipated that students will have an enhanced learning experience through the use of team-based learning and peer teaching. The tutorials intended to extend and amplify your understanding of material presented in lectures in an interactive format, where you are given opportunities to seek clarification on any aspect of the topics covered, as well as to tackle concepts that might be difficult to grasp.
3. Practical classes that incorporate clinico-pathological correlation sessions are intended to allow you to apply your understanding of disease processes to microscopic and macroscopic appearances of disease in tissues (lesions), and to correlate these with the clinical manifestations. Computer-based virtual microscopy is utilised together with a variety of diagnostic imaging modalities and laboratory investigations, in order to permit correlation between disease processes, changes in cells and tissues at the microscopic level and the clinical manifestations of disease.
4. Evidence based symposia based on cutting edge topics in musculoskeletal diseases that are organised, designed, delivered and assessed by students working in small groups.

5. A midsession written exam with group and individual feedback aimed at familiarising students with the end of the year practical and written exams and providing students with tailored feedback.
6. Two sessions of group discussion/feedback on homework tasks, the midsession exam and specific student questions.
7. Learning is supported via an eLearning Moodle module (accessible via student number and zPass at <https://moodle.telt.unsw.edu.au/>). Announcements, timetables, lecture slides and other resources will be made available during the course.
8. The PATH3207 Student Manual contains specific learning objectives for tutorials and practical classes, together with the course timetable and useful background information.

Recommended Text

You are expected to use the following text available online via the UNSW library Sirius website at <http://sirius.library.unsw.edu.au> (zID and zPass required). Search for the database MD Consult, then search for Robbins Basic Pathology. *Robbins Basic Pathology*. 9th edition. V. Kumar, A.K. Abbas, & J.C. Aster (2012). Saunders & Co. Philadelphia PA; Elsevier Saunders.

Robbins and Cotran Pathologic Basis of Disease 8th edition. V. Kumar, A.K. Abbas & N. Fausto (2010) Elsevier Saunders is highly recommended for students wishing to study the molecular biology or clinical features of diseases in greater depth.

Research Opportunities

Opportunities exist for all students wishing to undertake undergraduate and postgraduate research program within the School of Medical Sciences. Information can be accessed via the Faculty of Medicine directory for the School of Medical Sciences at: <http://notes.med.unsw.edu.au/home/medweb.nsf/website/5.1.MedicalSciences?OpenDocument>

Students are also encouraged to communicate with invited guest lecturers that are active in research and clinical practice.

Course Evaluation and Development

Periodically student evaluative feedback on the course is gathered, using UNSW's Course and Teaching Evaluation and Improvement (CATEI) Process and an in-house course evaluation questionnaire. This questionnaire is included in the manual to be completed by all students during Practical Class 11 (week 11) to provide feedback on the course. Student feedback is taken seriously, and continual improvements are made to the course based in part on such feedback.

Course Schedule

Week	Date	Time	Location	Lecturer	Title
1	31/7/2013	11	Biomed ThD	de Permentier	Lecture - Revision of Bone and Joint Histology
	01/8/2013	14	Biomed ThF	Kumar	Lecture - Pathological Basis of Bone/Joint pain and limitation of movement
	02/8/2013	12	WW G6/G7 WW G10/G16	Goh/Shum/ Magarinos/Zarzour	Tutorial - Anatomy of Bone and Joints
	02/8/2013	13	WW G6/G7	Tedla/Goh/Shum/ Magarinos/Zarzour	Practical - Histology of Bone and Joints
2	07/8/2013	11	Biomed ThD	Tedla	Lecture - Fracture Healing I
	08/8/2013	14	Biomed ThF	Tedla	Lecture - Fracture Healing II
	09/8/2013	12	WW G6/G7 WW G10/G16	Goh/Shum/ Magarinos/Zarzour	Tutorial - Fracture Healing and Complications
	09/8/2013	13	WW G6/G7	Tedla/Goh/Shum/ Magarinos/Zarzour	Practical - Histopathology of Fractures
3	14/8/2013	11	Biomed ThD	Tedla	Prelude to evidence-based symposium
	15/8/2013	14	Biomed ThF	Grassi	Lecture - Back Pain
	16/8/2013	12	WW G6/G7	Grassi/Shum/ Magarinos/Zarzour	Combined Tutorial and Practical - Back pain
4	21/8/2013	11	Biomed ThD	Dziegielewski	Lecture - Bone Tumours I
	22/8/2013	14	Biomed ThF	Dziegielewski	Lecture - Bone Tumours II
	23/8/2013	12	WW G6/G7 WW G10/G16	Goh/Shum/ Magarinos/Zarzour	Tutorial - Primary and Secondary Bone Tumours
	23/8/2013	13	WW G6/G7	Tedla/Goh/Shum/ Magarinos/Zarzour	Practical - Histopathology of Bone Tumours
5	28/8/2013	11	Biomed ThD	McNeil	Lecture - Arthritis I
	29/8/2013	12	Biomed ThF	McNeil	Lecture - Arthritis II
	30/8/2013	12	WW G2/G4 WW G10/G16	Goh/Shum/ Magarinos/Zarzour	Tutorial - Arthritis
	30/8/2013	13	WW G6/G7	Kumar/Goh/Shum/ Magarinos/Zarzour	Practical - Histopathology of Arthritis and Clinical correlations
6	04/9/2013	11	Biomed ThD	Vu	Lecture – Strains, Sprains and Dislocations
	05/9/2013	14	Biomed ThF	Morris	Lecture - Diagnostic Imaging of Musculoskeletal Diseases
	06/9/2013	12	WW G6/G7		MIDSESSION EXAM
7	11/9/2013	11	Biomed ThF	Duflou	Lecture - Forensic Pathology of the Musculoskeletal System
	12/9/2013	14	Biomed ThF	McFarland	Lecture - New approaches in Musculoskeletal Repair
	13/9/2013	12	WW G6/G7 WW G10/G16	Goh/Shum/ Magarinos/Zarzour	FEEDBACK – MIDSESSION EXAM
	13/9/2013	13	WW G6/G7	Tedla/Goh/Shum/ Magarinos/Zarzour	Revision – Homework Tasks

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Week	Date	Time	Location	Lecturer	Title
8	18/9/2013	11	Biomed ThD	Tedla/Herbert	Evidence-based symposium
	18/9/2013	11	Biomed ThA	Polly/Velan	Evidence-based symposium
	19/9/2013	14	Biomed ThF	Tedla/Velan	Evidence-based symposium
	19/9/2013	14	Biomed ThE	Polly/Herbert	Evidence-based symposium
8	20/9/2013	12	Biomed ThD	Tedla/Herbert	Evidence-based symposium
	20/9/2013	12	Biomed ThF	Polly/Velan	Evidence-based symposium
	20/9/2013	13	Biomed ThD	Tedla/Herbert	Evidence-based symposium
	20/9/2013	13	Biomed ThF	Polly/Velan	Evidence-based symposium
Midsession Break					
9	25/9/2013	11	Biomed ThD	Meerkin	Lecture – Metabolic Bone Diseases
	26/9/2013	14	Biomed ThF	Buckland	Lecture - Pathological basis of neuromuscular diseases
	27/9/2013	12	WW G6/G7 WW G10/G16	Goh/Shum/ Magarinos/Zarzour	Tutorial – Metabolic Bone Diseases
	27/9/2013	13	WW G6/G7	Meerkin/Goh/Shum/ Magarinos/Zarzour	Practical – Clinico-pathological correlations of metabolic Bone Diseases
Midsession Break					
10	09/10/2013	11	Biomed ThD	Polly	Lecture - Muscular Dystrophies
	10/10/2013	14	Biomed ThF	Velan	Lecture – Pathogenesis of Shock
	11/10/2013	12	WW G6/G7	Polly/Shum	Combined Tutorial and Practical - Muscle Diseases
Online Revision Quiz with Automated Feedback Opens on 16/10/2013					
11	16/10/2013	11	Biomed ThD	Dziegielewski	Lecture - Head Injury
	17/10/2013	14	Biomed ThF	Bowring	Lecture - Rehabilitation of Neuro-Musculoskeletal Diseases
	18/10/2013	12	WW G6/G7	Tedla/ Goh/Shum/ Magarinos/Zarzour	Combined Tutorial and Practical -Head injury and Shock
12	23/10/2013	11	Biomed ThD	Krishnan	Lecture – Pathological Basis of Upper and Lower Motor Neuron Lesions
	24/10/2013	14	Biomed ThF	Tedla	Revision - Homework Tasks
	25/10/2013	12	WW G6/G7	Tedla/Shum/Magarinos	Practical Examination
Online Revision Quiz with Automated Feedback Closes on 11/11/2013					

NOTE: Any changes in timetable will be announced on Moodle at <https://moodle.telt.unsw.edu.au/>

Lecture Program Outline

Lecture Title	Lecturer	Content outline
Revision of bone and joint histology	PD	Types of bones and joints, histology of synovial joint, micro architecture of bone, processes of bone formation and bone re-modelling
Pathological bases of bone/joint pain and limitation of movement	RKK	Aetiology, pathogenesis and diagnosis of bone and joint pain
Fracture healing I	NT	Types of fractures, stages of fracture healing, determinants of traumatic fracture healing and assessment of bone healing
Fracture healing II	NT	Acute, intermediate and chronic complications of fractures
Prelude to evidence-based symposium	NT	Introduction to the protocols and guidelines of the symposium, selection of topics and outline of timetable.
Back Pain	TG	Aetiology and pathogenesis back pain: Comparison of intervertebral disc disease, degenerative joint disease and inflammatory arthropathies
Bone Tumours I	MD	Types of bone tumours, macro and microscopic features, clinical features and complications
Bone Tumours II	MD	Metastases to bone; sources of metastases, histopathological features; Involvement of the bone in haematological malignancies
Arthritis I	PMcN	Rheumatoid arthritis: Aetiology, pathogenesis, clinical features, diagnosis and complications
Arthritis II	PMcN	Causes of arthritis; pathogenesis and clinical features of osteoarthritis and crystal induced arthropathies
Strains, sprains and dislocations	DV	Clinical evaluation of muscle, tendon, ligament and meniscus injuries with special emphasis to shoulder and elbow dislocation and knee and ankle injuries.
Diagnostic imaging of musculoskeletal diseases	SM	An outline of types of imaging techniques available for musculoskeletal diseases and their indications, cost, advantages and disadvantages
Forensic pathology of musculoskeletal system	JD	Medico-legal relevance of investigation of death; Comparisons of coronial and hospital autopsy; Forensic investigation of musculoskeletal injuries
New approaches to musculoskeletal repair	CM	Summary on a cutting edge research on new approaches in treatment of musculoskeletal damages
Metabolic bone disease	MM	Classification; macroscopic, microscopic, radiological and clinical features; complications
Pathological basis of neuromuscular diseases	MB	Clinical and histo-pathological features of myopathy, myasthenic disorders, and neurogenic disorders resulting in muscle disease; investigation of muscle diseases and indications for muscle biopsy.
Muscular dystrophies	PP	Causes and effects of muscular dystrophies, histo-pathological diagnosis and indications for muscle biopsies
Rehabilitation of musculoskeletal diseases	GB	Outline indications, general approaches and effectiveness of rehabilitation programs in common Neuro-Musculo-Skeletal diseases; Discuss cost effectiveness of rehabilitation.
Pathogenesis of shock	GV	Definition, pathophysiology, causes and effects
Head injury	MD	Intracranial haemorrhage-epidural, subdural, subarachnoid, intracerebral: causes and effects
Upper and lower motor neuron lesions	AK	Pathological basis of UMN and LMN lesions, compare and contrast clinical manifestations and discuss underlying aetiology

KEY:

Bowring	Dr Greg Bowring	Senior lecturer, FAFRM (RACP), UNSW; Staff Specialist, POWH
Buckland	A/Prof Michael Bucland	MBBS PhD FRCPA, Head Neuropathology, Royal Prince Alfred Hospital, SydU
Dziegielewski	Dr Mark Dziegielewski	Lecturer, SOMS, Department of Pathology, UNSW
Grassi	Dr Tanya Grassi	Lecturer, SOMS, Department of Pathology, UNSW
Hawkins	Prof Nicolas Hawkins	Professor, Head of SOMS, Department of Pathology, UNSW
Krishnan	A/Prof Arun Krishnan	A/Prof, SOMS, Department of Neurophysiology, UNSW
Kumar	Prof Rakesh Kumar	Professor, SOMS, Department of Pathology, UNSW
McFarland	A/Prof Clive McFarland	A/Professor, Graduate School of Biomedical Engineering, UNSW
McNeil	Prof HP McNeil	Professor of Rheumatology, UNSW and SWAHS, Liverpool Hospital
Meerkin	Dr Mathew Meerkin	Senior lecturer, SOMS, Department of Pathology, UNSW
Morris	Dr Sarah Morris	Senior lecturer, Department of Radiology, POWH
Polly	Dr Patsie Polly	Senior lecturer, Department of Pathology, UNSW
Tedla	A/Prof Nicodemus Tedla	A/Professor, Department of Pathology, UNSW
Velan	A/Prof Gary Velan	A/Professor, SOMS, Department of Pathology, UNSW
Vu	Dr Dzung Vu	Senior Lecturer, SOMS, Department of Anatomy, UNSW

Guide to Practical Classes

Practical classes and tutorials in Musculoskeletal Diseases are aimed at amplifying and extending your understanding of the topics gleaned from attendance at lectures and reading of the recommended text, as well as correcting any misconceptions. Hence, adequate preparation and active participation are essential.

Practical classes will reinforce the clinico-pathological correlations involved with each topic. They are intended to help you to acquire the ability to recognize the macroscopic and microscopic features of pathology specimens and to relate the pathology to clinical application. The format of each practical class will be at the discretion of the tutor. Macroscopic “pots” will be generally used in conjunction with projected microscopic slides, x-rays and other materials. Materials for the practical classes are located at <http://vslides.unsw.edu.au/>. Remember, it is much better to make a mistake in the relative safety of a practical class, than to make a critical error in an essay or exam because of misconception of basic pathological principles. *It is highly recommended that you regularly visit the Museum of Human Disease.*

A simple guide to description of macroscopic specimens (“pots”)

The best approach to the study of macroscopic specimens in the Museum is to be systematic. As you cover each lecture topic this year, you should make it a point to visit the Museum to become familiar with macroscopic examples of that disease process, and other related conditions. One of the major tasks for you will be to learn how to differentiate with the naked eye between disease processes that at first glance have similar appearances. Sometimes this cannot be accomplished even by close examination, in which case you should formulate a list of differential diagnoses, in order of decreasing likelihood. All this takes time and careful attention to honing your skills of observation in the Museum. *In addition to the specimens and related conditions covered during practical classes, you are expected to cover all specimens in Bay 6 ,Bay 16, Bay 17 and Bay 29.*

1) Anatomical description

Almost all macroscopic specimens will contain sufficient “normal” tissue for you to identify the organ(s) of origin. Hence a good appreciation of normal anatomy is required (i.e. pathology requires integration with your previous studies). Knowledge of the normal dimensions of organs is important in order to comment on pathological enlargement, distortion or shrinkage of tissue. The way in which the tissue has been mounted is also relevant. For example, bones are usually kept intact or cut longitudinally to display abnormalities in the bone marrow and medulla.

2) Description of the lesion(s)

A “lesion” is a recognisable abnormality in an organ or tissue caused by injury or disease. Lesions can be sub-classified into “focal” (localised), “multifocal” and “diffuse” (an abnormality of the entire organ or tissue). An example of a focal lesion is a tumour in the lower part of femur. You should describe focal lesions as you would describe a lump in a surgical patient, e.g. “There is a mass lesion 5 cm in diameter above the knee, pushing the periosteum and extending to the overlying muscle. The mass is predominantly solid and whitish in colour, with focal areas of brown-red discolouration (haemorrhage) and softening (necrosis).”

3) Identification of the major pathological process

Once you obtain a basic knowledge of the classification of disease, it is possible to categorise abnormalities in tissue as traumatic, inflammatory (acute or chronic), vascular (thrombosis, embolism, infarction, haemorrhage), disorders of growth (atrophy, hyperplasia, hypertrophy, hamartoma, neoplasia - benign or malignant, primary or metastatic), metabolic or degenerative. For example, the qualities of the bone lesion described above are typical of a primary malignant tumour - a single, abnormal, invasive mass that has overgrown the surrounding tissue, with areas of necrosis and haemorrhage (indicative of rapid growth).

4) Related lesions and complications

It is important to integrate your description with your theoretical knowledge of disease causation and complications. For example, wrinkled skin (solar elastosis) surrounding a skin cancer on the back of the hand is caused by the same agent as the tumour - ultraviolet radiation. In the above example, it is important to note whether the bone tumour has been complicated by invasion to the blood vessels and or spread to other bones (as osteosarcomas often do), because this has prognostic implications.

5) Anatomical diagnosis

The diagnosis is no longer a guessing game once you become aware of the basic pathological principles - your description justifies the selection of which pathological process(es) are operative, which you then relate to the anatomy and to your knowledge of the natural history of disease to formulate a tissue diagnosis. In the above example, the diagnosis is “primary osteosarcoma of the lower femur, complicated by metastases to the vertebrae”.

Remember: Your descriptive skills will only improve with practice. It is recommended that students work through the Museum in pairs or small groups - one student is armed with a textbook, lecture notes and Museum catalogue, while the other(s) act as “the guinea pig” and are required to describe and identify the specimens. **Be warned: it is useless for you to look at a number on a specimen, refer to that number in the Museum catalogue and learn it by rote.** That is not an approach befitting thoughtful prospective professionals. It is much better to look carefully at a specimen, attempt to identify the disease process, justify your diagnosis, and only then refer to the catalogue, textbook and lecture notes. In the event that you are unable, even after careful thought and referral to the text, to work out why a particular diagnosis was made, then you should ask your tutor at a convenient time.

A simple guide to writing histopathological descriptions

Haematoxylin and eosin are used for staining all routine sections, and special stains are used only to confirm or refute the presence of a particular substance in the tissue. In addition, histochemistry, immunohistochemistry and electron microscopy may be used extensively in the hospital situation to confirm a clinical diagnosis. Haematoxylin is preferentially taken up by nucleic acids and stains them blue, hence any highly cellular tissue will appear blue (basophilic). Other sources of basophilia include hyaline cartilage, calcium salts and bacterial colonies. Eosin is preferentially taken up by proteins, hence any proteinaceous tissue will appear pink (eosinophilic). Clear spaces may be caused by fat (washed out by aqueous fixatives), water or air. If you have an atlas of histology you may find it useful at these classes. We assume that you are acquainted with the normal histological appearances of human tissues - if not, revise this prior to examining the histopathology slides.

Armed with the basics outlined above, it is possible to write a histopathological description, which should possess the following components:

1) Anatomical and General Description

- **Draw a simple sketch of the main features** to remind you of these areas when you look at the screen or look down at the microscope. This can be used to clarify your description, e.g. area A in the sketch is strongly eosinophilic and is an area of haemorrhage, B is palely eosinophilic and is an area of fibrosis, etc.
- **Make a general statement that both identifies the tissue and indicates whether the lesion is focal or diffuse.** For example, "*Slide 44 is a 2 X 2 cm section of peripheral lung tissue (i.e. it contains no major bronchi) including one pleural surface that contains a focal basophilic lesion labelled area A. The surrounding normal lung tissue is labelled area B.*" Or "*Slide 25 is a section through the left ventricle measuring 2 X 1.5 cm including pericardium, myocardium and endocardium. The tissue is diffusely abnormal.*"

2) Description of the Major Lesion and Identification of the Major Pathological Process

- These elements require a thorough appreciation of the entirety of the section. Such an appreciation cannot be achieved by using only the 40X objective, which will result in failure to see the forest for the trees. Remember the following maxim: Use a low-power objective and a high-powered mind (not *vice versa*)!
- Avoid the trap of describing each abnormal feature in the order that you discover it, without any regard to its relationship to the totality of the lesion. That is, your description requires prior thought, interpretation and planning. By all means jot down your observations on scrap paper, but then order them (so as to exhibit your understanding of "the big picture"). The major pathological process (e.g. acute inflammation, malignant neoplasia) should then become obvious to the informed reader even before you have named it.

3) Identification of Related Lesions

- Sections may contain abnormalities that either share a common aetiology with or predispose to the major lesion (e.g. solar damage to dermal collagen in skin adjoining a melanoma), or else complicate the main lesion (e.g. invasion of dermal lymphatic vessels by melanoma cells). Linking of these elements requires an alert mind (which we hope you already possess) and an understanding of the natural history of disease (which you will acquire with study). Some complications are so important that it is necessary to comment on their absence (e.g. lymphatic or venous invasion by malignant neoplasms).

4) Tissue Diagnosis

- This should bring together the anatomy, major lesion and any related lesions in a concise fashion with the use of all relative descriptive adjectives (e.g. chronic osteomyelitis with multiple areas of acute inflammation and bacteria).

Team-based learning

At the commencement of this course you will be divided into four tutorial groups and each tutorial group will be subdivided into four teams, each consisting of six students. Each team will have a mixture of abilities and backgrounds. The aim of this teaching approach is to enhance your learning experience through the use of individual and team quizzes and peer-teaching and discussions.

The role of the tutor is not to give you another lecture; but to facilitate your interactive discussions and assist you clarify some challenging concepts presented in your lectures, practical classes and/or text books. You are therefore strongly urged to make adequate preparation for these tutorials and encouraged to participate. Attendance at all these tutorials is mandatory and is assessable.

Pre-reading will be allocated prior to each tutorial. Each tutorial will commence with a quiz (based on the pre-reading), which will first be attempted individually and the answers submitted to your tutor. The same quiz questions will then be attempted in teams, with each team submitting their consensus answers. The tutor will guide you through the answers, encourage discussion and provide clarifications regarding of the challenging questions and concepts. Some of the tutorials will have additional tasks to be completed on a worksheet in your course manual. *Please bring your course manual to all the tutorials and practical classes.*

You will receive a maximum of **2%** towards your final course mark for each tutorial quiz, comprising **1%** for your individual performance and **1%** for your group's performance. Over the course of 5 tutorials, this will contribute up to **10%** of your final mark.

The names in each tutorial group and team will be posted on Moodle at <https://moodle.telt.unsw.edu.au/>. The same teams will work together to develop presentations for the Evidence-Based Symposium.

Evidence based symposium

The evidence based symposium is a collection of group presentations on cutting-edge topics in musculoskeletal diseases. These presentations are aimed to enhance students' skills in team work, effective communication and peer-review processes in line with learning outcomes 5, 6 and 7 described in the Course Outline.

The selection of topics will take place in week 3, **Tuesday 14th August 2013**. On this day teams will be allocated a random topic by a lottery from a pool of relevant topics.

Students will submit a 400 word Abstract by e-mail to n.tedla@unsw.edu.au in week 6, no later than 5 pm on **13th September 2013**. This abstract will outline each team's forthcoming presentation in week 8. *Please follow the strict Abstract format outlined below.* Late submission and/or inappropriately formatted abstracts will not be accepted.

In week 8, each team of students will give a 10-minute (maximum) group presentation followed by an additional 5 minutes for question time as part of a symposium. Several one hour sessions will be set aside for students to present their work to the rest of the group. Presentation style is at the discretion of each group (examples include PowerPoint presentations, Video, You Tube, role play, Interview, etc.). Groups can choose their spokesperson beforehand, although all students are expected to contribute equally and the performances of each individual may affect the group's overall score. The presentation will need to be supported by a thorough literature review. At the end of the presentation, questions can be asked to any member of the group by students and members of academic staff.

15% of the final mark for the course is allocated for this task, of which **2.5%** will be determined by members of the group, who will provide their collective score for each group member at the end of their presentation. **2.5%** will be determined by peers in the audience and **10%** will be allocated by academic staff (see assessment criteria on the following pages). Attendance at all of the presentations is mandatory. Students will lose 1 mark for each day they do not attend and will lose an additional 2 marks if they do not attend their own group presentation.

The timetable for the Evidence Based Symposium will be posted on Moodle at <https://moodle.telt.unsw.edu.au/>

Format for Evidence Based Symposium Written Abstract

Time New Roman, font 12, justified

Title and headings in Bold → **Joints Replacement - The Advances and Pitfalls of Current Research Aimed at Improving Duration:** Smith J, Kane SL, Lim K, Kwok J and Krishnan G.

Address in Italics → *School of Biomedical Engineering, University of NSW, 2052 Australia*

Objective: The average life span for a typical joint replacement is between 10 to 12 years. The objective of this presentation is to investigate current advances and pitfalls in surgical techniques and materials used aimed at improving durability of joint replacements

Methods: Research for the presentation began by seeking council with Professor William Walsh who provided us with first hand information as well as resources, including textbooks and joint prosthetics. The other information was obtained through the UNSW Sirius application. Search engines such as Science Direct, Compendex, MEDLINE and Pub Med provided abstracts on journal articles relative to our presentation question. We selected studies published from 1966-2009 and refined our search scope using the key words joint replacement, joint arthroplasty and total hip replacement. Statistics were also obtained from the Australian Orthopaedic Association National Joint Replacement Registry.

Presentation Style: The presentation method incorporated the use of PowerPoint while utilising three different speakers. The first speaker represented the patient, who discussed the need for increased duration of replacements and outlined relevant statistical information on the subject. The second speaker is representative of the surgeon/specialist, who explained the importance of good surgical technique in prolonging duration, while demonstrating that every advance in materials or treatment appears to bring with it several disadvantages. The third speaker is the researcher who outlines the importance of material research in joint arthroplasty. The use of a PowerPoint presentation allows us to explore several examples of current research in more detail than other forms of media. It was also selected because of its reliability, ease of use and familiarity amongst group members. As part of our presentation, several replacement hip prosthetics were distributed to the audience. By having a tangible example of a replacement accessible we believed that a greater connection and understanding of the subject would be attained.

Results: Although joint replacement surgery has advanced significantly there are still major improvements and advancements needed if researchers expect to extend the duration of joint replacements. It appears that with any new breakthrough in material, fixation or treatment there are several pitfalls and disturbances that challenge surgeons and researchers assessments of what is and what is not appropriate for implantation.

Conclusions: There are still significant challenges and pitfalls in obtaining joint longevity primarily related to lack of suitable materials that have the desired strength, flexibility and biological properties.

400 words

Due on 14/09/12 before 5:00PM

Submitted by e-mail to n.tedla@unsw.edu.au

Margins 2.2 cm all around

Marking scheme for peer assessment

Presenting group:.....

Topic:

Student Assessor: Name..... Group No.

	0	0.5
Originality of presentation		
Clear explanation of the most important aspects of topic		
Evidence of inclusion of recent medical literature		
Evidence of critical evaluation of the literature		
Answering relevant questions		
Total		

Comments:

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Signature:

Date:

Marking scheme for assessment by academic staff

Group number:

Assessor's name:

	0	0.5	1.0
Demonstrate an understanding of the topic and how it fits into the point of discussion			
Demonstrate effective communication of the most important aspect of the topic			
Ability to effectively discuss questions on the topic			
Demonstrate an ability to utilise the current medical literature to support argument			
Clear and justified conclusions			

Comments:

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Signature:

Date:

Revision Quiz On-line

This quiz consist 20 questions focusing on learning outcomes 1, 2, 3 and 4 described in the Course Outline. These on-line questions encourage independent and reflective learning. This occurs in a non-threatening environment, without fear of embarrassment for making errors. The aim of this assessment is to provide students with prompt feedback on their progress that would allow them self-adapt their preparation for the exams rather than to rank students. The online quiz is aligned to the end of the course and will be available from the 16th of October to 11th of November 2013.

Assessments

Students will undertake multiple forms of assessment during the session

- 1) **Evidence based symposium** is a group presentation that comprises **15%** of the final mark. Of the **15%** total mark, **2.5%** will be determined by members of the group, **2.5%** by peer assessment and **10%** will be allocated by academic staff on the basis of content, presentation, use of relevant literature and ability to answer questions on the topic.
- 2) **Tutorial quizzes** are **weekly** individual and group assessments in a form of multiple choice questions and will comprise **10%** of the final mark (**1%** for each 5 individual quizzes and **1%** for each 5 group quizzes). Each tutorial will commence with a quiz which will first be attempted individually and the answers submitted to your tutor. The same quiz questions will then be attempted in teams, with each team submitting their consensus answers. The tutor will guide you through the answers, encourage discussion and provide clarifications regarding of the challenging questions and concepts. Some of the tutorials will have additional tasks to be completed on a worksheet in your course manual. *Please bring your course manual to all the tutorials and practical classes.* Each quiz is primarily based on the two lectures given during same week and a pre-reading that will be allocated prior to each tutorial. You are therefore strongly advised to attend and review the lectures and perform the allocated pre-reading before you come to the tutorial. The recommended pre-readings are only a guide, additional reading on the subject from the prescribed textbooks is highly recommended.
- 3) **Mid-session written exam.** Students will complete a 45 minute written mid-session exam on week 6, Friday the 6th of September 2013 at 12:00. The exam will contain 5 multiple choice questions and 3 short answer questions that may include interpretation of diagnostic image(s), describing pathophysiological processes, describing and diagnosis of macroscopic specimen and/or writing histopathological reports. This will constitute **10%** of the final mark of the course. A general feedback to each tutorial group will be provided on week 7, Friday the 13th of September 2013, 12-2. Students who performed poorly in this exam may receive individual feedback either face-to-face or electronically. The aim of this assessment is to provide timely feedback on your progress and provide you with remedial assistance if needed.
- 4) **A practical examination in week 12.** Students will complete a practical exam during the final week of term (scheduled in normal teaching time) constituting **20%** of the final mark for the course. **This will** consist of a series of 10 stations, each with questions based on material presented during term focused on learning outcomes 2, 3, 4 and 5 described in the Course Outline. Students will rotate around the stations, spending 3 minutes per station.
- 5) **End of course written examination.** At the end of the session there will be written exam that accounts for **45%** of the final mark. The questions assess all the learning outcomes and encourage an in-depth understanding of the pathology of musculoskeletal diseases in a clinical and research context. Marks will be weighted as follows: Short answer questions 25% and Objective items 20%. The short answer questions vary in style, but are intended to provide you with the opportunity to demonstrate your understanding of the topic and your ability to integrate ideas rather than simple “regurgitation of facts”. The objective items consist of 20 multiple choice questions where the best or most appropriate answer is chosen from among alternatives.

Sample Examination Paper

SAMPLE END OF COURSE EXAMINATION FORMAT FOR 2013

- (1) TIME ALLOWED: 1.5 HOURS.
- (2) ANSWER ALL QUESTIONS.
- (3) ANSWER **PART A** QUESTIONS 1 AND 2 IN SEPARATE BOOKS. WRITE LEGIBLY IN INK.
- (5) ANSWER **PART B** USING THE GENERALISED ANSWER SHEET PROVIDED.
- (6) THIS PAPER MAY NOT BE RETAINED BY THE CANDIDATE.

PART A (25 Marks)

1. Explain to a healthy 20-year-old female how she might be able to prevent herself from developing osteoporosis later in life.
(10 marks)
2. A 22-year-old man was brought by ambulance to the Emergency Department. One hour previously, he had been driving a car and was involved in a high-speed head-on collision. He had not been wearing a seat belt. Immediately after the accident, he briefly lost consciousness and recovered soon after. On arrival to the hospital he was disorientated and was gradually losing consciousness. Initial examination revealed multiple abrasions to the head, fracture on the left side of the skull and some bleeding from the left ear. What injuries might this patient have sustained? Explain how these might have developed.
(10 marks)

PART B (20 marks)

This part of the examination consists of 20 questions, each containing 5 statements. For each question, select the **BEST or MOST APPROPRIATE** answer (i.e that which is most relevant for the disease and/or its consequences) from among the alternatives, several or all of which may be true. On the supplied generalised answer sheet, **FILL IN** the corresponding circle. **USE PENCIL.**

1. Antibody tests are useful in the diagnosis of:
 - (A) Parkinson disease
 - (B) Multiple sclerosis
 - (C) Segmental demyelination
 - (D) Myasthenia gravis
 - (E) Motor neuron disease
2. Osteosarcomas:
 - (A) May arise in bones affected by Paget's disease
 - (B) Usually metastasise to local lymph nodes
 - (C) May show areas of cartilage formation
 - (D) Commonly arise in the metaphyses of long bones

- (E) Are associated with exposure to ionising radiation
3. Intervertebral disc herniation:
- (A) Characteristically occurs at L3/L4
 - (B) Is commonly associated with facet joint degeneration
 - (C) Typically leads to spondylolisthesis
 - (D) Usually results in anterior protrusion of the nucleus pulposus
 - (E) Affects athletes more frequently than the elderly
4. Duchene muscular dystrophy:
- (A) Dystrophin is present in large quantities
 - (B) Clinical expression occurs in adolescence and progression inevitable
 - (C) It is the most common of the X-linked muscular dystrophies
 - (D) Is commonly associated abnormal muscle and nerve fibres
 - (E) Pulmonary infection is a rare complication
5. Rheumatoid Arthritis:
- (A) Is associated with periarticular osteoporosis and juxta-articular erosions
 - (B) Is characterised by a florid polymorphonuclear cell infiltrate within hyperplastic vascular synovia
 - (C) Yields chronic inflammatory cells on aspiration of synovial fluid
 - (D) Is associated with elevated serum rheumatoid factor in approximately 95% of cases
 - (E) Typically presents as a chronic, asymmetrical, joint arthropathy

Answers: 1D, 2D, 3E, 4B, 5A

Resources for Students

You are expected to use the following text available online via the UNSW library Sirius website at <http://sirius.library.unsw.edu.au> (zID and zPass required). Search for the database MD Consult, then search for Robbins Basic Pathology. *Robbins Basic Pathology*. 9th edition. V. Kumar, A.K. Abbas, & J.C. Aster (2012). Saunders & Co. Philadelphia PA; Elsevier Saunders.

Students wishing to study the molecular biology, clinical features of diseases and diagnosis in greater depth might consider the purchase of the following texts:

1. *ROBBINS AND COTRAN, Pathologic Basis of Disease* 8th edition. V. Kumar, A.K. Abbas & N. Fausto (2010) Elsevier Saunders. (highly recommended)
2. *ORTHOPAEDIC, Examination, Evaluation and Intervention*. Mark Dutton (2004). McGraw Hill.
3. *DIAGNOSTIC MUSCULOSKELETAL IMAGING*. Theodore T Miller & Mark E. Schweitzer (2005). McGraw Hill.
4. *MUSCULOSKELETAL EXAMINATION*. Jeffrey Gross, Joseph Fetto & Elaine Rosen 3rd Ed (2009). Wiley Blackwell.
5. *Histology and Cell Biology. An Introduction to Pathology*. Abraham L. Kierszenbaum. Mosby (2002).

Additional Learning Resources

In addition, there are many resources available on the web, which vary from simple patient information brochures to on-line pathology courses to information on the latest research. Some general sites you may find useful are:

Medline Plus ('health topics' index of disease with information)

<http://www.nlm.nih.gov/medlineplus/healthtopics.html>

University of Iowa (on-line histological slides on many of the topics covered)

http://www.medicine.uiowa.edu/pathology/nlm_histology/or

http://www.medicine.uiowa.edu/pathology/uarep_histopathology/

American Arthritis Foundation (Patient information and latest research on arthritis) <http://www.arthritis.org>

National Institute of Arthritis and Musculoskeletal and Skin Diseases

<http://www.niams.nih.gov/>

Neuromuscular Disease Centre, Washington University, St Louis, MO USA

<http://www.neuro.wustl.edu/neuromuscular/>

Muscle Physiology, University of California, San Diego

<http://muscle.ucsd.edu>

PATH 3207 Moodle course

The online module for the Musculoskeletal Disease course can be found by logging in to Moodle at <https://moodle.telt.unsw.edu.au/>, using your student number as the user name (e.g. z1234567) and your zPass as the password. The PATH3207 Moodle module will contain information directly related to the course such as tutorial lists, revisions to the lecture timetable, examination timetables, links to lecture slides and iLecture recordings etc.

You are expected to visit this site regularly during your course.

On-line lectures

PDF of most lectures will be up-loaded to Moodle together with corresponding recorded lectures (iLectures).

However, large numbers of lecture slides in this course are images that are not annotated but explained/discussed in during the lecture. Therefore, you are strongly advised to attend lectures in person. Note that no online recording will be available for lectures that are of sensitive nature and those that are intellectually protected.

PATH3207 Virtual slide box and images

Students will be able to access microscopic slides to all practical classes at: <http://vslides.unsw.edu.au/>

Images of Disease (IOD) database

This database is a collection of images used for teaching within the Department. The latest version is available online, optimised for smart phones and tablet computers as well as Firefox4+, Chrome 13+ and Safari browsers on laptop and desktop computers at <http://iod.med.unsw.edu.au/>. The IOD database contains over 3000 images relevant to your study as an undergraduate. Many of these images represent specimens from the Museum of Human Disease, histopathological images from the student histopathological slide set as well as some diagnostic images such as X-rays.

Many images used in this program are of a sensitive nature, and are intended for the purpose of private study by pathology students and graduates. You should exercise appropriate standard of professional ethics when using them.

Interactive images of disease

This is a collection of “hotspotted” images from the Department of Pathology’s database on the Museum of Human Disease web page. Images contain clickable “hotspots” allow identification of the normal features and pathological changes within each specimen. At present this is limited selection, intended for the education of senior high school students and interested members of the public. However, these might be useful tools for you to practice your skills in interpreting macroscopic specimens.

The Museum of Human Disease

The Donald Wilhelm Museum of Human Disease is located on the ground floor of the Samuels Building (Building F25). Originally located on the 5th floor of the Wallace Wurth Building, it was established by Professor Donald Wilhelm, the Foundation Professor of Pathology at this university. Thanks to his foresight, and to the tireless efforts of Dr G. Higgins (the Museum Curator until 2004), the Museum has been meticulously maintained and updated over the years to reflect the changing patterns of disease in our society. The Museum contains over 2,700 specimens (or “pots”), which display diseased human tissue at the macroscopic level, usually preserved in formalin. Specimens are obtained both from organs removed surgically and from tissue obtained at autopsy, where the natural history of disease is in full view. **Please take note that some specimens of diseases which have become rare, e.g. diphtheria, are over 60 years old, and are irreplaceable.** Each specimen is numbered and is accompanied by a clinical history (when known), a macroscopic description of the abnormalities displayed, and a histopathological description of changes at the microscopic level (where relevant). That information, specific to each of thirty areas (or “bays”), can be found in the Museum catalogues located in a bracket within each bay.

All the specimens in the museum are arranged in one or other of two major groups. One group comprises collections of specimens according to pathological processes such as congenital, inflammation and healing, vascular, neoplasia etc. The second group comprises collections of specimens under organ systems, such as cardiovascular, central nervous, renal etc. As responsible adults, we expect you to maintain decorum in the museum behave with care and respect for the integrity of the specimens, and help to keep the Museum tidy at all times. This means no eating or drinking in the Museum, and always returning specimens and catalogues to their allocated places. **Do not shake the pots!** This activity conveys no useful information, but often damages the specimens. If you discover that a specimen is leaking or broken, follow the instructions listed in the safety notice below. **Remember that the Museum is a precious learning resource, of which you are encouraged to make full use.**

The Museum of Human Disease page contains links to some excellent undergraduate and postgraduate educational resources that might be useful for you. The address is <http://web.med.unsw.edu.au/pathology/pathmus/>

Security in the Museum

It is a crime under the Human Tissue Act to steal or mistreat material preserved in the Museum or practical class laboratories. Anyone who contravenes the Act will be prosecuted.

In order to protect the collection of specimens, access to the Museum is restricted for students during weekdays from 9 a.m. to 5 p.m. The Museum is security locked, and can only be entered by using your student card to enable the doors to be opened. Mr Derek Williamson, Ms Bridget Murphy, Ms Julia Kiss and Mr Cutting play a supervisory role during office hours.

The Museum and practical class laboratories are under constant electronic surveillance.

Safety in the Museum

- Always handle museum specimens with care and respect. All specimens consist of generously donated human tissue.

- The specimens are preserved in Perspex and contain a range of preserving chemicals that may be harmful. Chemicals used may include formalin, pyridine and sodium dithionate. A full list of chemicals and associated information is available at the Health and Safety (H&S) station in the Museum and on the SoMS website.

Chemical	Max. Percentage Composition
Glycerol	17 (v/v)
Pyridine	0.8 (v/v)
Sodium Acetate	7 (w/v)
Formalin	<2 (v/v)
Sodium Dithionate	0.4 (w/v)

- For reasons of hygiene, never take food or drink into the museum.
- Never leave a museum specimen on the floor, or in any precarious position.
- If a specimen is leaking, turn it upside down to prevent further leakage, then immediately inform Mr David Cutting or a member of academic staff.
- If a specimen is broken, do not attempt to wipe up the spillage. Use the kitty litter provided in the central cupboards to absorb the fumes, then clear the area and immediately inform Mr Vincent Strack Van Schijndel or a member of academic staff.
- Remember that the museum is here for your benefit - your cooperation in maintaining neatness and safety at all times is appreciated.
- For more information on matters related to occupational and health safety policies of the UNSW visit the following web site: http://www.ohs.unsw.edu.au/ohs_policies/

Administrative Matters

You may also meet the following members of the School support staff during the course of the year:

Ms Soo Han Chup

Position: Administrative Officer, Department of Pathology

Location: Administrative Wing, 5th Floor New Wallace Wurth Building

Phone: 9385 2528

Ms Chup is responsible for the distribution of Pathology manuals to students, and will assist in arranging interviews with academic staff within the Department.

Ms Carmen Robinson

Position: Student Advisor

Location: Room G27, Biosciences Building

Ms Robinson is responsible for assistance with general enquiries, enrolment procedures and the collection of assignments.

Phone: 9385 2464

E-mail: Carmen.Robinson@unsw.edu.au

Fergus Grieve

Position: Administration officer, SOMS Web Manager & TELT Administrator

Location: Administrative Wing, 5th Floor New Wallace Wurth Building

Please contact Mr Grieve if you have any inquiries related to PATH3207 materials up-loaded to this site including lectures, assignments, timetables and communications. Mr Grieve also maintains materials up-loaded to Moodle.

Phone: 9385 8288

Fax: 9385 2866

E-mail: f.grieve@unsw.edu.au

Mr Derek Williamson

Position: Museum Manager

Location: Room G04 Ground Floor Samuels Building, Building F25

Mr Williamson provides support for all undergraduate teaching programs. He plays a major role in broadening the use of the Museum of Human Disease by introducing an integrated learning program for senior high school students and community interest groups. Mr Williamson Co-ordinates a network of volunteers, who assist with the supervision of visitors from outside the University.

Phone: 9385 2190

E-mail: derek.williamson@unsw.edu.au

Mr David Cutting

Position: Museum Technical Officer/Laboratory Manager

Location: Room G06 Ground Floor Samuels Building, Building F25

Mr Cutting is responsible for the mounting and maintenance of Pathology Museum specimens, both on campus and in the associated teaching hospitals. Contact Mr Cutting immediately if there are any broken or leaking specimens in the Museum.

Phone: 9385 1722

E-mail: davecutting@unsw.edu.au

Ms Bridget Murphy and Ms Julia Kiss

Position: Museum Education Officer

Location: Room G06 Ground Floor Samuels Building, Building F25

Ms Murphy assists Mr Williamson in delivering Museum learning programs and coordinating volunteers.

Phone: 9385 1522; E-mail: b.murphy@unsw.edu.au

Academic Honesty and Plagiarism

What is Plagiarism?

Plagiarism is the presentation of the thoughts or work of another as one's own.* Examples include:

- direct duplication of the thoughts or work of another, including by copying material, ideas or concepts from a book, article, report or other written document (whether published or unpublished), composition, artwork, design, drawing, circuitry, computer program or software, web site, Internet, other electronic resource, or another person's assignment without appropriate acknowledgement;
- paraphrasing another person's work with very minor changes keeping the meaning, form and/or progression of ideas of the original;
- piecing together sections of the work of others into a new whole;
- presenting an assessment item as independent work when it has been produced in whole or part in collusion with other people, for example, another student or a tutor; and
- claiming credit for a proportion a work contributed to a group assessment item that is greater than that actually contributed.†

For the purposes of this policy, submitting an assessment item that has already been submitted for academic credit elsewhere may be considered plagiarism.

Knowingly permitting your work to be copied by another student may also be considered to be plagiarism.

Note that an assessment item produced in oral, not written, form, or involving live presentation, may similarly contain plagiarised material.

The inclusion of the thoughts or work of another with attribution appropriate to the academic discipline does *not* amount to plagiarism.

The Learning Centre website is main repository for resources for staff and students on plagiarism and academic honesty. These resources can be located via: www.lc.unsw.edu.au/plagiarism

The Learning Centre also provides substantial educational written materials, workshops, and tutorials to aid students, for example, in:

- correct referencing practices;
- paraphrasing, summarising, essay writing, and time management;
- appropriate use of, and attribution for, a range of materials including text, images, formulae and concepts.

Individual assistance is available on request from The Learning Centre.

Students are also reminded that careful time management is an important part of study and one of the identified causes of plagiarism is poor time management. Students should allow sufficient time for research, drafting, and the proper referencing of sources in preparing all assessment items.

* Based on that proposed to the University of Newcastle by the St James Ethics Centre. Used with kind permission from the University of Newcastle

† Adapted with kind permission from the University of Melbourne.

The School of Medical Sciences will not tolerate plagiarism in submitted written work. The University regards this as academic misconduct <https://my.unsw.edu.au/student/academiclife/assessment/AcademicMisconduct.html> and imposes severe penalties. Evidence of plagiarism in submitted assignments, etc. will be thoroughly investigated and may be penalised by the award of a score of zero for the assessable work. Flagrant plagiarism will be directly referred to the Division of the Registrar for disciplinary action under UNSW rules.

The attention of students is drawn to the following extract from the above website:

"The basic principles are that you should not attempt to pass off the work of another person as your own, and it should be possible for a reader to check the information and ideas that you have used by going to the original source material. Acknowledgment should be sufficiently accurate to enable the source to be located speedily."

"The following are some examples of breaches of these principles:

a) Quotation without the use of quotation marks. It is a serious breach of these rules to quote another's work without using quotation marks, even if one then refers to the quoted source. The fact that it is quoted must be acknowledged in your work.

- b) Significant paraphrasing, e.g., several sentences, or one very important sentence, which in wording are very similar to the source. This applies even if the source is mentioned, unless there is also due acknowledgment of the fact that the source has been paraphrased.
- c) Unacknowledged use of information or ideas, unless such information or ideas are commonplace.
- d) Citing sources (e.g., texts) which you have not read, without acknowledging the 'secondary' source from which knowledge of them has been obtained."

Appropriate citation of sources therefore includes surrounding any directly quoted text with quotation marks, with block indentation for larger segments of directly-quoted text. The preferred format for citation of references is an author-date format with an alphabetically arranged bibliography at the end of the assignment. Note that merely citing textbooks or website URLs is unlikely to yield a bibliography of satisfactory standard. ***The internet should be avoided as a primary source of information.*** Inclusion of appropriate journal articles, both primary research publications and reviews, is usually expected.

Teaching Laboratories Risk Assessments

Medicine Teaching Laboratory

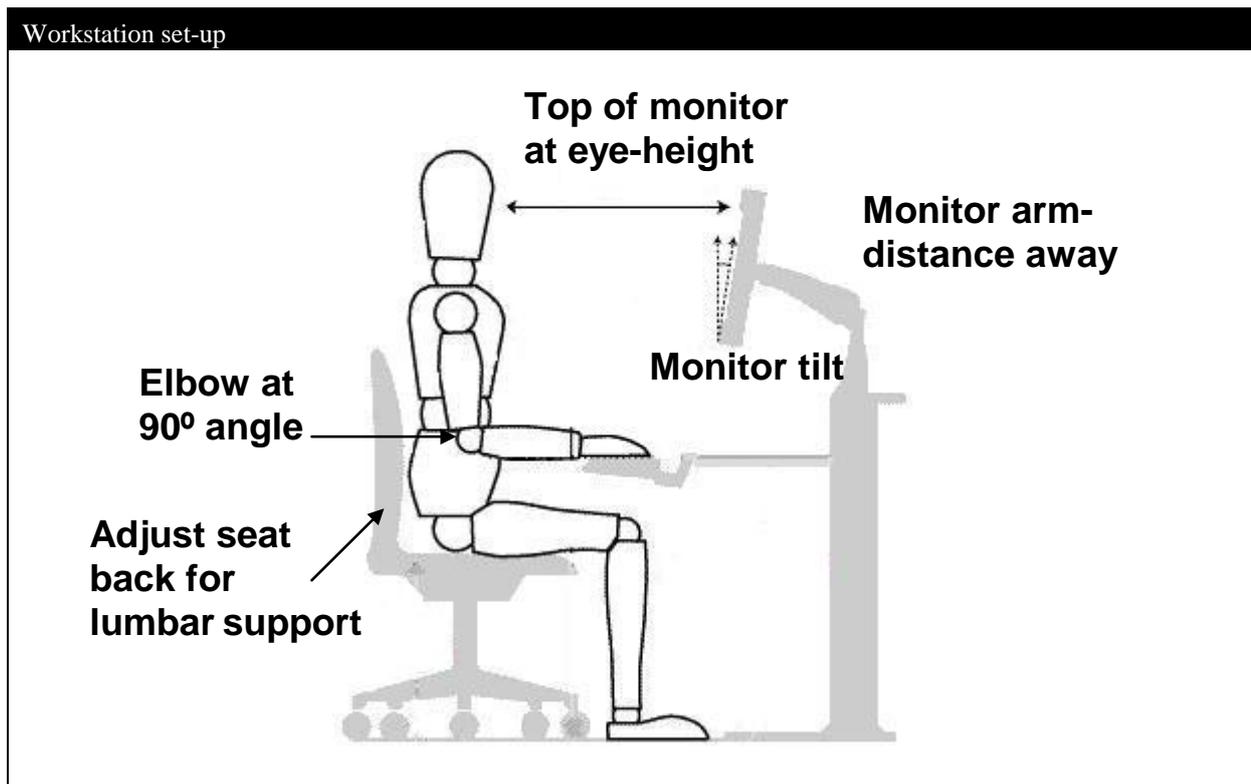
Student Risk Assessment



UNSW
THE UNIVERSITY OF NEW SOUTH WALES

Pathology practicals in G6/G7 & G08 & G16/G17 at Wallace Wurth for PATH 3207, 2013

Hazards	Risks	Controls
Ergonomics	Musculoskeletal pain	Correct workstation set-up.
Electrical	Electrical shock/fire	Check electrical equipment in good condition before use. All portable electrical equipment tested and tagged.
Handling pots	Chemical spillage	Instructions on correct manual handling of pots



Personal Protective Equipment

All pots contain real human tissue that has been generously donated to medical science and **must be treated with appropriate respect and dignity.**
 Specimens are preserved in Perspex and contain a range of preserving chemicals that may be harmful. Chemicals used include **formalin, pyridine, sodium dithionate.** A full list of chemicals and associated MSDS information is available in the H&S Station and on the SoMS website.
MANUAL HANDLING OF POTS
 It is recommended that all students wash their hands thoroughly as they leave practical class Chemical residues may be present on pots.
Carry one pot at a time. Use two hands at ALL TIMES and support the base of pot.
Avoid rough handling and/or tilting of pots. This can cause leaking joints or tear tissue in specimen.
Limit the number of pots on a table at any one time.

SPILLS AND LEAKAGES

If a specimen is leaking or broken, do not attempt to wipe up the spillage. Clear the area and immediately inform the Museum Manager or a member of academic staff. A spill kit will then be used to absorb the fumes.

Emergency Procedures

In the event of an alarm, follow the instructions of the demonstrator. The initial sound is advising you to prepare for evacuation and during this time start packing up your things. The second sound gives instruction to leave. The Wallace Wurth assembly point is in the lawn in front of the Chancellery. In the event of an injury inform the demonstrator. First aiders and contact details are on display by the lifts. There is a first aid kit in the laboratory and the Wallace Wurth security office.

Clean up and waste disposal

Not necessary in these practicals.
 No open-toe shoes allowed

Declaration

I have read and understand the safety requirements for this practical class and I will observe these requirements.

Signature:.....Date:.....
 Student Number:.....

Reviewed on 04/07/2013

Science Teaching Laboratory

Student Risk Assessment



Pathology practicals in G6/G7& G08 & G16/G17 in Wallace Wurth for PATH3207, 2013

Hazards	Risks	Controls
Physical Sharp plastic	'Stabbing' wound of hand	<ul style="list-style-type: none"> Use disposable gloves
Biological Antibody	Inoculation/Irritant	<ul style="list-style-type: none"> Do not eat, drink or smoke in the teaching laboratory Use disposable gloves
Chemical Acrylamide Azide ...PBS	Corrosive/Flammable Irritant/neurotoxic Irritant Mild Irritant	<ul style="list-style-type: none"> Low concentrations of chemicals used Use disposable gloves

Pipetting ergonomics: to avoid aches and pain due to repetitive pipetting follow the following guides

- Adjust your chair or stool so that your elbow is at a 90° angle while pipetting.
- Adjust the height and position of sample holders, solution container, and waste receptacle so that they are all approximately the same.
- Try to work with your hands below shoulder height.
- Let go of the pipette from time to time and give the fingers/hand a break
- Do not twist or rotate your wrist while pipetting; Use minimal pressure while pipetting
- Try to switch periodically between different types of work.

For more information on preventing repetitive strain while pipetting click on <http://www.anachem.co.uk/rsi>

Personal Protective Equipment required			
<p>Closed in Footwear</p>	<p>Lab. Coat</p> <p>optional</p>	<p>Gloves</p>	<p>Safety Goggles</p> <p>optional</p>

Emergency Procedures

In the event of an alarm sounding, stop the practical class and wait for confirmation to evacuate from demonstrators. Then wash your hands and pack up your bags.
Follow the instructions of the demonstrators regarding exits and assembly points.

Clean up and waste disposal

- Remove your gloves and dispose in the biowaste bins provided.
- Dispose of all pipette tips in the bin provided.

Ethics Approval

This type of practical does not require ethics approval.

Declaration

I have read and understand the safety requirements for this practical class and I will observe these requirements.

Signature:.....Date:.....

Reviewed on 04/07/2013

Tutorials and Practical classes

Tutorial 1 –Anatomy of Bone, Cartilage and Joints

Aim:

This Tutorial aims to revise the basic anatomy of bone and joints.

Learning objectives:

At the completion of this tutorial you should be able to:

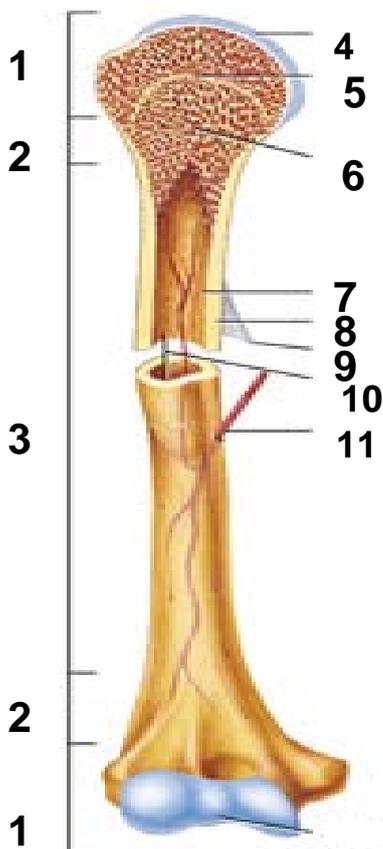
1. Outline the structure of long bones.
2. Explain the functions of the bone cells, including the osteoprogenitor cells, osteoblasts, osteocytes and osteoclasts.
3. Describe the constituents of bone, broadly represented by organic matrix and inorganic elements.
4. Discuss the structure of hyaline cartilage.
5. Outline the structure and function of synovial joints.

Task 1

You will complete an individual and group quiz, based on this week's lectures and allocated pre-reading from *Histology and Cell Biology. An Introduction to Pathology*. Abraham L. Kierszenbaum. (2002): Page 114-123; page 143. This will be followed by discussion of the answers with your tutor (20 min).

Task 2

Working with your small groups, describe the general architecture of a long bone on the following schematic picture of the humerus and discuss your answers with your tutor (10 min).



Task 3

Working with your small groups, match cells that are found in the bone to the characteristics and/or function listed below and discuss your answers with your tutor (10 min).

Cell types

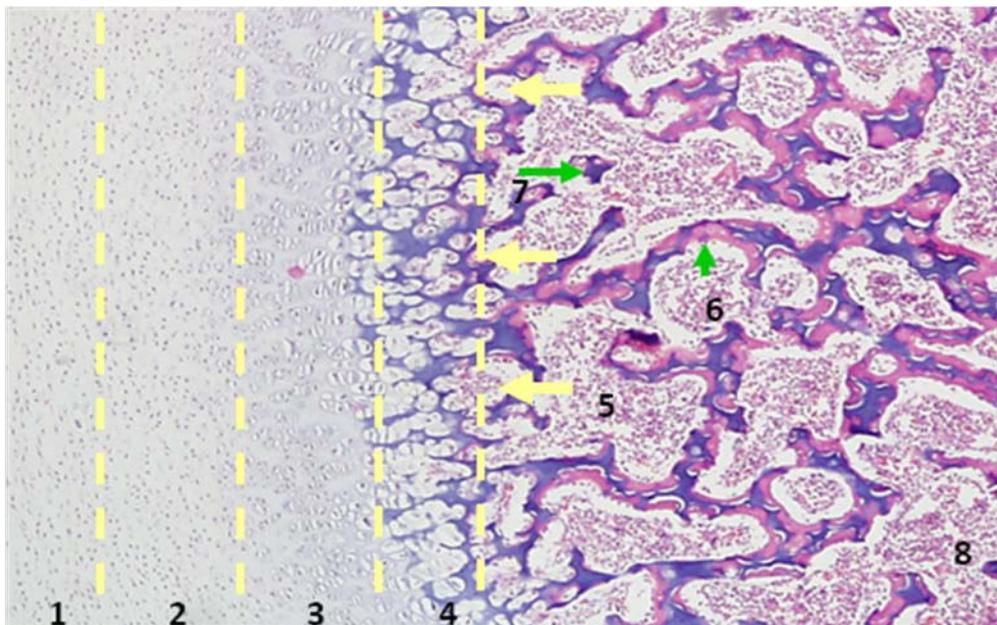
- A. Osteoprogenitor cells
- B. Osteoblasts
- C. Osteoclasts
- D. Osteocytes
- E. Chondroblasts
- F. Chondrocytes

Functions/characteristics

- 1. Bone forming cells located at edges of bones
- 2. Secrete organic matrix including collagenous fibers, establish conditions favorable for calcification
- 3. Mature bone cells in lacunae surrounded by calcified matrix
- 4. Unspecialized mesenchymal cells
- 5. Do not form matrix but do recycle Ca^{+2} salts in matrix; can revert to osteoblasts at breaks in the bone
- 6. Large motile multinucleated cells at bone edges that break down bone
- 7. Convert to osteocytes when surrounded by calcified matrix
- 8. Secrete acids and enzymes that dissolve matrix
- 9. Derived from monocytes
- 10. Produce osteoblasts
- 11. Active surface facing the Howship's lacuna with a ruffled border

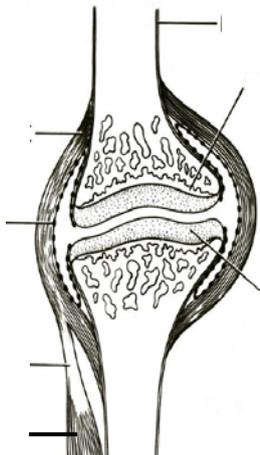
Task 4

Name the different zones of hyaline cartilage on a developing bone labelled 1-5 and the components indicated as 6, 7 and 8 and discuss your answers with your tutor (10 min).



Task 5

The following figure is a schematic representation of synovial joint. Working in your small groups, label the different parts of the joint and discuss your answers with your tutor (10 min).



Task 6

Quizzes for next week's tutorial on Fractures and Osteomyelitis will be based on the lectures from the 07/08/2013 and 08/08/2013 and pre-reading of *Pathologic Basis of Disease*, 8th Ed. V. Kumar, R. Cotran & S Robbins (2010): Pages 1219- 1221 or *Robbins Basic Pathology*, 9th Ed (2012) Pages 772-774. Students are expected to complete these tasks prior to their quizzes.

Practical 1– Histology Bone, Cartilage and Joints

Aim:

This practical class aims to revise the basic histology bone, hyaline cartilage and synovial joints. This will lay a foundation to facilitate better understanding of musculoskeletal pathology.

Learning objectives:

At the completion of this practical class you should be able to:

1. Identify histologically normal bone cells, including, osteoblasts, osteocytes and osteoclasts.
2. Discuss the difference between compact and spongy bone; the difference between woven and lamellar bone, and the significance of the presence of woven bone in the adult.
3. Understand the important relationship between osteoblasts and osteoclasts as the basic multicellular unit involved in bone remodelling.
4. Identify the histological features and constituents of hyaline cartilage
5. Discuss the role of hyaline cartilage and, more specifically, its chondrocytes.
6. Explain the process of endochondral and intramembranous ossification.
7. Describe the structure and importance of synovium, its constituent cells and synovial fluid.

Task 1

Examine virtual slides 1 and 2 at <http://vslides.unsw.edu.au/> that show a compact bone. Identify the different structures and cells in these sections. Use Image 3 in the attached document at <http://vslides.unsw.edu.au/> as a guide.

Task 2

Name the two types of lamellar bone and describe their differences. How does lamellar bone differ from a woven bone? Use Image 4 in the attached document at <http://vslides.unsw.edu.au/> as a hint.

Task 3

Examine virtual slide 3, which is a section of hyaline cartilage. Identify and describe the cartilage forming cells in this slide. What are the functions of hyaline cartilage? Name three anatomical locations where you can find hyaline cartilage.

Task 4

Examine Virtual slide 4 that shows a section of developing bone and write a brief description. Refer to Images 5 and 6 in the attached document at <http://vslides.unsw.edu.au/> as guide. What is this type of osteogenesis called?

Homework Tasks

1. Examine Virtual slide 5 at <http://vslides.unsw.edu.au/> and try to identify the different components of the synovial joint. What is the origin of synovial lining cells and what are their functions?
2. Label the different parts of the Lumbar vertebrae shown on Image 8 of the attached document at <http://vslides.unsw.edu.au/>. Use your lecture notes as a reference.
3. What is the clinical significance of: A. excessive osteoblast activity; B. lack of osteoblast activity; C. defect in the mineralisation of osteoid; D. increased osteoclast activity; E. Absence of osteoclast activity?

Tutorial 2 – Fracture Healing and Complications

Aim:

This tutorial aims to provide insight into the mechanism of fracture healing, and the pathogenesis of associated local and systemic complications.

Learning objectives:

At the completion of this Tutorial you should be able to:

1. Classify fractures as; complete or incomplete, closed (simple) or compound, comminuted, displaced or not-displaced.
2. Explain the difference between “pathological” and “traumatic” fractures.
3. Describe the sequence of events in fracture healing, along a time-line, beginning with development of haematoma and soft tissue callus and concluding with bony callus formation and maturation.
4. Discuss complications of fractures and consequences of abnormal healing including delayed union, mal-union, non-union and pseudoarthrosis.
5. Outline the local and systemic complications (both immediate and delayed) of a compound fracture of the femur.

Task 1

You will complete an individual and group quiz, based on the lectures and allocated pre-reading. This will be followed by discussion of the answers with your tutor (20 min).

Task 2

Working with your small groups, name 3 common conditions that may cause pathological fracture in 70 year old male and 3 conditions that may cause pathological fracture in a 70 year old female. Discuss your answers with your tutor (10 min).

Task 3

Working with your small groups, describe the stages of fracture healing and their approximate time line. Discuss your answers with your tutor (10 min).

Task 4

Based on your understanding of compound fracture of the femoral neck, name 3 early and 3 late complications of this fracture (10 min). Discuss your answers with your tutor (10 min).

Task 5

Identify some key local and systemic factors that may impede fracture healing and relate these to the possible undesired long term outcomes. Discuss your answers with your tutor (10 min).

Note: No quiz next week. All groups will attend a combined Practical and Tutorial class on Back pain from 12-2 pm at G6/G7.

Practical 2 – Histopathology of Fractures

Aim:

This practical class aims to provide insight into the clinical and histopathological features of different types of fractures and their associated local and systemic complications as well as the histopathology of osteomyelitis.

Learning objectives:

At the completion of this practical class you should be able to:

1. Distinguish different types of fractures and their clinical significances.
2. Explain the difference between “pathological” and “traumatic” fractures.
3. Describe the sequence of events in fracture healing, along a time-line, beginning with development of haematoma and soft tissue callus and concluding with bony callus formation and maturation.
4. Identify all cellular, matrix and inorganic components that contribute to the process of fracture healing
5. Determine the clinical, histopathological and radiological features of osteomyelitis.

CASE 1

A 16 year-old boy collapsed after being tackled during a game of football and was completely paralysed below the neck and could not breath unassisted. His cervical spine was immediately immobilised and he was intubated and ventilated. He died 2 hours after an X-ray was taken, and specimen 2419.16 was obtained from tissue removed at autopsy.

Task 1

Examine specimen 2419.16 and the attached X-rays at <http://vslides.unsw.edu.au/> and describe the type of fractures present. Which X-ray is consistent with this patient’s lesion? What was the major clinical problem that led to the death of this patient? Refer to the Images of Diseases site as a guide.

Virtual slide 1 is tissue removed at autopsy from a 35 year-old man who died 5 weeks after motor vehicle accident.

Task 2

Examine virtual slide 1 and state your diagnosis. Use Image labelled “Callus” at <http://vslides.unsw.edu.au/> as a guide.

Task 3

Based on your understanding of stages of fracture healing, is your finding consistent with a fracture that occurred 5 weeks ago?

CASE 2

A 36 year-old man developed serious complications after he sustained a fracture of the lower third of the left tibia and fibula that were protruding out through the skin after a football accident. A diagnosis of chronic osteomyelitis was made based on clinical and laboratory findings as well as X-ray Images on admission and six months after open reduction and immobilisation shown at <http://vslides.unsw.edu.au/>. Long-term antibiotic treatment and bone grafting were unsuccessful, and a below-knee amputation was performed. Specimen 486.6 and virtual slide 2 were prepared from tissue removed surgically.

Task 4

What type of fracture did the man sustain? Describe the X-ray appearances of the fracture on admission. What abnormalities are present in the X-ray and clinical Image taken six months after the fracture? Describe the pathological changes in specimen 486.6. Use the Images of Diseases site as a guide.

Task 5

Examine virtual slide 2 and list the features consistent with the man's history. What other investigations would you perform to confirm the diagnosis? What is the most likely causative agent in this patient?

CASE 3

Specimen 1329.15 was obtained at autopsy on a 47 year-old man who died from complications of bronchopneumonia 9 months after sustaining a fracture of the lower third of the femur due to a fall from a roof. Archival X-rays at the time of his accident and 8 months after treatment are shown (linked from <http://vslides.unsw.edu.au/>).

Task 6

Examine the X-ray on admission and determine the type of fracture in this man. What abnormalities are present in the X-ray 8 months after the fracture?

Task 7

Describe specimens 1329.15 and explain the sequence of events that resulted to this outcome. What are the long term consequences of such outcome?

Homework Tasks:

Specimen 514.6 and virtual slide 3 were obtained at autopsy on a 63 year old male who died due to complications related to this disease. The appearance of the lower legs and an X-ray at the time of his accident and 8 months after treatment are shown (linked from <http://vslides.unsw.edu.au/>).

Task 1

Examine the images of the patient and the X-ray and state your diagnosis? What type of fracture would this disease cause?

Task 2

Examine virtual slide 3 and write a histopathological report. What are the complications of this disease and which complications are the likely causes of death? Name 4 other causes of pathological fracture in adults?

Task 3

Visit the Museum of Human Disease and examine the following additional specimens: 250.6, 763.6, 743B.6, 1071.6 1223.6 and 743A.15. Use Images of Diseases site as a guide.

Practical/Tutorial 3 – Back pain

Aim:

This aim of this topic is to alert you to the wide differential diagnosis of back pain (an extremely common symptom). In particular, you should become familiar with the pathological processes that may induce back pain. You should also be able to briefly outline the investigations that can establish the diagnosis.

Learning objectives:

At the completion of this practical class you should be able to:

1. List the common sources of back pain, both somatic and visceral.
2. Distinguish between the classical clinical features of mechanical and inflammatory back pain.
3. Outline the pathogenesis of lumbar intervertebral disc degeneration and prolapse, and describe the clinical syndromes that may ensue.
4. Define the terms “spondylolisthesis” and “facet joint degeneration”, and explain how these disorders may cause back pain.

CASE 1

A 42 year-old man was admitted to hospital with severe back pain after a fall from a ladder. On examination he was able to move his extremities and his sensory and motor functions were intact. His lower back was bruised and was tender on palpation.

Task 1

Examine the X-ray at <http://vslides.unsw.edu.au/> and locate the likely source of his back pain. List other possible causes of back pain in this patient. What are the possible complications of this injury?

Task 2

Specimen 1318.16 was obtained from a patient who was admitted to the hospital with upper back pain after a motor vehicle accident and died two weeks later. Describe the abnormalities in the specimen and discuss the possible causes of death in this patient. Use Images of Diseases site as a guide.

CASE 2

A 35 year-old Caucasian woman visited her local GP with severe lower back pain shooting down her right buttock and stiffness that started after she lifted a heavy object 2 months ago.

An MRI of the lumbosacral spine, performed after the visit to her GP is displayed at: <http://vslides.unsw.edu.au/>

Task 3

Examine the image and state your diagnosis. Are the location of the lesion and the age of the patient common for this condition? How does this condition differ from spinal canal stenosis shown at: <http://vslides.unsw.edu.au/>?

Despite treatments with NSAIDs and physiotherapy over the next 6 months, her pain persisted and extended to her right leg reaching the little toe.

Task 4

How do you explain the progression of her pain? What are the complications of this condition if left untreated?

CASE 3

A 64 year-old man presented with loss of weight and a persistent cough with greyish sputum, which had become streaked with blood. A chest X-ray showed a mass occupying the upper 1/3 of the right side of the thorax. He developed progressive paraparesis 4 months before his death and an X-ray of the lumbosacral spine was performed.

Task 5

What is your diagnosis? What other investigations would confirm your diagnosis?

The X ray showed at <http://vslides.unsw.edu.au/> was taken at the time when he developed paraparesis.

Task 6

What abnormalities are evident on the X-ray? Are these abnormalities consistent with the patient's history?

Task 7

Examine Specimen 250.6 taken at autopsy and list the complications arising from these lesions. Use Images of Diseases site as a guide.

CASE 4

A 30 year-old man complained of recurrent episodes of back pain and joint stiffness for the past 2 years. On investigation, he had a raised erythrocyte sedimentation rate of 48 mm/h, a mild anaemia (Hb 106g/L) but no detectable serum rheumatoid factor. He responds to low dose of steroids and NSAIDs and gets into remission for few weeks. His X-ray displayed at <http://vslides.unsw.edu.au/> shows vertebral fusions at multiple levels.

Task 8

What is your diagnosis? What other clinical manifestations would you expect? What other investigations would help to confirm the diagnosis?

Task 9

What is known about the pathogenesis of this disease?

CASE 5

A 20 year-old woman presented with back pain and difficulty in mobilising following a motor vehicle accident 3 months previously. Her X rays, CT scan and MRI did not reveal any fracture, dislocation or prolapsed disc other than the accidental finding as shown at <http://vslides.unsw.edu.au/>

Task 10

What is this condition? What possible surgical emergency is this associated with?

Task 11

In the absence of any radiological abnormalities, what are the most common causes of back pain after a motor vehicle accident?

Task 12

Quizzes for next week's tutorial on Bone Tumors and Multiple Myeloma will be based on the lectures from the 21/08/2013 and 22/08/2013 and pre-reading of *Pathologic Basis of Disease*, 8th Ed. V. Kumar, R. Cotran & S Robbins (2010): Pages 609-611; pages 1225-1228; page 1232 and page 1235 or *Robbins Basic Pathology*, 9th Ed (2012): Pages 774-781 and 437-439. Students are expected to complete these tasks prior to their quizzes.

Tutorial 4 – Bone tumours

Aim:

The aim of this tutorial is to review the pathophysiology and briefly describe investigation of common neoplasms occurring in bone.

Learning objectives:

At the completion of this tutorial you should be able to:

1. List the common neoplasms occurring in bone in approximate order of frequency.
2. Compare the epidemiology, cell of origin, clinical behaviour and radiographic appearances of osteosarcoma, chondrosarcoma, Ewing sarcoma and giant cell tumour of bone.
3. Outline risk factors for the development of osteosarcoma.
4. Name primary neoplasms that are most likely to metastasise to bone and describe the common locations, and their clinical effects on the bone.
5. Understand the pathophysiology of multiple myeloma in terms of infiltration of bone, the presence of secreted paraprotein in serum and/or urine and processes leading to renal impairment and immunodeficiency.

Task 1

You will complete an individual and group quiz, based on the lectures and allocated pre-reading. This will be followed by discussion of the answers with your tutor (20 min).

Task 2

Working with your small group, identify common genetic and acquired risk factors associated with osteosarcoma and discuss underlying pathological mechanisms. Discuss your finding with your colleagues and tutor (10min)

Task 3

Name one of each common primary tumour that metastasises to the bone in adult female and male. Discuss their preferred location in the bone and their clinical effects (10 min).

Task 4

Working with your small group, define multiple myeloma, its local effects to the bone and bone marrow, its systemic effects to the kidney, joints and hematopoietic system (10min).

Task 5

Quizzes for the tutorial on Arthritis in Week 5 will be based on the lectures from the 28/08/2013 and 29/08/2013 and pre-reading of *Pathologic Basis of Disease*, 8th Ed. V. Kumar, R. Cotran & S Robbins (2010): Pages 1235-1240 and pages 1242-1245 or *Robbins Basic Pathology*, 9th Ed (2012): Pages 782-790. Students are expected to complete these tasks prior to their quizzes.

Practical 4 – Bone tumours

Aim:

The aim of this practical is to review the histopathology and briefly describe the clinical appearances and investigation of common neoplasms occurring in bone.

Learning objectives:

At the completion of this practical class you should be able to:

1. Describe the clinical, histological and radiographic features of benign and malignant primary bone neoplasms.
2. Understand osteolytic and osteosclerotic effects of metastatic bone tumours and provide typical examples.
3. Describe clinical effects and investigative abnormalities associated with bony metastases.
4. Define the term “multiple myeloma” and discuss the pathophysiology of this disorder in terms of infiltration of bone and the presence of secreted para-proteins in serum and/or urine.
5. Explain the development of renal impairment and immunodeficiency in patients with multiple myeloma.

CASE 1

A 66 year-old man experienced an episode of pain at the right costal margin, which soon subsided. Chest X-ray at the time of admission shown in the attached document at <http://vslides.unsw.edu.au/> revealed a circumscribed lesion, thought to be in the lung.

Task 1

Examine the Chest X-ray and describe the lesion. What further information from the patient would you seek and/or what examinations would you perform exclude a lesion originating from the lung?

CT scan shown at <http://vslides.unsw.edu.au/> indicated an expansile lesion (arrow) in a right upper rib exhibits several benign features: sharp margins, no aggressive periosteal reaction, and no associated soft tissue mass. Some cartilaginous tumour matrix is evident. On the basis of this report thoracotomy was performed, specimen 362.6 was removed and virtual slide 1 prepared. Postoperative progress was satisfactory.

Task 2

Examine specimen 362.6 and describe the lesion. Refer to the Images of Diseases site for a guide. Write a brief histopathological report on virtual slide 1 and provide your final diagnosis.

CASE 2

A 13 year-old boy presented with a three week history of intermittent ache above his left knee. He had not suffered any recent injury. On examination there was a tender swelling on the lateral aspect of the distal left femur as shown at <http://vslides.unsw.edu.au/>. There were no other abnormal physical findings.

Task 3

Describe the X-ray, bone scan and MRI of his left femur shown at <http://vslides.unsw.edu.au/>. What further investigations do you consider appropriate to establish a definitive diagnosis?

On the basis of the results of a biopsy of the lesion, the boy underwent major surgery. Virtual slide 2 was prepared from tissue removed at operation. Specimen 237.6 is representative of the tissue removed at operation.

Task 4

Write a brief description of the abnormalities in virtual slide 2, correlate the microscopic appearance with the macroscopic appearances in Specimen 236.7 and state your diagnosis.

CASE 3**Task 5**

Virtual slide 3 was obtained from the head of femur surgically removed from an elderly man who complained lower back and hip pain for 4 weeks that limited his mobility. He had a long history of urinary tract obstruction that was surgically treated followed by radiotherapy. Bone scan at the time of the operation is shown at <http://vslides.unsw.edu.au/>

What abnormalities are present in the Bone scan and histological tissue? What is the diagnosis?

Task 6

Examine specimen 1469.20, 1293.20, 2959.20, 507.8 and 1206.9. Which of the following specimens is likely to be obtained from this man? Which of the specimens is unlikely to cause metastasis to the bone? Describe the predominant radiological appearances produced by bony metastasis of each cancer?

Homework Tasks

A previously healthy 60 year-old man presents with 2 to 3 months of back pain. Over the last 3 weeks, he has developed a cough and increasing fatigue. On examination he has generalized bone pain with crepitation and abnormal movement of left upper arm and evidence of pneumonia. It is noted on radiography to have osteolytic lesions and a pathological fracture of left humerus shown at <http://vslides.unsw.edu.au/>

The results of four investigations are shown below:

Full Blood Count			
Haemoglobin (g/L)	90	115-165	*
RCC (x 10 ¹² /L)	2.9	3.8-5.8	*
PCV	0.32	0.37-0.47	*
MCV (fL)	95	80-100	
MCH (pg)	31	27-32	
MCHC (g/L)	326	300-350	
WCC (x10⁹/L)	9.0	4.0-11	*
Neutrophils	8.1	2.0-7.5	*
Lymphocytes	0.5	1.5-4.0	*
Monocytes	0.1	0.2-0.8	*
Eosinophils	0.1	0.04-0.4	
Platelets (x10⁹/L)	158	150-400	
ESR (mm/hr)	120	3-12	*

Clinical Biochemistry		
Sodium (mmol/L)	148	135-145
Potassium (mmol/L)	2.8	3.5-5.0*
Chloride (mmol/L)	100	95-107
Calcium (mmol/L)	3.6	2.10-2.55*
Phosphate (mmol/L)	2.3	0.8-1.5*
Uric Acid (µmol/L)	525	200-500*
Alkaline Phosphatase (U/L)	70	36-126
Urea (mmol/L)	9.8	3.0-8.0*
Creatinine (µmol/L)	125	60-110*
Parathyroid hormone (pmol/L)	1.3	1.1-6.9

Urinalysis: Large amount of protein, neutrophils and bacteria detected

Serum Protein Electrophoresis and Bone Marrow Aspiration and Biopsy: see <http://vslides.unsw.edu.au/>

Task 1

Examine virtual slide 4 and write a brief report.

Task 2

Putting together the history and the investigative findings, state your diagnosis? What is the prognosis of this condition?

Task 3

Review the following additional specimens: 867.6, 2303.3, 2004.6, 3230.6, 2498.6, 2320.6, 922.6, 1260.6 and 1469.20.

Tutorial 5 – Arthritis

Aim

The aim of this tutorial is to understand the aetiology, pathogenesis and clinical features of common joint disorders.

Learning objectives

At the completion of this practical class you should be able to:

1. Compare and contrast the clinical features of osteoarthritis with those of rheumatoid arthritis.
2. Describe the changes in the synovial fluid in the common types of arthritis.
3. Explain the pathogenesis of the extra-articular complications of rheumatoid disease.
4. Outline the features of "anaemia of chronic disease".
5. Define the term "tophus" and describe its macroscopic features.

Task 1

You will complete an individual and group quiz, based on lectures and allocated pre-reading. This will be followed by discussion of the answers with your tutor (20 min).

Task 2

Working with your small group, construct a table that compare and contrasts the clinical features of rheumatoid arthritis and degenerative joint disease.

Task 3

Complete the table summarising the appearance and pathological features of synovial fluid in osteoarthritis, rheumatoid arthritis, gout and acute gonococcal arthritis. Discuss the underlying processes leading to each synovial fluid profile (20 min).

	Osteoarthritis	Rheumatoid Arthritis	Gout	Gonococcal Arthritis
Appearance				
Colour				
Viscosity				
White Cells (cells/mm³)				
Neutrophils %				
Protein content				
Culture				
Polarising Microscopy				

Note: No quiz on week 6, week 7 and week 8. There will be MIDSESSION EXAM ON week 6, 06/09/2013; 12-2pm, FEEDBACK/REVISION ON week 7, 13/09/2013, 12-2pm and all groups will attend Evidence Based Symposium on their designated lecture theatres on week 8 (see Timetable for Evidence Based Symposium on Moodle at <https://moodle.telt.unsw.edu.au/>)

Task 4

Quizzes for week 9 tutorial on Metabolic Bone Diseases will be based on the lecture from 26/09/2013 and pre-reading of *Pathologic Basis of Disease*, 8th Ed. V. Kumar, R. Cotran & S Robbins (2010): Pages 1214-1219; pages 1209-1210 and pages 433-436 or *Robbins Basic Pathology*, 9th Ed (2012): Pages 298-300; 735-738; 766-772. Students are expected to complete these tasks prior to their quizzes.

Practical 5 – Histopathology of Arthritis and Clinical correlations

Aim

The aim of this practical class is to correlate the macroscopic and microscopic features of various types of arthritis with their clinical manifestations.

Learning objectives

At the completion of this practical class you should be able to:

1. Contrast the macroscopic and microscopic features of osteoarthritis with those of rheumatoid synovitis.
2. Describe the changes in the synovial fluid in the common types of arthritis.
3. Define "rheumatoid factor" and explain its diagnostic significance.
4. Explain the pathogenesis of the extra-articular complications of rheumatoid disease.
5. Define the term "tophus" and describe its macroscopic and microscopic features.

CASE 1

The X-ray and MRI of the knee joint at <http://vslides.unsw.edu.au/> were obtained from an elderly male, who had a long history of pain in the right hip which limited his mobility.

Task 1

Describe the abnormalities in the Images and the associated specimen 993.18? Which of the two images provide more detail about the extent of the lesion? What is the likely diagnosis?

Task 2

Virtual slide 1 at <http://vslides.unsw.edu.au/> was obtained from the head of a femur removed surgically from the same patient. What abnormalities are present in this tissue? Are the findings consistent with the patient's history and the Images of his joint? Using Images at <http://vslides.unsw.edu.au/> as a hint, list other sites where this condition commonly occurs.

Task 3

What type of operation did the patient undergo? (Use images at <http://vslides.unsw.edu.au/> as a hint). What complications may occur in this patient following this operation?

CASE 2

A 37 year-old woman presented with the insidious onset of polyarthritis in her hands and feet with early morning stiffness, weight loss and low grade fever. Examination revealed symmetrical synovitis involving the metacarpophalangeal and proximal interphalangeal joints of hands as well as her elbows, knees and feet. There was limitation of movement in all involved joints. The patient appeared pale and unwell.

Task 4

What is your provisional diagnosis? What extra-articular complications of this condition may occur? (Use images at <http://vslides.unsw.edu.au/> as a hint)

The results of her investigations are shown below:

Full Blood Count			
Haemoglobin (g/L)	100	115-165	*
RCC (x 10 ¹² /L)	3.3	3.8-5.8	*
PCV	0.35	0.37-0.47	*
MCV (fL)	95	80-100	
MCH (pg)	31	27-32	
MCHC (g/L)	326	300-350	
WCC (x10⁹/L)	14.0	4.0-11	*
Neutrophils	12.1	2.0-7.5	*
Lymphocytes	1.5	1.5-4.0	
Monocytes	0.3	0.2-0.8	
Eosinophils	0.1	0.04-0.4	
Platelets (x10⁹/L)	258	150-400	
ESR (mm/hr)	86	3-12	*

RHEUMATOID FACTOR: Positive with a titre of 1: 640.

ANTI-CYCLIC CITRULLINATED PEPTIDE (CCP) ANTIBODIES: Strongly positive.

X-RAY and MRI OF THE HANDS: attached links at <http://vslides.unsw.edu.au/>

Task 5

How do you explain the leucocytosis and raised ESR? What are rheumatoid factor and anti-CCP antibodies? How do they compare as diagnostic tools for rheumatoid arthritis?

Task 6

Examine specimen 791.6 and virtual slide 2, which are surgical specimens of this woman's synovium. What abnormalities can you see? What would examination of the synovial fluid in this case reveal?

Homework Tasks

An obese 64 year-old man had a 3 cm diameter, irregular, firm lump removed from the subcutaneous tissue along the ulnar surface of his forearm. He was hypertensive and had a past history of recurrent arthritis. Specimen 1755.6 and Virtual slide 3 was prepared from the excised tissue.

Task 1

Write a brief description of specimen 1755.6 and virtual slide 3 at <http://vslides.unsw.edu.au/>. What is your diagnosis? (Use Images at <http://vslides.unsw.edu.au/> as a hint)

Task 2

What other conditions may cause subcutaneous nodules or lumps in the forearm?

Tutorial 6 - Metabolic bone diseases

Aim:

This tutorial class focuses deals with the definition, aetiology and pathogenesis of metabolic bone diseases.

Learning objectives:

At the completion of this practical class you should be able to:

1. Outline the pathogenesis and clinical course of osteoporosis (primary and secondary).
2. Discuss the methods of prevention of post-menopausal osteoporosis.
3. Construct a table comparing the biochemical and radiographic abnormalities associated with Osteoporosis, Osteomalacia, Rickets, and Hyperparathyroidism, Paget disease of bone and Renal osteodystrophy.

Task 1

You will complete an individual and group quiz, based on the lectures and allocated pre-reading. This will be followed by discussion of the answers with your tutor (20 min).

Task 2

Working with your small group, describe the pathogenesis of the following metabolic diseases and construct a table that compare the serum biochemical investigations and radiological finding of each disease (20 min).

	Primary Osteoporosis	Rickets	Primary Hyperthyroidism	Paget disease	Renal Osteodystrophy
Underlying cause					
Sodium (mmol/L) Potassium (mmol/L) Chloride (mmol/L) Calcium (mmol/L) Phosphate (mmol/L)					
Alkaline Phosphatase (U/L)					
25-Hydroxy vitamin D (nmol/L)					
Parathyroid hormone (pmol/L)					
Urea (mmol/L) Creatinine (µmol/L)					
Radiological findings					

Practical 6 – Clinico-Pathological Correlation of Metabolic bone diseases

Aim:

This practical class focuses on Osteoporosis as a major cause of morbidity and mortality in the community. Other metabolic bone diseases and their clinical features are also covered.

Learning objectives:

At the completion of this practical class you should be able to:

1. Define the term “Osteoporosis”.
2. Outline the pathogenesis and clinical course of Osteoporosis (primary and secondary).
3. Discuss the methods of prevention and the investigation of post-menopausal osteoporosis.
4. Explain the pathophysiology of the other metabolic bone diseases; Osteomalacia, Rickets, Hyperparathyroidism, Paget disease of bone and Renal osteodystrophy.

CASE 1

A 70 year-old Caucasian woman with a history of chronic back pain was recently admitted to the hospital with a femoral neck fracture after a fall in her backyard. In the past 3 years she has become increasingly inactive and lost 5 cm of her original height.

Her Clinical Biochemistry is shown below:

Clinical Biochemistry		
Sodium (mmol/L)	140	135-145
Potassium (mmol/L)	3.8	3.5-5.0
Chloride (mmol/L)	99	95-107
Calcium (mmol/L)	2.2	2.10-2.55
Phosphate (mmol/L)	1.3	0.8-1.5
25-Hydroxy vitamin D (nmol/L)	65	50-140
Alkaline Phosphatase (U/L)	78	36-126
Urea (mmol/L)	6.5	3.0-8.0
Creatinine (µmol/L)	85	60-110

Task 1

How do you interpret the above results? What is your diagnosis? What further investigations would you perform to confirm your diagnosis?

Examine the results of her L2-L4 dual energy X-ray absorbitometry (DEXA) plotted (black spot) over a reading from a woman of the same age and race that is shown on attached Image at <http://vslides.unsw.edu.au/>

Task 2

Is the bone mineral density (BMD) of this woman normal? What is her T score and what does it mean? How severe is her condition?

The X-ray from her hip and the vertebrae are shown on attached Images at <http://vslides.unsw.edu.au/> and specimen 682.6 was obtained from another 70 year old patient who died due to the complication of her fracture.

Task 3

Examine specimen 682.6 and the X-ray images and list your observations. How do you interpret the results? How might have this prevented? What are the clinical consequences of this condition? What are the primary and secondary causes of this disease?

CASE 2

A 40 year-old woman was well until two and a half years before admission. She then sustained several fractures from minimal trauma. These included fracture of the left ankle and metatarsal bones and upper femoral shaft. She was weak and tired, and suffered from generalised pain in her bones.

The X-rays of her hands are shown on attached Image at <http://vslides.unsw.edu.au/> and Her Clinical Biochemistry showed below:

Clinical Biochemistry		
Sodium (mmol/L)	140	135-145
Potassium (mmol/L)	3.8	3.5-5.0
Chloride (mmol/L)	100	95-107
Calcium (mmol/L)	3.2	2.10-2.55*
Phosphate (mmol/L)	0.5	0.8-1.5*
25-Hydroxy vitamin D (nmol/L)	95	50-140
Alkaline Phosphatase (U/L)	142	36-126*
Urea (mmol/L)	6.5	3.0-8.0
Creatinine (μ mol/L)	85	60-110
Parathyroid hormone (pmol/L)	173	1.1-6.9

Task 4

How do you interpret the results of the X-rays and the Clinical Biochemistry?

A radionuclide scan of the neck shown at <http://vslides.unsw.edu.au/> was performed and a bone biopsy was taken for histopathological examination. She underwent explorative surgery of the neck and specimen 1141.22 was removed from below the lower left pole of the thyroid gland.

Task 5

Examine the radionuclide scan and specimen 1141.22 and state your diagnosis. How this condition might have led to the abnormal serum calcium and phosphate levels?

Virtual slide 1 was prepared from the bone biopsy.

Task 6

Examine virtual slide 1 at <http://vslides.unsw.edu.au/> and identify the microscopic features that are consistent with hyperparathyroidism.

Task 7

What are the complications of hyperparathyroidism and give the main causes of primary and secondary hyperparathyroidism.

CASE 3

Specimen 2997.6 and virtual slide 2 were obtained from a 66 year-old man who died from serious complications of a long-standing metabolic bone disease. The X-rays of this man's skull and humerus are shown in slides 6 and 7 and a CT scan is shown at <http://vslides.unsw.edu.au/>.

Task 8

Describe the abnormalities shown in specimen 2997.6 and the diagnostic imaging investigations. What is your diagnosis? How might this condition have presented clinically?

Task 9

Examine virtual slide 2 at <http://vslides.unsw.edu.au/> and write a brief histopathological report. Is this slide consistent with the patient's history?

Homework Tasks

A 2 year-old child presented with shortened and deformed lower extremities and failure to walk properly. She was recently adopted from a poorly managed Romanian state orphanage where she spent most of her life confined to her bed. On physical examination she was irritable and apathetic with a height and weight below the 5th percentile. There was delayed psycho-motor and speech development. Multiple deformities of the skeleton including poorly formed teeth, severe bowing of the tibiae bilaterally (45 degrees), pigeon chest (pectus carinatum) and bilateral widened wrists were evident.

Her lower limb and wrist X-rays are shown at <http://vslides.unsw.edu.au/> and her clinical biochemistry is shown below:

Clinical Biochemistry		
Sodium (mmol/L)	140	135-145
Potassium (mmol/L)	3.8	3.5-5.0
Chloride (mmol/L)	100	95-107
Calcium (mmol/L)	1.8	2.10-2.55*
Phosphate (mmol/L)	0.5	0.8-1.5*
25-Hydroxy vitamin D (nmol/L)	25	50-140*
Alkaline Phosphatase (U/L)	155	36-126*
Urea (mmol/L)	4.5	3.0-8.0
Creatinine (μ mol/L)	75	60-110

Task 1

How do you interpret the above results? What is your diagnosis?

Task 2

Examine virtual slide 3 taken from a child with rickets. Identify evidences of insufficient bone formation. Describe the aetiology and pathogenesis of rickets. What are the clinical consequences of this disease?

Task 3

What are the causes of vitamin D deficiency in children? Name 3 causes of osteomalacia in adults.

Practical/Tutorial 7– Skeletal Muscle diseases

Aim:

This practical class aims to familiarise you with diseases of muscle and places particular emphasis on the clinical features of these diseases.

Learning objectives:

At the completion of this practical class you should be able to:

1. Outline the components of normal skeletal muscle.
2. Describe the clinical course, pathogenesis and genetics of the X-linked muscular dystrophies.
3. List the autosomal muscular dystrophies.
4. Compare the clinical course and histopathologic changes of myotonic dystrophy with Duchenne muscular dystrophy.
5. Classify the various myopathies based on the major disease processes and appreciate their relative importance.
6. Discuss polymyositis as an example of inflammatory myopathy.
7. Understand the indications for muscle biopsy.

Aim: To set-up and analyse laboratory assays and histology used to diagnose muscular dystrophy

Methods: Western blotting, Autoradiography, Immunohistochemistry

Materials: SDS-PAGE gels, buffer and electrophoresis equipment, patient and transgenic mouse muscle samples from biopsy, images showing immunohistochemistry from normal and dystrophic patient samples

Results: Attempt to answer tasks on sheets provided

Outcomes: Discuss the types of assays we have used today and their relevance in the diagnosis of this disease. Describe your interpretation of the results.

STATION 1:

SDS-PAGE (Sodium Dodecyl Sulphate-PolyAcrylamide Gel Electrophoresis):

Muscle samples were taken from normal and dystrophic patients and prepared to enrich for dystrophin protein; 10% mini-gels will be loaded with normal and dystrophic protein samples; These gels (which now contain protein are blotted to membranes and assayed with an antibody to dystrophin); This assay is called Western blotting and is used to detect levels of expressed protein.

Tasks:

1. Why are molecular weight markers used in this assay?
2. In which direction do proteins migrate when being electrophoresed through a PAGE gel?
3. Discuss the relevance of this assay.

STATION 2:

AUTORADIOGRAPHY

Once Western blotting with the dystrophin antibody has been performed, detection of protein expression is facilitated by exposure of the antibody signal to an X-ray film.

This film is then developed and the result can be visualised.

Tasks:

1. Assess the banding pattern on the autoradiogram and describe what is seen in each lane.
2. Which lane contains the individual with a dystrophic muscle?
3. What is your diagnosis of the protein expression seen?
4. Discuss the interpretation of this result.

STATION 3:

IMMUNOHISTOCHEMISTRY

Biopsies have been taken from patients with normal and dystrophic muscles;

Histological techniques were then applied to these samples;

Immuno-staining with the same dystrophin antibody (as the primary) used in Western blotting;

Detection of dystrophin was visualised with a second antibody (as the secondary) which contains a fluorescent label.

Task:

1. Describe the location of the fluorescent signal.
2. Is this the case for a patient with muscular dystrophy?
3. Comment on the value of this technique in the analysis of muscular dystrophy.

Note: No quiz next week. All groups will attend a combined Practical and Tutorial class on Head injury and Shock from 12-2 pm at G6/G7

Practical/Tutorial 8 – Head injury and Shock

Aim:

The aim of this topic is to review the common causes of head injury and discuss the pathophysiology of shock.

Learning objectives:

At the completion of this practical class you should be able to:

1. Describe the various types of intracranial haemorrhage, their common sites and factors predisposing to their development.
2. Summarise the effects of raised intracranial pressure.
3. Outline the causes of shock.
4. Discuss the pathophysiology of acute renal failure and adult respiratory distress syndrome following severe trauma.

CASE 1

A 30 year-old horse trainer was brought to hospital by ambulance 45 minutes after being kicked by a horse on the left side of the head. He had briefly lost consciousness immediately following the blows. On arrival at hospital he was confused but conscious, and was therefore sent for an urgent skull X-ray. While in the radiology department, his condition deteriorated rapidly and he lapsed into coma. He died 8 hours after admission. Post mortem examination revealed a depressed skull fracture. Images shown at <http://vslides.unsw.edu.au/> and specimen 675.29 were taken short before death and at autopsy respectively.

Task 1

What is your diagnosis?

Task 2

What is the main pathological change in specimen 675.29? Use the Images of Disease website (<http://iod.med.unsw.edu.au>) as a guide

Task 3

What might have caused his death?

CASE 2

Specimen 1479.17 and Images at <http://vslides.unsw.edu.au/> were obtained from a 46 year-old man who suffered a severe head injury after falling from the 4th floor of a building. He lost consciousness immediately after the accident and rapidly deteriorated and died as a consequence of increased intracranial pressure (ICP).

Task 4

What is your diagnosis?

Task 5

What are the clinical manifestations of raised ICP?

Task 6

What are the complications of raised ICP?

CASE 3

Specimen 2277.17 and the image at <http://vslides.unsw.edu.au/> were obtained from a 60 year-old male alcoholic who was found in a park semi-conscious with minor bruises to his face and his scalp. He was suspected by the police to be intoxicated, and was kept at the police station for the night to sober up un-attended. Next morning he was found dead in his cell. Post-mortem examination found an old blood clot covering 2/3rd of the parietal lobe of his brain.

Task 7

What is your diagnosis, and how could this condition have occurred?

Task 8

What abnormalities might you expect to see in the underlying brain tissue?

Task 9

What might have been the clinical manifestations of this condition prior to the death of the patient?

CASE 4

Specimens 288.29 and 399.29 were obtained at autopsy from a 58 year-old woman who suddenly collapsed in her bathroom. Apart from a history of occasional headaches and mild hypertension, she was well prior to her death. Use Images at <http://vslides.unsw.edu.au/> as an additional guide.

Task 10

What is your diagnosis?

Task 11

What are the main pathological changes in specimens 288.29 and 399.29?

Task 12

How might this condition occur?

CASE 5

Specimens 2913.29 and 732.29 were obtained from two patients who died after intracerebral bleeding. Patient A had a history of chronic hypertension and patient B had a history of coronary heart disease. Use the images at <http://vslides.unsw.edu.au/> as additional guide

Task 13

What are the main pathological changes in specimens 2913.29 and 732.29?

Task 14

Which specimen is likely to belong to patient A and which one to patient B?

Task 15

Name three other causes of intra-cerebral haemorrhage.

CASE 6

Virtual slide 1 at <http://vslides.unsw.edu.au/> was prepared from tissue taken at post mortem from a woman who died due to massive blood loss. Firstly, determine the source of the tissue and decide which part of the slide shows the most prominent change. List any of the changes you can observe. Are these changes consistent with your understanding of massive blood loss and hypovolemic shock? What is your diagnosis?

CASE 7

Examine virtual slide 2 at <http://vslides.unsw.edu.au/> that shows one of the consequences of septic shock. Write a histopathological report. What is your diagnosis? What clinical manifestations could arise? Name two major pathogenetic mechanisms leading to this condition.

CASE 8

Virtual slide 3 at <http://vslides.unsw.edu.au/> is an example of acute respiratory distress syndrome (ARDS), which often complicates massive trauma and shock. Write a histopathological report. How does this process lead to impaired oxygenation of the blood and increase in the work of breathing? What are the consequences of this problem?

Homework Task

1. Which organs are commonly injured in prolonged shock? What lesions result?
2. Review the additional notes provided in the attached document at <http://vslides.unsw.edu.au/> that describe clinical assessments of head injury and monitoring of head injury by Glasgow Coma Scale.

Course evaluation questionnaire

This questionnaire is included in the manual to be completed by all students during Practical Class 12 (during week 12) to provide feedback on the course. The questionnaire is designed to be completed anonymously, but if you wish to include your name or discuss your comments with me please feel free to do so during the session. If you feel a specific area did not meet your expectations, your suggestions on how it may be improved are most valuable to us. Please refer to the table of contents of this manual for a list of lecture and practical titles.

Thanks for your help.

A/Professor Nicodemus Tedla, Course Convenor.

Lectures:

Please indicate the extent to which you agree/disagree with the following statements:

	Strongly agree			Strongly disagree		
The lecture series focused on interesting topics:	1	2	3	4	5	N/A
The lecture series was challenging:	1	2	3	4	5	N/A
The lectures attempted to cover too much material:	1	2	3	4	5	N/A
The lecture series was too content driven:	1	2	3	4	5	N/A

Please select one aspect of the lecture course you liked and suggest one way in which it could be improved.

Liked: _____

Could be improved by: _____

Team-based learning/tutorials:

Please indicate the extent to which you agree/disagree with the following statements:

	Strongly agree			Strongly disagree		
They added to my understanding:	1	2	3	4	5	N/A
They helped me to focus my study:	1	2	3	4	5	N/A
They attempted to cover too much material:	1	2	3	4	5	N/A
The sessions were not challenging enough:	1	2	3	4	5	N/A

Please select one aspect of the tutorials and team-based learning you liked and suggest one way in which it could be improved:

Liked: _____

Could be improved by: _____

Practical classes:

Please indicate the extent to which you agree/disagree with the following statements:

	Strongly agree			Strongly disagree		
They added to my understanding:	1	2	3	4	5	N/A
They helped me to focus my study:	1	2	3	4	5	N/A
Feedback on homework tasks was helpful:	1	2	3	4	5	N/A
They attempted to cover too much material:	1	2	3	4	5	N/A
The sessions were not challenging enough:	1	2	3	4	5	N/A

Please select one aspect of the practical classes you liked and suggest one way in which it could be improved:

Liked: _____

Could be improved by: _____

Evidence-based Symposium:

Please indicate the extent to which you agree/disagree with the following statements:

	Strongly agree			Strongly disagree		
They were a useful addition to the course:	1	2	3	4	5	N/A
The topics were interesting:	1	2	3	4	5	N/A
The aims were clear:	1	2	3	4	5	N/A
I learned useful skills in making the presentation:	1	2	3	4	5	N/A
The presentations were not challenging enough:	1	2	3	4	5	N/A

Please suggest one aspect of the presentations you liked and suggest one way in which it could be improved:

Liked: _____

Could be improved by: _____

Midsession exam and feedback:

Please indicate the extent to which you agree/disagree with the following statements:

	Strongly agree			Strongly disagree		
The midsession exam provided useful feedback:	1	2	3	4	5	N/A
Feedback on midsession exam was useful:	1	2	3	4	5	N/A
Midsession exam helped me focus on my study:	1	2	3	4	5	N/A
Midsession exam was not challenging enough:	1	2	3	4	5	N/A

Please select one aspect of the on-line assessments you liked and suggest one way that they could be improved:

Liked: _____

Could be improved by: _____

The course overall:

Please indicate the extent to which you agree / disagree with the following statements:

	Strongly agree			Strongly disagree		
I generally appreciated the standard of work expected:	1	2	3	4	5	N/A
The course developed my analytical skills:	1	2	3	4	5	N/A
The workload was too heavy:	1	2	3	4	5	N/A
To do well in this course all you need is a good memory:	1	2	3	4	5	N/A
The course manual assisted my learning:	1	2	3	4	5	N/A
The aims and objectives in the manual were useful:	1	2	3	4	5	N/A
The course encouraged critical thinking and understanding:	1	2	3	4	5	N/A
The staff worked hard to make their subjects interesting:	1	2	3	4	5	N/A
Overall I was satisfied with the quality of the course:	1	2	3	4	5	N/A
I would recommend this course to my friends:	1	2	3	4	5	N/A

Please list two aspects of the course you liked and two ways in which you think the course could be improved.

Liked: _____

Liked: _____

Could be improved by: _____

Could be improved by: _____

Other comments:

Name (optional): _____

***Thank you for your assistance.
 Please tear out these sheets and hand them in.***

Glossary of terms used in Pathology

Abscess

a localised collection of pus in an organ or tissue

Acquired

a lesion occurring due to an event after birth (cf. congenital)

Adenoma

a benign neoplasm derived from glandular (secretory) epithelial cells

Acute respiratory distress syndrome (ARDS)

respiratory failure caused by diffuse damage to type 1 pneumocytes and alveolar capillaries, often secondary to shock (hence the term "shock lung")

Aetiology

cause of a disease

Agensis

congenital absence of an organ or structure

Allele

one of two alternative genes at a locus that controls a particular characteristic

Allergen

antigen which gives rise to allergic reactions, usually mediated by IgE antibody

Allograft

a tissue graft between two individuals of the same species (synonymous with homograft)

Anaemia

a significant reduction in the level of circulating haemoglobin below the normal range

Anaplasia

less than normal differentiation of cells; an important feature of malignant neoplasms

Aneurysm

a localised abnormal dilatation of a vessel due to weakness of its wall

Anorexia

loss of appetite

Antibody

immunoglobulin specifically reactive with a particular antigen

Antigen

a substance which can induce a detectable immune response

Appendicular skeleton

the skeleton associated with the appendages; ie scapula, clavicle, arms and legs

Aplasia

congenital disturbance leading to failure of development of a part (synonymous with agenesis)

Apoptosis

a form of individual cell death, particularly observed in physiological turnover, in which the morphological changes consist of nuclear condensation and fragmentation (cf. necrosis)

Arthralgia

pain (of any cause) in a joint or joints

Arthritis

inflammation of a joint or joints; usually signified by pain (arthralgia), erythema and swelling

Arthrodesis

surgical fusion of a joint usually performed to relieve pain and instability . Arthro= joint; Desis= fusion
abnormal accumulation of fluid in the peritoneal cavity

Atrophy

diminution in size of an organ or tissue which had previously reached mature size, due to a decrease in size and/or number of its constituent specialised cells (cf. agenesis, aplasia and hypoplasia)

Arthroplasty

surgery to relieve pain and/or restore range of motion, by realigning or reconstructing a joint

Autoantibody

antibodies against self antigen

Autoimmunity

a disease caused by failure of normal immunological tolerance, such that the immune system identifies "self" antigens as foreign

Axial skeleton

the skeleton of the axis, including skull and hyoid, vertebral column, ribs and sternum

Bacteraemia

the presence of bacteria in the blood (cf. pyaemia and septicaemia)

Benign

in reference to neoplasms, the term indicates strict localisation, growth by expansion, and frequent encapsulation (synonymous with innocent)

Biopsy

sampling of tissue for diagnosis, includes excisional, incisional and needle procedures, and also subsumes many cytological procedures

Callus

tissue of repair that bridges the fractured ends of bone, made up of osteoprogenitor cells and their products as well as new blood vessels; forms bones by endochondral or intramembranous ossification.

Cachexia

extreme wasting of the body, accompanied by weakness, anorexia and anaemia; most commonly seen in the terminal phase of malignancy

Calculus

a stone formed in a hollow tube or viscus, e.g. gallbladder, renal pelvis

Cancellous or Spongy bone

bone arranged in plates or struts (trabeculae) with many large irregular marrow spaces

Cancer

often used synonymously with carcinoma (see below); also a general term for all malignant neoplasms

Carbuncle

a multilocular abscess resulting from extension of a boil into the subcutaneous tissues

Carcinogen

an agent which can cause a cell to undergo neoplastic transformation, or which may initiate such a process by permanently altering cellular DNA

Carcinoma

a malignant neoplasm derived from epithelium

Carcinoma in situ

a malignant epithelial neoplasm which has not yet invaded through the basement membrane

Cartilage

tough, resilient, pliable, avascular compact connective tissue; provides support for some soft tissues, reduces friction at joints and is used as a template for the development of fetal skeleton. Divided into Hyaline, Elastic and fibrocartilage on basis of the amount of matrix present and the fibre type

Cell-mediated immunity (CMI)

immune response in which T-cells and macrophages predominate

Cellular differentiation

process of development of phenotypic characteristics of a mature tissue by selective gene expression

Chondroblast

immature cells that form cartilage matrix

Chondrocyte

mature cells located in lacunae of cartilage matrix

Chronic inflammation

an inflammatory response evoked by a persistent stimulus and characterised by aggregation of inflammatory cells and tissue proliferation rather than exudation

Circumscribed

well defined or demarcated, e.g. circumscribed lesion

Compact bone

densely packed bony substance arranged in regular lamellae in Haversian systems (osteon).

Congenital

literally, "born with" a disease; a condition attributable to events prior to birth

Clone

a group of cells, all of which are the progeny of a single cell

Clot

a semi-solid mass formed from constituents of the blood after death (post-mortem clot), following haemorrhage, or in vitro (cf. thrombus)

Consolidation

becoming firm or solid: usually applied to the lung in which the alveolar spaces are filled to varying degrees with inflammatory exudate, retained secretions, neoplastic tissue or scar tissue

Cytokines

protein or peptide molecules mediating pathologically significant cellular reactions

Degeneration

a change in structure and function caused by injury to cells; the change is often reversible

Degenerative joint disease

see osteoarthritis

Delayed hypersensitivity (DTH)

cell-mediated immune response elicited by the subcutaneous injection of an antigen, with subsequent oedema and inflammation which are maximal between 24 and 48 hours (cf. immediate hypersensitivity)

Demyelination

loss of myelin ensheathing axons

Diaphysis

shaft of a long bone

Disseminated intravascular coagulation (DIC)

widespread thrombosis in the microvasculature arising secondary to another illness, resulting in consumption of platelets and clotting factors (often leading to severe haemorrhage), traumatic damage to red cells and ischaemia to vital organs; common causes include septicaemia, obstetric emergencies and malignancy

Dysplasia

atypical cellular differentiation; may be observed histopathologically within neoplasms or pre-neoplastic lesions

Dystrophic calcification

the localised deposition of calcium salts in dead or degenerate tissue (in the presence of normal plasma levels of calcium and phosphorus)

Epiphysis

ends of a long bone containing red bone marrow

Epiphyseal line (growth plate)

boundary zone between Epiphysis and Metaphysis during growth; in adults identified radiologically as a line

Endochondral ossification

bone formation by replacement of a preexisting hyaline cartilage

Endosteum

squamous cells and connective tissue layer lining medullary cavity; contains osteoprogenitor cells, osteoblasts and osteoclasts

Embolism

the transportation by the blood of abnormal material and its impaction in a vessel at a point remote from its entry into the circulation

Epidemiology

the study of the incidence, distribution, and determinants of disease in a population, and its application to the control of health problems

Exudate

proteinaceous fluid resulting from the selective extravasation of intravascular plasma in response to an inflammatory stimulus; exudate usually has a specific gravity exceeding 1.020 due to its relatively high content of protein and cellular debris (cf. transudate)

Fibrinoid

a descriptive term for a variety of microscopic changes that occur in various tissues under dissimilar circumstances, in which the affected tissues stain brightly with eosin

Fibrinous

the adjectival form of fibrin - the protein formed by interaction of thrombin and fibrinogen

Fibrous

literally, containing fibres; but often used in Pathology to refer to collagenous connective tissue

Fine needle aspiration (FNA)

a form of biopsy in which a fine needle (usually 25 gauge) is inserted into an area of tissue and a number of cells are collected, then expelled onto a slide and stained for cytological examination

Fistula

an abnormal communication between two body surfaces (cf. sinus)

Fracture

a break in the continuity of bone

Gangrene

necrosis with putrefaction of macroscopic portions of tissue

Grade

degree of malignancy of a neoplasm, judged from histological features

Granulation tissue

consists of newly formed blood vessels, fibroblasts and their products, and inflammatory cells: the tissue of repair

Granulomatous inflammation

a form of chronic inflammation; characterised by focal aggregations of chronic inflammatory cells, principally macrophages and their derivatives, e.g., epithelioid cells; these focal lesions are known as granulomas, and may exhibit central necrosis

Haematoma

localised collection of blood or clot in solid tissues

Healing

the process by which the body replaces damaged tissue with living tissue; healing includes both regeneration and repair

Hernia

the abnormal protrusion of the whole or part of a viscus or other internal structure through an opening

HLA (Human Leucocyte Antigen)

the major histocompatibility (MHC) genetic region in man; important in control of immune responses and graft rejection

Humoral immunity

immune response in which the predominant effector mechanism involves antibodies

Hyaline

a descriptive term for homogeneous, somewhat glassy or refractile microscopic appearance exhibited by various extracellular tissue elements or by the cytoplasm of cells

Hydroxyapatite

inorganic matrix of the bone accounting for 65% of the bone content; made up of calcium phosphate salts

Hyperaemia

an increased volume of blood within actively dilated vessels in an organ or part of the body (cf. congestion)

Hyperplasia

an increase in size of an organ or tissue due predominantly to an increase in the number of its constituent specialised cells

Hypertrophy

an increase in size of an organ or tissue due predominantly to increase in size of its constituent specialised cells

Hypoplasia

the failure of development of an organ to a full, mature size (cf. aplasia)

Iatrogenic

implies 'caused by doctors', incorrectly derived from Greek root

Immunity

a state of reactivity following exposure to an antigen

Infection

the invasion of the body by pathogenic micro-organisms

Inflammation

the process by means of which exudate and cells accumulate in irritated tissues and usually tend to protect them from further injury; may be acute or chronic –when unqualified, the term "inflammation" usually refers to acute inflammation

Intramembranous ossification

bone formation directly in primitive connective tissue or mesenchyme

Joint capsule

includes an outer layer of dense connective tissue with blood vessels and nerves, and an inner layer of synovial membrane

Juvenile osteomalacia (rickets)

defect in mineralization of cartilage in the growth plate

Lacunae

small cavities in the matrix of cartilage or bone that are occupied by chondrocytes or osteocytes; two chondrocytes may occupy a single lacuna (isogenous group)

Lamellar bone

see compact bone

Ischaemia

a state of inadequate blood supply to a tissue or organ - potentially reversible

Keloid

hypertrophic cutaneous scar, in excess of that necessary to heal the original defect

Lesion

an alteration of structure or of functional capacity due to injury or disease

Leucocytosis

an elevated number of circulating white blood cells

Leucopenia

a decreased number of circulating white blood cells

Malignant

literally means virulent or life-threatening; in reference to neoplasms, the term indicates rapid growth, invasion of neighbouring tissues, potential for spread by metastasis, and frequently a fatal outcome; the single most important histopathological criterion of malignancy is tissue invasion

Medullary cavity

large central cavity containing bone marrow; in adults it is fatty yellow marrow whereas in fetal bones it is cellular red marrow

Metaplasia

An adaptive substitution of one type of differentiated cell(s) by another type of differentiated cells

Metaphysis

distal end of diaphysis connects; the region of elongation in growing bone

Metastasis

in reference to malignant neoplasms, the term refers to the development of secondary growths which arise from, but are discontinuous with, the primary lesion; such is termed a metastasis or metastatic lesion (synonymous with secondary)

Metastatic calcification

precipitation of calcium salts in apparently normal tissue as a result of disturbed calcium-phosphorus metabolism (e.g., hypercalcaemia) (cf. dystrophic calcification)

Monoclonal

attributable to a single clone of cells, and so more characteristic of a neoplastic than a reactive process (polyclonal)

Morphology

the structure of tissues and organs

Muscular Dystrophy

inherited disorder, characterised by the reduction or absence of dystrophin protein in the muscle fibre

Mutagen

an agent capable of damaging the DNA structure of cells; initiators of neoplastic transformation are mutagenic

Myalgia

pain in one or more muscles

Myelin

consists of lipid bilayers in which transmembrane proteins are embedded

Myesthenia Gravis

caused by immune-mediated loss of acetylcholine receptors

Myopathy

muscle disease

Myositis

inflammatory muscle disease

Myotonia

The sustained involuntary contraction of a group of muscles

Necrosis

death of cells in a restricted portion of tissue, recognisable by the autolytic changes undergone after the cells have died

Neoplasm

an abnormal mass of tissue, the growth of which exceeds and is uncoordinated with that of the normal tissues, and persists in the same excessive manner after cessation of the stimuli which evoked the change

Oedema

excessive accumulation of fluid causing swelling of tissues

Organisation

a part of the healing process, occurring after an injury that has destroyed tissue which is unable to regenerate; involves the ingrowth of granulation tissue

Osteoarthritis

characterised by a progressive deterioration and breakdown of articular cartilage, mainly in the weight bearing joints

Osteoblasts

bone forming cells, typically located at edges of bony trabeculae, which secrete organic matrix that establishes conditions favorable for calcification. They are derived from osteoprogenitor stem cells and convert to osteocytes when surrounded by calcified matrix.

Osteoclasts

large motile multinucleated cells derived from monocytes, located at bone edges with active surface facing the Howship's lacuna. They secrete acids and enzymes that dissolve matrix and break down bone.

Osteocytes

mature bone cells in lacunae surrounded by calcified matrix.

Osteogenesis

the process of bone formation by replacement of preexisting connective tissue. (see intramembranous vs endochondral ossification).

Osteoid

Organic matrix of the bone. It accounts for 35% of the bone content. It is made up of Type 1 collagen fibers (90%), proteoglycans (hyaluronic acid) and non-collagenous matrix proteins (osteocalcin, osteopontin and osteonectin)

Osteomalacia

phosphate depletion resulting in defective matrix mineralisation

Osteoid Osteoma

small, benign neoplasm without malignant potential

Osteomyelitis

inflammation of the bone and medullary cavity

Osteopetrosis

overgrowth and sclerosis of bone

Osteoporosis

a reduction in bone mass due to small, incremental losses of bone matrix during turnover

Osteosclerosis

increased bone mass due to increased osteoblast activity

Ossification

See osteogenesis

Pannus

proliferating mass of hyperplastic synovial lining cells admixed with inflammatory cells, fibrin and granulation tissue. (see rheumatoid arthritis)

Pathogenesis

mechanism(s) by which the cause (aetiology) of a disease produces the clinical manifestations

Pathology

the scientific study of diseases

Periosteum

outer dense fibrous layer covering bone surface (except articular cartilage and at the insertion site of tendons and ligaments). Continuous with tendons via Sharpey's fibers (collagen fibers embedded in bone lamellae). Inner osteogenic layer contains vessels, osteoprogenitor cells and osteoblasts.

Phagocytosis

ingestion of foreign or particulate matter by cells

Polymerase chain reaction (PCR)

a molecular diagnostic technique based on amplification by DNA polymerase of a known sequence of genomic DNA isolated from cells, or of DNA reverse-transcribed from mRNA or viral RNA; permits rapid, sensitive and specific detection of e.g. genetic mutations

Prognosis

forecast of the outcome of an illness, based on the natural history of the disease and the likely response to treatment

Pus

typically a semi-fluid of creamy colour, pus is composed of necrotic and living neutrophils, together with necrotic tissue cells and exudate

Pyaemia

the presence of pus-inducing micro-organisms in the circulation with resultant formation of abscesses at sites of their lodgment (cf. bacteraemia and septicaemia)

Pyknosis

shrinkage and increased basophilic staining of the nucleus in a necrotic or apoptotic cell, caused by reduced pH (cf. karyolysis, karyorrhexis)

Regeneration

replacement of parenchymal cells by multiplication of similar surviving cells

Repair

replacement of lost tissue by connective tissue elements and parenchymal cells in varying proportions; when replaced completely by granulation tissue, which later matures to fibrous tissue, the result is referred to as a scar

Resolution

the return of a diseased tissue or organ to normal without residual scarring

Rheumatoid Arthritis

a severe form of chronic synovitis that can lead to the destruction and ankylosis of affected joints

Sarcoma

a malignant neoplasm arising from mesenchymal tissue

Sclerosis

hardening of tissue, especially from overgrowth of fibrous tissue

Septicaemia

severe infection with marked systemic clinical features; septicaemia is usually the expression of rapid and continuous invasion of the blood stream by microorganisms from the tissues, or multiplication in blood stream (cf. bacteraemia and pyaemia)

Shock

a clinical state in which there is widespread inadequate perfusion of tissues

Sign

a clinical feature identified by observation or examination of the patient (cf. symptom)

Staging

assessment of the size and extent of spread of a malignant neoplasm, important in determining the treatment and prognosis

Stem cell

a primitive cell from which differentiated cells arise during development, renewal and maintenance

Subchondral bone

the bone adjacent to (ie underlying) the articular cartilage of a joint

Symptom

a manifestation of disease which the patient may be aware of, or describe

Syndrome

a group of symptoms and signs which, when considered together, characterise a disease or lesion

Synovium

the inner layer of the joint capsule; normally covered by one or two layers of synovial cells

Teratogen

an environmental agent which acts in utero to cause abnormal development, resulting in malformation of the fetus; teratogens include infective agents, radiation, drugs and chemicals

Teratoma

a true neoplasm arising from totipotential cells and therefore composed of numerous tissues which may not be indigenous to the part in which it occurs

Thrombus

a solid or semi-solid mass formed from the constituents of blood within the intact vascular system during life (cf. clot)

Tolerance

a state of non-responsiveness of cells of the immune system to a particular antigen

Trabeculae

latticework of cancellous bone that orient along stress lines with cross-bracing; can withstand stress from many directions although less strong and lighter than osteon of compact bone. Contains red marrow

Transudate

fluid accumulated in tissue planes or spaces which is low in protein and which has leaked into the tissues from the micro-circulation; it occurs in non-inflammatory disorders such as congestive cardiac failure and venous obstruction (cf. exudate)

Tumour

a lump or swelling; however, the term is frequently used as a synonym for neoplasm

Tumour suppressor gene

a gene present in normal cells, which acts to suppress cellular proliferation (e.g. *TP53*, *RB*); when both alleles are inactivated, may cause or accompany the onset of neoplastic transformation

Western blotting (immunoblotting)

a molecular diagnostic technique involving separation of proteins by gel electrophoresis, transferring them to a solid membrane via a blotting procedure, incubating with specific antibodies and applying a sensitive technique for detection of bound antibody; often used to detect specific proteins (e.g. viral) present in the serum

Woven bone

observed in developing bone, fracture healing, tumour-derived bone and other pathological states; its trabeculae are not oriented along stress lines and it is less strong than mature lamellar or compact bone.