Submissions

Dr James GAFFIELD

FAX TRANSMISSION

AMA QUEENSLAND

TO:	Jarrod Cowley Grimmond		
FAX:	3109 9151		
FROM:	Colleen Smyth		
PAGES:	Cover + 3		
DATE:	24 October 2005	**TIME:**	10.00am
SUBJECT:	Dr James Gaffield		

☐[P21] **Urgent** ☐ **For Review** ☐ **Please Comment** ☐ **Please Reply**

Please find herewith a letter from Dr James Gaffield in response to your correspondence of 14 October 2005.

There was difficulty transmitting this via fax on Friday, but the Secretary to the Inquiry should have been received a copy via email.

Please call me on (07) 3872 2268 if there are any difficulties.

Kind regards

Colleen Smyth
Senior Policy Officer

AMA Queensland
88 L'Estrange Terrace, Kelvin Grove 4059 • PO Box 123, Red Hill 4059
Telephone (07) 3872 2222 • Facsimile (07) 3858 4727 • Email amaq@amaq.com.au • Web www.amaq.com.au
ACN 009 660 380 • ABN 67 009 660 380

21 October 2005

Mr Cowley Grimmond
Principal Lawyer
Queensland Public Hospitals Commission of Inquiry
PO Box 13147
George Street Qld 4003

Dear Mr Cowley-Grimmond

Thank you for your letter dated 14 October 2005 addressed to Hall Payne Solicitors, which I received on 18 October 2005.

I have attempted to respond to the main areas I feel need further deliberation by the Commission before making the general or adverse findings under consideration. I believe some of the proposed findings are untrue or the testimony on which they are based is misleading. It is possible to prepare a more detailed response, if the Commission wishes me to, but to do so I would need to seek an extension.

Alternatively, I feel these matters are dealt with adequately in my testimony to the Bundaberg Hospital Commission of Inquiry on Day 45, 19 August 2005. It may be useful for the Commission to refer to this transcript.

The comments that I wish to make on the findings under consideration by the Commission follow, and are numbered as per your letter:

1(d) Patient 26 (P26) did show some signs of clinical improvement during his hospital stay in Bundaberg. To say that he showed no significant signs of clinical improvement is untrue. It would be more appropriate to say he did not show dramatic improvement, but certainly did show some signs of improvement prior to his deterioration in his final time in Bundaberg. He improved in terms of the colour of his leg (less apparent ischemic tissue), and the swelling (reduced). Please refer to my testimony at pp4584 – 4585.

1(f) The patient had a normal white cell count on 28 December, not one that had "increased to an extent that it should have been clear to those caring for him that he was septic.". His white cell count was 10 on 28 December, 10 on 29 December, and rose to 17 on 30 December. A white cell count of 10 is within normal limits. As detailed in my earlier testimony, it was two days later that P26's white cell count rose to a level that indicated he was possibly becoming septic. When Dr Woodruff's testimony from the Bundaberg Hospital Commission of Inquiry is read I think it is clear he meant to say P26 was "clearly septic" on 30 December when he actually stated 28 December:

"...and I would like to draw the Commission's attention to the white cell count, and you'll see that on the - around the 29th of December, it's remained roughly normal from the 26th through to the 29th, but it's grossly abnormal on the 30th, it's risen from 10 to 18 and then goes on to 19 and a half on the 31st. And if one looks also at the nutrafills (sic) or the reactive white cells in the bloodstream, they're becoming elevated on the 28th. So this patient is becoming septic, clearly septic around the 28th, and should not have been allowed to get into that state."

Day 42, 18 August 2005, p 4322

2(a) Obviously, looking at this case retrospectively is quite different to prospectively, as I was forced to do. There was nothing in his clinical presentation to suggest that he needed to be transferred to Brisbane, nor were there clinical guidelines made available to me to suggest that this was the case (i.e. any guidelines from Queensland Health, Medical Board, or the Bundaberg Base Hospital). At the time, I didn't transfer the patient to Brisbane earlier due to Dr Patel's reassurance that the patient's injuries had been fixed appropriately and that there was no reason for concern that he may develop complications or not have undergone appropriate initial surgical management. I transferred P26 to Brisbane when I discovered this not be the case (i.e. when he clinically deteriorated).

2(b) With regard to P26's urine pathology test of 29 December 2004, it is easy to create some particular significance of this test in hindsight, however I maintain that this should not be so, and moreover reject that my action or inaction as a result of it is of any particular relevance, as:

- I was on holidays at the time the urine analysis in question was conducted and was not aware of its existence;
- I do not think it is reasonable to suggest that it is standard practice for a doctor covering someone else's patients to read through every single portion of the patients' charts before providing advice or treatment to them. Rather, it is common to take a briefing from his treating doctor, as I did so in the case of Dr Patel with regard to P26; and
- In any case, had I reviewed the test I would have concluded that the patient had muscle necrosis, which would be expected after a period of transient ischaemia to the leg. It is commonly used to assess the need to prevent renal failure (the muscle products clog up the kidneys and cause acute renal failure). If the level is high, measures to protect the kidneys need to be instituted. Dr Patel had told me that the patient initially had renal failure, but that it had cleared with appropriate treatment. This can be shown in his subsequent normal kidney function tests (blood tests). It did not require ongoing monitoring since the kidney problem had ceased. The test, theoretically, could be used to measure ongoing muscle death, but I don't think this was why it had been ordered by Patel, merely that it was used to judge the need for renal function monitoring. If one thinks that there is ongoing tissue ischaemia (eg, from faulty vascular repair, as was the case here), the appropriate pathway is either to re-

explore tha vascular repair or perform imaging (ultrasound) to look at the vascular repair, not to follow the trend on this urine test.

2(c) I believe the statement that I "failed on 30 December 2004 to identify the patient's increasing white blood cell count in his pathology test dated 30 December 2004" is untrue. I noticed P26's increasing white cell count on 30 December 2004 and I considered potential sources of infection and took measures to resolve them. These measures included removing of the central intravenous line, taking urine and blood cultures, and examining the wound.

These are the major areas I feel it necessary to comment on in the time available.

Yours sincerely

James Gaffield

Dr James Gaffield