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

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VOLUME 2

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requirements for a Doctor of Philosophy

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Contents

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 ([redacted]), 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 ([redacted]), 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 [redacted] 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, u:h 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: 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 happ what how 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 (.)

40	Fay	{(hi)} 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:
41	Students	moyamoya
42	Ron	) moyamoya diagnosed to have moyamoya disease
43	Fay	(.) that
44	Jan	moyamoya disease m-o-y-a
45	Keith	) m-o-y-a
46	Fay	) yeh but I don't know what is this disease
47	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
48	Fay	how could that cause intracellular haemorrhage?
49	Ron	(.) um: I'm not particularly sure about this {(smiling & looking at Tutor)} (.)
50	Fay	because you said that it's the cause (.) cause like the collaterals is fragile vessels so (.)
51	Ron	{(smiling & looking at Tutor)} maybe
52	Fay	you're not quite sure
53	Keith	actually for the surfing was there any travel history involved with that?
54	Ron	travel history,
55	Keith	) or was it done in Hong Kong?
56	Ron	oh it was done in Hong Kong
57	Keith	) right by surfing do you mean like wakeboarding or surfing, cos wakeboarding is much more higher much high impact kind of sports?
58	Ron	he went (.) surfing / really surfing (.)
59	Trudy	did he have any headache in the past? (.)
60	Ron	(.) no it was new onset after the activity
61	Jan	{(lo)} (.) (.)
62	Ron	{(shaking head)} (.) (.)
63	Tutor	so how severe was the headache?

64	Ron	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 (.)
65	VS	so he does take any medicine /
66	Ron	he did took medicine from uh the Princess Elizabeth Hospital but I did not identify uh what kind of medicine was that /
67	VS	but before?
68	Keith	) was he on any long term medications?
69	Ron	no // our patient enjoyed good past health there was no long term medication (.)
70	Tutor	was it a type of (pulse thing) any more than headache?
71	Ron	uh there was no diurnal variation of the headache so no (.)
72	Tutor	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? (.)
73	Ron	yes
74	Tutor	) do you find any significant (.)functional impairment? {(lo)}
75	Ron	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
76	Tutor	)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
77	Trudy	could he walk?
78	Tutor	)can he walk yes very good {(lo)}

79	Ron	no he could not walk it was actually a right sided paralysis uh
80	Keith	) left sided
81	Ron	) left sided (.)
82	Tutor	so how about the upper limbs?
83	Ron	definite it was paralysis
84	Jan	he hav has to use the right upper limb to move his left upper limb
85	Keith	so the right side was five five
86	Ron	yes the right side was intact (.)
87	Keith	{nodding} how about the sensation?
88	Ron	there was a decreasing sensation on the left side mm {nodding} {Keith making gestures with arms to ask Ron to move on?} (.)
89	Fay	is there any bowel symptoms, urinary symptoms?
90	Tutor	) yes very good
91	Ron	uhhh (.) {looking at Jan} I did not ask about this (.)
92	Tutor	any incontinence, any accidents?)
93	Fay	) (.)
94	Keith	) did the patient have a diaper?
95	Ron	no the patient did not had {Keith nodding} (.)
96	Keith	how long has the patient been in hospital uh since:: you clerked him (.) before you clerked him?
97	Ron	he was in the hospital since thirty-first of May so it was: two days
98	Fay	(.) ah since you clerked him?
99	Ron	uh it was two or three days (.) in hospital {nodding} (.)
100	Tutor	I think everyone knows about the approach in: making a neurological diagnosis (.)
101	Students	mm mm {nodding}
102	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?

103	Trudy	) what type of sensory loss was that?
104	Tutor	what (kind of information) would you like to seek?
105	Ron	{looking at Trudy}) loss of fine touch sensation, (.)
106	Tutor/Sue	how about the pain?
107	Ron	(.) uh: (.) the pin prick sensation was reduced and the fine touch was reduced uh: so it was affecting both
108	VS	and how about the reflex?
109	Ron	uh reflex was:
110	Tutor	) can we can we concentrate on the history first? OK and then we can focus on the physical examination (.)
111	Keith	uhuh (when) the patient (complained of) the lower motor neurone facial palsy like it suddenly progressed to being complete or,
112	Jan	) no it just
113	Trudy	) it was a long time
114	Ron	) it was a few years ago that our patient had lower motor neurone facial palsy
115	Keith	) (.)
116	Ron	with a residual weakness but no recovery during these few years
117	Fay	was any diagnosis made in that time or was he told to be like (.) (.)
118	Ron	{looking at notes and shaking head}
119	Keith	but the patient said it's now complete?
120	Ron	(.) the patient is still in complete
121	Fay	) (.) facial palsy
122	Ron	(in complete) facial palsy now (.)
123	Tutor	so any speech problem, any,
124	Ron	) no dysarthria (.) no dys uh dysphasia (.)
125	Tutor	any swallowing problem:? (.)
126	Ron	I asked him whether he choked on food or drinks and he said he did not / (.)
127	Tutor	so the patient remained: conscious all along?
128	Ron	yes yes there was no episode of loss of consciousness no head injury:

129	Tutor	mm how about the vision:?
130	Ron	our patient complained a blurring of vision on the right side together with the onset of headache but the left sided vision was normal /
131	Tutor	mm mm no double vision?
132	Ron	no (.)
133	Tutor	) no so can we localise the lesion based on the history?
134	Sue	this patient presented with um (simile) weakness of: uh left haemiparalysis and haemiparesis and without any cranial nerve deficits (.) from the history (.) so we would think that the lesions would be above the brain stem /
135	Tutor	mm mm that's fair enough mm mm (.) can it could it be a spinal cord problem?
136	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 haemipares haemiparalysis (.)
137	Tutor	mm mm
138	Ron	and the sensory loss and the motor loss is on the same side (.) of the:
139	Tutor	) (.) yes that's right yes
140	Ron	)so it's suggested that the lesion should be uh above the uh brain stem
141	Tutor	mm mm (.) OK (.) so any other relevant findings?
142	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,
143	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
144	Ron	{nodding} mm
145	Tutor	OK
146	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
147	Jan	) (.) {lo}
148	Ron	on the right side (.) (.) (.) {lo}
149	Students	(.) orientation {lo}
150	Ron	{lo} conscious and alert, (.)
151	Jan	) orientation
152	Ron	he was: he was oriented to time, place and person,
153	Tutor	and that's all for the high cerebral function, high mental function? (.)
154	Sue	calculation and language
155	Ron	mm I did not perform the mini mental state examination (.)
156	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:,
157	Ron	attention
158	Tutor	)attention:,
159	Keith	) concentration
160	Fay	memory
161	Ron	)memory
162	Tutor	OK attention:, {writing}
163	Keith	concentration:, short-term memory
164	Tutor	memory, very good, what else? {writing}
165	Sue	language
166	Tutor	yes very good, language: (.) {writing}
167	Sue	calculation
168	Tutor	) mm mm
169	Keith	)calculation

170	Tutor	attention, calculation alright {writing} what else?
171	Trudy	planning
172	Tutor	yes very good planning {writing} executive functioning, what else?(.)
173	Trudy	{lo} (.) (.)
174	Tutor	mm mm (.) some abstract thinking, logical thinking mm mm and what else? (.)
175	Ron	visual spatial
176	Tutor	) yes very good the visual perceptual function OK (.) so do you know ho- how to assess these various domains? (.)
177	Ron	for the attention and calculation we can do the Serial Seven
178	Tutor	) yes very good mm mm
179	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 (.)
180	Tutor	mm mm
181	Ron	and ask him about the: recent news, and
182	Tutor	mm
183	Ron	) historical events
184	Tutor	OK (.) uh this will (tap) on the uh: the long term memory?
185	Ron	yes
186	Tutor	OK (.) a common thing is is: uh I think uh for this memory part you have to judge the 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?
187	Ron	I did not (.)
188	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,
189	Students	{nodding}
190	Tutor	) so your assessment may be incomplete OK?

191	Students	{nodding}
192	Trudy	can I ask a question when we say cortical sign what exactly
193	Tutor	) that tha 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?
194	Students	{nodding}
195	Tutor	and the perceptual function, (.) OK? (.) so you have missed all this high mental function assessment OK, so how about the cranial nerve? (.)
196	Ron	um except) the cranial nerve seven
197	Tutor	) so how about the speech? speech is the ver uh uh
198	Ron	uh there is no dysarthria no dysphasia
199	Tutor	mm so you don't have any difficulty in communicating with him
200	Ron	{nodding}
201	Tutor	OK (.) so apparently the language function is intact
202	Ron	mm
203	Tutor	yeh (.) can you go on to the cranial nerve (.) OK
204	Ron	um: except the cranial nerve seven on the right side other cranial nerves was intact uh: including
205	Tutor	) so do you think it's a new lesion or:, (.)
206	Ron	) um: our patient suggested that it was uh: a residual weakness a few years ago
207	Tutor	so how can you tell whether it's an old lesion or a new lesion? (.)
208	Ron	uh we can assess the: mus:cle: if there is any muscle atrophy:,
209	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 (.)
210	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?

229	Ron	no (.) actually the partial ptosis I had noticed from the case notes and I only noticed the right sided miosis yes (.) (nods) so um what are the uh uh how can we correlate the physical finding with the history in this patient?
230	Tutor	So first question is: get the answer you know the question where is the site of the lesion? (.)
231	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:: (.)
232	Tutor	mm mm
233	Jan	and because sensory is also involved so it's not in the internal capsules so I guess it's somewhere sub-cortical: (.) (shrugs)
234	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
235	Tutor	no (.)
236	Sue	but I'm not sure how does the Horner's related to the: sub-cortical lesion (.)
237	Keith	were there any marks of sweating on the face? did the patient notice any?
238	Ron	no (.) I notice: uh from the case notes that there is anhidrosis on the right side
239	Jan	uhhh (laughs) how can you feel anhidrosis in the hospital air-conditioning environment?
240	Ron	mm so I only observe it on the case notes (general amusement)
241	Keith	did you ask the patient afterwards?
242	Ron	(shakes head))
243	Sue	) but it's difficult to notice that
244	Jan	) you
245	Sue	you don't even sweat in the uh hospital
246	Keith	but the Horner's could have occurred previously?
247	Ron	yes it's possible (nods)

248	Jan	(laughs)
249	Keith	so it may not be the stroke that caused the Horner’s syndrome
250	Ron	(nodding) yes
251	Jan	) (laughing) ohh
252	Ron	(shrugs)
253	Keith	) (.) (.)
254	Tutor	mm so
255	Ron	) I also do not know how to correlate the Horner’s with uh the this clinical picture
256	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(.)
257	Ron	upper (.)
258	Tutor	) if the autonomic system is compromised
259	Students	) sympathetic ganglion
260	Tutor	mm mm (.) you know the sympathetic system supplying the the the: where does this locate do you know?
261	Trudy	t1 to L2
262	Tutor	mm mm (.)
263	Ron	it’s in the sympathetic trunk
264	Tutor	) yes
265	Ron	(.) column and in this case the Horner’s syndrome is suggested for cervical sympathetic trunk (.) affected
266	Tutor	mm mm (.)
267	Ron	and it can be due to compression, ischaemia, vasculitis,
268	Tutor	mm mm
269	Ron	different causes
270	Tutor	mm mm (.)
271	Ron	so actually in this case
272	Tutor	) so you don’t think the facial palsy is relevant for this case? (.)
273	Ron	mmm: I: don’t think so

274	Tutor	mm mm (.)
275	Keith	mm (.) multiple neurological lesions occurring,
276	Ron	nn?
277	Jan	nn?
278	Keith	multiple neurological lesions could it be (possibly) multiple sclerosis?
279	Jan	oh:
280	Ron	I guess multiple sclerosis would be uh progressive onset rather than: the patient come uh sudden onset complete paralysis
281	Keith	(.)
282	Jan	) not that acute not that acute
283	Keith	how acute was the sudden left side weakness at 2 a.m.?
284	Ron	yes our patient can tell the exact time when he felt the weakness
285	Keith	does that mean he was woken up by it or he was still asleep]
286	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]
287	Jan	] mm mm (.)
288	Keith	this mean he couldn’t walk but he also how about his upper limbs?
289	Ron	mm
290	Keith	muscle (.) function
291	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?
292	Tutor	yes possibly
293	Ron	yes]
294	Jan	but isn’t it that the sensory fibres not really directly passing through the internal capsule?
295	Tutor	well you know the internal capsule is not a single (.) you know some some disease may affect the the: anterior or posterior you know affecting the 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?

296	Ron	so is it true that it is very difficult to differentiate between sub-cortical lesion and internal capsule lesion? (.)
297	Tutor	well the internal capsule is a sub-cortical lesion
298	Ron	oh I see]
299	Tutor	] OK (.) so mind you know the history and phys 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 (.)
300	Jan	mm:
301	Ron	I would think it is uh the site of lesion would be uh: the sub-cortical region on the right side affecting the mid s: mid ce m the mid cerebral artery region (that's it)
302	Tutor	yes OK it's possible (.) (nodding)
303	Ron	so shall I go on with the investigations after the admission?
304	Tutor	mm mm
305	Ron	so:]
306	Tutor	]so any (vascular risk) factors for this gentleman,
307	Sue	patient was]
308	Tutor	] stroke (.) you know in this age group it's not that common]
309	Ron	] (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
310	Tutor	mm mm that's possible (.) especially because of the Horner's syndrome (.) OK
311	Ron	(.) do you mean that um: Horner's syndrome can arise from uh: a neck trauma injury?
312	Tutor	yes possible
313	Ron	ss
314	Tutor	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
315	Ron	mm (.) (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 lectiform 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 mi 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 cervical 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 (.)

326	Tutor	OK very good
327	Ron	and: (.)
328	Tutor	young stroke
329	Ron	causes of stroke in young patient and uh what should we need to look out for
330	Tutor	) yes
331	Ron	in patients presenting with this history (.) some uh we can discuss now young patient with stroke / we can discuss the causes of it
332	Tutor	mm well as you've (.) OK you can reserve for the the: later part of the tutorial
333	Ron	OK
334	Tutor	or just a follow up question, (.) is OK
335	Ron	OK (.)
336	Fay	so I will uh I will uh deal with the next patient
337	Tutor	(.) do you know the the: causes of ischaemic stroke you know in young patient
338	Fay	(.) deficiency
339	Tutor	mm mm
340	Fay	mm (.)
341	Sue	it could be cardio (.)
342	Jan	(.)
343	Tutor	yes very good
344	Ron	vasculitic changes
345	Tutor	so you have checked the VSL, this is normal?
346	Ron	did not notice
347	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
348	Tutor	) yes it's possible it's possible
349	Fay	OK
350	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? (.)
351	Ron	I think it's possible to be relevant because of the trauma to the neck
352	Tutor	uh huh
353	Ron	and actually the the case MO has asked for any uh traumatic history or any neck manipulation
354	Tutor	yes very good
355	Ron	(.) mm mm and the patient denied any history]
356	Tutor	] we are very near to the border you know / people are you know]
357	Ron	] I didn't actually ask]
358	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
359	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	diplopea, 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

406	Sue	any facial asymmetry? (.)
407	Fay	mm: (.) ye:s 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 (.)
408	Trudy	you mean left
409	Ron	right or left
410	Fay	right
411	Trudy	you mean] uh
412	Fay	] the problem is on the left side but the facial palsy is on the right side
413	Tutor	so, did the patient complain of facial palsy?
414	Fay	no he did not complain it was just on examination
415	Tutor	OK]
416	Sue	] diplopia]
417	Tutor	] 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
418	Sue	{whispering}
419	Fay	I mean for symptom-wise he just had slurring of speech and weakness, which are]
420	Tutor	]you know slurring of speech is a relatively non-specific term (.)
421	Fay	mm mm (.)
422	Tutor	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: (.)
423	Fay	I’m not quite sure]
424	Trudy	] if the content is understandable
425	Fay	he’s uh communicable
426	Tutor	what kind of patient can he may have difficulty in expressing himself you know
427	Fay	no he’s communicable
428	Tutor	any evidence of so-called of finding difficulty
429	Fay	no he’s (.))
430	Tutor	(or just you know)
431	Fay	he’s now communicable]

432	Trudy	] so does he he's recovered now,
433	Sue	you mean the slurring of speech has got]
434	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:]
435	Tutor	] you know this slurring of sp 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
436	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]
437	Tutor	] OK
438	Fay	] and no delays in responding to us (.)
439	Ron	is there any problem with the tongue control?
440	Fay	uh:: symptom-wise, no / (.)
441	Tutor	so on presentation any other uh swallowing problem
442	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 (.)
443	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}
444	Tutor	(so you) it's it's already one month past the uh from the occurrence, how how did the symptoms and signs progress
445	Fay	the left side weakness remains (.)
446	Tutor	mm mm]
447	Fay	] and the (.) I'm not quite sure about the slurring of speech (.)
448	Tutor	so no sign of recovery
449	Fay	uh
450	Tutor	not even a trace]
451	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]

452 Tutor] mm mm

453 Fay (.) speaking (.)

454 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

455 Fay I think so

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

457 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

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

459 Fay] mm mm yes

460 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 (.)

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

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

463 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

464 Tutor mm mm

465 Fay um: family history uh no uh]

466 Tutor] but the schizophrenia history is very important OK

467 Fay mm

468 Tutor do you know why? (.)

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

470 Tutor yes what else

471 Sue affects his compliance too

472 Tutor] yes very good (.) so what's your (.) what's your name

473	Fay	Fay
474	Tutor	Fay
475	Fay	] mm
476	Tutor	) OK Fay well you haven't mentioned about the poor control of diabetes (.) how about the blood pressure control
477	Fay	uh: he: was told to be hypertensive but he was not on any medication and he defaulted follow up
478	Tutor	mm mm
479	Fay	(.) 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]
480	Keith	(^^^)
481	Tutor	]OK (.) Family history social history that kind of (.) important for the management?
482	Fay	uh: 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 (.)
483	Sue	is he a smoker?
484	Fay	uh: chronic smoker, non-drinker (.)
485	Tutor	OK so apparently the social support of this gentleman is not that good?
486	Fay	mm (.) when we clerk him]
487	Tutor	] so we expect some discharge problem]
488	Fay	]we saw that uh: he's uh:]
489	Tutor	] this gentleman
490	Fay	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 (.)
491	Tutor	mm mm (.) OK anything from the physical examination?
492	Fay	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)
493	Tutor	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

513	Fay	we should not do anything that can cause unpleasant to the patient
514	Tutor	so what is your comment (.)
515	Fay	only with touch I think
516	Jan	maybe not done in every single patient but uh in the case that you have some trigeminal nerve problem]
517	Tutor	](^^^)
518	Fay	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}
519	Tutor	that's OK again you can you can go back to your basic neuroanatomy (.) so you you've if the tongue cannot move (.) mm (.) so where is the lesion?
520	Sue	brain stem]
521	Jan	] brain stem
522	Tutor	if it is the brain stem it should be bilateral (.) so do you think other (.) signs are compatible with bilateral (.) involvement? (.)
523	Jan	mm (.)
524	Tutor	bilateral sub-cortic bilateral cortical bilateral: so do you think it's likely? mm mm (.)
525	Fay	we are not quite sure about that
526	Jan	(.)
527	Sue	because the (.) both are bilateral
528	Trudy	(neurone)
529	Keith	it can protrude right
530	Fay	yes it can protrude]
531	Keith	] protrude (but not left right)]
532	Fay	] yes yes
533	Jan	then that should (.)]
534	Tutor	] so he's not (.)
535	Fay	he can understand other instructions well so I don't think it's that he has a it's a problem in understanding instructions (.)
536	Jan	but how will it protrude

537	Ron	I think if he can protrude then there is no problem with the uh:]
538	Jan	] power of the tongue]
539	Ron	] involvement
540	Sue	it's just some coordination
541	Jan	the coordination between you and his tongue {laughter}
542	Fay	but he has no problem understanding other instructions (.)
543	Jan	but in case he can protrude the tongue and no deviation so the power should be OK on both sides
544	Keith	mm mm
545	Fay	(^^^)
546	Ron	is there any cerebellar signs, because he has uh]
547	Jan	] a nystagmus]
548	Ron	] nystagmus and (dys)coordination of tongue
549	Fay	there is (pass pointing)
550	Ron	oh
551	Sue	how about (.)
552	Ron	which side (.)]
553	Keith	] when I did it he was OK there was no (pass pointing)
554	Sue	how about other cerebellar sign]
555	Keith	] maybe some tremor (.) but generally he was OK]
556	Fay	] I was not (.)
557	Jan	so there is no cerebellar signs except nystagmus?
558	Keith	yes (.)
559	Tutor	and you know nystagmus is not a ~pure cerebellar sign
560	Students	mm mm
561	Trudy	brain stem)
562	Tutor	) 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
563	Fay	usually if it's central the fast phase is towards the lesion side
564	Tutor	mm mm
565	Fay	so this nystagmus we think that it's to the left side (.) which is not the lesion side

566	Tutor	mm mm
567	Fay	{laughs} I'm not quite sure about the (RV)
568	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?
569	Fay	I could not identify any]
570	Tutor	] just (.)
571	Fay	(.) yeh (.)
572	Tutor	any sign of any vertigo
573	Fay	no not that the patient complained of
574	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?
575	Fay	so (.) on examination of the limbs, uh for the upper limbs]
576	Tutor	] just focus on the cranial nerve
577	Fay	uh cranial nerve uh there's no other abnormalities on that (.)
578	Tutor	it's not a taxic type of nystagmus (.)
579	Fay	] ataxic type, what is this so how it does it look like]
580	Tutor	] the abducting uh (AV) abducting is more (affected) (.)
581	Fay	I am not su]
582	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 (.)
611	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]
612	Tutor	] go to go to the patient and check
613	Fay	but we did quite a few times and really the two sides are upgoing so]
614	Tutor	] but not one side
615	Fay	not one side
616	Ron	is there any old stroke?
617	Fay	no, no history of previous stroke / it's the first time]
618	Tutor	] so any possibility]
619	Fay	]we (.) for many times but
620	Jan	is it just withdrawing rather than (.))
621	Fay	no it's quite obvious is that the fing that the toes are (.) back
622	Ron	is that a cord lesion
623	Fay	uh what]
624	Ron	] was that a cord lesions that (would make) uh bilateral]
625	Fay	] but he has no cranial nerve symptoms
626	Ron	oh
627	Fay	his cranial nerve symptoms are]
628	Tutor	so] you found a mixture of upper and lower motor neurone sign
629	Fay	I'm not quite sure so I]
630	Tutor	] OK so we can go to the patient and see:
631	Fay	yes I would like us to go and see the patient
632	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?
633	Fay	uh the sensation is uh: is the same for both sides
634	Tutor	so: normal?
635	Fay	(nodding)
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 (.)
658	Fay	we did not ask the patient to go down (.) I could not remember (.) {laughter}
659	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}]
660	Jan	] a pass
661	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)
662	Fay	um no uh: no abnormality is noticed
663	Tutor	no abnormality]
664	Fay	] is detected (.) no abnormality is detected
665	Tutor	so how about blood pressure?
666	Fay	I did not complete that
667	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?
668	Fay	mm:]
669	Tutor	] (.) further giving you hints that the blood pressure is not that good probably (.)
670	Fay	as far as I can rec remember there was no uh ab uh abnormalities that I could identify
671	Tutor	mm mm (.) good (laughter) (.) OK the diabetic control any any evidence of diabetic problem?
672	Fay	symptom-wise he has (.) he has polydipsia, polyuria,]

673	Tutor	] what I want is]
674	Sue?	] {whispering} physical findings
675	Tutor	]from the physical findings, any complication of diabetes, (myelitis)
676	Fay	we did not look]
677	Sue	] fundus]
678	Fay	]at the fundus but we test the sensation(.)
679	Sue	urine urinalysis
680	Tutor	urine very good
681	Fay	we did not do the urine {laughter}
682	Jan	under (.) pulses?
683	Fay	uh present (.)
684	Tutor	you mentioned the diminished reflex
685	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
686	Tutor	have you performed the reinforcement test (.) {laughter}
687	Fay	{shaking head}
688	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
689	Fay	{nodding} (.)
690	Tutor	how about the sensory testing any:
691	Fay	we just did the pinprick but it's uh equal on both sides both upper and lower limbs]
692	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
693	Fay	we did not test vibration
694	Tutor	so you suspect the patient having proven neuropathy (.) you know which sensory modality would go first will go first?
695	Students	vision
696	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 you 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?

720	Fay	he can feed himself but we did not ask him to walk (.)
721	Tutor	mm mm and the patient is living alone there is no social support (.)
722	Fay	but his (.) function is quite good he has a stable job
723	Tutor	mm
724	Fay	he has well-controlled]
725	Tutor	] that's the question you know can the patient get uh go back to his previous uh: occupation his previous job? (.)
726	Fay	(.) 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 (.)
727	Tutor	how about his home environment (.) do you think his home environment is conducive for his return
728	Fay	then we have to ask an occupational therapist for an assess
729	Tutor	well (.) in an examination situation you can't ask the occupational therapist to do the assessment for you (laughter) (.)
730	Jan	and to ask the speech therapist to assess the speech for you
731	Tutor	I think a simple question is good enough since several simple questions
732	Fay	whether there's a lift
733	Tutor	OK (.)
734	Fay	uh::
735	Tutor	what sort of pulsing: you know
736	Fay	mm mm
737	Tutor	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
738	Fay	mm mm (.)
739	Tutor	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]

766	Fay	] left side weakness
767	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
768	Sue	but where is the decussation of the: (.)
769	Trudy	motor neurone
770	Tutor	you mean]
771	Sue	] is it
772	Tutor	uh which which which nerve
773	Sue	of the: seventh nerve
774	Tutor	I think seven is (.) in the pons
775	Ron	(.) {to Jan and then a Student replies to him while Jan asks the following question}
776	Sue	(.)
777	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?
778	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 (.)
779	Jan	(^^^)
780	Trudy	(.) above]
781	Ron	] the facial nerve (.)]
782	Tutor	] OK you should read about the new anatomy OK (.) please take time to digest everything OK (.)
783	Sue	{Cantonese}
784	Jan	{Cantonese}
785	Sue	haih ah)
786	Jan	(.) go dih
787	Sue	haih ah]
788	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 ccase 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?

14	Ron	acute onset bilateral lower limb progressive and ascending numbness for three) days
15	Tutor	) three days good
16	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 (0.2)
17	Tutor	spinal cord pro:blem (.) 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 (0.2_ yes what do you think Mr Lee?
18	Cathy	uh I'm wondering about onset {ac} of the numbness {dc} is it uh how acute is acute is is there may be like a vascular cause say inflammatory cause so I'm wondering like if there were any systemic symptoms (.)
19	Tutor	well if it is a vascular cause it's less likely to be progressive over the past three days (0.2) you know for vascular it's usually sudden (.) when it happens it happens (.) it rarely progress over the next three days in asc<u>end</u>ing you know (.) that is less likely (.) infection cause you are thinking abou:t (0.2) what kind of infection would you, are you worried about? yes Miss Fay?
20	Fay	mm:
21	Tutor	everybody gets a {smiling} chance (.) to involve in the discussion (0.3)
22	Fay	it could be viral infection
23	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?
24	Sue	myelitis?)
25	Tutor	) yes myelitis, myelitis is possible, yes? {turning to Jan} (0.2) besides myelitis can this be just uh: some kind of uh:
26	Fay	it could be just some compression)

27	Tutor	) 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
28	Sue	(^^^)
29	Tutor	no we are still talking about the history not the investigations at this moment (.) specifically ask
30	Fay	whether there's any back pain (.) um:
31	Tutor	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)
32	Students	(^^^)
33	Tutor	ye:s then we will specifically ask what about) (^^^)
34	Jan	(^^^))
35	Tutor	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
36	Students	yes
37	Tutor	housewife?
38	Ron	{nodding} housewife mm
39	Tutor	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

40	Ron	mm {nodding}
41	Tutor	good (.) then you can give us uh the present illness part
42	Ron	for the history of present illness, (.) uh she described the numbness as a tingling sensation which is parasthesia 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, (.)
43	Tutor	it's not three days, it's seven days?
44	Ron	uh: because she was admitted quite some time
45	Tutor	ok so the symptom onset was three days before the admission
46	Ron	yeh
47	Tutor	but by the time you clerked the case) it's
48	Ron	) yes)
49	Tutor	) she has been in this hospital for quite some time)
50	Ron	)yes {nodding}
51	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:
52	Ron	{nodding} it was acute onset she can describe the day she had the symptoms on her) foot
53	Tutor	) and it was it was not triggered by any specific movement, (.) no preced) ing
54	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,
55	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
56	Fay	) progressive?
57	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
58	Ron	{nodding}
59	Tutor	ok?
60	Ron	{nodding} (^^^) the upper limb was normal (.)

61	Tutor	so any viral illness preceding this event?
62	Ron	it was not preceded by any uh upper respiratory tract infection, by any uh or vaccine injection
63	Tutor	ok good
64	Ron	there was no associated weakness, there was no fecal or urinary incontinence,
65	Tutor	mm mm
66	Ron	she volunteered the history of taking herbal medicine two weeks prior to the onset of the symptoms)
67	Tutor	) why does why did she take herbal medicine two weeks before the onset?
68	Ron	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
69	Tutor	good
70	Ron	yes and there was no heavy metal exposure in her occupation and the environment (0.2)
71	Tutor	any motor involvement
72	Ron	no there was no weakness
73	Tutor	and upper limb)
74	Ron	) were spared
75	Tutor	were spared (0.2) because,
76	Ron	{smiling}
77	Tutor	it is only ascending to T4 level
78	Students	{laughing})
79	Tutor	) of course
80	Ron	right yes
81	Tutor	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)
82	Keith	myelitis, transverse myelitis,
83	Tutor	{dc} transverse myelitis yes (.) anything else
84	Fay	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

123	Tutor	why
124	Trudy	it usually affect the (^^^)
125	Tutor	no because we need to (^^^) the patient (.) we have to repeat the OGD (.) for gastric ulcer we usually repeat
126	Trudy	oh yeh
127	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,
128	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 (.)
129	Tutor	two thousand and,
130	Ron	* November)
131	Tutor	) well
132	Ron	) she presented with vertigo, diplopia, headache, vomiting and later progressive (^^^) tetraplegia
133	Tutor	tetraplegia {Ron nodding} and uh but then the diagnosis was uh
134	Ron	Bickerstaff's brainstem encephalitis
135	Tutor	how was the diagnosis made
136	Ron	the diagnosis was made uh:: based on the clinical uh presentation and the uh (0.2) (^^^) test (0.2) uh::)
137	Tutor	) did she)
138	Ron	) the (^^^) test was done (^^^)
139	Tutor	have an MRI (0.2) of the brain?
140	Ron	uh:)
141	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
142	Ron	yes they checked the (^^^) antibody it was negative)
143	Tutor	) mm mm
144	Ron	and uh: they: I'm not sure whether they had the imaging done I assume they had the imaging MRI

145	Tutor	and then the patient had a very good recovery
146	Ron	uh not exactly
147	Tutor	not exactly because then she could still visit Thailand
148	Ron	the tetraplegia recovered but there there were residual neurological deficits
149	Tutor	as reflected by?
150	Ron	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)
151	Tutor	) ok
152	Ron	) residual weakness uh: the neurological deficits were ataxic gait and diplopia
153	Tutor	ok (0.2) do you have any questions? (0.2) what about family history
154	Ron	uh she had no family history of uh similar illness
155	Tutor	and besides these besides h pylori and this uh brain stem encephalo)pathy
156	Ron	) she had good past health
157	Tutor	) she enjoyed good past health (.) no other problem at all:
158	Ron	no
159	Tutor	ok social history?
160	Ron	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
161	Tutor	mm mm (0.2) good (.) any questions (0.2)
162	Fay	was a lumbar puncture done at the time that encephalitis was) (^^^)
163	Ron	) yes it was done
164	Tutor	you mean the last time or this time
165	Fay	last)time
166	Ron	) last)time
167	Tutor	) last time
168	Fay	what was the (finding) (0.2)
169	Tutor	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 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

181 Ron part of) them

182 Tutor) 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

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

184 Ron 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
203	Tutor	fusion test, no?
204	Ron	uh: fusion tests were (0.3) uh)
205	Jan	{whispering} (^^^)
206	Ron	no
207	Jan	no)
208	Tutor	) ok (0.2)
209	Ron	there were no other cerebellar signs
210	Tutor	ok (.) so it's purely sensory right?
211	Ron	yes
212	Tutor	{dc} it's true purely sensory (.) ok (0.2) so do you think the physical examination part contributes to the (.) diagnosis?
213	Jan	actually the involvement of the sensory modalities of pain, and also the uh::
214	Tutor	(light) touch?
215	Jan	pain? {turning to Trudy and Ron}
216	Trudy	(light) touch
217	Fay	pain, light touch) vibration
218	Ron	pain, light touch,) and vibration
219	Jan	pain and) vibration
220	Ron	)(^^^))
221	Fay	)(^^^)
222	Jan	that proprioception is (spared)
223	Tutor	quite odd right? well you know sensory test is always difficult (.) you you you you do the sensory test in the morning and you repeat in the afternoon and you 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)

224	Students	yes
225	Fay	(^^^) she has
226	Tutor	probably not because the diplopia)
227	Jan	) oh {smiling at R}
228	Tutor	therefore fusion test is more, can more truly reflect the cerebellar function
229	Fay	{looking at R} but diplopia is it only in extreme uh gaze or even the looking uh
230	Ron	uh:: sometimes she complained of that even in the central gaze there is diplopia
231	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?
232	Ron	there were some investigations (.) done in the ward during this admission
233	Tutor	yes if you you you are you were the doctor in charge what kind of investigations would you ask (0.2)
234	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)
235	Tutor	) yes
236	Ron	to exclude any cord compression
237	Tutor	would you do a lumbar puncture? (0.3)
238	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
239	Tutor	) unlikely peripheral right sensory level up to T4 {draws line across chest}
240	Ron	{nodding} yeh so it depends on (^^^) so yes I will perform this {laughing}
241	Tutor	to look for?
242	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

243	Tutor	ok (.) and so some must have been done right since she has been here for quite some days
244	Ron	{smiling} (0.2) uh yes
245	Tutor	was MRI performed?
246	Ron	MRI was awaiting (.) awaiting
247	Tutor	still waiting) ok
248	Ron	) still waiting and the nerve conduction study was done (0.2)
249	Tutor	and?
250	Ron	and found that there was no acute neuritis, there was no demyelination of the peripheral nerves, there were no peripheral nerve channelopathy (0.2)
251	Tutor	but those were actually expected right? it's unlikely to be peripheral cause (.) ok (.) what other tests have been performed?
252	Ron	{shaking head}
253	Tutor	no
254	Ron	just the nerve conduction study and some uh routine blood tests
255	Tutor	but you said you said this patient has been with us for some time?
256	Ron	yeh just a few days
257	Jan	{laughing})
258	Tutor	no LP at the moment
259	Ron	no (0.2)
260	Tutor	they're waiting for the MRI
261	Ron	yes {nodding}
262	Tutor	no treatment at this moment
263	Ron	(0.2) vitamin b complex (0.2)
264	Tutor	excellent
265	Jan	{laughing})
266	Ron	{laughing}
267	Tutor	what about immune immune test
268	Jan	any blood test for) immune markers
269	Fay	) (^^^) antibodies
270	Tutor	) yes
271	Ron	yes uh: I didn't specifically look for that (0.2)

272	Tutor	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
273	Ron	mm {nodding}
274	Tutor	because the disease will not wait 9.) that’s the problem (0.2) well it’s a good thing (.) you said it’s not a common problem it’s not commonly seen (0.2) ok good (.) anything you would like to uh ask, discuss (.) yes Miss Fay
275	Fay	(^^^) {laughing} what was the result of the last lumbar puncture in two thousand and six I mean
276	Ron	{laughing} I: don’t know
277	Tutor	you can say the patient did not know so that (^^^) save you
278	Students	{laughing}
279	Tutor	well it’s unrealistic right the patient could tell you what was the LP)
280	Fay	) I think she he has looked at the charts (^^^)
281	Tutor	you know during exam)
282	Fay	) oh ok)
283	Tutor	) there’s no chance to read the charts
284	Fay	yes (^^^))
285	Tutor	) 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
286	Fay	mm mm
287	Tutor	ok?
288	Ron	so the working diagnosis of the uh Miss Wong is acute transverse myelitis multiple sclerosis and to exclude cord compression
289	Fay	(^^^)

290	Tutor	you know (.) if they put to rule out cord compression as the first working diagnosis
291	Jan	maybe MRI should be earlier
292	Tutor	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
293	Students	{nodding}
29	Tutor	so without investigation (.) we can get to the diagnosis (.) go back in two weeks time and you will uh where's the patient now
294	Ron	D seven
295	Tutor	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)
296	Ron	) because she was transferred on Wednesday {smiling}
297	Tutor	good timing
298	Students	{laughing}
299	Tutor	{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
300	Ron	{nodding}
301	Fay	with this history of uh: brain stem encephalitis still is it not possible for multiple sclerosis (^^^) brain stem and also the T4 spinal cord
302	Tutor	uh::
303	Fay	can it be (^^^) lesion (0.2)
304	Tutor	I guess first of all (0.2) MRI must have been done last time (.) if this is multiple sclerosis
305	Fay	{nodding} mm
306	Tutor	the diagnosis was already made (.) right (0.2)
307	Fay	but clinically do you know

308	Tutor	ok there are some specific signs for multiple sclerosis when it’s uh known as inter nucleus ophthalmoplegia
309	Students	{nodding}
310	Tutor	do you know what this is?
311	Students	{nodding}
312	Tutor	very complicated
313	Students	{laughing} (^^^)
314	Fay	very complicated
315	Tutor	yes (.) do you know the sign)
316	Jan	) nystagmus of the (abductive) eye and the other eye cannot abduct
317	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
318	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)
319	Tutor	fourteen years history of,
320	Trudy	SLE (.)
321	Tutor	ok

322	Trudy	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 appetitie after haemodialysis
323	Tutor	so do you think the chief complaint is a little bit too long
324	Trudy	{nodding} (0.2) so um: patient with a fourteen years year history (.) of um uh lupus nephritis on haemodialysis complained of ten times vomiting
325	Tutor	do you think fourteen years is an important number
326	Trudy	(0.2) uh:: long history of SLE
327	Tutor	well basically it's it's)
328	Jan	) (^^^)
329	Tutor	end stage renal failure due to SLE on dialysis presented with how many days history of (.) GI symp)toms
330	Trudy	) two days)
331	Tutor	) 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
332	Trudy	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)
333	Tutor	) 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

334	Trudy	) no uh no family members with uh similar symptoms (0.2)
335	Tutor	no family members probably yes no well other people share the same food, did not have similar features (.) ok, so uh::)
336	Trudy	)and no travel history
337	Tutor	is she still on haemodialysis
338	Trudy	{nodding}
339	Tutor	she's unlikely to have a travel history right, she needs two times per week or three times per week,
340	Trudy	(^^^)
341	Tutor	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
342	Ron	um::)
343	Tutor	}besides gastro-enteritis, what else can give rise to vomiting (0.4)
344	Ron	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
345	Tutor	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)
346	Ron	it may also be due to distal pathology for example increase in renal pressure,
347	Tutor	ye:es 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)

348	VS	) could it be)
349	Tutor	) yes
350	VS	could it be (^^^) symptom of the SLE
351	Tutor	SLE give rise to dysphagia: it's not common (.) it's not scleroderma (0.2) ri:ght 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 int(ra) 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
352	Keith	lupus cerebritis
353	Tutor	uh,
354	Keith	lupus cerebritis
355	Tutor	yes you can say that can be due to the disease per se: vasculitis vasculitis involving the the the brain or::, (0.2) due to problems related to, (0.2)
356	Sue	the patient is immuno-suppressed
357	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
358	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
359	Tutor	) sorry just one more dx we haven't discussed yet (.) if the patient's pregnant (.) she's a young lady
360	Students	{nodding}
361	Tutor	right, that should always be remembered
362	Jan	{smiling}

363	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?

380	Trudy	) uh sorry graft uh causing re causing graft rejection
381	Tutor	ye:s) that's better
382	Trudy	) and uh so uh the transplanted kidney was removed in may this year (.) and she referred to uh the haemodialysis (.)
383	Tutor	ok (0.3)
384	Trudy	and this is about the uh renal problem (.)
385	Tutor	no other history, present illness?
386	Trudy	history of present illness she also had um uh other like complications of SLE (.) for the) (^^^)
387	Tutor	} no no no I mean uh what about (.) can you describe in detail about this episode uh the reason for this admission yes
388	Trudy	um this time she was she had uh haemodialysis last Saturday and
389	Tutor	she had haemodialysis last Saturday) and
390	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
391	Tutor	) but did anything unusual happen during the haemdialysis (0.2)) was there any hypo
392	Trudy	uh)
393	Tutor	) tensive event et cetera during the dialysis
394	Trudy	I think (^^^) not event
395	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,
396	Trudy	but then because of the heavy rain she did not come to hospital till Monday (.) oh until)
397	Tutor	) Sunday
398	Trudy	{looking at notes} like the day after the rainstorm warning she came
399	Students	{laughing}
400	Tutor	it was Sunday it was Sunday
401	Students	{laughing}
402	Tutor	ok (0.3)
403	Trudy	and uh yeh so there was no change in bowel habits

404	Tutor	so by the time she came to hospital she still had a lot of) vomiting
405	Trudy	) after admission her vomiting subsided
406	Tutor	ok
407	Trudy	and she (end of tape) only vomited once after admission
408	Tutor	ok (.) then
409	Trudy	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? (.) and oh)
8	Zelda	) the blistering eruption
9	Tutor	) yeh
10	Zelda	) that's fine yes (0.1) um: (0.1)
11	Tutor	so perhaps you'd like to repeat the the major clinical features of this patient? (0.2)
12	Zelda	um so)
13	Tutor	) I'll tell you that I want you not to refer to your notes {laughs}
14	Zelda	um do do you want me to uh: include um the drugs this time and also the)
15	Tutor	) yeh yeh)
16	Zelda	) some of the physical examination)
17	Tutor	) mm
18	Zelda	uh so uh Mr Lau um a sixty-five year old um retired um government servant um presented with um blistering um (.) blistering
19	Tutor	(^^^)

20	Zelda	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)
21	Tutor	) 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)
22	Zelda	) 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)
23	Tutor	) 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?
24	Zelda	um those over the lower limbs are ruptured and when I see the patient um actually those uh over the palms are uh)
25	Tutor	) still intact)
26	Zelda	) 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)
27	Tutor	) mm)
28	Zelda	) um that even contains some pus over the right dorsum
29	Tutor	) mm)
30	Zelda	) and um there are also uh some erythromatous 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 stoo:l

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
49	Tutor	so he is well otherwise?
50	Zelda	uh but because he has had uh several vascular accidents um in 2003 which left him having a left (hemiparesis)
51	Tutor	^^^^ past health (.) you can mention that later OK (.) I can't remember whether you have mentioned whether the rash is itchy or not)
52	Zelda	) uh he said it's itchy but it's not too (.) it's both itchy and painful but not to a very severe extent
53	Tutor	) OK
54	Zelda	) s s it's moderately itchy)
55	Tutor	) 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 <u>might</u> be taking some form of traditional Chinese medicine, which (.) they might <u>think</u> (.) 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
56	Zelda	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
57	Tutor	mm
58	Zelda	and um (0.2) the uh the uh left hemiplegia ^^^^ from the the)
59	Tutor	) ^^^)
60	Zelda	since then he has become retired and he requires a quadropod to walk
61	Tutor	OK)
62	Zelda	) and)
63	Tutor	so you mean he had to retire early and is it because of this illness?)
64	Zelda	) because of the illness um but he said that he doesn't really have any financial problems uh and he was alright with that
65	Tutor	perhaps this is so because he mentioned that he is a civil servant)

66	Martin	) mm
67		) so with retirement he would be entitled to what?
68	Zelda	um the {(gong sik gam)})
69	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)
70	Zelda	um I did)
71	Tutor	) originally
72	Zelda	) I didn't specify because it seems that he was a bit reluctant to tell me
73	Tutor	really?
74	Zelda	I I I'm a bit unsure
75	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)
76	Zelda	) no)
77	Tutor	) what he is doing professionally (.) why should he why should he be so evasive?
78	Zelda	because I don't know when when I asked about the family uh things)
79	Tutor	) he's also evasive as well
80	Zelda	he feels a bit I just have this feeling that
81	Tutor	{(^^^ Cantonese) someone enters the room} (0.3) are you alright?
82	Student	yes
83	Tutor	because you don't feel too well
84	Student	(^^^)
85	Tutor	OK (0.4) thank you thank you (0.6) so we have been discussing the first patient with blistering eruption OK
86	Zelda	(^^^) concerned when I asked about any forms of family history
87	Tutor	OK OK (.) so you have no idea at all whether he is married or)
88	Zelda	) oh he is living with his son
89	Tutor	his son?
90	Zelda	yes who is also taking care of him
91	Tutor	mm how old is his son?

92	Zelda	I haven’t specified
93	Tutor	mm mm right: so anything else? (0.3) did you did you tell me that you want to elaborate on his drugs?
94	Zelda	oh yes um for so as for the last time um the um the red blood cells was elevated and also the (^^^))
95	Tutor	) no but the drugs)
96	Martin	) the drug)
97	Zelda	) oh yes the drugs he’s on aspirin, metformin, (^^^) some dologesic and (^^^)
98	Tutor	are you familiar with all these kinds of medications?
99	Zelda	um it)
100	Tutor	) you are quite clear about all their indications?
101	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
102	Tutor	you just used the term *some* dologesic what do you mean?
103	Zelda	um I think that (^^^))
104	Tutor	) he’s taking that)
105	Zelda	) request)
106	Tutor	) so we usually call it dologesic PRN)
107	Zelda	) PRN)
108	Tutor	) on demand basis
109	Zelda	on demand basis
110	Tutor	we don’t usually have *some* analgesics OK?
111	Zelda	OK >
112	Tutor	alright
113	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 tha that case mentioned that um he was known to have poor foot care
114	Tutor	mm mm mm
115	Zelda	OK
116	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 to (.) 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

154	Zelda	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)
155	Tutor	)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)
156	Zelda	) 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)
157	Tutor	) mm mm
158	Zelda	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
159	Tutor	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 stools 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)yria

171 Tutor) porphyria isn’t it)

172 Zelda) oh yes (^^^)

173 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 epidermylolosis 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)

174 Zelda) oh sorry ah)

175 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)

176 Zelda) redness

177 Tutor) redness multiforme means)

178 Zelda) every)where

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

180 Zelda) blisters

181 Tutor perhaps blisters OK and very often it could be a drug induced problem,

		which which are the most common drugs
182	Zelda	I've just seen a Chinese medication induced one
183	Tutor	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?
184	Martin	) I think it's usually some)
185	Students	) (^^^)
186	Martin	) septrin
187	Tutor	septrin, sulphur containing drugs sulphonomanides, (^^^) septrin)
188	Zelda	) carbamezepine)
189	Tutor	) carbamezepine yes)
190	Martin	)allopurinol
191	Tutor	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
192	Students	yes
193	Tutor	last Saturday so drug is always important (.) OK and then? what is the other big categories of differential diagnosis?
194	Zelda	um auto-immune
195	Tutor	auto-immune OK and then such as?
196	Zelda	um pemphigus um uh vulgaris
197	Tutor	mm) OK
198	Zelda	) pemphigoid)(^^^)
199	Tutor	) 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?
200	Zelda	(laughing) um:
201	Tutor	SLE
202	Zelda	oh SLE
203	Tutor	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,

247	Tutor	) gus yes OK so
248	Zelda	) (^^^)
249	Tutor	if mucosa is involved it's pemphigus (.) which is the one to give you high incidence in the elderly?
250	Zelda	uh pemphigus uh oh {laughing} bullous pemphigoid
251	Tutor	bullous pemphigoid OK and which)
252	Zelda	) (^^^sixty) years)
253	Tutor	) and which one requires a big a big dose of steroid treatment? (0.2)
254	Martin	(^^^))
255	Zelda	) I think pemphigus because it's life) threatening
256	Tutor	} 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)
257	Zelda	) would be immunoglobulin the IgG
258	Tutor	anything else any specific antibody?
259	Martin	some form of anti epidermal antibody that will be positive in pemphigus but not in pemphigoid
260	Tutor	yeh it is the anti yeh anti skin antibody or the anti-epidermal antibody OK so)
261	Zelda	) 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:)
262	Tutor	) yeh
263	Zelda	) is it true that
264	Tutor	) yes I think skin biopsy obviously will also give you the relevant changes uh uh relevant features as)well
265	Zelda	)I thought is it a bit invasive or is it often that they perform
266	Tutor	in general principle any biopsy would be invasive but of all the biopsies I would feel most comfor)table
267	Zelda	)yes
268	Tutor	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?

288	Zelda	mm)
289	Martin	) (^^^)
290	Zelda	) (^^^)
291	Tutor	mm
292	Zelda	azathioprine or)(tetracycline)
293	Tutor	) mm
294	Zelda	)(^^^)
295	Tutor	)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 we 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?
296	Zelda	oh oh maybe the some local creams may be given to treat the tinea pedis)
297	Tutor	) mm 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
298	Martin	mm (0.3)
299	Tutor	OK? alright? so perhaps we (.) go back to our objectives, about the past health isn’t it?
300	Martin	) mm ha stroke
301	Zelda	) mm about the stroke
302	Tutor	yeh
303	Zelda	um the common types of cerebrovascular accident)
304	Tutor	) mm mm
305	Zelda	) and the sequelae social and economical point of view
306	Tutor	mm so in clinical practice what are the common types of strokes that you’ll see?

307	Zelda	ischaemic hemorrhagic ones or maybe)
308	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,
309	Martin	mm
310	Tutor	what are the two common types of ischaemic stroke?
311	Martin	uh: you mean the) in situ thrombosis or embolic?
312	Student	) (^^^)
313	Tutor	) thrombotic yeh)
314	Martin	) embolic
315	Zelda	(^^^)
316	Tutor	thrombotic or embolic isn’t it?
317	Zelda	lo{thrombotic or embolic}
318	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?
319	Student	) (^^^)
320	Martin	hypotensive or:
321	Tutor	hemorrhagic stroke)
322	Students	)(^^^)
323	Tutor	no no not the cause OK?
324	Student	(^^^)
325	Tutor	yes intracerebral and:?
326	Students	subarachnoid
327	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:)
328	Zelda	) mm mm
329	Tutor	) how will this how will the clinical features give us hints (.) as regards how to categorise these types of strokes?
330	Martin	mm (0.2)
331	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

350	Martin	) (^^^) heart disease)
351	Tutor	) heart mur)mur
352	Zelda	) heart murmur
353	Martin	)heart murmur)
354	Tutor	heart murmur anything else related to the heart?
355	Students	(^^^)
356	Tutor	arrhythmia OK any others any other positive features to suggest a focus for embolisation?
357	Martin	) carotid bruit
358	Zelda	) carotid bruit
359	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) cer)ebral
360	Zelda	) in the um: maybe a very insidious onset)
361	Tutor	) yes of:?
362	Zelda	ischaemic stroke)
363	Tutor	) which type of ischaemic stroke?
364	Zelda	um for the {lo} embolic
365	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?)
366	Zelda	) um
367	Tutor	so the suddenness of onset would be very im very suggestive of an embolic stroke) whereas
368	Zelda	) this is (^^^)
369	Tutor	insidious onset is suggestive of thrombotic stroke
370	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)
371	Tutor	) with what, sorry?
372	Zelda	evolving um)
373	Tutor	) yes yes
374	Zelda	neuro)logical deficits do we still call that) insidious onset

375 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 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

376 Zelda um I I didn't specifically ask him)

377 Tutor) OK

378 Zelda) in detail

379 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?

380 Zelda um no,)

381 Tutor) has he got arrhythmia?

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

383 Tutor) heart bruit

384 Zelda no no no

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

386 Zelda yes um like

387 Tutor which)suggests

388 Zelda) hypertension,

389 Tutor is his hypertension well controlled now?

390 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 reasona)ble
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 prohylactically 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

416	Martin	for the hypoglycaemia
417	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)
418	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)
419	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
420	Zelda	mm mm
421	Tutor	OK? and then? what other risk factors? did you say hyperlipidaemia?
422	Zelda	yes but he’s not on any medication
423	Tutor	did you say Zo)cor
424	Martin	) Zocor
425	Zelda	oh oh yes he is sorry
426	Tutor	are you aware that Zocor is a stat statin?
427	Zelda	um because sorry um because I have two patients’ notes over here so
428	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?
429	Martin	because the (^^^) may not know high LDL or which HDL)
430	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
431	Martin	mm
432	Tutor	alright? any other risk factors?

433	Martin	{lo-laughing} sex
434	Zelda	{lo} what
435	Martin	male uh male risk factors
436	Tutor	yes male)
437	Zelda	) yes oh the) mono (^^^)
438	Tutor	) you are the weaker sex {laughs}
439	Zelda	{lo} I remember) that
440	Tutor	) any other risk factors?
441	Zelda	gener generally um um (^^^) status, age, gender,
442	Tutor	is he at risk?
443	Martin	(^^^)
444	Zelda	yes sixty)(^^^)
445	Tutor	2-0-0-3
446	Zelda	oh)
447	Tutor	how old
448	Zelda	by 2003 he was sixty
449	Tutor	sixty just reached risk age OK six)ty
450	Zelda	and um he has also the drinking history
451	Martin	mm
452	Tutor	smok)ing
453	Zelda	) non-smoker
454	Tutor	any family history?
455	Zelda	uh no
456	Tutor	probably he's not too happy to answer that question either
457	Zelda	no no family history that I've asked that's specific
458	Tutor	alright (.) so he did have quite a number of risk factors (.) isn't it? OK? so: how has the stroke affected him(.) socially and psychologically? you have mentioned that he has to retire from work, but you are not quite sure whether it's because he chose to do it, maybe because he's a civil servant he thought it's earlier to get the pension {Students laughing} OK, and you don't you have not been successful to ask him what he has been doing professionally isn't it so we can't really gauge how disabled he would be uh uh uh with this impact of stroke isn't it? (0.2) how about

		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 (0.3)
481	Tutor	anything else about this patient that we haven't really catered for? have we finished all the objectives? (0.2) 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? (0.4) {Zelda shuffling through notes}
486	Zelda	um yes I have I think so
487	Tutor	OK perhaps perhaps back to the 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		) (^^^)
499	Tutor	macrocytosis is a is a sign obviously it's a a a afeature of chronic alcoholism OK so is the MCP alright with him?
500	Zelda	um it's all normal
501	Tutor	OK
502	Zelda	I I'm pretty sure
503	Tutor	ah hah how about the biochemistry? what might be indicative of telltale (.) finding?
504	Students	{whispering}
505	Zelda	mm {laughing} he could say
506	Tutor	yes yes
507	Martin	increase in AST more than ALT the gamma increase in gamma GT
508	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 al)right?
509	Zelda	yes it's alright
510	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 uh result OK?
511	Martin	mm (0.3)
512	Tutor	{lo} alright? (0.2) so: any yes
513	Harry	may I know the mechanism for the low potassium with patients with alcoholism?
514	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)
515	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)?
516	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 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

524	Tutor	why did you have to think?
525	Harry	um I didn’t ask
526	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?
527	Harry	) mm
528	Tutor	so did you ask her?
529	Harry	I didn’t went back to see the patient
530	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?
531	Martin	it may affect our drug treatment
532	Tutor	may (.) affect (.) 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
533	Kevin	because the patient presented with lower limbs swelling so the patient is more (^^^) {Students laughter}
534	Martin	(^^^)
535	Tutor	OK anything else?
536	Harry	auto-immune disease can cause failure)
537	Tutor	) yeh
538	Harry	) of the ovaries mm (.) or the endocrine problem
539	Tutor	uh: not not really but nearly you are nearly there)
540	Martin	) is it is it some auto-immune disease like SLE may play up during pregnancy?
541	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 (.)
542	Martin	uh huh

543	Tutor	or less sort of smooth in patients with connective tissue disorders because they are more prone to have what?
544	Martin	anti-phospholipid (^^^))
545	Tutor	) no no yes for anti-phospholipid but in general
546	Martin	{lo} they are more prone to
547	Tutor	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-eclam 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
548	Martin	hah:?){Students' laughter})
549	Tutor	)(^^^) I was so anxious to know what)
550	Students	){laughing}
551	Tutor	because so why should you have mixed up this (^^^)
552	Joy	because I was standing at the end of the bed) and um
553	Tutor	) but they are discussing on the patient next to her bed
554	Joy	we didn't hear the content about the argument)
555	Tutor	)mm
556	Joy	so we just)
557	Students	){laughing}
558	Tutor	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?

559 Harry I haven’t (.) sorry

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 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,

591	Tutor	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)
592	Martin	) mm
593	Harry	)mm
594	Tutor	we very often mention about criteria)
595	Martin	)mm
596	Tutor	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)
597	Harry	no,
598	Tutor	no what are these criteria meant for(.) basically?
599	Martin	for research purpose
600	Tutor	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)
601	Harry	I didn't have the exact criteria but just some features {lo} of Sjogren's syndrome)
602	Tutor	) 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?

603 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)

604 Tutor) are you are you aware of what I mean by Schirmer's test?

605 Joy yes

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

607 Martin) ah)

608 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)

609 Zelda){laughs}

610 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?

611 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 paroti 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 scintography 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 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 <u>even</u> uh 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 uhor 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)
647	Harry	for the learning objectives uh:)
648	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:
649	Zelda	do we need to) rule out
650	Tutor	leukaemia, yes?
651	Zelda	oh oh oh sorry I was thinking about it might be a mixed connective tissue so do we have to:
652	Tutor	no (.) that would bring us to the question of whether this is primary Sjogren's or secondary)
653	Zelda	oh:
654	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)
655	Zelda	oh
656	Tutor	with secondary Sjogren's so this is still Sjogren's syndrome) OK?
657	Students	mm
658	Tutor	but what I'm trying to say is graft versus host disease)
659	Students	oh
660	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?
661	Zelda	{whispering} sarcoidosis
662	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)
663	Student	I yes I saw yes
664	Tutor	you will have) sarcoidosis clinic isn't it?
665	S	) (^^^) yeh
666	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 (0.3) 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? (0.2) do you think she qualifies for the diagnosis of Sjogren's syndrome? (0.2)

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	(^^^))diagnosis
674	Tutor	) her other major symptom would be Raynaud's pheno)menon
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

691	Tutor	yeh ribonuclear protein (.) a high titre of circulating anti-RNP (.) OK? so: has he got RNP?
692	Harry	no
693	Tutor	no isn't it so he's just got anti-Ro so again he she does not seem to fit isn't it
694	Harry	mm
695	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?
696	Harry	include the reason of the uh intestinal obstruction, in our patient
697	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?
698	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
699	Tutor	mm mm mm
700	Zelda	and due to the nature of that it involves the (whole thickness) of the um)
701	Tutor	) yeh
702	Zelda	intestine, and sometimes this is also a single joint pain which can also be an extra GI tract manifestation of um Crohn's disease) then I
703	Tutor	) yeh but Crohn's disease patients would be seldom seldom be under our care (.) they are under the care of the GI physician
704	Martin	hhh
705	Tutor	but how about patients under our care
706	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 sequelae 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?

713	Harry	) mm
714	Zelda	) dermatomy) ositis
715	Tutor	) dermatomyositis yeh
716	Zelda	the mouth being uh the tense skin
717	Tutor	no dermatomyositis would actually lead to the muscles involvement OK?
718	Martin	mm mm
719	Tutor	which part uh of the uh uh es esophagus would be affected? (0.2) it's the laryngeal muscle
720	Zelda	oh:
721	Tutor	it's the initial part of the esophagus because this is the skeletal muscle isn't it?
722	Zelda	oh
723	Tutor	whereas in scleroderma?
724	Zelda	oh it's the (^^^))
725	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?
726	Students	{whispering} (0.3)
727	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?
728	Zelda	) I have I heard heard about this term
729	Tutor	mm you are you must be very familiar with (nephrotic) syndrome isn't it?)
730	Martin	) mm
731	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 de 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

732	Zelda	um what is the mechanism again for the protein losing enter)opathy?
733	Tutor	) probably it's probably due to the vasculitis of the gut so)
734	Zelda	) right:)
735	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)
736	Zelda	but then it only affects protein absorption but not all the other)
737	Tutor	) not absorption
738	Zelda	) (^^^)
739	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
740	Zelda	(^^^) they are losing the protein from the body)rather than
741	Tutor	) yeh yeh yeh (0.4) {Students whispering}
742	Martin	is it associated with (other) sorts of connective tissue diseases?
743	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
744	Martin	B I P?
745	Tutor	yeh {Students whispering BIP} (0.4) OK so other objectives?
746	Harry	that's all for the objectives yes
747	Tutor	I think have I not mentioned that (.) we seem to have rather uh inappropriate or discrepancy)
748	Harry	) yeh
749	Tutor	) between the signs and the symptoms as regards her pain in the ankle?

750	Harry	mm mm
751	Tutor	and so we would like to find out a more objective way to assess whether her ankle is really that sort of affected to induce this her (^^^) 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
752	Harry	) ultrasound
753	Tutor	) 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?
754	Students	{whispering}
755	Tutor	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)
756	Martin	) skin (^^^))
757	Tutor	) skin, cellulitis might also give you a swollen joint which might be misleading but in fact this is the cellulitis OK? and then?
758	Martin	the synovium
759	Tutor	OK before the synovium
760	Martin	the (^^^) subcutaneously
761	Tutor	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?
762	Students	the capsule)
763	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?
764	Student	{lo} (aspirate)
765	Tutor	yeh we have tried to aspirate the joint and somehow um zero point five cc of fluid)
766	Students	) (hh)
767	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, they have sent it to the biochemistry department (.) for what? what might be more (.) most important what might be the most important) (^^^)
768	Martin	) crystals
769	Tutor	uh: for crystals? it's not done by the biochemistry department (.)
770	Martin	{whispering} biochemistry
771	Tutor	sorry not for the biochemistry the mi by the hematology department
772	Students	{whispering}

773	Tutor	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
774	Martin	zero point five cc
775	Tutor	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 OKso 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
776	Martin	mm
777	Tutor	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?
778	Harry	the patient mentioned that she has to undergo uh routine check up of her serum marker to see if there is any disease
779	Tutor	mm

780	Harry	so what should be monitored in the blood test?
781	Tutor	what do you think? (0.2)
782	Harry	I think it should be rheumatoid is it rheumatoid rheumatoid factor?
783	Martin	mm
784	Tutor	mm
785	Harry	or the SR
786	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?
787	Harry	anti (DS)) DNA
788	Tutor	) sorry
789	Harry	anti (DS) DNA
790	Tutor	yes (.) the DNA titre, and:?
791	Harry	and the ESR Chris Chris four four Chris Chris three Chris four
792	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?)

793 Harry) mm

794 Tutor) 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 <u>serious</u> connective tissue) disease

795 Martin) mm

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

797 Martin)mm

798 Tutor)isn’t it? I’m sure all of you are familiar with the fact that false positive (NA) can be present in otherwise normal healthy) individuals

799 Martin) mm

800 Harry) mm

801 Tutor) isn’t it? OK? by the same token I think that anti-Ro positive could be present in otherwise normal healthy individuals as well

802 Harry mm

803 Tutor 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) <u>years</u> 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 <u>all</u> 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?

804 Martin 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 (.)

7 Tutor don't be shy, don't be shy, just go on

8 Eddie yeah we got two cases

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

10 Harry Madam Wu a eighty three years old woman uh presented with three week history of generalised weakness (.) previously uh: Miss uh: Madam Wu wa has been 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 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

36	Harry	uh: because of the weakness of the muscles of her: hand
37	Eric	of her hands
38	Joy	which part of her body is most suffering from the generalised weakness (or is it equally distributed)?
39	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
40	Students	{whispering} (0.2)
41	Gladys	for the lower limb weakness how long have it been start
42	Harry	uh: she she said that it's about three weeks or so
43	Zelda	(so (^^^) quadropod (for three weeks)
44	Harry	(uh for several years
45	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)
46	Harry	toge)ther (0.3)
47	Zelda	) together (0.4)
48	Tutor	oops
49	Zelda	(^^^))
50	Tutor	so: satisfied) with all the history?
51	Zelda	umm no uh so you said she was ADL dependant who is it she lives with and who is she dependant on?
52	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)
53	Zelda	mm mm and you mention about depressive symptoms have you actually assessed her suicidal risk?
54	Harry	(ummm
55	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
56	Zelda	uh but you didn't ask her

57	Becky	um we didn't
58	Zelda	mm mm but did she actually attempt it in the notes
59	Becky	um: (.) not) attempt
60	Harry	) not attempt
61	Zelda	not attempt but) (trying to)
62	Becky	) but trying to kill)
63	Zelda	) was there any plans (or
64	Becky	) (^^^)
65	Zelda	I just thought)
66	Becky	(planning to die (^^^)
67	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
68	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)
69	Students	mm mm (0.5) {whispers}
70	Eric	then do you (think
71	Joy	how) you first you first you first
72	Eric	do you think it's possible in this case the generalise weakness it is due to psychogenic factors
73	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)
74	Eric	mm)
75	Harry	so I think that generalised weakness can be due to hyponatraemia
76	Zelda	mm (right)
77	Martin	what but what hypertensive any hypertensive medication is the patient taking
78	Harry	um: I can recall uh she she was on a HCI and also on a diuretic
79	Martin	aah)
80	Zelda	where is she following up for her hypertension?
81	Harry	I didn't ask because she was quite tired at that time

82	Zelda	oh: so um but uh did she mention the reason for changing her medication a month ago?
83	Harry	uh no
84	Martin	so you mean the new drug has been added to the)
85	Harry	) the dose have been changed
86	Martin	oh change in dose
87	Harry	yes
88	Eric	that means uh still the two drugs for the hypertension
89	Harry	yes
90	Zelda	have you assessed her compliance
91	Harry	no
92	Becky	we think we think that um she is living in an old age home, uh so)
93	Zelda	so) uh people are taking
94	Becky	likely (0.3)
95	Martin	(^^^)
96	Becky	((^^^)
97	Zelda	how about) her hyperlipidaemia is she on any medication
98	Harry	um:: yes yes
99	Zelda	oh on on statins,
100	Harry	I think so (0.4)
101	Zelda	any so how about her past medical history any previous (0.1) stroke or
102	Harry	uh no previous stroke and only hyper tension hyperlipid lipidemia and a minor surgery
103	Zelda	minor surgery?
104	Harry	thyroid for (nodule ^^^)
105	Students	mm mm
106	Harry	(so total) for that (0.3)
107	Martin	(mm
108	Zelda	mm)
109	Eric	did the patient have any uh hypothyroid symptoms?
110	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

111 Zelda but the thyroid nodule was it functioning or diseased

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

113 Zelda so how was it discovered (0.3)

114 Harry mm: I don't know (0.6)

115 Tutor {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 think 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

116 Harry uh yes

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

118 Harry mm

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

120 Harry 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

121 Tutor uh huh

122 Harry and an:d also (.) and that's also systemic and (because

123 Tutor just (hyponatraemia

124 Harry and because (uh the

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

126 Harry um:)

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

128 Harry other causes

129 Tutor what other causes you better try something more common

130 Harry an:d also hypokalaemia

131 Tutor OK potassium

132 Harry yes the potassium level of the patient is low

133 Tutor 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

134 Harry ((^^^)

135 Zelda myasthenia (gravis

136 Tutor no no it’s the neurology) (systemic causes

137 Zelda oh)

138 Students ((^^^)

139 Harry other) causes include hypothyroidism, or adrenal gland insufficiency

140 Tutor now adrenal insufficiency,

141 Harry yes

142 Tutor but could it be the other way round too?

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

144 Tutor how about the thyroid

145 Harry uh: hypothyroidism

146 Tutor hyperthyroidism can also cause generalised (weakness

147 Harry yes)

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

149 Students {whispering}

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

151 Tutor mm mm

152 Harry that is cephalomyelopathy

153 Tutor mm mm)

154 causing) the weakness of the four limbs (.)

155 Tutor mm mm)

156 Harry 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 abou:t 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,

180	Harry	mm yes (.)
181	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)
182	Zelda	uh I asked if she has recent change of the dosage
183	Tutor	Ok how is medications if we uh I mean referring to this case (.) how can medications affect the muscle power
184	Zelda	because she has been taking diuretics so I'm thinking of the elect(rolyte
185	Tutor	OK) so it is uh just electrolyte disturbance, muscle weakness so it actually uh it just induce systemic causes right what else
186	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 (
187	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
188	Harry	(no pain
189	Zelda	(no pain)
190	Tutor	so it's one of the things you need to uh further confirm by examinations (.) what else, (.) still on drugs
191	Kevin	can the patient taking beta blocker have increased (^^^) causing tiredness and present as weakness from ((^^^)
192	Tutor	(beta blocker and tiredness
193	Harry	(^^^) beta blocker)
194	Kevin	tiredness but not weakness
195	Tutor	it's very common to be very tired (with beta blockers) (0.4)
196	Harry	mm
197	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 (.)
198	Tutor	OK: (0.2) we can you need to uh also look at the history here (.)
199	Zelda	mm mm
200	Tutorutor	a bit slightly more common slightly more common OK (0.5) OK is he on is she on T4 replacement,
201	MS	mm mm

202 Tutor 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)

203 Students (mm mm

204 Zelda hyper) thyroid ism,

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

206 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

207 Tutor OK)

208 Gladys they are likely to have diarrhea and uh muscle weakness also ((^^^)

209 Tutor 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

210 Students {whispering}

211 Harry steroid

212 Tutor sure steroid is she taking steroid, (0.2)

213 Harry no

214	Tutor	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 ((^^^)
215	Joy	sta)tins
216	Gladys	a sta)tin
217	Tutor	sure (.) statin
218	Zelda	mm::
219	Tutor	sure you need a low a statin (0.2) OK, is she on statins
220	Harry	uh:: because (
221	Tutor	(do you know what drugs she is on naturally, before (^^^)
222	Harry	because I previously (^^^) lost my notes so I can only remember some of them
223	Tutor	does she have a history of hyperlipidaemia
224	Harry	uh yes so I think so
225	Tutor	(^^^^)
226	Harry	mm
227	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 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,
228	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
229	Harry	I didn’t ask about this
230	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 mmmm

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

268 Tutor her son works in Macau

269 Harry yes

270 Tutor so when did she go to Macau (to live) (0.3)

271 Harry I didn't ask about that

272 Tutor mm mm (.) so

273 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

274 Tutor who uh take her took her to the old age home

275 Harry I assume to be her son

276 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

277 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

278 Tutor so she never went to Macau

279 Harry I didn't ask her whether she went to Macau before

280 Tutor OK anyone clerk the case together with him and can give more history

281 Joy basically he asked about her history and I perform the physical examination umm so

282 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 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:)

296 Harry) I didn’t ask about that

297 Tutor) income

298 Harry I didn’t ask about that (.) I think she is retired,

299 Tutor I know but uh you even after retirement you can still have income

300 Harry yes yes (0.2)

301 Zelda and what was her previous job?

302 Harry sorry

303 Zelda what did she work as (.) before she retired

304 Tutor he mentioned about the factory work

305 Zelda oh OK

306 Tutor factory worker,

307 Harry yeh yes maybe

308 Tutor so the history is not too)

309 Harry because the patient was not very conscious uh not very uh)(^^^)

310 Tutor) (^^^)

311 Harry yes

312 Tutor it depends on your you need to have charm

313 Students {laughter}

314 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 (^^^) I give you a (^^^) 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

315 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

316 Tutor mm mm mm OK (.) thank you for your honesty

317 Zelda {smiling}

318 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

25	Tutor	alcohol … yes,
26	Harry	that’s all I can think of
27	Tutor	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
28	Harry	anti-convulsants
29	Tutor	sorry?
30	Students	anti-convulsants
31	Tutor	anti
32	Students	anti-convulsants
33	Tutor	anti
34	Students	anti-convulsants
35	Tutor	such as?
36	Kevin	phenothrin
37	Tutor	what
38	Kevin	phenothrin
39	Tutor	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
40	Students	) on the central nervous system on the CNS
41	Martin	= on the CNS system
42	Tutor	specifically more
43	Martin	the brain stem
44	Tutor	) brain stem could be on the brain stem but also on the
45	Students	cerebellum
46	Tutor	= 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
47	Martin	central causes by the history of the
48	Tutor	= louder, talk to your

49	Martin	= you mean by the history of the physical examination
50	Tutor	yeh any features
51	Eddie	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
52	Tutor	would you get that in peripheral as well, nausea vomiting would you get those
53	Eddie	uh depending but the pattern
54	Tutor	= you seem to be shaking your head would they get it
55	Student	I think so
56	Tutor	eh you can so your colleague seems to disagree with you
57	Eddie	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 (…)
58	Tutor	= 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
59	Eddie	uh because I thought uh for a CNS tumour uh I mean I mean brain tumour uh usually
60	Tutor	) 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
61	Zelda	) depends on the site
62	Tutor	) depends on the site
63	Zelda	let's say if it's brain stem then you might get the signs
64	Tutor	Right you might get other brain stem signs
65	Zelda	and also but sometimes it can be mixed features let's say (….) you might get mixed central and peripheral

66	Tutor	) 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 wha what other features what's what's a key feature that wo might help you distinguish between peripheral and central from your reading
67	Joy	) I guess
68	Zelda	) I think the central is more persistent but
69	Tutor	) more
70	Zelda	more persistent
71	Tutor	that's right more persistent would you like to expand on that
72	Zelda	so uh so um but I 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
73	Tutor	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
74	Zelda	yes
75	Tutor	you're nodding
76	Joy	I read something in Davidson's about that
77	Tutor	louder yeh
78	Joy	(….)
79	Tutor	) yeh you read in Davidson's which is good yeh (laughter)
80	Joy	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)
81	Tutor	(mm mm) (0.2) do you agree? would you like to expand on that?
82	Chris	ahh and also (.) then for the po peripheral it was usually posturally related
83	Tutor	post what
84	Chris	posturally related because (^^^)
85	Tutor	postural (related)
86	Chris	(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 ^^^

87	Tutor	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
88	Chris	um benign paroxysmal positional vertigo
89	Tutor	alright benign positional vertigo benign positional vertigo right (.) would you like to expand a bit about that?
90	Chris	(umm)
91	Tutor	(perhaps) you can just write that down first benign positional benign positional vertigo (0.1) BPV (0.2) we we'll keep that in mind alright but are there other peripheral causes which might not be postural related
92	Chris	umm like um vest uh uh labyritis
93	Tutor	OK labyrinthitis: and?
94	Chris	vestibular neuritis,
95	Tutor	vestibu vestibular neuronitis (.) is that postural related?
96	Zelda	that was not postural (related)
97	Tutor	(right) so can you use that (.) to distinguish between peripheral and central then? (posture)
98	Zelda	(um) not so
99	Tutor	(not) so yeh: cause you can't use it because (.) the problem with it is that neuronitis is not postural related (0.1) OK? which then comes back to:?
100	Students	*persistency* ^^^^
101	Tutor	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?
102	Martin	mm: I think like (^^^^)
103	Tutor	(louder)
104	Martin	I would check in the examination for nystagmus uh
105	Tutor	sorry?
106	Martin	usually for the central cause they will have uh vertical nystagmus (.) as well as ^^^^
107	Tutor	mm mm
108	Martin	but whereas the for the (.) peripheral cause they usually do not present with vertical nystagmus

109	Tutor	I see OK
110	Martin	but these tend not to be very (.) ss absolute (0.2)
111	Tutor	mm (0.2) *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? (0.2)
112	Harry	the managements (^^^^)
113	Tutor	(louder)
114	Harry	their managements are different
115	Tutor	mm mm (0.2) and how do you mean by that?
116	Harry	mm:: if if central lesion is suspected then the patient should receive a CT scan to localise the lesion (0.1) otherwise if it is peripheral I think the patient can be just given some antihistamines to try if the vertigo can subside or not
117	Martin	I think it should be more related to the symptom (severity)
118	Tutor	(louder)
119	Martin	symptom severity (.) if it's severe we'll we will proceed with the CT scan even if ^^^^ peripheral
120	Tutor	I see (.) so if somebody comes in with not very severe symptoms are you saying that: the: thing is not central
121	Martin	no no I'm just (saying)
122	Zelda	I should say it's a collection of the symptoms because I I just remembered
123		another (cause)
124	Tutor	(OK)
126	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
127	Tutor	so she disagrees with you (.) how can you defend yourself?

128	Martin	mm::: of course we need to get confirmation of signs
129	Tutor	you’ve got to talk to her I’m not I’m not arguing with you you’re arguing with her (laughter)
130	Martin	I think that what she says is correct we have to con 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)
131	Zelda	are you meaning one man two (disease)
132	Tutor	(you) have to talk a bit louder because
133	Zelda	oh sorry
134	Tutor	(you know to be)
135	Zelda	(so so) are you meaning that this is one man having two disease because the peripheral causes are so common then?
136	Martin	(0.2) mm mm (.)
137	Tutor	do you agree?
138	Zelda	umm I think it’s quite rare (but)
139	Tutor	(mm mm)
140	Zelda	it’s always possible (0.5)
141	Tutor	what are you going to (.) withdraw your your thoughts or are you going to defend your turf?
142	Martin	mm::
143	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
144	Vicky	I think the severity is important but I think you also have to consider that the vertigo is very subjective
145	Tutor	mm mm? (0.3) so would you tend to agree with him (0.1) that severity is important?

146	Vicky	I think it's important but I think you still have to look at the other signs as well
147	Tutor	what would be a key feature if one were to determine (0.2) whether it's peripheral or central there were a few things why don't we just write down 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
148	Martin	mm (0.3)
149	Tutor	distinguishing features of central and peripheral (0.2) ((Eddie writes on whiteboard))
150	Zelda	I thought the peripheral one might some of them might be self-limiting)
151	Tutor	)self-(limiting)
152	Zelda	(or they might be coming across) like that like kind of (relapses)
153	Tutor	(mm)
154	Zelda	but for the central one it's it's much more persistent (0.1)
155	Tutor	would you agree with that
156	Student	not sure
157	Tutor	what do you think (0.1)
158	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)
159	Tutor	have you read up
160	Chris	the BBPV (^^^)
161	Tutor	(you have read up)
162	Chris	those will be episodic and be quite long and ((Menieres disease)) those will not be uh very self-limiting but for labyrynthitis um the patient are the lateral cause are we expect to (have)
163	Tutor	so what does B (.) BBPV means
164	Chris	uh it was uh (.) uh I read some there was a problems in the autolymph
165	Tutor	yes
166	Chris	and so that the patients um because the autolymph is inside the (uh)
167	Tutor	(is it) self-limiting
168	Chris	um: I think it was being eh episodic but postural related (but)
169	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)

199	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
200	Chris	umm (.) yes
201	Tutor	right (0.3) what do you think (0.2)
202	Joy	uh: BBPV is not lethal ((laughter))
203	Tutor	is what
204	Joy	it’s not not (not)
205	Tutor	(it’s not) what
206	Joy	it’s not lethal fatal ^^^
207	Tutor	not ohh initially I thought it was not legal but ((laughter)) but not lethal
208	Zelda	but because if for my thought
209	Tutor	yes
210	Zelda	I think about it from the ^^^manoevres
211	Tutor	yeh
212	Zelda	it’s just that you want to test if it's really a BBPV
213	Tutor	yes
214	Zelda	and after you have put the patient’s head down and after five or ten seconds you see the (nystagmus)
215	Tutor	(yes)
216	Zelda	but after thirty seconds it subsides itself
217	Tutor	right
218	Zelda	and then if you expect something central then you expect that it just keeps going on once you have (tested)
219	Tutor	(right)
220	Zelda	the nystagmus
221	Tutor	OK
222	Zelda	and that’s why that’s why I call (it)
223	Tutor	(does) that does that sound logical to you
224	Chris	yeh
225	Tutor	do you want to change your mind
226	Chris	oh oh OK
227	Tutor	sorry

228	Chris	so self-limiting can be used can be ^^^ used for paroxysmal ^^^ type
229	Tutor	yeh: what do you think
230	Chris	I'm not really sure about that
231	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 (.)
232	Martin	mm
233	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
234	Eddie	uh no no no
235	Kevin	I I
236	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
237	Kevin	yes
238	Tutor	so:: and he also had other (.) features didn't he (0.3)
239	Kevin	the: vertigo is uh: constant and sustained (.) but subside after I think (.) uh

subside after one to two day (.) 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 ische(mic)
246 Tutor (no) louder does it
247 Martin (it can be)
248 Tutor make sense
249 Martin it can be some transient ischaemic (^^^)
250 Tutor do you think this was a transient ischaemic 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 ischaemic 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)

265	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
266	Martin	collateral (^^^)
267	Tutor	do you think he's talking nonsense (laughter) or do you think he's uh (0.3)
268	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)
269	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)
270	Tutor	(right)
271	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)
273	Tutor	(correct) isn't it I mean you have to make I mean it 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)
274	Kevin	after hearing (^^^)
275	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)
276	Martin	mm
277	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?
278	Kevin	uh pardon me
279	Tutor	where do you think the lesion is?
280	Kevin	I think the lesion is uh in the posterior circulation

281	Tutor	alright, (0.2)
282	Kevin	(^^^)
283	Tutor	(and let) it stand on (that)
284	Kevin	(^^^)
285	Tutor	(could) you be a bit more more specific?
286	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
287	Tutor	what do you* think (0.2) do you agree with him? (0.3)
288	Kevin	less likely to have that those uh: features
289	Chris	you mean in the anterior circulation
290	Kevin	uh because anterior circ there 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
291	Tutor	what do you think? you clerked him as well didn't you?
292	Harry	I also (have)
293	Tutor	(louder)
294	Harry	I also have similar thoughts with him
295	Tutor	mm mm and so specifically where do you think the lesion could be?
296	Harry	it should be in the (.) brain stem in the region of the: pons
297	Tutor	of the pons (.) why would you think that?
298	Harry	because it is the: region where the brain stem connect to the cerebellum
299	Tutor	just only at the pons? (0.2)
300	Harry	and also the medulla
301	Tutor	mm mm what do you* think? what does anybody think?
302	Joy	mm actually I think that it is affecting the cerebellar (^^^)
303	Tutor	(mm mm)

304	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
305	Tutor	OK:, what do you* think? (.) do you agree?
306	Chris	agree
307	Tutor	yeh mm mm
308	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 (.)
309	Tutor	mm mm
310	Zelda	brain stem wall
311	Tutor	mm mm yeh
312	Zelda	because of the mixed picture picture of ^^^
313	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 (.) a:rea of the brain stem (.) would (.) you think would most fit his symptoms?
314	Martin	mm: ^^^
315	Tutor	you must remember that he's got a whole set of symptoms do you need to revisit those symptoms?
316	Martin	mm
317	Tutor	remember he's got (.)
318	Martin	dysarthria nystagmus)
319	Tutor	) vertigo, he's got) 33.36/ 30.42
320	Martin	) vertigo, hystagmus ^^^)
321	Tutor	) nystagmus, vertigo, dysarthria, slo he's got difficulty swallowing (.)
322	Martin	) ^^^

323	Tutor	) 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)
324	Kevin	medulla
325	Tutor	sorry?
326	Kevin	medulla
327	Tutor	would that fit? what does everybody think?
328	Martin	medulla
329	Tutor	what do <u>you</u> think? (to visiting student)
330	Martin	is there facial numbness?
331	Kevin	yes
332	Students	facial numbness
333	VS	^^^
334	Tutor	I see your English is not as uh (.) what do <u>you</u> think (Students discussing among themselves)
335	Chris	I think it's quite ^^^)
336	Tutor	) so let's just draw the brain stem first wha wha what what what structures are in the brain stem?
337	Martin	midbrain pons medulla)
338	Tutor	) you've got the midbrain (0.2) what else is there apart from midbrain what you've got
339	Martin	^^^ pons) (Students reciting medulla, pons)
340	Tutor	) you've got pons and then medulla right so that's your brain stem
341	Students	(laughter at Eddie's drawing of brainstem on whiteboard))
342	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?
343	Martin	the third nerve is there)
344	Tutor	) third nerve (.) that's right you've got third nerve (.) that controls what?
345	Martin	uh: eye movement)
346	Tutor	) <u>eye</u> movement you've got third nerve eye movement you've got <u>eye</u> movement haven't you (.) what else is there apart from third?
347	Joy	Edinger-Westphal ^^^

348	Tutor	well that’s the third isn’t it I think Edinger-Westphal is third isn’t it? what else? (0.2)
349	Kevin	^^^
350	Tutor	sorry
351	Zelda	fourth nerve)
352	Student	) fourth nerve)
353	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)
354	Martin	)mm
355	Tutor	)and fourth (.) and then in the pons:? in fact it’s one continuous area isn’t it so third fourth and what else?
356	Zelda	trigeminal trigeminal
357	Tutor	sorry?
358	Students	trigeminal
359	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
360	Martin	pons)
361	Students	)^^^
362	Tutor	)similarly isn’t it
363	Zelda	)a little bit lower down
364	Tutor	yes that’s right slightly lower down so perhaps fourth sorry fifth sixth and seventh are over in the pons (.) right?
365	Martin	mm
366	Tutor	(.) and then what happens in medulla? (.)
367	Martin	the vagus nerve
368	Kevin	lower cranial
369	Martin	the lower cranial nerve)
370	Tutor	)sorry
371	Kevin	the lower cranial nerve nucleus nuclei)
372	Martin	the vagus eighth nerve
373	Tutor	so like what?

374	Kevin	like the: fifth nerve
375	Students	eighth
376	Tutor	no no no I'm talking about motor now
377	Zelda	glossopharyngeal
378	Martin	glossopharyngeal hypoglossal
379	Tutor	right so ninth tenth eleventh twelfth isn't it ninth tenth eleventh twelfth all down in the gullet area isn't that right?
380	Martin	hah
381	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 ha:s (0.2) oh don't look at me look at the picture (.) what do you think?
382	Zelda	somewhere in the medulla region)
383	Students	)medulla
384	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?
385	Zelda	cerebellum midbrain)
386	Students	midbrain
387	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?
388	Harry	mm: I can't remember if there is any (.) documentation of the: partial ptosis but I think it was there (0.2)
389	Tutor	did you see it? did you see a bit of partial ptosis?
390	Harry	from the patient I saw it

391	Tutor	you saw it OK alright (.) but did it fit in with any syndrome)
392	Harry	)no
393	Tutor	)that you know
394	Martin	)of partial ptosis
395	Harry	because there are no other eye signs so I don’t think)
396	Tutor	)you didn’t think that it was significant (.) do you think he has partial ptosis now)
397	Harry	)no no
398	Tutor	at least not this morning
399	Harry	no
400	Tutor	so it could be a visual hallucination was it alright OK
401	Harry	yes {Martin muttering}
402	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
403	Students	^^^ reactive
404	Tutor	what would you like to do
405	Kevin	uh:: I would like to ^^^ signs
406	Tutor	such as?
407	Kevin	^^^ anhydrosis
408	Tutor	right so did you check for anhydrosis?
409	Harry	uh no because)
410	Tutor	)why?
411	Harry	both pupils are equal in size
412	Tutor	so you didn’t bother
413	Harry	yeh
414	Tutor	yeh (.) is it a good move (.) do you think?
415	Harry	^^^ backward I should check it
416	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?

417	Martin	mm mm
418	Tutor	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
419	Students	mm yes
420	Tutor	yeh? OK (.) so let's assume that this guy did have partial ptosis
421	Martin	mm
422	Tutor	(.) and let's assume that he did have eh unequal pupils (.) what are you going to do?
423	Harry	umm: check whether the pupils are reactive to light
424	Tutor	mm OK but what else
425	Harry	anhydrosis)
426	Tutor	)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
427	Zelda	chest chest and (impacts)
428	Tutor	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
429	Martin	mm
430	Tutor	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?
431	Harry	well essentially it have the central (thalamic) tract
432	Tutor	right right to where
433	Harry	to:
434	Martin	^^^

435	Tutor	sorry
436	Martin	(spinal ^^^)
437	Tutor	to where
438	Martin	thalamus
439	Tutor	to the thalamus but which part of the body does it enervate?
440	Martin	the contra)
441	Harry	)contralateral
442	Martin	)contralateral side
443	Tutor	mm mm
444	Harry	^^^
445	Tutor	did he have any contralateral sensory deficits?
446	Harry	no)
447	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
448	Martin	)mm
449	Tutor	it's Guillain Barre umm:)
450	Zelda	)I was very lost when I was trying to think about a more likely picture for that patient)
451	Tutor	)yeh right
452	Zelda	)because another variant that he doesn't the weakness is not that (common) for that he doesn't fit into ^^^)

453 Tutor)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 <u>not</u> 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) <u>half</u>-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 we have seen a case of multiple myeloma on Tuesday an:d 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 <u>more</u> 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

11	Tutor	where is the site?
12	Harry	um: involved the bilateral lower limbs
13	Tutor	bilateral lower limb
14	Harry	the bone below the shin area,
15	Tutor	right
16	Harry	as well as the ribs and the hip
17	Tutor	OK as well as the:)
18	Harry	) ribs
19	Tutor	ribs
20	Harry	and the hip
21	Tutor	the hip (.) OK (0.4) what else?
22	Harry	um:: there are no other symptoms
23	Tutor	no other symptoms OK (.) I guess it's the pain that brought her to the hospital
24	Harry	yes
25	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?
26	Joy	fifty)three
27	Zelda	) fifty three
28	Tutor	fifty three (.) what can give rise to chronic pain in uh: in a: lady of fifty three? {lo} what would you consider?
29	Students	{whispering}
30	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

		diagnoses?
31	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,
32	Tutor	) mm mm OK so that's part of the history
33	Zelda	then)
34	Tutor	) so uh what is this
35	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
36	Tutor	right (.) so what do you think? what did you get from the history? is it related to movement
37	Harry	it was not related to any movement um::)
38	Tutor	) mm
39	Harry	) it is constant and the pain also persists during the night time night time
40	Tutor	does she still have pain now? did you ask her?
41	Harry	uh:
42	Tutor	any (wrist) pain you know those sites
43	Harry	uh: she said the pain relieved
44	Tutor	it has been relieved
45	Harry	mm mm
46	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?
47	Harry	mm yes
48	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?
49	Students	left) side
50	Tutor	) left side left hip now OK she has pain in the left hip (.) how does the pain come on? (0.2)

51	Eddie	in fact it also uh involves uh: physical examination
52	Tutor	mm mm
53	Eddie	uh during the exam uh in a resting state the patient uh did not complain of any pain,
54	Tutor	mm mm
55	Eddie	even even the left hip, uh: but on uh: active external rotation,)
56	Tutor	) mm
57	Eddie	uh when the patient did it uh the left hip pain appeared with radiated down to the:) foot
58	Student	) foot
59	Student	) foot
60	Eddie	so (.) does that answer your question?
61	Tutor	and you are I've got to try and remember your names
62	Zelda	um Z
63	Tutor	Z? right (.) start (.) good (.) so what would you say? mechanical or non-mechanical?
64	Zelda	mechanical) because it
65	Tutor	) mechanical good)
66	Zelda	because it (.)
67	Tutor	but what does that mean mechanical? (0.2) is it pain which is elicited or the person complains of it when: when she is in a certain kind of manoevre (.) what information does it give you (0.4)
68	Zelda	um:)
69	Harry	it is likely due to degeneration or due to trauma to that area
70	Tutor	de:generation (.) maybe not necessarily degeneration (0.2) 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 {hi} be:: not in the joints but around the joints because tissue around the joints also moves maybe in the capsule, or in the surrounding auxiliary muscles, in the in the around the joint (0.2) 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 (0.2) 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? (0.2) right (0.2) hip pain (.) anything else? so that’s asking about the symptoms (^^^) information (.) how how would you go about thinking about the differential diagnosis? (0.2) 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?
77	Martin	mm sometimes on the basis of)(^^^)
78	Zelda	) vindicate {laughing})
79	Martin	)pathological system like the inflammatory)
80	Tutor	) I see I hear this word every time I {Students laughing} so what is that word?
81	Martin	vascular)
82	Zelda	) vascular infection inflammation degenerative
83	Tutor	how how do you spell it? I don’t know the word)
84	Students	) V I N
85	Chris	vindicate followed by
86	Tutor	vin: {writing on board}
87	Chris	followed by indicate I N D I
88	Students	C A T E
89	Tutor	E what is that?
90	Students	vascular inflammatory
91	Tutor	inflammatory
92	Chris	neoplastic
93	Students	neoplastic
94	Tutor	neoplastic
95	Zelda	con
96	Students	degenerative
97	Tutor	degeneration, yes
98	Chris	infection
99	Zelda	infection
100	Tutor	infection
101	Martin	clinical
102	Zelda	congenital
103	Tutor	con:genital what else
104	Students	autoimmune
105	Tutor	congenital autoimmune OK well never mind what about the next one?

106	Martin	) trauma
107	Zelda	trauma
108	Tutor	trauma
109	Chris	maih toxins
110	Martin	toxin
111	Chris	or toxin toxin
112	Zelda	or toxin trauma or toxin
113	Tutor	or: toxin OK (0.2)
114	Zelda	environment uh
115	Joy	) endocrine
116	Tutor	endocrine
117	Chris	or metabolic
118	Tutor	or metabolic
119	Zelda	or environment {Cantonese}
120	Chris	(^^^)
121	Tutor	is that where it comes from? who told you that?
122	Zelda	since the PBL era
123	Tutor	PBL?
124	Zelda	in year one)
125	Tutor	) in year one)
126	Zelda	) two
127	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?
128	Student	) influenza
129	Joy	) infection
130	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?
131	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

157	Zelda	inflammation
158	Tutor	ischemia ischemia) is end the end result don't forget
159	Zelda	) infla infla in inflammation?
160	Tutor	inflammation is part of the pathogenic process (.) but what is the basic lesion?
161	Eddie	(local) embolism
162	Tutor	this is atherosclerosis (.) what is the basic ab abnormality in atherosclerosis? (0.2)
163	Zelda	(^^^) {laughing}
164	Joy	cholesterol
165	Zelda	chole
166	Tutor	cholesterol doing something {laughing} what does it do?
167	Zelda	block (^^^)
168	Tutor	in fact uh: it's a vessel uh inside of the vessel the intima and the thelium (.) 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
169	Student	mm
170	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 (.)
171	Martin	mm
172	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

muscle (0.4) {Students whispering} autoimmune of course involves the immune system (.) ok? (.) so a lot of the time it involves inflammation G cells, P cells, and there is also conditions that involves especially B

Zelda cells which (.) (dc) doesn't give rise to significant inflammation for example myasthenia gravis its blocking up the neuromuscular junction))mm

186 Tutor it's the antibody (0.2) OK? for example, uh:: (0.2) hyperthyroidism, (0.2) PSI thyroid stimulating immunoglobulin which it stimulate the thyroid cells, secretes a lot of uh: up-regulate the thryroid gland (.) and then secretes the hormone not not much of inflammation it's not (^^^) to thyroiditis it's different (0.2) alright? (.) and then of course endocrine and metabolic ok (.) 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 (0.3) 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 (0.2) uh:: patient that you see then 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 (0.2) what is possible (0.3) uh 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 (^^^)

191 Zelda HYPERparathyroidism

192 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

194	Tutor	avascular necrosis AVN (0.02) ~possible, (.) ok lets just name it first what else?
195	Zelda	autoarthritis in inflammation that also can be O A in degeneration
196	Tutor	OK O A (.) is it like RA?
197	Zelda	hmm not in the not in these big joints)
198	Tutor	) yes so it's not like
199	Zelda	) I would expect more in the finger joints)
200	Tutor	) finger possible uh likely ones more probable ones anything else? (0.10)
201	Chris	septic arthritis (^^^)
202	Tutor	septic arthritis
203	Joy	one year (0.2) yaat lin
204	Tutor	I dunno)(^^^) infection (^^^)
205	Chris	) (^^^ Cantonese)
206	Tutor	septic arthritis let's put it down first
207	Chris	for one year (^^^ several Students speakings at same time)
208	Tutor	anything else? (0.09)
209	Chris	neoplastic
210	Tutor	(^^^) let's talk about the bone pain first
211	Chris	neoplastic primary or secondary
212	Tutor	good
213	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 (
214	Zelda	trauma but you can get that from the history like
215	Tutor	right, ok so we don't have it in the history?
216	Zelda	mm
217	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?

218	Joy	there should be history for example steroid chronic steroid use (^^^) occupation
219	Tutor	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?
220	Zelda	the septic arthritis because it has been (^^^) for quite a long time and)
221	Tutor	) yes it has been for a long time
222	Zelda	) and she's now feverish
223	Tutor	uh:: when you talk about septic septic arthritis what are what kind of organism are you referring to most of the time?)
224	Students	) (star form)
225	Tutor	{Students muttering} good its pyogenic)
226	Student	pyogenic
227	Tutor	(^^^) 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 (.)
228	Students	{whispering}
229	Tutor	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)

230	Martin	TB joint
231	Tutor	TB joint (.) is it common?
232	Students	um {whispering}
233	Tutor	have you done your orthopaedics? how many TB joints have you seen?
234	Martin	{laughing} TB spine)
235	Zelda	{Students laughing} one TB spine) (0.2)
236	Joy	) TB spine
237	Zelda	one TB kidney
238	Tutor	TB kidney and (^^^)
239	Zelda	not
240	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)
241	Martin	marginal (0.03)
242	Tutor	we haven't gone over the physical examination
243	Chris	hmm
244	Students	{whispering}
245	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)
246	Student	) (^^^)
247	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?
248	Eddie	hmm, I couldn't feel any crepitation over the hip
249	Tutor	ok (0.03) and osteoarthritis can: (0.02) you 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)

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	(^^^)

274		{Tutor and Students laughing})
275	Zelda	) hyper peptic ulcer (.) peptic ulcer
276	Tutor	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?
277	Zelda	mm
278	Tutor	I can assure you I didn't go back to study it for years and years (.) it's (.) these little tricks to remember things (.) so in fact it's important to ask for other symptoms so did you ask it in your history?
279	Harry	mm there)
280	Tutor	) definitely no pain?
281	Harry	no symptoms of hypercalcaemia so no no no pain or no yes no pain and no peptic ulcer history
282	Tutor	mm mm so she doesn't have dypspeptic or dys abdominal pain, any problem with her: passing water? or no womb pain?
283	Harry	no pain or no increase (^^^))
284	Tutor	) and then something, may not be that ser:ious um some other symptoms which are less serious but still can be very troubling, constipation?
285	Student	oh uh
286	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 hyperthroidism 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 othopedic 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)

342 Zelda) structure of tissue {laughs}

343 Tutor 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?

344 Students trauma

345 Tutor 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?

346 Zelda mm

347 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

348 Chris hereditary telangietis (0.2)

349 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

361 Martin) uh huh

362 Tutor 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?

363 Eddie mm as the patient is currently on chemotherapy there is partial hair loss)

364 Tutor) uh what)

365 Zelda) partial

366 Eddie) hair loss hair loss

367 Tutor OK

368 Eddie and the um patient was on a central venous catheter on the right side)

369 Tutor) yes

370 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

371 Tutor mm

372 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,

373 Tutor you said she had a stroke

374 Eddie yes

375 Tutor when was that?

376 Eddie mm?

377	Tutor	the stroke when was that the stroke?
378	Eddie	in 200X four years ago
379	Tutor	(^^^) OK
380	Eddie	yeh basically this uh the uh positive signs (^^^)
381	Tutor	so mainly it's the hip pain
382	Martin	mm
383	Tutor	the other things are the consequence of her immunotherapy (0.2) good (.) did you ask her to stand and walk?
384	Eddie	yes)
385	Tutor	yes
386	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
387	Tutor	hemiplegic gait is it uh: significant? is it is it obvious?
388	Eddie	yeh I would say it's uh obvious
389	Tutor	OK so fine (0.2) so that's a past problem which is not the present more acute problem
390	Eddie	mm
391	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?
392	Zelda	mm no (0.2)
393	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

394	Zelda	{smiling} start with the simple test so we do)
395	Tutor	) good
396	Zelda	) the um for example because there is bleeding then we can do the complete blood picture)
397	Tutor	) good
398	Zelda	) look for the platelet count, and um also uh (heui) what uh and also can do the uh)
399	Martin	) {lo} clotting profile
400	Zelda	) do the calcium level,)
401	Tutor	) calcium level OK
402	Zelda	) mm (0.2) mm do the clotting
403	Tutor	clotting
404	Zelda	clotting profile PT APTT (0.2) mm although those are more likely to present with hemoarthrosis rather than bruises if it's coagulation
405	Tutor	more likely to be:, sorry?
406	Zelda	if it's a coagulating problem then I expect more more thing things like hemoarthrosis rather than {lo}) maybe
407	Tutor	) OK yes,
408	Zelda	and also can check the urine for paraprotein
409	Tutor	urine for paraprotein (0.3) or the:)
410	Zelda	) oh and also do the x-ray of the hip (.) very simple too
411	Tutor	urine for para what kind of paraprotein you you may get to see in the urine?
412	Eddie	immunoglobulin light chain
413	Tutor	and what do we call that (^^^)
414	Students	estrogen protein
415	Zelda	(^^^)
416	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
417	Zelda	mm
418	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?
419	Zelda	mm mm
420	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
421	Martin	one five four?
422	Zelda	two five four (.) normal) {Students whispering in Cantonese} normal
423	Tutor	) yes (0.3) (^^^) equal what seconds
424	Students	{whispering in Cantonese}
425	Student	(gau dim uh APDTT normal))
426	Tutor	)any comment? anyone?
427	Zelda	prolong APT
428	Martin	that is not quite long
429	Students	{discussing together in Cantonese})
430	Tutor	) it is normal in fact this is a little bit shorter this is a little bit shorter)
431	Zelda	) oh
432	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 li:ttle bit low: maybe even within the normal ranges I would think probably
433	Joy	normal
434	Zelda	uh I would like to see)
435	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)
436	Martin	(^^^) bruises {laughs} this is at the presentation?
437	Tutor	at the presentation yes
438	Zelda	{gam} the bruises
439	Tutor	real life case {laughs} I'll try to find) the calcium level
440	Zelda	) how severe (^^^)
441	Students	{Students speaking at same time}

442	Student	how severe the) (^^^)
443	Tutor	) anything else uh::) I can give you the calcium if I can find it
444	Martin	) {Cantonese}
445	Eddie	have the globulin) (^^^)
446	Student	{laughs}
447	Tutor	) calcium levels:
448	Zelda	) {haih la} globulin hah
449	Tutor	calcium levels again normal even adjusted calcium is normal two point three six
450	Zelda	how about the globulin level? (0.2)
451	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?
452	Joy	phosphate)
453	Eddie	) phosphate
454	Tutor	what phosphate? other: inorganic phosphate OK what else?
455	Martin	ALP
456	Tutor	) ALP why?
457	Zelda	) mm
458	Joy	increased (^^^) will have increased ALP
459	Tutor	good (.) let's say this is hyperparathyroidism what happens to ALP? (0.2)
460	Zelda	) increase
461	Student	) hyperparathyroidism?
462	Tutor	yeh hyperparathyroidism
463	Students	increase
464	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)
465	Martin	mm
466	Zelda	mm:
467	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?
468	Zelda	low (.) if the calcium is high phosphate has to be low because they form uh
469	Tutor	mm mm
470	Zelda	(a golden) balance
471	Tutor	what type of hyperparathyroidism are you talking about?
472	Zelda	primary or tertiary
473	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
474	Zelda	mm
475	Tutor	and also the uh: there will be increased phosphaturia the phosphate goes out of the kidney
476	Zelda	) mm
477	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 (0.2) it's easy to pick it up uh 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 (0.2) 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 (0.2) albumin, globulin we'll come to that later (.) so this is the X ray (.) have a look (0.2) you can come round and sit there (0.3) uh I don't have a whole set of X rays (^^^) {noise of movement of furniture throughout this turn and next few turns} take a look see if you find anything (0.16) this is the AP (.) this is the: (^^^) this is a bit rotated (0.2) this kind of view is particularly good for to look for uh fracture in the (^^^) because it shows up clearly (0.3) any: anything? (0.8) {Students whispering} 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 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 which is very clear, (.) goes all the way around but what happens here? (0.3)

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 point 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) {lo} 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)

565 Tutor 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

566 Zelda I remember a (^^^) burns

567 Tutor 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)

568 Student) mm:?

569 Tutor) in the fluid (.) did you try to do that?

570 Students (Cantonese)

571 Tutor very interesting so you measure the the colour of that fluid, at different temperatures and then the 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

572 Student (^^^)

573 Tutor 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?

574 Students dissolve

575 Tutor 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
576	Students	{whispering in Cantonese}
577	Tutor	sorry say this in Chinese (yeung cho daan)
578	Zelda	huh?
579	Tutor	(yeung cho daan)
580	Zelda	oh because (^^^) eggs
581	Tutor	these eggs you put in you boil it for these uh: post partum ladies it's a sweet vinegar with ginger and uh:)
582	Chris	)(Cantonese)
583	Tutor	(^^^) the longer you boil it the harder the egg will become (.) if you go to Taiwan, you have these tihn daan) iron eggs
584	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
585	Zelda	) overboiled
586	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
587	Eddie	uh excuse me I want to ask about uh immunoglobulin assay
588	Tutor	yes?
589	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?
590	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

(0.3) go back and have a look (0.3) we're bound to go over time

591 Joy SP electrophoresis

592 Zelda mm

593 Harry mm

594 Joy SP la)

595 Tutor) two seven two this page or this page

596 Harry (^^^) (0.2)

597 Chris IgG ah (.) {Cantonese}

598 Tutor this picture)(^^^)

599 Students {whispering in Cantonese})

600 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

601 Zelda m mm (0.2)

602 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 sma:ll blue peaks (.) and you see these peaks are sort of quite broad because the molecular si:zes 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 gammglobulin 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

603 Students {discussing together})

604 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)

605 Eddie it's on

606 Tutor dim the lights (0.2) should have turned it on earlier (.) need time to warm up

607 Zelda mm yes (^^^) (0.5)

608 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

609 Student (^^^)

610 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 <u>nor</u>mal, 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 it 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 sca:n, 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}

630	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
631	Chris	) (^^^)
632	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 (0.2) 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 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? (0.2) seventy per cent of plasma cells this is definitely myeloma (.) and where 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 immunoparesis (.) but most multiple myeloma patients do have immunoparesis (0.2) 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?
633	Martin	mm

634	Tutor	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 Ig(D) (.) now why is the total globulin not high? it is becau:se 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?
635	Student	mm mm
636	Tutor	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 (0.2) no problem his donor is a sister a nursing officer in the orthopedic ward in fact {Students laughing} (0.3) {lo} 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 (0.2) 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 a 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 cyt oma 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 yu 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 (0.2) 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 (0.2) 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 myelo:ma, or plasmacytoma (.) sometimes it behaves very:: um: in 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:gammopathy (.) 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 tha:t, 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 for 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 dow:n 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

671	Martin	(^^^)
672	Tutor	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
673	Students	thank you
674	Tutor	(^^^) so far it's illegal

Tutorial 7

Medicine Specialty PBL Session

5th Year Students: Keith, Ron, Trudy, Jan (chair), Sue, Fay and Larry

1	Jan	first of all after yesterday it's about the investigation of uh…
2	Tutor	no it's not that
3	Jan	about the history of this patient
4	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
5	Tutor	you may …. at the side. *Noise of chairs scraping floor as late arriving students come in and sit down*
6	Anne	ok
7	Trudy	so the antihypertensive medication was prescribed about ten years ago by a private doctor and uh for the
8	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?
9	Trudy	He said he has some generalised malaise
10	Tutor	And then how was it diagnosed first?
11	Trudy	He said the private doctor diagnosed it.
12	Tutor	Mm for the first time….
13	Trudy	(nods)
14	Tutor	And he was only given drugs for the hypertension?
15	Trudy	Twenty years after
16	Tutor	I know I know but he he is not given any drug for the rheumatic heart disease
17	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 ….
18	Tutor	When wa the when was he started warfarin?

19	Trudy	He wasn't started ….
20	Tutor	When was he suggested to have had warfarin? ….What information would that how would that information help you?
21	Trudy	Um with the onset of the atrial fibrillation
22	Tutor	Yes not the onset the first detection atrial fibrillation
23	Trudy	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
24	Tutor	So thirty years ago ….are you surprised?
25	Trudy	…. I think it should occur later
26	Tutor	Thirty years ago
27	Trudy	than the onset
28	Tutor	Why? ….
29	Trudy	Maybe ….
30	Tutor	I thought you had all read up on ?? heart disease….
31	Jan	So the rheumatic heart causes damage to the valves and like if there's MS there may be affecting the atrium
32	Tutor	Mm mm
33	Jan	so leading to
34	Tutor	so **how** does it affect the atrium?
35	Jan	Increasing the atrial pressure
36	Tutor	And then?
37	Students	it causes dilatation of the dilatation
38	Tutor	**Yes** dilatation of the left atrium. Is it only mitral stenos..I agree mitral stenosis is most damaging but is it only mitral stenosis?
39	Students	Mitral dilatation will also cause
40	Students	Mitral regurgitation
41	Tutor	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? ….
42	Larry	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
43	Tutor	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 **re**present

the whole history of presenting illness in chronological order and dates mmm

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…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	Electrophoresis
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….

123	Tutor	How do you more or less how can one more or less deduce that it's due to TCM?.... ….
124	Trudy	Because uh it's acute increase
125	Tutor	Yeh ….maybe….and what?
126	Trudy	And also there's some increase in the liver [….]
127	Tutor	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?
128	Trudy	Find out what kind of TCM he's taking
129	Tutor	Possible and what else?....Yeh?
130	Ron	Um there will be showing um acute uh tubular acute and chronic renal failure
131	Tutor	Yeh and then what ….
132	Ron	Mmm….
133	Tutor	…. 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?
134	Keith	Maybe what if we do a toxicology screen possibly find the specific [….]
135	Tutor	OK ….which is probably unlikely for TCM….OK there should be complete recovery ….go on
136	Trudy	and at the same time he was diagnosed with uh drug-induced intra hepatic cholestasis
137	Tutor	yeh
138	Trudy	and uh….
139	Tutor	Which resolved completely again I presumed
140	Trudy	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
141	Keith	So it's only this episode I thought yesterday you said he's got [breathlessness] I'm waiting for the breathlessness to come actually….

142	Trudy	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
143		For what?
144	Trudy	Uh the one is this the chest infection
145	Tutor	and what?
146	Trudy	and one this
147	Tutor	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
148	Trudy	And uh
149	Tutor	So that’s all made up ….
150	Trudy	I asked the patient like in the past he said the shortness of breath started for about five years ago and I asked
151	Tutor	Only five days?
152	Trudy	Five years …uh and I asked him how many episodes he had he usually had and he said he forgot and
153	Tutor	why did you not mention about the breathlessness just now? ….
154		Sorry I should have….um …. he could only remember the two episodes this time [….] he forgot ….
155	Tutor	Can you describe the breathlessness? ….
156	Trudy	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
157	Tutor	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 ….
158	Trudy	I think uh like the the previous uh uh shortness of breath episodes were due to heart failure but
159	Tutor	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		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 ….

219	Tutor	For?
220	Keith	for the for example giving diuretics
221	Tutor	For? For]
222	Students	Ischemic heart disease
223	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
224	Fay	Because there could be hypotension …. And also decrease the contractility of the heart
225	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 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?
226	Jan	Like the shortness of breath
227	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. …..
228	Jan	like the shortness of breath…. like in these five years how how does it progress or is it [….] frequent exacerbation …. Is it worsening ….
229	Trudy	[….] Because I think like the patient said more than ten [….] but I don't remember ….
230	Tutor	Just a moment but in between he was OK?

231	Trudy	He said it got worse and worse especially these days so because
232	Tutor	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
233	Trudy	He said he got worse
234	Tutor	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?
235	Students	yesss
236	Trudy	Discharged at lunchtime]
237	Tutor	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? ….
238	Keith	I can think of drug compliance in this case]
239	Tutor	Yeh?
240	Keith	because the symptoms seems to improve so quickly after admission and then the drug may be still OK]
241	Tutor	[….] and anything else? ….
242	Jan	any concomitant illness like infection?
243	Tutor	Yeh infection he just has infection …. For someone with heart disease actually a very trivial let's say influenza infection probably worsen it ….
244	Trudy	I asked the patient in clinical [….]
245	Tutor	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?
246	Ron	Giant B wave pulsatile liver, and uh uh sys weak systolic murmur because it's covered by [….]

247	Tutor	[….] systolic murmur [….] he's got this what kind of liver has he got?
248	Ron	Enlarged
249	Tutor	What has not got of the three signs of [….]
250	Students	[….] pulsatile liver
251	Tutor	[….]
252	Ron	Cirrhosis
253	Tutor	But one thing that he says that his TI [….] well-controlled now is
254	Larry	[….]
255	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? ….
256	Jan	Only the history part of the clerking?
257	Tutor	Yeh …
258	Jan	I think we've done quite much
259	Tutor	I think you have there are certain aspects I personally am interested …. What about his hypertension? ….
260	Larry	[….] does he present with any complications
261	Students	[….]
262	Tutor	he was actually first diagnosed to have hypertension at what age?
263	Trudy	]Twenty years ago
264	Larry	]Twenty years ago
265	Ron	Look for any secondary causes

266	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?
267	Students	[….]
268	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?
269	Trudy	Around uh five to ten years ago
270	Tutor	So he was already what?
271	Keith	Sixty-seven
272	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?
273	Fay	Twenty-four
274	Tutor	At that time what?
275	Fay	Twenty-four hour [….] to estimate the GFR?
276	Tutor	Yeh OK
277	Fay	(See how bad it is) ….
278	Tutor	Or maybe the cause is the renal failure ….
279	Sue	Could it be]
280	Fay	Hypertension
281	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? …
282	Larry	As the patient has a history of TCM maybe the patient was taking
283	Tutor	Bit I thought we concluded there is that it causes acute [….] necrosis which he should completely recover ….
284	Sue	Could be the drugs for treating gouty arthritis like NSAIDS to?
285	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 antihypertensive 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

316	Keith	Pardon
317	Tutor	and how to measure the heart etc let's just repeat it just ....*[Fay placing film in light box]*
318	Keith	So Professor L said yesterday that
319	Students	*[Laughter]*
320	Keith	(^^^)
321	Tutor	[….] it's not me OK
322	Students	*[Laughter]*
323	Keith	We should separate the heart into uh half right and then we find uh the maximum …..um
324	Larry	distance
325	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
326	Students	(….)
327	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?
328	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
329	Tutor	But what about the cardiac border come on there are mmmultiple abnormalities in this chest X-ray
330	Sue	[….] aortic [….] pulmonary [….]
331	Tutor	yeh
332	Sue	This is the aortic uh knuckle]
333	Keith	Knuckle
334	Tutor	which is quite normal actually it's on the small side for somebody who is seventy seven yeh yeh
335	Sue	This is the left atrium
336	Keith	[…]
337	Tutor	noo [….] it is actually a little bit prominent yeh
338	Keith	and …

339	Tutor	and what
340	Keith	we have the left atrium over here
341	Tutor	Where?
342	Keith	[….]
343	Tutor	Yeh And where else can you see the left atrium
344	Keith	It goes all the way down to this area
345	Tutor	Nuh which where is it?
346	Students	*[Cantonese/ English switching]*
347	Tutor	Speak in English
348	Anne	It's alright
349	Keith	Sorry and then we have the [….]
350	Tutor	So can you please hold on the left ventricle is …the girl ..he seems un a little bit un a little bit ..
351	Fay	This is the left ventricle
352	Tutor	No the left atrium where is it? Left atrium
353	Fay	The left atrium is here and then this is the double atrium double atrium side
354	Tutor	double atrium side it's not …. double atrium side where is double atrium side?
355	Fay	*[getting up to point to film]*[leung go….]
356	Keith	Nido this um
357	Tutor	Yes the right atrium and the left atrium where is
358	Keith	The left atrium
359	Tutor	[addressing chair] why don't you why don't you point it out because
360	Jan	this one um
361	Tutor	Yes one rim is the left atrium the left atrium
362	Jan	This is the left atrium [tracing on film with hand] and this is the right atrium so these two
363	Tutor	It's a double atrium [….] and the left ventricle is where
364	Keith	[*pointing]*
365	Tutor	And the right ventricle?
366		……

367 Fay *[shakes head] [Tutor stands up and goes to light box]*

368 Tutor 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

383	Tutor	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?
384	Fay	[….] wave […]
385	Tutor	right so he's what
386	Sue	he's in atrial fibrillation
387	Tutor	Yeh he's got atrial fibrillation ….left axis deviation anything else ….
388	Sue	left ventricular hypertrophy
389	Tutor	Yes quite marked left vent- on it's own already it's what what how many
390	Students	Si…five and a half
391	Tutor	Ok and this one a little bit. OK ….so he's got left eh left ventricular hypertrophy with what anything else
392	Ron	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
393	Tutor	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 haptogloblin, increase in urobilinogen 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

		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 (.) {ac} uh no no no
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 (.) okay (.) good anything else?
27	Jan	mm: any questions? (0.4)
28	Tutor	any questions?
29	Sue	how 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 complement activating uh pathway and at the end it will form membrane attack complex MAC (.) so if the pathway uh the pathway is very we:ll 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 (^^^)

32	Sue	mm)
33	Jan	) mm (0.2)
34	Tutor	good that’s that’s a very good presentation (.) it’s quite a big subject isn’t it (.) very complicated thank you (0.2) right uh: next uh: paraprotein]
35	Jan	]Trudy (0.2)
36	Trudy	now paraprotein
37	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 immunoglobin (.) 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

66	Tutor	okay so go on and tell us more
67	Sue	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 macroglobulinemia (.) so uh but uh concerning the signs or symptoms it's usually asymptomatic (.) yeah (0.2)
68	Tutor	yes uh it can also reflect underlying lymphoid proliferative disorder uh some B cell neoplasm
69	Sue	uh hum)
70	Tutor	) like uh chronic lymphocytic leukaemia for example
71	Sue	yeh there is a chance that it will progress to lymphocytic leukaemia (.)
72	Tutor	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)
73	Trudy	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
74	Ron	neutrophils

75 Trudy uh

76 Fay that is the neutrophils not lymphocytes (^^^) nucleus

77 Trudy 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 hemoglobulin 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 com 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 spherocytes don't have any (.) usually due to hereditary spherocytosis (.) G6PD-deficiency (.) or uh autoimmune uh haemolytic anaemia and also uh white target cells due to thalassaemia 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 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)

78 Tutor that was a very excellent uh" presentation uh thank you it's a very visual subject isn't it?

79 Trudy mm

80 Tutor can you uh you mentioned the rouleaux what's the uh significance of rouleaux? (0.2)

81 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 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 poikylocytosis (.) (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	hemoglo)bin
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?

104	Sue	alcoholism)
105	Jan	)alcoholism
106	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
107	Sue	mm (0.5)
108	Jan	because less segmented cells less (^^^) cell less (hybrid) segmented cells
109	Tutor	d'you mean the white cells)the neutrophils?
110	Jan	)yeh
111	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)
112	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

which may give rise to a slight anaemia um which is called the anaemia of chronic diseases, and we have to as:k 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 {ac} any pallor in the patient, whether there is {dc} 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,

		uh: how would you: proceed with your approach to the patient?
126	Ron	we 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
127	Tutor	so if fecal occult blood is positive, what would you do then?
128	Ron	um 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 (.)
129	Tutor	and what if those tests are both normal? (0.2)
130	Jan	there may be)bleeding in the small intestine
131	Ron	) (^^^)
132	Jan	which is less common but not detectable by OGD or colonoscopy
133	Tutor	{nodding} what sort of diseases could affect the small intestine?
134	Fay	the AV malformation, it can cause or uh AV displacement can causes bleeding in the small intestine as especially in the elderly (.) people
135	Tutor	AV dysplasia?
136	Fay	mm
137	Tutor	wh where's the commonest uh: commonest location for uh vascular malformation causing iron deficiency in the (^^^)
138	Ron	in the ascending colon)
139	Tutor	) the ascending colon yes (.) how is that diagnosis made?
140	Ron	colonoscopy)
141	Jan	) colonoscopy
142	Tutor	it's made by colonscopy (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)
143	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 an:d total conjugated and unconjugated bilirubin (and) performed at the time (.) so for the parenchymal en:zymes 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 biliary caniliculae 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)lly
144	Tutor	) what about in uh: hereditary spherocytosis (.) what do you see there?
145	Keith	well that would be a pre-hepatic
146	Tutor	pre-hepatic OK
147	Keith	so we would have an excess of unconjugated bilirubin (.)
148	Tutor	{nodding}
149	Keith	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?)
150	Jan	disseminated intravascular) coagulation
151	Keith	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 choleangicarcinoma, 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 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

165 Fay {walking around table to computer} 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 super 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) {moves back to seat} so for esophageal varices it take uh the uh the it will

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 <u>all</u> 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 intra-hepatic 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 itncan 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 intra-hepatic 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 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: uh tapping and uh so we have to do uh give IV abdomen infusion in order not to cause hyperalbuminia 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}

184	Keith	tubular)
185	Fay	) jugular)
186	Keith	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}
187	Tutor	wha what's the name of the antibiotic?
188	Fay	nor norfloxacin)norfloxacin
189	Tutor	) norfloxacin
190	Fay	it's a third generation cyclosporine)
191	Tutor	that's quinalone isn't it it's not it's not cyclo)sporine
192	Fay	it's not,
193	Tutor	no quinalone
194	Jan	(^^^)
195	Fay	quinalone

196	Jan	does it start with (^^^)
197	Tutor	{laughs}
198	Fay	{smiling}
199	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 com) complicate ascites
200	Fay	(^^^) mm)
201	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
202	Fay	yeh
203	Tutor	good thank you very much
204	Sue	thank you
205	Tutor	see you again
206	Fay	{students switch to Cantonese just before Tutor leaves room and appear to be discussing the presentations} (^^^) unconjugated hyperbilirubinaemia (^^^)