The display and negotiation of expertise and uncertainty in problem-based tutorials in medicine: a discourse analytic approach

Anne M. Storey

VOLUME 2

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Appendix A – Information Sheet and Consent Form
A Study of Communication Skills in Bedside Problem-Based Learning

You are invited to participate in a study of communication skills in Bedside Problem-based Learning (PBL). The purpose of the study is to investigate the communication skills that English as a Second Language students use in Bedside PBL in the clinical years at the University of Hong Kong.

The study is being conducted by Anne Storey (Pesep), Language Instructor at the English Centre at the University of Hong Kong, to meet the requirements for the degree of PhD under the supervision of Srikant Sarangi at Cardiff University (United Kingdom).

You will be asked to:
• fill out a questionnaire about Bedside Problem-based Learning (approx. 10 minutes);
• allow the bedside PBL sessions to be video taped;
• allow your notes from the patient history taking to be photocopied (optional);
• attend a short review session of approx 1 hour where you will be shown the video and asked questions about the tutorial (optional) and lunch will be provided.

Any information or personal details gathered in the course of the study are 100% confidential. No names will be used in any publication of the results. All participants will be identified with a pseudonym. Short segments of the video may be shown only during conference presentations or in the classroom, at the consent of each participant, and during that time, no personal details about the participants will be given. No one else but myself and my supervisors will have access to the data.

Benefits of the research will include a deeper understanding of how second language students manage the demands of clinical bedside PBL in the HKU context and an understanding of how students develop into medical professionals who are effective communicators.

If you wish to gain details of the results of the research, you may contact Anne Storey on Pesep at any stage.

Participation is voluntary and you are free to withdraw from further participation in the research within 6 months of the video taping, without having to give a reason and without adverse consequence.

I, __________________________ have read and understand the information above and any questions I have asked have been answered to my satisfaction. I agree to participate in this research, knowing that I can withdraw from further participation in the research at any time without consequence. I have been given a copy of this form to keep.

I ALSO do / do not give my consent to have my notes from patient history taking photocopied (please circle).
I ALSO do / do not give my consent to allow my participation to be seen at conferences and or in the classroom without my name being given or any other personal details (please circle).

Participant’s Name:

Participant’s Signature: ___________________________ Date:

Investigator’s Name:

Investigator’s Signature: ___________________________ Date:

The ethical aspects of this study have been approved by the University of Hong Kong Human Research Ethics Committee for Non-Clinical Faculties
Appendix B - Transcription symbols
Final rise, questioning

Slight rise (more is expected)

[Overlapping speech; first bracket indicates approximately where overlap begins in utterance of speaker holding floor and second bracket indicates beginning of overlap of speaker taking or trying to take the floor;]

: Lengthened segment

~ Fluctuation over one word

___ Extra prominence

CAPITAL LETTER increased volume

[ac] Non-lexical phenomena, both vocal and non-vocal which overlays the lexical stretch i.e. ac = accelerate; dc = decelerate; hi = high pitch; lo = low pitch

{ } Non-lexical phenomena, both vocal and non-vocal which interrupt the lexical stretch e.g. [laughing], [chairs scraping]

( . ) untimed short pause

(0.2 ) pause exceeding 2 seconds with approximation of length in seconds e.g. (0.5)

(did) transcriber’s guess at unclear word

(^^^) unclear utterance

Adapted from Celia Roberts, Sarangi (chapter from Language and Interaction: Discussing the state-of-the-art. Celia Roberts’ conventions based partly on Jefferson
Appendix C - Bedside PBL Tutorial Transcripts
Tutorial 1

Medicine Specialty PBL Session

5th Year Students: Keith, Ron (chair), Trudy, Jan, Sue, Fay, Visiting Student (VS), Student (unidentifiable)

Note: Patient is called Lam Siu An

1  Tutor   so: uh: this is the PBL so how many cases do we have
2  Sue     two
3  Sue     ) two
4  Tutor   OK so just uh: uh:: some housekeeping rules uh: well since this is a PBL tutorial, uh I would like to elect the chairperson first (.) OK so who are going to present the two cases (.)
5  Ron     I’m going to present one ]
6  Tutor   ] OK
7  Fay     ] and I am going to present the other one ]
8  Tutor   ] maybe the student who are presenting take turns, you know, to be the chairperson of this tutorial / OK so the time is running late, maybe we have to make a start (.)
9  Ron     {looking at notes} my patient um Lam Siu An is um a thirty-five year old man an ex-smoker and non-drinker and worked as a driver he had a good past health and complained of a three day history of right sided headache, and sudden onset of left sided weakness (.) uh: for the headache the onset was three days ago, right sided, it was a constant pain, he consulted the uh: the outpatient department of Princess Elizabeth Hospital, and diagnosis was made to be a cluster headache together with the eye pain um and uh lachrymation uh: rhinorrea (.) and the headache, uh but the headache persisted uh after the treatment and uh: together with a:: a:: blurring of vision on the right side, it was not accompanied by vomiting, there was no diurnal variation of the headache, it was not preceded by any aura, there was no pre dromal or post dromal symptoms, and there was no clear precipitating or relieving factors // (.) for the sudden onset of left sided weakness our
patient suggested that it was a sudden onset while he was sleeping, it occurs at two a.m. when the patient uh uh wanted to go uh for a toilet, (. and: the uh the weakness was not associated with loss of consciousness, there was no increasing of the right sided headache, there was no head injury, (. there was also a: a decreasing sensation on the left side, there was no chest pain, no fever: and there was no history of hypertension and diabetes, (. m~ so uh up to this point maybe we would like to discuss the um: clinical presentation and to uh postulate any differential diagnosis at this point, (. so ({ac}) in some way this patient presented with a three day history of right sided headache and: sudden onset of left sided weakness (.)

10 Fay can I ask a question, like during these three days what happened what has the condition progressed, like it’s deteriorating or it’s better?

11 Ron the headache was persistent despite the treatment / it persisted (.)

12 Fay and the weakness?

13 Ron it was sudden onset the night before admission / yes / (.)

14 Jan so for acute onset of weakness a vascular cause may be possible, (.)

15 Ron {( nodding)} mm mm

16 Sue {( nodding)} ) mm

17 Fay so the first the first differential diagnosis is stroke, (.)

18 Ron yes

19 Sue is there any risk factors with this patient, associated with stroke

20 Ron yes exactly this {((hi)} the point is that this patient is a young patient thirty five years old, um: the only risk factor we can identify is (. he is an ex-smoker, uh:: but he smoked very lightly (. and: there is no other risk factor, but on further e further eh questioning um our patient did volunteer the history that he went (. surfing uh two days before uh three days before the uh onset of uh left sided weakness (. and also um there was a family history which his sister had a history of moyamoya disease presented with seizure with sudden collapse found to have right side intra cerebral hemorrhage (. the patient’s sister was now thirty three years old, the accident of intra cerebral hemorrhage happened when his pu sister was thirty years old (. {(nodding)} um however for
the surfing uh activity he denied any injury to the neck or to the head (. )
{(nodding)}

21 Sue does he had hypertension or any (. )

22 Ron uh no he enjoyed good past health

23 Keith so the surfing occurred {(cough)} before or after the onset of the
headache,

24 Jan ] (. )

25 Ron ] before the onset of headache ]

26 Sue ] before

27 Ron so he went surfing three days ago

28 Trudy ] the first episode

29 Ron and there was onset of headache and: three days later during the night
he suddenly developed a left sided weakness

30 Fay apart from that is there any cerebral (speech) like any cranial nerve
involved, like the facial palsy, any things like that,]

31 Ron mm mm: uh: during that episode of sudden onset of left sided
weakness there were it was not associated with any right sided
weakness however, in the:: uh:: three years ago our patient had lower
motor neurone facial palsy and he was left with residual weakness and
now there was a complete lower motor neurone weakness on the left
side / (. )

32 Jan {(smiling)} so I just wonder whether the surf-ing have any relationship
with this episode of weakness, because the surfing was three days ago:
and

33 Ron ) yes

34 Jan it seems that there isn’t any: any specific things that happened during
that activity, (. )

35 Ron ) yes:

36 Jan ) and no injury

37 Ron ) yes that is what I was thinking

38 Trudy ) does he go surfing regularly or

39 Ron uh no just that those two days of activities (. ) it may be related and may
be not related (. )
Fay: I want to know more about the sister’s condition, so the sister was found to have: intra cerebral haemorrhage at age of thirty, any investigations done of like what was the cause of uh:

Students: moyamoya

Ron: moyamoya diagnosed to have moyamoya disease

Fay: that

Jan: moyamoya disease m-o-y-a

Keith: m-o-y-a

Fay: yeh but I don’t know what is this disease

Ron: moyamoya disease is uh:: (.) mm: was the uh as partial stenosis of the circle of Willis and on the digital subtraction scan there will be um: opening of collateral vessels uh to the brain, appear like a smoke like appearance uh supplying from the circle of Willis

Fay: how could that cause intracelluar haemorrhage?

Ron: I’m not particularly sure about this {(smiling & looking at Tutor)}

Fay: because you said that it’s the cause (.) cause like the collaterals is fragile vessels so (.)

Ron: {(smiling & looking at Tutor)} maybe

Fay: you’re not quite sure

Keith: actually for the surfing was there any travel history involved with that?

Ron: travel history,

Keith: or was it done in Hong Kong?

Ron: oh it was done in Hong Kong

Keith: right by surfing do you mean like wakeboarding or surfing, cos wakeboarding is much more higher much high impact kind of sports?

Ron: he went (. ) surfing / really surfing (.)

Trudy: did he have any headache in the past? (.)

Ron: no it was new onset after the activity

Jan: {(lo)} (. ) (. )

Ron: {(shaking head)} (. ) (. )

Tutor: so how severe was the headache?
mm: I didn’t particularly ask about the headache but it uh seems that it was constant pain, and troubled him so that he consulted outpatients OPD but with medication the headache persisted (.) it was not associated with any vomiting, there was no precipitating or relieving factors (.)

so he does take any medicine /

he did took medicine from uh the Princess Elizabeth Hospital but I did not identify uh what kind of medicine was that /

but before?

) was he on any long term medications?

no // our patient enjoyed good past health there was no long term medication (.)

was it a type of (pulse thing) any more than headache?

uh there was no diurnal variation of the headache so no (.)

so this episode must be a very severe pain: that: you know when the patient is seeking medical advice (.) you know, headache is a very common: complaint in the community well you know some studies show that it’s more than ten per cent (.) in the community who are troubled by headaches / so (.) for a male patient: well: not troubled by headache before well: (.) if a patient seeks medical attention well usually there may be something sinister going on OK, (.) so concerning the weakness / well do you think there is any significant functional impairment? (.)

yes

) do you find any significant (..functional impairment? {(lo)}

yes / on physical examination the uh muscle power on the left side u-upper limb was zero and the lower limb was one / (. that means it was just based on the history / because some patients are (.) figuring out you know their complaint you know (.) we don’t know whether this so-called weakness is genuine or not (.) so a functional history is very important

could he walk?

)can he walk yes very good {(lo)}
Ron: no he could not walk it was actually a right sided paralysis uh
Keith: ) left sided
Ron: ) left sided (.)
Tutor: so how about the upper limbs?
Ron: definite it was paralysis
Jan: he hav has to use the right upper limb to move his left upper limb
Keith: so the right side was five five
Ron: yes the right side was intact (.)
Keith: {nodding} how about the sensation?
Ron: there was a decreasing sensation on the left side mm {nodding} {Keith making gestures with arms to ask Ron to move on?} (.)
Fay: is there any bowel symptoms, urinary symptoms?
Tutor: ) yes very good
Ron: uhhh (. ) {looking at Jan} I did not ask about this (.)
Tutor: any incontinence, any accidents? )
Fay: ) (.)
Keith: ) did the patient have a diaper?
Ron: no the patient did not had {Keith nodding} (.)
Keith: how long has the patient been in hospital uh since:: you clerked him (.)
Ron: he was in the hospital since thirty-first of May so it was: two days
Fay: (. ah since you clerked him?
Ron: uh it was two or three days (. ) in hospital {nodding} (.)
Tutor: I think everyone knows about the approach in: making a neurological diagnosis (.)
Students: mm mm {nodding}
Tutor: so first of all you determine the site of the lesion and possibly with the history and the physical examination you can derive the uh the likely pathology (. ) can you determine you know just based on the history where is the site of the lesion you know supposing there is (genuine) weakness, sensory loss, (. ) whether you can locate whether the lesion is in the cortex, sub-cortex, brain stem, spinal cord? (. ) (. ) what kind of information you would like to seek?
what type of sensory loss was that?
what (kind of information) would you like to seek?
{looking at Trudy} ) loss of fine touch sensation, (.)
how about the pain?
(.) uh: (.) the pin prick sensation was reduced and the fine touch was reduced uh: so it was affecting both
and how about the reflex?
 uh reflex was:
) can we can we concentrate on the history first? OK and then we can focus on the physical examination (.)
uhuh (when) the patient (complained of) the lower motor neurone facial palsy like it suddenly progressed to being complete or,
) no it just
) it was a long time
) it was a few years ago that our patient had lower motor neurone facial palsy
) (.)
with a residual weakness but no recovery during these few years
was any diagnosis made in that time or was he told to be like (.). (.)
{looking at notes and shaking head}
but the patient said it’s now complete?
(.) the patient is still in complete
(.) facial palsy
( in complete) facial palsy now (.)
so any speech problem, any,
) no dysarthria (.) no dys uh dysphasia (.)
any swallowing problem?: (.)
I asked him whether he choked on food or drinks and he said he did not / (.)
so the patient remained: conscious all along?
yes yes there was no episode of loss of consciousness no head injury:
Ron: our patient complained of a blurring of vision on the right side together with the onset of headache but the left sided vision was normal. 

Tutor: mm mm no double vision?

Ron: no.

Tutor: ) no so can we localise the lesion based on the history?

Sue: this patient presented with um (simile) weakness of: uh left hemiparesis and hemiparesis and without any cranial nerve deficits from the history so we would think that the lesions would be above the brain stem /

Tutor: mm mm that’s fair enough mm mm (. can it could it be a spinal cord problem?

Sue: mm: if it is the spinal cord problem at least it should be at the cervical region that it would affects both upper limb and lower limb {ac} but then uh it should be uh both side um would be weak instead of hemiparesis-hemiparalysis (.)

Tutor: mm mm

Ron: and the sensory loss and the motor loss is on the same side (. of the:

Tutor: ) (. yes that’s right yes

Ron: )so it’s suggested that the lesion should be uh above the uh brain stem

Tutor: mm mm (. OK (. so any other relevant findings?

Ron: so on physical examination um I noticed that the patient {hi} had uh a complete lower motor neurone facial nerve palsy, as a result of the few years ago onset with a residual weakness (. and: there was no pallor, no clubbing,

Tutor: ) no no uh well my comment is well: if it is a very definite neurological case (. I think you should have some system in your presentation (. when (. a traditional one is you start with a high cerebral function and then (bring on then) what is sensory so it will help you to remember not to miss something important

Ron: {nodding} mm

Tutor: OK

Ron: our patient is uh alert and conscious and the GCS was fifteen over
fifteen, (.) and: (.) uh on general examination we can observe a lower motor neurone facial nerve palsy

Jan ) (.) {lo}
Ron on the right side (.) (.) (.) {lo}
Students (.) orientation {lo}
Ron {lo} conscious and alert, (.)
Jan ) orientation
Ron he was: he was oriented to time, place and person,
Tutor and that’s all for the high cerebral function, high mental function? (.)
Sue calculation and language
Ron mm I did not perform the mini mental state examination (.)
Tutor mm mm well mini mental state is a a screening tool you know, sometime well: we don’t we can’t remember all the item in the in the mmse OK (.) but (.) if you know about neurology and all what I’m what I’m what I mean is the high skill function what I mean is you can remember several domain you know cortical function and then you can ask some screening question I think that that’s that’s good enough (.) OK so what are the what are the {ac} domains in the high skill function? (.) so you you mentioned about the: orientation:, Ron attention
Tutor )attention:,
Keith ) concentration
Fay memory
Ron )memory
Tutor OK attention:, {writing}
Keith concentration:, short-term memory
Tutor memory, very good, what else? {writing}
Sue language
Tutor yes very good, language: (. ) {writing}
Sue calculation
Tutor ) mm mm
Keith )calculation
Tutor attention, calculation alright {writing} what else?

Trudy planning

Tutor yes very good planning {writing} executive functioning, what else? (.)

Trudy {lo} (.)(.)

Tutor mm mm (.). some abstract thinking, logical thinking mm mm and what else? (.)

Ron visual spatial

Tutor ) yes very good the visual perceptual function OK (.). so do you know ho- how to assess these various domains? (.)

Ron for the attention and calculation we can do the Serial Seven

Tutor ) yes very good mm mm

Ron ) for the memory we can divide it into short term memory, intermediate and long term memory: asking about uh uh digit span for the short term memory and attention and uh also ask him to remember a few items and ask him a few minutes later (.)

Tutor mm mm

Ron and ask him about the: recent news, and

Tutor mm

Ron ) historical events

Tutor OK (.). uh this will (tap) on the uh: the long term memory?

Ron yes

Tutor OK (.). a common thing is is: uh I think uh for this memory part you have to judge the the educational level of this patient OK / so for example for this very young patient well {ac} he must remember the date of the handover, OK? So you have to ask something relevant (.). so have you (.). uh: assessed the mem the memory function the visual perceptual function of this gentleman?

Ron I did not (.).

Tutor OK (.). mm mm (.). so this is important because well: if you didn’t assess these high mental functions you you don’t know whether there’s a cortical involvement or not OK,

Students {nodding}

Tutor ) so your assessment may be incomplete OK?
Trudy: can I ask a question when we say cortical sign what exactly

Tutor: that that’s (.) some (.) OK (.) there’s also some uh uh uh visual uh visual field defect, you know, language, these are the some of the cortical signs, OK?

Students: {nodding}

Trudy: and the perceptual function, (.) OK? (.) so you have missed all this high mental function assessment OK, so how about the cranial nerve? (.)

Ron: um except ) the cranial nerve seven

Tutor: ) so how about the speech? speech is the ver uh uh

Ron: uh there is no dysarthria no dysphasia

Tutor: mm so you don’t have any difficulty in communicating with him

Ron: {nodding}

Tutor: OK (.) so apparently the language function is intact

Ron: mm

Tutor: yeh (.) can you go on to the cranial nerve (.) OK

Ron: um: except the cranial nerve seven on the right side other cranial nerves was intact uh: including

Tutor: ) so do you think it’s a new lesion or:, (.)

Ron: ) um: our patient suggested that it was uh: a residual weakness a few years ago

Tutor: so how can you tell whether it’s an old lesion or a new lesion? (.)

Ron: uh we can assess the: muscle: if there is any muscle atrophy:,

Tutor: so you don’t know whether it’s uh due to a complete or inc o o or so-called you know, Bell’s palsy (.) recovery, you don’t know (.) so any tricks, (.) that you can differentiate whether it’s a (.) an old lesion or a new lesion? (.) a simple way is to ask the patient to produce some old photo for you (.) OK? or you can also you can inspect the ID card, you know, you know when the ID card was taken, issued and then you can compare about it you know for the so-called facial palsy is taken well before the the the the you know the ID card was issued OK you can s-compare the present facial features and the present facial features (.)

Ron: so I did not compare uh so I do not know it was an old lesion or a new
lesion (. ) uh: other cranial nerves was intact, uh: (. ) however I noticed that there was uh: right-sided miosis, partial ptosis which is suggestive of a right side Horner’s syndrome, (. )

211 Tutor mm mm (. )

212 Ron and the cerebellar signs was normal, the muscle power on the right side was intact, it was zero on the left side of right the right upper limb, uh left upper limb, and uh grade one on the left lower limb/ the reflexes were symmetrical and normal, the plantar re:flex was (. )

213 Tutor so-sorry where where’s the which side did the patient have Horner’s syndrome?

214 Ron on the right side/

215 Tutor {lo} right side OK (. ) OK

216 Ron and: the reflexes was symmetrical and normal and there was an up-going plantar reflex on the left side suggesting upper motor neurone lesion on the left side (. ) on the cardiovascular examination there was no murmur, no carotid bruit (. )

217 Tutor mm mm

218 Ron (. ) {nodding towards Tutor} (. ) yes that was about all that I can find from the

219 Tutor ) so other systems were unremarkable? (. )

220 Ron yes / (. ) mm so in

221 Tutor ) (^^^)

222 Ron summary our patient has got a left sided lower motor neurone facial palsy, and a right-sided uh: uh suspected Horner’s syndrome, and a left-sided uh: haemi:plegia with an out going plantar reflex, cerebellum and cranium was intact (. )

223 Keith sensation? (. )

224 Tutor ) sensation?

225 Ron sensation was decreased on the left side uh both the pin prick sensation and the plantar sensation (. )

226 Trudy did the patient say whether the ptosis was (. )?

227 Ron our patient did not actually notice the ptosis (. )

228 Tutor so it’s jus it was just a partial ptosis not a complete ptosis?
Ron: no. Actually the partial ptosis I had noticed from the case notes and I only noticed the right sided miosis yes. So um what are the uh uh how can we correlate the physical finding with the history in this patient?

Tutor: So first question is: get the answer you know the question where is the site of the lesion.

Jan: So suppose the high mental function is alright: then it is something sub-cortical, I guess, because there’s uh due to the distribution of the weakness and the sensation on the same side so we have mentioned that it’s like need to be above: the spinal cord since cranial nerves are intact so it should be above the brain stem so:

Tutor: mm mm

Jan: and because sensory is also involved so it’s not in the internal capsules so I guess it’s somewhere sub-cortical: (.) shrugs

Trudy: sorry can I ask like do we call uh: like motor weakness a cortical sign, (. ) because the motor cortex is part of the cortex

Tutor: no.

Sue: but I’m not sure how does the Horner’s related to the: sub-cortical lesion.

Keith: were there any marks of sweating on the face? did the patient notice any?

Ron: no. I notice: uh from the case notes that there is anhidrosis on the right side

Jan: uhhh (laughs) how can you feel anhidrosis in the hospital air-conditioning environment?

Ron: mm so I only observe it on the case notes.

Keith: did you ask the patient afterwards?

Ron: (shakes head).

Sue: ) but it’s difficult to notice that

Jan: ) you

Sue: you don’t even sweat in the uh hospital

Keith: but the Horner’s could have occurred previously?

Ron: yes it’s possible.
Jan (laughs)

Keith so it may not be the stroke that caused the Horner’s syndrome

Ron (nodding) yes

Jan ) (laughing) ohh

Ron (shrugs)

Keith ) (...) (...)

Tutor mm so

Ron ) I also do not know how to correlate the Horner’s with uh the this clinical picture

Tutor ) mm (general nodding) well you have a good learning objective (.) what are the different causes of Horner’s syndrome, (.) you know (.) so where where is the site of the lesion if a person has Horner’s syndrome( )

Ron upper (...)

Tutor ) if the autonomic system is compromised

Students ) sympathetic ganglion

Tutor mm mm (.) you know the sympathetic system supplying the the the: where does this locate do you know?

Trudy t1 to L2

Tutor mm mm (...)

Ron it’s in the sympathetic trunk

Tutor ) yes

Ron (...) column and in this case the Horner’s syndrome is suggested for cervical sympathetic trunk (.) affected

Tutor mm mm (...)

Ron and it can be due to compression, ischaemia, vasculitis,

Tutor mm mm

Ron different causes

Tutor mm mm (...)

Ron so actually in this case

Tutor ) so you don’t think the facial palsy is relevant for this case? (...)

Ron mmm: I don’t think so
Keith mm mm (.).

multiple neurological lesions occurring,

Ron nn?

Jan nn?

Keith multiple neurological lesions could it be (possibly) multiple sclerosis?

Jan oh:

Ron I guess multiple sclerosis would be uh progressive onset rather than: the patient come uh sudden onset complete paralysis

Keith (.)

Jan ) not that acute not that acute

Keith how acute was the sudden left side weakness at 2 a.m.?

Ron yes our patient can tell the exact time when he felt the weakness

Keith does that mean he was woken up by it or he was still asleep ]

Ron ] he was woken up. he was want he was going to the toilet, and he found he had a weakness he found he cannot walk ]

Jan ] mm mm (.)

Keith this mean he couldn’t walk but he also how about his upper limbs?

Ron mm

Keith muscle (. ) function

Ron there was also paralyis of the left upper limb (nodding) so it’s a total left body paralysis with a sudden onset (. ) actually I want to ask if there is a stroke in a internal capsule can the sensory be affected?

Tutor yes possibly

Ron yes ]

Jan but isn’t it that the sensory fibres not really directly passing through the internal capsule?

Tutor well you know the internal capsule is not a single (. ) you know some disease may affect the the anterior or posterior you know affecting the the whole internal capsule (. ) so it’s a it’s more classical in a person with a very dense you know motor and sensory loss without any (clinical sign) or high cerebral function deficit, it’s usually due to a sub-cortical or internal capsule lesion because the fibre (track) are densely packed in this area OK?
so is it true that it is very difficult to differentiate between sub-cortical lesion and internal capsule lesion? (.)

well the internal capsule is a sub-cortical lesion

oh I see ]

OK (. ) so mind you know the history and physical examination just give you a clue you know the: may not sometime may not hundred per cent give you you know the the: can tell you the site of the lesion but it can approximately indicate where is the lesion. OK? (. ) so uh you’re based on based on this account you know the where is the most likely site of the pathology? do you know (. )

I would think it is uh the site of lesion would be uh: the sub-cortical region on the right side affecting the mid cerebral artery region (that’s it)

yes OK it’s possible (. ) (nodding)

so shall I go on with the investigations after the admission?

mm mm

so:

] so any (vascular risk) factors for this gentleman, patient was ]

] stroke (. ) you know in this age group it’s not that common ]

] (nodding) he was a young patient uh: the only risk factor we can identify that he was an ex-smoker and the uh: possibly the trauma from the activity

mm mm that’s possible (. ) especially because of the Horner’s syndrome (. ) OK

(.) do you mean that um: Horner’s syndrome can arise from uh: a neck trauma injury?

yes possible

ss

because in: in the (pure) sub-cortical region we don’t expect the patient would have Horner’s syndrome, it’s a bit unusual in this case

(. ) (nodding) (. ) so from the investigations,]


316 Tutor yes

317 Ron {reading from notes} after admission, from the complete blood count uh: our patient had a thalassaemia trait (ac) as evidenced by decrease in MCV MCH increase in red blood cell count, increase in red cell distribution, with a normal haemoglobin of thirteen/ the random glucose was seven, which is not diagnostic of diabetes, uh: the CK was normal, uh: a hundred and thirty five, uh: the ECG and clotting were all normal, from the CT brain uh revealed a right-sided infarct on the MCA region, there is uh hypodensity on the right leucentiform nucleus / from the MRI brain it showed an acute ischaemia of the MCA region / there was loss of signal on the right (.) and cavernous intern internal carotid artery and the right middle cerebral artery / (.) the anterior cerebral artery and the posterior cerebral artery were patent, uh MRI of the neck showed occlusion of the right sided cerebral internal carotid artery ]

318 Tutor ] mm

319 Ron there was uh: the occlusion was one cm from the carotid bifurcation and: the radiologist suggested it was]

320 Tutor ] infarct {laughing}

321 Ron yeh su suggested a possible thrombosis from an acute dissection of the internal carotid artery, so the impression was uh: an MCA infarct as a result of right sided carotid dissection /

322 Tutor OK very good

323 Ron so the management was uh uh: a close neurological observation mm and it’s to repeat the CT if there is any decrease in GCS or in uh: signs of increased intracranial pressure, and specific treatment for the carotid dissection uh: low molecular weight heparin and warfarin was offered were offered / (.) so in summary our patient had uh dissection of the right sided internal carotid artery resulting in, a mid cer a middle cerebral artery infarct, and possibly also resulting from the right sided Horner’s syndrome/

324 Tutor mm very good (.) ss learning objectives this case?

325 Ron so I think the learning objectives in this case is the causes of Horner’s syndrome, and to know the anatomy of the sympathetic trunk (.)
Tutor: OK very good
Ron: and: (.)
Tutor: young stroke
Ron: causes of stroke in young patient and uh what should we need to look out for
Tutor: ) yes
Ron: in patients presenting with this history (. ) some uh we can discuss now young patient with stroke / we can discuss the causes of it
Tutor: mm well as you’ve (. ) OK you can reserve for the the: later part of the tutorial
Ron: OK
Tutor: or just a follow up question, (. ) is OK
Ron: OK (.)
Fay: so I will uh I will uh deal with the next patient
Tutor: (. ) do you know the the: causes of ischaemic stroke you know in young patient
Fay: (. ) deficiency
Tutor: mm mm
Fay: mm (.)
Sue: it could be cardio (.)
Jan: (.)
Tutor: yes very good
Ron: vasculitic changes
Tutor: so you have checked the VSL, this is normal?
Ron: did not notice
Fay: uh could I ask a question, if it’s a vasculitic type of stroke will it be like only affect one side, because the vasculitic lesion should be
Tutor: ) yes it’s possible it’s possible
Fay: OK
Tutor: some vessels are more affected than the other (. ) do you know the incidence of this kind of this sort of carotid artery dissection in uh in young ischaemic stroke patients (. ) I think it’s now increasingly
recognised to be a more prevalent condition why I think in some studies it’s almost ten to twenty per cent. because of this advance in the imaging technique you have more of this uh entity so back to this patient OK do you think the uh so-called surfing you know very vigorous exercise before this incident is relevant?

Ron

I think it’s possible to be relevant because of the trauma to the neck

Tutor

uh huh

Ron

and actually the the case MO has asked for any uh traumatic history or any neck manipulation

Tutor

yes very good

Ron

(.) mm mm and the patient denied any history]

Tutor

] we are very near to the border you know / people are you know]

Ron

] I didn’t actually ask ]

Tutor

] people going across the border go to Shenzhen and have some manipulation some massage mm mm we do see a few cases you know either of carotid or (. ) dissection / I think (. ) dissection is a bit more a bit more common due to this sort of manipulation (. ) OK very good / I think it’s a very interesting case (. ) OK so we can proceed to the second case

Fay

so our patient is Mr Yeung a forty-nine year old gentleman working as a security guard, a chronic smoker non-drinker, presented one month ago with a sudden onset of left sided weakness and slurring of speech while gambling uh playing cards / so for history of uh presenting illness uh: patient was found to have hypertension, and uh diabetes two years ago, uh but he def defaulted follow up and he did not on uh was not on any medication / patient had a history of nocturia for uh usually two times uh a night uh polydipsia, polyuria, weight loss of fifteen pounds over the last two years despite good appetite / there’s also (. ) urine and haematuria which may point to the fact that the diabetes is not well-controlled, uh so uh the before admission the patient uh has bilateral ankle oedema (to the shins) there was no shortness of breath no orthopnea and no uh paroxysmal nocturnal dyspnea / patient uh: presented with left sided weakness and slurring of speech and was
brought uh to the A and E by ambulance and patient denied uh denied any loss of consciousness nor had trauma before the uh incident (.) uh in a CT a CT scan uh a CT brain in the A and E was found uh the patient has a haemorrhagic stroke of the right side (.) for part]

360 Tutor ] so we lost the joy of making a diagnosis
361 Fay oh sorry oops (.) so: yep (.) (laughing) shall I continue (laughing) the presentation?
362 Tutor mm mm
363 Fay so uh past medical history the patient did not have any hyperlipidaemia, uh ]
364 Tutor ] so just (recite for everybody) OK
365 Fay mm mm
366 Tutor uh: the: presentation of a long case / I think first of all you have to delineate focus yourself whether it’s a diagnostic problem or management problem OK / so (.) diagnostic problem normally we don’t give the: you know the diagnosis right at the beginning you know (.) you paint the picture and then you know try to leave the (.) tests at the end OK so we lost all the joy of making a diagnosis and you can’t arouse the interest of the examiner OK
367 Fay {nodding}
368 Tutor so what what’s your focus / it’s a diagnostic problem or a management problem (.)
369 Fay I think for this patient it’s more on the management side
370 Tutor OK let’s find out
371 Fay mm mm because the diagnosis was uh pretty obvious ]
372 Tutor ] straightforward yes
373 Fay the patient has uh uh cardiovascular risk factors ]
374 Tutor ]mm mm
375 Fay which were not well-controlled and uh: the sudden loss (.) also pointing to vascular causes
376 Tutor mm mm (.)
377 Fay and also the typical half body involvement is also typical cereb of a cereb uh above brain stem stroke
378 Tutor  yes that’s fair enough / OK well with this sort of background the psychosocial history is will be more will be you know also of great importance

379 Fay  mm mm

380 Tutor  so in your in your presentation you should also elaborate on that

381 Fay  mm mm

382 Sue  I’m sorry ]

383 Ron  ] sorry did you mention that the patient had ) a slurring of speech ]

384 Sue  ] a slurring of speech

385 Fay  yes

386 Sue  so it’s not above brain stem but rather at the brain stem or maybe cerebellar

387 Tutor  OK maybe we can forget about the CT results OK (Fay laughing)

388 Sue  ] yes

389 Tutor  so back to the basic OK .)

390 Fay  well:

391 Tutor  ] elaborate the history for us (.)

392 Fay  the slurring of speech was uh together with the onset of the left side weaknesses so it’s also yeh it occurs together

393 Sue  is there any other cranial nerve deficits ]

394 Fay  ] yes uh:

395 Sue  ] symptoms

396 Fay  symptoms

397 Sue  diploea, dysphasia:

398 Fay  there’s dysarthria, dysarthria uh (.)

399 Tutor  so how come the patient complained of dysarthria

400 Fay  slurring of speech

401 Tutor  OK

402 Sue  how about double vision?

403 Fay  um: no / no double vision

404 Sue  any choking?

405 Fay  no
any facial asymmetry? (.)

mm: (.). yes yes I think so (.). there’s right side upper motor uh neuron
type upper motor neuron lesion type so it’s right lower facial palsy (.).

you mean left

right or left

right

you mean ] uh

] the problem is on the left side but the facial palsy is on the right side

so, did the patient complain of facial palsy?

no he did not complain it was just on examination

OK ]

diplopia ]

so uh in your presentation you should focus on the history first OK

and don’t say don’t mix up the history and the physical findings OK

{whispering}

I mean for symptom-wise he just had slurring of speech and weakness,

which are ]

]you know slurring of speech is a relatively non-specific term (.).

mm mm (.).

usually due to dysarthria but sometimes it may be due to dysphasia (.).

so how can you know? (.). can you gather more information from the

history whether it’s due to a dysphasic problem or: (.).

I’m not quite sure ]

] if the content is understandable

he’s uh communicable

what kind of patient can he may have difficulty in expressing himself

you know

no he’s communicable

any evidence of so-called of finding difficulty

no he’s (.).)

(or just you know)

he’s now communicable ]
Trudy: so does he he’s recovered now,

Sue: you mean the slurring of speech has got 

Fay: no the slurring of speech is still remain, we talked to him (.) and he still have dysarthria but he’s well understanding what we’re asking him, and he can also respond well / (. ) mm:

Tutor: you know this slurring of speech is not very localising you know in a sense, OK, so we need more information (.) besides the slurring of speech any diff any difficulty expressing himself or understanding others

Fay: while we are talking to him, no / he has no problem in talking to us and we did not notice him having any problem with any words]

Tutor: OK

Fay: ] and no delays in responding to us (. )

Ron: is there any problem with the tongue control?

Fay: uh:: symptom-wise, no / (. )

Tutor: so on presentation any other uh swallowing problem

Fay: mm: (. ) no/ I’m not quite sure because I did not ask uh him whether he had a swallowing problem right at symptom onset so I’m not sure about that, but now he can (. )

Sue: another differential for slurring of speech can be due to cerebellar problems so did the patient complain of any dizziness, or (. ) tremor

END OF DISC {missed approx. 2 minutes of talk while changing tape}

Tutor: (so you) it’s it’s already one month past the uh from the occurrence, how how did the symptoms and signs progress

Fay: the left side weakness remains (. )

Tutor: mm mm ]

Fay: ] and the (. ) I’m not quite sure about the slurring of speech (. )

Tutor: so no sign of recovery

Fay: uh

Tutor: not even a trace ]

Fay: ] for the left side weakness (. ) I mean for while we are testing (. ) for the physical examination we are not too convinced about that but the patient did complain that the left side weakness is still a problem (. ) for
me I didn’t]

Tutor ] mm mm

Fay (.) speaking (.)

Tutor so all of you are convinced that the patient had a stroke (.) or what (.) just based on the history (.) so you know stroke is (.) take the you know very acute onset and then a trough and gradually recover mm do you think this gentleman followed this usual pattern

Fay I think so

Tutor mm mm (.) so you think so but you said there is no recovery

Fay I’m not quite sure about the slurring of speech but the left side weakness the patient still complained that it is present but actually when we were doing the physical examination it was only 5 minus

Tutor mm so it’s just just very mild you know uh uh uh (degree of mobility)

Fay ] mm mm yes

Tutor but he can probably function (.) (.) somehow can the patient walk? Can the patient go to the toilet himself? Can the patient use a spoon and chopsticks you know (.)

Fay {turning to Keith } we did not ask about that, sorry

Tutor (. mm (. any other relevant history you want to ask

Fay uh:: (.) so the patient has no hyperlipidaemia, he’s a chronic schizophrenic patient follow up at Victoria Hospital / only had an appendicectomy appendectomy done in the sixties / so he is not, is only on anti-hyp uh anti-psychotics and his uh schizo his schizophrenia is well-controlled, no uh: uh hallucinations or the usual symptoms

Tutor mm mm

Fay um: family history uh no uh ]

Tutor ] but the schizophrenia history is very important OK

Fay mm

Tutor do you know why? (.)

Keith it affects the pre morbid uh: idio (.)

Tutor yes what else

Sue affects his compliance too

Tutor ] yes very good (. so what’s your (. what’s your name
OK Fay well you haven’t mentioned about the poor control of diabetes. How about the blood pressure control?

uh: he was told to be hypertensive but he was not on any medication and he defaulted follow up.

(.) he said that at that time he was on the high side but then he was not uh: he did not need to put on any medication but since then he had no follow up.

(.) his family history uh (lots of) cardiovascular family history uh the father and mother died of uh lung cancer in their old age, social history he is a security guard, uh: single and lives alone.

is he a smoker?

uh: chronic smoker, non-drinker.

OK so apparently the social support of this gentleman is not that good?

mm (.). when we clerk him.

] so we expect some discharge problem.

] we saw that uh: he’s uh:]

] this gentleman.

uh his sister and his cousin was there to take care of him and actually they are quite concerned about his situation, and they know quite a lot about him.

mm mm (.). OK anything from the physical examination?

so for the physical examination the patient is uh alert and well communicable uh his uh GCS is uh (full), we did not do the MMSE examination.

oh forget about the MMSE I can’t remember it I can’t remember the MMSE there are about twenty items with the MMSE how can you
remember OK? (.) but you can remember the six domains OK, it’s more easy (.). OK

494 Fay uh:: we performed the cranial nerve examination, uh:: the only (.). we identify is the nystagmus to the first phase to the left side (.). there is uh: (.).

495 Jan (whispering to Ron)

496 Fay decrease (.). as for as for the trigeminal nerve we are not quite sure because there is decrease in left facial sensation but apparently when we feel the masseter muscle and the temporalis muscle there is dec uh there is decreased muscle bulk on the ~right side (.). so we’re not quite sure (what was this) (laughter) we have repeated and were worried by about the: uh: right side uh masseter and temporalis uh decreased muscle bulk but the patient apparently said that there’s a left facial uh decreased left facial sensation/ we can try to see afterwards because the patient is outside (.). there’s a right lower facial palsy ]

497 Tutor ] but what sense of modality you have checked for this gentleman?

498 Fay uh touch touch

499 Tutor just light touch?

500 Fay yes

501 Tutor what about (pinprick)? (.).

502 Fay we didn’t do that (looking at K) I’m not quite sure

503 Keith I don’t think so

504 Fay no we just did light touch

505 Tutor mm mm (.). temperature (.). you can you can (.). you can also check the temperature (.).

506 Fay we have not done that

507 Tutor OK (.).

508 Fay uh:: there’s ]

509 Tutor ] how about the corneal reflex (.).

510 Keith (.).

511 Fay no also we did not do that (.). but actually can we do that because I mean like in an exam ]

512 Tutor ] usually
we should not do anything that can cause unpleasant to the patient
so what is your comment (.)
only with touch I think
maybe not done in every single patient but uh in the case that you have
some trigeminal nerve problem ]

J(^^^)
we can do that then and uh for the tongue we are also not quite sure
(laughter) because the patient cannot move to either side so uh we are a
little bit frustrated about the tongue {laughter}

that’s OK again you can you can go back to your basic neuroanatomy
(.) so you’ve if the tongue cannot move (.). mm (.). so where is the
lesion?

brain stem ]

] brain stem

if it is the brain stem it should be bilateral (.). so do you think other (.)
signs are compatible with bilateral (.). involvement? (.)

mm (.)

bilateral sub-cortic bilateral cortical bilateral: so do you think it’s
likely? mm mm (.)

we are not quite sure about that
(.)
because the (.). both are bilateral
(neurone)

it can protrude right

yes it can protrude ]

] protrude (but not left right) ]

] yes yes

then that should (.). ]

] so he’s not (.).

he can understand other instructions well so I don’t think it’s that he has
a it’s a problem in understanding instructions (.)

but how will it protrude
I think if he can protrude then there is no problem with the uh: ]

] power of the tongue ]

] involvement

it’s just some coordination

the coordination between you and his tongue {laughter}

but he has no problem understanding other instructions (.)

but in case he can protrude the tongue and no deviation so the power should be OK on both sides

mm mm

(^^^)

is there any cerebellar signs, because he has uh ]

] a nystagmus ]

] nystagmus and (dys)coordination of tongue

there is (pass pointing)

oh

how about (.)

which side (. ]

] when I did it he was OK there was no (pass pointing)

how about other cerebellar sign ]

] maybe some tremor (. but generally he was OK ]

] I was not (.)

so there is no cerebellar signs except nystagmus?

yes (.)

and you know nystagmus is not a ~pure cerebellar sign

mm mm

brain stem )

) yes it can be a brain stem or (cerebral) problem or peripheral problem (. so how can you differentiate whether it’s a central, a peripheral

usually if it’s central the fast phase is towards the lesion side

mm mm

so this nystagmus we think that it’s to the left side (. which is not the lesion side
Tutor: mm mm

Fay: {laughs} I’m not quite sure about the (RV)

Tutor: (nystagmus) is a very difficult topic, you know I think it’s a very confusing uh you know when you are just faced with a patient with just purely nystagmus I don’t think you can localise the site of the lesion (.).

In my experience (.), I think you can only tell whether it’s central, peripheral, or what, you know (.), unless there’s some very specific type of the nystagmus, for example the toxic nystagmus, OK so you can more localise it, otherwise you can’t tell, differentiate whether it’s a cerebellar:, a vestibular problem (.), it’s very difficult (.). so uh you found nystagmus OK, so any vertical component or any (.). component?

Fay: I could not identify any]

Tutor: ] just (.).

Fay: (.). yeh (.).

Tutor: any sign of any vertigo

Fay: no not that the patient complained of

Tutor: mm OK (.), so it’s uh: quite classical for some central cause (.). usually the patient didn’t complain much about vertigo you know but you found very clear cut clinical signs (.), and another problem is we don’t know whether this gentleman have genuine (pager beeping) genuine nerve cause trigeminal problem (.). am I correct? (.). I think you should be more vigilant, more careful about your testing (.). you should check for every (common) reflex, general sensory modality (.). of the face, you know (.). the muscle, (.), masseter, temporalis, (.). so any other?

Fay: so (.). on examination of the limbs, uh for the upper limbs ]

Tutor: ] just focus on the cranial nerve

Fay: uh cranial nerve uh there’s no other abnormalities on that (.).

Tutor: it’s not a taxic type of nystagmus (.).

Fay: ] ataxic type, what is this so how it does it look like ]

Tutor: ] the abducting uh (AV) abducting is more (affected) (.).

Fay: I am not su ]

Tutor: ] maybe you should go back and read about it OK it’s a difficult topic / I think you have to take a few hours to master the uh you know the
different presentation of different type of nystagmus

583  Sue   
      (.)

584  Trudy (horizontal)

585  Tutor ] how about the uh facial nerve

586  Fay   uh: the patient noticed a right lower facial palsy (.)

587  Tutor mm mm (.)

588  Fay   signifies a upper cranial uh upper motor nerve ]

589  Tutor ] just the lower part of the face affected

590  Fay   yes

591  Tutor not the full not the whole face

592  Fay   yes

593  Tutor OK (.), so where is the site of the lesion?

594  Fay   (.)

595  Tutor how about the cranial (.), the tongue is alright the tongue is alright

596  Fay   the tongue we are not quite sure, as I said that ]

597  Tutor ] but apparently it’s OK (.)

598  Fay   there’s no fasciculation

599  Tutor (.), he has stuck out the tongue, and there’s no deviation, no wasting, no fasciculation

600  Fay   no fasciculation

601  Sue   what about his pulse

602  Fay   no weakness actually it’s so strong ]

603  Tutor ] so you should compare both sides ]

604  Keith ] yeh we did it was quite even

605  Fay   but as for me I think it (.)

606  Keith the patient subjectively said the ]

607  Fay   ] left side

608  Keith left side was weak but then when we did the testing of everything it just seemed: mu much equal (.)

609  Ron   (.)

610  Tutor that’s fair enough because uh it’s already one month after the acute onset (.), maybe the power: (.), have returned to normal, OK, (.), so that’s
why you know you are dealing with a patient who have with a very long history / the functional side is also important because you know everything you know go back to normal / you don’t know whether it’s a true account or not / whether the patient is telling you a true story (.)

Fay: uh: for the lower limbs uh: (.) the tones are normal, the power is: four: for both sides, and the: there is also decrease in the bilateral reflex (.) there is upgoing plantar for both sides(.) (laughter) we are very confused about the history so I would like to like us to go and see the patient together ]

Tutor: ] go to go to the patient and check
Fay: but we did quite a few times and really the two sides are upgoing so ]
Tutor: ] but not one side
Fay: not one side
Ron: is there any old stroke?
Fay: no, no history of previous stroke / it’s the first time ]
Tutor: ] so any possibility ]
Fay: ]we (. ) for many times but
Jan: is it just withdrawing rather than (. )
Fay: no it’s quite obvious is that the fings that the toes are (. ) back
Ron: is that a cord lesion
Fay: uh what ]
Ron: ] was that a cord lesions that (would make) uh bilateral ]
Fay: ] but he has no cranial nerve symptoms
Ron: oh
Fay: his cranial nerve symptoms are ]
Tutor: so ] you found a mixture of upper and lower motor neurone sign
Fay: I’m not quite sure so I ]
Tutor: ] OK so we can go to the patient and see:
Fay: yes I would like us to go and see the patient
Tutor: first you should check whether it’s due to withdrawal or: whether it’s a true one upgoing plantar response, OK, (.) I think it’s still possible you know in a patient with long-standing diabetes you know, you have to make sure there are upgoing, up up upper motor and lower motor site is
possible Ok / I think in medicine there are few entities of so-called of mixing upper and lower motor neurone lesion OK I think it’s frequently pointed out in MCQ questions I think you should revise and remember some of this OK (. ) OK (. ) OK how about the sensory? OK ( . ) OK ( . ) OK how about the sensory?

633 Fay uh the sensation is uh: is the same for both sides
634 Tutor so: normal?
635 Fay (noding)
636 Tutor on both sides (. ) so which sensory modality you have checked?
637 Fay uh: pinprick
638 Tutor just pinprick?
639 Fay mm:: yeh we should have ]
640 Keith ] and light touch
641 Fay yeh
642 Tutor pinprick and light touch
643 Fay we should do proprioception also
644 Tutor yes very good (. )
645 Fay ( . )
646 Tutor ) so which sense of modality do you think is important more important? ( . )
647 Fay proprioception?
648 Tutor yes: (. ) very good (. ) what’s the reason?
649 Fay because it affects the patient when he, when he can walk later, 
650 Tutor yes very good you know your subject (. ) yes what else you want to tell?
651 Fay I think for the history and physical exam (. )
652 Tutor so how about the cerebellar sign
653 Fay ( . ) ah (pointing to K) it’s OK
654 Keith ( . ) not present and uh: finger nose: (. ) there was some slight tremor but there was no pass pointing (. )
655 Tutor what about the heel shin you know the heel shin test
656 Fay (shaking head) (laughter)
657 Tutor so you just focused on the upper limb and forget about the upper limb ( . ) so you don’t bother whether the patient can walk or not {laughter}
(.) so how about the gait (.)

Fay  we did not ask the patient to go down (. I could not remember (.)
{laughter}

Tutor  so: having a system in approaching a neurological patient is very
important so I’ve always I always teach my patient in this format (.)
you know in your presentation in your assessment you should go from
you know the top to bottom the top to bottom approach/ sign of high
cerebral function, cranial nerve, motor sensory, coordination and gait
you know, and in your presentation you just follow similar format so
you will not miss out anything important/ I think this is very important
to get a pass you know {laughter}]

Jan  ] a pass

Tutor  you know remember all the all these points the format even though you
make some you know a few mistake I think we will let you let you pass
(.) if you forgot all your basics and you don’t have a good approach I
don’t think we can let you let you pass (. OK how about the general
examination the cardiovascular examination)

Fay  um no uh: no abnormality is noticed

Tutor  no abnormality ]

Fay  ] is detected (. no abnormality is detected

Tutor  so how about blood pressure?

Fay  I did not complete that

Tutor  I think in a long case you have to measure the blood pressure yourself
(.) OK so (pager beeping) so how about the apex any (. apex signifying
a cardiomegaly?

Fay  mm: ]

Tutor  ] (. further giving you hints that the blood pressure is not that good
probably (.)

Fay  as far as I can rec remember there was no uh ab uh abnormalities that I
could identify

Tutor  mm mm (. good (laughter) (. OK the diabetic control any any
evidence of diabetic problem?

Fay  symptom-wise he has (. he has polydipsia, polyuria, ]
Tutor: what I want is

Sue?: {whispering} physical findings

Tutor: from the physical findings, any complication of diabetes, (myelitis)

Fay: we did not look

Sue: fundus

Fay: at the fundus but we test the sensation(.)

Sue: urine urinalysis

Tutor: urine very good

Fay: we did not do the urine {laughter}

Jan: under (. ) pulses?

Fay: uh present (. )

Tutor: you mentioned the diminished reflex

Fay: mm mm , but it’s all: the reflexes are diminished I’m not sure if it’s quite normal for some patients they just don’t have so much reflexes

Tutor: have you performed the reinforcement test (. ) {laughter}

Fay: {shaking head}

Tutor: so you miss you missed a lot (. ) so the history account should be comprehensive and appropriate and the physical examination should be accurate OK and to the point OK

Fay: {nodding} (. )

Tutor: how about the sensory testing any:

Fay: we just did the pinprick but it’s uh equal on both sides both upper and lower limbs ]

Tutor: ] OK: as mentioned you have to assess the uh (both) for the proprioception because it’s important for the gait, stability (. ) how about the vibration sense

Fay: we did not test vibration

Tutor: so you suspect the patient having proven neuropathy (. ) you know which sensory modality would go first will go first?

Students: vision

Tutor: yes very good (. ) so all the questions are you know tailored to the specific aetiology in the mind it’s in your mind OK because of the time limitation well: you can have to select the most appropriate
examination (. ) OK (. ) I think detecting a (. ) neuropathy, the vibration sense and (the point discrimination) is most sensitive (. ) but we seldom perform the (discreet point discriminations) you know it’s a bit more you know it’s a bit difficult it’s more time-consuming (. ) usually we perform the vibration sense you know ]

697 Trudy ] (^^^^)
698 Tutor because patients have different neuropathy OK
699 Trudy do we test at the big toe and what are what we do
700 Tutor OK any idea from the (floor)
701 Sue I think only if the big toe vibration is lost ]
702 Jan ] lost ]
703 Sue ] yeh then you should go for more proximal ]
704 Jan ] (^^^^)
705 Tutor any reason for that?
706 Jan because peripheral neuropathy affect the longest nerve first
707 Tutor mm mm
708 Jan the length ]
709 Tutor ] yes except
710 Jan ] (. ) length dependent
711 Sue ] {Cantonese to Trudy} ]
712 Tutor ] what do you mean by length dependent
713 Jan so longer than earlier involvement
714 Tutor yes (. ) so have we completed our assessment?
715 Fay that’s all I have
716 Tutor OK
717 Jan shall we go and check
718 Fay yeh I I would like us to go and check this one (. )
719 Tutor well you mentioned about the management problem with this gentleman: (. ) I think a functional assessment is also important so up to this point now can the patient you know walk to the toilet you know (. ) get up and down from the bed, (. ) can the patient feed himself, dress himself?
he can feed himself but we did not ask him to walk.

and the patient is living alone there is no social support.

but his function is quite good he has a stable job.

he has well-controlled]

] that’s the question you know can the patient get uh go back to his previous occupation his previous job?

I think it depends whether he can walk uh: and whether: mm I don’t think speech is much of a problem it’s not much of a problem to affect his uh social functioning because whether he can walk is important.

how about his home environment do you think his home environment is conducive for his return.

then we have to ask an occupational therapist for an assessment.

well in an examination situation you can’t ask the occupational therapist to do the assessment for you (laughter).

and to ask the speech therapist to assess the speech for you.

I think a simple question is good enough since several simple questions

whether there’s a lift

what sort of pulsing: you know

any surrounding environment, whether there’s any slope, stairs, you know {Fay nodding} do the patient need to share toilet facilities, this sort of thing you know, simple but important details so the power of this gentleman is pretty good

but the limiting step is whether the patient’s stability is OK, whether the patient have sufficient ataxia, stability, to prevent him to go home or to prevent him from going back to his previous occupation OK these are some of the management issues that you should look into OK? I think that’s all we need to talk about for this you know for this second
case (. ) any further questions or learning objectives? We’ll go back and spend more time with (. )

740  Fay  is the uh progression of uh he hemorrhagic stroke and an ischaemic stroke the same because I was actually quite surprised that the patient could survive a hemorrhagic stroke and progressed that well I mean

741  Tutor  mm mm

742  Fay  hemorrhagic stroke is usually points to poor prognosis, higher mortality but the patient is actually quite well (. ) I mean if it’s a hemorrhagic stroke ]

743  Tutor  ] it’s a hemorrhagic stroke? (Students nodding)

744  Sue  how did you take a chance to look at the imaging? how ]

745  Fay  ] we saw the CT

746  Sue  how big is the hemorrhage and where is it?

747  Fay  it’s in the cerebral area but how big I cannot remember

748  Sue  mm cor]

749  Tutor  ] cerebral area?

750  Sue  cortex (. ) cortical (. )

751  Tutor  for brain stem?

752  Fay  brain stem

753  Tutor  which is which area do you ]

754  Fay  ] I don’t know  {general laughter}

755  Jan  let us go and see the patient and also the investigation ]

756  Fay  ] do you remember (to K)

757  Jan  {Cantonese}

758  Trudy  {Cantonese}

759  Sue  haih brainstem meih (. ) tunga

760  Fay  yeh the right (. ) lower (. ) facial (. ) weakness ]

761  Ron  ] (. ) upper

762  Jan  lower face )

763  Ron  ] ah lower face

764  Fay  right lower facial weakness ]

765  Sue  ] gam jeh brain stem ]
Fay] left side weakness

Tutor it’s still possible in a high relation about the pons you know it’s possible it’s still possible (Sue and Trudy whispering in Cantonese)
(. the facial supply is in the pons OK

Sue but where is the decussation of the

Trudy motor neurone

Tutor you mean

Sue ] is it

Tutor uh which which which nerve

Sue of the; seventh nerve

Tutor I think seven is in the pons

Ron (. {to Jan and then a Student replies to him while Jan asks the following question}

Sue (. )

Jan (. (. so the lesion for the left weakness is in the right uh right hemisphere? and then (. but then the upper motor neuron facial paralysis for the right face?

Tutor if it crossed then that area is affected / if it’s select if the same side is affected then should be above the the crossing of the facial nerve (.

Jan (^^^)

Trudy (. above ]

Ron ] the facial nerve (. ]

Tutor ] OK you should read about the new anatomy OK (. please take time to digest everything OK (.)

Sue {Cantonese}

Jan {Cantonese}

Sue haih ah )

Jan (. go dih

Sue haih ah ]

Tutor ] well localisation is always difficult

Students and tutor prepare to leave
Tutorial 2

Medicine Specialty PBL Session

5th Year Students: Keith, Fay, Jan, Ron, Sue, Trudy, Cathy and Visiting Student (VS)

1  Tutor  Why because I think we have quite a number of patients in the ward (.) and you are very busy

2  Jan  yesterday I have one but she just came in and left after a few hours

3  Tutor  well that kind of case is not really a good case because it’s uh: the admission is uh basically a scheduled admission (.) so we: already know what is the main problem (0.2) good (.) let’s start (0.2)

4  Ron  uh my patient a sixty-one year old)

5  Tutor  I ) would like you to just pre give me the uh: chief complaint first )

6  Ron  ) ok

7  Tutor  then we can discuss to see whether this is a good chief complaint or not

8  Ron  ok um our patient, sixty-one year old Miss Wong, uh:: a {ac} non-smoker non-drinker {dc} complained of acute onset bilateral lower limb progressive and ascending numbness for three days (.)

9  Tutor  excellent (.) so: {gesturing to Sue} what do you think?

10 Sue  um::)

11 Tutor  ) well if you let’s assume this is a good chief complaint, hearing this what will appear in your mind: and what questions would you like to ask, and can you have a list of differential diagnoses just based on this chief complaint? we should have one because when we hear a chief complaint a number of dx a number of things will jump into your mind (.) then detail after asking detailed questions and excluding other things then we can have a working diagnosis (.) yes?

12 Sue  um from the chief complaint we know that um uh the deficits mainly involve the sensory but I would still like to know whether the motor is involved (.) and um from the chief complaint we know that it’s involving the lower limbs, (.)

13 Tutor  ) what was the chief complaint again?
Ron: acute onset bilateral lower limb progressive and ascending numbness for three days.

Tutor: ) three days good

Sue: um:: it seems mainly involving the lower limbs but I would still like to know whether the upper limb is spared and um if it is involved the lower limb I would think more about uh: pathology in the spinal cord, um if it is involving the spinal cord I would like to know whether there is any uh sphincter disturbance, um so I um after these few questions I would uh think of some spinal cord problem or peripheral nerve problem.

Tutor: spinal cord problem. yes I agree with you we would not usually limit to the sensory part especially if it is bilateral. that means involvement is not that limited. therefore sparing the motor part is unlikely. if it only affects the sensory let’s assume if it only if it because it’s numbness not weakness right if it is motor involvement the patient will complain of weakness. yes what do you think Mr Lee?

Cathy: uh I’m wondering about onset of the numbness is it uh how acute is acute is there may be like a vascular cause say inflammatory cause so I’m wondering like if there were any systemic symptoms.

Tutor: well if it is a vascular cause it’s less likely to be progressive over the past three days. you know for vascular it’s usually sudden. when it happens it happens. it rarely progress over the next three days in ascending you know. that is less likely. infection cause you are thinking about: what kind of infection would you, are you worried about? yes Miss Fay?

Fay: mm:

Tutor: everybody gets a {smiling} chance to involve in the discussion.

Fay: it could be viral infection.

Tutor: yes it’s usually a viral infection so we are thinking about can this be: if it’s a viral infection, what kind of terms would we give to that?

Sue: myelitis.

Tutor: ) yes myelitis, myelitis is possible, yes? {turning to Jan} besides myelitis can this be just uh: some kind of uh:

Fay: it could be just some compression.
compression due to mechanical reason right, maybe just a prolapsed disc, (
) progressively increases severity, and numbness is usually the uh: well sensory deficit is usually the first presenting features ok? but of course if you have that kind of uh di differential diagnosis you will specifically ask

no we are still talking about the history not the investigations at this moment (. ) specifically ask

whether there’s any back pain (. ) um:

yes how does this happen yes maybe the patient can tell you she tried to pick up something (. ) from the ground, or when she was trying to lift a weight, that kind of thing (.0 so (. ) must be some triggering things (.0 and if we worry about infection, trans say it’s transverse myelitis, it’s usually preceded by:; (0.3)

yes then we will specifically ask what about ) (^^^)

right so yes just do you have do you have flu-like symptoms or common cold like symptoms, let’s just say one week ago or even within two weeks time (. ) or maybe viral infection can present as diarrhoea, gastro-enteritis, right? so these are the I think basically the differential diagnosis (0.2) because uh can brain lesion present like this (0.2) unlikely because it’s bilateral right unless you’re assuming it’s (^^^) on the top it’s a brain tumour a space-occupying lesion in the brain (. ) yes maybe but it’s less likely (. ) and this is a non-smoker, non-drinker, sixty year sixty-one year old lady

yes

so occupational risk (. ) ruled out, uh:: non-smoker the chances of malignancy is well {shrugging} probably less, should be less, and good (.0)these are the things probably that we have to discuss in detail during history-taking part right (. ) I hope you have asked all these
Ron: mm {nodding}
Tutor: good (. ) then you can give us uh the present illness part
Ron: for the history of present illness, (. ) uh she described the numbness as a tingling sensation which is paraesthesia and there was associated decrease in sensation, and it was ascending in nature (. ) it started off on her bilateral foot and within seven days it spread up to the T4 level, (. )
Tutor: it’s not three days, it’s seven days?
Ron: uh: because she was admitted quite some time
Tutor: ok so the symptom onset was three days before the admission
Ron: yeh
Tutor: but by the time you clerked the case ) it’s
Ron: ) yes )
Tutor: ) she has been in this hospital for quite some time )
Ron: )yes {nodding}
Tutor: ok; excellent so you see now more you haven’t described the onset yet it’s sudden onset, in the morning when she wakes woke up, and then noticed there’s some numbing or:
Ron: {nodding} it was acute onset she can describe the day she had the symptoms on her ) foot
Tutor: ) and it was it was not triggered by any specific movement, (. ) no preceding
Ron: ) there is associated back pain with the onset three days before the onset of the numbness, (. ) but there was she denied any trauma to the back,
Tutor: but then you see it’s uh: that does not really fit because if there is (. ) we assume (. ) can this be prolapsed disc or even vertebral collapse, then there’s a cord lesion (. ) it should not (. ) something which is contradictory to uh a cord lesion that it ) should
Fay: ) progressive?
Tutor: yes whilst ascending upwards right it’s less likely so this is unlikely to be a mechanical cause, therefore basically (. ) that can be excluded
Ron: {nodding}
Tutor: ok?
Ron: {nodding} (^^^^) the upper limb was normal (. )
so any viral illness preceding this event?

it was not preceded by any uh upper respiratory tract infection, by any uh or vaccine injection

ok good

there was no associated weakness, there was no fecal or urinary incontinence,

mm mm

she volunteered the history of taking herbal medicine two weeks prior to the onset of the symptoms )

) why does why did she take herbal medicine two weeks before the onset?

mm mm I particularly asked her about this and she said she took the herbs just for uh:: maintenance of the health and not for any particular illness

good

yes and there was no heavy metal exposure in her occupation and the environment (0.2)

any motor involvement

no there was no weakness

and upper limb)

) were spared

were spared (0.2) because,

{smiling}

it is only ascending to T4 level

{laughing} )

) of course

right yes

not yet ok (.) yes now by knowing this part of the history (0.3) what would be uh what would be {turning and gesturing to Sue} your diagnosis differential diagnosis? (0.6)

myelitis, transverse myelitis,

{dc} transverse myelitis yes (.) anything else

could be a presentation of multiple sclerosis but uh: at age of sixty-one
the onset is a bit late
85 Tutor uh:: well for multiple sclerosis (.) the typical feature is )
86 Fay ) (progressive loss of (^^^)
87 Tutor a (^^^) lesion (0.3) demyelination demyelination in different part of the therefore it’s unlikely to be a continuous path all involved (.) and MS usually have a motor component (.) ok?
88 Sue but still the acute (^^^) although it is not preceded by )
89 Tutor yes if some kind of viral illness can be so subtle, maybe just a little bit of malaise or that kind of thing yes it’s still possible (.) can this be Guillain- Barre syndrome?
90 Trudy is it more motor weakness than sensory )
91 Tutor ) normal?
92 Trudy like motor involvement rather than )
93 Tutor yes no motor function is not involved (.) good (0.2) anything else?
94 Ron she had a travel history (.) uh she travelled to Japan and Thailand but she stayed in the city area ) and
95 Tutor ) when was the travel
96 Ron she travelled to Japan in January, and stayed in Tokyo for ) eight days
97 Tutor ) January uh do you think that’s uh: a little bit,
98 Ron yes and and March (.) she travelled to Thailand
99 Tutor March (.) so you know when we talk about infection I guess you attend the uh you attend the (^^^) {SS laughing} lecture therefore it depends on what kind of bacteria what kind of infection are we worrying about we’re looking for a viral infection, incubation period is usually one or two weeks right, usually (.) so do you think travel to Thailand in March will give rise to this kind of clinical feature just because she get something she got infected in Thailand, (0.2) the )
100 Ron ) no
101 Tutor chance is extremely small (.) extremely small extremely small unlikely unless you can say this is HIV infection (.0 it could be what (^^^) three months yes now it’s June three months apart, it is possible but then this is not a typical feature of a (0.2) primary HIV infection (.) so (.) I I don’t think the travel history is that important in this particular patient but
you’re yes it’s always correct you should always ask about travel history
(.) because that may give us a hint (.) ok yes any other questions you
would like to ask (0.3) no yes (0.3) no questions from Mr Kong

102 Sue what about functional impairment like activities of daily living because
any disturbance with ) (^^^)

103 Tutor ) can she walk

104 Ron uh yes she can walk (.) but uh: very clumsily uh because of the residual
illness of her past health which I will continue

105 Tutor so other questions then we’d like to know is now we basically know that
because the uh:: history of present illness is not that complicated right
then we would like to know something about her past health, what
questions would you ask

106 Fay whether the patient has dia diabetes mellitus, whether )

107 Tutor )why why are you interested in diabetes mellitus

108 Fay because for peripheral neuropathy it can it’s also uh: ascending from the
most distal part first,

109 Tutor so rapidly, within a few days time,)

110 Fay ) but it’s (^^^)

111 Tutor {laughing} it’s extremely unlikely but yes you should always ask for
diabetes, what else (0.3)

112 Trudy {turning to Ron) is it the first episode

113 Ron uh yes it is the first episode for the numbness

114 Tutor the answer seems not to be completed yet right {laughing}

115 Ron I shall continue with the past )

116 Tutor )yes

117 Ron history (. ) in the year 200* she has a helicobacter pylori positive gastric
ulcer and in the next year repeated OGD was normal, and in November,)

118 Tutor so helicobacter, ulcer of?

119 Ron ) gastric ulcer

120 Students ) gastric

121 Tutor gastric ulcer so basically for even we do not know if it’s gastric or
duodenum (. ) we can assume this is (. ) gastric or duodenal ulcer

122 Students gastric
Tutor: why
Trudy: it usually affect the (^^^^)
Tutor: no because we need to (^^^^) the patient (.) we have to repeat the OGD (.) for gastric ulcer we usually repeat
Trudy: oh yeh
Tutor: for duodenal ulcer there’s no need to repeat the OGD therefore on reassessment that means the patient had a even we don not know whether it’s gastric we can guess (.) mostly that this is gastric ulcer (0.2) but on reassessment it’s cured right no more {Ron nodding} h pylori, that’s in 200* ok,
Ron: and in the next year November she presented with vertigo, diplopia:, headache, vomiting and progressive (^^^^) tetraplegia and a diagnosis of (.) brainstem encephalitis was made (.)
Tutor: two thousand and,
Ron: * November )
Tutor: ) well
Ron: ) she presented with vertigo, diplopia, headache, vomiting and later progressive (^^^^) tetraplegia
Tutor: tetraplegia {Ron nodding} and uh but then the diagnosis was uh
Ron: Bickerstaff’s brainstem encephalitis
Tutor: how was the diagnosis made
Ron: the diagnosis was made uh:: based on the clinical uh presentation and the uh (0.2) (^^^^) test (0.2) uh:: )
Tutor: ) did she)
Ron: ) the (^^^^) test was done (^^^^)
Tutor: have an MRI (0.2) of the brain?
Ron: uh: )
Tutor: ) because if it’s encephalitis, and then you’d also like to have interest to know what would be what would be the CSF finding etc
Ron: yes they checked the (^^^^) antibody it was negative )
Tutor: ) mm mm
Ron: and uh: they: I’m not sure whether they had the imaging done I assume they had the imaging MRI

55
and then the patient had a very good recovery

not exactly because then she could still visit Thailand

the tetraplegia recovered but there there were residual neurological
deficits

as reflected by?

uh as reflected by she had ataxic gait, and diplopia on horizontal gaze,
neuralgic pain on the at the tongue, uh but there was no problem
swallowing or drinking (.) so the )

) ok

) residual weakness uh: the neurological deficits were ataxic gait and
diplopia

ok (0.2) do you have any questions? (0.2) what about family history

uh she had no family history of uh similar illness

and besides these besides h pylori and this uh brain stem
encephalo)pathy

) she had good past health

) she enjoyed good past health (.) no other problem at all:

no

ok social history?

for social history she was uh: a non-smoker, non-drinker, she is a
housewife, she lives with her family both in Macau and Hong Kong (.)
half of the time in Macau and half of the time in Hong Kong

mm mm (0.2) good (.) any questions (0.2)

was a lumbar puncture done at the time that encephalitis was ) (^^^)

) yes it was done

you mean the last time or this time

last )time

) last )time

) last time

what was the (finding) (0.2)

well on taking the history a lot of the time the patient do not know the
finding (.) yes Ok we can trace back from the computer (^^^) of the time
but you know this lecture is talking about history-taking, physical
examination, and well if the patient can tell, (.) well it is unlikely to be a
very honest patient who can tell you what was the uh (LP) finding or the
(^^^)

170  Fay  no no not that but uh I was thinking that uh at least she would have a: uh
the doctor would have told her a diagnosis {Keith nodding smiling} )
(^^^)

171  Tutor  ) that is the uh well brain stem encephalopathy (. ) I don’t think this was a
diagnosis that’s a mistake mister ken {Tutor indicates Ron} got from the
patient right, it’s from the notes ) rather cos how

172  Ron  ) from the notes yes )

173  Tutor  because do you expect the patient can tell you {laughing} I’ve got brain
stem encephalopathy two years ago it’s extremely unlikely (.0 probably
then the patient can tell you (.)

174  Fay  (^^^) )

175  Tutor  what ) was the (. ) clinical features by then (. ) and you could guess maybe
it’s some kind of brain stem lesion, because of diplopia et cetera right
{Fay nodding} ataxia, diplopia, but then uh: it’s unlikely we we could
know the detail of the: especially those complicated illness (. ) it’s unlike
diabetes or hypertension (. ) the patient can tell you yes I’m on
medication for that (. ) otherwise it is extremely unlikely (0.2) but then
now with the knowledge with that kind of past history, do you think it is
related to the present illness (. ) or you think it is actually uh completely
not related

176  Jan  but it’s been some years already like ) between

177  Tutor  ) two years right

178  Ron  yes )

179  Jan  she’s well in between and no similar episodes

180  Tutor  yeh (0.2) but why so unfortunate (. ) brain stem encephalopathy, I would
assume this is also due to viral, or you may think if it’s not due to viral
can this be be due to some kind of auto-immune disease, vasculitis or
especially female patient (. ) right, bacteria is unlikely but viral if it’s
auto-immune (. ) if it’s auto-immune then it is well (^^^) episode
vasculitis yes if it’s viral why is she prone to viral infection, that’s why I would like to know is she prone to uh infection based on the past history (. ) then other than h pylori there’s no hint at all that she is prone to infection that she has immune deficiency et cetera (. ) it’s a bit a bit odd then we may still think ok, probably viral, again unfortunately or: could this be auto-immune disease and of course then it would be interest it would be of interest to know what kind of investigations were performed (. ) during her last admission because you know she must have undergone thorough investigation during the last episode (. ) and you uh probably you have already got some of the investigation result from the uh record part of ) them

181 Ron part of them (. ) ok (. ) so now well if we just go back to the history part, do you think there’s other things we would like to know (. ) beside the chief complaint what we have heard from mister ken (0.3) is there any other things you think might be important we have missed (0.8) {Keith coughs} yes mister lee you want) to say something

182 Tutor any (^^^) seizures, any SLA

183 Keith any (^^^) seizures, any SLA

184 Tutor um {dc} I have checked actually the Bickerstaff type brain stem encephalitis is an immune disorder

185 Tutor ok (. ) so then I would ask basically for female sixty-one I would ask about history of joint pains, stiffness of fingers, hair loss, sunlight exposure, (^^^) rash, asking about things related to rheumatoid arthritis, SLE that kind of thing (. ) right (. ) so if it’s not (. ) then we go back to find out what were the investigations last time (. ) if this was only the first episode, then probably we would order a number of investigations which we think is appropriate (. ) you know the most difficult part is always the the diag the diagnosis part (. ) because whenever you know the disease well you know what is the diagnosis (. ) you can search the internet you can know what is the most appropriate treatment right, (. ) good (. ) anything else, history part of this is quite simple right, not much differential because it’s such a specific problem (. ) so if not then we will proceed to the physical examination part

186 Ron uh for the physical exam uh Miss Wong was afebrile, uh blood pressure
and pulse were normal, uh)

187 Tutor sorry uh besides well I think we do mention this was it’s probably auto-immune in nature but then (.) just remember you have Guillain-Barre transverse myelitis (.) or auto-immune in nature right just sum up I mean it can trigger the immune response so of course it’s not due it’s not directly due to the infection just due to some abnormal behaviour of the immune system ok (.)

188 Ron she was afebrile, BP and pulse were normal, she had uh: diplopia on horizontal gaze (.)

189 Tutor that was probably the remnant of the last ) episode wasn’t it

190 Ron ) yes yes and she also had nystagmus on the right hand side which was also a residual neurological deficit (.) and the facial nerve was (.) normal, uh:: the u the uvula was deviated ) from the

191 Tutor ) facial nerve you mean cranial nerve

192 Ron yes

193 Tutor or facial nerve, specifically facial nerve

194 Ron {nodding} facial nerve ) (^^^)

195 Tutor ) seventh cranial nerve seventh ) cranial nerve

196 Ron the seventh cranial ) nerve

197 Tutor ) ok

198 Ron and the uvula was deviated towards the right side which again was probably residual neurological weakness, and the {hi} tongue: there was tongue fasciculation on the right side with no deviation of the tongue, (0.2) uh which is probably also the residual weakness (.) the power was five over five, reflexes were)

199 Tutor ) power of

200 Ron all limbs ) all limbs

201 Tutor {nodding} all limbs ok

202 Ron the reflexes were normal and with a downgoing plantar, no sphincter disturbance 9.) and: the sensory part, there was (.) decreasing pinprick sensation (. ) T4 level and below, there was (.) decrease in vibration sensation (. ) on all lower limb joints (. ) there was decrease in fine touch sensation T4 and below, uh however, the proprioception were all
preserved, on the gait (.) she had an unstable (^^^) walking gait uh: which she required a stick since (.) the: episode in year two thousand and six the Romberg sign was negative, there was no (tremble ataxia), and there were no other cerebellar signs

Tutor  fusion test, no?
Ron  uh: fusion tests were (0.3) uh )
Jan  \{whispering\} (^^^)
Ron  no
Jan  no )
Tutor  ) ok (0.2)
Ron  there were no other cerebellar signs
Tutor  ok (.) so it’s purely sensory right?
Ron  yes
Tutor  \{dc\} it’s true purely sensory (.) ok (0.2) so do you think the physical examination part contributes to the (.) diagnosis?
Jan  actually the involvement of the sensory modalities of pain, and also the uh::
Tutor  (light) touch?
Jan  pain? \{turning to Trudy and Ron\}
Trudy  (light) touch
Fay  pain, light touch) vibration
Ron  pain, light touch, ) and vibration
Jan  pain and ) vibration
Ron  )(^^^))
Fay  )(^^^)
Jan  that proprioception is (spared)
Tutor  quite odd right? well you know sensory test is always difficult (.) you you you do the sensory test in the morning and you repeat in the afternoon and you may have different findings (.) just it’s sensory right it’s not that objective (.) so anyway (.) first of all it does contribute to the diagnosis because you have ruled out motor involvement, there is no cerebellar sign, (.) can we use uh:: uh past-pointing test to test the cerebellar (0.2) function of this lady? (0.2)
Students: yes
Fay: (^^^) she has
Tutor: probably not because the diplopia)
Jan: ) oh {smiling at R}
Tutor: therefore fusion test is more, can more truly reflect the cerebellar function
Fay: {looking at R} but diplopia is it only in extreme uh gaze or even the looking uh
Ron: uh:: sometimes she complained of that even in the central gaze there is diplopia
Tutor: so therefore you have to choose the right test for the right patient otherwise it’s uh it could be quite misleading (. ) good (. ) so what’s next?
Ron: there were some investigations (. ) done in the ward during this admission
Tutor: yes if you you you are you were the doctor in charge what kind of investigations would you ask (0.2)
Ron: um:: (0.2) because the patient presented with sensory loss I would like to perform nerve conduction study, and also we suspect some cord compression and she had some low back pain and so (. ) uh: we can order an MRI spine )
Tutor: ) yes
Ron: to exclude any cord compression
Tutor: would you do a lumbar puncture? (0.3)
Ron: uh depends on uh the finding on the nerve conduction study (. ) if it is uh a peripher, a lesion of the peripheral nerve then I would not ) proceed
Tutor: ) unlikely peripheral right sensory level up to T4 {draws line across chest}
Ron: {nodding} yeh so it depends on (^^^) so yes I will perform this {laughing}
Tutor: to look for?
Ron: to look for any inflammatory markers, for example um {smiling} including cell protein dissociation, and uh: (0.2) some (oligochromal bands) or auto-immune auto-immune antibodies
ok (. ) and so some must have been done right since she has been here for quite some days

(smiling) (0.2) uh yes

was MRI performed?

MRI was awaiting (.) awaiting

still waiting ) ok

) still waiting and the nerve conduction study was done (0.2)

and?

and found that there was no acute neuritis, there was no demyelination of the peripheral nerves, there were no peripheral nerve channelopathy (0.2)

but those were actually expected right? it’s unlikely to be peripheral cause (.) ok (.) what other tests have been performed?

{shaking head}

no

just the nerve conduction study and some uh routine blood tests

but you said you said this patient has been with us for some time?

yeh just a few days

{laughing})

no LP at the moment

no (0.2)

they’re waiting for the MRI

yes {nodding}

no treatment at this moment

(0.2) vitamin b complex (0.2)

excellent

{laughing})

{laughing}

what about immune immune test

any blood test for ) immune markers

) (^^-^) antibodies

) yes

yes uh: I didn’t specifically look for that (0.2)
you know it’s well based on the past history we would tend to believe this is probably uh auto-immune disease and the disease is actually progressing right so that means we start the investigation probably some treatment should be given as well based on the nature of the illness it’s unlikely to be an infectious cause or problem and you know we do not have much choice in treating auto-immune disease other than the steroid {laughing} right, so uh: I guess you would push for a so-called early MRI it’s uh: that would be of fundamental importance and then proceed to (LP) to get more information and then probably can start treatment

mm {nodding}

because the disease will not wait that’s the problem well it’s a good thing you said it’s not a common problem it’s not commonly seen ok good anything you would like to uh ask, discuss yes Miss Fay

(^^^) {laughing} what was the result of the last lumbar puncture in two thousand and six I mean

I: don’t know

you can say the patient did not know so that (^^^) save you

(^^^) {laughing}

well it’s unrealistic right the patient could tell you what was the LP)

I think she he has looked at the charts (^^^)

you know during exam)

) oh ok)

there’s no chance to read the charts

yes (^^^)

) you would just assume you get everything from the patient no more than that even if {laughing} if you have you had read the chart

mm mm

ok?

so the working diagnosis of the uh Miss Wong is acute transverse myelitis multiple sclerosis and to exclude cord compression

(^^^)
you know (. ) if they put to rule out cord compression as the first working diagnosis

maybe MRI should be earlier

the MRI should be done already (. ) but then of course we do not think this is compatible with cord compression and this is not compatible with MS multiple sclerosis, therefore this is most likely a case of transverse myelitis (0.2) right

so without investigation (. ) we can get to the diagnosis (. ) go back in two weeks time and you will uh where’s the patient now

why don’t you clerk a case in B seven? you know when we are doing problem-based learning I would expect you to go and clerk a case in acute medical ward (. ) problem-based learning is so patient is admitted to the hospital with a )

) because she was transferred on Wednesday {smiling}

good timing

{laughing} excellent (. ) so basically yes I would I would encourage you go back and monitor the progress of this particular patient and see what is the final diagnosis ok (0.2) so we can only learn from these longitudinal and monitor the patient if we do not know the diagnosis yet ok (. ) this is most likely a case of transverse myelitis ok

{nodding}

with this history of uh: brain stem encephalitis still is it not possible for multiple sclerosis (^^^) brain stem and also the T4 spinal cord

uh::

can it be (^^^) lesion (0.2)

I guess first of all (0.2) MRI must have been done last time (. ) if this is multiple sclerosis

{nodding} mm

the diagnosis was already made (. ) right (0.2)

but clinically do you know
ok there are some specific signs for multiple sclerosis when it’s uh known as inter nucleus ophthalmoplegia

Students {nodding}

Tutor do you know what this is?

Students {nodding}

Tutor very complicated

Students {laughing} (^^^)

Fay very complicated

Tutor yes (.) do you know the sign)

Jan ) nystagmus of the (abductive) eye and the other eye cannot abduct

Tutor yes but then only when looking to one side and the other side is quite normal right (.) and then there’s also hyperreflexia (.) it’s quite common in multiple sclerosis ok? we do have well there are there are a few patients with multiple sclerosis and they commonly appear in different kinds of examinations (.) but usually at higher level Royal College examination, and I did have the luck to examine one of the patients during exam {laughing} yes multiple sclerosis (0.2) less likely ok (.) you can uh what is why multiple sclerosis is not common here (.) people did have some hypothesis, I guess one of the hypotheses is that it is triggered by infection (.) some kind of infection triggered the auto-immune system but then that particular infective source that agent is only at certain geographic area, therefore MS is only appear in countries with whe:::re specific geographic regions (.) I can’t remember the details but when you search you can find this kind of information (.) you know why suddenly people got auto-immune response? probably something triggered it (.) and most likely the triggering factor will be viral infection 9.) right (.) good (.) who’s next

Trudy {raises hand} 0.2 our patient Miss Wong uh a twenty six year old um non-smoker, non-drinker, working as a clerk, with a fourteen year history of systemic lupus erythematosis, complicated by uh)

Tutor fourteen years history of,

Trudy SLE (.)

Tutor ok
um complicated by um lupus nephritis (.) leading to end stage renal failure on haemodialysis, this time admitted because of um uh generalised malaise, vomiting for more than ten times of undigested food and (.) decrease in appetite after haemodialysis

so do you think the chief complaint is a little bit too long

so um: patient with a fourteen years year history (.) of um uh lupus nephritis on haemodialysis complained of ten times vomiting

do you think fourteen years is an important number

(0.2) uh:: long history of SLE

well basically it’s it’s )

Jan ) (^^^)

end stage renal failure due to SLE on dialysis presented with how many days history of (. ) GI symp)toms

two days )

) like that you can simplify because history of how many years of SLE might not be that important, but then including SLE in the chief complaint does have a meaning because (. ) due to (. ) hypertension, diabetes, because SLE per se can give rise to these symptoms (0.2) right three days history, she’s on haemodialysis, the symptoms appear after haemodialysis ok, (.) good

um I’ll expand more on the chief complaint so um she had vomiting more than ten times of undigested food um os um however there was no um diarrhoea, and um she um did not have any special food taken um for that (.) she also complained of decrease in appetite, and some uh dizziness but uh there was no vertigo and there was no fever, no cough, and no sputum (.) and um on admission she was found to have hypoglycaemia um her (haem ^^) was three point six (A and E) um )

) so basically when someone presented with uh: vomiting (0.2) with it is quite common if we think about gastro-enteritis, if we do suspect gastro-enteritis, (0.2) yes we will ask about specific food what kind of food have you taken, before that, and we would also like to know cluster (.) is there any ) cluster
no uh no family members with uh similar symptoms (0.2)
no family members probably yes no well other people share the same food, did not have similar features (. ) ok, so uh:: )
and no travel history
is she still on haemodialysis
{nodding}
she’s unlikely to have a travel history right, she needs two times per week or three times per week,
yes no travel well it’s less likely (. ) ok GI symptoms lead to hypoglycaemia (0.2) that can be explained, so then of course the next question would be what leads to the vomiting, what do you think mister mister ken
besides gastro-enteritis, what else can give rise to vomiting (0.4)
it can be uh: (0.5) it can be related to the haemodialysis and may get some (0.2) uh form of infection through the haemodialysis machine and then present as vomiting (0.3) you mean uh well what we what can people get from the haemodialysis machine (0.2) hepatitis right hepatitis well but then it’s unlikely I mean in Hong Kong right (0.3) then usually they hep for hepatitis patients the chief complaint would be malaise, jaundice, poor appetite (. ) it is not common to be acute onset (. ) when you when people talk about what do you think is the reason for the for the vomiting, then do you think it can be due to local pathology or distal pathology (0.2) let’s give you some time to think about what to talk next (. ) yeh local thing can this be due to the bowel obstruction, maybe maybe not (0.2)
it may also be due to distal pathology for example increase in renal pressure,
yes or maybe just after haemodialysis due to imbalance of the electrolytes (. ) sometimes this can also give rise to vomiting (0.2) vomiting is not that difficult can this be due to things not related to the haemodialysis, yes)
could it be) could it be (^^^) symptom of the SLE

Tutor SLE give rise to dysphagia: it’s not common (.) it’s not scleroderma

(0.2) right dysphagia is not one of the common features of SLE (.)

rather then think of if it’s not due to local, because there is no diarrhoea,

the next question would be is there normal bowel opening, maybe it’s

just because of bowel obstruction (.) if yes the patient told you yes I do

have normal bowel opening (.) then you have already ruled out bowel

obstruction, then think out can this be increase in intra-cranial pressure

because headache is also one of the chief complaints right, why, what

can give rise to intra-cranial pressure in SLE patients, then you can have

a long long list (.) which will include, yes mister lee

Keith lupus cerebritis

Tutor uh,

Keith lupus cerebritis

Tutor yes you can say that can be due to the disease per se: vasculitis vasculitis

involving the the brain or::, (0.2) due to problems related to, (0.2)

Sue the patient is immuno-suppressed

Tutor yes related to the immuno-suppressed patient (.) they can develop all

kinds of infections (.) (CNS) infection can give rise to vomiting as well

(.) and headache and malaise (.) there are always a lot of things you can

discuss (.) due to the disease or due to treatment (.) then in the history

part you will provide what kind of (immunomodulating agents) is she on

et cetera et cetera (.) ok, good (.) let’s proceed

Trudy so um for the um: history of the present illness she was uh diagnosed to

have SLE fourteen years ago when she presented with malaise and

dizziness and also uh hemat)uria

Tutor ) sorry just one more dx we haven’t discussed yet (.) if the patient’s

pregnant (.) she’s a young lady

Students {nodding}

Tutor right, that should always be remembered

Jan {smiling}
69

Students {nodding}

364 Trudy at that time

365 Tutor ) not possible do you think

366 Students {laughing}

367 Tutor yes why not

368 Students {laughing}

369 Tutor yes maybe you can say yes then she will ask in the history as well (.) (because) it’s significant illness, could just be amenorrhea that we can basically rule out (^^^) (0.4)

370 Trudy oh at that time she had she also had hemoturia, proteinuria, and (^^^) urine and renal biopsy was done and she was diagnosed to have some form of acute glomerulonephritis, and since then she had been put on steroids and um but no other immuno-modulating agents was given (.) and in nineteen ninety nine she um pro uh proceeded to uh also at the same time she was diagnosed to have some (beta) thalassaemia trait (.) and in nineteen ninety nine too she progressed to end stage renal failure requiring um: like CAPD (.) and um later in two thousand and two it was complicated by peritonitis with severe adhesions so she switched to haemodialysis (.)

371 Tutor {nodding}

372 Trudy and in two thousand and six she had a cadaveric renal transplant (.) uh

373 Tutor ok so:: start she was put on haemdialysis in two thousand and two and then two thousand and six she had uh ) renal transplant

374 Trudy renal transplant

375 Tutor done in (0.1) ) Hong Kong

376 Trudy ) Hong Kong

377 Tutor Hong Kong (.) good (0.1) and?

378 Trudy and then in two thousand and eight she had some dysuria and um she was admitted to (^^^) and they found she had some urinary tract infection, and it ascended to the transplanted kidney causing um uh acute graft rejection (.) um there was)

379 Tutor ) acute (.) graft (.) rejection (.) what is the definition for that particular ) medical term?
Trudy: uh sorry graft uh causing rejection uh causing graft rejection
Tutor: yes that’s better
Trudy: ) and uh so uh the transplanted kidney was removed in May this year (.)
and she referred to uh the haemodialysis (.)
Tutor: ok (0.3)
Trudy: and this is about the uh renal problem (.)
Tutor: no other history, present illness?
Trudy: history of present illness she also had um uh other like complications of
SLE (. for the ) (^^^)
Tutor: no no no I mean uh what about (. can you describe in detail about this
episode uh the reason for this admission yes
Trudy: um this time she was she had uh haemodialysis last Saturday and
Tutor: she had haemodialysis last Saturday ) and
Trudy: ) and then after she went home uh a few hours after she went home she
started to have uh like vomiting of uh more than ten times and
undigested food, and like malaise and poor ) appetite
Tutor: ) but did anything unusual happen during the haemodialysis (0.2 ) was
there any hypo
Trudy: uh)
Tutor: ) tensive event et cetera during the dialysis
Trudy: I think (^^^) not event
Tutor: it’s just like the usual haemodialysis, nothing abnormal happened, and
she was send home after the haemodialysis, and then after a few hours at
home she developed vomiting,
Trudy: but then because of the heavy rain she did not come to hospital till
Monday (. oh until)
Tutor: ) Sunday
Trudy: {looking at notes} like the day after the rainstorm warning she came
Students: {laughing}
Tutor: it was Sunday it was Sunday
Students: {laughing}
Tutor: ok (0.3)
Trudy: and uh yeh so there was no change in bowel habits
so by the time she came to hospital she still had a lot of vomiting

after admission her vomiting subsided

ok

and she (end of tape) only vomited once after admission

ok (.) then

however she still uh have some hypoglycaemia
Tutorial 3
Medicine Specialty PBL Session

Students: Eddie, Martin, Kevin, Harry, Zelda, Joy, Chris, Vicky

Note: The venue for this tutorial had changed at the last minute unknown to the researcher. The tutor came to the original venue to collect the researcher after approx 5 minutes in which time the chief complaint and symptoms of the first case under discussion had been presented so Zelda began in Turn 8 below by repeating this.

1 Tutor alright?
2 Anne I think so (. ) I’ll sit at the back if that’s alright
3 Tutor so we’ll pretend that that you are not there
4 Anne as much as possible yes thank you
5 Tutor so (. ) I think we have uh: decided uh to talk about two cases already )
6 Martin ) mm
7 Tutor so shall we talk about the first patient with this blistering eruption? (. )
8 Anne and oh )
9 Zelda ) the blistering eruption
10 Tutor ) yeh
11 Zelda ) that’s fine yes (0.1) um: (0.1)
12 Tutor so perhaps you’d like to repeat the the major clinical features of this patient? (0.2)
13 Zelda um so )
14 Tutor ) I’ll tell you that I want you not to refer to your notes {laughs}
15 Zelda um do do you want me to uh: include um the drugs this time and also the )
16 Tutor ) yeh yeh )
17 Zelda ) some of the physical examination )
18 Tutor ) mm
19 Zelda uh so uh Mr Lau um a sixty-five year old um retired um government servant um presented with um blistering um (. ) blistering
20 Tutor (^^^)
blistering eruptions over uh bilateral uh palms also on the dorsum of the lower limb to the Accident and Emergency Department (.). um: so on the ninth ( {laughs} ) of November um and um the physicals were uh itchy and uh painful but uh the pain is not so severe that would prevent the patient from sleeping (.) and the blisters gradually increased in size and um the one over the um lower limbs actually ruptured with some watery discharge and um )

) actually you have been using on the one hand vesicle and on the other hand blisters (.). do you think there are any differences between these two terms )

uh yes um: blistering is um refers uh to um a lesion that contains a fluid and vesicles are those smaller than 0.5cm and bullas would be greater than )

yeh I think the size is the determining uh factor to describe the lesion OK anything smaller than 0.5cm would be called vesicles OK (.). but bigger than those then we call it either bulla or blister OK but both of them are entitled to contain clear fluid pussy fluid or even blood in the case of either of these might have blood pus or clear fluid alright? so now that you have been using two terms because perhaps originally: the vesicles seemed to be small but then they seemed to get larger to be qualified to be called blisters (.). isn’t it OK? (.). and then you have specified that they are ruptured isn’t it?

um those over the lower limbs are ruptured and when I see the patient um actually those uh over the palms are uh )

) still intact )

) are still intact (.). there’s one that measures around three to four cm over the left palm and there is one that um basically um involves the whole whole palm palmar area )

) mm )

) um that even contains some pus over the right dorsum

) mm )

) and um there are also uh some erythematous spots over the right upper limbs which are itchy and the patient have um developed before um the
bulla’s formation )

31 Tutor ) mm )
32 Zelda ) and um there were no mucosal involvement, no constitutional
symptoms, um no urinary or bowel symptoms, and the patient has uh no
recent travelling history or contact history, vaccination, um use of
Chinese medication, and he hasn’t um been started on any new
medication um within um that’s half a year um )

33 Tutor ) good )
34 Zelda ) the patient has a history of )
35 Tutor ) before we go any further, so: for the present illness, here is a patient
sixty-five years old (.) with a rather acute onset of a blistering eruption
(.) OK (.) mainly over his four limbs isn’t it, especially over the palms
isn’t it (.) OK and um (.) for how long by now? cos you haven’t really
told us since when )

36 Zelda ) um yes )
37 Tutor ) because he was admitted on the ninth )
38 Zelda ) um yes )
39 Tutor ) and had he been having this problem ) before he presented himself?
40 Zelda ) it’s one week before he presented to the A and E )
41 Tutor rather acute onset isn’t it? OK? and you have taken pains to tell us that
the patient did see actual vesicles isn’t it? OK and also you have
mentioned about no mucosal involvement (.) would you like to
elaborate on this? (.) what do you mean by no mucosal involvement?

42 Zelda um the:: the oral area is not involved )
43 Tutor ) yes )
44 Zelda ) and um there are no tarry stool, um any uh urinary symptoms, the
patient isn’t ^^ dysuria ) or any tarry stool
45 Tutor ) anywhere else (.) you would like to highlight to suggest whether the
mucosa is involved?
46 Zelda oh um there are no eye symptoms (.) either
47 Tutor no eye discharges? of each of the ^^ OK (.) and then (.) has this been
associated with any systemic upset?
48 Zelda uh there is no uh fe fever no chills or (aigres) no (constitutional)
symptoms, no joint pain

so he is well otherwise?

uh but because he has had uh several vascular accidents um in 2003
which left him having a left (hemiparesis)

past health (.) you can mention that later OK (.) I can’t remember
whether you have mentioned whether the rash is itchy or not )

uh he said it’s itchy but it’s not too (.) it’s both itchy and painful but
not to a very severe extent

OK

) s s it’s moderately itchy )

) I think itchiness and pain is also uh to be noted whenever you are
confronted with a patient with a rash: OK? so:: and you have
specifically mentioned that he has not been changing his medications (.)
and in particular he has not been taking any traditional Chinese medicine
OK? {ac} I think this is a very important aspect because in Hong Kong
(.) most of our patients might be taking some form of traditional Chinese
medicine, which (.) they might think (.) it’s important so they might not
have (.) directly or uh uh uh purposely told you if you have not asked of
them OK so it’s important that you should have a direct question on
these issues OK (.) and: so: you are about to tell us his past health

ye:s um so um he has um the left hemiplegic stroke in um 2003, and um
he has had a long standing history of hypertension, type two um diabetes
um and also had lipidemia and uh is on uh medications

mm

and um (0.2) the uh the uh left hemiplegia ^^^ from the the )

) ^^^ )

since then he has become retired and he requires a quadrapod to walk

OK )

) and )

so you mean he had to retire early and is it because of this illness? )

) because of the illness um but he said that he doesn’t really have any
financial problems uh and he was alright with that

perhaps this is so because he mentioned that he is a civil servant )
Martin  ) mm
) so with retirement he would be entitled to what?
Zelda  um the  {(gong sik gam)}  )
Tutor  ) no it’s a pension for the civil servants that means he maybe he may
have the advantage of being paid regularly every month despite the fact
that he has stopped working OK? but what did he work )
Zelda  um I did )
Tutor  ) originally
Zelda  ) I didn’t specify because it seems that he was a bit reluctant to tell me
Tutor  really?
Zelda  I I I’m a bit unsure
Tutor  OK because you would really like to know whether in fact he would
have become disabled because of this cerebrovascular accident and
that’s why he has to retire or whether in fact um he chose to retire a bit
early OK (.) so you have no idea at all )
Zelda  ) no )
Tutor  ) what he is doing professionally (.) why should he why should he be so
evasive?
Zelda  because I don’t know when when I asked about the family uh things )
Tutor  ) he’s also evasive as well
Zelda  he feels a bit I just have this feeling that
Tutor  {(^^^ Cantonese) someone enters the room}  (0.3) are you alright?
Student  yes
Tutor  because you don’t feel too well
Student  (^^^)
Tutor  OK (0.4) thank you thank you (0.6) so we have been discussing the first
patient with blistering eruption OK
Zelda  (^^^) concerned when I asked about any forms of family history
Tutor  OK OK (.) so you have no idea at all whether he is married or )
Zelda  ) oh he is living with his son
Tutor  his son?
Zelda  yes who is also taking care of him
Tutor  mm how old is his son?
Zelda: I haven’t specified

Tutor: mm mm mm right: so anything else? (0.3) did you did you tell me that you want to elaborate on his drugs?

Zelda: oh yes um for so as for the last time um the um the red blood cells was elevated and also the (^^^)

Tutor: ) no but the drugs )

Martin: ) the drug )

Zelda: ) oh yes the drugs he’s on aspirin, metformin, (^^^) some dologesic and (^^^)

Tutor: are you familiar with all these kinds of medications?

Zelda: um it )

Tutor: you are quite clear about all their indications?

Zelda: yes the antihypertensive the um diabetic drug and um aspirin is for blood thinning and dologesic is I think uh just for the pain for um the lesion

Tutor: you just used the term some dologesic what do you mean?

Zelda: um I think that (^^^))

Tutor: ) he’s taking that )

Zelda: ) request )

Tutor: ) so we usually call it dologesic PRN )

Zelda: ) PRN )

Tutor: ) on demand basis

Zelda: on demand basis

Tutor: we don’t usually have some analgesics OK?

Zelda: OK >

Tutor: alright

Zelda: um actually and uh also for one part of the history he didn’t volunteer himself I found from the case notes in um May 07 actually he was admitted with uh left foot cellulitis and was given some antibiotics and that case mentioned that um he was known to have poor foot care

Tutor: mm mm mm

Zelda: OK

Tutor: that would be important to note because as you might know we usually invite the podiatrist in our team approach in managing patients with
diabetes mellitus (.) isn’t it OK? that’s all about the history?

117  Zelda  um (0.2) and also um for um I want to highlight one point again chronic alcoholic and he has been drinking regularly um three times a week and every time around two cans of beer

118  Tutor  mm and he: was quite (. ) relaxed to tell you about this isn’t it? he’s not trying to hide away this particular fact from you?

119  Zelda  um I just asked him bit by bit (. ) so I said at first do you drink and he said three times and then I asked a bit more

120  Tutor  because uh I hate to say so but alcoholics are known to be great liars OK (.) most alcoholics would not tell you the truth as regards how much they are drinking OK so I’m a bit surprised that he should be so evasive in some other aspects of his social life then he confesses to you that he is eh drinking regularly isn’t it? OK, how about his smoking history?

121  Zelda  he is a non-smoker

122  Tutor  non-smoker OK

123  Zelda  and there was no known drug allergy and no any history of allergy disease

124  Tutor  OK so that’s about it isn’t it? OK?

125  Zelda  and um for the um physical uh the neurological exam because I didn’t perform it last time and um I the power is four on the right side and on the left side um uh I would uh grade it around three and the (^^^) are brisk brisk and there is an upgoing plantar response and )

126  Tutor  ) so it’s quite in keeping with

127  Zelda  upper motor neurone lesion

128  Tutor  OK

129  Zelda  mm mm and the sensation is um intact but he said that it feels slightly diminished when compared to the right side (0.2)

130  Tutor  how: is he or is he able to walk himself adequately, well,

131  Zelda  yes with the quadropod and he said he can take care of himself for example getting changed or eating or taking a shower

132  Martin  mm mm

133  Tutor  I believe he must have developed a bit of friction contracture of the
relevant (. ) limbs?

134 Zelda um yes the tone is um increased )
135 Tutor ) increased (0.2) alright so: um (. ) he you are quite convinced that probably he has some sort of stroke previously isn’t it? OK?
136 Zelda yes yes
137 Tutor and the skin lesions we have all of us have checked those already, ok we did we do recognise that he has blistering eruption and we did uh uh uh observe an actual intact blister isn’t it ok and you remember that on his left foot there seemed to be some additional (. ) rashes isn’t it?
138 Zelda yes um it looks more hyper pigmented and uh a bit um (. ) swollen over the upper left ankle (0.2) with a lot of scaling and there’s uh quite obvious um tines pedis over the foot )webs
139 Tutor um um ) ok so we are suspecting that the patient may have been having super imposed infection on that particular site (. ) probably fungal as well as perhaps bacterial (. ) isn’t it? OK? (0.5) that’s all mm?
140 Zelda yeh yes
141 Tutor so: any relevant investigations that we haven’t really talked about last time that we like to highlight (0.7) so nothing impressive?
142 Zelda mm not much
143 Tutor OK (. ) alright (. ) so perhaps what are our our objectives set last time? (0.3) the first one was the one )
144 Zelda ) the differential diagnosis of blistering disease)
145 Tutor ) yeh
146 Zelda and um they and uh primary or secondary dermatological disorder,
147 Tutor mm
148 Zelda and also the difference between and ruptured and unruptured blisters
149 Tutor OK so basing on those (. ) can’t you tell us (. ) with (. ) reference to our patient,
150 Zelda so sh uh I I have a definition for blisters )
151 Tutor ) yes
152 Zelda and it’s a accumulation of fluid within or under the epidermis and the diagnosis will depends on the site of the intercellular split
153 Tutor mm
and um (. . .) so for example um if it’s uh subcorneal they are very thin and the roof will break easily so they might present with just ruptured vesicles, and um some example would be impetigo, or um SSSS the staphylococcus scaly skin syndrome (smiling) and if its uh further down intra-epidermal it’s still within roof an unruptured with denuded surface so it looks uh slightly depressed, and these include um acute eczema, um varicella, um herpes, (. . .) and if it’s uh sub-epidermal then usually they present with te with a tense roof and the um um blisters the vesicles (laughing) or bullas will still be intact (. . .) and um these include bullous pemphigoid um dermatitis, herpetic bullas, um erythema multiform

mm mm so I think its very important when you are confronted with a patient with blistering eruption to see whether you are able to detect (anything) vesicle or blister OK and if they are present you could infer that the lesions are probably sub-epidermal and that will help you to exclude other possibilities OK? (0.3) so )

and um uh also because uh actually I think I think it’s not the (. . .) one in clinical examination it’s not just by if they they ruptured or not but asking if there’s mucosal involvement)

) mm mm

and also exclude many differential diagnosis (. . .) for example, those with mucosal involvement uh might be pemphigus or some kind of maybe drug reaction say Stevens-Johnson syndrome

so again that’s why again another very important leading question may be to ask for mucosal involvement and I have reminded you of what uh we call mucosal symptoms isn’t it (. . .) most obviously it would be the oral ulcerations so do remember to look into the oral mucosa (. . .) despite the fact that the patient might not be complaining of it (. . .) OK and (. . .) pay attention to the lips as well because uh angular kelosis is also that is some uh uh erosions on the angle to the mouth might also be telltale alright? OK and the eye is a very important area for attention because the mucosal uh uh lining uh conjunctiva is a very great uh very important area for attack isn’t it OK and then other mucosa would
involve the genitals uh area so the patient might complain a bit of
dysuria, worse still hematuria and then involvement of the mucosa of the
gut would lead you to abdominal symptoms, and tarry or blood-stained
stoools OK? and worse still if the mucosa of the bronchial tree are
involved then the patient might be coughing and irritating and even
hemoptysis though that would be very uncommon (.). OK? for practical
purposes mucosa is present mucosal involvement is present um you
would like to think of Stevens-Johnson syndrome as an evolution from a
erythema multiforme isn’t it OK? and the other important uh uh uh
differential diagnosis would be pemphigus, and pemphigoid OK the
mucosal involvement would help you to suggest that this is more likely
to be pemphigus rather than pemphigoid isn’t it? OK (0.2) so (.)
anything else?

160 Zelda um (0.2) without um and also um: there are other ways of classifying
them as well but (some) can put them into auto-immune and non auto-
immune cause

161 Tutor mm

162 Zelda and: um because then uh treatment might be different and um to go
further there are some form of bullous um um disease which are um
congenital and um but those would have been presenting um since uh
childhood and wouldn’t be applicable ) to our case

163 Tutor ) which is the most famous congenital fistular eruption?

164 Martin epidermolysis bullosa

165 Tutor epidermolysis bullosa isn’t it I don’t think I have seen any one such case
I don’t know whether )in the pediatric

166 Martin ) but they did have )

167 Tutor ) you’ve seen one already?

168 Martin ) they have one in ICU )

169 Tutor ) oh really so you are very lucky then (laughing) and anything else when
you are thinking about congenital would there be any again quite well
unknown perhaps metabolic error (. ) that might have led to this
blistering eruption?

170 Martin porph)ydria
porphyria isn’t it)

Zelda

) oh yes (^^^)

Tutor

porphyria is very uncommon again and they come in different forms OK porphyria cutaneous tarda might be the one who is most famous for giving you this uh blistering eruption (0.2) right? so as I’ve hinted for the last in the last session OK when you are thinking of differential diagnosis you would like to think of the aetiology then we usually go by congenital, infection, metabolic, uh and so on OK so congenital wise we have epidermyolosis bullosa, metabolic wise we have this uh porphyria OK and then infection wise (.) all kinds of infection can lead to blistering eruption OK (.) be it uh bacterial as you have mentioned, it is scalded skin syndrome usually associated with staphylococcal infection or even streptococcal isn’t it? (.) fungal infection is a very important uh no fungal it’s not herpes )

Zelda

) oh sorry ah )

Tutor

) fungal infection can also induce blistering eruption although this is not that common OK there is virus infection herpes simplex, herpes zoster OK (.) and a typical micro-bacterial sorry a typical bacterial infection like mycoplasma can also give the blistering eruptions (.) well so infection is a great category for you to remember (.) and then we come to of course drug isn’t it OK drug eruption, which would give you erythema multiforme and one of the form would be this blistering eruption which we call um with mucosal involvement which we call Stevens-Johnson syndrome isn’t it (.) what do you mean by erythema multiforme? (.) I think the term itself is very uh uh self explain explanatory (.) erythema means )

Zelda

) redness

Tutor

) redness multiforme means )

Zelda

) every)where

Tutor

) in multiple form so patient would have all kind of skin rashes (.) macules, papules, nodules )

Zelda

) blisters

Tutor

perhaps blisters OK and very often it could be a drug induced problem,
which which are the most common drugs

I’ve just seen a Chinese medication induced one

yeh in Hong Kong yes we tend to blame it on Chinese medication when
we can’t find a good cause (Martin laughing) although it’s not too fair
but this is something definitely important OK but (.) in Western
medication?

) I think it’s usually some )

) (^^^)

) septrin

septrin, sulphur containing drugs sulphonomanides, (^^^) septrin)

carbamezepine )
carbamezepine yes)
allopurinol

allopurinol is perhaps one of the most common uh uh culprit
as you might have mentioned uh uh heard Professor (Kumana) say
telling us isn’t it

yes

last Saturday so drug is always important (.) OK and then? what is the
other big categories of differential diagnosis?

um auto-immune

auto-immune OK and then such as?

um pemphigus um uh vulgaris

mm ) OK

pemphigoid )(^^^)

we are not too sure whether this uh actual auto-immune yes but what
are the more auto-immune conditions (0.2) when you are thinking of
me?

(laughing) um:

SLE

oh SLE

auto-immune diseases SLE patients are entitled to have blistering
eruption although this is not that common OK and so uh there is
something to remember as well (0.2) any other big categories for the
differential diagnosis?
204 Zelda how about the one during gestations for the ladies
205 Tutor yes yes but that is not that common to induce blistering it’s more of a pruritus isn’t it?
206 Zelda mm
207 Tutor OK? but that would bring us to a very important point (. ) in fact the most common cause for a blister would be what?
208 Zelda co um)
209 Martin ) (^^^)
210 Zelda ruptured I I think )
211 Tutor ) trauma
212 Student (^^^)
213 Zelda ) trauma
214 Tutor which would include scratching so any itchy eruption would induce blisters isn’t it? and you might have witnessed that yourself isn’t it? be it a burn, an actual scald, a chemical burn, fire OK? what else? (0.2) what is a another very common scenario which perhaps all of us might have experienced when we have to hike a )lot or
215 Zelda ) insect bites
216 Tutor ) jog a lot )
217 Zelda ) insect bites
218 Tutor no I’m talking about trauma (. ) um
219 Zelda oh
220 Tutor friction )
221 Zelda ) friction
222 Tutor ) on the soles of feet can you recall )
223 Martin ) mm
224 Tutor ) so pok haime uh the haiam je (Cantonese) is coming up isn’t it so uh (. ) all those participants would end up with blisters on the soles of feet so trauma is always important for you to remember (. ) as a cause for blister OK? so this will come into differential diagnosis of the big categories
225 Zelda mm (0.2)
226 Tutor what else what is another important big category for differential
diagnosis that would be applicable to all kinds of (.)

227 Zelda ) um cancer?
228 Tutor yeh malignancy )
229 Zelda ) {lo} malignancy
230 Tutor very good do remember malignancy can induce {dc} any form of dermatosis OK? (0.7)
231 Tutor so )
232 Martin ) is it is it uh neuroplastic ) periplastic or is it a
233 Tutor ) yeh it could be a kind of periplastic manifestation OK and then you would like to think of primary skin diseases and of which I think there are two very important uh conditions for you to remember the pemphigus (.) vulgaris and the pemphigoid bullous pemphigoid isn’t it the third one would be as you have suggested earlier dermatitis herpetiformis OK (0.3) when you are going for the higher professional examination which we call MRCP exam a very favourite question to ask of you would be the differentiating features between pemphigus and pemphigoid OK? so I believe you might have read about this in your (.) preparation for this morning’s session, can you enlighten us on this?
234 Zelda um for the uh uh pemphigus uh there are two forms the vulgaris or the {lo} foliaceus I don’t know how to pronounce that )
235 Tutor ) OK
236 Zelda and the pemphigus vulgaris is actually life-threatening because it involves um extensively the whole body um and might lead to um )
237 Tutor ) could you just highlight the differentiating features between these two?
238 Zelda ) it it )
239 Tutor )first of all from the experience appearance point of view (.)
240 Zelda um )
241 Tutor which is the la one to give you intact (.)
242 Zelda first pemphigoid will be intact )
243 Tutor ) because there are sub-)epidermal
244 Zelda ) sub-epidermal,
245 Tutor yeh which is the one to give you mucosal involvement? (0.2)
246 Zelda um the pemphi)gus,
if mucosa is involved it’s pemphigus (.) which is the one to give you high incidence in the elderly?

uh pemphigus uh oh {laughing} bullous pemphigoid

bullous pemphigoid OK and which ()

) (^^^sixty) years)

) and which one requires a big a big dose of steroid treatment? (0.2)

) I think pemphigus because it’s life) threatening

} pemphigus usually requires bigger doses of fifty or sixty milligram per day whereas pemphigoid hopefully will respond to a moderate dose of steroid about forty OK so (.) basically pemphigus is more sort of life threatening OK because it will include involve the mucosa, and so on, and as you have sa hinted this is approach to the auto-immune condition and so blood tests to (.) go for when you are thinking of this would be )

) would be immunoglobulin the IgG

anything else any specific antibody?

some form of anti epidermal antibody that will be positive in pemphigus but not in pemphigoid

yeh it is the anti yeh anti skin antibody or the anti-epidermal antibody OK so)

) but because I’ve heard but is it frequent that um what I read they say is that they will actually do the skin biopsy and stain:

) yeh

) is it true that

) yes I think skin biopsy obviously will also give you the relevant changes uh uh relevant features as )well

)I thought is it a bit invasive or is it often that they perform

in general principle any biopsy would be invasive but of all the biopsies I would feel most comfor)table

)yes

about requesting for a skin biopsy because I I have never seen any
complications of a skin biopsy {Students’ laughter} so I won’t have any reservation about that OK? but to the patient it might still mean something very (. ) uncomfortable so they might still not be able to consent to that (. ) OK? so have has some biopsy been done on this patient?

269  Zelda  no they haven’t um:)

270  Tutor  ) why? because the patient )refuses it or

271  Zelda  ) because the:

272  Tutor  ) because the dermatologist is very confident with the diagnosis? (0.3)

273  Zelda  maybe I thought maybe it was that the patient is not that acutely ill it’s just the bullous (. ) formation over the palm

274  Tutor  so what does that mean? {laughs} a patient not not too ill does not mean that we don’t need a diagnosis isn’t it?

275  Martin  so )probably

276  Zelda  )I think maybe the (^^^) with the bullous pemphigus

277  Tutor  bullous what?

278  Zelda  um pemphigoid {laughs}

279  Tutor  so in this particular patient I think for the cutaneous involvement I think bullous pemphigoid is the most likely diagnosis here (. ) isn’t it he is in the right age group, OK? and there doesn’t seem to be any culprit, and we did see an intact blister and there’s no mucosal involvement isn’t it? OK so what has he been given from the treatment point of view?

280  Zelda  I haven’t followed up on that

281  Tutor  why? are you not interested in how the patient is being treated?

{Students laughing a little} (0.2)

282  Zelda  uh no actually it’s because I was looking at other patients yesterday so (^^^)

283  Tutor  so what do you think (. ) might be the appropriate treatment?

284  Zelda  um steroid um maybe with some um (. ) um immunosuppressive drug uh uh low dose

285  Tutor  what do you mean by immunosuppressive drug?

286  Zelda  or or immunomodulating drug

287  Tutor  which one?
azathioprine or (tetracycline)

azathioprine might be a milder form of what we call probably a steroids (bearing) drug here would be most more appropriate because uh uh uh we would have to we are obliged to treat them with steroids but we don’t want to put the patient on too long a period of steroid treatment isn’t it? and therefore we need to put on another what we call steroids bearing drug (0.2) anyway the other thing is um does the lesion on the left foot require extra treatment?

oh oh maybe the some local creams may be given to treat the tinea pedis )

I think probably he would need at least a bit of antifungal treatment probably but depending on whether the cellulitis is getting worse he might even need a course of systemic antibiotic isn’t it anyway a patient with um um profuse blistering eruption might be also entitled to have secondary skin infection so a a course of broad spectrum antibiotic may not be too unreasonable

OK? alright? so perhaps we (.) go back to our objectives, about the past health isn’t it?

 ) mm ha stroke
 ) mm about the stroke
 yeh
 um the common types of cerebrovascular accident )
 ) mm mm
 ) and the sequelae social and economical point of view

mm so in clinical practice what are the common types of strokes that you’ll see?
Zelda: ischaemic hemorrhagic ones or maybe)

Tutor: two big groups OK ischaemic stroke and hemorrhagic stroke and under each of these two possibilities, there are also two other (.) differential diagnoses for the ischaemic stroke the two common most would be what? (0.2) cerebral,

Martin: mm

Tutor: what are the two common types of ischaemic stroke?

Martin: uh: you mean the ) in situ thrombosis or embolic?

Student: ) (^^^)

Tutor: ) thrombotic yeh)

Martin: ) embolic

Zelda: (^^^)

Tutor: thrombotic or embolic isn’t it?

Zelda: lo{thrombotic or embolic}

Tutor: so cerebral thrombosis and cerebral embolism isn’t it (.) for the hemorrhagic stroke we will have a a a as well, two kinds? what are they?

Student: ) (^^^)

Martin: hypotensive or:

Tutor: hemorrhagic stroke )

Students: )(^^^)

Tutor: no no not the cause OK?

Student: (^^^)

Tutor: yes intracerebral and?:

Students: subarachnoid

Tutor: ) subarachnoid (.) we usually don’t include subdural because that is supposed to be traumatic (.) OK so from our medical perspective these are the four main types of stroke that we will encounter and very often history itself (.) will give you the hints OK so: )

Zelda: ) mm mm

Tutor: ) how will this how will the clinical features give us hints (.) as regards how to categorise these types of strokes?

Martin: mm (0.2)

Tutor: first of all how to differentiate between ischaemic and hemorrhagic
stroke (.) in a broad sense OK as you might know there’s always exceptions in clinical medicine Ok but you could just base on general principles

332 Martin uh for the hemorrhagic stroke because there are bleeding inside in the brain there may be signs and features of intra raised intracranial pressure like headache or vomiting um)

333 Tutor ) mm yeh for practical purposes ischaemic stroke would usually give you a more conscious patient whereas hemorrhagic strokes are more entitled to give you a semi-comatose or even comatose patient OK

334 Zelda ) mm mm
335 Martin ) mm mm
336 Tutor so probably the distinguishing feature between subarachnoid hemorrhage and intracerebral hemorrhage it’s quite easy isn’t it? anybody could quote )

337 Zelda ) look at the eye:
338 Tutor the eye, for what?
339 Zelda um for subarachnoid and (^^^) hemorrhage)
340 Martin ) some meningeal signs)
341 Zelda ) oh look for meningeal sign
342 Tutor meningeal sign yes the key word in subarachnoid hemorrhage yes basically the seeping into the meninges and therefore the meninges are being irritated so the patient will complain bitterly of headache, photophobic, and when you do the physical examination there will be Kernig sign, and so so so this is very tell-tale isn’t it?

343 Martin mm
344 Tutor how about cerebral thrombosis and cerebral embolism? (0.3)
345 Martin (^^^))
346 Tutor ) can you differentiate between these ) two?
347 Martin ) we: we need some peripheral signs of history of like uh you uh repl replacement of a heart valve or a history of rheumatic heart ) disease
348 Tutor ) OK this is the first point, source of emboli (.) which would include (.) not necessarily a heart valve replacement but, what)
349 Zelda ) rheumatic heart disease
Martin: ) (^^^) heart disease
Tutor: ) heart murmur
Zelda: ) heart murmur
Martin: ) heart murmur
Tutor: heart murmur anything else related to the heart?
Students: (^^^)
Tutor: arrhythmia OK any others any other positive features to suggest a focus for embolisation?
Martin: ) carotid bruit
Zelda: ) carotid bruit
Tutor: carotid bruit OK so these are always important feel for the pulse, listen to the heart, and listen to the neck OK (.) what else? (0.2) how about the mode of onset? would that be very informative? (0.2) cerebral
Zelda: ) in the um: maybe a very insidious onset )
Tutor: ) yes of?:
Zelda: ischaemic stroke )
Tutor: ) which type of ischaemic stroke?
Zelda: um for the {lo} embolic
Tutor: are you sure? embolic? this is exactly where you would be wrong isn’t it? it’s thrombosis is an insidious onset of hemiparesis hemiplegia whereas embolic stroke it could happen in a split of a second isn’t it? )
Zelda: ) um
Tutor: so the suddenness of onset would be very im very suggestive of an embolic stroke ) whereas
Zelda: ) this is (^^^)
Tutor: insidious onset is suggestive of thrombotic stroke
Zelda: um actually because what I read as well um they said that in haemorrhagic stroke sometimes you can get a patient with uh evolving symptoms as well but do these )
Tutor: ) with what, sorry?
Zelda: evolving um )
Tutor: ) yes yes
Zelda: neurological deficits do we still call that ) insidious onset
Tutor: yes hemorrhagic stroke can also take the form of either a suddenness of onset or insidious one because the blood might be leaking gradually OK? as I have told you nothing is absolutely ^^^^ in clinical medicine, we are encountering more and more patients who come in very conscious, insidious onset of neurological deficit, and then we will have suspected him to have thrombotic or ischaemic stroke (...) but with the advent of the availability of CT brain OK, we are very surprised to find out that in fact those patients are having hemorrhage into the brain as evidenced by the CT (...) OK? so therefore we we have always to be open-minded OK but these are the general symptoms that you might try to (...) uh: make the best bet (...) I believe you haven’t really gone into the details of how he presented in two in 2003 with the stroke (...) or else you might have speculated what is the type of stroke that he might be having then (...) could he recall or he might not be too happy to reiterate the unhappy events isn’t it

Zelda: um I I didn’t specifically ask him

Tutor: OK

Zelda: ) in detail

Tutor: mm but mm {hi} I don’t know but he seemed to recover quite well but hopefully this is an ischaemic stroke isn’t it because cerebral hemorrhage carries with it a worse prognosis OK but at least um have you got any hints that he might have a focus of embolisation?

Zelda: um no, )

Tutor: ) has he got arrhythmia?

Zelda: I there is no arrhythmia and no heart) murmur

Tutor: ) heart bruit

Zelda: no no no

Tutor: mm OK but he did have quite a number of risk factors for stroke

Zelda: yes um like

Tutor: which )suggests

Zelda: ) hypertension,

Tutor: is his hypertension well controlled now?

Zelda: I remember he said on home monitoring {laughs} it’s around a hundred
and twenty for the um) systolic

391 Tutor: ) systolic yeh)

392 Zelda: ) and around seventy to eighty) for the

393 Tutor: ) that’s quite reasonable

394 Zelda: so it’s reasonable

395 Tutor: ) but being a diabetic we would we would like to go for even stricter BP control isn’t it

396 Zelda: {lo} yes

397 Tutor: you’ve just listed his medications, and indeed he has been on quite a number of hypertensive including the ACDI

398 Martin: ) mm

399 Tutor: isn’t it

400 Martin: (^^^))

401 Tutor: (^^^)

402 Zelda: ) yes)

403 Tutor: including the calcium channel blocker, including the beta blocker isn’t it so at least he’s on three types of anti-hypertensives so: uh he it would not be too I think his blood pressure may not be that easy to control (.) although (.) having said that (.) a diabetic patient might have been put on ACDI in quotation prophylactically to protect the kidneys so there’s a low threshold for starting ACDI

404 Martin: ) mm

405 Tutor: on a patient that is diabetic isn’t it OK?

406 Zelda: mm

407 Tutor: what type of beta blocker is he on?

408 Martin: Betaloc)

409 Zelda: ) Betaloc

410 Tutor: is that alright? for a diabetic?

411 Martin: oh ai ah)

412 Tutor: ) do we have to be careful about using beta blockers)

413 Zelda: oh because um for the uh renal)

414 Martin: ) no no for the)

415 Tutor: ) no
Martin for the hypoglycaemia

Tutor yeh because non-selective beta blockers would (reduce) the sympathetic response of a diabetic patient to hypoglycaemia so you might have been alerted that when you are giving beta blocker try to avoid non-selective beta blocker mainly (^^^) isn’t it but Betaloc is a selective beta blocker so it’s perhaps (. ) marginally better OK? (0.2)

Zelda so meaning that um they have to be more hypoglycaemic to present with all the hypoglycaemic um symptoms if they’re on the non-selective )

Tutor {laughing} more hypoglycaemic {Students laugh} yes OK you can say that yeh they are they because they have blunted their response OK for example they won’t run tachycardia because of the beta blocker

Zelda mm mm

Tutor OK? and then? what other risk factors? did you say hyperlipidaemia?

Zelda yes but he’s not on any medication

Tutor did you say Zo)cor

Martin ) Zocor

Zelda oh oh yes he is sorry

Tutor are you aware that Zocor is a stat statin?

Zelda um because sorry um because I have two patients’ notes over here so

Tutor OK. Zocor is a statin which is an anti-lipidaemic agent but I think a more fashionable term to use instead instead of hyperlipidaemia as regards lipid profile risk factors for stroke would be dyslipidaemia OK? why do I say this?

Martin because the (^^^) may not know high LDL or which HDL )

Tutor ) because in the old days we believed that the cholesterol being high would be bad (. ) now we come to realise that the total cholesterol might not be high but it’s the distribution of the lipid profile which is not that healthy OK (. ) a lowish HDL high density lipid has been known to be a risk factor for stroke so that is why uh you call it hyperlipidaemia and you find that the lipid the cholesterol is normal you feel a bit uneasy about using this term OK so dyslipidaemia would be a better term to use

Martin mm

Tutor alright? any other risk factors?
Martin {lo-laughing} sex
Zelda {lo} what
Martin male uh male risk factors
Tutor yes male)
Zelda ) yes oh the ) mono (^^^)
Tutor ) you are the weaker sex {laughs}
Zelda {lo} I remember ) that
Tutor ) any other risk factors?
Zelda gener generally um um (^^^) status, age, gender,
Tutor is he at risk?
Martin (^^^)
Zelda yes sixty ^(^^)
Tutor 2-0-0-3
Zelda oh )
Tutor how old
Zelda by 2003 he was sixty
Tutor sixty just reached risk age OK six)ty
Zelda and um he has also the drinking history
Martin mm
Tutor smok)ing
Zelda ) non-smoker
Tutor any family history?
Zelda uh no
Tutor probably he’s not too happy to answer that question either
Zelda no no family history that I’ve asked that’s specific
Tutor alright .) so he did have quite a number of risk factors .) isn’t it? OK? so: how has the stroke affected him(. socia
psychologically? (0.2) has he become rather depressed all these years after the stroke? has he been receiving adequate care (.) from the family the care givers?

459  Martin   mm
460  Tutor     you said his son is taking care of him
461  Zelda    {lo} yes (.) um sorry actually I should have asked him in detail but from what I learnt that he is only living with his son but no any other family members

462  Tutor     mm mm
463  Zelda     uh: so I didn’t specify about his marital status)
464  Tutor     ) at least you seem to think that his activity of daily living is fairly alright isn’t it )
465  Zelda     ) yes
466  Martin   ) mm
467  Tutor     ) he can walk by himself, he could toilet, he could bathe, OK?)
468  Zelda     ) yes
469  Tutor     alright
470  Zelda     um when I was talking to him um I didn’t really notice any partic he doesn’t look particularly depressive but I would say um he’s not very talkative

471  Tutor     mm
472  Zelda     because I also (^^^) that stroke especially in older patients there is a much increased incidence of depression (. ) post-stroke

473  Tutor     mm mm mm (. ) alright
474  Martin   mm (0.4)
475  Tutor     anything else? have we ) (^^^)
476  Zelda     ) um this is from the case note and not from myself but he is not very keen on the rehabilitation and the podiatry program and he hasn’t been um follow ) up

477  Tutor     ) he defaulted he has defaulted
478  Zelda     yes
479  Tutor     it’s not too uncommon sometimes patients get fed up with all this rehabilitation program so I think he needs a lot of encouragement both
from us the caregivers and from the family members OK? he might have problem going to the rehabilitation centre because there is nobody else in the family to escort him isn’t it that might be the reasons (.). OK? alright? but do pay attention to the psychological aspect of your patient.  

480 Zelda  {lo} mm mm (.3)  
481 Tutor  anything else about this patient that we haven’t really catered for? have we finished all the objectives? (.2) is there something about alcoholism?  

482 Zelda  um I think I haven’t put down)  
483 Martin  ) alcoholism {Cantonese}  
484 Zelda  we didn’t we didn’t put down an objective for)  
485 Tutor  OK so have you finished the job then? (.4) {Zelda shuffling through notes}  

486 Zelda  um yes I have I think so  
487 Tutor  OK perhaps perhaps back to the the point on alcoholism we have discussed briefly about how we might detect on that side whether the patient might be lying to you or not (.). he claimed that he’s not been drinking when in fact he must have been drinking because of his Cushing eye his pseudo-Cushing eyed face because of this Dupuytren contracture, in fact the biochemistry might also have helped (.). but you mentioned that it’s completely normal  

488 Zelda  yes except the whites are under (^^^)  
489 Tutor  how could the CPP and the clin uh chemistry help you to (.). suspect or strengthen the suspicion that the patient might be alcoholic and yet he’s lying to you that he’s not drinking alcohol regularly?  

490 Martin  {laughing}  
491 Zelda  {laughing}  
492 Tutor  how would the CPP help? what would happen to the )  
493 Zelda  ) um:  
494 Martin  ) there might be microcytic anaemia  
495 Tutor  microcytic)  
496 Students  macrocytic)  
497 Martin  uh macro ) macrocytic macrocytic yes
498 Tutor: macrocytosis is a sign obviously it’s a feature of chronic alcoholism OK so is the MCP alright with him?

499 Zelda: um it’s all normal

500 Tutor: OK

501 Zelda: I’m pretty sure

502 Tutor: ah hah how about the biochemistry? what might be indicative of telltale finding?

503 Students: {whispering}

504 Zelda: mm {laughing} he could say

505 Tutor: yes yes

506 Martin: increase in AST more than ALT the gamma increase in gamma GT

507 Tutor: yeh I think if the patient has really been drinking a lot the liver enzymes will be deranged usually (SGOT) more than (SGPT) but if it’s a moderate intake probably the gamma GPT would be elevated because that might be the most sensitive index (.) and it’s quite alright is it? for him, it’s quite alright?

508 Zelda: yes it’s alright

509 Tutor: as I may have mentioned earlier the (^^^) might as well be high uh sorry may as well be low in a patient with alcoholism and that might also be a telltale uh uh uh result OK?

510 Martin: mm (0.3)

511 Tutor: {lo} alright? (0.2) so: any yes

512 Harry: may I know the mechanism for the low potassium with patients with alcoholism?

513 Tutor: uh: I’m not too sure um myself maybe it’s related to the renal tubular uh loss OK because of this renal uh alcohol effect OK but ah I must say I’m not too sure myself about the actual mechanism (0.3)

514 Martin: actually if the patient pre has a uh history of some transient ischaemic of uh transient ischaemic attack before will it be more likely to be embolic or thrombotic (event)?

515 Tutor: um: yes in a way it is more suggestive of embolic problem if there have been frequency of TIAs preceding the actual stroke (0.5) alright OK (.)
so no further queries on this patient?

517 Martin extra (oestrogen can also lead to) blistering formation?

518 Tutor yes blisters uh: it’s one of the cutaneous manifestations of LE but this is very rare and this is not a specific cutaneous manifestation these are non-specific manifestation as you may know, in SLE patients we usually classify skin lesions into specific lesions and non-specific lesions (. specific lesions means ) acute (malar) rash,

519 Zelda ) (^^^)

520 Tutor the discoid lupus and also the (subacute) cutaneous lupus in the two forms isn’t it the psoriaform and the uh uh uh (papillomatosis) form OK? (0.3) right (.) so we’ll move on to the next patient, are you going to present her?

521 Harry yes (. ) our patient a forty-one years old woman presented with two year history of um: (monoarthralgia) of the right ankle, and one year ago the patient also noticed to have a dry dry dry mouth with uh dry eye, and the patient also noticed to have Raynaud’s phenomenon on her hands, and also some systemic symptoms including fever, ma including weight loss and malaise (. ) an: d (there were) no other features of connective tissue disease, an: d on the blood investigation the patient found to have increase in the um: ah: anti(viral) antigen but the (^^^) factor was not increased, and also the patient had a bone marrow biopsy and found to have hypoplastic (. ) uh features in the bone marrow and: this time the patient was admitted for bilateral limb swelling and: and pain, and for the past medical history the patient also has admitted for an episode of intestinal obstruction requiring laparotomy and later she did undergo another episode of intestinal obstruction but she uh treated treated conserv conservatively, (0.2) and: for the family history it was not remarkable, social history she worked as a clerk and: the: (monoarthralgia) didn’t affect much of her daily uh activity activity of daily living, and she was a non-smoker, non-drinker (0.2) that’s: about all for the history of the patient

522 Tutor is she married? (0.3)

523 Harry um: (0.2) no
Tutor: why did you have to think?
Harry: um I didn’t ask
Tutor: OK that’s most likely because otherwise you don’t have to think isn’t it {Students laughing} but anyway I forget whether I may have reminded you, when you are taking history from a patient, especially a female patient OK, another important point would be to ask for menstrual and obstetric history OK, have I reminded you about this last time?
Harry: ) mm
Tutor: so did you ask her?
Harry: I didn’t went back to see the patient
Tutor: OK ) do remember a sing a patient an an unmarried patient does not mean that she might not have been pregnant before OK? so don’t take for granted that if she’s unmarried you can bluff that oh she has never been pregnant OK, this is wrong ) why do we have to be so particular about obstetrical history? (0.2) or for that matter menstrual history?
Martin: it may affect our drug treatment
Tutor: may ) drug treatment OK but 9.) anything else? when we are trying to ) when we are clerking a new patient ) we won’t be thinking of using drugs yet and
Kevin: ) because the patient presented with lower limbs swelling so the patient is more (^^^) {Students laughter}
Martin: (^^^)
Tutor: OK anything else?
Harry: auto-immune disease can cause failure)
Tutor: ) yeh
Harry: ) of the ovaries mm ) or the endocrine problem
Tutor: uh: not not really but nearly you are nearly there)
Martin: ) is it is it some auto-immune disease like SLE may play up during pregnancy?
Tutor: mm OK I think this is still controversial but there’s something more definite is that the obstetrical history is more likely to be more (.) complicated (.)
Martin: uh huh
or less sort of smooth in patients with connective tissue disorders because they are more prone to have what?

anti-phospholipid (^^^))

) no no yes for anti-phospholipid but in general

{lo} they are more prone to

liable to develop miscarriages OK, they might have abortions, in the first trimester or late abortions, they might have greater tendency of intrauterine growth retardation, they might also develop they might also have higher chances of there being pre-eclampsia, OK, and they might also have higher chances of premature labour OK? so all this would be very important (.) especially as you have suggested, if we are thinking of anti-phospholipid antibody syndrome OK? the other issue is is about your your contraceptives, OK, the fact that you have to ask whether the patient is single or married, whether she’s sexually active is because you want to know whether she might be using oral contraceptives OK which might be of relevance if we are dealing with some kind some form of connective tissue diseases like SLE isn’t it OK? (0.3) so in fact just um I want to clarify one point (.) two days ago you tell me that the relatives of this patient seemingly are arguing with the uh nursing staff and I feel so uneasy about this because I don’t see why this patient should have kicked up such a big fuss and I’ve clarified that in fact it’s the patient next to her

hah:? ){Students’ laughter}')

(^^^) I was so anxious to know what)

){laughing}

because so why should you have mixed up this (^^^)

because I was standing at the end of the bed) and um

) but they are discussing on the patient next to her bed

we didn’t hear the content about the argument)

)mm

so we just)

){laughing}

but at least I was relieved to know that it was not her OK? anyway I
would have thought that if this is really the case you would like to explore why isn’t it (.) have you tried to go up and explore why? you haven’t?

560 Tutor mm so I think you need to be more motivated you need to be more (.) nosy for patient (^^^) when I am clerking taking history from my patient I always do apolise to my patient at the end of our interview that I’ve been a bit nosy because I’ve been exploring into or probing into many of their sort of uh uh private uh uh part uh histories but I think that this is important and as long as the patient realises that this will be of help to help (.) analyze her problem she will be more than welcoming to let you know more about herself isn’t it OK so this is one of the techniques that you would like to gain rapport with your patient OK so (0.3) so that’s all for this patient?

561 Harry for the history that’s all

562 Tutor so what are our objectives?

563 Harry um: how to how to define dry mouth and dry eye

564 Tutor yeh (.) so do you think have you read about this?

565 Harry uh )(^^^)

566 Tutor ) do you think that this patient should qualify for the actual dry mouth?

567 Harry I read that for dry mouth we can ask about something called cracker signs which is ask whether the patient have difficulty in swallowing dry food

568 Tutor mm mm

569 Harry and also ask about complications of dry mouth including dental caries, candidiasis or inflammation of the angle of the mouth (.)

570 Tutor mm

571 Harry and:

572 Tutor and also? how about the duration of the actual symptom of dry mouth? you want it to be how long? you want it to be )three months

573 Students {whispering} three months three months

574 Harry )three months)

575 Tutor ) so I think as I’ve told you dry mouth is a very common symptom isn’t
it? all of us might have been having dry mouth in these few weeks uh few days isn’t it? but we are not having sicker symptoms OK because it’s not lasted for more than three months, I’m sure all of you have been swallowing crackers or biscuits with no problem OK, but in those patients with genuine dry mouth they need some water in order to swallow the the biscuits this is what we call the cracker sign OK? and again as you have highlighted they might develop complications from the dry mouth, I think a more specific symptom to qualify for dry mouth in order to help you diagnose (Sjogren’s) syndrome would be parotitis OK? so if you’d like to ask her if she might have episodes of pain over the parotid areas OK because the saliva hasn’t has been so thick and so viscous that they have blocked the salivary duct and to induce this inflammation alright? how about dry eyes?

576 Harry dry eyes you can ask about you can ask the patient about whether there is (set like) substance on the patients eye)

577 Tutor ) we usually describe it as gritty (.) sensation OK? and?

578 Harry and also complications like infections of the eyes because of the dry eye)

579 Tutor ) mm (0.2) anything else?

580 Harry and we can also do some investigations for the:

581 Tutor ) no how about from asking the history what else might be helpful?

582 Chris use of artificial ) tears

583 Tutor ) yes you would like to ask him definitely whether he uh uh uh directly whether he might have been using artificial tears you know in order to help relieve the symptoms of dry eyes OK? again this needs to be there for three months OK?

584 Harry \\{lo\} three months

585 Tutor so I believe you haven’t gone up to the patient to ask her again whether she qualifies for this \\{laughing\} genuine dry mouth?

586 Harry no I didn’t

587 Tutor OK (.) anyway alright so:

588 Harry um:

589 Tutor second learning objective

590 Harry for the next learning objective is talk about the Sjogren’s syndrome,
somehow somebody has diagnosed her as having possible Sjogren’s syndrome, yes but do you think she qualifies for the diagnosis? are you aware that in rheumatology uh: you must have read from the textbook)

) mm

we very often mention about criteria)

mm

for classification or diagnosis of a specific connective tissue disease isn’t it? what do you think of this criteria? (0.2) do you really think we should stick to this criteria like a Bible that we have to follow? (0.2)

no,

no what are these criteria meant for(.) basically?

for research purpose

mm yeh mainly for epidemiological studies, OK, to make sure that the investigators concerned are comparing or are uh doing things on comparable patients OK because it’s no good that you have to stand up to say I am an expert in treating SLE but in fact your group of SLE patients are quite different from a universally accepted group of SLE so we have to have standards of comparison OK so these set of guidelines are not uh meant to be followed when you are dealing with a single patient in the clinical setting (.) but this set of guidelines would obviously be helpful to remind you of the important features that might have to be present if you are suspecting that particular diagnosis and therefore that would uh help you to prioritise your sort of investigation to further consolidate your suspicion mm OK, therefore I don’t think you really need to memorise all those guideline but I think it would be nice for you to be aware of certain guidelines uh: to help you go along when you are clerking a single patient OK? so: I believe you might have read up some criteria for that Sjogren’s syndrome (0.2)

I didn’t have the exact criteria but just some features {lo} of Sjogren’s syndrome)

) yeh I think for that matter for Sjogren’s syndrome again we have got some criteria and basically there are six criteria OK? two of them we
have mentioned already that is the eye and mouth OK so the symptom of dry eyes the symptom of dry mouth, what are the other four?

Joy: oh the other four are ocular signs, for example um the Schirmer’s test positive or the rose Bengal score or ocular dye score may indicate some kind of conjunctivitis yes uh)

Tutor: ) are you are you aware of what I mean by Schirmer’s test?

Joy: yes

Tutor: mm in the old days some of our students would try to do that )

Martin: ) ah)

Tutor: ) on a patient they just grasp hold of some filter paper in the bench (^^^) and then uh uh stick the paper onto the patient’s eye I think this is absolutely forbidden because uh this Schirmer’s paper are specially made OK and they come in uh uh intact forms been sterilised, OK and you have to rip off the package in order to set it onto the eyes of your patient OK? in the old days I tend to carry some of this Schirmer’s paper here in order to demonstrate to the students, but uh subsequently this uh Schirmer’s test bare a cost OK so )

Zelda: ){laughs}

Tutor: so (^^^) we leave it up to the ophthalmologist to do it OK? so basically you will be uh putting this strip-like thing in the outer one-third and inner two-third junction of the lower eyelid, and then put it there for about five minutes (.) as you might know, with some foreign body there your eye will be stimulated and so it will tend to secrete tears OK? and after five minutes this strip should have been wetted five millimeter OK so this is what we call the Schirmer’s test (.) but as you could reckon this is a very crude type of test because obviously it would depend on the humidity of the environment isn’t it you know doing it on a very dry day then the Schirmer’s test might not be that easily wetted isn’t it, OK so but it does give you some idea of whether the tear production is really (sufficient) whereas the Bengal test would obviously be done by the ophthalmologist (.) they are trying to stain the conjunctiva with special uh stain to see whether there’s actual corneal ulcers developing OK?

Zelda: but um then wouldn’t the rose Bengal staining not that be not that
specific to the um um uh syndrome )

612 Tutor it’s more specific

613 Zelda it’s more specific )

614 Tutor ) because they will show up actually the ulcers isn’t it? as I have said Schirmer’s test will test uh uh only the tears secretion so it might not be too:)

615 Zelda ) but then how can we know the ulcers are definitely due to dryness or not?

616 Tutor oh probably you are suspecting (. ) a patient with such a problem already OK? the patient is probably having a dry eye problem coupled with this (^^^))

617 Zelda ) oh:

618 Tutor obviously you have to decide whether there might be some secondary infection to the ulcer as well isn’t it OK?

619 Zelda yes

620 Chris but is this Bengal test similar to the immunofluoroscence stain that you uh)

621 Tutor ) yeh probably yeh (0.3) so: the other?

622 Joy so the fourth criteria will be the doing um biopsy and lymphocytic infiltration, the parotid gland )

623 Tutor ) mm nowadays we seldom go for biopsy of the parotid scan uh gland uh because we found it too intrusive or )invasive

624 Joy ) mm mm

625 Tutor nowadays we usually go for a minor salivary gland biopsy, which is inside the buccal mucosa and usually in the lower lip OK? and basically this is a very uh uh uh mini trauma type of procedure uh and and there has been some (. ) uh histological requirement before you think this is positive you look for what we call a focus which would mean an infiltration of mononuclear cells of more than fifty cells over a specified area of four millimeters square uh uh uh of the microscope field OK? and you would like to count the number of uh this score, if there is present one or more that would be good enough to satisfy for this criteria OK? again, a minor salivary gland biopsy originally I thought it’s rather
non-invasive until one fine day when one of my patient on coming back
to uh the follow up complained to me that after the biopsy he has been
having numbness over this part) of

626 Martin ) hah?)
627 Zelda ) oh:
628 Tutor ) his lower lip for two months OK {Students whispering} and and then I
realised I I I tried to uh feed this back to our surgeon, and the surgeon
indeed told me that this is a recognised sort of complication but in the
old days (.) or previously uh we have not met with this and so we have
not really reminded our patient that this might occur so I think this is
important isn’t it?

629 Zelda mm
630 Tutor try I think this is a learning process for all of us in clinical medicine isn’t
it when you come to know something about your patient’s problem you
would like to integrate it into your subsequent interview with your
patient so that the patient would be better informed isn’t it? OK?

631 Martin mm
632 Tutor so a minor salivary gland biopsy would be much much less invasive than
a parotid gland biopsy but having said that if the parotid glands
definitely show a lump, or a a a a a induration you would like also to do
a a a biopsy on it isn’t it to make sure that you are not dealing with
something else

633 Martin mm
634 Tutor OK? so the last two criteria would be:? 
635 Joy um the salivary gland involvement mainly that you’ve mentioned the
amount of saliva production, uh or you do a (sinography) or
(scimometry) (. ) uh scint scintigraphy sorry

636 Tutor in fact (.) we are not doing all none of this now OK? first of all
unstimulated salivary flow probably is a rather complicated or um
cumbersome procedure so uh: we don’t routinely do it, OK, and um
parotid um gland um what dya call it (0.3) adductogram? I ca can’t
remember (. ) they actually cannulate the duct and then to to see whether
the ducts have been narrowed with this inflammation, or the other one
would be the scint scin scintigraphy that is to the the radio isotope of uh skin OK? but perhaps nowadays we would like to go for a MRI scan if we are aware or we are worried about some tumor uh arising from this paro uh (^^^) OK so the last category would be autoimmune markers isn’t it? anti-ro or anti-la, anti-nuclear factor or rheumatoid factor as I may have highlighted to you last session, rheumatoid factor positivity is almost uh a very well recognised feature of Sjogren’s and and I highlighted uh indeed very often it would give you the highest (^^^) isn’t it?

637 Students  mm

638 Tutor  so if the patient has three out of these six criteria present you could suspect the patient highly that the patient is having Sjogren’s (.) if he is having four out of six it will be even uh uh more suspicious OK? when we are having this criteria for diagnosis very often you might come up with terms which we call sensitivity and specificity isn’t it?

639 Students  mm

640 Tutor  so the test is sensitive, that means it’s very commonly present but it might have a lot of false positive uh: situations (.) when a test is very specific it will have very little uh or very few false negatives uh values isn’t it ) so

641 Martin  ) mm

642 Tutor  so this is uh how we would like to (avail) of this test (.) but having said that (.) what preliminary conditions we have to rule out before we apply this criteria to the uh diagnose our patient as having Sjogren’s

643 Martin  (^^^))

644 Tutor  ) that is what conditions might in fact give you all this set of symptoms but in fact the patient is not having Sjogren’s just because he is having that particular condition(0.4) what is the most commonly talked about most dreaded but luckily very uncommon complication of Sjogren’s?

645 Zelda  )may be lymphoma

646 Tutor  ) what may be lymphoma OK, a small proportion of such patients might evolve into lymphoma, and that is something we would always like to monitor OK? so if the patient is having lymphoma this set of criteria is
not applicable (.) OK? (.) anything else? (0.2)

Harry for the learning objectives uh:)

Tutor ) no no no no no (.) I’m still mentioning about some conditions which have to be excluded before you would like to apply this criteria to diagnose Sjogren’s syndrome (0.4) in relationship to:

Zelda do we need to ) rule out

Tutor leukaemia, yes?

Zelda oh oh oh sorry I was thinking about it might be a mixed connective tissue so do we have to:

Tutor no (.) that would bring us to the question of whether this is primary Sjogren’s or secondary )

Zelda oh:

Tutor Sjogren’s (.) so the patient could have a primary disease of Sjogren’s but if the patient has mixed connective tissue disease he’s certain of having MCT)

Zelda oh

Tutor with secondary Sjogren’s so this is still Sjogren’s syndrome ) OK?

Students mm

Tutor but what I’m trying to say is graft versus host disease)

Students oh

Tutor might also sometimes give you this set of symptoms you might remember, those patients might complain of dry mouth, dry eyes and so on isn’t it? and a very uncommon condition in this locality, perhaps in the Caucasian world it’s much more common, is sarcoidosis OK?

Zelda {whispering} sarcoidosis

Tutor I bet you haven’t seen any one patient with sarcoidosis (.) in clinical teaching? I think I have only three or four such patients after so many years of {laughing} uh service here OK so but I think in the UK)

Student I yes I saw yes

Tutor you will have ) sarcoidosis clinic isn’t it?

S ) (^^^) yeh

Tutor this is very interesting isn’t it? Chinese patients are sort of immune to sarcoidosis although admittedly we seem to be seeing a bit more (.) OK
but as with many other diseases the local diseases pattern seem to be changing we are not too sure whether it might be related to the dietary change isn’t it? because our diet is more and more Westernised now isn’t it OK? and the fourth one would be the HIV infection OK so we would like to exclude all these four conditions before we apply these criteria to the diagnosis of Sjogren’s alright? so again we would like to know whether this is a Sjogren or secondary Sjogren isn’t it so how do we apply this to our patient then? do you think she qualifies for the diagnosis of Sjogren’s syndrome?

667 Harry mm:

668 Tutor you haven’t gone back to ask her about the dry mouth?

669 Harry no:

670 Tutor I don’t think the dry mouth is too: impressive OK she hasn’t got any dry eyes she is positive for anti-Ro OK? but otherwise

671 Harry (^^^)

672 Tutor I don’t think she qualifies for that diagnosis yet OK? but as I’ve said this set of criteria are not very important when you are dealing with a single patient, you would still like to bear this suspicion at the back of your mind as you are monitoring his her progress isn’t it? OK? but then what might be another uh possible diagnosis for her?

673 Harry (^^^)

674 Tutor her other major symptom would be Raynaud’s phenomena

675 Martin mm

676 Tutor wouldn’t it? how how would you like to qualify a patient as having Raynaud’s phenomenon? what are the features? what kind of changes?

677 Harry yes uh the hands will turn pale uh they turn blue and then they turn red because of the vessel change

678 Tutor yeh so basically these patients would have vascular instability so on cold exposure the vessels would be constricting and so it would induce a pale or white colour (.) and then: obviously when the supply blood supply has been has been compromised for some time the tissue would become anoxic, anoxic, and they become blue, and then, as a physiological reaction to hypoxemia the vessels would dilate and so finally we will
have the reddish discoloration so it’s a change from white, blue to red which constitutes the Raynaud’s phenomenon but in clinical practice you need a very observant and intelligent patient to tell you these changes isn’t it? but I don’t advise you to provoke this Raynaud’s phenomenon in your patient (.) some students are so so: motivated they would like to ask the patient to immerse a pair of hands {laughing} in icy ice cold water (.) I think this is forbidden again OK because this could be basically dangerous and induce cold injury to the patient OK? but as long as the patient complains to you of some bluish discoloration, I think it would be reasonable for you to suspect her to be having Raynaud’s phenomenon OK? so (.) if this is really Raynaud’s what would be the most important or common connective tissue disease that you would like to bear in mind?

679 Harry (^^^) connective tissue disease and systemic sclerosis
680 Tutor yeh I think systemic sclerosis or scleroderma which tops the list when you are dealing with patients with Raynaud’s phenomenon OK? and then the next one would be the mixed connective tissue disease (.) although I think I may have hinted to you that this mixed connective tissue disease the term is being a bit controversial (.) some investigators do not believe that this is a separate entity OK but historically what do we mean when we try to label a patient as having mixed connective tissue disease?

681 Harry it means the clinical the clinical features of the patient have the overlapping in the systemic (^^^) SLE and then myocites
682 Tutor OK and then in their blood we would like them to have what?
683 Harry anti-RNF pattern
684 Tutor anti?)
685 Zelda ) RNP)
686 Harry ) RNP
687 Tutor what is it?
688 Harry it is a kind of (.)
689 Tutor what is the full term for RNP?
690 Zelda ribonuclear protein
Tutor  yeh ribonuclear protein (.) a high titre of circulating anti-RNP (.) OK? so: has he got RNP?

Harry  no

Tutor  no isn’t it so he’s just got anti-Ro so again he she does not seem to fit isn’t it

Harry  mm

Tutor  so therefore in this particular patient she seemed to have a bit of dry eyes uh dry mouth, she had Ro positivity, she had Raynaud’s so perhaps the best term to label her here would be undifferentiated connective tissue disease (.) I think this is a very useful term to use when you are suspecting a patient to have some form of connective tissue disease, but not that classical of any defined pattern OK? (0.2) so what are the other objectives?

Harry  include the reason of the uh intestinal obstruction, in our patient

Tutor  I’m afraid you haven’t gone up to ask her whether she has an operation in the past which might have started off the intestinal uh the abdominal uh peritoneal adhesion which might induce this recurrent intestinal obstruction isn’t it OK? but (.) in a patient with a connective tissue disease, do you think she is entitled to have symptoms of intestinal obstruction?

Zelda  {softly} I worry about Crohn’s disease as well or is that too far {louder} because um I what I read is that since Crohn’s disease is also autoimmune disease

Tutor  mm mm mm

Zelda  and due to the nature of that it involves the (whole thickness) of the um)

Tutor  ) yeh

Zelda  ) yeh but Crohn’s disease patients would be seldom seldom be under our care (.) they are under the care of the GI physician

Tutor  ) yeh but Crohn’s disease patients would be seldom seldom be under our care (.) they are under the care of the GI physician

Martin  hhh

Tutor  but how about patients under our care

Chris  a patient with scleroderma will have impaired GI motility constipation
or intestinal obstructions (.) or sometimes uh pseudo ( ) obstructions

707  Zelda  ) pseudo yeh paralytic hernias

708  Tutor  yes yeh I think pseudo-intestinal obstructions is the key word that I would remind you of in patients with connective tissue disease OK? probably it’s related to a segment of gut being immobi uh uh hypomo hypo hypo um: uh hypomobile perhaps I don’t know hypomotility of the gut of a particular section of intestine, which would um uh uh literally uh stop moving that’s why the patient would present as if she’s having an (^^^) obstruction

709  Zelda  mm mm

710  Tutor  but this is a very rare occurrence this is something for you to bear in mind because if you’re not aware of this you might like to go in ( . ) to explore and once you go for laparotomy ( . ) as I’ve told you earlier you induce scarring round the gut and that would activate further obstruction but this time it’s a real mechanical obstruction because of the adhesion isn’t it OK? so the classical teaching on managing patient with pseudo intestinal obstruction is not to do laparotomy as far as possible because the scarring, as a sequela to the laparotomy, would further aggravate the intestinal obstruction by mechanical means ( . ) OK? I think GI tract involvement is not that common in connective tissue disease OK but somehow scleroderma perhaps is somehow one of the conditions that might have more of GI manifestation, and the other common uh uh condition which we might encounter in hypomotility of the gut would be the stagnant gut syndrome where part of the gut is being stagnant, so, not to the extent of causing obstruction but they would lead to an overgrowth of bacteria isn’t it and that would also induce non-absorption of the patient ( . )

711  Students  ) mm mm

712  Tutor  isn’t it? dysphagia is also a uh uh a very interesting symptom in connective tissue disease OK? patient with Sjogren’s syndrome would be ( . ) uh having dysphagia because of this lack of saliva, isn’t it ( . ) OK? but can you quote other examples of connective tissue disease patients having dysphagia?
Harry mm

Zelda ) dermatomyositis

Tutor ) dermatomyositis yeh

Zelda the mouth being uh the tense skin

Tutor no dermatomyositis would actually lead to the muscles involvement OK?

Martin mm mm

Tutor which part uh of the uh uh esophagus would be affected? (0.2) it’s the laryngeal muscle

Zelda oh:

Tutor it’s the initial part of the esophagus because this is the skeletal muscle isn’t it?

Zelda oh

Tutor whereas in scleroderma?

Zelda oh it’s the (^^^^)

Tutor ) the patient yeh the patient would also have dysphagia but it would tend to affect the lower part the lower one third of the esophagus because it would tend to affect the sphincter and the smooth muscle OK? so these are something of interest isn’t it?

Students {whispering} (0.3)

Tutor another important and not uncommon not not common but uh very specific GI manifestation in connective tissue disease would be what we call protein losing enteropathy isn’t it? are you familiar with this term? (0.2) have you) heard of this?

Zelda ) I have I heard heard about this term

Tutor mm you are you must be very familiar with (nephrotic) syndrome isn’t it?)

Martin mm

Tutor in which the patient with connective tissue disease (^^^^) nephritis is losing protein excessively through the kidney (.) isn’t it? and that would induce fluid retention, (bilateral^^^) oedema, puffiness of face, and even uh ascites isn’t it? so in the same token a patient with connective tissue disease might be losing protein through the gut so that the patient might
be equally hypo(^^^^) to induce this generalised fluid retention as if she is having nephrotic syndrome (.) OK? so the lesson to remember is that where the patient is suspected to have SLE, scleroderma or for that matter UCTD, when the albumin is very low and the (^^^^) value and (quantification) of urine is normal, try to think of this particular entity (.)

protein losing enteropathy and we need a very sophisticated and special test to delineate this and this is what we call a (^^^^) (clearance) this test (.).

have you heard about this test before? I don’t think this is too important but I would like you to remember this particular entity.

Zelda um what is the mechanism again for the protein losing enter)opathy?
Tutor ) probably it’s probably due to the vasculitis of the gut so)
Zelda ) right:)
Tutor ) the gut vascular uh vascular the vascular uh: permeation of this protein (.)

uh and then they are all lost in the stool OK? (0.3)

but then it only affects protein absorption but not all the other )
Tutor ) not absorption
Zelda ) (^^^^)
Tutor ) it’s secretion yes (.)

they are losing the gut (.)

they are not it is not a malabsorption syndrome, it’s different (.)

OK so ) they are just losing the protein
Zelda (^^^^) they are losing the protein from the body )rather than
Tutor ) yeh yeh yeh (0.4) {Students whispering}
Martin is it associated with (other) sorts of connective tissue diseases?
Tutor uh most likely lupus related diseases OK? and in the past I think we find that anti (BIP) perhaps is a is a risk factor for this particular complication
Martin B I P?
Tutor yeh {Students whispering BIP} (0.4) OK so other objectives?
Harry that’s all for the objectives yes
Tutor I think have I not mentioned that (.)

we seem to have rather uh inappropriate or discrepancy )

Harry ) yeh
Tutor ) between the signs and the symptoms as regards her pain in the ankle?
We would like to find out a more objective way to assess whether her ankle is really that sort of affected to induce this (^^^) symptoms all the time she has been complaining of pain around the ankle so that she has not been moving uh: very comfortably OK? so: again I think you haven’t gone to see the patient (.) what particular investigation has been done on her(.) which perhaps might have been of help to assess her ankle pain? we discussed about the fact of X-raying her ankle which is likely to be normal, because the X-ray is not a very sensitive method to detect changes in the acute stage, so what may be the ) other ultrasound OK OK in fact we have done an ultrasound right ankle on her OK um nowadays ultrasound and perhaps magnetic resonance imaging have become more and more important in the assessment of musculoskeletal symptoms in our patients because they are found to be much more sensitive in detecting early changes and they would also probably in due course become the definitive outcome marker for treatment responses OK? so as I have told you when we examine her ankle the ankle did not seem to be swollen, did not seem to be too tender, and we can’t really detect any collection of fluid at all OK? when the joint is swollen what might be the cause for it?

obviously all the components of the ankles might contribute to the swelling so it’s easy for you to remember OK? so the most superficial part of the)

) skin (^^^))

) skin, cellulitis might also give you a swollen joint which might be misleading but in fact this is the cellulitis OK? and then?

the synovium

OK before the synovium

the (^^) subcutaneously

muscle muscle involvement OK but usually the muscle would not be that plentiful around the joint OK so it’s very uncommon but what
would be covering the joint?

Students the capsule)

Tutor ) the capsule the tendon OK so it could be the tendonitis or the capsulitis (.) and then we go into the joint with the synovial synovitis, and then in the joint cavity itself it would be the joint fluid OK? and then still deeper would be the actual bones the joining surfaces of the two bones in the joint OK so all these structures could become swollen OK? and so the ultrasound would be able to detect all these perhaps probably not all that good for the bone texture but for the synovium, for the fluid, for the capsulitis, it would be very uh: adequate (.) and in fact it did show up a bit of fluid collection in the interior recess of the right ankle but the synovium is not supposed to be inflamed and the tendons are also alright OK? so what do you think the radiologist might have done on her? can you detect something?

Student }lo} (aspirate)

Tutor yeh we have tried to aspirate the joint and somehow um zero point five cc of fluid)

Students ) (hh)

Tutor is drained out so it’s just a tiny drop and even with that tiny drop they are able to send it out for analysis OK? in this particular patient I don’t think septic arthritis is too likely isn’t it? but that will come to the issue of when you are getting some precious specimen you have to decide on what to do with it OK? I would think this tiny drop would be very precious to be sent to the microbiology department if you are worried about her having septic arthritis isn’t it? but somehow this has not been sent to the microbiology department (.) for what? what might be more (.) most important what might be the most important ) (^^^)

Martin ) crystals

Tutor uh: for crystals? it’s not done by the biochemistry department (.)

Martin }whispering} biochemistry

Tutor sorry not for the biochemistry the mi by the hematology department

Students }whispering}
for counting the cells in the joint fluid OK I think joint cell count is also helpful because there would be good ranges of cell counts which would help us to define whether the joint fluid is inflammatory, inflammatory or infective (.) these are the three main types of joint (effusions) that you will see (.) and the results come back to show only five cells per cc which is very very low I don’t know why it’s so low so five wbc

zero point five cc

oh they would just do the calculation OK but that means that we are quite comfortable that the patient is probably not having an inflammatory response isn’t it? an inflammatory synovitis would induce much much higher wbc count OK? so all in all we think this is probably a mechanical uh cause to cause the joint pain and it might uh the continuous irritation might lead to this very tiny fluid collection OK so what we have done would be to convince the patient uh that she is not likely to be having inflammatory arthritis but as she has been labeled as being anti-ro positive she is all the time worried about her evolving into a more definitive connective tissue disease on the other hand indeed she had past history of trauma to her right leg and so when you look at her she might have a sort of uh uh unhealthy uh uh sort of mechanical uh uh structure of the right leg and so we are uh sending her to the physiotherapist for uh gait analysis to see whether in fact some adjustment of her shoes or whatever might have uh offset this uh mechanical insufficiency that she might be having (.) at the same time we have also referred her to the podiatrist for the specific uh uh tailormade footwear for her OK? so hopefully that might have solved her problem

OK? (0.3) so: any any other objectives that we haven’t really touched upon? (0.2) alright? so: I think that’s about all about this patient, any questions on her?

the patient mentioned that she has to undergo uh routine check up of her serum marker to see if there is any disease

mm
Harry so what should be monitored in the blood test?

Tutor what do you think? (0.2)

Harry I think it should be rheumatoid is it rheumatoid rheumatoid factor?

Martin mm

Tutor mm

Harry or the SR

Tutor yeh if you are suspecting Sjogren’s syndrome, um for the markers serological markers perhaps, yes it would be reasonable for you to mention the rheumatoid factor because I have hinted to you that perhaps less than five per cent of such patients might evolve into lymphoma (.) and we reckon that rheumatoid factor titre would tend to fall when they are evolving into lymphoma problem so if the originally sky high rheumatoid factor suddenly becomes low (.) we have to be worried about this evolution, OK? so that is something that you would like to monitor OK? but (.) for lupus like disorders what do we usually monitor?

Harry anti (DS) DNA

Tutor sorry

Harry anti (DS) DNA

Tutor yes (.) the DNA titre, and:?

Harry and the ESR Chris Chris four four Chris Chris three Chris four

Tutor yeh I think the anti DNA titre and the complement levels are the most important uh parameters for us to monitor a patient with lupus or lupus related diseases OK? um: the ESR is worth monitoring but it’s too non-specific isn’t it? it would be monitored anyway OK? in her I think it would be a bit difficult with regards to monitoring her serum markers because first of all we can’t really define a specific diagnosis on her yet isn’t it? uh I think for her the monitoring would be more a sort of clinical monitoring to ensure that she’s not developing more uh features to help us to delineate her connective tissue disease better OK? for example if she happens to have more and more joint pain, in a more symmetrical distribution, that would be ind more compatible with a lupus like or (RA) problem OK?)
or if she evolves into having more rashes on her face no matter erythema or discoid lupus rash then again we have to be more worried about lupus isn’t it but (. .) at this moment I’m always trying to be to reassure our my patient that she’s not having any serious connective tissue disease

indeed, anti-Ro antibody um could be found in otherwise normal healthy individuals although this is much much less uncommon than (^^^)

I’m sure all of you are familiar with the fact that false positive (NA) can be present in otherwise normal healthy individuals as well.

although having said that it’s interesting to remind you that there has been a very important study uh: a few years back to show that in fact many lupus patients their serum would be positive for ANA, positive for anti DNA, positive for anti (SM) years before they actually develop the disease so that particular paper would make us a bit worried (. .) and that would mean that the so-called false positive ANA patients might perhaps not that false positive different time they really might be relevant to lupus patient but um still I can’t really uh uh uh um um concur with the fact that all of them would evolve into SLE (. .) I’m sure there are some genuine false positive ANA patients OK? so (. .) that is why up till now a patient with anti-Ro positivity we dare not not to follow up that OK? but for patients with only positive ANA and nothing else and if they don’t really have any other features we are still relaxed about it and sometimes we would tell that probably they would not need any follow up (. .) OK?

and you mentioned that we have to monitor the patient for lymphoma do
we do it clinically or by:)

805 Tutor } just clinically

806 Martin clinically

807 Tutor by uh feeling for lymph nodes and again watching for the systemic symptoms

808 Martin mm (0.6)

809 Tutor OK? any other questions? (0.4) so if not we will call it a day thank you

810 Students thank you
Tutorial 4
Medicine Specialty PBL Session

Students: Harry, Eddie, Zelda, Martin, Eric, Joy, Gladys, Becky, Kevin

1 Tutor I believe some of you may be taught by her or some of you at least during your uh first and second year for English right sorry her English is better than me (.) during my university I didn’t have this sort of opportunity it’s a bit scary (.) I mean we didn’t uh we didn’t have any uh: English uh teaching at that time so we all learned by ourselves (.) anyway so these are problem based learning, and (.) do you want to explain something to (them (^^^)).

2 Anne no ( they know me they know me yeh this is how many times now four or five already I think.

3 Tutor oh they have been taped four or five times already, ( oh::

4 Anne yeah ( they have yeah.

5 Tutor OK so you’re used to that, I’m not used to that, so you speak more ok (.) anyway so uh: let’s start because I need to end at ten sharp because I have a grand a round (chairs scraping) (0.2) I hope they are not bored (.) ok oops your badge (.) ok so today we have two: cases (.) I know I think you are all familiar with the uh problem based learning right (.) problem based learning is something that uh: you learn (.) mostly by yourself (.) I am the facilitator (.) of course uh now in the last few years um: as some of the students are very good they talk (.) they are well prepared so that they can have good discussions for many (.) many minutes but some are not too good (.) they can only talk less than three minutes and then everything’s silent and then I need to teach spoon feed again so I don’t want to be uh like this (.) hopefully (.) so ah you know what I mean uh so at least my deadline is at least you need five minutes of talking by yourself (.) OK it is a minimum time for me OK (.) alright so who is the one who clerked the first case and want to discuss with the group?

6 Harry ye:s (.)
Tutor: don’t be shy, don’t be shy, just go on.

Eddie: yeah we got two cases.

Tutor: yeah so you decide which is the first case and then you present OK (. ) how you decide yourself ok please.

Harry: Madam Wu a eighty three years old woman uh presented with three week history of generalised weakness (. ) previously uh: Miss uh: Madam Wu was activity of daily living dependant and having coughing and dressing uh need to be held by others (. ) three weeks ago uh the patient uh uh noticed to have generalised weakness involving all the four limbs and: the patient can only rise her arms but she uh but she cannot eat or write (. ) and also the patient prefers can walk with a quadropod but three weeks ago the patient start to (. ) uh unable to walking, umm further questioning there wa have been um no history of dysarthria, diplopia or respiratory distress from the patient (. ) and it was not associated with any sensory deficits or um uri uh urinary or bowel incontinence (. ) her weakness is not associated with any (flex) ability and also (^ ^) muscle tenderness (. ) from further questioning the patient had changed (. ) his anti antihypertensive medication and had a flu vaccination one month ago, otherwise the patient didn’t have any alcohol history or chronic liver disease or diabetic (. ) um um: the patient also have some uh specific complaints and the patient has been having (sternal) chest pain and headaches for one year (. ) regarding the (sternal) chest pain it is central in the heart in the chest without radiation (. ) and it is not related to exertion (. ) and the duration is about a few minutes each time and uh and there mm no otherwise problem (. ) and for the headache the patient also having headache for one year and it is general generalise headache without any neurological deficits or it is it is not related to uh vomiting (. ) um:: from the uh from the history taking the patient also have some depressive symptoms and the patient have been unhappy for about a few months (. ) and (. ) she also claim to have loss in interest and lack of energy and for most most of her time in the week (. ) and her appetite is not good (. ) and for the past medical history she has been uh diagnosed to have hypertension for several years and also hyperlipidaemia (. ) she is under follow-up with medication.
and her blood pressure uh: was uh: about one hundred and eighty during her follow up in clinic

11 Tutor OK up to now any questions you want to ask (.) so he has mentioned some of the problems of this lady (.) it’s a lady right?

12 Harry a lady

13 Tutor which ward is the lady in?

14 Harry B1

15 Tutor OK (.) so anything:, is it the fat lady or thin lady?

16 Harry the fat lady

17 Tutor OK OK I I I may have come across this lady already (.) so anyone who wants to ask the questions about this old lady

18 Zelda umm I want to ask about the onset of the generalised weakness when did it come on and under what condition

19 Harry three weeks ago (about)

20 Zelda no I mean um was it a gradual or acute ( onset

21 Harry ahh ( yes it is gradual

22 Zelda gradual ( onset

23 Harry gradual ( yes

24 Zelda and under what circumstances she first noticed it?

25 Harry (0.2) mm: I didn’t ask about this (.) but the patient said that then she cannot walk (.) previously she can walk with a quadrupod but she she cannot walk since three weeks ago

26 Zelda mm mm

27 Martin is there any reason the patient need to walk with a quadrupod

28 Harry uh:: because the patient complain of uh lower limb weakness before

29 Martin already have lower limb ) weakness

30 Harry yes ) but there have been no history of stroke

31 Zelda but does she um complain of like pain in her knees or

32 Harry uh it’s not pain related

33 Zelda ) it’s not pain related

34 Martin ) just weakness

35 Eric did you say the patient couldn’t eat and write
Harry: because of the weakness of the muscles of her hand of her hands which part of her body is most suffering from the generalised weakness (or is it equally distributed)?

Harry: umm the generalised weakness is symmetrical and for the upper limbs it is the distal part that is more affected (.) for lower limbs I think the whole limb is affected

Students: {whispering} (0.2)

Gladys: for the lower limb weakness how long have it been start

Harry: uh: she she said that it’s about three weeks or so

Zelda: ( so (^^^) quadropod (for three weeks)

Harry: ( uh for several years

Zelda: for several years (0.2) uh does the so for the lower limbs is there like the onset of the weakness does it start in the lower limbs first or the upper limbs first or does it start )

Harry: toge)ther (0.3)

Zelda: ) together (0.4)

Tutor: oops

Zelda: (^^^))

Tutor: so: satisfied ) with all the history?

Zelda: umm no uh so you said she was ADL dependant who is it she lives with and who is she dependant on?

Harry: uh she is living in a old age home, for fam for social history, uh: she has a husband but died and she has a son but working in Macau so nobody so no body take care to take care of her, so she is living in an old age home (0.5)

Zelda: mm mm and you mention about depressive symptoms have you actually assessed her suicidal risk?

Harry: ( ummm

Becky: ( actually we have read uh by the time we are clerking the case, a psychiatrist um is coming to consult her, and we can read from the notes that um she is suicidal

Zelda: uh but you didn’t ask her
Becky: um we didn’t

Zelda: mm mm but did she actually attempt it in the notes

Becky: um: (.) not ) attempt

Harry: ) not attempt

Zelda: not attempt but ) (trying to)

Becky: ) but trying to kill )

Zelda: ) was there any plans ( or

Becky: ) (^^^)

Zelda: I just thought )

Becky: ( planning to die (^^^)

Martin: mm (0.3) is is is the weakness started after the depressive symptoms (. or is it because he she can cannot walk or something like that that she develop depressive symptoms

Harry: um:: I think um: just the depressive symptoms have been for several months but uh because it’s about three weeks only (0.2)

Students: mm mm (0.5) {whispers}

Eric: then do you ( think

Joy: how ) you first you first you first

Eric: do you think it’s possible in this case the generalise weakness it is due to psychogenic factors

Harry: um: it is possible but medical factors um: seems to be more more reasonable (. ) because from the investigation results the patient has um: the sodium level of the patient is uh reduced)

Eric: mm )

Harry: so I think that generalised weakness can be due to hyponatraemia

Zelda: mm (right)

Martin: what but what hypertensive any hypertensive medication is the patient taking

Harry: um: I can recall uh she she was on a HCI and also on a diuretic

Martin: aah )

Zelda: where is she following up for her hypertension?

Harry: I didn’t ask because she was quite tired at that time
Zelda: oh: so um but uh did she mention the reason for changing her medication a month ago?
Harry: uh no
Martin: so you mean the new drug has been added to the )
Harry: ) the dose have been changed
Martin: oh change in dose
Harry: yes
Eric: that means uh still the two drugs for the hypertension
Harry: yes
Zelda: have you assessed her compliance
Harry: no
Becky: we think we think that um she is living in an old age home, uh so )
Zelda: so) uh people are taking
Becky: likely (0.3)
Martin: (^^^)
Becky: ( (^^^)
Zelda: how about ) her hyperlipidaemia is she on any medication
Harry: um:: yes yes
Zelda: oh on on statins,
Harry: I think so (0.4)
Zelda: any so how about her past medical history any previous (0.1) stroke or
Harry: uh no previous stroke and only hyper tension hyperlipid lipedemia and a
minor surgery
Zelda: minor surgery?
Harry: thyroid for (nodule ^^^)
Students: mm mm
Harry: (so total) for that (0.3)
Martin: (mm
Zelda: mm)
Eric: did the patient have any uh hypothyroid symptoms?
Harry: because it was a (^^^) so I so I don’t think she has she she has she has a
problem (. ) and from the medication they have no history of long term
thyro(^^^) so I think the thyroid function should be OK

but the thyroid nodule was it functioning or diseased

I can’t know about this because it was done in 1994 and the patient has no idea about it

so how was it discovered (0.3)

mm: I don’t know (0.6)

coughs} so so far can you just with the history come up with any differential diagnosis (0.1) before you go to exam of patient) you know some of the questions are quite valid (.) so uh I know you are thinking something so (.) can you just list (.) on the possible (.) make it a um possible (orders) for (^^^) diagnosis for this lady (.) anyone can do that (.) or the one who clerked

uh yes

I think you you sense what your colleagues ask you should be meant, I mean they are they are hinting of some diagnosis right (.)

mm

so what kind of diagnosis can you think of just from the history

um: for generalised weakness I think uh the causes can be divided into central nervous system or systemic problem (.) for systemic problem I think it is it can be due to the hyponatraemia

uh huh

and an: d also (.) and that’s also systemic and ( because

just ( hyponatraemia

and because ( uh the

but uh: in other cases what other systemic symptoms can lead to generalised weakness)

um:)

you mentioned hyponatraemia but this is not so common actually for hyponatraemia

other causes

what other causes you better try something more common

an: d also hypokalaemia
OK potassium

yes the potassium level of the patient is low

I know I know but ah you don’t know, you just look at the history, right, you look at the file you know the potassium sodium is low but you are thinking of a fresh case maybe you are the intake MO right, so you know you don’t know everything (^^^) at all right, so what are the other causes possible causes or systemic causes

Harry ( (^^^)

myasthenia ( gravis

no no it’s the neurology ) (systemic causes

oh )

Students ( (^^^)

other ) causes include hypothyroidism, or adrenal gland insufficiency

now adrenal insufficiency,

yes

but could it be the other way round too?

uh Cushing’s syndrome uh can can also cause general generalised weakness

how about the thyroid

uh: hypothyroidism

hyperthyroidism can also cause generalised (weakness

yes )

OK (^^^) (0.2) what else (.)

{whispering}

and (.) and then I can think about the neu neurological problems

mm mm

that is cephalomyelopathy

mm mm )

causing ) the weakness of the four limbs (.)

mm mm )

and also myasthenia gravis but the patient did not have any (fatiguability) so it is unlikely, and then I can think of about the uh um peripheral
neuropathy

157 Tutor mm mm

158 Harry for example uh: uh: Gu Gu Guillan-Barre syndrome, because the patient also have a previous influ influenza vaccination taken about one: month ago

159 Tutor mm mm

160 Harry yes but physical examination findings are incompatible

161 Tutor oh we are not talking about physical examination now (. ) forget about (^^^)
   forget about the actual(ise) just based on the history

162 Harry and ( 

163 Tutor this ) is always a possibility but uh:: it is actually not not very common (. )
   it’s very rare

164 Harry mm

165 Tutor so you can think of this as a academic sort of possibility

166 Harry mm

167 Tutor but I don’t think it’s really)

168 Harry mm

169 Tutor if it’s a real case then the vaccine, we’d be quite scared about the vaccine

170 Harry mm yes yes

171 Tutor OK do you have vaccine

172 Harry uh no

173 Tutor some of you you have right

174 Students mm

175 Tutor so uh let’s keep our fingers crossed and don’t don’t hope that this is a real case of TBS

176 Harry mm

177 Tutor my parents also have vaccine too (. ) I didn’t have yet so I can wait and see OK no other disease

178 Harry other disease uh:

179 Tutor OK you have neurology ok neurology can mean a lot of things so
   (^^^myelopathy ^^^) peripheral neuropathy (^^^) syndrome blah blah blah all these right,
Harry mm yes (.)

Tutor right so: on top of that other you asked some questions that were irrelevant some of you asked about medications who asked about medications (0.2)

Zelda uh I asked if she has recent change of the dosage

Tutor Ok how is medications if we uh I mean referring to this case (. ) how can medications affect the muscle power

Zelda because she has been taking diuretics so I’m thinking of the elec( rolyte

Tutor OK) so it is uh just electrolyte disturbance, muscle weakness so it actually uh it just induce systemic causes right what else

Zelda just now I also asked about pain um because um I also thought maybe there can be um pain from the um diuretics may be causing gout and ( 

Tutor oh ) you are thinking about gout ok (. ) so you do you do have quite good lateral thinking but uh: there is no pain right ( you further confirm by examination

Harry ( no pain

Zelda ( no pain)

Tutor so it’s one of the things you need to uh further confirm by examinations (. ) what else, (. ) still on drugs

Kevin can the patient taking beta blocker have increased (^^^) causing tiredness and present as weakness from ( (^^^)

Tutor (beta blocker and tiredness

Harry (^^^) beta blocker )

Kevin tiredness but not weakness

Tutor it’s very common to be very tired (with beta blockers) (0.4)

Harry mm

Zelda I want to ask about the compliance of the drug if she’s overdosing herself but since we have mentioned she lives in an old age home so (.)

Tutor OK: (0.2) we can you need to uh also look at the history here (.)

Zelda mm mm

Tutor a bit slightly more common slightly more common OK (0.5) OK is he on is she on T4 replacement,

MS mm mm
if she is on T4 replacement just a (sidetrack here) she will (^^^) but if she is on T4 replacement and she has full compliance then the overdose of T4 can lead to weakness in elderly (. ) you know in hyperthyroidism in elderly, you know in young what are the symptoms of hyperthyroidism (0.4)

Students ( mm mm

Zelda hyper) thyroid ism,

Tutor yeah any, some of the ladies are more silent and can you tell me what are the symptoms )

Gladys OK ) heat intolerance, the patient may generalise may generally be more uh metabolically active like the patient will be having a heat intolerance, sweaty palms, sweaty hands, and maybe trauma uh tremor, palpitations ( um

Tutor OK)

Gladys they are likely to have diarrhea and uh muscle weakness also ( (^^^) yes and also ) (^^^) and also quite thin right they will be thin (. ) very anxious (. ) so if there is a T4 but {ac} in elderly we have a syndrome called the um apathetic hyperthyroidism that means that they seem to be in a burnt out state now they they have no response tissues so they are very apathetic (. ) not not anxious but on the contrary they are apathetic, uh they are:: exhausted, they don’t want to talk much, look depressed, instead of anxious, sort of and also they will have deconditioning (. ) OK so in hyperthyroidism in the elderly may be present you may present differently (. ) compared with the young (. ) so the patient’s deconditioning will check the file in front of you we would expect we would like to (note) hyper and hypothyroidism (. ) you don’t know right especially alright so in this case we’ll also check the file definitely especially as she has a history of the thyroid surgery (0.2) but then for the drugs we need we don’t know whether this is T4 or not right (. ) what other medications that can lead to (^^^) on the same line

Students {whispering}

Harry steroid

Tutor sure steroid is she taking steroid, (0.2)

Harry no

132
you don’t know because sometimes the over the counter pain medications and even um: so called Chinese medicine may contain steroid right so you need (.) to have a very very detailed to ask what kind of drugs she’s taking besides that prescribed by the doctor (0.3) right, Chinese medicine you don’t know (^^^) OK (0.3) any more drugs, that are important, (0.2) suppose the CPK now you go to the drug CPK is three thousand four thousand in this lady (.) so what are the drugs in this (^^^) previous ( (^^^)

Tutor you need a very very detailed to ask what kind of drugs she’s taking besides that prescribed by the doctor (0.3) right, Chinese medicine you don’t know (^^^) OK (0.3) any more drugs, that are important, (0.2) suppose the CPK now you go to the drug CPK is three thousand four thousand in this lady (.)

Joy statins

Gladys a statin

Tutor sure (.) statin

Zelda mm:

Tutor sure you need a low a statin (0.2) OK, is she on statins

Harry uh:: because ( do you know what drugs she is on naturally, before (^^^)

Tutor ( do you know what drugs she is on naturally, before (^^^)

Harry because I previously (^^^) lost my notes so I can only remember some of them

Tutor does she have a history of hyperlipidaemia

Harry uh yes so I think so

Tutor (^^^)

Harry mm

Tutor (^^^) statins nowadays are quite cheap they have a lot of generic drugs now you don’t know what the whether generic drugs are as good as the uh the uh real I mean the uh the uh trade the the the um original drugs (.) OK (.) some of the generic drugs are made in Europe, some are made in Israel, some are made in Thailand, the quality control is very good or not (.) OK (.) so: we’re still in the (^^^) systemic causes, drugs causes, neurologic causes,

Zelda can it because since you mention the lady is quite overweight as well does she have any like um maybe polyuria, polydypsia symptoms suggestive of DM, umm maybe it can be a new onset

Harry I didn’t ask about this

Tutor yeah this is a good point (0.3) there is DM: patients that generalise it can be DM control is very poor control is very poor (.) especially in the state of
and they may present with weakness, right, very high glucose level and high glucose level can be can link to the hyponatremia too as well we call this pseudohyponatremia (.) high tryglyceride, high glucose can lead to pseudohyponatremia (.) because of the laboratory interference not due to the real sodium ( low

231 Zelda mm )

232 Tutor (0.5) the method is interesting because what you learn we just can’t get enough from one chapter you need to reread the whole textbook again, one I (^^^) here (^^^) you know there together that’s why we need problem based learning (.) usually we just learn one chapter ok that’s a (^^^)opathy (.) to one case right so you need to dig up all the background knowledge to put in one case (0.3) what are other details come on not enough you still have have you watched the series House yeah you have whole lot of details you are the people who advise people on the details (0.3)

233 Gladys metabolic process like (^^^) because the patient have hypertension then maybe (^^^)

234 Tutor ummmm

235 Gladys )(^^^)

236 Tutor )OK in general (^^^) the body process renal fail, liver fail, actually any organ failure can (^^^) but there should be other symptoms on top like generalised oedema, shortness of breath (acidosis ^^^) do you think the vaccine is really related to this weakness {cough}

237 Zelda vaccine?

238 Tutor you mentioned vaccine was given was given one month ago right

239 Harry mmmm

240 Tutor so do you really think that it is related, if it is GBS what sort of symptoms is the patient expect

241 Zelda If it starts from the lower limbs first it would effect the (^^^)

242 Tutor OK,

243 Zelda gradually moving up but then in the history it said that the upper limbs and the lower limbs symptoms start together

244 Tutor Ok it’s rare (0.2) and uh: also it should be a little bit more (slowly) I mean it suddenly occurs in a few days time and then from normal to very weak
person but you mention she’s in (^^^) for a few weeks already

245 Harry yes yes

246 Tutor and uh she needs to walk with a cane for a few months already,

247 Harry yes uh yes

248 Tutor so before she has the vaccine she’s not normal

249 Students mm

250 Tutor right so it’s a bit odd to have a (GBS) on top another neurological problem

so I think if I need to place my bet I don’t bet on the (GBS) so how exactly

how long was she be unable to walk

251 Harry umm about six to seven months

252 Tutor so she has a quite long history right (0.5) any other possible causes so

imagine: some of the important causes

253 Gladys any infections maybe for three weeks some chronic infections causing

some general weakness like in cases of TB the patient in old age home may

have) (^^^)

254 Tutor ) infections could be one of the causes yes it can be (0.4) any other,

255 Zelda mmmmm

256 Tutor actually you have mentioned something already that hints that it may be a

possibility it is something related to the social background of this lady

257 Zelda oh the depression

258 Tutor ye:es

259 Zelda medically unexplained symptoms usually it’s more difficult to

260 Tutor yeah (. ) anyone want to elaborate on this how this is related to her unable to

walk (. ) why you need to think of this in this case

261 Eric maybe attention seeking for family members

262 Tutor OK can you elaborate on the social background and effects on this lady I

think you mentioned some points but not in great detail

263 Harry she was married but her husband passed away about ten years ago

264 Tutor OK,

265 Harry and her son uh work as a labour worker in Macau

266 Tutor so she used to live in Macau or not

267 Harry uhh she live in Hong Kong but uh her son work uh work in Macau
Tutor: her son works in Macau

Harry: yes

Tutor: so when did she go to Macau (to live) (0.3)

Harry: I didn’t ask about that

Tutor: mm mm (.) so

Harry: so because there is no other people look after her in Hong Kong who so she was she was taken to the old age old age home

Tutor: who uh take her took her to the old age home

Harry: I assume to be her son

Tutor: but she I’m not too sure of the sequence she is living in Macau she has been living in Macau for some time (.) when did she come back to Hong Kong

Harry: my understanding is uhh she lives in Hong Kong but her son have to work in Macau so no one care about her in Hong Kong so she needs to be has to live in the old age home

Tutor: so she never went to Macau

Harry: I didn’t ask her whether she went to Macau before

Tutor: OK anyone clerk the case together with him and can give more history

Joy: basically he asked about her history and I perform the physical examination umm so

Tutor: so basically no other (^^^) so I think uh if I’m correct she has been living in Macau for some time (.) because her son work in Macau but about in the last one year she is not able she she has difficulty walking in Macau already (.) and she need to walk with the cane indoors and wheelchair outdoors for nearly one year (.) and then in the last three months she came back to Hong Kong (.) for some reason because no one take care her in Macau and she came back to Hong Kong and then find a old age home and found an old age home (.) for a few weeks already (.) I don’t know exactly the time (.) so some people ask her it’s ten days some people ask her it’s a few weeks (.) so she’s very unreliable in the history (.) especially the date and then in the in the old age home she said that she’s completely wheelchair bound and cannot walk (.) this has been confirmed by the old age home staff (.) by telephone (.) so the sequence is that she start to unable
to walk or at least I mean disability disa uh a decreasing ability to walk for nearly one year and then gradually um like this (.) OK that’s um what I got from the history when the nurses asked about the patient’s relatives as well as the old age homes (.) so it’s not new (^^^) but surprisingly the muscle did not did not uh during examination I think you may see her (^^^) it’s not too severe wasting and there’s no contractures at all during the examination so a bit odd

283 Zelda mm:

284 Tutor and uh:: you mentioned about the suicidal ideas (. ) you mentioned about the suicidal ideas,

285 Joy mm I read from the um psychiatrist (testament)

286 Tutor uh alright (. ) so uh could you relate it to the weakness

287 Joy uh I think it can both be associated with the depression and also it can also because of the weakness because of the weakness she suddenly become chair bound (. ) and so may have and also um her son is in Macau, and so altogether may contribute to the depressive mood and uh the suicidal

288 Tutor mm mm so sometimes psychological evidence pyschosomatisms are (. ) need to be looked at (. )OK occasionally uh we see some cases but it’s hysteria but they may be sign of stroke )

289 Zelda )hysteria,

290 Tutor ) very: I don’t know they do that but they mimic a stroke (. )

291 Students {smiling}

292 Tutor the only thing you can know is that when you are not (. ) you’re you’re hiding and thinking she’s normal walking around and then suddenly when the doctor comes (^^^) she cannot move her right arm again but when (^^^) they sometimes unconsciously can move their arm and do something so sometimes maybe it may be like this they can be (^^^) (. ) but this need to be excluded by exclude by (^^^) so the work up need to be done (. ) so don’t think (^^^) do all the things especially the uh (^^^) very important (^^^) (. ) OK so any other things you want to confirm the history, (0.6)

293 Tutor smoking alcohol

294 Harry she said she was a non-smoker non-drinker

295 Tutor mm mm (0.2) um: how about the education level and the:)
Harry: I didn’t ask about that.

Tutor: income.

Harry: I didn’t ask about that. I think she is retired.

Tutor: I know but uh you even after retirement you can still have income.

Harry: yes yes.

Zelda: and what was her previous job?

Harry: sorry.

Zelda: what did she work as before she retired?

Tutor: he mentioned about the factory work.

Zelda: oh OK.

Tutor: factory worker.

Harry: yeh yes maybe.

Tutor: so the history is not too.

Harry: because the patient was not very conscious uh not very uh.

Zelda: sorry.

Tutor: it depends on your you need to have charm.

Students: {laughter}

Tutor: your charm and your skill {more laughter} ninety–nine per cent in the ward are elderly ninety–nine per cent are not very cooperative {more laughter} so it depends on the technique OK, give them some {laughter} I give you a {laughter} so you can talk better {more laughter} so you need to have charm right to get a history OK, well anyway so uh: let’s go to phys examination.

Joy: I did the physical examination, um but however in the middle of the examination um she complained of headache and tiredness and refused to be uh so I will just report I uh I copied from some of them I copied from the notes.

Tutor: mm mm mm OK. thank you for your honesty.

Zelda: {smiling}

Joy: Madam Wu uh when I examined her uh she was conscious and alert and the GCS was fifteen over fifteen, and the blood pressure was a hundred and seventy three over seventy–nine millimeter mercury, and the pulse was
eighty-five um beats per minute regularly regular (.) she was afebrile at the
time um and also there was no jaundice pallor (^^^) and also there was no
lymph nodes palpable um however she has ankle oedema up to the shin (.)
umm uh for the neurological examination I was just able to complete um
the upper limb um (.) uh actually uh um she she has uh um normal tone and
also there was no muscle wasting, no fasciculations and the limb power
was better over the proximal than the distal area (.) however um when I
examined the uh proximal umpower, uh she complained of shoulder pain
on both sides but more severe on the right shoulder (.) and the proximal
power was uh about uh four minus and the distal power was about three
plus (.) um and the (^^^) are normal and symmetrical on both sides (.) and
um I also examined the sensation and um actually there was generalised
decrease of pinprick um sensation, sparing the face

319 Tutor mm mm
320 Joy um: um and then uh I copied the rest from the notes because uh she
refused)
321 Tutor mm mm mm mm)
322 Joy ) and also the distal uh the lower limb power was above three plus and the
plantar was downgoing the reflexes also normal (.) there was no (^^^)
trauma and the cranial nerves was grossly intact um she was not in any
respiratory distress and the SAO 2 was normal, the chest was clear, and the
heart sound was uh normal without any added sounds or murmur (0.2) and
the um yeh basically and uh I also copied the MMSE was um fourteen over
thirty
323 Martin {whispering} fourteen over)
324 Joy ) yes
325 Zelda fourteen
326 Tutor so in a in a nutshell the notes doesn’t suggest it’s cervical myopathy
327 Joy um the notes suggest there is no signs of any cervical myopathy
Tutorial 5

Medicine Specialty PBL Session

Students: Eddie, Martin, Kevin, Harry, Zelda, Joy, Chris, Vicky, and two Year 5 students

Note: This tutorial followed from the examination of a patient at the bedside. The researcher observed the examination following the tutor’s request for consent to the patient. The bedside session focused on diagnostic tests to establish the site of a brain lesion as in a stroke. The tutor mentioned that he had taught the students these tests some days previously.

What follows in the transcription took place in a tutorial room outside the ward. Eddie was both scribe and presenter. The tutor sat directly opposite Eddie at the end of the table and other participants on the other sides of the table.

1 Tutor OK go on
2 Eddie In layman terms)
3 Tutor Try to write a bit smaller because you know the problem with it is that we have so much difficulty in rubbing it off
4 Eddie Uh …when we clerk cases uhhh dizziness is a very common complaint uh that we encounter and under dizziness uh we could interpret it uh under three categories uh one is uh syncope (writing on board) and the second one could be vertigo …and the last one it could be uh disequilibrium uh which is um mo motion sickness …and under each category uh there . is a list of differential diagnosis that we need to consider uh say in vertigo as in my case, vertigo we could differentiate under central and peripheral causes … uh for central causes uh we consider brain stem and cerebellum problem it could be uh a space occupying lesion like uh hematoma uh tumour it could also be a stroke affecting the cerebellum uh these are classified under the central causes while uh for another sub-category the peripheral causes which affect the inner ear uh inner ear problem uh it could include uh vestibulitis uh lab labyrinthitis or even uh some problem with the vestibular cochlear nerve um yeh that’s for the vertigo … and
then for the syncope

5 Tutor before you go on any thoughts or comments on what he’s said so far do you want to add to that one let’s just consider vertigo first before we go to syncope anybody wants to add, correct, amend …. you look as if you want to say something

6 Kevin He’s presenting quite good (laughter)

7 Tutor Right OK anybody no anybody thought want to add on to this discussion …. 

8 Kevin Maybe the characteristic of the vertigo in central …. 

9 Tutor OK … well perhaps you’d like to just add to that

10 Kevin Well in the case of uh central vertigo usually it is more severe constant and not related to position and do not have any ear symptoms like tinnitus and hearing loss and sometimes associated with cerebellar sign … while that for peripheral vertigo it’s opp it’s just opposite but the but this not always true you may have hearing tinnitus or hearing loss in the case of central not a hundred per cent…. 

11 Tutor Any other thoughts? ….. any other thoughts?

12 Harry I think the (in deciding the) exact lesions)

13 Tutor ) speak a bit louder

14 Harry Oh I think (…. ) exact lesions in the brain um drugs or alcohol can cause vertigo

15 Tutor So you think that there could be other drugs as well acting where peripherally centrally?

16 Harry Peripherally

17 Tutor You think so? Drugs adding

18 Harry Um on the on the

19 Tutor ) such as what

20 Harry aminoglycosides

21 Tutor ) so aminoglycosides OK aminoglycosides where would that act mainly

22 Harry on the inner ear

23 Tutor on the inner ear OK autotoxicity OK alright drugs any other drugs since you are on the (.5) topic of drugs?

24 Harry alcohol
alcohol … yes,

that’s all I can think of

that’s all / any other any other thoughts on drugs? Can you think of any
other side effects of drugs that could give you … cerebellar signs or
cerebellar symptoms

anti-convulsants

sorry?

anticonvulsants

anti

anticonvulsants

anti

anticonvulsants

such as?

phenothrin

what

phenothrin

Yeh I suppose it could do yeh … and where would these act (.5) where
can they act …. would they act on the nerves …. like your
aminoglycosides just now you were mentioning aminoglycosides right
they act on the nerves on at least on the inner ear …. but where would
things like alcohol and anti-convulsants if they were going to act

) on the central nervous system on the CNS

= on the CNS system

specifically more

the brain stem

) brain stem could be on the brain stem but also on the
cerebellum

= cerebellum yeh could be acting on the cerebellum as well so it could be
central isn’t it so drugs need not necessarily just only act peripherally they
can act centrally as well …. are there any thoughts I mean how are you
going to distinguish between central and peripheral causes but also on the

central causes by the history of the

= louder, talk to your
= you mean by the history of the physical examination

yeh any features

as mentioned by Kevin, um peripheral peripheral causes of vertigo are often associated with hearing problem, tinnitus while for central causes uh like cerebellar uh space-occupying lesions the patient uh may complain of nausea, vomiting, headache

would you get that in peripheral as well, nausea vomiting would you get those

uh depending but the pattern

= you seem to be shaking your head would they get it

I think so

eh you can so your colleague seems to disagree with you

yes but the pattern is different for peripheral causes uh the nausea vomiting may be uh occurring throughout the day but uh while for uh central causes uh the nausea and vomiting may be more severe uh in the morning after (…)

= is that right is that right is he making it up or do you think uh (.) where’s the evidence for that I mean where do you do you have any any way to defend your uh thing is that is that you quoting a new publication or somewhere that you’ve read that this was the case

uh because I thought uh for a CNS tumour uh I mean I mean brain tumour uh usually

) let’s not forget let’s forget about brain tumour first I mean let’s concentrate on the question of: features between central and peripheral what are the differences so you’ve mentioned that differences could be the fact that central could have other signs: that’s correct is that is that is that correct do you think

) depends on the site

) depends on the site

let’s say if it’s brain stem then you might get the signs

Right you might get other brain stem signs

and also but sometimes it can be mixed features let’s say (….) you might get mixed central and peripheral
so how can you tell then between central and peripheral I mean it’s not just only based on one sign is it or one feature but what what other features what’s what’s a key feature that we might help you distinguish between peripheral and central from your reading

I guess

I think the central is more persistent but

) more

more persistent

that’s right more persistent would you like to expand on that

so uh so um but I I don’t quite get that actually I I didn’t read up about that and it said that the peripheral causes (…) go away quite quickly but I don’t actually get what it means

you might have to talk a bit louder because they’re not getting it on the microphone but uh but it’s more persistent, lasts longer is that what you’re trying to say

yes

you’re nodding

I read something in Davidson’s about that

 louder yeh

(…) yeh you read in Davidson’s which is good yeh (laughter)

actually um for the peripheral cause of vertigo although it’s positional after sometimes um a short period the um the vertigo and also the associated nystagmus will go away (.) but for the central cause even after a long time um that you check it it (persists)

(mm mm) (0.2) do you agree? would you like to expand on that?

ahh and also (.) then for the po peripheral it was usually posturally related

post what

posturally related because (^^^)

postural (related)

(postural) related but for the central although it can also be postural related but usually would be having in any head movement but in the peripheral usually only one specific postural related ^^^
right: so:: can you think of a (. ) postural related (.1) type (. ) dizziness (. ) that (. ) would give you that which is quite dependent on the posture of the head

Chris um benign paroxysmal positional vertigo

Tutor alright benign positional vertigo benign positional vertigo right (. ) would you like to expand a bit about that?

Chris (umm)

Tutor (perhaps) you can just write that down first benign positional benign positional vertigo (0.1) BPV (0.2) we'll keep that in mind alright but are there other peripheral causes which might not be postural related

Chris umm like um vest uh uh labyritis

Tutor OK labyrinthitis: and?

Chris vestibular neuritis,

Tutor vestibular neuritis (. ) is that postural related?

Zelda that was not postural (related)

Tutor (right) so can you use that (. ) to distinguish between peripheral and central then? (posture)

Zelda (um) not so

Tutor (not) so yeh: cause you can’t use it because (. ) the problem with it is that neuritis is not postural related (0.1) OK? which then comes back to?:

Students *persistence* ^^^^ persistency right *OK* (0.2) would you like to talk a bit more about that? (. ) the difference between central and peripheral (. ) or anybody would like to talk about it? (0.2) would you like to talk about that?

Martin mm: I think like (^^^^)

Tutor (louder)

Martin I would check in the examination for nystagmus uh

Tutor sorry?

Martin usually for the central cause they will have uh vertical nystagmus (. ) as well as ^^^^

Tutor mm mm

Martin but whereas the for the (. ) peripheral cause they usually do not present with vertical nystagmus
Tutor: I see OK

Martin: but these tend not to be very ss absolute

Tutor: *OK* so: anything else that you’d like to add to so I think that this will be something that those of you who have not actually read up that well on uh on the differences between central and peripheral I think this is something that you should know about the differences between central and peripheral wha wha what’s the significance of that?

Harry: the managements (^^^^)

Tutor: (louder)

Harry: their managements are different

Tutor: mm mm and how do you mean by that?

Harry: mm:: if if central lesion is suspected then the patient should receive a CT scan to localise the lesion otherwise if it is peripheral I think the patient can be just given some antihistamines to try if the vertigo can subside or not

Martin: I think it should be more related to the symptom (severity)

Tutor: (louder)

Martin: symptom severity if it’s severe we’ll we will proceed with the CT scan even if ^^^ peripheral

Tutor: I see so if somebody comes in with not very severe symptoms are you saying that: the: thing is not central

Martin: no no I’m just (saying)

Zelda: I should say it’s a collection of the symptoms because I I just remembered another (cause)

Tutor: (OK)

Zelda: for example the uh vertebrobasilar ischemia you can ask the patient just to extend the neck and see if it aggravates at all because it’s because I it’s because the nerve runs such a whole course and maybe you can just test it step by step to delineate the exact site and also by the onset or you may observe the patient for a while so that if it goes away then you can decide mm it’s half the severity but not just the severity of the symptoms I mean it’s how sinister the causes

Tutor: so she disagrees with you how can you defend yourself?
Martin: mm::: of course we need to get confirmation of signs.

Tutor: you’ve got to talk to her I’m not I’m not arguing with you you’re arguing with her (laughter)

Martin: I think that what she says is correct we have to consider all the signs together in order to get a whole picture of the situation but I think that what I’m saying is that even for the peripheral cause even if the if you’re suspecting the constellation points to the peripheral cause but however if the symptoms itself is so very severe to affect the patient’s quality of life worse than it was before or something like that then uh we should still suspect some organic cause that may be causing the problem (0.2)

Zelda: are you meaning one man two (disease)

Tutor: (you) have to talk a bit louder because

Zelda: oh sorry

Tutor: (you know to be)

Zelda: (so so) are you meaning that this is one man having two disease because the peripheral causes are so common then?

Martin: (0.2) mm mm (.)

Tutor: do you agree?

Zelda: umm I think it’s quite rare (but)

Tutor: (mm mm)

Zelda: it’s always possible (0.5)

Tutor: what are you going to (. ) withdraw your your thoughts or are you going to defend your turf?

Martin: mm:::

Tutor: who agrees? who agrees with (. ) with the colleague here? (0.2) about that severity does not play uh play a: that significant a role in you know in determining whether it is peripheral or central? (0.1) whereas on this corner you’ve say no ((emphasises by beating fist on table)) severity is important what do you think

Vicky: I think the severity is important but I think you also have to consider that the vertigo is very subjective

Tutor: mm mm? (0.3) so would you tend to agree with him (0.1) that severity is important?
I think it’s important but I think you still have to look at the other signs as well.

What would be a key feature if one were to determine whether it’s peripheral or central there were a few things why don’t we just write those things that we are talking about those issues about whether peripheral or central what are the features distinguishing features why don’t we do it on the right hand side.

Tutor

Distinguishing features of central and peripheral ((Eddie writes on whiteboard))

Zelda

I thought the peripheral one might some of them might be self-limiting.

Tutor

Self-limiting

Zelda

(or they might be coming across) like that like kind of relapses

Tutor

(MM)

Zelda

but for the central one it’s much more persistent

Tutor

Would you agree with that?

Student

Not sure

Tutor

What do you think?

Chris

Umm: (0.4) I was: I’m not very um quite agree with self-limiting for peripheral because sometimes the um like for example (the)

Tutor

Have you read up

Chris

The BBPV (^^^)

Tutor

(YOU HAVE READ UP)

Chris

Those will be episodic and be quite long and those will not be uh very self-limiting but for labyrinthitis um the patient are the lateral cause are we expect to (have)

Tutor

So what does B (. ) BBPV means

Chris

Uh it was uh (. ) uh I read some there was a problem in the autolymph

Tutor

Yes

Chris

And so that the patients um because the autolymph is inside the (uh)

Tutor

(Is it) self-limiting

Chris

Um: I think it was being episodic but postural related but

Tutor

Do they get it twenty four hours seven days a week
170 Chris uh: not (so: ^^^)
171 Tutor (is it) self-limiting
172 Chris um yes (it is)
173 Tutor (it’s) self-limiting yeh
174 Chris (self-limiting)
175 Tutor so does (BPPV)
176 Chris (^^^) adapted ^^^ (vertigo)
177 Tutor (is that something that you’ve read up)
178 Chris (^^^)
179 Tutor do you agree with her
180 Martin yeh based on the main ((paroxysmal)) it usually comes and goes ^^^ benign (^^^)((making up and down hand movement))
181 Tutor (everybody) agrees with her (. so far
182 Martin mm
183 Eddie mm ((Students around table nodding)
184 Tutor all of you agree (. is that right (0.2) you don’t look very convinced .)
185 Student (^^^)
186 Student (^^^)
187 Tutor do you are you convinced with her think that that she thinks that it is not self-limiting (. she’s she’s are you are you disagreeing with her that she says that she’s that it’s self-limiting but you think you don’t think that it is self-limiting you think it is
188 Chris I think it was paroxysmal (to) ((making up and down hand movement))
189 Tutor (so) if it’s paroxysmal what does paroxysmal mean
190 Chris paroxysmal is like (. episodic)
191 Tutor )episodic (yeh)
192 Chris (with) relapse over (time) ((making up and down hand movement))
193 Tutor (mm mm) mm mm (. but does it relapse all the time
194 Chris when the patient maintain the specific posture
195 Tutor is that true (0.1)
196 Chris uh (. not maintain but have that uh movement (0.2)
197 Tutor mm I see yeh)
198 Chris )so that the patient will avoid to have (. that movement on that side (0.1)
Tutor: yeh (.) so you don’t think that self-limiting uh issue is a major issue (.)
who thinks jus just by the raise of hands who thinks that she (0.1) that this
is not self-limiting (0.1) is that is that is that your argument

Chris: umm (.) yes

Tutor: right (0.3) what do you think (0.2)

Joy: uh: BBPV is not lethal ((laughter))

Tutor: is what

Joy: it’s not not (not)

Tutor: (it’s not) what

Joy: it’s not lethal fatal ^^^

Tutor: not ohh initially I thought it was not legal but ((laughter)) but not lethal

Zelda: but because if for my thought

Tutor: yes

Zelda: I think about it from the ^^^manoevres

Tutor: yeh

Zelda: it’s just that you want to test if it's really a BBPV

Tutor: yes

Zelda: and after you have put the patient’s head down and after five or ten
seconds you see the (nystagmus)

Tutor: (yes)

Zelda: but after thirty seconds it subsides itself

Tutor: right

Zelda: and then if you expect something central then you expect that it just keeps
going on once you have (tested)

Tutor: (right)

Zelda: the nystagmus

Tutor: OK

Zelda: and that’s why that’s why I call (it)

Tutor: (does) that does that sound logical to you

Chris: yeh

Tutor: do you want to change your mind

Chris: oh oh OK

Tutor: sorry
Chris: so self-limiting can be used can be used for paroxysmal type

Tutor: yeh: what do you think

Chris: I’m not really sure about that

Tutor: yeh: (. ) time for you to read up a bit more about this isn’t it whether it’s self-limiting yeh it is important because let’s move on because we have got to move on to the next stage (. ) talking about this patient (. ) important that the central part just now you were talking about the issues about uh other neurological deficits in central I think that’s very important (. ) it’s important to know that there are other features apart from: um just the deafness or the tinnitus and of course the self-limiting issue is a very very important distinguishing feature (. ) I’m a bit surprised that those of you that think you have read it somehow (. ) misinterpreted the uh your reading (. ) it is self-limiting and that is one of the major distinguishing features between central and peripheral (. ) severity is not important so much because there could be some people who comes in with extremely severe vertigo (. ) OK (. ) and they have a peripheral cause (. )

Tutor: so: it is not a not a a distinguishing feature on its own severity is not a distinguishing feature on its own but rather a uh issue of uh so uh it’s obvious that some of you that think you have read it obviously have not done your homework and I would strongly suggest that you actually go back to do that (. ) OK (. ) uh: I’m a bit surprised that uh: some of you had a completely wrong concept of the distinguishing features of between central and peripheral (. ) let’s come back onto this chap who had vertigo ((points at whiteboard)) who was the one who clerked him (. ) yeh ((Eddie raises hand) you you clerked him this gentleman bed two

Kevin: yes

Tutor: actually yeh bed two let’s go back because the chap had tinnitus I mean he didn’t have tinnitus but he had vertigo he had dizziness OK

Kevin: the: vertigo is uh: constant and sustained (. ) but subside after I think (. ) uh
subside after one to two days of the symptom of the

240 Tutor so what are you trying to say with this
241 Kevin so it is self-limiting but it is central
242 Tutor so I see (you think it’s self-limiting)
243 Zelda (no no I think) because a vascular cause is just like when you take history then somehow collaterals develop or they just improve a bit you get you get some perfusion it’s due to the vascular cause rather than the central peripheral that

244 Tutor does that make sense?
245 Martin yeh it can be seen transient ischemic
246 Tutor (no) louder does it
247 Martin (it can be)
248 Tutor make sense
249 Martin it can be some transient ischemic
250 Tutor do you think this was a transient ischemic attack?
251 Martin uh:: (0.2) how long how long has the patient been admitted?
252 Kevin the patient was admitted uh three days (ago)
253 Martin (oh) then uh mm not likely
254 Tutor (so it’s not) a transient ischemic attack (what do you think it was then? (0.2)
255 Martin (mmm)
256 Tutor (you think) it was a peripheral cause
257 Martin I still think it’s a central cause
258 Tutor but why do you agree with him? (pointing to Joy)
259 Martin yeh
260 Tutor you are agreeing with him, that he’s saying that it’s a peripheral cause
261 Martin he’s saying a peripheral cause?
262 Tutor because he’s saying that it’s self-limiting hence it must be peripheral
263 Kevin no no no (laughter) I’m saying it is central but I don’t know why self-limiting (because it’s)
264 Tutor (but) what about your colleague she just mentioned to you do you accept her argument? (0.2)
Kevin: yes yes I accept (. ) there may be some (. ) perfusion back to the brain stem area that that (. ) that supply the inner ear so maybe (0.3)

Martin: collateral (^^^)

Tutor: do you think he’s talking nonsense (laughter) or do you think he’s uh (0.3)

Kevin: I mean there is some perfusion reperfusion back to the (. ) lesion in the brain or brain stem so there is resolution of the vertigo (. ) do you mean that? (addressing Zelda) (0.2)

Zelda: it’s it’s just like when you have the hemiplegia I don’t know to me all vascular causes the onset is acute, and then when you take the history you usually after several after a longer time they improve (a bit)

Tutor: (right)

Zelda: that’s just a cause that will make me think of a vascular cause but doesn’t stop um it’s not a feature that helps me to analyze if it’s a central or peripheral cause of (^^^ of vertigo )

Tutor: (correct) isn’t it I mean you have to make I mean it’s a well-known feature of vascular cause isn’t it (. ) that you have got sudden onset, and you’ve got a recovery phase you’ve got a recovery phase of course you might not necessarily recover all you know of ^^^ so your* argument the fact that it was self-limiting makes it (. ) unusual for a vascular cause do you think that makes sense? (0.2)

Kevin: after hearing (^^^)

Tutor: I see so do you want to withdraw that whatever you said just now about the fact that you thought it was unusual the fact that it was unself-limiting (0.2) yeh yeh I thought that it was um your argument was uh do you agree do you agree that her argument (. ) sounds (. ) reasonable? (Students nodding)

Martin: mm

Tutor: yeh it’s a central cause affecting the cerebellum it’s a vascular issue (. ) and: he’s just recovering from a stroke so that he’s recovering but you’ve clerked him so where do you think the lesion is?

Kevin: uh pardon me

Tutor: where do you think the lesion is?

Kevin: I think the lesion is uh in the posterior circulation
Tutor alright, (0.2)
Kevin (^^^)
Tutor (and let) it stand on (that)
Kevin (^^^)
Tutor (could) you be a bit more more specific?
Kevin uh:: because uh: vertigo, nystagmus and ataxia uh: (0.5) they are the symptoms of a brain stem lesion in case of a lesion higher up in the subcortical region or cortex there will not be uh vertigo (0.3) but nystagmus
Tutor what do you* think (0.2) do you agree with him? (0.3)
Kevin less likely to have that those uh: features
Chris you mean in the anterior circulation
Kevin uh because anterior circ there there is a syndrome called anterior circulation syndrome and (.). in this syndromes there are no: ataxia vertigo or nystagmus or any uh cranial nerve cause so when there is presence of cranial nerve cause vertigo nystagmus cerebellar sign then it promise more likely to the posterior circulation that is the uh the brain stem lesion the brain stem yes (.). that is what I learn
Tutor what do you think? you clerked him as well didn’t you?
Harry I also (have)
Tutor (louder)
Harry I also have similar thoughts with him
Tutor mm mm and so specifically where do you think the lesion could be?
Harry it should be in the (.). brain stem in the region of the: pons
Tutor of the pons (.). why would you think that?
Harry because it is the: region where the brain stem connect to the cerebellum
Tutor just only at the pons? (0.2)
Harry and also the medulla
Tutor mm mm what do you* think? what does anybody think?
Joy mm actually I think that it is affecting the cerebellar (^^^)
Tutor (mm mm)
Joy: so uh umm I want to mention about ataxia and nystagmus and also if the patient comes in with clumsiness they are also um some signs of some cerebellar signs so obviously I think the cerebellum is affected, and if patients look present with um uh (0.1) loss of gag reflex obviously this is because the nucleus um and the brain stem is affected

Tutor: OK; what do you* think? (.) do you agree?

Chris: agree

Tutor: yeh mm mm

Zelda: I can’t remember exactly the: I’m sorry I can’t remember exactly the names of the vasculature but I would expect it to be somewhere before branching off to the cer separation to the cerebellum or to the um (.)

Tutor: mm mm

Zelda: brain stem wall

Tutor: mm mm yeh

Zelda: because of the mixed picture picture of ^^^

Tutor: if somebody had* slurring and slurring was one of the: problems and as you’ve heard from the patient he had some choking didn’t he he had choking he had slurring of speech (.). along with this clumsiness (.). if you’re going to localise it down to the brain I mean is everybody happy that it’s going to be somewhere in the brain stem yeh so that seems to be generally agreed is that right? (.). so if it was within the brain stem what (.). area of the brain stem (.). would (.). you think would most fit his symptoms?

Martin: mm: ^^^

Tutor: you must remember that he’s got a whole set of symptoms do you need to revisit those symptoms?

Martin: mm

Tutor: remember he’s got (.).

Martin: dysarthria nystagmus)

Tutor: ) vertigo, he’s got) 33.36/ 30.42

Martin: ) vertigo, hystagmus ^^^)

Tutor: ) nystagmus, vertigo, dysarthria, slo he’s got difficulty swallowing (.)

Martin: ) ^^^
so: out of these symptoms (0.2) where do you think within the brain stem where do you think (.) it would most likely be? (Students discussing among themselves)

Kevin medulla

Tutor sorry?

Kevin medulla

Tutor would that fit? what does everybody think?

Martin medulla

Tutor what do you think? (to visiting student)

Martin is there facial numbness?

Kevin yes

Students facial numbness

VS ^^^

Tutor I see your English is not as uh (.) what do you think (Students discussing among themselves)

Chris I think it’s quite ^^^ )

Tutor ) so let’s just draw the brain stem first wha wha what what what structures are in the brain stem?

Martin midbrain pons medulla)

Tutor ) you’ve got the midbrain (0.2) what else is there apart from midbrain what you’ve got

Martin ^^^ pons ) (Students reciting medulla, pons)

Tutor ) you’ve got pons and then medulla right so that’s your brain stem

Students (laughter at Eddie’s drawing of brainstem on whiteboard))

Tutor ) oh very nice brain stem it looks more like uh but it’s OK alright you’ve still got your brain stem there (.) so let’s just deal with it (.) up in the midbrain (.) what sort of things have you got up there in the midbrain?

Martin the third nerve is there)

Tutor ) third nerve (.) that’s right you’ve got third nerve (.) that controls what?

Martin uh: eye movement)

Tutor ) eye movement you’ve got third nerve eye movement you’ve got eye movement haven’t you (.) what else is there apart from third?
well that’s the third isn’t it I think Edinger-Westphal is third isn’t it? what else? (0.2)

Kevin ^^^

Tutor sorry

Zelda fourth nerve )

Student ) fourth nerve)

Tutor third fourth nerve as well isn’t it yeh third and fourth (.) let’s just deal with the motor first right let’s just deal with the motor first (.) motor aspect third)

Martin )mm

Tutor )and fourth (.) and then in the pons:? in fact it’s one continuous area isn’t it so third fourth and what else?

Zelda trigeminal trigeminal

Tutor sorry?

Students trigeminal

Tutor trigeminal yeh OK:: but it is a bit longer than that but yes (.) so that would be in the pons wouldn’t it so: yep (.) fifth what about sixth seventh where are the sixth and seventh

Martin pons)

Students )^^^

Tutor )similarly isn’t it

Zelda )a little bit lower down

Tutor yes that’s right slightly lower down so perhaps fourth sorry fifth sixth and seventh are over in the pons (.) right?

Martin mm

Tutor (.) and then what happens in medulla? (.)

Martin the vagus nerve

Kevin lower cranial

Martin the lower cranial nerve)

Tutor )sorry

Kevin the lower cranial nerve nucleus nuclei)

Martin the vagus eighth nerve

Tutor so like what?
Kevin: like the fifth nerve
Students: eighth
Tutor: no no no I’m talking about motor now
Zelda: glossopharyngeal
Martin: glossopharyngeal hypoglossal
Tutor: right so ninth tenth eleventh twelfth isn’t it ninth tenth eleventh twelfth all
down in the gullet area isn’t that right?
Martin: hah
Tutor: OK? alright? now let’s just revisit that diagram it’s a terrible diagram but
nnn: it’s about right I mean it’s sort of sort of give you some idea (.) but (.)
if we were going to look at this area here where do you think (.) with the
symptoms that this patient has (0.2) oh don’t look at me look at the
picture (.) what do you think?
Zelda: somewhere in the medulla region)
Students: )medulla
Tutor: yeh isn’t it it’s a medullary lesion isn’t it? it’s a medullary lesion (.)
because you’ve got dysarthria you’ve got (.) slurring you’ve got gag reflex
yes you’ve got ^^^ symptoms there (.) with eye movement what do you
have to worry about if it with eye movements (.) you’ve got disconjugate
eye movements it’s gonna be in the?
Zelda: cerebellum midbrain)
Students: midbrain
Tutor: )midbrain (.) you can see right third fourth and sixth near the pons and the
brain (.) do you understand OK? (0.2) but this one is is mainly along the
medulla (.) isn’t that right? (.) OK (0.2) so you were (.) early on you
(pointing at Kevin) were you said or at least he disagrees with you that he
thought he’d had a visual hallucination but uh and you thought that he had
a bit of partial ptosis why why were you so concerned that he had a bit of
partial ptosis (.) had you read the notes or what?
Harry: mm: I can’t remember if there is any (.) documentation of the: partial
ptosis but I think it was there (0.2)
Tutor: did you see it? did you see a bit of partial ptosis?
Harry: from the patient I saw it
Tutor: you saw it OK alright, but did it fit in with any syndrome?

Harry: no

Tutor: that you know

Martin: of partial ptosis

Harry: because there are no other eye signs so I don’t think

Tutor: you didn’t think that it was significant. do you think he has partial ptosis now?

Harry: no no

Tutor: at least not this morning

Harry: no

Tutor: so it could be a visual hallucination was it alright OK

Harry: yes {Martin muttering}

Tutor: but if it did if it did let’s let’s let’s for example take it if he did and he had unequal pupils what would you like to do wha what’s your next step that you’d like to do to confirm whatever that you wanted to do

Kevin: ^^^ reactive

Tutor: what would you like to do

Kevin: uh:: I would like to ^^^ signs

Tutor: such as?

Kevin: ^^^ anhydrosis

Tutor: right so did you check for anhydrosis?

Harry: uh no because)

Tutor: )why?

Harry: both pupils are equal in size

Tutor: so you didn’t bother

Harry: yeh

Tutor: yeh (.) is it a good move (.) do you think?

Harry: ^^^ backward I should check it

Tutor: yeh right OK so whenever you remember what I said about the principles of clinical medicine if you are gonna try to get some signs you really want to get the whole spectrum as much as possible isn’t it (.) of course the diagnosis might not necessarily give you the whole set of clinical signs isn’t that right? remember what I said to you?
OK remember what I said about the issue about showing you what what number is this what number is this what number is this and then you get a certain diagnosis (.) you might not necessarily get all those additional signs and symptoms (0.2) but you should look for them

Students mm yes

yeh? OK (.) so let’s assume that this guy did have partial ptosis

and let’s assume that he did have eh unequal pupils (.) what are you going to do?

umm: check whether the pupils are reactive to light

mm OK but what else

anhydrosis)

check for anhydrosis OK (.) and with anybody with Horner’s what do you need to do? (0.2) time is moving on so we need to move on

chest and (impacts) absolutely (.) additional reading up Horner’s syndrome (.) learning objectives Horner’s syndrome (0.3) OK? alright? (.)

so this gentleman had: do you know the diagnosis? he’s got some kind of lesion in the medulla, yes? it looks as if it’s pro:bably on the right side

yes? and: what (. ) sort of structures lie within the medulla, wha what do you think? yeh he’s got (0.2) he’s got a right-sided medullary lesion in the brain stem isn’t it, sudden onset (0.3), OK (.) from posterior circulation infarct now I don’t really need you to tell me whether it’s medial lateral or whatever but this gentleman had a lateral medullary syndrome (. ) whether he had right Horner’s we don’t know I mean from what you have seen uh I he presumably had it (.) do you know what other structures lie in that area (.) in the lateral medulla?

well essentially it have the central (thalamic) tract
Tutor sorry

Martin (spinal ^^^)

Tutor to where

Martin thalamus

Tutor to the thalamus but which part of the body does it enervate?

Martin the contra)

Harry )contralateral

Martin )contralateral side

Tutor mm mm

Harry ^^^

Tutor did he have any contralateral sensory deficits?

Harry no)

Tutor )no he can’t he didn’t (.) mm mm (.) alright time is moving on I suspect I suggest that you and read up a bit more about lateral medullary syndrome (.) and compare it to the: structures in the medulla (0.4) I want you actually to take a neuroanatomy book, look at the medulla, and the midbrain and the pons, and then just see what structures are affected there (.) that will be helpful (.} scribe writing on whiteboard and students writing instructions down) and then correlate it to whatever signs that you’ve got (.) OK? now I would also strongly suggest that you go back to see this patient, go through the signs again after you have done your reading (0.2) to then go back on (that) OK? I think we can make a stop here alright unfortunately bed three is not very good because bed three in fact is not Guillain Barre I’m not convinced

Martin )mm

Tutor it’s Guillain Barre umm:)

Zelda )I was very lost when I was trying to think about a more likely picture for that patient )

Tutor )yeh right

Zelda )because another variant that he doesn’t the weakness is not that (common) for that he doesn’t fit into ^^^)
) yeh so whenever you have that whenever you don’t have a thing do you understand because one of the things is that you then grasp onto something: (.) which you are absolutely paranoid about and absolutely convinced that it’s that and then it turns out that you ignore everything else (.) you need to look at the whole complete picture so just now you sort of said you know oh because you know it seems to be self-limiting hence it cannot be central, that is not the sort of thinking that you should be doing (.) you understand? because you need to look at what is the picture and obviously this is the most dangerous you know (0.2) half-baked knowledge is about the most dangerous issue you can have because if you think you know everything unfortunately it’s not, do you understand? so that’s why I encourage you whenever you have seen a case like this, don’t just base it on your own notes taken at the teaching session, go back to (0.2) your text and read it in its entirety (0.2) never just take bits of information and then imagine the rest (.) that’s the easiest way of confusion d’you understand, the easiest way to confuse yourself (.) you have got bits of information, gives you some idea of what the condition is, you go back to your text, read up a bit more, put it into context (.) put your knowledge into context, do you understand? and then go back to that patient, and that’s the way you should do it (0.2) it was obvious during the discussion that some of you are: not (.) quite (.) interpreting the knowledge interpreting your your your your your text and seeing it in a in a in a logical manner and we’ll come back to this do you need to go {addressing observer}
Tutorial 6

Medicine Specialty PBL Session

Students: Eddie, Martin, Kevin, Harry, Zelda, Joy, Chris, Vicky, Anne, 2 Visiting Students (VS)

Note: Throughout the tutorial the tutor acts as scribe and writes and draws on the whiteboard.

1 Tutor (^^^) I’m trying to remember all your names but uh: speak up if you have any questions alright (0.5) now (. ) we have a bit to cover today (. ) if there’s time uh uh I’ll show you uh a few cases which I’ve prepared for the class (. ) on this topic (0.2) now let’s uh: go over today (0.2) we have seen a case of multiple myeloma on Tuesday and you’ve taken the history, and I suppose you have done a physical examination, so we’re going to talk about this in a different in the context of how to make a diagnosis, OK remember the: the pathway I mentioned last time? making a diagnosis, taking the information, doing the investigations to get more information to confirm the diagnosis and then to treat the patient (. ) OK now let’s start with this: at the beginning coming on to the history, signs and symptoms (. ) wha what is the history? just remind us (. ) briefly

2 Harry mm the history of this uh the patient have uh bone pain involving the lower limbs and the ribs and hips and the patient also has history of increased bleeding tendency (. ) then the patient )

3 Tutor ) OK just one minute so she had bruises right {Tutor writing notes on board}

4 Harry yes

5 Tutor bruises for how long

6 Harry for about one year

7 Tutor for about one year OK and then what else

8 Harry and uh also bone pain

9 Tutor bone pain

10 Harry chronic constant bone pain for about six month
Tutor: where is the site?
Harry: um: involved the bilateral lower limbs
Tutor: bilateral lower limb
Harry: the bone below the shin area,
Tutor: right
Harry: as well as the ribs and the hip
Tutor: OK as well as the:
Harry: ) ribs
Tutor: ribs
Harry: and the hip
Tutor: the hip (. ) OK (0.4) what else?
Harry: um:: there are no other symptoms
Tutor: no other symptoms OK ( . ) I guess it’s the pain that brought her to the hospital
Harry: yes
Tutor: right OK so the history seems quite long (0.3) now this uh: let’s say there are two (0.3) uh two fairly different symptoms, that she complained of (. ) now what would you have to consider if the lady (. ) complained of bone pain? forget about the (^ ^ ^ ) concentrate on the bone pain (. ) a person complaining of bone pain for quite a long time, how (. ) what is her age?
Joy: fifty three
Zelda: fifty three
Tutor: fifty three (. ) what can give rise to chronic pain in uh: in a: lady of fifty three? {lo} what would you consider?
Students: {whispering}
Tutor: because once you started to ask a history and get the information out from the patient, (. ) then you’ve already got some information (. ) alright? so you want to (. ) start to formulate (. ) or to analyze (. ) what is or what are the likely differential diagnoses OK? once you start to ask the history the next thing you want to get is the differential diagnoses (. ) how how do you go about thinking of of possible differential
Zelda: uh is it for example in the history I want to know more. Is it mechanical or non-mechanical bone pain if there is resting pain?

Tutor: mm mm OK so that’s part of the history.

Zelda: then.

Tutor: so uh what is this

Zelda: because if it’s for example if it is a resting pain down at the worrying about the age as well worrying about metastasis some tumour like breast CA

Tutor: right. So what do you think? What did you get from the history? Is it related to movement?

Harry: it was not related to any movement um::

Tutor: mm

Harry: it is constant and the pain also persists during the night time

Tutor: does she still have pain now? Did you ask her?

Harry: uh:

Tutor: any (wrist) pain you know those sites

Harry: uh: she said the pain relieved

Tutor: it has been relieved

Harry: mm mm

Tutor: the: (0.6) maybe she’s better in these few days but uh: on the sixth of November (0.2) about two weeks ago she still had a set of X-ray on hip (.). So the pain hasn’t been gone for long (0.2) right? (.). Did you ask her whether she still has the hip pain?

Harry: mm yes

Tutor: yes so: what’s the hip pain like? (.). In fact when I asked her yesterday she said she still says she has the pain in the hip (.). Which side of the hip?

Students: left side

Tutor: left side left hip now OK she has pain in the left hip (.). How does the pain come on? (.2)
Eddie: in fact it also involves physical examination.

Tutor: mm mm

Eddie: during the exam in a resting state the patient did not complain of any pain,

Tutor: mm mm

Eddie: even the left hip, but on active external rotation,

Tutor: mm

Eddie: when the patient did it the left hip pain appeared with radiated down to the foot

Student: foot

Student: foot

Eddie: so does that answer your question?

Tutor: and you are I’ve got to try and remember your names

Zelda: um Z

Tutor: Z? right start good so what would you say mechanical or non-mechanical?

Zelda: mechanical because it

Tutor: mechanical good

Zelda: because it

Tutor: but what does that mean mechanical? is it pain which is elicited or the person complains of it when she is in a certain kind of manoeuvre? what information does it give you

Zelda: um

Harry: it is likely due to degeneration or due to trauma to that area

Tutor: degeneration maybe not necessarily degeneration when you try to move, when you try to walk, or when you try to bend over in certain positions, it is it may be related to the movement, which may be: you know what what moves when you start to move? the joint moves right? so it may be pain in the joints or it may be around the joints because tissue around the joints also moves maybe in the capsule, or in the surrounding auxiliary muscles, in the around the joint and sometimes when you do certain things, let’s
say when you ask the patient to lift a weight then she starts to have the pain or when the patient is standing she has the pain, or sitting no pain lying down no pain (. ) it may be related to the weight there (. ) it’s related to the force that you are trying to apply (. ) in the joint OK? so: in that case it may be part of the bone which stands the force (. ) if the bone is having a problem then it’s going to have pain (. ) OK? so much for that (. ) if it is not uh non-mechanical just pain all the time it may be inflammation just inflammation chronic (. ) or infection (. ) it doesn’t go away because with infection there is swelling there’s ab maybe even abscess so the pain is always there the tissue is always stretched (. ) if you move it, it gets even worse (. ) but if you don’t move it, it’s still there (. ) OK? (. ) right (. ) hip pain (. ) anything else? so that’s asking about the symptoms (^^^) information (. ) how how would you go about thinking about the differential diagnosis? (. ) do you have a system that you sort of go through when you ask the history uh to think of the differential diagnoses? any system that you use? (. ) I try to stress stress on system because I I mean even though hematology (0.2) this is the book I mentioned about this is the present edition (. ) I don’t this is not my book this is colourful I have the second edition {lo} which is black and white (. ) this is from my colleague (0.2) even for hematologists a simple hematology book is (static) a more detailed one is (static) but

71 Zelda {laughing}
72 Tutor it’s like my pillow
73 Students {laughing}
74 Tutor my pillow (0.2) and then you have all the specialties right? and you have different things like surgery obstetrics and within each specialty there are sub specialties how can you remember so many things if you don’t have a system?
75 Martin mm
76 Tutor no way (. ) OK? but but just mind you in the en:d, sometimes you have to go beyond systems (. ) sometimes you have to use your imagination but (. ) never mind (. ) systems first (. ) at least you have some ground
work (.) how how how do you go about thinking of differential diagnoses? (.) any systems that you have adopted or: or heard about?

Martin mm sometimes on the basis of )(^^(^)

Zelda ) vindicate {laughing})

Martin )pathological system like the inflammatory)

Tutor ) I see I hear this word every time I {Students laughing} so what is that word?

Martin vascular)

Zelda ) vascular infection inflammation degenerative

Tutor how how do you spell it? I don’t know the word)

Students ) V I N

Chris vindicate followed by

Tutor vin: {writing on board}

Chris followed by indicate I N D I

Students C A T E

Tutor E what is that?

Students vascular inflammatory

Tutor inflammatory

Chris neoplastic

Students neoplastic

Tutor neoplastic

Zelda con

Students degenerative

Tutor degeneration, yes

Chris infection

Zelda infection

Tutor infection

Martin clinical

Zelda congenital

Tutor con:genital what else

Students autoimmune

Tutor congenital autoimmune OK well never mind what about the next one?
Martin: trauma
Zelda: trauma
Tutor: trauma
Chris: maih toxins
Martin: toxin
Chris: or toxin toxin
Zelda: or toxin trauma or toxin
Tutor: or: toxin OK (0.2)
Zelda: environment uh
Joy: ) endocrine
Tutor: endocrine
Chris: or metabolic
Tutor: or metabolic
Zelda: or environment {Cantonese}
Chris: (^^^)
Tutor: is that where it comes from? who told you that?
Zelda: since the PBL era
Tutor: PBL?
Zelda: in year one )
Tutor: ) in year one)
Zelda: ) two
Tutor: this is a very (.) well you have the foundation (.) it’s very good
{Students laughing} but do you apply it? I don’t have this word I I I
hear this for the third time uh in the last month {Students laughing} I
never had this word in my mind (.) I think of it in a very simple way or
maybe I’m used to it so I think mine is simple (.) just ask yourself what
is the commonest condition in the whole world (.) that gets people sick?
or group of conditions shall we say?
Student: ) influenza
Joy: ) infection
Tutor: infection of course (.) you or I maybe a couple of you have it right now
huh but to me infection is the commonest alright? and then what is the
other or another common condition that you see around in this hospital?

Students degenerative)

132 Tutor ) degenerative what?

133 Martin in the elderly people

134 Tutor elderly people

135 Zelda ageing population

136 Tutor OK degeneration (0.2) uh: what else? what are other common things around (.) that you see?

137 Chris ) malignancies

138 Tutor ) or you see in the newspaper or you hear it you hear it on the on the radio talking about health health program

139 Martin (^^^)

140 Zelda vas)cular

141 Joy ) vascular

142 Tutor vascular yes vascular but uh: in fact what (.) what is the commonest vascular problem?

143 Zelda hypertension)

144 Tutor ) or couple of common vascular problems?

145 Zelda hypertension

146 Joy stroke

147 Tutor hypertension,

148 Martin atherosclerosis

149 Students atherosclerosis

150 Tutor atherosclerosis giving rise to?

151 Martin multiple (^^^) stroke, peripheral vascular diseases

152 Tutor stroke, peripheral vascular disease,

153 Martin coronary heart disease

154 Tutor coronary artery disease or ischaemic heart disease (. ) is it a vascular one? basically what is the pathology that lead leads to all this uh ) stroke

155 Zelda ) ageing degeneration

156 Martin ) ischemia
inflammation

ischemia is end the end result don’t forget

inflammation in inflammation?

tutor is ischemia ischemia) is end the end result don’t forget

inflammation is part of the pathogenic process (.) but what is the basic
lesion?

Eddie (local) embolism

this is atherosclerosis (.) what is the basic ab abnormality in
atherosclerosis? (0.2)

Zelda (^^^) {laughing}

Joy cholesterol

Zelda chole

Tutor cholesterol doing something {laughing} what does it do?

Zelda block (^^^)

Tutor in fact uh: it’s a vessel uh inside of the vessel the intima and the theilum
(.) which gets lipid deposit, you get a plaque, right (.) and then the
plaque somehow well various things get it enflamed, somehow the
plaque may rupture and block the the thrombus and then block up the
vessel and you get a stroke going uh: ischaemic heart attack or
sometimes, because the vessel is so weakened that it might just burst up
and uh end up with a hemorrhage in the brain (.) right? it’s part of
degeneration in fact (.) not just degeneration it’s also metabolic,
because it involves (.) the lipid

Student mm

Tutor so it’s a combination of two (.) metabolic problem starting off with the
lipid metabolism and then inflammation, and then thrombosis, and then
I can say it’s haematological right? it’s thrombosis (.)

Martin mm

Tutor so I have more job to do (.) I won’t run out of a job right? {Students
laughing} if you are if you are fully a doctor you can think of ways to
get yourself involved in things (.) if you just lower the the let’s say you
can lower the threshold for fasting blood glucose then you get more
doctors getting into endocrine {Students laughing} very simple uh to
get a job (.) and uh: of course nowadays uh the common thing is to ask
ladies to get themselves more beautiful {Students smiling} so you get lots of people into dermatology right? {Students laughing} good (.) now (.) so degeneration (.) vascular is in fact part of degeneration this is something in there what else? { door opening} oh

173 Student uh sorry
174 Tutor go over there too late (^^^) remember the (^^^) read the Bible? you have ten girls in here (.) whoever comes late is not welcome {Students laughing}
175 Student huh?
176 Tutor alright what would be the next thing that you consider common around this hospital?
177 Students neoplasm
178 Tutor neoplasm of course (.) those three (.) already takes up uh probably seventy per cent of our most of our workload (.) and then, what else? (0.3) it’s the same thing (^^^) (0.3) then there’s also inflammation I agree inflammation what but if this is inflammation uh if this is infection infection causes inflammation but let’s say how about inflammation on its own without infection what kind of condition(  
179 Chris autoimmune
180 Tutor autoimmune in fact, inflammation a lot of it is autoimmune (0.2) they don’t need a separate category of autoimmune (.) to me I think that is redundant (.) autoimmune {whispering} autoimmune sometimes doesn’t involve inflammation (.) when you think of autoimmune you think of conditions which are non-inflammatory autoimmune? name me one condition
181 Zelda {whispering} non-inflammatory
182 Eddie chlamydien
183 Tutor hmm chlamydien
184 Eddie chlamydien 17:25
185 Tutor um (.) OK (0.2) well depends, well some would say its also uh starts uh somewhat uh: either (0.3) cell inflammation and then it starts to degenerate (0.2) for example let’s say: (.) something wrong with the thyroid OK? (0.4) {Students whispering} something wrong with the
Zelda: autoimmune of course involves the immune system, so a lot of the time it involves inflammation. G cells, P cells, and there is also conditions that involves especially B cells which doesn’t give rise to significant inflammation for example myasthenia gravis its blocking up the neuromuscular junction.

186 Tutor: it’s the antibody OK? for example, hyperthyroidism, (0.2) PSI thyroid stimulating immunoglobulin which it stimulate the thyroid cells, secretes a lot of uh: up-regulate the thyroid gland and then secretes the hormone not not much of inflammation it’s not to thyroiditis it’s different alright? and then of course endocrine and metabolic it’s the same thing so I don’t care if you use that system this system is just um trauma is nothing to do with the uh de degeneration it’s just accidents, traffic accidents whatever, having a ball game, then you twist your ankle whatever a lot of it is orthopaedic problems OK and then some hereditary congenital problems coming to the bottom in paediatrics mostly, not not with us not with adult alright so this is something that you should go over with every uh patient that you see then you’ve got some symptoms what are the possible things that occurs in this particular patient alright now for example let’s take hip pain what is possible in this lady would you like would you like to give me a couple of conditions

187 Zelda: endocrine hyperparathyroidism
188 Tutor: so what of this hyperthyroidism(189 Zelda: ) hyperthyroidism
190 Tutor: hyperparathyroidism good PH because it increases bone metabolism the bone gets weaker and maybe reaches uh osteoporosis and you get pain in the good ok that’s one thing what else? anything anywhere

193 Joy: vascular say avascular necrosis
Tutor: avascular necrosis AVN (0.02) ~possible, (.) ok lets just name it first what else?

Zelda: autoarthritis in inflammation that also can be O A in degeneration

Tutor: OK O A (.) is it like RA?

Zelda: hmm not in the not in these big joints )

Tutor: ) yes so it’s not like

Zelda: ) I would expect more in the finger joints )

Tutor: ) finger possible uh likely ones more probable ones anything else? (0.10)

Chris: septic arthritis (^^^)

Tutor: septic arthritis

Joy: one year (0.2) yaat lin

Tutor: I dunno )(^^^) infection (^^^)

Chris: ) (^^^ Cantonese)

Tutor: septic arthritis let’s put it down first

Chris: for one year (^^^ several Students speakings at same time)

Tutor: anything else? (0.09)

Chris: neoplastic

Tutor: (^^^) let’s talk about the bone pain first

Chris: neoplastic primary or secondary

Tutor: good

Tutor: good plastic primary or secondary (0.2) and then you start to think of a whole big list {Students laughing} it’s a two page long list {laughing} (. ) anything else? inflammation, congenital (.

Zelda: trauma but you can get that from the history like

Tutor: right, ok so we don’t have it in the history?

Zelda: mm

Tutor: good at least you’ve got some (0.3) uh a few differential diagnoses now just just concentrate on what you’ve already (.) come up with (.) wha which is (.) tell me which amount is uh categories are unlikely (0.05) I’ll start with the first one avascular necrosis does it come on spontaneously?
there should be history for example steroid chronic steroid use (^^^) occupation

good so (.) if you have a patient that you suspect this may be the case then probably in the background then there may be some other condition that requires the patient to have long term steroid (.) uh but there are also other conditions without steroid that can cause you something uh sorry avascular necrosis for example not here in Hong Kong uh go back to UK you see it (.) they have a big haematology unit they have sickle cell disease there {laughing} the first time I see a sickle cell patient is is in UCH {laughing} {aww} and that poor young man had avascular necrosis {aww} from sickle cell anaemia (.) alright so (.) trauma can also give rise to secondary avascular necrosis (^^^) but anyway in this particular lady this is I would say pretty unlikely (.) its not in the history, the pain is not like that, {hmm} (.) OK so this is less likely as a prospect what else?

the septic arthritis because it has been (^^^) for quite a long time and )

) yes it has been for a long time

and she’s now feverish

uh:: when you talk about septic septic arthritis what are what kind of organism are you referring to most of the time?)

) (star form)

{Students muttering} good its pyogenic )

pyogenic

(u^^^) uh sometimes uh (0.02) sometimes even gram negative rods salmonella (.) these are infections that tend to give acute inflammation, great pain, sudden quick onset uh (.) patient go to see a doctor very very early right (.) I don’t know, no delay OK (0.05) another type of more chronic infection that involves the joint (.)

{whispering}

tuberculosis (0.02) not not so much (.) this usual this pyogenic (sepsis) PD OK chronic OK can this be the case? (.) it’s possible we have quite a number of tuberculosis patients around, (.) uh: the prevalence is high in Hong Kong, and uh (0.02) possible but is it common? (0.2)
Martin: TB joint

Tutor: TB joint, is it common?

Students: um {whispering}

Tutor: have you done your orthopaedics? how many TB joints have you seen?

Martin: {laughing} TB spine

Zelda: {Students laughing} one TB spine (0.2)

Joy: ) TB spine

Zelda: one TB kidney

Tutor: TB kidney and (^^^)

Zelda: not

Tutor: OK so it’s uncommon, it’s possible but quite quite uncommon, it can be: in fact it can present without much other history, just involve (^^^) starting to have a lot there of pain in the hip very chronic (0.02) so: it’s possible but uh it’s uncommon (0.02) anything else? osteoarthritis, is it likely in this lady? (0.04)

Martin: marginal (0.03)

Tutor: we haven’t gone over the physical examination

Chris: hmm

Students: {whispering}

Tutor: something from the history (0.02) fifty-three well you do have osteoarthritis at that age, but the important thing is not just with the history OK well we we are doing it very artificially we’re just doing it bit by bit but you go to see the lady, look at the joints, do you think that it’s osteoarthritis? (0.02) does she have?

Student: ) (^^^)

Tutor: these big deformed joints? (0.02) could you move them properly? one of you said you start to move her limb did you get this crepitation in her joints?

Eddie: hmm, I couldn’t feel any crepitation over the hip

Tutor: ok (0.03) and osteoarthritis can: (0.02) you roughly you can divide it into two types (0.02) OA involving certain isolated joints and OA involving a lot of joints, which is the more common one? (0.03)
177

250  Students  {whispering} (0.03)
251  Tutor  it’s usually: you did it (.) it’s a degeneration
252  Chris  multiple joints
253  Tutor  multiple joints (0.2) OK it’s the commoner one (.) sometimes you only
think this is well (.) uh: what we call osteo osteoarthrosis OK (.)
involving isolated single joints it’s usually due to? (0.6)
254  Students  {whispering}
255  Martin  secondary joints trauma
256  Tutor  yes secondary trauma because of dislocation or:: a fracture or involving
the joint you get secondary uh (.) joint degeneration (0.2) so if this is
osteoarthritis it’s likely to involve uh (.) more than one joint in this lady
and this is this is not the case (^^^) so this is a bit unlikely (.) or quite
unlikely in fact (.) anything else? (.) how about this PTH? (0.4)
257  Martin  she had quite diffuse pain
258  Tutor  diffuse pain? yes (.) anything else that would help you to (.) decide
whether this can be the case
259  Zelda  (the) typical stones and also abdominal pain ( (^^^)
260  Tutor  ( stones of ) what?
261  Zelda  ) kidney stones and ) also
262  Tutor  ) kidney stones
263  Zelda  also some uh some may complain of some abdominal pain then you can
ask in the history also maybe a (
264  Tutor  ( why do they get abdominal pain? (0.3)
265  Zelda  mm:
266  Students  {whispering}
267  Eddie  secondary to hypercalcaemia,
268  Tutor  so?
269  Eddie  causing increasing (calcium) transmission
270  Tutor  in the end?
271  Eddie  uh increase uh: excess secretion in the stomach (.)
272  Tutor  so what do you get (  
273  Eddie  (^^^)
{Tutor and Students laughing}

Zelda: hyper peptic ulcer right? so it’s hypercalcaemia and uh: the chain of events leading to peptic ulcer, pain, we have this term moans, bones, groans and stones right? do you still remember these four words?

Tutor: mm

Zelda: I can assure you I didn’t go back to study it for years and years (it’s these little tricks to remember things (fact it’s important to ask for other symptoms so did you ask it in your history?

Harry: mm there)

Tutor: ) definitely no pain?

Harry: no symptoms of hypercalcaemia so no no no pain or no yes no pain and no peptic ulcer history

Tutor: mm mm so she doesn’t have dypspeptic or dys abdominal pain, any problem with her: passing water? or no womb pain?

Harry: no pain or no increase (^^^)

Tutor: ) and then something, may not be that serious um some other symptoms which are less serious but still can be very troubling, constipation?

Student: oh uh

Tutor: you can get pretty: marked constipation because of the hypercalcaemia (0.2) OK you can go back to ask her (so this is just to illustrate when you ask (when you come to see a patient the history is important (you should ask for these things (when is the onset, what are the associated symptoms, and then of course in the end (^^^) the other systems whether there are also other problems (0.2) and anyway (even if you don’t ask the rest of the history is hypercalcaemia a common condition so by itself it’s uncommon so the chance of it being uh hypercal uh hyperthyroidism is low huh? and with her sitting in a hematology ward it’s of course not the case {Tutor laughs} (0.2) but uh it tend to come up in exams uh somehow the incidence increases with examination {Tutor and Students laugh} alright? so what what what is more common? trauma you can get it in the history which she doesn’t
have any history of trauma OK did you: (0.2) there are a lot of conditions that give rise to this kind of picture (. ) congenital then it’s extremely unlikely because she’s fifty-three that means this (. ) is this possible? (0.5)

287 Students {whispering}
288 Tutor neoplasm what could be a primary neoplasm? tell me one of them
289 Martin osteosarcoma
290 Tutor osteosarcoma everytime it’s the same {Students laughing}
osteosarcoma OK another one primary bone problem
291 Students {whispering}
292 Martin (^^^)
293 Tutor mm?
294 Students {whispering}
295 Tutor myeloma
296 Students (^^^)
297 Tutor myeloma is it a primary bone tumour?
298 Students {whispering}
299 Tutor if you turn to your orthopedic books I’m sure it is in your book because uh:: the malignancy of the bone is always mentioned there
300 Martin still a primary tumour?
301 Tutor we’ll we’ll come to that
302 Students {laughing}
303 Students ) {whispering}
304 Tutor ) but it is certainly something that you need to consider in a bone with a problem OK
305 Student {Cantonese}
306 Tutor secondary? I wouldn’t exclude it but I think it is good to keep it as a primary bone tumour so the orthopedic surgeons can remember
307 Students {laughing}
308 Tutor and be very alert to this {laughing} (0.3) OK? how about secondary secondary
309 Chris ) breast CA
(310) Zelda breast
(311) Tutor breast OK,
(312) Martin ) lung
(313) Zelda ) lung
(314) Tutor lung
(315) Zelda (^^^)
(316) Tutor these are the more common OK cancers oh (^^^) thyroid anything that we haven’t gone over> (0.2) a lady of fifty-three bone pain let’s say uh: )
(317) Zelda ) (^^^) thyroid
(318) Tutor ) if this is a lady who is older seventy pain in the back what would you consider? chronic pain in the back?
(319) Martin osteoporotic fracture
(320) Tutor osteoporotic or osteoporosis (. ) where do you put it you put it here OK (0.5) is this is it likely in this lady fifty-three? (0.3)
(321) Martin not likely
(322) Tutor not likely why?
(323) Martin because fifty something is still too young to have significant osteoporosis )
(324) Tutor speak louder)
(325) Martin yeh)
(326) Tutor ) speak to them not to me to them
(327) Martin yeh I think that fifty something is still too young to have uh significant osteoporosis )
(328) Tutor do you do you think it’s too young (. ) anyone agree?
(329) Eddie depends on when uh when did the menopause start
(330) Tutor good so when she start to have the menopause? so did you ask the menstrual history?
(331) Joy one year ago (0.2) uh one year ago the patient was menopause just one year ago
(332) Tutor Ok one year ago she was still having menses( .) is this likely? quite unlikely (. ) {hi} very important because it depends on when she started
to have menopause (.) let’s say sometimes you have a patient (0.2) this is not uncommon now uh:: late twenties or early thirties who uh: they have uh they have carcinoma of the breast or carcinoma of the ovary and they start to have chemotherapy in the end they get premature ovarian failure you start to have ovarian failure at the age of thirty (.) can they get osteoporosis by the age of fifty-three?

333 Martin mm:)

334 Tutor ) possible if they’re not on hormonal therapy OK so just don’t take the age at face value you have to ask the history (.) OK (.) so I’m emphasising time and again ask a detailed history (.) and how would you ask a history it’s only when you have something in the back of your mind that you want to clarify then you know what to ask for (.) which is the more important information which are less important (.) OK so I hope by going through this you see the point of asking a history and going over the differential diagnoses because it really helps you to think of what is the possible diagnosis (0.2) alright so much for this (.) leave it there for the time being (.) the other is bruises {coughs} I can’t go over it in great detail because if I do that you you would have to spend three hours here and then uh I’d have to show you my nice slides huh? {laughs} but bruises is a if you go over bruises it’s a bleeding problem but bleeding mind you is not just a problem with the blood (0.2) did I take you for bleeding disorders teaching clinic you should have one it may not be myself

335 Martin (^^^) disorder

336 Tutor is it? the teaching clinic on bleeding disorders

337 Students {murmuring}

338 Tutor you haven’t gone through it yet

339 Students {Cantonese}

340 Student not in my group

341 Tutor not in your group? oh I thought that everyone of you should have (.) but uh:: how does bleeding come about? this is the skin, this is the subcutaneous tissue, then you have blood vessels running underneath, you have the organ that (^^^) OK? if you have an internal organ this is
You can see the stomach)

) structure of tissue {laughs}

what is bleeding? bleeding means there’s a rupture in the vessel and the blood starts to come out this side, or that side, OK (. ) gastric ulcer, bleeding inside, trauma you have bleeding outside (. ) the blood in a way most of the bleeding is due to what?

Tutor trauma

trauma so in fact most of the bleeding is nothing to do with the blood (. ) the blood is just a passive uh uh: participant in the bleeding it’s because the vessel that ruptured the tissue that has been damaged and then the blood starts to come out (. ) the bleeding first of all is there something wrong with the tissue around?

Zelda mm

Tutor an ulcer, an aortic aneurysm, um (0.2) a trauma which is the commonest sometimes even things like less common things like arteritis the vessel wall itself and the vessel ruptures, so bleeding is just a sign that tells you there is something wrong there (. ) it’s not necessarily something wrong with the blood (0.2) so all those and {hi} then uh people who tends to bleed easily, uh (. ) so sometimes it is not a problem with the with the with the blood for example uh one common condition is called hereditary hereditary: uh: what is it

Chris hereditary telangiectasis (0.2)

Tutor hemorrhagic telangiectasis (. ) patients that get dilated vessels (. ) they tend to bleed easily (. ) again it’s a problem with the tissue OK? uh rare things like Ehlers-Danlos syndrome you have to remember this it’s it’s in my notes if you have it if you don’t have it you can ask ask around (. ) I’m sure you can get it on some web sites of the university these are problems with the tissue collagen formation defect most certainly alright? then once you start you have uh damage to the vessel, it is the duty of the blood to stop the bleeding OK not just the blood it’s (^^^) the blood vessels should contract restrict the uh the the amount of bleed involved in the clotting process it’s the platelets and the (^^^) OK so go over that I’m sure you have notes and whatever references can refer to
it’s not totally top rank ask his help (. ) uh but remember one important thing platelets and the clotting factors help you to form the clot to patch up patch up the wall in the vessel (. ) but that is not the end of hemostasis (0.2) OK the vessel is damaged fine you want to stop the bleeding but what is the ultimate aim of (. ) with with the tissue?

350 Martin regeneration?

351 Tutor regeneration (. ) do you want the blood vessel to be blocked forever? put a patch there?

352 Students {laughing}

353 Tutor no (0.2) if you have a hole in the wall of course you may put in a wooden board to seal off the wind but in the end you want to (. ) put in bricks to seal it up, put on paint, make it beautiful (. ) you want to regenerate (. ) and how can you start to make uh regeneration of course regeneration involves smooth muscle, fibroblasts, all those things but first of all you have to remove the board and then put in the bricks and cement )

354 Zelda ) mm

355 Tutor ) and the paint (. ) what helps to remove this clot that this clot of course (^^^) as well what helps to remove it?

356 Zelda plasma

357 Tutor OK you remember this plasma activator:: something like that

358 Zelda yes

359 Tutor but if you have a problem with {coughs} uh:: this part of the pathway you can still have bleeding if you have excessive up regulation of (plasma) for which there are certain conditions (. ) excessive which we call fibrinolysis (0.2) OK? the fibrin is being broken up too quickly and you start to have bleeding again (. ) and that kind of bleeding is very unusual because it is not a problem with forming the clot initially, so the patient can stop bleeding in in the natural process but the unusual thing is it starts to bleed again later on because whatever clot is formed there starts to break up early so this problem here with excessive fibrinolysis is usually delayed bleeding (0.2)

360 Zelda ) mm
OK? this is unusual OK every time you talk to a doctor or hematologist doctor I would say students would always think of this platelet clotting that’s not the end of the story there’s a bit further beyond alright? so {lo} now so much for those two (0.2) as I said I don’t have time to go over everything (. ) based on that information you can go and read up (. ) now anything in the physical examination that is worth mentioning?

Eddie mm as the patient is currently on chemotherapy there is partial hair loss )

Tutor ) uh what)

Zelda ) partial

Eddie ) hair loss hair loss

Tutor OK

Eddie and the um patient was on a central venous catheter on the right side )

Tutor ) yes

Eddie ) of the chest with some mild erythema around the inser insertion site ( . ) um: because of the lack of pain we have carried out a focal exam on the left hip ( . ) and the left hip pain uh occurred again when the patient actively externally rotated her the um no actively rotated her left hip um

Tutor mm

Eddie uh but the range of movement was normal and there is no um uh inflammatory signs that uh I could observe, any redness, uh increased temperature and or swelling ( . ) also the patient got a past history of stroke affecting the right side of her body so I’ve carried out a neurological exam ( . ) uh the cranial nerve was grossly intact, but for the upper limb and the lower limb the right-sided uh is general generally rigid in tone and also the power was decreased to four plus, (0.2) and for the reflex upper limb was normal but for the lower limb the reflexes uh was hyper-reflexive with ankle clonus on the uh right knee,

Tutor you said she had a stroke

Eddie yes

Tutor when was that?

Eddie mm?
the stroke when was that the stroke?

Eddie in 200X four years ago

(T^^^) OK

Eddie yeh basically this uh the uh positive signs (^^^)

so mainly it’s the hip pain

(Tutor)

the other things are the consequence of her immunotherapy (0.2) good

(. ) did you ask her to stand and walk?

Eddie yes )

Tutor yes

Eddie ) um I would say the gait was uh no not normal uh the patient was

having a hemiplegic gait affecting her right side of the ~leg causing uh

causing some circum circum circum gait

Tutor hemiplegic gait is it uh: significant? is it is it obvious?

Eddie yeh I would say it’s uh obvious

Tutor OK so fine (0.2) so that’s a past problem which is not the present more

acute problem

Eddie mm

Tutor now (0.3) we are trying to get at a diagnosis we have gone through

some differential diagnoses (. ) so out of all those: I hope I have

convinced you if you are not convinced (. ) some of those are very

unlikely like the vascular necrosis, septic arthritis, (^^^) but uh things

like TB hip, uh: hyperparathyroidism, uh: malignancies certainly still

quite possible with of course you now see the line there with the hair

loss and the history of myeloma but let’s say if you saw the patient

right at the beginning (. ) you don’t know where we where where you

know what is the exact problem so how would you go about trying to

get a diagnosis? (0.2) her bruises, and pain, (0.3) you get bruises in

hyperthyroidism?

Zelda mm no (0.2)

Tutor so how would you go about trying to get a diagnosis? so the second part

( . ) history, physical examination, then comes investigation I talked

about last time
Zelda (smiling) start with the simple test so we do)
Tutor ) good
Zelda ) the um for example because there is bleeding then we can do the complete blood picture)
Tutor ) good
Zelda ) look for the platelet count, and um also uh (heui) what uh and also can do the uh)
Martin ) {lo} clotting profile
Zelda ) do the calcium level,)
Tutor ) calcium level OK
Zelda ) mm (0.2) mm do the clotting
Tutor clotting
Zelda clotting profile PT APTT (0.2) mm although those are more likely to present with hemoarthrosis rather than bruises if it’s coagulation
Tutor more likely to be:, sorry?
Zelda if it’s a coagulating problem then I expect more more thing things like hemoarthrosis rather than {lo} ) maybe
Tutor ) OK yes,
Zelda and also can check the urine for paraprotein
Tutor urine for paraprotein (0.3) or the: )
Zelda ) oh and also do the x-ray of the hip (. ) very simple too
Tutor urine for para what kind of paraprotein you you may get to see in the urine?
Eddie immunoglobulin light chain
Tutor and what do we call that (^^^)
Students estrogen protein
Zelda (^^^)
Tutor Ok you don’t get the whole immunoglob you don’t get the whole paraprotein you only get the light chain coming out OK
Zelda mm
Tutor now now go let’s go over that one by one (. ) CPT she has bleeding you want to know the platelet (. ) of course if she has bleeding, besides the
CPT what else would you like to know? (0.2) how low is the hemoglobin, how bad is the bleeding, right?

Zelda mm mm

Tutor let’s see {reading from patient’s notes} (^^^) diagnosis (0.3) and of course: this is always given the white cell in fact in diagnosis (eleven) point nine, (^^^) two five four

Martin one five four?

Zelda two five four (.) normal ) {Students whispering in Cantonese} normal

Tutor ) yes (0.3) (^^^) equal what seconds

Students {whispering in Cantonese}

Student (gau dim uh APDTT normal))

Tutor )any comment? anyone?

Zelda prolong APT

Martin that is not quite long

Students {discussing together in Cantonese} )

Tutor ) it is normal in fact this is a little bit shorter this is a little bit shorter )

Zelda ) oh

Tutor maybe there is a reason why it is shorter {Students whispering} {lo} (^^^ calcium ^^^) (0.4) {hi} in fact she: her white cell count is normal, platelet count is normal, hemoglobin a little bit low: maybe even within the normal ranges I would think probably

Joy normal

Zelda uh I would like to see )

Tutor ) within the normal range because the lower limit here is eleven point seven ah it’s just within the normal range (.) it’s not even anaemic (.) PTA PTC normal (0.4)

Martin (^^^) bruises {laughs} this is at the presentation?

Tutor at the presentation yes

Zelda {gam} the bruises

Tutor real life case {laughs} I’ll try to find ) the calcium level

Zelda ) how severe (^^^)

Students {Students speaking at same time}
Student: how severe the (^^^)
Tutor: anything else uh:: I can give you the calcium if I can find it
Martin: {Cantonese}
Eddie: have the globulin (^^^)
Student: {laughs}
Tutor: ) calcium levels:
Zelda: ) {haih la} globulin hah
Tutor: calcium levels again normal even adjusted calcium is normal two point three six
Zelda: how about the globulin level? (0.2)
Tutor: before we go to that two point three six (.). now when you think of calcium what is also important in calcium besides calcium (^^^) ) calcium metabolism?
Joy: phosphate )
Eddie: ) phosphate
Tutor: what phosphate? other: inorganic phosphate OK what else?
Martin: ALP
Tutor: ) ALP why?
Zelda: ) mm
Joy: increased (^^^) will have increased ALP
Tutor: good (.). let’s say this is hyperparathyroidism what happens to ALP? (0.2)
Zelda: ) increase
Student: ) hyperparathyroidism?
Tutor: yeh hyperparathyroidism
Students: increase
Tutor: increase OK so uh taking a note of the uh alkaline phosphates would tell you whether this is hyperparathyroid or not this is an uncommon condition or it’s easy to pick up? (0.2)
Martin: mm
Zelda: mm:
Tutor: this is normal and the phosphate is: ? (0.2) point two zero yes it’s also
normal. What happens with phosphate when it’s hyperparathyroidism?

Zelda: Low if the calcium is high phosphate has to be low because they form a golden balance.

Tutor: What type of hyperparathyroidism are you talking about?

Zelda: Primary or tertiary.

Tutor: Good. When it is primary hyperparathyroidism in fact, what you are saying is the calcium phosphate product OK, one side is high, the other will be low.

Zelda: And also the uh: there will be increased phosphaturia the phosphate goes out of the kidney.

Tutor: So the uh phosphate level will go down. So it’s this PTH which is uncommon I would say even it’s rare OK. It’s easy to pick it up uh in the laboratory results OK so lookout for that. People have bone pain unexplained bone pain chronic especially chronic bone pain uh so this is high this is low this is high well look out for that. Some sort of unusual increased turnover of the bone if it is secondary, due to renal: chronic renal renal failure then of course the phosphate will be high. OK because it cannot be excreted so this already tells you this is not likely to be PTH, not likely to be hyperparathyroid. Protein we’ll go to that later: you mentioned about albumin, globulin we’ll come to that later. So this is the X ray. Have a look. You can come round and sit there. Uh I don’t have a whole set of X rays. Take a look see if you find anything. She in fact actually had other X rays at another hospital because she was...
previously seen at another hospital I don’t have the initial X rays but
this is the more recent one (0.5) (^^^) something wrong with the
acetabulum what is wrong? where?

478 Martin (^^^)
479 Tutor where?
480 Martin in the middle
481 Tutor point it out clearly (0.2) somewhere here? (0.3) you mean here? (0.4) now because she has this pain it is not obvious uh? so the thing is you have a history, you have your physical examination, if I just put it up for you without any history you’d probably miss it (. ) but if you have that history and findings and physical examination and you look carefully at the left hip, and I: just (. ) look at the right side first (. ) this is the pelvic rim right? inner pelvic brim you see this line: which is (^^^) of the pelvic bone whi
482 Students {murmuring}
483 Tutor uh see it? (0.3) it’s not so smooth
484 Zelda mm
485 Tutor overlapping
486 Students mm
487 Tutor a fracture (. ) fractured hip is not bad (. ) don’t expect any significant displacement huh (. ) fractured hips do not get significantly displaced but just uh compared to this side this is a ~very smooth curve, (.) symphysis pubis (^^^) this is a fracture (0.2) anything else?
488 Zelda um just this point (not quite sure what is this )
489 Tutor mm mm
490 Zelda not sure because (^^^)
491 Tutor the shadow the shadow very
492 Zelda not a shadow but I thought (^^^)
493 Tutor this jutting out?
494 Zelda I’m not sure about the: along just along (^^^) I’m unsure if it’s (^^^)
495 Tutor compared to the other side
496 Zelda because (^^^^)
497 Tutor this little bump here is the lesser trochanter (.) OK? this is the cortex
498 Zelda oh I thought the cortex looks a bit
499 Tutor compared to the other side does it does it look the same? {Zelda
laughs} any difference between the two? (0.2) this is subtle
500 Martin thinning of the ) cortex
501 Zelda ) mm thinning of the cortex
502 Tutor thinning of the cortex, on which side
503 Martin left ) side
504 Tutor left side (.) thinning because of what? compared to this side, you see
this is a little bit darker
505 Zelda mm
506 Martin mm (0.2)
507 Tutor that’s an osteolytic lesion very subtle (0.2) uh: this what we would
describe uh this is not a not a not a punched out lesion as such but what
we call )
508 Students {whispering} osteolytic
509 Tutor ) moth-eaten lesion a moth-eaten appearance (.) we seldom have moth-
eaten clothes now, we used to have it
510 Zelda mo mo
511 Tutor moth
512 Zelda moth what is the meaning
513 Tutor moth-eaten you have these bugs these moths )
514 Zelda ) {lo} oh::
515 Tutor uh sometimes they lay eggs in your your clothes and whatever they
start to rot away (.) there’s a little patch there not really bad but uh:
starting to get thinner compared to this side (.) that’s why you see this
cortex is sort of more healthy, the whiteness on this side, you start to
see this darker patch (.) alright?
516 Martin mm (0.2) {lo smiling} subtle
517 Tutor in fact uh: I got it in the notes you should have she should have shared
some lytic lesions in the skull as well (.) I don’t have those things (.)
this is good enough

518 Martin  so this is what we have for the bone multiple myeloma

519 Tutor  yes this can be anything . multiple myeloma, CA breast, CA lung, just a lytic lesion in the bone (0.2) talking about the X ray itself (can’t get) anything just a lytic lesion in the bone but it is not a bone cyst . bone cysts are very clear with a very clear margin

520 Martin  mm

521 Tutor  this is moth-eaten that means the margin is not well-defined (.) it’s very sort of blurred (.) OK? (0.2) alright let’s move on (0.2) we should have more X rays (^^^) so with those investigations I think we can do (pretty) (0.9) so: it is not likely to be hyperparathyroidism, but just to mention that this is osteoporosis, calcium phosphate alkaline phosphates can all be normal(.) osteoporosis you can diagnose it on an X ray (.) very uh: osteopenic bones with degenerative uh: edges (^^^) and this is a uh obviously uh a malignancy most likely a malignancy in the tissue which is really common (.) the question is whether this is primary or secondary (.) you mentioned about osteosarcoma, multiple myeloma, breast:, let’s say lung OK so in your examination check for clubbing, check the trachea, the chest whether there’s any fusion, things like that, uh any lymph nodes, examine the breast, this can tell you, not all of the time but a lot of the time (.) even the thyroid as I said (^^^) osteosarcoma, is it likely to start off? this is a primary bone tumour, but she’s fifty-three (.) it tends to occur when? in what age group? and what site? (0.3)

522 Chris  around )

523 Tutor  ) come on our visitors are very quiet {laughs} (0.5) where do they tend to occur in the body? (0.2) any idea?

524 Student  long bones, femur )

525 Student  ) femur,

526 Tutor  femur yes that’s common (.) it’s a common site (.) anywhere else? (0.2)

527 Student  the long bones

528 Tutor  the long bones yes (.) not so much in the axial skeleton (.) the axial skeleton means the skull, ribs, (^^^) body, pelvis, (^^^) in the limbs
basically the long bones (.) not not the extremities (.) this part (.) this part (.) OK not the extremities (.) so this is it occurs in young people and it’s very easy to detect because it swells up (.0 very painful usually not a chronic pain quite quite sub-acute (.) the point is to if it is primary then we have to consider these things (. ) multiple myeloma there are other primary bone tumours such as osteosarcoma, uh: giant cell tumour, those (are common) things (. ) those mostly diagnosis with biopsy, or sometimes they get very uh: peculiar, specific features on the X ray (. ) now with her I think the primary type of common bone tumour it’s multiple myeloma (.) now we I wouldn’t mind people calling it a primary bone tumour (.) it’s in the bone anyway (.) and it is multiple (.) that’s why you do multiple X rays at different in different sites (.) so the important thing is what one of you mentioned A and G, albumin and globulin was: (0.3) (^^^) (0.2)

529 Zelda mm:
530 Tutor (^^^) is seventy (0.3) so what do you notice? (0.2)
531 Student increase (^^^) increased globulin
532 Zelda increased globulin
533 Tutor albumin is: upper limit is fifty (^^^) I didn’t put down upper limit is normally the upper limit is forty yes forty (0.2) this is the upper limit of the normal range
534 Student increase globulin
535 Tutor OK so the normal is (^^^) and the albumin?
536 Students decrease )
537 Tutor ) decrease (.) so for a normal person the A/G ratio, (0.2) is weighted at one (.) albumin should be greater than the globulin (.) and there are conditions that give rise to a reduced A/G ratio such that it becomes less than one (.) and of course the reason is either the albumin that goes down for example in nephritic syndrome albumin that goes up or in conditions that increases the albumin uh sorry the globulin uh:: of course you would think of multiple myeloma but there are a lot of other conditions (.) other inflammatory conditions (.) for example rheumatoid arthritis, (^^^), chronic inflammatory conditions that increases uh
chronic liver disease, cirrhosis in the that means cirrhosis of course the albumin also goes down (.) globulin goes up because in chronic inflammatory conditions you will produce more globulin especially the hema gammaglobulin (.) so this ration will reduce (0.2) and in malignancy as well not just multiple myeloma a lot of malignancies the albumin will go down (^^^) poorly and then the albumin will go down (.) this this is a reverse (^^^) but obviously in this particular case it is the globulin which has gone up quite a lot (.) almost double yeh (.) considered higher than the than the normal range which is a lot (.) but this is in fact a very obvious um result to alert you to multiple myeloma (.) so the next thing of course would be to do? (0.3)

538 Student ) Ig pattern
539 Tutor ) you almost have the diagnosis
540 Chris ) pattern )
541 Student ) (^^^)
542 Chris Ig pattern
543 Tutor immunoglobulin assay yes gives you the IgG, IgA, (^^^) these are the things you read in your reports right? have you seen this?
544 Students mm
545 Tutor so let’s see (0.2) what was it (0.5) {lo} (^^^) one and sixteen seventeen OK and in fact they actually measured the paraprotein (.) obviously you see this is a lot higher than normal the upper limit is about hundred and sorry seventeen hundred this is almost double (.) this is still within the normal range but on the low side (.) this is in fact uh again within the normal range on the low side OK? this has gone up a lot and then in fact you can measure paraprotein which is what is this this must be IgG (0.6) ten (^^^) thirty five two grams per litre (.) that means that out of the seventy grams of uh: globulin you have thirty five half of it is paraprotein (.) IgG kappa (.) OK, this is kappa only because this is monoclonal (.) alright? so you get the diagnosis? IgG kappa (0.3) OK? you don’t do investigations for no good reason (.) the other thing: now that’s why I don’t I didn’t give you the urine (^^^) protein at the beginning (.) there’s no need to do it initially (.) because: (0.2) a lot of
the other results will tell you whether this is necessary

546 Zelda mm mm

547 Tutor I seldom ask for that uh:: in the beginning but if you’ve already got X
ray evidence uh: (plates) globulin of course do it by all means (0.2)
I don’t have it here this is protein

548 Students {whispering}

549 Tutor not recorded ah yes in fact they did it they did it in uh: Princess Diana
Hospital (.) it’s uh:: it’s kappa light chain (^^^) together this is a this is
a definite case of multiple myeloma (.) what’s what’s the feature what’s
the characteristic feature of with (^^^) protein? (0.4)

550 Martin (^^^)

551 Tutor anyone (^^^) )

552 Kevin ) (^^^) when you heat to a certain degree then when you further heat up
it will dissolve

553 Tutor wha wha if you heat up protein just usual normal protein, cause usually
you don’t have protein in your urine let’s say nephritic syndrome you
heat up the urine wha what happens?

554 Kevin it will denature ) coagulate

555 Tutor ) it will form, coagulate coagulate and form what?

556 Kevin a solid

557 Tutor not exactly a solid

558 Students precipitate )

559 Tutor ) precipitate becom the urine becomes sort of uh murky with little
particles suspended inside OK? and what temperature? any idea?

560 Joy sixty

561 Tutor any one of you study biology? you know: matriculation days (0.2) don’t
tell me all of you studied maths (.) {laughs} I studied maths I didn’t
study biology )

562 Zelda I studied biology

563 Tutor uh? you studied biology (.) did you do an experiment with these little
sugar beet? chop it into dices?

564 Zelda I went to UK so)
you were in UK never mind they can do the same experiment {Students laughing} put this sugar um sugar beet dice into a beaker, and then start to heat it up, measure the different temperatures of the beaker, and look at the colour change (. ) did you try to do that? this is a very interesting experiment

I remember a (^^^) burns

no: the solution this sugar beet the solution at first the water the water is of course clear uh it’s not just simple water water uh water of the same tonicity otherwise just plain water would burst out of the cells so you keep the same tonicity I can’t remember the tonicity (^^^) but you start to heat up, and then the cells will start to denature and the uh: the uh what do you call it the pigments inside the sugar beet will start to leak out and then you start to get a colour in the solution )

) mm:? ) in the fluid (. ) did you try to do that?

(Cantonese)

very interesting so you measure the the colour of that fluid, at different temperatures and then the the you put it on the graph the the intensity starts to increase increase increase the temperature on this side the intensity on this side starts increasing up to a certain point it flats out because all the cells have bursted you don’t get any more (^^^) plateau (. ) remember that temperature oh you don’t do this interesting experiment, even I who studied mathematics did it

it’s around forty five to sixty degrees around fifty odd it flats out, the cell bursts, protein damage, membrane gone (. ) OK? it starts to coagulate the the pigment comes out (. ) so around sixty degrees you get this precipitate but with (^^^) it’s different (. ) you heat it up further, close to boiling point, what happens?

dissolve

dissolves again (. ) different from the usual protein that you see because the other proteins if you heat them up further what do you get? (0.5) you don’t have you taken down these uh: you see you have to make it
interesting

Students {whispering in Cantonese}

Tutor sorry say this in Chinese (yeung cho daan)

Zelda huh?

Tutor (yeung cho daan)

Zelda oh because (^^^) eggs

Tutor these eggs you put in you boil it for these uh: post partum ladies it’s a sweet vinegar with ginger and uh:)

Chris )(Cantonese)

Tutor (^^^) the longer you boil it the harder the egg will become (. ) if you go to Taiwan, you have these tihn daan) iron eggs

Chris ) ohh: tiny tiny little eggs you throw it at people and hurt {Students laughing} this is if you boil it further the cooking just gets stiffer and ) stiffer

Zelda ) overboiled

Tutor overboiled yes that’s the usual protein you get it hardens up (^^^) because by that time the chains are deformed (. ) the globin the globulin chains are deformed and start to spread out (. ) good (. ) now

Eddie uh excuse me I want to ask about uh immunoglobulin assay

Tutor yes?

Eddie for the paraprotein uh IgG counter uh uh do we need uh another specific test to order or does the result come from uh come with the immunoglobulin assay also?

Tutor uh:: the immunoglobulin assay what we usually do is is order for the globin content immunoglobulin pattern (. ) then they will give you the IgG, A, and M alright? if it is normal they will also do no if it is normal they won’t do anything else but they will also do the serum protein (^^^) to find out whether there is a monoclonal (^^^) OK? but that will not tell you the uh kappa or lambda (. ) to tell the difference between whether this is kappa or lambda you have to do what we call an immunofixation assay a few different assays (. ) the reason I bring along this book (. ) it’s just to show you one picture (. ) it’s about this SPE serum protein electrophoresis {lo} because I didn’t take a picture of it
go back and have a look we’re bound to go over time

Joy  SP electrophoresis

Zelda  mm

Harry  mm

Joy  SP la )

Tutor  ) two seven two this page or this page

Harry  (^^^) (0.2)

Chris  IgG ah {.Cantonese}

Tutor  this picture )(^^^)

Students  {whispering in Cantonese})

Tutor  ) stand a bit closer now the blue: the blue shadow this is a serum electrophoresis and how is it done? it’s done with two electrodes, uh: OK one is positive one side is positive one side is negative, you put the protein I think in the middle, or somewhere in the middle, and then it will start to spread out usually it’s closer to the negative pole because most of the proteins are negative and will start to move towards the positive pole

Zelda  m mm (0.2)

Tutor  OK? so the: smaller ones will start to move quicker (0.2) alright? and the smallest one is albumin and the amount is quite quite a lot so you have a big blue peak normally, and then you have little peaks that follow so that the albumin globulin the cut off is around here: the blue peak here is all the all the uh: albumin the rest of it if you add it all together, that is the globulin that you get the globulin is not just one single globulin there are different types of globulin alpha one alpha two beta delta OK? these these small blue peaks and you see these peaks are sort of quite broad because the molecular sizes of these molecules are spread out alright? now just to mention what would they do? most a lot of them are binding protein for example haptaglobin you’ve probably heard about, you have a binding protein for uh uh:: hormones, especially the steroids steroidal hormones, a lot of these are binding proteins uh things like transferrin that carries iron around, OK? but then the last peak gammaglobulin is the thing that
we’re interested particularly in (.) (female) globulins (. it should be a
broad peak because you have IgG A M D E and M is much bigger, the
usual size is IgG the molecule size is much smaller so even within the
gamgoglobin it’s spread out (. a a broad peak (. if you have multiple
myeloma, then: because of the malignant condition the production of
albumin will reduce, to the red peak you see it’s lower than the blue
peak and the rest is about the same, then you have a big huge sharp
peak at the end here which is the monoclonal protein that you get OK?
so this is the SPE (. this of course doesn’t tell you whether this is
kappa or lambda (.) it’s the immunofixation (. OK remember this (.)
now let me show you a few slides

Students  {discussing together})

Tutor now of course you know multiple myeloma when you start to treat
them, uh:: hopefully you will expect the uh: expect the paraprotein to
come down right? {lo} can you turn on this thing? (0.4)

Eddie it’s on

Tutor dim the lights (0.2) should have turned it on earlier (. need time to
warm up

Zelda mm yes (^^^) (0.5)

Tutor this one (0.2) good enough right? (0.11) (^^^) (0.5) in fact this is a
grand round presentation that I prepared a couple of years ago (. there
are four cases in here which I I don’t think I have time to go over every
one but I just want to highlight a few things (0.2) now all these are real
cases I’ve treated, uh a thirty eight year old gentleman, an engineer,
pain in the back for some time, went to see the orthopedic surgeon,
examination mainly showed of course there are many other
examinations mainly tenderness at the T6 level, localised so uh (0.3)
{lo} let’s go down next page page down

Student (^^^)

Tutor {lo} oh I see I see I see it doesn’t turn why (. come on OK there’s a
little bit of compression at that level (. you can see the spine is a little
bit bent (. can you see? a collapsed vertical body (. this may not be
very clear (. we’ll go to the next one (. it’s actually a collapsed
fracture OK if you can’t see it trust me (^_^) {Students laughing} so: as usual OK? this patient have a batch of investigation and you notice that his calcium level is high, you’ll notice in this particular patient A/G ratio is normal, alkaline phosphate is a bit high, which is explained by the fracture (.). OK in fact remember that patient we just talked about (.). the alkaline phosphotase is normal, even with osteolytic lesions in the bone (0.2) and that is typical with multiple myeloma (.). although they have osteolytic lesions the alkaline phosphate doesn’t go up unless they have a fracture (.). if there are other secondary lesions let’s say cancer of the breast ongoing to the bone usually the alkaline phosphate will be up (0.2) so this is this the peculiar thing about multiple myeloma (.). and this one the alkaline phosphotase is up because a little bit up because of the collapsed fracture (.). so this is a fracture no trauma so is this multiple myeloma? we’ll see (.). all these these two are normal OK (.) now the orthopedic surgeons did a series of uh uh investigations this this is of course a CAT scan in fact if you see this is the T6 you see these lesions in the vertical body, it’s holes OK? sort of like Swiss cheese like holes (.). it’s inside the vertical body (.). you see that? it’s like holes yeh not through your telescope yeh this is even through the telescope you can’t see black holes (.). now these are even more obvious (.). and you see this is not just in the T6 level (.). these other lesions in the lower levels, which doesn’t seem to show up on the X ray actually it shows up on the uh CAT the CAT scan yes (0.3) alright? (0.2) and this is a collapsed T6 very clear alright? fortunately it has not compressed on the vertical: I mean the spinal cord right? now the the it pick up a few gallstones but that is not important (.). she also have uh renal stone (.). so there are multiple lesions with the MRI (and CT) so uh:: they even went on to do a PET scan but I would say uh: we’ll come to that a bit later (.). you can see that the T6 shows up with a slight increase in uptake that should be slightly increased but the rest of the spine looks OK (0.2) OK? (0.5) when I was asked to go and see the patient I asked for one thing (.). which they haven’t done (.). a skull X ray (.). you see these little pinpoint uh not pinpoint quite small size, punch out lesions
all over the skull (. ) alright? so a skull X ray is much much cheaper and quicker to do than a MRI:, or CAT scan, or a PET scan OK? if you have that, collapsed spine, umm: what do we call this, pepper pot skull you know the first thing I will think of is uh: multiple myeloma ( . ) you remember this globulin is normal ( . ) it’s not the usual type of multiple myeloma, so start thinking ( . ) can multiple myeloma give rise to no increase in globulin? ( 0.2 )

611 Zelda non-secretory ) type

612 Tutor ) non-secretory good ( . ) any other type? ( 0.3 ) (lo) uh we can skip these ( . ) (lo) never mind so the next thing, is a bone marrow biopsy ( . ) you see these uh plasma cells, if I can (lo) (^^^) these are the plasma cells ( . ) now plasma cells produces immunoglobulin ( . ) that’s why they stain up very blue (lo) plasma cells ( . ) so they are active cells, they produce immunoglobulin so they have to make RNA ( . ) that is why their nucleus is not condensed ( . ) cells which have condensed nucleus can you name me a couple of them? (^^^) cells

613 Students {whispering}

614 Tutor the simplest one is cells without nucleus

615 Zelda {laughing} red blood ) cells

616 Tutor ) red blood cells why? because it has already condensed while it is inside the marrow it is before it comes out of the marrow that the nucleus is struck out ( . ) you don’t need a nucleus OK? so: that means cells that are inactive and not actively producing protein you don’t need a nucleus OK throw it out ( . ) so what what so red cells is not really red cells but normoblasts ( . ) the nucleus is condensed ( 0.2 ) and later on they will be thrown out ( . ) to form the red cell, no more nucleus ( . ) uh: {hi} neutrophils this is not really a neutrophil but close to a neutrophil ( . ) it’s by the time they come out to the circulation they are they are inactive, they look for a fight ( . ) right? to fight off bacterias ( . ) but if there is no bacteria around they don’t fight ( . ) so they don’t need to produce any protein ( . ) it’s not active ( . ) they don’t need any sort of (excessive) metabolism ( . ) but cells that are producing protein the nucleus is not condensed ( . ) that’s why you often see this desc this
description of clock face nucleus (0.2) that is the: and then the
cytoplasm is (blue) due to what? (0.2) they are producing proteins,
immunoglobulins, and what do you need to produce protein?

617  Student  RNA

618  Tutor  not in the cytoplasm (^^^) it’s produced and then transported out (.) but
with the RNA what do you do?

619  Eddie  ribosomes

620  Tutor  ribosomes yes (.) it stains up blue (0.2) the ribosomes (.) cells that are:
very active in producing protein it stains up blue 9.) but then it
produces a light chain and a heavy chain, these are produced separately
(.) you need to link them together to form this this Y shaped structure
right? and where do you:: uh what does where does it take place in the
cell?

621  Students  {whispering}  (^^^) Golgi apparatus

622  Tutor  Golgi apparatus yes and where is the Golgi apparatus?

623  Students  {whispering}

624  Tutor  paranuclear hoff (.) the clear zone (0.2) OK? so even the morphology
tells you something (.) it’s not just (0.2) any cell (.) it’s a cell that is
actively producing protein with post-translational modification (^^^)
secretion and there are lots of plasma cells taking up quite a percentage
of (^^^) cells and you can easily find some binucleated ones (.) {lo}
this is starting to split (.) alright?

625  Martin  mm

626  Tutor  so this is why morphology is so interesting because you have lots of
pictures to look at (0.2) now: this is a bigger magnification, alright, you
see the very clear paranuclear zone which is the hoff where the Golgi
apparatus is (.) you see you see the red cells with dense nucleus,

627  Zelda  mm:

628  Tutor  alright? now this is a marrow oh sorry the aspirate when you take the
(trephine) the thing is slightly different that is why it is not so blue, but
then you can still find some blueness in the cytoplasm, it’s not so:: )
uh:: not so diffuse

629  Students  )  {whispering}
Tutor: a bit of blueness uh usually um: on one side of the cell and a background of pink alright because it stains a bit different.

Chris: (^^^)

Tutor: and you see the nuclei they are very rounded most of them are rounded or ovoid and it’s all packed up OK it’s diffusely involving the marrow now you see the blueness clearer alright? you see in some of those cells uh depending on the cut of the uh slide, some of them you see the blueness under the cytoplasm OK? that’s a typical and the nucleus is the nucleus is pushed to one side because of all the other activities in the cell it’s not in the centre OK now this is staining this is doing immunostaining staining for kappa, is it kappa? oh this is a different patient not the one that we are talking about so this kappa stain doesn’t stain up anything so if you compare it with the lambda stain it’s all stained up so this is light chain restriction um uh a clone of cells producing all of them producing the same light chain OK? very clear now the important thing is we went on to do so how to explain this absence of hyperglobulinaemia? seventy per cent of plasma cells this is definitely myeloma and where where has the globulin gone? is it a non-secretory one? well we’ll see in fact the IgG IgM are low this is very common in multiple myeloma and this is what we call immunodiuresis if you produce too much of the abnormal one thenormal one somehow gets suppressed OK in this particular patient that we were talking about, this lady, that you presented, there’s no obvious immunopareshis but most multiple myeloma patients do have immunopareshis so: uh: we look for other things now the Ig the usual immunoglobin pattern will give you the A G and M, so: in this particular case we look for something else and ask for the IDD as well, now what conditions will not give rise to a raised IgG or A M? either this is a myeloma that involves the other chains, the D or the E. but then most of the time you will think oh well even if there is D or E around then the total globulin should also be high right?

Martin: mm
but this is not so then let’s say this is a non-secretory one, the immunoglobulin will not be (high) or if this is just a light chain myeloma some myeloma cells only produce this kappa or lambda light chain so again in those cases the globulin will not be high so there are a few reasons that the globulin may not be high for this particular patient that I present this is the IgD now why is the total globulin not high? it is because IgD the normal level is in brackets, it’s only around three hundred milligrams per litre compared to the upper one you see. it’s let’s say the uh IgD is can be up to over a thousand milligrams per decilitre that means even the normal concentration or amount of IgD in the blood in very low OK very little amount of IgD around (0.2) so even if it is raised ten times it doesn’t show up much in the total globulin you understand?

so remember multiple myeloma doesn’t always have raised globulin most of the time yes but not always OK light chain myeloma no raised globulin, non-secretory no raised globulin OK? so forget about this I don’t think you need that anyway you see this patient is treated and you see the globulin went back to normal in fact, later on, and the protein is completely gone and then he had a bone marrow transplant, a few years ago and I can tell you he is still very happy, doing his own job now no problem his donor is a sister a nursing officer in the orthopedic ward in fact {Students laughing} do we have time? I’ll just show you: forget about these {lo} these are uh::: uh: forget about all these no time let’s go to the last bit now OK maybe this is worth telling you this is another patient who presented a few years ago of course with a swelling in the sternum a large very large bulging out in the centre of the chest, and in fact it was very easy to do a biopsy, a direct biopsy of the mass and it’s a plasma cell tumour now this is growing from the in fact it’s growing into the tissue the subcutaneous tissue OK the marrow you see the marrow percentage of plasma cells is less than ten OK but then it’s also producing a lot of IgG and it’s monoclonal IgG lambda so in this
particular patient it’s what we call a plasmcytoma, a big lump of growing in the chest wall, and it’s easy to diagnose by a direct biopsy somehow the marrow is not particularly involved alright? so it can be very localised that’s why we call this oma a tumour plasma cyte plasma cell tumour plasma cytoma OK? and she was treated with a lot of things uh: radiotherapy, multiple chemotherapy, even thalidomide and so on still with progressive disease and I can show you this is the biopsy you can even see a trinucleated cell in the centre, you see that? big round cell with three nuclei? all these are plasma cells staining with lambda OK? tumour in the sternum, but extending beyond the sternum, you see both the outer table and inner table of the sternum are completely destroyed, getting into the subcutaneous tissue, but no no particular other lesions around this shows up in the (CAT) scan, a huge hot lump and uh: even on the chest wall later on this is a picture later on when it starts to spread not just in the sternum but when it starts to spread around in the subcutaneous tissue alright? in fact it grows on the (^^^) line OK? I presented this because we used a new drug, oh by the way it’s also in the pancreas we did a because she complained of some abdominal pain the tail of the pancreas is increased, of course we didn’t do a biopsy, but we think this is most likely the tumour also inside the pancreas and this is after one course of the new drug (^^^)

637 Students {whispering}
638 Tutor uh?
639 Student (^^^)
640 Tutor this is not dermatology huh not a cosmetic lesson huh {Students whispering} but (they) like to show these pictures and the pancreas has shrunk in size alright? so I I uh: I just want to show you that this can present in some other atypical ways forget about that, now the last case is also worth mentioning a bit because don’t think that plasma cells can only give rise to uh myeloma, or plasmacytoma sometimes it behaves very:: um: in a very benign manner this is a gentleman OK and he’s still around he was sixty six uh thirteen
years ago, now seventy odd, I still see him once in a while, once a year I think (.) he went to see the cardiologist because of his hypertension (.) he also had gout and they checked the blood and so on (.) a bit of impaired renal function and then they found that the IgA is increased, it’s IgA kappa monoclonal, did the bone marrow, but unfortunately the bone marrow not done by ~me, it wasn’t adequate you can’t see much in the bone marrow (.) and then (^^^) is is normal no obvious lesion (.) and A/G ratio is normal so the question is is this multiple myeloma? is the renal impairment due to his plasma cell (^^^) problem? time will tell (.) he is clinically quite well (.) so uh I just decided to follow him up (.) so that’s the beginning uh a few years later, quite a few years nine, ten years later you can see immunoglobulin level IgA hasn’t gone up much, a little bit down maybe (.). laboratory fluctuation(^^^) nothing significant this time we repeated the marrow, six per cent plasma cells, creatinine same (.) so the paraprotein is not causing any organ damage in terms of cell count, that is the marrow, in terms of organ damage in terms of say the the the kidney, no bone lesion, no fracture, no bone pain, OK? this is MGus monoclonal gammopathy in the old days we called it benign idiopathic benign mono:gammpathy (.) now the new term MGus alright? so multiple myeloma and MGus they survive a long time OK

641 Zelda mm mm
642 Tutor {lo} never mind never mind (.) so this is why my title was M and M
643 Zelda {laughing} M and M
644 Tutor I don’t have the chocolate today {Students laughing} I thought of getting it for you (.) so: they can present in different patterns {lo} come on (.) it can present as a malignant condition like multiple myeloma which is the commonest condition, OK, involves the marrow everywhere, OK? the typical feature of course is the M component and you have multiple lesions sometimes in the bones of course and also involves the kidney, sometimes it can even give rise to things like uh amyloidosis things like that, but that is a less common my myeloma (.) OK multi organ involvement, kidney, bone, as I have mentioned,
sometimes it presents as a mass (.) plasmacytoma and uh: in a number of patients it is a quite milder condition uh: which we call MGus and they live for many many years alright? so do you have a copy of this (^^^) because it’s for you to remember not so you to copy {Students laughing} OK (0.3) that’s the end (.) OK? no::w (0.2) in fact there is a very interesting case right now outside in got admitted I think got admitted yesterday or the day before (.I’m really tempted to tell you about this case because it is so much (0.3) {hi} so what what do you expect with a paraprotein level if you treat the patient with chemotherapy and they respond?

645  Martin  decreasing )
646  Zelda ) decreasing
647  Tutor decreases of course (.) it sounds very stupid to us this question (.) so if you see a reduction in the paraprotein: or even the complete absence of the paraprotein you will expect the patient to be (0.2) well

648  Martin  mm
649  Tutor OK you’ve got rid of the uh: condition right? (.but sometimes things do not follow the normal pattern, especially in malignancy OK remember this (.malignancies are conditions that do not follow the normal pattern because they: to to start off a malignant cell is not a normal cell, they don’t behave in a normal physiological way (0.2) {lo} (^^^) (0.4) so if you go out and see a patient, alright I’ll tell you which bed now I remember the name Lau Lok Yung look up the name list and find him (. he’s at the back there this is another patient with multiple myeloma who {lo} I can’t remember his IgG or IgA (.raised paraprotein levels, we treated him with a few courses of chemotherapy to (.his paraprotein went very nicely down to nothing even with the immunofixation it’s completely absent (.it means (.what we call complete remission (0.2) it seems to be a good result, but unfortunately he developed a lump in the in the wall in the abdominal wall (.the lump is still there go and have a look (.just like the lumps that you saw just (^^^) (.we did a biopsy, confirmed plasma cells, here, in the subcutaneous tissue (0.2) so what is happening? use your imagination I
said use your imagination

650  Eddie  relapse?
651  Tutor  re:lapse (0.2) uh OK)
652  Zelda  (^^^) lymphoma)
653  Tutor  we can’t really say it’s relapse because he never really got into a coma
  {laughs} uh
654  Martin  ah?
655  Tutor  remission because as the paraprotein went down this lump came up (.)
  because there’s no period of complete clearance of everything (.)
  anyway the disease is still there
656  Zelda  ) (^^^) )
657  Martin  ) another plasma?
658  Tutor  hm?)
659  Zelda  )because (^^^))
660  Martin  ) another lymphoma in the
661  Zelda  lymphoma in the ?
662  Tutor  this is plasmacytoma
663  Martin  another (0.2)
664  Tutor  first of all this is is this tumour secretory?
665  Martin  non-secretory
666  Tutor  must be non-secretory because you don’t detect anything in the: serum
  (. you’ve confirmed it to be (plasma) cells (. is it responding to the
  chemotherapy you have been giving?
667  Zelda  no
668  Tutor  no but the others are (.) the oth the bone marrow is completely clear
  (0.2) so you see when you start to treat patients it’s not just multiple
  myeloma but with any other patient with cancer or you can patients
  with infection, resistance can develop (. if you imagine the bacteria (.)
  you are more familiar with bacterias right? it’s an organism,
669  Martin  mm
670  Tutor  you give antibiotics, it responds, it dies away but uh but sometimes a
  few of them develop resistance
OK this is evolution of the organism. The bacteria they develop resistance and the sub-clone of bacteria behaves slightly differently from the original bacteria. Same thing can happen with cancer. That is why a lot of cancer patients can have relapses. Not all the cells respond to the chemotherapy that you can give, and some of them, as you treat, the others escape and (grow) they can behave differently. In this case it doesn’t secrete anymore, it becomes resistant to the drug. Some cells they remain dormant for many years, and then relapse later. That’s why you have late relapses. Even patients with breast cancer five years is not absolutely safe. Ah waiting for five years (we see) patients relapsing after seven or ten years alright? So this is real life not just pictures. OK I have to go I’m late for my meeting. Now if you have any questions you are welcome to ask me, you know where I am walking around the corridor (0.3) if you don’t have time to ask now ask later bring all these back to the patient’s bedside.
Medical Specialty PBL Session

5th Year Students: Keith, Ron, Trudy, Jan (chair), Sue, Fay and Larry

Jan first of all after yesterday it’s about the investigation of uh…

Tutor no it’s not that

Jan about the history of this patient

Trudy well I went back to the patient uh yesterday so uh regarding theee uh chronic rheumatic disease uh it was discovered about uh thirty years ago uh patient had uh malaise at that time and went to see a private doctor and he was also diagnosed with hypertension in that time but he did not take any drugs until about …until about ten years ago um

Tutor you may …. at the side. Noise of chairs scraping floor as late arriving students come in and sit down

Anne ok

Trudy so the antihypertensive medication was prescribed about ten years ago by a private doctor and uh for the

Tutor just a moment how you still have not given us sufficient detail this is still the history of the present illness WHY was he diagnosed with rheumatic heart disease thirty years ago? he may might have forgotten but he can say that he might have forgotten but why did he go to see the doctor?

Trudy He said he has some generalised malaise

Tutor And then how was it diagnosed first?

Trudy He said the private doctor diagnosed it.

Tutor Mm for the first time….

Trudy (nods)

Tutor And he was only given drugs for the hypertension?

Trudy Twenty years after

Tutor I know I know but he he is not given any drug for the rheumatic heart disease

Trudy (Shakes head) …..and uh for the warfarin um I asked him why was it necessary and he couldn’t say …and uh for the ….

Tutor When wa the when was he started warfarin?
He wasn’t started ….

When was he suggested to have had warfarin? ….What information would that how would that information help you?

Um with the onset of the atrial fibrillation

Yes not the onset the first detection atrial fibrillation

I asked him when was the onset of the atrial fibrillation and he said at the same time when the chronic rheumatic heart disease was

So thirty years ago ….are you surprised?

…. I think it should occur later

Thirty years ago

than the onset

Why? …. 

Maybe …. 

I thought you had all read up on ?? heart disease…. 

So the rheumatic heart causes damage to the valves and like if there’s MS there may be affecting the atrium

Mm mm

so leading to

so how does it affect the atrium?

Increasing the atrial pressure

And then?

it causes dilatation of the dilatation

Yes dilatation of the left atrium. Is it only mitral stenos..I agree mitral stenosis is most damaging but is it only mitral stenosis?

Mitral dilatation will also cause

Mitral regurgitation

Yes mitral regurgitation too….So are you Back to my original question are you surprised that the patient was detected with atrial fibrillation when he was detected with rheumatic heart disease? …. 

I think it takes time to develop before uh before the atrium become dilated and causing arrhythmia and first uh other uh ??flow problems may present

Because he hasn’t got any symptoms other than malaise which is probably
not related

44  Jan  But as you have mentioned yesterday um the cardiac problem may have
      started since ch uh youth so there may be

45  Tutor  Since childhood yes so what?

46  Jan  So there may already be heart damage before this malaise ever started

47  Tutor  When was he detected? How old was he when it was detected?

48  Trudy  Maybe fifteen

49  Tutor  He’s now what?

50  Trudy  Seventy seven

51  Tutor  So he’s had thirty years ago so he would be what

52  Trudy  Forty four

53  Tutor  Forty four or forty five or thereabout and you are surprised that he should
      have atrial fibrillation?

54  Trudy  Probably maybe the heart problem already started in his

55  Tutor  Probably probably in his t teens or before he was ten years old so I’m
      NOT surprised at all actually I thought you people have taken the studied
      this history rheumatic heart disease and you still …have to have no idea
      that was the first time it is detected does it mean it is the first time he
      developed the rheumatic heart disease? He must have it years ago for
      decades so I am actually not surprised….

56  Trudy  And uh for the renal problem I asked him when this started and he actually
      said a few years…uh I gave him some suggestion and he said about five to
      ten years

57  Tutor  OK

58  Trudy  and then

59  Tutor  which means at what age?

60  Trudy  Sixty …seven

61  Tutor  Seventy….OK

62  Trudy  And he presented with again some weakness and he went to […]…. For
      the thrombocytopenia he said it was discovered around two years ago
      during the blood taking in the regular follow up for […] However he did
      not have any admission because of that
63 Tutor  What about his bone marrow done?
64 Trudy  ….Uh ….it was…. 
65 Tutor  As an outpatient?
66 Trudy  (low voice) I think he needs to be admitted for bone marrow
67 Tutor  I know Ok you can well you can do it as an outpatient but but most of the
time you do actually admit the patient.…. 
68 Trudy  And for the gastritis he said he did not remember…. For the gout it started
uh he said twenty years ago and uh he just sometimes bought some over
the counter ointment
69 Tutor  Wh what about the history of the gout?
70 Trudy  …. 
71 Tutor  Are you really incapable I was I was I’m going to ask you to repeat the
whole history again you seem why are you why are you not capable of
giving a good history you only have got seven more weeks to go…. Yeh
for the gout what are you going did he say? ..... 
72 Trudy  Said just that buy some over the counter ointments sometimes to
73 Tutor  But how was it diagnosed the gout but he’s on allopurinol
74 Trudy  And then later the private doctor gave him some medication
75 Tutor  But where oh come on what do you people want to ask?
76 Jan    How did he presented with?
77 Larry  Has he had any acute attacks of gout
78 Fay    Is it one side or two side ..... ...... 
79 Tutor  How long ago was it again?
80 Trudy  He said he actually forgot most of the dates I only gave him suggestions
81 Tutor  Yes OK I understand you give some suggestions after he’ll probably give
you some date after ultimately what does twenty years five years or four
years or two years actually mean to you in the long term?
82 Trudy  He said twenty years
83 Tutor  Twenty years yeh
84 Trudy  And then for any uh like symptoms of infection before this admission he
said he had some cough for uh ten days with uh whitish sputum
85 Tutor  Now you people start to present the history can you start present represent
the whole history of presenting illness in chronological order and dates

86 Trudy Mmm for the history of present illness our patient was discovered to have
uh chronic rheumatic heart disease uh thirty years ago when he presented
to a private doctor with uh malaise and uh was diagnosed to have uh uh
moderate uh MRN, AR and mild AS and [TI??]

87 Tutor Was he already diagnosed with all that in the first place?
88 Trudy Umm…. he had follow up in Grantham after….
89 Tutor So he was followed up in Grantham for thirty years …. Mm mm
90 Trudy At the same time he was also diagnosed with hypertension but he was was
not put on any antihypertensives …and…also um ….
91 Tutor It was also suggested that he should have warfarin
92 Trudy It was also suggested that he should have warfarin but patient refused
because the warfarin required dietary restriction and uh twenty years ago
um he also
93 Tutor So he received nothing… or….for his rheumatic heart disease. When was
this […] whatever?
94 Trudy He said the private GP gave him some medication about ten years ago
95 Tutor You said he was not given any drugs in Grantham …it’s almost
unbelievable
96 Trudy He said he wasn’t…. and uh uh twenty years ago he was um diagnosed
with gout and over the years he purchased some over the counter ointment
and a GP gave him some medication and uh ….five …. around five to ten
years ago he had renal impairment discovered by a private GP in
Central…..
97 Tutor but no symptoms
98 Trudy He just said this….and uh
99 Tutor uh uh what symptoms did you ask for?
100 Trudy I was ask I would ask for like any polyuria
101 Tutor I thought he had polyuria, polydipsia ….even now….
102 Trudy ….and so any malaise or […]
103 Tutor No polyuria polydipsia is probably the most definite ….malaise is so
indefinite anything you give us with malaise look listening to your history
now will give me some malaise ………. But I thought he had ….he still has polyuria polydipsia

104  Trudy  Polyuria
105  Students  yeh
106  Trudy  And also um…. um for these uh few years he also complained of some [BPH??] symptoms such as weak stream and slow stream but he did not he did not seek any treatment for that …. And uh…around uh…two to uh two years ago he was found to have thrombocytopenia after follow up and the bone marrow uh biopsy uh found some uh uh benign monochromal [??]
107  Tutor  He started bone marrow how can a bone marrow biopsy found some benign monochromal renopathy
108  Trudy  I’m sorry I mean only six per cent blood cells [??]
109  Tutor  OK what blood cells
110  Trudy  Immature uh..
111  Jan  Plasma cells
112  Trudy  Plasma cells
113  Tutor  Plasma cells and then benign monochromal um benign monochromal renopathy was found by what?
114  Trudy  Um ….I think they had some blood
115  Tutor  What blood test would show
116  Jan  [serum??…..]
117  Tutor  She she’s supposed to have gone over the history.
118  Trudy  Electrophoreshis
119  Tutor  Yes …. 
120  Trudy  ….And also um uh for the uh he had according to the case notes he had gastritis in 2007 but the patient forgot
121  Tutor  OK …. [that I can accept]
122  Trudy  And for uh …. And then I in uh 2007 he had an episode of um admission it was uh drug-induced uh TCM induced uh acute interstitial nephritis …. and he presented with uh increase of uh creatine in like the blood test was … um follow up at Grantham…..
How do you more or less how can one more or less deduce that it’s due to TCM?.... ....

Because uh it’s acute increase

Yeh ....maybe....and what?

And also there’s some increase in the liver [....]

How can one blame it on TCM and it’s acute tubular nephrosis you’ve already mentioned he’s got renal failure ....how did he did he how he expect the doctor to diagnose it’s possibly acute tubular nephrosis due to TCM?

Find out what kind of TCM he’s taking

Possible and what else?....Yeh?

Um there will be showing um acute uh tubular acute and chronic renal failure

Yeh and then what ....

Mmm....

.... the patient will recover to the basal level ....after a while ...there should be OK the patient’s kidneys are normally the first stage in the complete recovery and you may [...] something ....like what?

Maybe what if we do a toxicology screen possibly find the specific [....]

OK ....which is probably unlikely for TCM....OK there should be complete recovery ....go on

and at the same time he was diagnosed with uh drug-induced intra hepatic cholestasis

yeh

and uh....

Which resolved completely again I presumed

And for 2008 ...um....he was he uh in March in March he presented with an episode of uh chest infection, uh resulting in hemoptysis and hospitalisation for two days and this for this.... this episode he presented with uh progressive shortness of

So it’s only this episode I thought yesterday you said he’s got [breathlessness] I’m waiting for the breathlessness to come actually....
Um I asked him how many episodes for this year he could not remember the exact number he said two or three and I looked in the case notes there are two episodes in QM

For what?

Uh the one is this the chest infection

and what?

and one this

But yesterday you said that he’s he’s been breathless for a long long time and you gave and unconvincing history because you mentioned about seven alternatives and also orthopnea

And uh

So that’s all made up …. I asked the patient like in the past he said the shortness of breath started for about five years ago and I asked

Only five days?

Five years …uh and I asked him how many episodes he had he usually had and he said he forgot and

why did you not mention about the breathlessness just now? …. Sorry I should have….um …. he could only remember the two episodes this time […] he forgot …. Can you describe the breathlessness? …. So um the …. The he had increased breathlessness for one week uh exercise tolerance was uh reduced to um shortness of breath on um even on showering um uh compatible with [heart association] class 3 and heart failure …. And um there was

Just a moment just a moment …. only if he’s really heart failure but he’s not even so far not according to previous during the intervals he’s not got any symptoms of heart failure….is anyone who cannot shower is your class 3 heart failure?….even he let us say got bronchogenic carcinoma with pleural effusion …. I think uh like the the previous uh uh shortness of breath episodes were due to heart failure but

I thought before I thought before we had gone over this yesterday since
he’s got [...] tricuspid regurgitation…he really [would not have palpable]

160  Trudy  Yeh I also asked him whether there was a period of uh relief in between like the first onset it was more serious and more relief and then it like worse again and he said it-it was just progressive and getting worse

161  Tutor  No no no I’m not talking about the breathlessness I’m talking about orthopnea I’ve already said actually after […] breathless because he’s still his oxygenating is still going to be bad he’s still going to have pulmonary hypertension

162  Trudy  Yeh I have

163  Tutor  His orthopnea is going to be relieved

164  Trudy  Yes I asked about

165  Tutor  You all understood what I said yesterday?.... You asked what?

166  Trudy  He I asked whether orthopnea improved

167  Tutor  You haven’t really described orthopnea today do you realise? How could he have it if you have not yet described orthopnea? ….

168  Trudy  so um ….I asked him he said he need to uh like prop up

169  Tutor  And then? Was it promptly relieved?

170  Trudy  It was relieved

171  Tutor  When was when did he start to have this?

172  Trudy  I think for the past few months….  

173  Tutor  OK ….

174  Trudy  And I uh asked about the paroxysmal nocturnal dyspnea …um this time he said he was seldom seldom suddenly woken up

175  Tutor  It’s not just a matter of suddenly waking up I said with paroxysmal nocturnal dyspnea with a classical description it should not be relieved by by anything it should be quite severe

176  Trudy  And so I

177  Tutor  It’s not the waking up I said but if you fall asleep immediately on lying in bed then you’ll be woken up even if you’ve got orthopnea orthopnea is promptly relieved by sitting up….

178  Trudy  so I don’t think it’s any paroxysmal heart disease …. And over this time the shortness of breath was not associated with chest discomfort but ….uh
associated with bilateral uh pitting [...] oedema. ...he also reported

179  Tutor  I really just find the history far from **far** from excellent actually I will probably just pass you in the [...] with this this history.... You can’t even you don’t even know how the gout presented which are [...] simply unforgettable by the patient. How could if he realise that he’s got gout he **would** remember what is his pain .... OK go on ....do you mean you haven’t given me as any clue as to when he started diuretics or ooorr hypertension he did

180  Trudy  [inaudible]

181  Tutor  I know what about the diuretics? .... Never mind ....

182  Trudy  ....I think the anti-hypertensive [...] and the this episode of admission ended before that he had some uh cough with whitish sputum and 2132

183  Tutor  Just a moment what just a moment what the anti-hypertensive patient is [...]diuretic] what is he on?

184  Trudy  [...] And previously

185  Tutor  You give you give furosemide for treatment directly of hypertension ....

186  Tutor  He said before he was he was given he was given the same drug and then after admission doubled it

187  Tutor  That’s what you furosemide is for for treatment of hypertension you would give furosemide for treatment of hypertension .... What diuretics if you are going to give diuretics at all if you wish to treat hypertension?  
[...**looking through notes**]  

188  Students  Thiazides

189  Tutor  Yes thiazides .... [...] what drug is actually what drug is the patient on [handing notes to Trudy .....] Or rather what drugs are the patient on?

190  Trudy  Patient on uh allopurinol for the gout and uh aspirin uh

191  Tutor  for what? What do you think?

192  Trudy  To uh to prevent any [...] of heart problems

193  Tutor  yes the patient refuses to [...] probably we’ll put him on aspirin

194  Trudy  And also uh the [...] ....

195  Tutor  **IS THAT ALL I THOUGHT THERE WERE SIX DRUGS?**

196  Trudy  Sorry .... On on discharge the drugs are those dologesics and
[antihypertensive] drugs

197 Tutor Yep
198 Trudy and so uh now on discharge he’s only on allopurinol aspirin and furosemide
199 Tutor is it? What about the fifth agent?
200 Trudy Because it says treatment [….pointing to notes]
201 Tutor What?
202 Trudy It says keep record only does it mean that it’s ….[hands notes to Tutor]
203 Tutor […..]
204 Trudy Beta beta blockers
205 Tutor What do you think it’s being given for? ….
206 Trudy For the heart failure …. 
207 Tutor Only uh actually beta blockers the treatment of heart failure is usually only for ischemic heart disease …. 
208 Trudy Or maybe for lowering
209 Tutor Thank you …. [hands notes back to Trudy] ….why do you think […..] right now
210 Trudy I think [
211 Tutor Wow do you mean you don’t know you use beta blockers to treat heart hypertension? …. 
212 Trudy I think they uh because they increase the dosage …. Of furosemide
213 Tutor What?
214 Trudy they increased the dose of furosemide that it was […..]
215 Tutor What uh what do you people think why was it done […..] positive for a while?
216 Students Because [……] of the heart failure symptoms
217 Tutor Yeh number one thank you yes be beta blockers can actually worsen heart failure in […..] severe heart disease number one why else what other reasons has he been […pale?] for a while? [ …. [Students writing notes] when will you actually prescribe beta blocker for heart failure actually I’ve already given you some inf but you should tell me again ….when
218 Keith When you stabilise the patient …. 

220
Tutor: For?
Keith: for the for example giving diuretics
Tutor: For? For 
Students: Ischemic heart disease
Tutor: Ischemic heart disease NOT for rheumatic heart disease ….for ischemic heart disease AFTER you’ve actually controlled the heart valve which is […] heart failure heart disease then the beta blocker is supposed to improve the uh survival rate. I’m only but when during the acute phase of heart of failure why would beta blocker be worsen heart failure why would it
Fay: Because there could be hypotension …. And also decrease the contractility of the heart
Tutor: yes it decreases the heart rate …. heart contractility ….is there any other reason you can think of OK this is probably the major reason you want him to be out of heart failure first you already prescribe […] antihypertensive agent why else do you think the patient may not have beta blocker […] ….. they probably find uh blood pressure on admission is probably too low …. And then there will probably be assessment in out patients to see whether he should need to be started on hypertension ….on any antihypertensive agents …. Look you you you not actually be able to tell the […] but he actually refused to list out the word […]metopelo? […] Go on …. Actually I don’t think we’ll waste time much on on the history again ….so what aspects what aspects do you think you want to discuss again?
Jan: Like the shortness of breath
Tutor: It’s actually I know it’s more uncomplicated slightly complicated but after two days of preparation you should have plenty of time to arrange it well.
Jan: like the shortness of breath…. like in these five years how how does it progress or is it […] frequent exacerbation …. Is it worsening ….
Trudy: […] Because I think like the patient said more than ten […] but I don’t remember ….
Tutor: Just a moment but in between he was OK?
He said it got worse and worse especially these days so because

Actually when we saw him yesterday he was really absolutely absolutely OK not a single hint of breathlessness much less orthopnea .... that he requires morphine on admission doesn’t mean that he’s getting worse and worse but you you didn’t even ask

He said he got worse

Ju jus just now actually yesterday he was breathing you all saw him he was really actually completely normal not not there’s not a hint of breathlessness or - I haven’t really counted the breath rate .... is the patient discharged?

Discharged at lunchtime ]

Yeh that’s the reason he was so well he was so well.... Actually can ....can you suggest for example why the patient would be periodically admitted let’s say ten times? ....

I can think of drug compliance in this case]

Yeh?

because the symptoms seems to improve so quickly after admission and then the drug may be still OK]

[...] and anything else? ....

any concomitant illness like infection?

Yeh infection he just has infection .... For someone with heart disease actually a very trivial let’s say influenza infection probably worsen it ....

I asked the patient in clinical [....]

Yeh Ok it may be just trivial chest infection ....after all I I I have probably had two or three actually this winter .... In spite of the flu vaccination .... So just any mild mild respiratory infection may probably make it worse .... And and your history of orthopnea I still really doubt it so OK right now yesterday he maybe he did not manage to um his TI is probably not very prominent uuh yesterday um because go on how do you diagnose decide what’s his TI?

Giant B wave pulsatile liver, and uh uh sys weak systolic murmur because it’s covered by [....]
Tutor: [...] systolic murmur [...] he’s got this what kind of liver has he got?

Ron: Enlarged

Tutor: What has not got of the three signs of [...]?

Students: [...] pulsatile liver

Tutor: [...] ?

Ron: Cirrhosis

Tutor: But one thing that he says that his TI [...] well-controlled now is

Larry: [...]?

Tutor: Mmm He has a mild [...] with his TI I would expect him to be normal no [...] No orthopnea .... Someone who is breathless actually very often do sit up ..... most people just don’t lie flat in bed having feeling breathless .... And he he I just don’t trust the history if you ask her the history a little bit it’s actually it won’t take you that much time .... I think unfortunately I was asking letting you ask it with me it would probably be in five minutes I would disentangle it his so-called breathlessness or orthopnea .... Less than five minutes .... Without leading him too much either he’s but allowing him to actually give .... But because if you even after two days you still cannot ask him so ask him [...] and can’t even get the history of gout .... Which to me is obviously almost un un unbelievable that you cannot actually get a history of which joint are being involved .... OK anything else you would like to discuss about the history? ....

Jan: Only the history part of the clerking?

Tutor: Yeh ...

Jan: I think we’ve done quite much

Tutor: I think you have there are certain aspects I personally am interested .... What about his hypertension? ....

Larry: [...] does he present with any complications

Students: [...]?

Tutor: he was actually first diagnosed to have hypertension at what age?

Trudy: Twenty years ago

Larry: Twenty years ago

Ron: Look for any secondary causes
Tutor: Yeh well OK he was diagnosed with it thirty years ago he was would have been around forty-seven um I would pass I would probably […] maybe he should be unlikely is it likely to be secondary hypertension or is it just essential hypertension he’s the age of forty-seven I think which one?

Students: […]

Tutor: I think it’s probably essential hypertension …. A little bit early but I still think it’s probably essential hypertension …. And then the renal failure …. When was that detected?

Trudy: Around uh five to ten years ago

Tutor: So he was already what?

Keith: Sixty-seven

Tutor: Two years ago he’s now seventy-seven ….So what will we …. what will we do we need to do some investigation about his renal failure?

Fay: Twenty-four

Tutor: At that time what?

Fay: Twenty-four hour […] to estimate the GFR?

Tutor: Yeh OK

Fay: (See how bad it is) ….

Tutor: Or maybe the cause is the renal failure ….?

Sue: Could it be]

Fay: Hypertension

Tutor: Yeh may be um hypertension which is not very well-treated it probably wasn’t actually since he’s not a very compliant patient …. What else what else can cause the renal failure? …

Larry: As the patient has a history of TCM maybe the patient was taking

Tutor: Bit I thought we concluded there is that it causes acute […] necrosis which he should completely recover ….?

Sue: Could be the drugs for treating gouty arthritis like NSAIDS to?

Tutor: has he been given NSAID? …. He hasn’t even got any history ….OK possibly you should think about whether we don’t know that he’s taken NSAIDs and anyway analgesic properties is not really that commonly encountered nowadays ….most people know about this ….therefore they
do tend to avoid it .... Anyway you’d need quite a few dose of NSAID before you can cause it OK yeh you should bear that in mind yeh 3537

286 Fay Could it be obstructive nephropathy
287 Tutor yeh possibly yeh
288 Fay for a patient with uh gout then we can think of the patient have uh uric stone
289 Tutor Uric acid nephropathy….yeh so he’s got he OK he’s already seventy years old OK and he may just be atherosclerotic anyway yeh we’ve already mentioned that it’s hypertension um gout and also he also have BPH …. all these he could have three major causes for for his renal failure….yes I think this is OK uh yes the gout I mean OK the gout is it primary gout or secondary gout?
290 Trudy I think it’s uh primary
291 Tutor Why
292 Trudy Because um first it started before [....] and secondly he’s not he’s not on any any antihypertensive drugs for gout
293 Tutor On what?
294 Trudy On any any anti hypertensive drugs
295 Tutor How can antihypertensive drugs cause gout
296 Trudy I think like [....] diuretic ....
297 Tutor any other thing that can give rise to ....anyone.... I thought we I mentioned it yesterday actually……. Furosemide can also increase uric acid .... But we don’t know when he was started actually I think it’s unlikely I do not believe that he was only given furosemide ....um I uh I would have thought that he was probably given furosemide right at the beginning .... Um I’m not sure of course since you haven’t given us a clue as to when he has he has uh any breathlessness oor uh until five years ago uh and any any ankle oedema I haven’t got a clue as to when he was started on furosemide I would have thought that he uh since he was diagnosed to have uh uh [....haemolytic??] disease uh thirty years ao he probably have been started on furosemide ages ago um if he only had gout around ten years ago it was probably unrelated whereas if he was given
um if um if he was given um gout the gouty arthritis related furosemide I would have expected him to have started um the furosemide around that time …..OK [...] going on addresses chair]

298 Jan [...] the history

299 Tutor I think we’ve gone we’ve gone through..

300 Trudy [...] 

301 Tutor OK we’ve already gone through the aspects of hypertension, um renal failure and also gout….yeh…. 

302 Jan Would you like to like us to discuss about the investigation?

303 Tutor Yeh …OK….. …..How will we discuss the investigations?

304 Jan For this admission?

305 Tutor Yeh first emergency investigations first …. And then and then for the back backdating history repeat it afterwards 

306 Jan So for this episode of acute shortness of breath what investigations you would like to do with this kind of patient?

307 Fay I think in the physical examination also do the [...] because with this kind of patient [...] check whether there’s any [...] proteinuria or blood in the urine check and then other basics exams like taking the blood for CBC because there’s a chance for chest infection check whether there’s any any elevated white cell count um and then liver renal function because um patient had um deranged renal function and also 

308 Tutor [...] what about an emergency like stress

309 Students We check the saturation and ECG, chest X-ray, heart failure

310 Tutor Yes yes you do the chest X-ray first …. Yeh you what

311 Students ECG

312 Tutor Yeh chest X-ray and ECG ….OK I would like to do another white cell count yeh ….chest X-ray we’ve looked at yesterday 

313 Students Yes

314 Tutor you were not here yesterday [addressing visiting student] why don’t you show it to her [addressing Trudy, hands X-ray to Keith] ….She can see it [pointing to light box on wall]

315 Tutor [...] why don’t you point it to her irregularities and how do you
Keith Pardon and how to measure the heart etc let’s just repeat it just …. [Fay placing film in light box]

Keith So Professor L said yesterday that

Students [Laughter]

Keith (^^^)

Tutor […] it’s not me OK

Students [Laughter]

Keith We should separate the heart into uh half right and then we find uh the maximum ….um

Larry distance

Keith The maximum length on the on each side and we add them up and divide that by the total […] uh width to find the [cardiothoracic ?….] ratio this is opposed to finding just any kind of random measurement […] the width

Students (….)

Tutor Yeh and then what else? OK it’s obviously large actually anyone can see the heart is large …so what other [measures?] are there?

Keith Oh sorry and then we also want to uh look for any uh maybe uncommon […] so there doesn’t appear to be very much [congestion] over here there doesn’t appear to be very much [congestion] over here

Tutor But what about the cardiac border come on there are mmmultiple abnormalities in this chest X-ray

Sue […] aortic […] pulmonary […]

Tutor yeh

Sue This is the aortic uh knuckle ]

Keith Knuckle

Tutor which is quite normal actually it’s on the small side for somebody who is seventy seven yeh yeh

Sue This is the left atrium

Keith […]

Tutor noo […] it is actually a little bit prominent yeh

Keith and …
Tutor: and what
Keith: we have the left atrium over here
Tutor: Where?
Keith: […]
Tutor: Yeh And where else can you see the left atrium
Keith: It goes all the way down to this area
Tutor: Nuh which where is it?
Students: [Cantonese/ English switching]
Tutor: Speak in English
Anne: It’s alright
Keith: Sorry and then we have the […]
Tutor: So can you please hold on the left ventricle is …the girl ..he seems un a little bit un a little bit ..
Fay: This is the left ventricle
Tutor: No the left atrium where is it? Left atrium
Fay: The left atrium is here and then this is the double atrium double atrium side
Tutor: double atrium side it’s not …. double atrium side where is double atrium side?
Fay: [getting up to point to film][leung go…]
Keith: Nido this um
Tutor: Yes the right atrium and the left atrium where is
Keith: The left atrium
Tutor: [addressing chair] why don’t you why don’t you point it out because
Jan: this one um
Tutor: Yes one rim is the left atrium the left atrium
Jan: This is the left atrium [tracing on film with hand] and this is the right atrium so these two
Tutor: It’s a double atrium […] and the left ventricle is where
Keith: [pointing]
Tutor: And the right ventricle?
……
[shakes head] [Tutor stands up and goes to light box]

367 Fay

So this is aortic knuckle which is more or less OK and then this OK actually this is aorta um which is slightly […] aortic knuckle […] and this is the left atrium actually it’s a straight line this left atrium also runs to this side so you imagine the whole left atrium like this …. This is the double atrium this is the left ventricle the right ventricle is probably this here a bit tilted up like in […]ology double straight (heart) ….OK …[returns to seat] are you all sure what I’m talking about? And [pointing to film] and there is some suggestion at that point […] it’s a good direction mainly because as I’ve said it’s the Trudy […] there’s really hardly any congestion that you can see….have I made myself ABsolutely clear? [students remove film and sit down] then you would like to do the ECG actually I’m trying to find why can’t I find the ECG in this version

369 Jan

I think the reason is it should be […]

370 Keith

Should be the paper fold ….is it…..

371 Tutor

here it is on the twenty seventh uh it was actually done yesterday…. What about what about the one on admission….

372 Fay

that’s because admitted on the twenty seventh

373 Tutor

Is he so he’s discharged within one day….uh uh ummm….OK [students lean over table to study ECG]….what does the ECG show? …. 

374 Jan

left [elasticity?] 

375 Tutor

Yeh why is it left elasticity? You are correct yeh

376 Jan

This one is positive and this one is negative

377 Tutor

Which actually which one is negative will give rise to you are correct but which one being negative would give rise to left axis…[nature??]

378 Students

B2

379 Tutor

B2 is what 

380 Students

sixty degrees

381 Tutor

fifty sixty therefore if it’s uh appreciation therefore if it’s equal then it means what […]appreciation?]

382 Jan

Negative thirty or one hundred twenty
Yeh it’ll be perpendicular to [...] lead to being sixty degrees uh if it’s negative it’s already LVH...[...dilation??] left axis deviation OK this have a look at this side and this one this will lead to which [...] if it’s epiphasic then it’s already minus thirty if it’s negative then it’s more minus than minus thirty...thing is this he she he has got quite marked left axis deviation ....anything else?

[....] wave [...]

right so he’s what

he’s in atrial fibrillation

Yeh he’s got atrial fibrillation ....left axis deviation anything else ....

left ventricular hypertrophy

Yes quite marked left vent- on it’s own already it’s what what how many

Si...five and a half

Ok and this one a little bit. OK .....so he’s got left eh left ventricular hypertrophy with what anything else

lateral deviation of eh the heart ....mm as shown by [pointing with pen to ECG] Actually it’s equi – equivocal and three three three four

Yeh OK ...[...] anything else .... there is actually some minor but not very marked TVA inversion in [E]6....which may be due to but not really very marked sorry not [EET ] SET inversion of just about one millimeter so probably left ventricular hypertrophy may be a bit of strain Ok ....left axis deviation umm atrial fibrillation left ventricular [reversion??]
Tutorial 8

Medicine Specialty PBL Session

5th Year Students: Keith, Ron, Trudy, Jan (chair), Sue and Fay

1 Tutor so we are ready to (. go
2 Ron (yes)
3 Tutor shall we
4 Ron yes
5 Tutor OK
6 Tutor so uh hum (. who would like to present (. the first
7 Jan shall we follow follow the order
8 Students (yeah follow the order)
9 Jan the first learning objective is (. uh haemolytic anaemia (0.5) so um as we
know that haemolytic anaemia is :: due to destruction of red blood cells (.)
they’re inside the uh blood vessels or outside so that’s intravascular or
extravascular (. um (. so um patient presenting with haemolytic anaemia
will have usual symptoms of anaemia like fatigue :: shortness of breath uh
uh more than that he or she may have signs of jaundice due to the
haemolysis and there may be darkening of urine colour like coca cola (.).
cough) um there may be splenomegaly in some cases in like in uh
lymphoid proliferative cases there will be enlargement of the spleen and
also um haemolytic anaemia may predispose to pigment gallstones (0.4) so
for the diagnosis of haemolytic anaemia other than the normal
investigations for anaemia like uh complete blood count (. reticulocyte
count (. blood film (. um other (^^^) parts (. there’s there are some
specific items for haemolytic anaemia like increase in um unconjugated
bilirubin, decrease in free haptoglobin, increase in urobinogen in the
urine and increased LDH in the blood (0.5) so you may have heard about
the Coombs test it is for uh auto uh it is for immune-mediated haemolytic
anaemia (. so the Coombs test is for uh testing the antibody acting uh to
the red blood cell (. so uh (0.3) if it is positive this indicates the:: anaemia
is due to immune haemolysis (0.6) so for the (. different causes of

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haemolytic anaemia

10 Tutor jus just before you go on (. ) are there any different kinds of Coombs test?

11 Jan um it can be direct or indirect (. ) so direct is antibody: to: red blood cell (. )

12 Fay (it's on the red blood cell)

13 Jan red (. ) antibody on the red blood cell (. ) and indirect is antibody in the serum (0.4)

14 Tutor {nodding} you you mentioned that uh:: (. ) you said these patients could have dark urine is that correct? so do you expect the patients to report dark urine?

15 Jan sorry I

16 Tutor you you you talked about darkening of the urine)

17 Jan (yeah)

18 Tutor (. ) uh is is that something that you expect with in a patient with (. ) most causes of haemolytic anaemia?

19 Sue {ac} If it is intravascular haemolysis then it would uh present with blood with tea colour urine dark colour urine due to increased urobilinogen in the urine

20 Tutor mm (0.5) I I think it is not usually tea-coloured (. ) I mean you you can detect urobilinogen in the urine (0.2) but huh really dark tea-coloured urine is usually the sign of conjugated hyperbilirubinaemia (0.2) so: (. ) although there may be uh: some (. ) uncommon causes of (. ) haemolytic anaemia with for example hemoglobin urine (. ) I’m not aware that (. ) very dark urine is is (. ) is is common in haemolytic anaemia (. ) uh: just thought I’d mention that (. ) okay so you are going to tell us some more

21 Jan mm so for the different causes of haemolytic anaemia it can be divided into uh: hereditary uh acquired (. ) so for the hereditary causes like including the red cell membrane problem, for example the hereditary spherocytosis um there may be some uh: enzyme problem like the G6PD deficiency or there can be problem of the hemoglobin like in thalassaemia (. ) then for the acquired cause, it can be divided into immune and non-immune (. ) and immune can be further classified into auto- or allo-immune (. ) like in uh autoimmune it can be some causes like idiopathic or SLA and
for allo-immune it can be some uh transfusion uh problem or the
haemolytic disease of newborn (. ) um it can be drug-induced too (. ) um:
{hi} actually drug-induced can cause immune and auto uh immune and
non-immune haemolytic anaemia so for the non-immune thing it’s like the
drug directly attack the red blood cell uh but for the immune cause it is the
drug that um act against the antibody red cell (0.2) antibody of red cell
(0.3) uh hum

22 Tutor do you (. ) you mentioned thalassaemia (. ) do you d’you classify
thalassaemia as uh haemolytic anaemia?

23 Jan but it will lead to haemolysis in some

24 Tutor is that is that the cause is that the main cause of the anaemia in
thalassemia?

25 Jan oh the main cause is the abnormal red blood cell but um (. ) well {ac} there
will be haemolysis but not the main main reason to cause anaemia

26 Tutor {lo} yes of course (. ) and so in effect erythopoiesis (. ) so that causes it (. )
ookay (. ) good anything else?

27 Jan mm: any questions? (0.4)

28 Tutor any questions?

29 Sue how do you classify the causes into intravascular haemolysis and
extravascular haemolysis (. ) because I always have a difficulty on like how
do you differentiate which causes actually would uh lead to the huh red
blood cell rupture intravascularly?

30 Jan mm I found something like if (. ) if the:: sorry (0.2) like in the blood there
is antibody acting against the red cell and if the cascade you know the com
plement activating uh pathway and at the end it will form membrane
attack complex MAC (. ) so if the pathway uh the pathway is very well
activated then it will cause direct direct damage to red cells in the blood (. )
so it will cause intravascular (. ) but if the complement response is not
sufficient to form the membrane attack complex (. ) uh the: complement uh
will cause opsonisation on the red cell and lead to the red cell destruction
in the spleen or extravascularly (. ) so I think it it is determined by whether
the complement pathway is very well activated (0.2)

31 Tutor yes I think most extravascular haemolysis occurs in the spleen (^^^)
Tutor: good that’s a very good presentation it’s quite a big subject isn’t it very complicated thank you right uh: next uh: paraprotein

Jan: ) mm (0.2)

Trudy: now paraprotein

Sue: ah paraprotein okay uh um um uh so it’s to know about paraproteinemia we first have to know about what is paraprotein paraprotein is actually some immunoglobulin from single clone of plasma cells so we sometimes call it uh monoclonal uh: immunoglobulin mono monoclonal immunoglobulin or um in the electrophoresis it’s marked as a M uh it’s marked with the letter M so it’s called M component um for paraproteinemia the most uh well-known cause would be multiple myeloma but in fact there are other reasons leading to paraproteinemia um last time we also mentioned the mono MGUS monoclonal um gammopathy of unknown significance um and there are other types of um monoclonal gammopathy like uh amyloidosis or uh: macro macroglobinemia so um I think because actually uh under each type of uh paraproteinemia they have different presentations so I’ll briefly first talk about um multiple myeloma and also the more common type of paraprotein uh monoclonal gammopathy that is the MGUS um for the multiple myeloma um just uh brief recap because we have been talking in systemic lectures um it is a malignant plasmacytoma leading to monoclonal gammopathy um that is there is an overgrowth of plasma cells um usually at the bone marrow we would found we would we would find more than 30 percent of plasma cells which is one of the diagnostic criteria for multiple myeloma and uh also uh we would find plasmacytoma at the bone marrow and uh one of the diagnostic uh investigations would be uh to do uh serum protein uh electrophoresis um for serum protein elec electrophoresis we would be able to found a increase in the amount of um monoclonal immunoglobulin um for 55 percents of the cases in multiple myeloma it would be increase in IgG other cases would be increase in uh IgA and then um other uh being the light chain um if we do uh urine
electrophoresis we would find uh increase uh in light chain um which is what we call Bence-Jones protein (. um (. um why do the light chain excrete in the urine is because uh it is its size is very small so we would uh usually would not find heavy chain in the urine but um rather it would be the light chain (. uh :: for the presentations of multiple myeloma uh mainly present um in three ways it affects the bone affects the renal function and um also affects the bone marrow (. for the bone it would lead to uh lytic lesions and the most well-known would be the uh lytic lesions at the skull that is the punched out lesions (. um :: other it would affect other parts of the bone mainly the truncal region um including the ribs or the uh pelvis uh leading to (. uh pathological fractures (. and also because of the bone (. uh boney lesions it would uh increase in hypercalcaemia (. and for the hypercalcaemia it is also one of the reasons leading to the renal function (. det deterioration (. um :: apart from bone and renal impairment it would also affect the bone marrow because of the increase in uh: monoclonal immuno uh because of the presence of plasmacytoma and increase in monoclonal gammopathy (. uh it would suppress other :: uh immunoglobulin leading to (. uh: easy infections like uh it suppress other (. um immune :: immune uh other defence systems so it would in uh increase the infection chance of the patients (. and also um it would lead to anaemia and thrombocytopenia

38 Tutor how how does that suppression go
39 Sue uh because there is increase in the single cell type like uh increase in plasma cells and so it suppress the other cell lines (. so leading to anaemia and thrombocytopenia (.)

40 Tutor (mm)
41 Sue so um for uh most patients with (. mon uh multiple myeloma they usually died of other infections or renal function deterioration (. uh :: there are als also other presentat uh presentations like uh it would lead to uh hyper hyperviscosity because of the: increase in monoclonal uh monoclonal immunoglobulin (. um hyperviscosity would lead to uh some uh retinopathy that uh: the patient would feel a slow (. loss of vision and also there would be retinal hemorrhage and oth (. there are also other
presentations like neuropathy (.). so um ::

42 Tutor  is hyperviscosity very common in multiple myeloma
43 Sue  um:: com compared with other monoclonogamopathy it’s it’s more
common to give hyperviscosity but among all the patients like only 10
percent will give {dc} hyperviscosity

44 Tutor  I think it would be less common than that (.).
45 Sue  OK
46 Tutor  not not common at all in multiple myeloma but
47 Sue  ){whispering} OK
48 Tutor  ) but there is there is another paraproteinemia which: it is common in (.)
which is what (.). we talked about it the other day

49 Ron  (^^^)
50 Tutor  yes (.). wh why is it more common in )
51 Ron  )because the IgM is a much larger molecule
52 Tutor  that’s right (0.4)
53 Sue  so that’s all for multiple myeloma (.). uh I ‘ll also
54 Tutor  jus just before you leave myeloma does the paraproteinemia itself (.)
mediate any of those :: symptoms or signs
55 Sue  uh:: the paraprotein would um deposit at the renal and leading to renal uh
renal function deterioration

56 Tutor  can it have any other effects
57 Sue  um ::]
58 Tutor  ] the paraprotein is of course an antibody (.)
59 Sue  mm mm
60 Tutor  so can it have (.). im immune-mediated effects
61 Ron  (^^^) amyloidosis
62 Tutor  ({dc} uh yes (.). I’m not sure if this (has to do with it) directly but (the
antibodies) (.). maybe (0.4)
63 Sue  um (.). I’m not sure whether it cause: haemolytic anaemia
64 Tutor  I think some of them can cause neuropathies (.). and uh:: haemolysis (^^^)
but the different paraproteins like IgD IgA have different patterns of
complications that (.). are occasionally associated with it (.)

65 Sue  mm mm
okay so go on and tell us more

um so an another common paraproteinemia would be monoclonal
gammopathy of uncertain significance um:: it actually is quite common uh
in the elderly that there is uh five percent in pat uh in persons over seventy
years old uh we would have uh this kind of monoclonal gammopathy (.)

um why we say that it is uncertain significance because it’s quite different
from the presentation of multiple myeloma that it would usually would not
give the it would not give the renal or bony uh :: (. it would not give the
bony lytic lesions and also the renal impairment as in multiple myeloma (.)
and also in the bone marrow uh if we observe at the bone marrow the
plasma cell is usually less than ten percent um but there is a chance that
the monoclonal gammopathy of uncertain significance will progress to
other types of monoclonal gammopathy like multiple myeloma or
mucroglobulinemia (. so uh but uh concerning the signs or symptoms it’s
usually asymptomatic (. yeah (0.2)

yes uh it can also reflect underlying lymphoid proliferative disorder uh
some B cell neoplasm

uh hum)

) like uh chronic lymphocytic leukaemia for example

yeh there is a chance that it will progress to lymphocytic leukaemia (.)

okay good (. anything else (0.2) any questions (0.8) okay: um so the next
problem that we were going to hear about was problems that can be
addressed using examination of the blood fluid {Trudy gets up and moves
along the table to the computer and shows slides} (0.10)

uh I’m going to talk about first the peripheral uh peripheral blood smear (.)
and how it can help us to diagnose (pathological) diseases (0.2) and in the
peripheral blood smear it’s usually bright-stained and uh being brought
under a hundred times power uh microscope and uh the things we are
looking for include uh uh platelets red cells white cells and also abnormal
cells (. so this is a normal blood smear uh {referring to image on screen}
under the uh the bright stain and red cells and we can see platelets and
lymphocytes (. and for the plat

neutrophils
and uh sorry (.) and so for the platelets … like uh how do we know how many platelets are there and usually or normally is that one to three platelets per twenty red cells (.) and uh the diameter is uh for platelet is usually one to two micrometers and if the platelets are large then uh it’s a problem indicating rapid platelet turnover for example myeloproliferative disorders (.) and also another problem is that if the platelets are clumped together it may give uh falsely low automated platelet counts so when we um take the blood again we need to put the blood in a (citrate pot) another cause of elevated uh falsely elevated platelet counts is neutrophil permutation (.) and we can see giant platelets in this slide (.) and after the platelets we look at the red cells and there are few things that we look for (.) um like the size of the red cells the shape and uh also the hemoglobin content (.) any red cell inclusions the age of the red cells or the distribution (.) and so about the size of the red cells um it’s usually eight micrometres in diameter and you can come and the blood film you can compare it with the nucleus of the smallest (neutrophils) (.) and uh for the size the problems include uh microcytosis (.) uh low MCV for example in uh iron-deficiency anaemia (.) and uh microcytosis or MCV for example (^^^) and also um if there’s a wide variation in size we call it anisocytosis (.) um examples include concomitant iron (^^^) deficiency uh (0.5) and this is a blood smear showing uh iron-deficiency anaemia it’s uh microcytic means small in size (^^^) um meaning that there is a low hemoglobin (.) and this is a (^^^) macrocytosis (0.2) and besides the uh size then we look at the shape of the red cells and it can tell us about like specific diseases and we call that (^^^) and there’s great variation (.) and different types of um like uh different shapes for example for a cancer site like reticulated red blood cells then it might point to renal disease or like patients are (^^^) and um for uh elliptocytes it’s like hereditary diseases or other uh blood diseases like iron-deficiency uh myelodysplastic uh disease and microblastic anaemia (.) and for schisto schistocytes it’s that usually due to hereditary haemolytic anaemia (.5)um sickle cells I’m sure you all
know about that (. ) the spheroocytes is are um mainly they are very spherical and the cells do not have the normal like the normal red cells they have a central um but the spheroocytes don’t have any (. ) usually due to hereditary spherocytosis (. ) G6PD-deficiency (. ) or uh autoimmun e uh haemolytic anaemia and also uh white target cells due to thalasssaemia and liver disease (. ) the tear drop cells in myelofibrosis and rouleaux formation meaning the red cells are stacked up in like (^^^) onset usually due to multiple myeloma and uh this slide shows the reticulated (^^^) associated with (^^^) patients (. ) this is (^^^) either hereditary or due to (^^^) and sickle cells (. ) and (atherocytosis) in hereditary uh spherocytosis or G6PD-deficiency (. ) and uh target cells (. ) so you can see that there is a ring of color in the centre so-called the bull eye uh bull eyes and this is the uh tear drop cells in the myelofibrosis (. ) and then after the after looking at the shape we will look at the hemoglobin content of the red cell (. ) and uh (. ) uh um we usually if it is hypochromic that means uh there is a iron-deficiency anaemia or (^^^) and and besides the hemoglobin content we should also look at any uh red cells like uh inclusions like inclusion of abnormal things inside the red cells (. ) for example the uh (^^^) bodies uh in like there’s some blue residual nuclear fragments that stains blue (^^^) patients um basal fluid stripping uh due to lead poisoning (. ) and uh (. ) Heinz bodies uh due to G6PD-deficiency and in malaria you can see some parasites being included (. ) (^^^) and this one is the (^^^) in the platelets after splenectomy you can see the residual nuclear fragments uh inside the residue (0.3) and then after looking at the inclusions we also (. ) also look at the distribution of the red cells of the blood smear (. ) if the red cells are glutinated together this may suggest (^^^) anaemia or auto uh immune uh haemolytic anaemia (. ) and also uh rouleaux formation for uh (. ) for example in the uh multiple myeloma (. ) and we also look at the age of the red cells uh you (. ) uh if the film shows some mixture of uh like they stain differently of different ages that they suggest uh there might be uh some like hemorrhage or anaemia so the bone marrow need to uh like produce a lot of uh young red blood cells to replace the destroyed blood cells (0.3) and this film shows the red cells like uh glutinated together (. ) and the
rouleaux formation like a like a stack of stones (.) and after red cells we look at the white cells, (^^^) for uh neutrophils um (0.2) these are usually associated with uh bands so we call that left shift which means that there is outpouring of immature neutrophils from the bone marrow (.) for example due to (infection) there is bands (.) and um (.) while associated with uh also if there is toxic (granulation) which which mean that there is some bacterial infection (.) with regard to haematological diseases if we see like hypersegmentation of the white cells suggestive of (myelodysplastic) anaemia but we also need to (^^^) marrow too uh uh and finally (hyperblastic) anaemia and if and if it’s hypersegmented (with white cells) it may suggest leukaemia (.) (0.3) and this uh: this film shows uh hypersegmented uh neutrophil (0.4) and also there might be uh Auer rods uh which they include some (^^^) like cytoplasmic cells uh cytoplasmic content inside the white cell and is suggestive of um AML as shown in this film uh slide (0.3) after looking at the neutrophils we look at the basophils and uh basophil count is increased in uh chronic myeloid leukaemia (.) this is uh (^^^) (0.2) and uh for white cells lymphocytes normally they are small with a dark nucleus (.) if you see that um: for example in CLL (as more) lymphocytes will increase in number and many of them will rupture and resulted in a (^^^) we call that (^^^) and also in (hairy cells) uh hairy cells and in CLL you see like (^^^) (0.2) and this one shows acute T-cell leukaemia with uh typical flower-shaped nucleus (.) and we also look for blast cells to diagnose uh uh hematological malignancy uh blast cells are abnormal immature nucleated precursor white cells that being pushed they are immature but they are being pushed by the (bone marrow) into the peripheral blood and it’s usually indicative of acute leukaemia or (myelocytosis)(0.6)

Tutor that was a very excellent uh” presentation uh thank you it’s a very visual subject isn’t it?

Trudy mm

Tutor can you uh you mentioned the rouleaux what’s the uh significance of rouleaux? (0.2)

Trudy like (it’s) a stack of uh coins being piled up and can be due to some uh like
some (^^^) or proteinemia like multiple myeloma or (^^^)

82 Tutor yes so if you see the comment of rouleaux on the blood film you
sometimes order a protein electrophoresis just to see if there is a
paraprotein (.) typically it’s associated with a very high ESR often over a
hundred which is due to a combination of the rouleaux and the paraprotein
(.) uh how how would you distinguish the blood the blood film of uh iron
deficiency and thalassaemia (. ) both are monochromic and microcytic
(0.15) any any idea

83 Sue there would be target cells in thalassaemia

84 Tutor yes

85 Sue and also under a certain s: stain (. ) if it is:: like there would be inclusion
(^^^) of golf ball appearance (0.2)

86 Tutor I’m not sure about) that

87 Trudy ) or elliptocytes (^^^)

88 Tutor yes and a lot of poikilocytosis (. ) (and) target cells so the film looks quite
different (. ) but if you’re unsure what what tests what tests can you do?

89 Ron ) iron profile

90 Keith ) hemoglobin

91 Tutor mm?

92 Keith hemoglobin

93 Ron and iron profile

94 Tutor iron profile

95 Keith and hemoglobin

96 Tutor check the hemoglobin the level?

97 Keith (^^^) specific

98 Tutor what’s the test )called?

99 Jan ) hemoglobin) pattern

100 Keith )pattern

101 Tutor uh?

102 Jan hemoglobin pattern

103 Tutor yes yes the hemoglobin like electrophoresis (0.2) and uh you you you
mentioned uh macrocytosis being due to folate and B twelve deficiency,
does anything else cause macrocytosis?
Tutor: is there any difference in the blood film between the macrocytosis of alcoholism and nutrition? (of course) some of these alcoholics may also be folate deficient.

Sue: mm (0.5)

Jan: because less segmented cells less (^^^^) cell less (hybrid) segmented cells

Tutor: d’you mean the white cells )the neutrophils?

Jan: yeh

Tutor: maybe (0.2) the the uh: the uh macrocytes in nutritional deficiency are typically oval in shape but in alcoholism they’re typically round uh macrocytes (0.4) are there any other questions about the blood film? (0.8) if not thank you very much for that and uh we’ll move on to the next problem which is to do with the patient presenting with uh anaemia (0.3)

Ron: (Trudy moves from the computer to her original chair) mm so when we are faced with a patient with anaemia either it’s by: (.) it’s by the symptom-wise or by the investigation will be found to be anaemic, then history wise we first have to ask the patient uh the symptoms of anaemia, for example, fatigue, (^^^^), a general malaise, or poor concentration or sometimes shortness of breath on exertion if there is severe anaemia, or any palpitation and we have to assess the severity of the anaemia (.). we may get a suggestion of the severity of the anaemia based on a past history of transfusion, or uh the progression of the anaemia of this kind uh based on the progression of the symptoms uh for example the dizziness or even loss of consciousness (.). and further, further on we have to delineate the e the uh causes of the anaemia, (0.2) um the most common cause of anaemia (^^^^) is of course bleeding and so we have to ask about uh the history of uh all the bleeding sites, uh for example in the GI tract, and the lungs, and form of hemoptysis, and the urinary tract and form of hematuria, amenorrhagia, and we have to ask um: symptoms and signs of haemolysis, for example dark urine, jaundice, and we and the family history we have to ask for the uh G6PD and thalassaemia, and we have to ask about the chronic diseases for example the rheumatoid arthritis or chronic infection.

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which may give rise to a slight anaemia um which is called the anaemia of chronic diseases, and we have to ask we have to screen a little bit about the other diseases the other auto-immune diseases for example SLE, and we have to ask uh in the history wise symptoms of other lineage um uh: low cell count, for example frequent infection for low white cell count, bleeding tendency for the low platelet count, and uh then drug history wise we have to ask for any herbal medication um because some herbal medicine may um lead to haemolysis, and we: mm in some occasion when there is some suggestion of malaria we have to ask about their travel history: of the patient and uh: for the diet history because of poor uh nutrition can uh lead to anaemia but this is very rare in Hong Kong, and we have to ask about the alcohol intake because sometimes alcohol is related to folate deficiency (. ) and uh also uh physical exam wise we have to assess whether there is any pallor in the patient, whether there is jaundice, and um: we have to palpate whether there is any lymph node and any ankle oedema, in relation to renal or liver disease, an: d we have to palpate for any organomegaly, actually the splenomegaly, and we have to look for any skin changes for example the bruises, the bleeding or any abnormal pigmentation (0.2) for the investigation wise, we: are most interested in the severity of the anaemia so we check the complete blood count, uh the complete blood count can also give us more information of the uh the affected lineages for example the white cell count, platelet count, and it also gives us um the MCV and MCH which further give us some clue to the cause of the anaemia ( . ) and we also have to look at the reticulocyte count, an: d because in anaemic patient if the marrow is uh: if the marrow is intact the reticulocyte count will be pushed out, and in case of marrow failure, even if even if there is anaemia, the reticulocyte will be low (. ) and a blood smear as suggested by Trudy will give us some valuable information, on the causes of the anaemia ( . ) and we have to look at the iron profile of the patient uh if there is uh if we suspect any iron deficiency anaemia an: d we can do the Schilling’s test if we suspect B twelve deficiency and we have we can do the liver function test for testing for the unconjugated bilirubin to see if there is any haemolysis, evidence
of haemolysis (. ) and we might (. ) do the ultrasound of the abdomen to see if there is any enlarged spleen, um when we cannot palpate for any splenomegaly (. ) and we suspect any underlying pathology which lead to splenomegaly and therefore hypersplenism, and increase in sequestration of uh: the platelets and other lineages, and we can do: the fecal occult blood test for detection of any bleeding (or for) bleeding in the GI tract, and we can do the urinalysis to detect any red blood cells in urine, and also the microscopy to detect any microscopic haematuria, and the renal function test will be very valuable to det if we suspect any chronic renal failure leading to uh anaemia (. ) and we can do the direct Coombs test for the haemolytic anaemia (. ) and finally we can resort to bone marrow aspiration or a (^^^) biopsy if we suspect any hemic malignancy or failure of the marrow (0.2) that’s about all of the approach when we are faced with anaemic patient (0.2)

113  Tutor  what is the Schillings test?
114  Ron  the Schillings test is the uh: used to detect any B twelve deficiency (. ) in the patient (  
115  Tutor  I think you can detect B twelve deficiency just by doing a B twelve level so I think a Schillings test must be different is it?
116  Sue  it’s for differentiating whether )
117  Ron  )(^^^)
118  Sue  yeh whether the cause is uh from the uh gastric or from the intestinal
119  Tutor  so how how is the test done?
120  Sue  um I’m not sure but it’s divided into part one and part two and then if both are low it is reduced in absorption
121  Fay  with increasing )(^^^)
122  Jan  ) radioactivity twelve is being ingested and uh see if the level in urine is (in)creased if it’s (in)creased that means it’s not absorbed (. ) so uh and then you give intrinsic factor to the patient, if the urine level of the B twelve increased that means the problem is uh is uh from the stomach
123  Tutor  {nodding} (0.2) yes that’s right (. ) what is intrinsic factor?
124  Jan  intrinsic factor is produced in the stomach for the absorption of B twelve
125  Tutor  {nodding} good (. ) and if uh if your tests show iron deficiency anaemia,
Ron: how would you proceed with your approach to the patient?

Ron: if we find an iron deficiency anaemia we uh this may indicate uh upward bleeding from um either the gastro-intestinal tract or uh the urinary tract then we have to check for that blood test or urinalysis to check for any bleeding sites and at the same time we can we may give iron supplement for the patient.

Tutor: so if fecal occult blood is positive, what would you do then?

Ron: if the fecal occult blood test is positive then that indicates some bleeding in the um GI tract we may do uh OGD or colonoscopy to detect anything inside (.) to visualise the gut (.)

Tutor: and what if those tests are both normal? (0.2)

Jan: there may be bleeding in the small intestine.

Ron: which is less common but not detectable by OGD or colonoscopy.

Tutor: {nodding} what sort of diseases could affect the small intestine?

Fay: the AV malformation, it can cause or uh AV displacement can causes bleeding in the small intestine as especially in the elderly (. people.

Tutor: AV dysplasia?

Fay: mm

Tutor: wh where’s the commonest uh: commonest location for uh vascular malformation causing iron deficiency in the (^^^)

Ron: in the ascending colon)

Tutor: ) the ascending colon yes (. how is that diagnosis made?

Ron: colonoscopy )

Jan: colonoscopy

Tutor: it’s made by colonoscopy (0.4) though it’s usually done (parietally the endoscopy) so it’s usually done by angiography (0.3) um: and you can sometimes do a (. radiolabeled (red) cell assay to visualise the bleeding site if it’s not apparent on endoscopy (. good OK that was very good (thank you (. so: any questions about approach to patients with anaemia, (0.3) if not we’ll move on to the: liver function tests (0.3)

Keith: OK for the last of the liver function tests first uh uh the common liver function tests done uh parenchymal enzymes ALT AST for enzymes ALP
GGT protein total protein and albumin globulin levels and total conjugated and unconjugated bilirubin (and) performed at the time (.) so for the parenchymal enzymes ALT and AST: they stand for alanine transaminase and (aspartate) transaminase AL uh these are both present in the parenchymal cells and they rise dramatically in acute liver damage (.) for example, viral hepatitis or overdose in paracetamol (.) for the ALT it can be raised up to (uh) thousands in acute hepatitis like A B Keith E and acute (^^^) and it will only be raised to the level of hundreds in say chronic hepatitis or drug-induced hepatitis (.) and: in a very severe form of hepatitis, it’s sometimes seen that ALT and AST will be normal or low (.) because there are less cells available to release the enzyme (^^^) uh AS uh ALT is more specific to the liver whereas AST is not as specific (.) so we can also expect to see it in red blood cells, and in the myocardium, and skeletal muscle (0.2) so: for example it may also be raised in acute myocardial infarction (.) so for the (ductal) enzymes, these are the ALP and GGT, so the alkaline phosphatase and (. ) gamma glutamyl transpeptidase {laughs} and these are present in the (^^^) biliary canaliculae and they are raised in ductal pathology (.) uh ALP is: not very specific cos it’s also present in bone and skeletal tissue so for example, it will be physiologically raised in say: puberty or pregnancy (.) though it will also be raised in elderly with like Paget’s disease (.) mm:: for GGT it will be: raised in alcohol toxicity, and drug toxicity (.) and both of these may be raised in a parenchymal disease which would (.) which acts as a space-occupying lesion compressing the ducts (0.1) and for: albumin, albumin is for the synthetic function of the liver (.) it has a turnover time of twenty-five days, (0.2) {coughs} and is often one of the first ones to decrease in cirrhosis so you would expect it to be decreased in cirrhosis also malnutrition, albumin and globulin we often use the A/G albumin globulin ratio (.) and in the A/G ratio: it’s: uh: normal for the albumin to be greater than the globulin (.) and: next we go on to the total bilirubin (.) so bilirubin is produced in the breakdown of haem (.) and the increased bilirubin causes the jaundice so: we can have a pre-hepatic, hepatic, or post-hepatic (0.2) and uh if there’s increased unconjugated bilirubin (.)
then we can expect a pre-hepatic or hepatic (.) problem, and if conjugated bilirubin is higher then we can expect a post-hepatic problem (0.2) {coughs} so for example bile duct obstruction (.) and: well fina)yly

) what about in uh: hereditary spherocytosis (.) what do you see there?

well that would be a pre-hepatic

pre-hepatic OK

so we would have an excess of unconjugated bilirubin (.)

and finally we have the prothrombin time and this is measured by: (0.2) sorry this is: a product of the factors 2 5 7 and 10, which are have decreased production in liver disease (.) and so: it would you would be expect it to be increased in liver disease (.) but we also must exclude vitamin K deficiency (.) which may be present if the patient’s on warfarin, or the patient has mal-absorption, or like in (^^^ baby) there is lack of the gastro-intestinal tract colonisation by bacteria, and maybe: uh and prothrombin may also be prothrombin time may also be increased for example in: disseminated in to {puts head on table} {Cantonese what is it?)

disseminated intravascular ) coagulation

coagulation {smiling} and may also be increased uh: in if the patient has lupus anticoagulant (.) and further to these tests we can also do some more tests, for example the (projected) glucose level, which may fall in a very severe form of liver failure, we can check the LDH the lactate dehydrogenase, but also this is quite non-specific cos it acts as a cardiac enzyme and: in: it’s also raised in haemolysis, and in states of (hyper) tissue turnover for example lymphoma (.) and we’re going to check the iron status, because haemochromatosis can lead to liver failure, and we can check for Wilson’s disease by checking for cerulo ceruloplasmin (.) um we can check the auto-immune antibodies, for example (primary biliary cirrhosis ^^^) and: (.) we can check the amylase and mitase for example if there are gallstones (.) due to biliary obstruction, we can check the tumor markers for example hepat hepatocellular carcinoma with alpha-fetoprotein, or for looking for secondaries for choleangic carcinoma, by
checking Keith A and Keith A (nineteen) point nine (. ) we can also do some imaging techniques, so for example we can do a (CC) abdomen or ultrasound of the hepatobiliary tree, but we can also check for the detoxification function of the liver by doing an (^^^) (0.5)

152 Tutor good that’s that’s a very thorough (. ) review, uh uh you uh mentioned that the ALP and LDH could be elevated in other disorders, do you know do you know um do do you know any ways to (. )

153 Keith ) well we ) can
154 Tutor to uh distinguish the origin of uh
155 Keith we can check the isoenzymes (. ) tha that would be specific for the liver (. )
156 Tutor mm mm ) and how is that done?
157 Keith (0.2) not sure
158 Tutor anybody know (. ) for ALP it’s done by heat heat fractionation (. ) so the uh the uh the ALP isoenzyme comes from bones (^^^) so for liver it’s (more heat stable) (. ) for the LDH they have the isoenzymes one to five but I can’t remember which one goes with ) which
159 Keith ) LDH five I think is for the liver (0.2)
160 Tutor uh in a a patient who’s jaundiced (. ) what uh different patterns of liver (. ) function test would you expect for extra hepatic obstruction (0.2) versus say uh: viral hepatitis
161 Keith well for extra hepatic we’d expect to see the ductal enzymes increased, and we expect to have an increase in conjugated bilirubin (. ) whereas for hepatitis we’d expect to have the parenchymal enzymes, which is ALT and AST increased (0.2) uh: because there is hepatocellular hepatocellular damage due to the hepatitis B we’d expect to have an increase of unconjugated bilirubin
162 Tutor good (. ) any uh any other questions about (. ) the interpretation of the functions
163 Fay {whispering to Keith} (0.4)
164 Tutor good alright (. ) thank you very much for that, uh now we move on to the final problem which is that of the patient with uh portal venous hypertension and or: encephalopathy
I just want to show some slides for the: to talk about the topic (0.2) the first part of the presentation (^^^) venous drainage of the (^^^) system uh: first of all uh the blood from the proximal GI will be collected by superior mesenteric vein, and (^^^) from the distal uh gastro-intestinal tract will be collected through inferior mesenteric vein, (. ) and together with this uh the splenic vein they join the superior (mesenteric) vein, and the left gastric vein to form one portal vein (. ) which uh: umm which collect all the uh all the blood drainage from various organs in the in the abdomen and then pass through the: (. ) uh pass through the liver, and drain into the portal vein before it goes to the inferior vena cava (. ) {hi} so inside the liver, the portal vein (with) branches with the blood supply uh the blood flow from peripheral to central while the bile will be uh will be transported in the opposite direction (0.2) so this is the portal systemic uh: anastamosis (. ) uh in case of portal hypertension all this uh: venous drainage we talk about will be uh: blocked, so therefore (. ) there’s and uh the collaterals will open up and causes this portal systemic anastamosis (. ) the (first) is the esophageal area called the left gastric because this uh left gastric vein is block up so therefore it will cause opening up of the esophageal vein, and the consequences is there will be esophageal varices (. ) the second one is the rectal area (. ) since this uh superior rectal vein is blocked block up so therefore the blood will uh go to the inferior and middle rectal vein and goes into the internal iliac uh vein and then goes to the inferior vena cava (. ) the consequences will be (^^^) (. ) the third one is the (^^^) area this is the (^^^) paraumbilical vein inside the uh: falciform ligament uh due to due to the block up so therefore it will causes the uh blood to flow into the inferior epigastric vein and clinically you will see caput medusae (. ) the fourth one is retroperitoneal area uh this (portal) vein will be block up so that will be going into the retroperitoneal vein (. ) so the problems associated with portal hypertension will be esophageal varices, (^^^) caput medusae, and also the uh hepatic encephalopathy ascites which is can cause causes bacterial peritonitis which I’m going to talk about (0.9)
present uh usually patient will present uh: (starting) doing an OGD surveillance or: or uh acute bleeding uh the management of which will uh categorise it into primary prevention uh primary prevention, acute management and uh secondary prevention (. ) for esophageal varices uh: primary prevention we can give the patient uh: the patient has a high grade esophageal varices we can give the patient beta blocker or: prophylactic prophylactic ligation of all the varices (. ) uh and if the patient presents with uh acute uh acute bleeding then we have to first uh stabilise the patient, remember our (ABC) (. ) treat uh if the patient’s in shock then we have to uh put in (^_^) uh and then afterwards we have to do an OGD to locate the sources of bleeding, and also to uh to stop the bleeding if possible (. ) the ways to stop the bleeding can be sclera sclerotherapy or: uh ligation (. ) uh: usually ligation is more difficult in acute setting because uh: the blood will block the view so therefore the uh we can use uh injection sclerotherapy injection sclerotherapy in which we don’t need to uh precisely locate the uh location of the bleeding (. ) uh we can also give drugs to a patient like some vasodi uh constrictors, to decrease the mesenteric uh superior mesenteric and splenic arterial flow, but meanwhile we can also give the vasodilators but it’s not so to dilate the uh intrahepatic vascular pressure but it’s usually not so useful (. ) and uh (0.2) in case all this fails, uh in the acute setting we can use (^_^) which is a pressure (^_^) to stop the bleeding for for a while but it’s never more than twenty-four hours because it can cause necrosis of the esophageal wall, uh: prevention of bleeding we can uh also use beta blocker to uh increase the cardiac output so that uh decrease the portal blood flow, and we can uh also use uh after after the uh bleeding is controlled for a while then we can use long-term band ligation to uh: to ligate all all the varices uh uh spot but if all these fail we can the last resort is to consider transjugular intrahepatic portal sys portal systemic shunt in which the uh:: uh we canalise the portal vein, the right portal vein and also to canalise the uh the uh right jugular and internal jugular vein and then we connect them together through a shunt to relieve the pressure in the portal uh system (. ) so this is the management of esophageal varices the hemorrhoids basically depends
on the grade of the hemorrhoid if it’s a high grade hemorrhoid and also if that’s there’s thrombosis of the hemorrhoid then we can consider haemorroidectomy (.) otherwise it can be symptomatic symptomatic relieved by some ointments or (^^^) (.) {hi} caput medusae basically it doesn’t have any clinical consequences apart from the cosmetic disturbance to the patient, so then I’ll proceed to uh if there are any questions I’ll proceed to the ) uh
166 Tutor ) haemorrhoidectomy do do you think that’s a very popular operation?
167 Fay mm: not really because it’s I heard {smiling} that it was the most extr uh ex uh most painful operation uh
168 Tutor yes yes yes that’s exactly right ) and
169 Fay ) so now they have this banding technique but (.) in cases of acute thrombosis then you ) still have to go to haemorrhoidectomy
170 Tutor ) an uh and uh I’ve never seen (^^^) haemorrhoids as a result of portal hypertension ) I have to say
171 Fay ) mm
172 Tutor ) so even though it’s spoken about (.) it’s it’s part of the {lo} (^^^)
173 Fay mm
174 Tutor (lesser varices )
175 Fay ) mm mm (0.2) OK so I will proceed to hepatic encephalopathy (.) uh: usually uh the pa the patient will present with uh confusion, and then uh for the uh uh its main cause is due to cirrhosis which uh uh open up the portal systemic shunt so that the uh: so that the the blood of goes to the systemic circulation without first being detoxified in the liver (.) so it can be uh it can be precipitated by several causes including increased uh nitrogen uh product uh absorption like uh high protein diet, or GI bleeding causes uh increased absorption of protein in the gut, and constipation which the bacteria in feces degrade uh the bacteria in the feces degrade to form ammonia (0.2) it can be also uh caused caused by a decrease in (intravascular) volume so that there is less oxygen supply to the liver (.) like over diuresis in in and also excess excess para parentecesis (.) it can also be caused by drugs like sedative and electrolyte imbalance (.) uh for example like diuretic caused by diuretics (.) uh (^^^) artificial portal
systemic shunt {lo} which we have talked about before uh: can also cause this hepatic encephalopathy (.) so the main pathogenesis is increase in the arterial ammonia in the blood uh: because of the decreased ability of the liver to detoxify the ammonia the ammonia uh waste to urea and also there’s an increase in cerebral metabolic rate and perme permeability of this ammonia into the brain, and cause extra(^^^) damage (0.2) so:

{leafing through notes} for: for manage first first of all we have to grade the patient according to the clinical features, and uh possibly his uh EEG findings, so it’s uh graded into four four categories from uh: grade one to four, uh in grade four the patient will be in coma and the uh clinical features will be (decerebrate) and in grade one the patient will just have some inverted sleep patterns and forgetfulness uh uh and some on clinical pictures you can see some tremor and (atrexia) (.)

176  Tutor  wh wh what sort of tremor?
177  Fay  uh: flapping tremor
178  Tutor  what is a flapping tremor?
179  Fay  uh it’s due to uh: high (CO2) retention
180  Tutor  can you show us a flapping trem)or?
181  Fay  ) {extending right arm with palm upwards} uh you can ask the patient to uh pull in this position, but then the patient has a ten then but then then they cannot hold this position but they have a tendency to restore it to the original position so therefore it’s like ) flapping
182  Tutor  ) yes
183  Fay  (0.3) so to manage it we uh mainly uh will use uh lactulose which is uh a laxative, to causes uh the uh to remove the protein from the gut (^^^) situation they can also use uh we can also use uh lactulose apart from lactulose we can also use (^^^) which is a gut bacterias a gut bacteriacidal agent but uh it’s uh but uh its use is very: controversial (.) uh we uh on the diet we can ask the patient to stop taking the high protein diet, if the patient’s having constipation, we can relieve it by giving laxatives to the patient, if all this fail then we can uh we have to think of hepatic transplantation (.) if the patient’s uh condition allows (.) and if there’s (supply) of course (.) {hi} so the last one I will talk about is ascites is
caused by the increased portal hypertension causes transudation uh: uh causes increased transudation so that accumulation of fluid in the: in the: abdomen, (. ) uh it can be relieved by di it can be relieved by grade first of all it has to be graded into mild moderate and severe, for mild and moderate we can treat it with diuretics, typically we use (spiro ^^) if it uh fails then we top up with uh (frusamide) and for severe asci ascites then the first intervention is uh paracentesis, uh: uh tapping and uh so we have to do uh give IV abdomen infusion in order not to cause hyperalbuminuria because of the: intravascular shift, uh if it fails then we add on uh: diuretics, if all this fails then we have to think of TIPS again the trans uh transhep trans ) {smiling}

Keith 184  tubular)
Fay 185  ) jugular )
Keith 186  jugular intra-hepatic portal systemic shunt {smiling} uh so uh: {hi} if the patient present with ascites the abdominal symptoms we have to investigate for the SBP which we can do ( . ) by the uh:: diagnostic ta uh diagnostic thoracentesis in which we can find that there is an increase in neutrophil, typical picture is that uh more than five hundred neutrophils that is diagnostic of SBP, uh: a patient can present uh a patient may not have any symptoms from uh SBP uh but we have to always rule out with the patient with ascites and abdominal symptoms (. ) the treatment of which will be uh: will be to give a patient third generation (cyclosporine) norfloxacin, (0.2) yes norfloxacin yeh (. ) because they are not my presentation {smiling}

Tutor 187  wha what’s the name of the antibiotic?
Fay 188  nor norfloxacin )norfloxacin
Tutor 189  ) norfloxacin
Fay 190  it’s a third generation cyclosporine )
Tutor 191  that’s quinalone isn’t it it’s not it’s not cyclo)sporine
Fay 192  it’s not,
Tutor 193  no quinalone
Jan 194  (^^^)
Fay 195  quinalone
Jan does it start with (^^^)
Tutor {laughs}
Fay {smiling}
Tutor good thank you that was a very nice presentation (.) any questions about that (0.3) the only other thing you might mention in this context is the possibility of thrombosis can also complicate ascites
Fay (^^^) mm 
Tutor ) in these patients (0.2) good well I learnt a lot from all those presentations thank you very much (.) I’m sorry we’re running late umm shall we stop there
Fay yeh
Tutor good thank you very much
Sue thank you
Tutor see you again
Fay {students switch to Cantonese just before Tutor leaves room and appear to be discussing the presentations} (^^^) unconjugated hyperbilirubinaemia (^^^)