Correspondence

Bones, groans, moans and salivary stones?
A 46 year old man was referred to hospital by his general practitioner with abnormal bone biochemistry. He had presented with poor appetite, fatigue, myalgia, and backache. Serum calcium, corrected for albumin, was 2.63 mmol/litre (reference range, 2.12–2.62), serum phosphate was 0.85 mmol/litre (normal range, 0.7–1.4), and alkaline phosphatase was 367 IU/litre (normal range, 0.085–0.65). Bone densitometry provided evidence of osteoporosis (T score, −3.05). Ultrasound of his neck revealed a solid lesion of low echodensity at the lower pole of the right lobe of the thyroid gland, typical of a parathyroid adenoma. At surgery the lower right parathyroid gland was excised, and confirmed by histology to be an adenoma.

At outpatients one week before elective parathyroidectomy, the patient reported that he had passed a stone from a salivary gland. He had attended hospital as an emergency two months previously and had been diagnosed as having sialadenitis of the left submandibular gland with a small calculus present into the duct. Subsequently, he became exasperated with the pain and manually forced the calculus out of the duct. There was no history of chronic infection or of other pathology to explain the presence of the calculus. The calculus weighed 2 mg and consisted of calcium phosphate (59%) and calcium oxalate (41%).

Sialolithiasis has been reported in hyperparathyroidism secondary to chronic renal failure, but not previously in primary hyperparathyroidism. Salivary stone formation may be promoted by the combined effects of calcium and phosphate release into the acinar lumina resulting in calcium phosphate aggregates. Such calcium phosphate intermediates may transform into more stable hard deposits. Their saturation in solution varies widely, partly because of the variation in pH that occurs in saliva. As a result, some of these calcium phosphate aggregates may precipitate.

Why do salivary stones occur so much less frequently than urinary stones in hyperparathyroidism? Some ions in saliva and urine, such as citrate, inhibit the growth of precipitated crystals, whereas others, like calcium and phosphate, accelerate growth. The balance of these and other molecules might favour stone formation in urine but not in saliva. Certainly, mechanisms invoked to explain urolithiasis in hyperparathyroidism include hypercalcuria, hyperphosphaturia, and hypocitraturia. However, salivary concentrations of calcium and phosphate also raise in primary hyperparathyroidism; the comparative rarity of salivary stones in hyperparathyroidism probably owes more to specific salivary proteins that control mineralisation, such as statherin and proline rich protein. It is possible that when salivary stones develop in hyperparathyroidism, they arise via the mechanism outlined above, with alterations in the concentrations of calcium and phosphate playing a primary pathogenic role. Such a putative similarity in the pathogenesis of salivary and urinary nephrolithiasis would be consistent with the observed association between the two conditions. In one large series, six of 56 patients with sialolithiasis were reported to suffer from nephrolithiasis as well.

It is impossible to estimate accurately the true extent of any putative link between hyperparathyroidism and sialolithiasis, precisely because such a link has not been widely recognised. Certainly, most patients with salivary stones are not investigated for abnormal bone biochemistry. The time honoured mnemonic has it that hyperparathyroidism and other hypercalcaemic states were classically associated with “bones, stones, abdominal moans, and psychic groans”. Although this full blown clinical presentation is rarely seen today, we suggest that it may include salivary as well as urinary stones.

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A case of purple urine bag syndrome associated with Providencia retgeri

We would like to report this interesting case of an elderly lady (85 years old) who has been passing violet coloured urine over the past four weeks. She is living in a nursing home and has a long term urinary catheter. There were no other symptoms but her general practitioner was worried about urine discolouration and sent three urine samples to the biochemistry department on three separate occasions to identify the cause of the violet colour. There was no history of intake of medication, food colourings, or any other substances that may alter the urine colour. The urine sample was alkaline (pH 8.5) with a strong smell of ammonia. It was centrifuged and a precipitate of fine blue crystals was identified in the sediment. The supernatant was clear and purple coloured, and was negative for haemoglobin, myoglobin, and porphyrins. At this stage, the purple urine bag syndrome (PUBS) was suspected and an aliquot was sent to microbiology for culture and sensitivity. There was heavy growth of a coliform species identified as Providencia retgeri, an ammonia producing bacterium, adding support to the diagnosis of PUBS. This interesting phenomenon in which the urinary catheter of some elderly patients develops intense purple colouration is thought to be caused by indirubin formation. Various observers stated that indigo producing bacteria, which possess indoxyl sulphatase activity, usually bring about the decomposition of urinary indoxyl sulphate to indigotin and indirubin.

Several bacterial species have been reported in association with PUBS including Escherichia coli, Proteus mirabilis, Morganella morganii, Klebsiella pneumoniae, and Providencia stuartii.

Providencia retgeri has been isolated from the urine of our patient; to our knowledge this organism has not been reported previously in PUBS cases. Awareness and prompt identification of this syndrome by biochemistry and microbiology departments should avoid the provision of unnecessary tests on such urine samples.

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Fatal legionella pneumonia after fludarabine treatment in chronic lymphocytic leukaemia

Treatment of chronic lymphocytic leukaemia (CLL) with nucleoside analogues may cause T cell dysfunction, thereby predisposing to opportunistic infections in addition to bacterial infections as a result of neutropenia and humoral immune dysfunction. The following case provides an example of fatal legionella pneumonia arising in a 62 year old male non-smoker who had obtained a good partial response after completing four courses of fludarabine treatment for relapse of stage B CLL. He had been treated at diagnosis 2½ years ago with chlorambucil and oxpurinol but had never received corticosteroids. His general health had been good and he had continued in full time employment throughout. He developed “flu-like” symptoms just before returning to the UK from holiday in Spain and was prescribed co-amoxiclav by his general practitioner immediately on arrival. The next day he was admitted to hospital as a general medical team with lobar pneumonia and commenced treatment with ceftazidime. Clarithromycin, ciprofloxacin, and rifampicin were added soon after Legionella pneumophila was suspected (and later confirmed) by the culture organism, but he died two days later.

There are approximately 200 cases of legionnaire’s disease notified each year to the National Surveillance Scheme in England and Wales, of which half have had overseas travel, mainly to Spain or Greece. Immunosuppression, usually from corticosteroids or human immunodeficiency virus (HIV) infection, is known to predispose to infection and to increase mortality, as is
chronic pulmonary disease. Of the haemato-
logical diseases, the risk of legionella seems
highest in hairy cell leukaemia, possibly because of impairment of monocyte function,
and has been seen after treatment with 2-chloro-2-deoxyadenosine. Opportunistic in-
fec tions after treatment with fludarabine are
usually seen with advanced Rai stage, severe neutropenia, impaired renal function, or con-
comitant prednisolone treatment. Legionella
is uncommon in CLL, although it has been
described after treatment with fludarabine.
Treatment with co-trimoxazole is recom-
mended for prophylaxis against pneu-
omocystis in patients receiving nucleoside ana-
logues but is not clear from its use in HIV
infected patients whether this decreases the risk of legionella infection. With the increas-
ing use of fludarabine as a first line treatment,
the number of treated patients with CLL who are fit enough to consider travelling abroad
will probably increase. Because patients may
present for medical help to those unfamiliar
with immunosuppression after treatment with
nucleoside analogues, the carrying of an alert card specifying infective and transfusion
risks seems warranted.
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imal residual disease and CD4+/lymphocytopenia
4 Byrd JC, Hargis JB, Kester KE, et al. Opportun-
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ancies. A role for pneumocystis carinii pneu-
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tivity of Pneumocystis carinii prophylactic regi-

Is it useful to test for antibodies to
extractable nuclear antigens in the
presence of a negative antinuclear
antibody on Hep-2 cells?
Antinuclear antibody (ANA) negative lupus has long been recognised as a distinct entity affecting a small number of patients with sys-
temic lupus erythematous (SLE). Initial estimates of the prevalence of this entity (5% of patients with lupus) were based upon studies using rodent tissues as substrate for antinuclear antibody testing. The increasing use of human epithelial cell lines (Hep-2 cells), which have greater sensitivity for extractable nuclear antibodies (ENA), has meant that new patients with true ANA negative lupus are now rarely encountered.
Many immunology laboratories are faced with a substantial number of requests for antibodies to ENA and double stranded DNA, even in patients with negative ANA, on the grounds that patients with ANA negative lupus might go undetected. Using Hep-2 cells, we have attempted to define the preva-
lence of ANA negative, anti-ENA positive disease in a series of consecutive, unscreened serum samples.
Over a 12 month period, all laboratory requests for antibodies to ENA (antibodies to Sm, Ro, La, and ribonuclear protein) were scrutinised to determine the number of sam-
ples that had antibodies to ENA despite a negative ANA on Hep-2 cells. The notes of patients who were ANA negative, anti-ENA positive were examined to verify the clinical diagnosis.
During the 12 month study period, 7077 Hep-2 ANA samples were processed and 468 patients had an anti-ENA positive performance determined by a positive Hep-2 ANA. Of these 468 patients, nine were identified who were ANA negative, anti-ENA positive. Review of their clinical notes indicated that six of these nine patients had previously been ANA positive and were known to have lupus but were receiving immunosuppressive treatment. Only three patients were persistently ANA negative despite positive anti-Ro antibodies before treatment. Thus, the prevalence of anti-ENA positivity combined with a negative ANA was three out of 468 (0.6%). Because ANA negative lupus characteristi-
cally presents with cutaneous disease the clinical notes of 90 of the dermatology patients were reviewed. Of these seven of these nine patients had confirmed lupus erythematosus. Only one patient from the dermatology group had ANA negative, anti-Ro positive lupus before the commencement of immunosuppressive treatment.
Our finding of a low prevalence of anti-ENA positivity in the presence of a negative ANA on Hep-2 cells is in keeping with other studies in the literature. Manousakis et al found that only 0.4% of 243 Hep-2 negative patients with systemic autoimmune disease had positive anti-ENA antibodies1 and Homburger,2 reporting on the experience of the Mayo Clinic immuno-
pathology laboratory, stated that anti-ENA antibodies were unlikely to be positive in the presence of a negative ANA result on Hep-2 cells. However, neither of these studies included a clinical evaluation of the ANA negative, anti-ENA positive patients.
We recognise the difficulty in subject to potential sources of bias. The failure to scruti-

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1 Ahmed AR, Workman S. ANA negative sys-
3 Homburger HA. Cascade testing for autoanti-

Handling of renal biopsies: different approaches reflect a lack of evidence for what constitutes “best practice”
We read ACP Best Practice No 160 “Renal biopsy specimens” with interest.1 Dr Furness rightly avoids providing a list of specific pro-
cedures to follow because, “there is a need to assess each case on its merits, rather than following rigid rules”. It is clear from an audit of handling of renal biop-
sies in the UK, performed in 1999, that standard operative procedures were not followed and that many laboratories fall short of “best practice”. A probable reason for this is that there is very little hard evidence to support any specific recommendations. In the UK audit, a questionnaire was circulated to all members of the UK Renal Pathology Group and returns were received from 50% of the 54 laboratories represented. It is interesting to compare current practices with Dr Furness’s guidelines.
Dr Furness recommended that all speci-
cmens should be examined in the biopsy room for adequacy, using a dissecting microscope. However, in only 15% of units is this performed as routine. Failure to confirm the presence of renal cortex in the specimen would be expected to increase dramatically the proportion of inadequate biopsies. This was not the experience of the audit, however, where in 1994, as a result of staff shortages, the practice of sending an MLSO to attend every biopsy procedure was stopped.2 In Oxford, the histopathology labo-
atory is on a different site to the renal and transplant units; neither an MLSO nor a pathologist attends biopsies, as was once the case. Furthermore, what constitutes an ad-
dequate specimen is difficult to define and to some extent depends on the nature of the pathology. More tissue is required to detect focal than diffuse lesions.3 This has been demonstrated in renal allograft biopsies; in the validation study of the CCITT classifica-
tion of allograft pathology, those biopsies showing acute vascular rejection contained the diagnostic arteritic lesion in only one of two cores taken in 82% of cases.4 In the UK audit it was found that 20% of cores of renal tissue routinely taken varied from one to four in different centres. Dr Furness recommends that division of the specimen should be done within minutes of the biopsy being taken, to avoid artefactual ultrastruc-
tural changes. Although subtle subcellular changes do develop if fixation is delayed, for routine diagnostic electron microscopy (EM) rapidity of fixation is much less crucial. Formaldehyde fixation alone may produce

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excellent ultrastructural detail and is the fave-
\text{r}o\text{r} of choice for EM in some laboratories. Occasionally, we have received specimens that have been stored unfixed in transport gel for two days, and found preservation to be adequate for the purposes of diagnostic EM.\textsuperscript{1}

There is also variation in the immunohisto-
chemical techniques used when handling native renal biopsies. A frozen sample for immunofluorescence (IF) is taken routinely in 81\% of laboratories, the remaining 19\% rely entirely on immunoperoxidase (IP) stains performed on paraffin wax embedded sections. This, in part, reflects varying success in achieving reliable results with IF for immunoglobulins and complement. In the case of early transplant biopsies, only 30\% of laboratories routinely take tissue for EM. In those that do, it is often taken for research purposes rather than for patient management. Similarly, most laboratories (88\%) routinely take tissue for EM from native renal biopsies. Because some of the most common renal diseases, such as thin membrane nephropathy, can only be diagnosed ultrastructurally, those laboratories that do not take tissue for EM are certainly falling short of “minimum adequate practice”. Although it may be “best practice” to perform EM in all cases, it is probably sufficient to store this tissue as a resin block and only perform EM if the light microscopy is non-diagnostic.\textsuperscript{2} In many instances, EM will not influence patient management and the “minimum adequate practice” would, therefore, be to consider each case on its own merits and perform further investigations only if necessary. At present, EM does not have a clearly defined role in the assessment of early transplant biopsies and the UK audit found that only 38\% of laboratories routinely take tissue for EM from these specimens.

The choice of which special investigations are performed should, at least in part, be determined by our clinical colleagues. Nephrologists differ widely in how aggressive they are in investigating patients with asymptomatic renal disease, such as those presenting with microscopic haematuria detected at a routine health check. In some centres a biopsy will only be performed if it is likely to affect management of that patient; in others, biopsy practice is partly driven by research interests. Equally, the information required from the pathologist will depend on its potential clinical value. For example, providing a measure of the severity of chronic tubulointerstitial injury in a patient with membranous nephropathy is of far more value to the nephrologist than knowing the glomerular disease stage, as defined by ultrastructural appearances.

In the UK audit, the number of paraffin wax sections routinely cut for native renal biopsies varied greatly—from two sections on two slides to ten sections on 10 slides—again reflecting a lack of evidence base. In his article, Dr Furness indicated that the number of sections that should be cut and examined depends on “the nature of the question”. A renal biopsy standard operative procedure should, however, include examination of sufficient sections to enable the diagnosis of conditions in which the pathology is usually focal. In the case of primary focal segmental glomerulosclerosis, this is considerably in excess of two. For renal transplant biopsies, the Banff classification\textsuperscript{3} recommends that at least three haematoxylin and eosin (H&E) and three periodic acid Schiff or methenamine silver stained sections should be examined. The rationale behind this is that the diagnostic lesions of acute rejection—tubulitis and arteritis—are often focal. A recent review of transplant biopsies in Manchester concluded that one third of diagnoses of acute vascular rejection would be missed if only one, rather than three, H&E sections were examined (GP McCarthy, ISD Roberts, 2000, unpublished data).

All laboratories that handle renal biopsies should review their standard procedures, particularly if they do not conform to Dr Furness’s guidelines or “usual practice”, as indicated by the UK Renal Pathology Group audit. As the diagnostic questions asked by nephrologists change and new techniques emerge, procedures will inevitably require updating, but we will need to provide the evidence that any changes introduced are of demonstrable benefit to patient management.

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I am grateful for the opportunity to respond to the letter of Drs Roberts and Davies on the ACP Best Practice article “Renal biopsy specimens”\textsuperscript{1}, although they say very little with which I disagree. Most of their points of difference relate to “current practice” or “minimum adequate practice” rather than “best practice”. For example, the observation that electron microscopy (EM) can provide useful information even if fixation is delayed for a day or more is interesting and useful information. It supplements my observation that tissue from the paraffin wax block can be reprocessed for EM, but it does not alter the fact that best practice is to get the tissue fixed quickly!

The UK audit that they describe is a welcome update of a similar study that we performed in 1995,\textsuperscript{4} which influenced the development of the ACP guidelines. There is one small point where I think that Roberts and Davies misrepresent my suggestions. In their discussion of identifying and dividing the sample under a dissecting microscope, they imply that this has to be done by a pathologist or an MISO. We have found that nephrologists and radiologists can identify renal cortex and divide the biopsy appropriately with only minimal training. Again, rapid division is best practice; taking a bit longer is probably quite adequate in most circumstances, but (for example) in the future a delay will probably invalidate studies of gene expression.

Apart from these rather trivial quibbles I welcome Roberts and Davies’s contribution to the discussion.

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2 Roberts ISD, Tarpey P. Use of a transport gel for antigen preservation in unfixed renal biopsies. \textit{J Pathol} 1996;179:31A.
Book reviews


To my knowledge, this is the first comprehensive textbook dealing exclusively with the histological interpretation of needle core biopsy samples. To date, there have been one or two books edited by radiologists on needle core biopsy, which include chapters on histological interpretation. These by their nature have been restricted to basic principles.

The author of this book is of course well known to pathologists involved in breast disease reporting. He has numerous widely cited publications in peer review and in recent years has produced a major textbook on breast pathology based on his personal experience. Personally, I am a great admirer of his achievement, enthusiasm, and dedication to the field of breast pathology. For this reason, reading this book has been a pleasure.

First, I would point out that this book although dealing principally with needle core biopsy interpretation is also a distilled version of Rosen’s textbook of breast pathology. Diagnostic entities are described in succinct detail and are well referenced.

The book includes 31 chapters, the first seven dealing with normal anatomy and benign conditions, including one chapter on molluscum contagiosum, which forms a diagnostic group that appears to be gaining prominence, particularly in the American literature. Rosen recognises that most adenomyoepitheliomas are variants of intraduct papilloma and closely related to ductal adenoma and pleomorphic adenoma.

There is a substantial chapter on ductal hyperplasia and intraduct carcinoma, which covers in detail the difficulties of distinguishing the microfocal changes present in core biopsy. In this chapter the author recognises that there are some challenging forms of atypical ductal proliferation that exhibit prominent architectural and architectural changes, which form a diagnostic group that appears to be gaining prominence, particularly in the American literature. Rosen recognises that most adenomyoepitheliomas are variants of intraduct papilloma and closely related to ductal adenoma and pleomorphic adenoma.

There are 12 chapters devoted to specific types of breast carcinoma including a chapter on rare special types. Lobular neoplasia, mesenchymal lymphoid, haemopoietic tumours, and mastitis are dealt with in separate chapters. There is a useful chapter on the pathological effects of radiation and chemotherapy and a short chapter dealing with the pathology associated with needle procedures. This chapter is controversial because it recognises that epithelial displacement can occur as part of the biopsy procedure. Such displaced cells may rest in the stroma or be found as carcinomatous lymphovascular emboli or groups of tumour cells in the subcapsular space. Lymphoid cells may be trapped in the field of in situ carcinoma. The clinical relevance of such findings is uncertain and controversial. Dr Rosen sticks his colours to the mast and states that he would regard these as evidence of invasive carcinoma and metastatic carcinoma, respectively.

The book concludes with three chapters on technical laboratory aspects, image guided techniques for needle core biopsy sampling, and the impact of needle core biopsies on the clinical management of breast disease. These are valuable chapters that I personally feel would have been better placed at the beginning rather than the end of the book. The chapter on pathological examination is succinct but I would give a critical analysis of the coverage of strategies for reporting core biopsies. Although specific diagnostic problems are covered in the various chapters on diagnostic entities, an overview on strategy and handling diagnostic problems, with guidance on reporting and avoidance of pitfalls would have been useful.

All of the chapters are well illustrated in colour, although the colour balance could have been improved.

Until relatively recently there has been little textbooks on breast pathology. This position has changed and we have several major textbooks from authorities. Does this book merit purchase for your reporting room? In my view there are two good reasons for considering the purchase of this book. First, the widespread option of needle core biopsy for diagnostic sampling of breast lesions clearly is the door to a textbook, such as this, which considers many of the diagnostic problems that are now being encountered. Second, it serves as an updated and concise version of Rosen’s major textbook. Those of you reporting breast disease who have not purchased this textbook could “kill two birds with one stone” by acquiring a copy of this book. I will be placing my copy in our reporting room and suspect that it will spend more of its time open on the bench top rather than gathering dust on our library shelf.

I. O. ELLIS


In this book, an impressive amount of different molecular techniques that can be used in vascular research are described in great detail.

In summary, methods of molecular biology are described related to gene isolation, characterisation, expression, and transfer, and (of course) cell death.

In each chapter, the principle of the technique is explained (of course a basic knowledge of molecular biology is necessary). Subsequent materials and methods sections are described stepwise. Each chapter ends with notes that give extra clues for doing the experiments, and also functions as a trouble-shooter. Also helpful are the illustrations of the outcome of the described experiments, when successful. In the last chapter, gene transfer protocols are described, according to recent developments in this field.

Although all these methods can be used in pathological specimens, for general histopathology it is probably less suitable. Nevertheless it is an extremely interesting book and I recommend it strongly for researchers, including pathologists, who are doing research in the field of vascular disease.

H. W. M. NIJSENN


I thoroughly enjoyed reading this compact, stimulating, and refreshingly thought provoking book. It really puts cancer into an evolutionary context. It was pitched just right for me; as for most of the doctors, even those involved with cancers and leukaemias, my knowledge of evolution, history, epidemiology, and molecular biology is very focused on and tends to be limited to what affects my daily practice. So essentially, most of us are laymen. It is the sort of book that will be enjoyed by scientists, doctors, and many of those whose primary interests lie in the arts and the humanities, not to mention pathologists too.

This book, with its almost conversational tone, allows us all to follow the arguments in what are potentially impenetrable arenas with surprising ease. Some of that ease is probably an illusion, but a well written book and (of course) cell death.

Mel Greaves to lead us through the jungle of cancer is probably as important as truly understanding the implications and fine detail of the paths and surrounding countryside through which he takes us. Personally, one can end up at a conclusion really believing one understands how one got there, only on reflection to realise that one might need to read the argument all over again. Perhaps I should replace all the “ones” with “I”. It’s a seductive story, well told too—that’s what carried me along, rather than my own intrinsic abilities to understand. But I do confess I got almost as much pleasure rediscovering how I got to some of his destinations as I had when I first arrived.

M. M. REID

International Consultation on the Diagnosis of Noninvasive Urothelial Neoplasms

11–12 May 2001, University of Ancona School of Medicine, Torrette, Ancona, Italy

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Human Adverse Drug Reactions

30 May 2001, Royal College of Pathologists, London, UK

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Calendar of events

Full details of events to be included should be sent to Maggie Butler, Technical Editor JCP, The Cedars, 36 Queen Street, Castle Hedingham, Essex CO9 3HA, UK; email: maggiebutler@pilottree.prestel.co.uk

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Correction


In table 1 the time of the first sample should have been at −11, −7, and −4 days in patients 1, 2, and 3, respectively; similarly, in table 2 the time of the first sample should have been at −12, −6, −4, and −1 days in patients 1, 2, 3, and 4, respectively. The authors apologise for this oversight.
A case of purple urine bag syndrome associated with Providencia rettgeri

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