

**Orthopaedic Surgeon, Dr B**  
**Consultant Physician, Dr C**  
**A District Health Board**

**A Report by the**  
**Health and Disability Commissioner**

**(Case 04HDC20951)**



Health and Disability Commissioner  
*Te Toihoa Hauora, Hauātanga*



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## Parties involved

Mr A	Consumer/Complainant
Mrs A	Complainant/Consumer's wife
Dr B	Provider/Orthopaedic surgeon
Dr C	Provider/Consultant physician
A District Health Board	Provider
Dr D	General practitioner
Ms E	Consumer's niece/Registered nurse
Dr F	General practitioner
Dr G	General practitioner
Ms H	Quality and Risk Manager, the District Health Board
Dr I	Physician
Dr J	House Surgeon
Dr K	Surgeon
Dr L	Physician

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## Complaint

On 17 December 2004, the Commissioner received a complaint from Mr and Mrs A about the services provided to Mr A by orthopaedic surgeon Dr B, consultant physician Dr C, and a District Health Board. The following issues were identified for investigation:

### Dr B

- *The appropriateness of the care and treatment Dr B provided to Mr A in February and March 2004.*

### Dr C

- *The appropriateness of the care and treatment Dr C provided to Mr A in March 2004.*

### A District Health Board

- *The appropriateness of the treatment and care provided to Mr A by the first public hospital from February to May 2004.*
- *The appropriateness of the treatment and care provided to Mr A by the second public hospital from February to May 2004.*

- *Whether the second public hospital staff provided Mr A with adequate information about the side effects, such as ototoxicity, when he was commenced on gentamicin and penicillin on 10 May 2004.*

An investigation was commenced on 27 April 2005.

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## **Information reviewed**

- Information was received from:
    - Mr A
    - Mrs A
    - Dr B
    - Dr C
    - Ms E
    - Dr F
    - Two friends of Mr A
    - Ms H, Quality and Risk Manager, the District Health Board (DHB).
  - Mr A's clinical records were obtained from the first and second public hospitals.
  - ACC provided a copy of Mr A's Medical Misadventure Unit claim file, which included the independent advice from an infectious diseases specialist, and an orthopaedic specialist.
  - Independent expert advice was obtained from Dr Stewart Mann, Associate Professor of Cardiovascular Medicine, and Dr Garnet Tregonning, orthopaedic surgeon.
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## **Information gathered during investigation**

### ***Overview***

Mr A, aged 58 years, has a history of congenital heart disease. In 1990 he underwent an aortic valve replacement, and in 1997 he had a Star Edwards valve fitted. He underwent further coronary surgery in 2003. Mr A also has diabetes and a history of intermittent lower back pain. On 12 January 2004, Mr A suffered a work-related back injury. When acupuncture and manipulation were ineffective, Mr A consulted his

general practitioner on 27 January. Blood tests were performed and analgesics prescribed. The pain did not settle and Mr A's general condition worsened.

On 7 February 2004, Mr A developed atrial fibrillation (AF) and was admitted to the first public hospital, where this was treated. On 10 February, Mr A was transferred to an orthopaedic ward at the second public hospital for further assessment of his back pain. He was seen by orthopaedic surgeon Dr B, who ordered X-rays, physiotherapy assessment and pain relief. Mr A's condition stabilised and he was discharged on 19 February. However, Mr A remained unwell, developing anaemia, and he was referred to Dr B for further assessment. Dr B saw Mr A at the second public hospital Outpatient Clinic on 4 March.

Mr A's condition deteriorated and, on 11 March 2004, he was admitted to the first public hospital, where he was seen by consultant physician Dr C, for assessment of his anaemia. Upper gastrointestinal bleeding was considered and Mr A was transferred to the second public hospital for a gastroscopy examination. When Dr C learned that the gastroscopy was negative, he arranged for Mr A to have further diagnostic examinations and assessment as an outpatient.

In April 2004, Mr A suffered an episode of melaena (black stools arising from bleeding in the upper gut) and, on 19 April, was admitted to the second public hospital for further investigations. An abdominal CT scan and colonoscopy revealed no cause for the anaemia. Endocarditis (an infection of a heart cavity and valves) was suspected, and blood cultures taken on the day of admission were found to be positive for the bacteria *Streptococcus*. A further set of blood cultures was taken and he was commenced on antibiotics. Mr A's back pain improved and he was discharged on 30 April.

In early May 2004, Mr A presented to the second public hospital with a further episode of melaena, and he was transferred to a city hospital and treated with high-dose penicillin and gentamicin. On 14 June, Mr A began to complain of poor balance, which proved to be the result of a toxic reaction to gentamicin. The penicillin therapy was stopped after six weeks, but Mr A continues to be troubled by ongoing balance problems.

## **Chronology**

### *Back injury — January 2004*

On 12 January 2004, Mr A sustained a work-related back injury. He consulted one of the doctors at his medical centre, who noted "Low back strain". (At about this time Mr A developed a swollen and painful left foot, as a result of a small puncture wound, which he treated with ice packs.) On 21 January, there was minimal improvement in Mr A's back pain and he consulted an acupuncturist, who, as well as providing acupuncture, manipulated Mr A's spine. Mrs A telephoned the medical practice on 23

January, stating that her husband was “still in a lot of pain and the Panadol is not enough”. Mr A was prescribed Paradex and “a few diazepam for the spasm”.

On 27 January, Mr A consulted one of the practice partners, Dr D. Dr D noted:

“Ongoing back pain still. Had acupuncture and manipulations with ‘the russian’ and has been worse since. No bowel or bladder problems.”

Dr D performed a physical examination, noting normal neurological signs and reflexes and minimal muscle spasm. He recorded that Mr A was taking “Paradex and Diclax [anti-inflammatory] with minimal success. Stop these and change to DHC [opioid analgesic].”

On 30 January, Mr A was no better and his wife contacted his usual general practitioner, Dr F, who visited Mr A at home. Mr A’s clinical records show that he was prescribed the analgesic tramadol 50mgs “2 as required for severe pain” on 29 January, and re-prescribed his usual medications of warfarin and aspirin (anticoagulants), and Renitec, Lipex and Sotalol (cardiac medications) on 30 January. Dr F recorded his impression that Mr A had suffered a lumbar disc prolapse. Mr A recalls that he was unable to weight-bear or sit upright, and the analgesia Dr F prescribed was insufficient.

On the morning of 7 February, Mr A suddenly experienced difficulty in breathing, and was sweating. He called out to his niece, Ms E, a registered nurse, who was visiting. Ms E assessed that Mr A’s pulse was not palpable peripherally. A call was made to the health centre, an ambulance was called, and Mr A, who was thought to be in atrial fibrillation, was taken to the first public hospital.

#### *The first public hospital— February 2004*

Mr A was assessed at the first public hospital emergency department at approximately 9.30am on 7 February by a doctor, who recorded a diagnosis of atrial fibrillation and recorded his plan to admit and monitor Mr A. He ordered blood tests and an intravenous digoxin infusion to be commenced when the results of the blood tests were known. The doctor also noted: “Arrange X-ray L [lumbar] spine when better — will need ortho f/up.” Mr A’s previous ECG records were retrieved from the second public hospital for review.

On 9 February, Mr A was reviewed by physician Dr I, who noted that the atrial fibrillation had settled and there were no cardiac complications. Dr I recorded that Mr A was not coping at home with his back pain. He ordered blood tests to check Mr A’s ESR (erythrocyte sedimentation rate — an indication of infection) and noted his plan to refer Mr A to the orthopaedic unit “for spinal management”.

Later that day Mr A was seen by a physiotherapist, who noted:

“Feels that his LBP [low blood pressure] has been slightly better since admission — continues bed exercises as prescribed by private PT [physiotherapist]. Has had no referred symptoms to LL [lower lumbar] or LL weakness with this episode. Prior to admission felt that his overall mobility was slowly improving.”

The physiotherapist noted that Mr A was mobilising “completely independently and safely using a deluxe frame”. Mr A was advised to mobilise every two hours and his pain was managed with Paradex.

In response to the provisional opinion, Mr A disputes that he told the physiotherapist he was improving. Mr A said that he was in severe pain at this time, was unable to weight-bear, and was becoming less mobile, and he considers that the severity of pain he experienced was a contributing factor in the development of his atrial fibrillation.

Mr A stated:

“I was taken off the monitor and drip on Sunday. I understand that [Dr F] phoned [the first public hospital] on the Monday and asked that I be transferred to the orthopaedic ward [at the second public hospital]. This did not happen. [Mrs A] was telephoned [on 9 February] by [the charge nurse] at [the first public hospital], to tell her I was to be discharged home. Even though I could still not weight-bear or sit, and that they intended sending me home by ambulance. [Mrs A] contacted [Dr F] and said that if this happened she intended contacting her MP. I believe that the treatment team in both [the first and second public hospitals] breached my rights ... by making the assumption that I was suffering from mechanical back pain because of my occupation [as a fencing contractor]. I believe this amounted to discrimination on the part of the treatment team and by making [this] assumption they neglected to consider any other differential diagnosis.”

On 10 February, it was noted that Mr A’s atrial fibrillation had resolved and that if he was to continue to be admitted for his spinal problems he “must come under the care of the orthopaedic team”. The transfer was discussed with orthopaedic surgeon Dr B. Arrangements were made to transfer Mr A to Dr B’s care at the second public hospital.

*The second public hospital— February 2004*

Mr A was admitted to the second public hospital on 10 February. House surgeon Dr J noted in his admission assessment that Mr A had experienced “back pain like this many times in his lifetime associated with lifting etc. This time is more severe but otherwise similar.” Dr J recorded that Mr A was not showing any signs of fever, weight loss or radiation of pain. (There was no note during this admission of Mr A reporting a puncture wound to his foot.)

Dr J discussed with Mr and Mrs A his plan to commence Mr A on the anti-inflammatory Voltaren to control his back pain. However, Mrs A pointed out that this medication was contraindicated for patients taking warfarin. Dr J prescribed an alternative anti-inflammatory, Vioxx, for Mr A. (*The New Ethicals Catalogue*, November 2001, states that Vioxx is to be used with “[c]aution with concomitant warfarin (monitor)”.)

On 10 February, Mr A’s spinal X-rays were repeated and showed multiple areas of degeneration throughout his spine.

On 12 February, Mr A was seen by Dr B, who noted:

“He got subjectively improvement yesterday and he is lying in bed for the whole day yesterday. I encourage him to do short distance walking today and we will continue to observe his progress.”

Mr A stated that during this admission his ESR was intermittently elevated and his temperature ranged between 37°C and 38°C. The medical records show that his temperature peaked above 37.5°C four times, but the remainder of the time it was within normal limits. On 10 February, his ESR was recorded at 68mm/hr (normal being between 1–10mm/hr); two days later the ESR had dropped to 40mm/hr.

Mr A recalled that on 11 February his wife suggested that he would benefit from physiotherapy, but this was declined. The clinical notes do not record this request. On 12 February, Mrs A requested a hospital bed for her husband to use at home. The occupational therapist explained to Mrs A (and recorded the conversation) that, as her husband’s condition was not one requiring long-term total- nursing care, this was not possible. Alternative measures were discussed, such as a new bed/mattress and hand rails in the shower.

Dr B reviewed Mr A on Saturday, 14 February, and noted that he was not confident to walk independently, and that bed-rest was to continue over the weekend with a further review planned for Monday, 16 February.

On 15 February, Mrs A asked to speak with Dr B about Mr A’s “recent admission to [the first public hospital] with AF. Wife feels that further investigation into why this occurred — needs to be looked into”.

Dr B reviewed Mr A on 16 February, noting: “[Mr A’s] back still aches and he is not very confident to go home yet. I let him stay here until he is confident to go.” The following day Mr A stated that he was prepared to be discharged in two days. He was seen by the occupational therapist with a view to discharge.



On 19 February, Mr A was discharged with an appointment to see Dr B at the first public hospital outpatient clinic in four weeks. The discharge letter sent to Dr F by Dr B regarding Mr A's 10 February admission advised:

“Admitted 10.02.04 with mechanical back pain. [Mr A] was referred from [the first public hospital] with lower back pain. He had been there for an episode of atrial fibrillation and had been recently started on Sotalol. [Mr A] said he had had back pain like this many times in his lifetime, associated with lifting. This time it was more severe but felt similar in character and position. There was no fever, weight loss, radiation. It started this time with some heavy lifting. Then he went to an ‘acupuncturist’ who manipulated his back, significantly worsening the pain. When examined he had negative straight leg raise tests and had no neurological deficit with normal reflexes. He was admitted for bed rest and analgesia. ESR on admission was raised at 68. Plasma electrophoresis was normal, and urine was sent for Bence Jones proteins — result not yet returned. With bed-rest and analgesia the pain significantly improved so that by 19.02.04 he was comfortable and confident enough for discharge home.

The cause for the raised ESR was not found during his admission, though his aortic valve replacement was considered a possible cause. Pain was thought to be of a mechanical origin and its improvement with bed-rest supports this. The ESR repeated closer to discharge was 40. Orthopaedic follow-up clinic is 18.03.04 in [the first public hospital].”

A friend of Mr A stated that he had observed the “continuous deterioration” in Mr A's health during February 2004. He said:

“On about 17/02/04 I was asked by [Mrs A] to pick [Mr A] up from [the second public hospital] and transfer him to his home. ... When I arrived at [the second public hospital] I was absolutely shocked to see the state in which [Mr A] was discharged in. ... I was sure I was taking him home to die.”

Mr A stated that he and his family were very concerned about his condition at discharge, as he had lost 12kg in weight and had become anaemic. He recalled that these findings were confirmed by Dr F, who arranged for him to be reviewed by Dr B on 4 March.

*GP management — February 2004*

Dr F's clinical record for 19 February states:

“Now out of hospital. Discharged with diagnosis of mechanical back pain, certainly loss of EHL [test for spinal nerve involvement] weakness now and can weight-bear. Coping with the pain although on Tramal and possibly side effects from this. A stone of weight loss. The wife says looks pale.

OE [On examination]: Clinically Slump Test pain free lying down and even sitting now. Pain mainly changing movements, translumbar and no referral down legs. The only outstanding fact is ESR of 68 which may be related to aortic valve.

NOTE: Despite an ESR of 68, protein electrophoresis and Bence-Jones proteins done in [the second public hospital] normal.”

Mr A stated that he was taking Vioxx daily and Tramal and Paradex every four hours for pain, but his condition was not improving. He said that he felt tired, lethargic and had a “fuzzy” head, but, although he still needed a walker to mobilise, his mobility was better.

Another friend of Mr A stated that she visited Mr A at home on 22 February 2004. She recalled:

“I called to say hello and drop off my personal blood pressure monitoring device. I was shocked to see how physically ill [Mr A] looked when I entered his room, his skin colour was pallid and grey with two bright red patches over his cheekbones. His eyes looked sunken & dull and he had lost a considerable amount of weight.”

On 1 March, Dr F made a home visit and took blood for testing. The following day Mrs A contacted the medical centre to obtain the results of the blood tests. Mr A recalled that his haemoglobin had dropped from 153 to 112G/L (normal is 130–180G/L) and he had lost more weight. Mrs A was so concerned about her husband’s condition that she telephoned the second public hospital to speak with Dr B about her concerns. Dr B agreed to see Mr A earlier than planned, and made an appointment for him for 4 March at the Outpatient Clinic.

Dr B stated:

“On the 4<sup>th</sup> March, the patient’s wife, the patient and myself had a lengthy discussion on this day and came to the conclusion that an MRI [magnetic resonance imaging] would be arranged as soon as possible to exclude any occult pathology accountable for the persistent low back pain. He is on warfarin and his haemoglobin was observed to have dropped from 150 to 114 in one month’s time and I have arranged patient to be admitted to [the first public hospital] again to arrange MRI and also for medical review.”

Ms E stated that she returned on 4 March 2004 to visit her uncle. She stated:

“I was shocked by [Mr A’s] deteriorating condition. He was still bedridden, he had lost a considerable amount of weight and looked pale and grey. This was clear to me that [Mr A’s] problem wasn’t a mechanical back problem.”

Dr F's records show that he was concerned that Mr A's haemoglobin had reduced dramatically and that he was considering a gastrointestinal bleed caused by the Vioxx and warfarin. On 5 March, Dr F arranged for Mr A to be admitted to the first public hospital for an urgent spinal MRI and assessment of his falling haemoglobin level.

*The first public hospital— March 2004*

Mr A was admitted to the first public hospital on 11 March. The admitting note records that Mr A had been admitted for investigation of recent weight loss, and had a history of back pain and melaena stools. An intravenous line was inserted and he was placed on "nil per mouth".

On 12 March, Mr A was seen by physician Dr C, who requested a faecal specimen for laboratory analysis before proceeding to gastroscopy. The intravenous line was discontinued and Mr A was commenced on morphine elixir for pain.

The following day, Mr A reported that he had "severe loss of power" in his legs when using the walking-frame, and had to take his weight on his arms. It was noted that he had no pain while on bed-rest. On 14 March, the nursing notes record that Mrs A stated that she wanted to be present at ward-round the next day to talk to the medical staff about her husband's treatment.

On 15 March, Dr C noted:

- Melaena probably 2° to Vioxx
- No further melaena
- am Losec
- Gastroscopy (Wt loss/anaemia)
- Back pain has returned since cessation of Vioxx. Under [Dr B].
- Try Tramadol — if problems
- Change to regular panadol
- to L A [long acting] morphine.
- Weight loss 15kg / easy ?
- AV [aortic replacement] / am warfarin
- NIDD [Non Insulin Dependent Diabetic]."

On 18 March, Mr A was transported to the second public hospital for a gastroscopy examination. The following day, Dr C noted that the gastroscopy examination was normal. A second request for an MRI was sent as there was no record that the first request (made by Dr F on 5 March) had been received. The clinical records note Mrs A's "dissatisfaction of service — complaints procedure discussed".

In response to the provisional opinion, Mr A disputes that his wife was given information about the DHB's complaints procedure at this time.

Dr C advised me that Mr A transferred back under his care and was seen on 19 March 2004.

On 19 March, the nursing notes record that Mr A was referred to a dietician as “pt & wife concerned about weight loss”, and that Mrs A requested that her husband be reviewed for depression. A further physiotherapy referral was also made as “pt & wife want more input from the physio eg passive exercises while lying in bed”.

Mr A stated, in response to the provisional opinion, that his wife did not request that he be reviewed for depression. He said that he was visited by his wife’s friend. The visit was prompted by a general concern among Mrs A’s colleagues about his deteriorating condition and the view that nothing was being done to address the situation.

On 21 March, the nursing notes record:

“Please follow up MRI scan tomorrow as [Mrs A] stated she heard it was scheduled on [5 March 2004], who is not impressed if this is so, wants it done urgently.”

The following day Mrs A escorted her husband in the ambulance for his MRI.

The pathologist reporting Mr A’s MRI, noted:

“As the bone marrow signal alteration is so well circumscribed around what is clearly a focal herniation and there is no evidence of any inflammatory phlegmon [tissue] anteriorly or posteriorly or in the disk this would appear to suggest that these changes are not due to infection. There is no clear evidence to suggest clear osteomyelitis or discitis.

I note the patient does have an elevated ESR. Is there any other potential infective focus?

If there is no cause for the elevated ESR and the pain continues it may be worth treating the patient conservatively (as there is no operative lesion here) and repeating the MRI.”

On 22 March, the MRI results were sent to Dr B for review. A physiotherapist assessed Mr A and documented a treatment plan of ultrasound, heat and mobilisation. An occupational therapist also reviewed Mr A.

On 23 March, Mrs A requested a “patient note release form” so that the results of her husband’s MRI could be sent to an orthopaedic surgeon for a second opinion. (Mrs A formalised this request in writing on 29 March.)

On 24 March, Dr C saw Mr A and noted that Mr A had a “low grade temperature”. He ordered four-hourly temperature recordings. The temperature recordings show that Mr A’s temperature ranged between 36.6°C and 37.8°C.

Dr B met Mr A on 24 March and reviewed his MRI film and report. He assessed Mr A’s back pain and found Mr A to be comfortable with a spinal corset and analgesics, and that there was no nerve involvement. Dr B stated:

“I clearly explained to him that the MRI shows degenerative changes at multiple levels and no sign of infection or malignancy. It is clearly documented in my notes that [Mr A] was able to fully understand my explanation and I was accompanied by the charge nurse when I saw [Mr A] in [the first public hospital]. The documentation from the nursing notes showed that [Mr A] was ‘alert and orientated’. He was advised to continue physiotherapy, using his corset when necessary and further review was arranged for 4 weeks in [the] clinic.

I received a telephone call via my mobile phone shortly after I interviewed [Mr A] in [the first public hospital] ward on the same day. [Mrs A] asked me to explain the MRI findings over again to her. I explained to her that I had already explained the full report to [Mr A] not long ago and I am sure that he fully understood my explanation. However, on strong request from [Mrs A], I emphasised the most important point on the report to her again over the phone.

This is the last encounter of myself with [Mr A]. Appointment was given but the follow-up was not attended.”

In response to the provisional opinion, Mr A stated that Mrs A did not call Dr B on his mobile phone. Mrs A called the first public hospital, and the call was put through to him by the hospital switchboard. Mrs A did not expect to meet with Dr B that day, but had hoped that he would make time to discuss with her and Mr A the expected long-term outcome.

On 26 March, the records show that there was a family group meeting. Dr C, the charge nurse, the physiotherapist, the social worker, Mrs A and her support person, met to plan Mr A’s discharge. Mr A stated that Dr B did not attend this meeting. The record of the meeting noted:

“[Mrs A] asked about plan for colonoscopy or falling haemoglobin. [Dr C] explained not expected to find anything as melaena indicates upper GI [gastrointestinal] bleed. [Dr B] has explained wear and tear to back, follow up with OP [Outpatient Clinic]. Will await iron studies/B<sub>12</sub> etc (also await chronic inflammation markers). Need to discuss package of care with ACC and have requested reassessment. Concerns from [Mr A] and [Mrs A] about ongoing physio — we will need to confirm, personal care and supervision/meal prep/transport etc

can be provided by ACC — [the social worker] to follow up. Plan dx [discharge] tomorrow with support to go in on Monday.”

Dr C advised that he would review [Mr A] at the Outpatient Clinic following his discharge and after he had had further blood tests for a complete blood count, ESR, additional inflammatory markers, B<sub>12</sub> and folic acid levels, and had had a colonoscopy. However, [Dr C] did not see [Mr A] again after his discharge on 27 March.

On 30 March, the first public hospital sent details of Mr A’s admission and discharge to the medical centre. (Dr F was on holiday at this time.) This included reports from the surgeon Dr K who performed the gastroscopy, and Dr B regarding the MRI results.

*GP follow-up — April 2004*

On 15 April, the medical centre general practitioner Dr G noted that Mr A’s haemoglobin had dropped from 103 to 90 G/L over a week, and noted his plan to urgently admit Mr A to the first public hospital for review. On 19 April, Dr G recorded a conversation he had with Mrs A. Mrs A had taken her husband to the first public hospital and he was declined admission. She said that she had been ringing the second public hospital and “finally managed to persuade them to do a colonoscopy next Tuesday under [Dr K]. ... I will visit [Mr A] later this morning then will discuss with [the cardiologist].” Dr G recorded his visit to Mr A at home later that day, noting:

“Heart sounds described as before middle last year, prosthetic with ejection systolic murmur AP [anterior/posterior] and L [lateral] sternal edge. Grade 3 out of 6 therefore assume nothing new but in view of high ESR and low-grade temperature that has now definitely been recorded several times over the last month we need to exclude the possibility of bacterial endocarditis and in view of ongoing melaena and risk of rapid AF suggest admit. Not at all keen to go to [the first public hospital] because of bad experience therefore will admit to [the second public hospital].”

*The second public hospital— April 2004*

A medical registrar admitted Mr A to the second public hospital on 19 April. He recorded that urgent bloods were taken for culture, and that the colonoscopy booking for Tuesday, 20 April, was confirmed. The following day, Mr A was reviewed by physician Dr L. The plan was:

“Try and bring forward colonoscopy ([Dr K])  
Echo [echocardiogram]  
Blood cultures X 2 more jets  
USS abdo [abdominal ultrasound scan], BA enema  
Leave off fentin  
2 units of blood  
remains on warfarin currently  
Physio referral please

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Repeat LFTs/Ca<sup>2+</sup>/ESR/CRP [blood tests].”

At 2.40pm, the ward was notified that Mr A’s blood cultures were positive and “looks like strep [Streptococcus]”. Further bloods were requested and he was commenced on Augmentin and was given a blood transfusion.

The following day, another doctor noted that the sensitivities for the blood cultures needed to be followed up, but Mr A was to continue with the Augmentin “for moment”.

On 21 April, Mr A had a trans-thoracic echocardiogram (TTE — heart examination). However, the TTE did not reveal any sign of infection in Mr A’s heart. The medical note regarding the results of the TTE suggested that Mr A have a trans-oesophageal echocardiogram (TOE) to “exclude endocarditis”.

On 23 April, Dr L recorded that he discussed these findings with a consultant cardiologist. He noted:

“USS [ultrasound scan] damage likely old.  
Features of endocarditis usually include organ infarcts.  
No need for a TOE at present, await culture sensitivity.”

The treatment plan was discussed with Mr A, as was the provisional diagnosis at that time, which was “not endocarditis”. Mr A had a colonoscopy performed on the afternoon of 23 April, which reported that “[t]here was no evidence of recent or active haemorrhage or any other mischief”. On 28 April, Mr A had an abdominal CT scan in an attempt to identify the cause of his gastrointestinal bleeding. The scan showed no cause for the bleeding, although there was a calcified lesion noted on his right kidney, which was reported as “small and of dubious significance. If a simple cyst can be confirmed on ultrasound then no further investigation is likely to be necessary.”

Mr A was treated with a short course of amoxycillin, which led to a significant improvement in his back pain. Mr A was discharged on 30 April to be followed up in two weeks. On discharge he was given a letter that stated that if his condition deteriorated or if he needed admission he was to go straight to the second public hospital.

*The second public hospital— May 2004*

On 6 May, Mr A presented to the second public hospital following a further episode of melaena. He was seen by Dr L, who ordered blood tests to monitor Mr A’s haemoglobin. Mr A’s temperature was recorded at 37.4°C. A gastroscopy examination found no cause for Mr A’s gastrointestinal bleeding. Mr A was informed of the findings.

On 8 May, further blood samples were taken for culture. Mr A was seen the following day by another doctor (at the request of Dr K). The doctor noted that Mr A did not present with additional heart sounds, and recorded “? Source of sepsis ? cause of hypoanaemia”.

On 9 May, the blood culture results showed “gram +ve [positive] bacilli resembling streptococcus”. Repeat blood tests were ordered. Mr A was commenced on intravenous penicillin and gentamicin. Mr A stated:

“Why weren’t [Mrs A] and myself informed of the possible devastating long term risks associated with gentamicin? I had no idea that I had been started on gentamicin in [the second public hospital] until I read this in my discharge summary. I still cannot believe that the medical profession would put you on an antibiotic with the potential to cause these side effects without informing you. Why in fact was I put on gentamicin at all? Was this because it was the cheapest option?”

A TOE was arranged for Mr A to be performed at a city hospital.

On 11 May, a house surgeon noted that he discussed Mr A’s case with a consultant cardiologist at the city hospital who had “concerns that IE [infective endocarditis] is likely”, and expressed concern about Mr A’s gastrointestinal bleeding. The cardiologist advised that he would arrange for Mr A’s transfer for a TOE. The house surgeon noted that he informed Mr A of “this progress”.

#### *The City Hospital — May 2004*

On 12 May, Mr A was transferred to the city hospital for further management. He was treated with high-dose penicillin. It was planned that he would stay on this medication for six weeks. His creatinine and gentamicin levels were monitored regularly, and the doses of gentamicin adjusted according to the city hospital protocol. Audiometry was performed regularly to check for hearing loss. Mr A began to improve, his back pain eased and he became more mobile.

#### *Gentamicin toxicity — June 2004*

On 14 June, Mr A began to complain of poor balance. A CT scan was performed along with a detailed clinical assessment and repeat audiology. No cause was found. A diagnosis of gentamicin toxicity was seriously considered but the gentamicin was continued according to the protocol. The penicillin therapy was stopped after six weeks.

Following his discharge, Mr A was troubled by ongoing balance problems and bouncing vision each time he moved his head. This has subsequently been diagnosed as vestibular damage secondary to gentamicin toxicity.



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**Additional information***Mr A*

Mr A stated:

“Initially both [the public hospitals] treated me as a back pain patient only, regardless of my deteriorating physical condition. The concerns of my family who knew I was acutely unwell, were not listened to by staff. Why, given my cardiac history, my raised ESR and CRP [C-reactive protein (inflammatory marker)] levels, pyrexia, loss of appetite and weight loss and the fact that my family and friends could clearly see that I was seriously ill, was [the infection] missed by both the orthopaedic unit in [the second public hospital] and all the staff at [the first public hospital] and just passed it off as a mechanical back, thus putting my life at risk and my recovery back months. [Dr G], who knew my medical history, was able to diagnose my condition without even having to see me. He asked [Mrs A], when she saw him on 19/04/04 had I been on any antibiotics, because I had an infection of some sort. Which I hadn’t? The point I want to make is that I was not treated holistically — I was seen as a back pain patient only, and treated accordingly with the stigma that is associated with back pain patients.

...

Why were the concerns of my family not listened to and had they been, the fact that I had bacterial Strep infection would have been picked up in February? Would I have needed the same dose and duration of gentamicin to treat my endocarditis, had I been diagnosed earlier? I may not now have ototoxicity. Why was I only treated with Augmentin for 10 days? According to the medical staff at [the second public hospital] this was the antibiotic of choice. Maybe if I had been kept on this longer, I may not have needed further treatment. Why did they not ensure that the infection had cleared up before they stopped the antibiotic treatment and discharged me. Why was I refused admission to [the first public hospital] on 15-4-04?”

*Dr B*

Dr B stated:

“I had a definite plan to investigate further on [Mr A’s] medical condition and review his back pain when I arranged for him to return to Orthopaedic Unit of [the second public hospital] on 8 March 2004. His failure to turn up upset my plan.

On review of the patient’s orthopaedic treatment, I think I had made a timely, prompt, appropriate orthopaedic response to patient’s needs with respect to his low back pain. We also had paid attention to and monitored [Mr A’s] medical conditions when the patient stayed in our unit. Communication was made with GP

upon discharge of patient. My enthusiasm for care was shown by putting forward the follow-up time to the next clinic on 4 March 2004, despite an already full clinic, upon acknowledge[ment] of the problem and immediate referral for hospital care on the same day. This discussion was lengthy, thorough, clear and had [Mrs A] actively involved.

Throughout the orthopaedic care process, [Mr A] and his family were adequately informed about progress and future outlook of the condition. The opinion from the patient's family was properly considered and well respected. This was well exemplified by the long discussion at the [the first public hospital] Orthopaedic Clinic on 4 March 2004.

Judging from the radiological and clinical information available to me, permanent damage to the back by streptococcal infection was not substantiated."

In response to my provisional opinion, Mr A explained that Mrs A telephoned the Orthopaedic Unit on 8 and 9 March but the Unit Manager advised that there were no beds and that she would follow it up with Dr B. Mr A advised me that when he was admitted to the first public hospital on 11 March, he gave Dr B's admission letter to ED staff so Dr B would know he had been re-admitted.

*Dr C*

Dr C stated:

"[Mr A] was admitted under my care on 11/03/04 with abdominal pain and anaemia.

I first saw him on 12/03/04. ... My clinical assessment of him at that time was that he had had an upper gastro-intestinal bleed, most likely secondary to erosions or peptic ulceration as a consequence of the use of Vioxx and Aspirin. However, the weight loss and easy satiety also raised the possibility of more significant upper gastro-intestinal pathology being present. Arrangements were made for him to undergo upper gastro-intestinal endoscopy.

At the time of my first consultation, I obtained a further history of chronic back pain for which he was under [Dr B], orthopaedic surgeon, and was awaiting an MRI of the vertebral column. He had also undergone aortic valve replacement for which he was on Aspirin and warfarin. INR [international normalisation ratio — monitors warfarin levels] at the time of admission was therapeutic at 2.1.

On 15/03/04 [Mrs A] suggested to me on my ward round that I speak with [Mr A's] cardiologist, regarding the management of his warfarin. I indicated that this was not necessary. The reasons for this being that his warfarin control was good and that there was no plan to take him off his warfarin. [Mr A] was transferred to [the second public hospital] for endoscopy.

He returned back under my care and was seen again on 19/03/04. Upper GI endoscopy had been unremarkable and it was suggested by [a surgeon], that at some stage colonoscopy be undertaken. On that visit it was apparent that he was having a lot of back [pain] despite the use of a combination of Tramadol and Panadol and, in view of this, long acting morphine was added to the analgesic regimen. He subsequently underwent MRI of the lumbrosacral spine on 22/03/04 and was followed up by [Dr B] after this.

On 26/03/04 I met with [Mrs A] and this meeting was attended by a number of allied health staff. [Mrs A] was concerned about his haemoglobin, which at the time was stable at 96gm/L. I felt that it was appropriate for him to be discharged from the hospital and to be followed up at Outpatients with repeat blood tests to include a complete blood count, ESR, additional inflammatory markers, B<sub>12</sub> and folic acid levels. I considered it more appropriate to review him at Outpatients after the colonoscopy had been performed. However, he was not seen again by myself after his discharge on 27/03/04.”

#### ACC

Mr A laid a claim with the ACC Medical Misadventure Unit. On 4 April 2005, ACC advised Mr A that his claim had been accepted as medical mishap based on the independent advice provided to ACC by an infectious diseases specialist, Dr Stephen Chambers, and an orthopaedic specialist, Dr William Taine.

Dr Chambers advised ACC:

“It is likely that [Mr A] had osteomyelitis of the spine or paraspinal structures and he probably had endocarditis as well. The organism causing this infection is a very unusual one but is well recognised as a cause of endocarditis and has been reported to cause other infections occasionally. It is part of the normal flora of [the] upper respiratory tract and gastrointestinal tracts of humans and is not regarded as an environmental organism. It is very unlikely to have entered the body through the foot.

... MRI scans have a sensitivity of about 95% if the infection was present for more than two weeks and changes become more obvious with time. In this case the MRI scan did not show convincing changes more than 3 months after the onset of symptoms which is most unusual. It is thus easy to see why the clinicians were misled and the back pain attributed to the more obvious pathology present in the spine.

Despite the scan appearance there was convincing evidence that an inflammatory process was present and that infection would be a likely cause. The evidence includes repeated observations that [Mr A] had an intermittently elevated body temperature, an elevated ESR, unexplained weight loss, and there was an

unexplained anaemia that is a common accompaniment of long standing infections. The question of infections elsewhere was raised in the report of the MRI scan. The prompt relief of pain following administration of antibiotics along with the isolation of the streptococcus from the blood is strong circumstantial evidence that there was infection present in the spine.

In retrospect it is easy to see that the features of chronic infection were present. However, there was a major distracting feature and that is the history of melaena. ... Because melaena may be caused by malignant disease, ulceration and other severe conditions that may also cause weight loss it is essential that this should be investigated thoroughly. It is understandable that this should be seen as a major need for investigation and explanation given that the back pain was apparently explained.

It is highly unlikely that the short course of Augmentin therapy in any way compromised [Mr A's] subsequent treatment. Once the diagnosis of endocarditis was made the treatment would have consisted of penicillin and Gentamicin and he would have been given 6 weeks treatment irrespective of the preceding short course of Augmentin."

Dr Taine advised ACC:

"[Mr A] presented with low back pain which only in retrospect can be considered to contain some pointers towards an underlying disease process other than a mechanical disorder. However, his initial history on admission to [the second public hospital] in February 2004 included [the] observation that he had had previous episodes of back pain similar in nature to that currently experienced. He also gave a history of a reasonably defined event serving to precipitate his pain, both of these factors would serve to direct attention towards a mechanical disorder rather than an infectious etiology. In spite of this, he underwent a series of blood tests, particularly when note was made of a raised ESR. However, while moderately raised, it should be noted that he had other co-morbidities which could be considered possible contributing factors ie. the heart valve replacement with anticoagulant therapy, and diabetes. With X-rays and other blood tests being satisfactory, and with no consistent pattern of temperature rise, particularly in the light of documentation of improvement over the period of admission, it seems reasonable to support a diagnosis at that time of mechanical back pain. ...

It subsequently became clear with a persistently falling haemoglobin, raised ESR, and several documented low grade rises in temperature, in the presence of an artificial heart valve, that the possibility of bacterial endocarditis needed to be investigated. Blood cultures subsequently were taken during symptomatic episodes confirming the presence of streptococcus species in the blood stream. Given his history, at that time one could consider the heart valve as the more likely

source, although his persistent back pain, particularly with the finding of no new vegetations or changes in the heart structure on investigation could raise the question that a low grade spinal osteomyelitis was present causing these episodes.

...

While the patient's submission contains a considerable number of grievances, there seems to be considerable discrepancy between these and the documentation provided. However, it is difficult to find a significant point of medical error. ... Particularly early in this gentleman's illness, there appears to be insufficient evidence to consider an infectious etiology, especially with the MRI scan findings. The presence of major co-morbidities to obscure and delay diagnosis, emphasises the difficulties in medical management in this case."

*The District Health Board*

Ms H, Quality and Risk Manager for the District Health Board (the DHB), stated, in relation to Mr A's concerns about bed availability and his MRI scan not being actioned appropriately in March 2004:

"Medical staff are involved in the prioritisation of patients and particularly if procedures require cancellation. There is a triage system to free up beds if that is required in times of emergency and involves medical staff reviewing existing patients with a view to moving them to another ward in the hospital where beds are available or discharging them into the community with appropriate support systems in place.

The systems at [the DHB] supports urgent MRI scan requests being acted on immediately both for inpatients and outpatients, and bed allocation is managed with clinical input according to the urgency of the required treatment. In reading [Mr A's] notes it would appear that the request for an MRI scan was managed within appropriate timeframes.

The clinical information system (CIS) at [the DHB] ensures that radiology and laboratory investigations and other relevant clinical information is available electronically as soon as it has been entered and this assists with consistent and timely information being made available for all clinicians across all sites including [the first public hospital].

While there may have been pressure on beds the day [Mr A] was booked to come into [the second public hospital] for his MRI scan there are systems and processes that ensure the clinical issues are considered before decisions made to cancel or defer patients and other alternatives are available if it is important the patient is admitted on that day for clinical reasons."

## Independent advice to Commissioner

The following expert advice was obtained from Dr Garnet Tregonning, orthopaedic consultant:

**“Re:           Complaint [Mr A]  
Your Ref:    04/20951/WS**

Herewith the report as requested by you on the 30<sup>th</sup> June 2005.

I have read the 756 pages of supporting information that you have provided to me and in particular:

- Letter of complaint to the Commissioner from [Mr & Mrs A] dated 29<sup>th</sup> November 2004.
- Notes taken during telephone interview with [Mr A] 6<sup>th</sup> April 2004.
- Copy of [Mr A’s] medical records from [the first public hospital].
- Copy of [Mr A’s] medical records from [the second public hospital].
- Response from [the medical centre] including Mr A’s clinical records.
- Copy of ACC’s file which includes the independent expert advice.
- The response to the Commissioner from [Dr B] received 6<sup>th</sup> May 2005.
- The response to the Commissioner from [Dr C] received 25<sup>th</sup> May 2005.

In addition I obtained verbal information relating to the MRI report from [...], Consultant Radiologist, [...] and advice concerning the microbiology from [...], Clinical Microbiologist, [...].

**1. In response to your question ‘was [Dr B’s] management of [Mr A’s] back pain appropriate?’ I propose to address this with respect to the three episodes of care provided by [Dr B].**

**A. February 10–19 2004 [the second public hospital].**

### Relevant Facts

[Mr A] was initially seen by [Dr B] approximately four weeks after the onset of his symptoms. Based on the document of the history as provided by [Dr B] and the Trainee Intern, the following facts arise.

- The patient had a history of definite trauma to his lower back with the onset of pain occurring after he had been lifting some heavy posts some four weeks previously. There was a background of similar episodes of low back pain going back many years.

- 
- The patient was complaining predominantly of low back pain with no complaints of sciatica or neurological disturbances.
  - It was documented that at the time of admission there was no history of fever or weight loss.
  - There was no documentation of the patient having a swollen foot with puncture wounds.
  - The documented findings of the clinical examination were of normal neurology and leg findings but there was no mention of findings of examination of the lower back or of general examination of the patient.
  - Investigations on admission revealed the presence of a raised ESR of 68 mm in one hour and raised CRP. It was noted at that time there was a normal white blood count with no leukocytosis.
  - Plain x-rays were reported by [the] Consultant Radiologist at [the second public hospital] showing some minor disc narrowing at L5S1 and degenerative changes all spaces. There was no report of any radiological findings supportive of infection.
  - The fact that the patient had diabetes was recognised and it was noted that he had had normal blood sugars during his stay.
  - A diagnosis of exacerbation of mechanical low back pain without neurological involvement was made.

Over the nine day period the patient was in hospital, the patient was seen daily by [Dr B]. It is noted that the patient had a gradual improvement in his clinical state with conservative treatment consisting of analgesia and bed rest. It was noted that his temperature was elevated on three occasions but was below normal on five occasions. The ESR, prior to discharge, had decreased to 40mm in one hour but was still recognised as being elevated.

The patient was discharged with arrangements made for follow up in four weeks and in the discharge summary it was recommended that the ESR be checked again.

### **Conclusion**

It is my impression that on the first admission there was a satisfactory management and treatment plan and appropriate follow up of the patient arranged. It also appeared that there was adequate communication with the patient. I note that the clinical progress was not consistent with an overt infective lesion of the spine.

## **B. March 4<sup>th</sup> [the first public hospital]**

In response to concerns that [Mr A's] condition was not improving and [he] had developed weight loss, [Dr B] appropriately responded by bringing the follow up appointment forward by two weeks.

There was no documentation of the clinical findings at this stage.

[Dr B] seemed to appreciate the need for further investigations given the ongoing back pain, weight loss and decreasing haemoglobin level and in particular the need to rule out occult pathology. I note that there was no specific mention of infection made at that time.

[Dr B] made arrangements to admit [Mr A] to hospital in [the second public hospital] four days later, specifically to have an MRI examination of the lumbar spine and a medical review including review by Haematology and Gastroenterology.

I comment that this was an appropriate response in management plan given the conditions at that time.

[Mr A] was not admitted as planned and this seems to be due to the lack of beds available at the hospital at that time.

Subsequently [Mr A's] condition deteriorated and he was admitted to [the second public hospital] under the physicians, where the focus was on investigation of a rapidly developing anaemia and suspected gastrointestinal bleeding.

Eventually an MRI of the lumbar spine was performed on the 22<sup>nd</sup> March. It is noted that this investigation occurred three months after the onset of symptoms. [The radiologist] performed the investigation with the background information suggesting possible infection and in particular the presence of a raised ESR.

Despite this, in summary the findings were that 'there is no clear evidence to suggest clear osteomyelitis or discitis'.

## **C. March 24<sup>th</sup> [the first public hospital]**

At that time [Mr A] was an inpatient.

[Dr B] reviewed the patient noting that the patient is 'comfortable in a spinal corset and taking oral analgesia'. There is no documentation of any other complaints.

[Dr B] informed the patient and subsequently his wife that the MRI showed no sign of infection or malignancy and confirmed degenerative changes at multiple levels.



The patient was then advised to continue physiotherapy, using a corset and a follow up appointment was arranged at [the first public hospital] four weeks later.

This appointment was not kept and [Dr B] did not see the patient again.

### **Comment**

In my view the report of the MRI of the lumbar spine proved to be a major distractor.

In consultation with radiology colleagues and in my own experience, an MRI examination of the lumbar spine two months after the onset of symptoms would certainly be expected to show changes consistent with infection. I have not seen the images myself but the report is quite clear that infective changes were sought and not definitely confirmed.

In general, MRI examinations for infection at this stage are a very reliable investigation.

In conclusion I comment that at this stage of [Mr A's] illness, given the above findings, it was not totally inappropriate to continue to treat the patient conservatively as advised by [Dr B].

### **2. What else should have been done?**

In commenting on this section I am fully mindful that any retrospective analysis is often misleading and does not necessarily reflect the situation faced by the clinician at the time.

With respect to the first admission on the 10<sup>th</sup> February I note that there is no documentation in the clinical records that the patient had previously had a swollen foot with possible puncture wound. There is also no documentation of the findings on clinical examination of the back itself which may have given some indication to a severe derangement such as infection. To balance this however at the time of this admission there was no history of fever or weight loss although both subsequently developed. The raised ESR and CRP which are both non specific indicators for infection were acknowledged but the ESR improved as did the patient's clinical condition.

In retrospect blood cultures or a bone scan may have proved helpful in identifying a cause.

**Clinic Appointment 4<sup>th</sup> March.**

There is no documentation or acknowledgement of weight loss which had occurred by that stage and which was considerable. Again there is no documentation of the findings of spinal examination, but I re-emphasise that, in discussion with both [Mr A] and his wife, [Dr B] acknowledged the need to investigate further to rule out any occult pathology accountable for persistent low back pain.

Thus it was entirely appropriate that [Dr B] arranged admission to [the second public hospital] for further investigations particularly an MRI examination and consultation with Haematologists and Gastroenterologists.

The fact that this did not happen, cannot be attributed to [Dr B].

**[The first public hospital] 24<sup>th</sup> March.**

[Dr B] states that he 'reviewed his low back condition again. He was comfortable in a spinal corset with satisfactory pain control on oral analgesic. No other nerve complaints arisen'. Again there is no mention of the clinical findings in his letter to the General Practitioner of 26<sup>th</sup> March. In addition there is no mention of continuing elevation of ESR, the investigation of anaemia or of the weight loss of some two stone.

However as mentioned above I believe [Dr B] was very strongly influenced by the report of the MRI.

**3. Was [Mr A's] discharge on 27<sup>th</sup> March appropriate?**

I find it difficult to answer this given the above. On balance it can be justifiable given the MRI findings and the fact that further follow up outpatient assessment was arranged.

**4. See above.**

**5. See above.**

**6. See above.**

**Summary**

I emphasise that this case is a complex presentation of probable bacterial endocarditis (although no vegetations were demonstrated), and possible low grade spondylodiscitis. However for both of these diagnoses, the presentation was not classical.

There were a number of distractors.

- A. The previous history of episodic low back pain and a traumatic incident which initiated the onset of back pain on this occasion.
- B. The plain X-ray findings at one month after onset showing no evidence of infection.
- C. The initial elevation of the ESR in the presence of normal white blood count, and the subsequent decrease in ESR associated with improved clinical findings during the first admission.
- D. The lack of evidence of infection on the MRI taken two months after the onset of symptoms. I have not seen the MRI and also understand that the second MRI was also not conclusive of infective changes and in particular evidence of destructive changes of the spine.
- E. The presence of anaemia and melaena which pointed towards a gastrointestinal disturbance.

I also note that after completing a full course of antibiotics, according to [the orthopaedic surgeon's] report, there was no evidence of residual destructive changes in the lumbar spine. Therefore there was no conclusive evidence that this patient had spondylodiscitis although I personally think it was probably present.

It is also possible that the low back pain was a component of bacterial endocarditis. It is my impression that it is not widely known in orthopaedic circles that bacterial endocarditis can cause low back pain in its own right.

I also point out that the diagnosis of spondylodiscitis, even in more straight forward cases, is frequently delayed up to 1–2 months. In this case despite repeated plain X-rays and MRI there was no conclusive evidence of spondylodiscitis.

Finally it is my opinion that, whilst it is unfortunate that an infective diagnosis was not made earlier (despite it being looked for), [Dr B's] management of this case could be considered to be appropriate in the circumstances.”

The following expert advice was obtained from an Associate Professor of Cardiovascular Medicine, Dr Stewart Mann:

“My full name is Stewart Mann. I undertook medical training at Oxford University and Kings College Hospital Medical School, London, qualifying MA, BM, BCh in 1973. I completed a succession of junior medical posts (internal medicine and cardiology) in UK and Australia and achieved MRCP (UK) in 1976 and a doctorate (DM Oxon) in 1985.

From 1986 until 2003 I practised as a physician and cardiologist at Hutt Hospital [...], becoming FRACP in 1989 and FRCP (London) in 1992. Since 2003, I have worked in a joint clinical and academic post as a cardiologist at Wellington Hospital and Associate Professor of Cardiovascular Medicine at the Wellington School of Medicine.

For 2002 and 2003, I was Chairman of the New Zealand Region of the Cardiac Society and, in that capacity, undertook a tour of all New Zealand Hospitals to document relevant human and physical resources available for the care of cardiac patients. In this capacity, I did visit [the hospitals involved], meeting [Dr C], several physicians at [the second public hospital] and cardiologists at [the city hospital]. I have no ongoing regular collegial contact with [Dr C] or other staff at [the first and second public hospital] who were involved in the care of Mr A in 2004. I do have collegial contact with some other physicians at [the second public hospital] and cardiologists at [the city hospital], including [the consultant cardiologist]. I do not believe any of the above constitutes a conflict of interest with the following report.

I have read the considerable amount of information supplied to me regarding the care of [Mr A] in the first half of 2004 at [the medical centre], [the first public hospital and the second public hospital]. In addition to the hospital and health centre records, this includes opinion from independent referees (from the fields of orthopaedics and infectious diseases) solicited by the Accident Compensation Corporation in respect of a claim for medical misadventure.

I have also read the Guidelines for Independent Advisors from the Health and Disability Commissioner and have undertaken to do my utmost to follow them in answering the specific questions put to me. The text below intersperses (under clear headings) my reading of the progress of the clinical problem, its diagnosis and treatment (categorised by month and hospital) followed for each episode of care by judgemental comments about the management. The report concludes with a summary.

**[Mr A] — (in 1994) A 57y old man with an aortic valve replacement (Homograft 1990, Starr-Edwards mechanical prosthesis 1997), Type 2 diabetes, paroxysmal AF and a history of mechanical back pain**

- 1. Was the management of [Mr A's] atrial fibrillation appropriate?*
- 2. If not, what else should have been done and whose responsibility was it?*

**[THE FIRST PUBLIC HOSPITAL] — FEBRUARY 2004**

From the notes, [Mr A] was admitted to [the first public hospital] on 7/2/04 ([Dr C]) with paroxysmal atrial fibrillation. He had had episodes of this before. He was hypotensive (had low blood pressure) with a limited response to a fluid challenge but not shocked and there was good renal output (urine flow). He was given a digoxin infusion over 1 hour on 7/2/04 and appears to have reverted to sinus rhythm on the afternoon of 8/2/04. The dominant symptom was of back pain and severity was such that inpatient referral was arranged with transfer to the orthopaedic unit at [the second public hospital] ([Dr B]) for further management.

**Comment**

There are a variety of options for treating an acute episode of paroxysmal atrial fibrillation including

- expectant treatment (no specific therapy but resuscitation with fluids etc as required)
- antiarrhythmic drugs (e.g. flecainide, sotalol or amiodarone)
- rate controlling drugs (e.g. digoxin, other beta blockers or verapamil) or electrical cardioversion.

Hypotension would have ruled out some choices. Of those remaining, digoxin is perhaps a little 'old fashioned' but perfectly reasonable. Although blood pressure was low, there was evidence of normal conscious level and good renal output and therefore no 'shock'. I feel it was reasonable for [Mr A] to be managed in [the first public hospital], especially as there had been previous episodes of paroxysmal AF, and I note he reverted to sinus rhythm the next day.

*3. Was the management of [Mr A] appropriate when he was noted to be losing weight, had an elevated ESR and unexplained anaemia?*

*4. If not, what else should have been done and whose responsibility was it?*

**[THE FIRST PUBLIC HOSPITAL] — FEBRUARY 2004**

The admission to [The first public hospital] (7-10/2/04) was brief and focused on the paroxysmal AF and the back pain. There was a history strongly suggesting mechanical back pain. There was a mild elevation in temperature on admission which settled and a further temporary mild rise on 8<sup>th</sup>-9<sup>th</sup> February. There is reference to broken skin on a pressure area.

### Comment

There was little or nothing to arouse suspicion of septicaemia at this stage and there were other explanations for a mild elevation in temperature at the time of admission.

### [THE SECOND PUBLIC HOSPITAL]— FEBRUARY 2004

The admission to [the second public hospital] (10-19/2/04) was under the care of the orthopaedic team ([Dr B]). Initial assessment (10-11/2/04) noted raised ESR (60) but absence of fever and noted ‘burns’ to [Mr A’s] back from hot packs. Investigations for myeloma (a malignancy affecting bone and often presenting as back pain along with raised inflammatory markers such as ESR) were done and found to be negative. Other explanations for a raised ESR were considered including the presence of a mechanical valve prosthesis. Mobilisation and Vioxx (an anti-inflammatory drug) were partly successful in relieving pain and this improved enough for [Mr A] to agree to discharge (there is disparity between the medical records and [Mr A’s] account of his readiness for discharge). No fever was recorded in written nursing notes. The ESR fell to 48. I note that haemoglobin level on 12/2/04 was 133g/l which is within the normal range — although it had been higher previously.

### Comment

Again, apart from the raised ESR, there is nothing specific to raise suspicion of an infective process. Attribution of a raised ESR to a normal mechanical valve replacement is inappropriate but this is a non-specific test and, in the absence of any other supportive evidence, does not point specifically to a likely septicaemia.

### [THE FIRST PUBLIC HOSPITAL] — MARCH 2004

[Mr A] was seen by [Dr B] at [the first public hospital] on 04/03/04. Readmission to [the second public hospital] was planned along with referral for MRI. A bed offered there on 9/3/04 was declined but he was **admitted to [the first public hospital] on 11/3/04** having deteriorated further. He was now anaemic (Hb 103 – > 92 – > 109) and gave a history of central abdominal pain and melaena (altered blood in the stools appearing black and arising from bleeding in the upper gut). The back pain continued and there was occasional mild pyrexia. There are notes from [Dr C] on 12/3/04 and 17/03/04 documenting anaemia and weight loss but not fever. Gastroscopy (done by [Dr K] at [the second public hospital] on 17/03/04) was negative.

The MRI of [Mr A’s] back was done on 22/03/04. The radiologist could find no conclusive evidence of inflammation although there was some focal pathology. The radiologist raised the question of an alternative focus of infection. [Dr C]

noted on 24/03/04 that the gastroscopy was negative (and colonoscopy planned); he also notes the 'low grade temp'[erature rise]. He intended to do further investigation in outpatients but indicated that current investigation and treatment as an inpatient was complete. [Mr A] was **discharged home on March 27<sup>th</sup>**.

### **Comment**

Gastrointestinal bleeding secondary to use of analgesics and warfarin was a logical primary diagnosis. The disparity between an apparently clear history of melaena (indicating an upper gut source of bleeding) and the negative gastroscopy is curious. [Mr A] was still being followed by [Dr B] and [Dr C] may have assumed the back pain problem was being supervised by him, especially while further investigations (that might have revealed a focus of inflammation responsible for the elevated inflammatory markers) were pending.

Once the results of MRI and gastroscopy were known, especially with the suggestion from the radiologist that other sources of inflammation should be considered, there should certainly have been a low threshold for doing blood cultures in a patient with a prosthetic valve. I believe most physicians would have done these in a patient with a prosthetic valve, raised ESR and CRP, low grade intermittent fever, weight loss and anaemia (unexplained after gastroscopy), despite some alternative explanations for some of these. I would hold this opinion even without the knowledge of eventual diagnoses (although I have made some relevant observations on these below as uncertainties remain). This would have potentially brought forward the diagnosis and treatment of the septicaemia and reduced the period of the patient's distress and discomfort, although there would probably have been no difference in the long-term outcome.

### **[THE FIRST PUBLIC HOSPITAL] — APRIL 2004**

[Mr A's] account documents ongoing discomfort and distress (from 29/03/04 to 19/04/04) with difficulty persuading [the first public hospital] staff of his condition and a need for further investigation and treatment.

### **Comment**

As above, a serious condition for which there were clues, was not investigated with all appropriate vigour resulting in a 3-week delay in diagnosis from the time this should have been considered.

### ***5. Was [Mr A] appropriately treated when he was diagnosed with infective endocarditis?***

**[THE SECOND PUBLIC HOSPITAL] — APRIL 2004**

Melaena and back pain continued and [Mr A] was **admitted to [the second public hospital] ([Dr L]) on 19/04/04**. The possibility of infective endocarditis (an infection of a heart valve) was realised at the time of admission (by his GP, [Dr G]) and blood cultures sent. These proved positive for streptococcus defectiva, a rare organism (which nevertheless has been implicated in endocarditis) sensitive to penicillin. No valve lesions were detectable clinically (or on subsequent ultrasound examinations) and there were no documented embolic phenomena (evidence of infection on a heart valve where bits of infected material break off and are carried by the blood stream to some other part of the body). The spine remained a possible focus for infection.

Augmentin was started as soon as the culture was known to be positive. A transoesophageal echocardiogram was not thought to be necessary (by [consultant cardiologist] at [the city hospital]) as, despite the septicaemia but in the absence of embolic phenomena, endocarditis was deemed unlikely. A colonoscopy was done on 23/04/05 with no abnormality found. Augmentin was given intravenously initially for 6 days and then orally for 3 more days. During the course, [Mr A's] back pain improved considerably and indices of inflammation resolved or were resolving. He was **discharged on 30/04/04** with provision made for rapid readmission to [the second public hospital] should his condition deteriorate again.

**[THE SECOND PUBLIC HOSPITAL] — MAY 2004**

[Mr A] was **readmitted to [the second public hospital] on May 6<sup>th</sup>** primarily under the care of the surgical team ([Dr K]) with further melaena; gastroscopy was again normal. His temperature was raised on May 8<sup>th</sup> and blood cultures again proved positive for the original organism. He was commenced on intravenous penicillin on May 10<sup>th</sup>. There was a medical review ([Dr ...] "in [Dr L's] absence", page 326 of notes as stamped by HDC) where this was confirmed and Gentamicin added. On May 11<sup>th</sup>, care was transferred to the medical team and there was a discussion with a cardiologist at [the city hospital]. The records do not indicate whether the choice of antibiotic regimen was discussed with a cardiologist or infectious diseases specialist (or with [Mr A]).

[Mr A] was **transferred to [the city hospital]** and completed treatment there. (I have not been supplied with notes from that admission). Unfortunately, despite monitoring of Gentamicin levels, he developed long-term toxic effects with vestibular damage leading to balance problems.

**Comment**

I am a little surprised by the conclusion in the April admission that septicaemia (or even 'bacteraemia' – organisms detected in the blood not causing illness) in a man



with a prosthetic heart valve did not need to be treated from the outset in the usual intensive way for endocarditis with several weeks of high-dose intravenous antibiotics. However, this was a considered decision made in discussion with a cardiologist and did not lead to unwarranted delay in formal treatment. Augmentin was a reasonable choice of antibiotic prior to sensitivities becoming available and, having started it and reached the determination for a limited course, it was reasonable to complete it with this antibiotic.

With recurrence of the infection later, the formal choice, dosage and monitoring of long-term antibiotic therapy would normally be done in collaboration with an infectious disease consultant. Starting the patient on high-dose intravenous penicillin and Gentamicin before transfer was, however, quite appropriate. Detailed discussion of choices of antibiotic regimen with the patient would not usually be undertaken at this stage, although some outline of the choice and need for it should of course be given. None was documented in the notes at the outset but the ongoing monitoring for ototoxicity would presumably have been explained as [Mr A's] treatment proceeded at [the city hospital]. Gentamicin is conventionally used as an adjunct to penicillin therapy in presumptive streptococcal endocarditis and it is not usual to offer patients an alternative unfunded antibiotic on the grounds of possibly less toxicity. The course of Gentamicin is commonly briefer than the extended one [Mr A] apparently received (with a consequently lower risk of toxicity) but this would usually be determined with the advice of an infectious diseases specialist (at [the city hospital]). Despite continuing surveillance for ototoxicity, this complication developed to a serious degree.

### **Underlying diagnosis**

Throughout the events and at follow-up there was no direct evidence of endocarditis. The valve remained normal clinically and on echocardiography, and there was no evidence of systemic embolisation. Even in retrospect, the diagnosis of endocarditis cannot be made with certainty although there was clearly a septicaemia with a focus of infection that was resistant to a conventional course of broad spectrum antibiotic therapy. The focus could have been in the prosthetic valve or could have been in the spine. Subsequent orthopaedic opinion and MRI scan suggests that there probably was a focus of infection in the spine although this was very difficult to diagnose on the initial assessments.

I do not therefore feel there was any deficient or clearly incorrect treatment of the septicaemia once it was identified. I would not claim expertise over the choice and administration or dosage of the subsequent antibiotic course and note that an infectious diseases consultant has already commented on this (for ACC) without finding fault.

## SUMMARY

The management of the episode of atrial fibrillation in February 2004 raises no concerns.

[Mr A] underwent a prolonged period of severe suffering in early 2004 with a variety of problems and delayed recognition of the serious diagnosis of septicaemia +/- endocarditis. I do feel that [Dr C] and staff at [the first public hospital] should have conducted the necessary investigations (including blood cultures) before [Mr A's] discharge in late March. This was, on the face of it, a moderately serious error leading to some weeks of probably unnecessary severe discomfort. In mitigation, there were several other contributors and potential contributors to his symptoms and findings, but enough clues to the likely presence of an additional cause of inflammation to warrant earlier investigation. In further mitigation, continued follow-up and investigations were planned but not with particular vigour in a patient with ongoing potentially serious systemic infection and disabling symptoms. In a patient with a prosthetic heart valve, a very low threshold for investigating the possibility of endocarditis should always exist, although this diagnosis cannot be proven here, even in retrospect.

Relations between clinical staff and [Mr & Mrs A] were not always the best, which would not have helped assessment and treatment. However, in the end [Mr & Mrs A] were fully justified in their belief that there was more to his condition than mechanical back pain and gastrointestinal bleeding. Thankfully, the delay in diagnosis of septicaemia did not result in any subsequently apparent long-term damage.

The long-term consequences of his Gentamicin therapy are deeply unfortunate. The commencement of the penicillin and Gentamicin regimen in [the second public hospital] was completely conventional. I would not like to comment personally (through lack of expertise) on appropriateness and detail of the continuing antibiotic regimen used but an infectious diseases referee has found no major fault with it. I believe that responsibility for keeping [Mr A] informed about the risks from continuing Gentamicin therapy lies much more with [the city hospital] given the relative duration of this treatment in the two institutions.”

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## Response to provisional opinion

*Dr C*

Dr C, commenting on Dr Mann's advice that there should have been a low threshold for doing blood cultures on a patient with a prosthetic valve once the results of the MRI and gastroscopy were known, stated:

“With the benefit of hindsight this is undoubtedly so, but at that time, [Mr A] had no clinical evidence of infection or endocarditis. Indeed, this was not the case until a month after he had been discharged.

[Mr A] gave a history of ingestion of Vioxx and Aspirin, melaena and had positive faecal occult blood testing. At the time it was our considered opinion that these factors were a reasonable explanation for his anaemia and ... a negative gastroscopy does not rule out upper gastrointestinal bleeding.

The ESR is a non specific marker of inflammation (also stated by Dr Mann). The ESR is not uncommonly elevated in anaemia. [Mr A] was anaemic. ...

At the time, there was, unfortunately, no evidence that the elevated ESR was a reflection of a more sinister process going on and that the elevation was readily explained by his poor nutrition as a consequence of ongoing pain plus the attendant general debility and anaemia.

[Mr A's] ESR in February was elevated. I agree with the statement of Dr Mann that in February 2004, 'there was little or nothing to arouse suspicion of septicaemia'. ...

At the time we placed greater weight on the likely explanation being that Mr A's chronic pain, poor nutrition, weight loss secondary to poor nutrition and general debility plus gastrointestinal bleeding and anaemia were a reasonable explanation for this modest elevation. A suprising feature of the case is that I would have expected that the CRP [C-reactive protein] elevation associated with septicaemia, endocarditis or abscess formation would be more robustly elevated and greater than 80 and more likely to be 100. [Mr A's] CRP in February was modestly elevated. ...

The low grade intermittent fever during [Mr A's] admission in February was very non-specific and was readily explained by his ongoing pain, general debility, gastrointestinal bleeding and anaemia. ... My entry in the notes on 24.03.04 indicates that I was aware of his aortic valve replacement and the fact that a low grade temperature could indicate endocarditis, and for that reason his temperature was observed for 3 days. The normal temperature, plus my clinical assessment and assessment of his various investigations did not lead me to the conclusion that Mr A had infection. Indeed, I was reassured to the contrary. His condition had also improved with pain control and hand in hand with this was the normalization of the low grade temperature.

A factor not referred to by Dr Mann is the importance of the white blood cell count as an indication of infection. At no stage did [Mr A] demonstrate an elevated white blood cell count and in particular he had normal segmented

neutrophils and no immature band cells present which I would have expected if this man was to have septicaemia or endocarditis. ...

I suggest that it would be most unlikely for ongoing septicaemia for at least 1 month (March to April when the blood cultures were positive) on the basis of endocarditis, to be associated with a normal valve clinically or on echocardiography.

MRI scan did not confirm a focus of infection. Subsequent orthopaedic opinion indicated that MRI was a very reliable investigation for infection and that it was unusual for [Mr A's] MRI, if infection was present, to have not shown changes consistent with infection despite the 2 month history of back pain.

If we could go back in time with the knowledge we have now, then I would completely agree with [Dr Mann's statement that the 'necessary investigations (including blood cultures)' should have been conducted before [Mr A] was discharged in March]. However, as stated earlier, there was no clinical evidence of infection present. Thus any blood cultures, in all likelihood, may not have revealed anything of concern. He had presented to us with anaemia supported by a history of gastrointestinal bleeding (melaena) and positive faecal occult blood testing. He was at risk for upper gastrointestinal bleeding having been consuming Vioxx and Aspirin. His elevated ESR and CRP were readily explained by his anaemia, ongoing pain, anorexia with attendant weight loss and general debility. MRI of the spine did not demonstrate any evidence of infection despite a 2–3 month history of back pain and the attending orthopaedic specialist considered his back pain was of mechanical origin, further supported by a history of the back pain having arisen as a result of a work related injury. A clear note in his case notes by myself indicates that he had an aortic valve replacement and a low grade temperature and that I requested 4 hourly temperature (TPR) observation. This observation took place over 3 days and no pyrexia was noted and it was at this stage his overall clinical condition had improved, mainly as a consequence of pain control and associated improvement in nutrition. As a consequence he was discharged home with arrangements for follow-up and further follow-up blood tests.

The aim was to see his mechanical back pain settled (under the supervision of the orthopaedic specialist). The expectation was that the relief of the back pain would result in improved nutrition, weight gain and eventual return back to normal. He was to avoid Vioxx and Aspirin and placed onto supplemental iron with the expectation that his anaemia would correct. Colonoscopy was to be undertaken to complete the investigation of gastrointestinal bleeding. If in follow-up he had not improved then further investigation would take place. This plan was discussed at the family meeting prior to his discharge. ...

The radiologist reported that the changes were due to disc herniation and that these changes were not due to infection. I am perplexed as to why the radiologist should raise the question of an alternative focus of infection having indicated that the MRI changes were not due to infection. I suggest that it may have been more relevant to have indicated that it was most unusual that no changes consistent with infection or malignancy were present bearing in mind the 2–3 month history of back pain. ... With the benefit of hindsight I absolutely agree [that most physicians would have done blood cultures in a patient with [Mr A's] history and symptoms], but at the time we were strongly reassured by a reasonable explanation for the symptoms and the negative MRI scan. ...

I am saddened and sorry that [Mr A] feels there is a 'stigma' that is associated with back pain patients. I would like to assure him that this is not the case. To the contrary we were concerned about his symptoms and falsely (and some might say understandably) were reassured by explanations for his symptoms that we felt were more likely with the knowledge that we had at the time. ... Our intentions were to assist him on the basis of the information known to us at the time. [A's] outlined above we all deeply regret and feel sorrow that he did not have his infection diagnosed earlier."

### **Additional expert advice**

Dr Stewart Mann was asked to review Dr C's response to the provisional report and provide additional expert advice. Dr Mann stated:

"I have read carefully [Dr C's] response to the expert opinions submitted and the HDC's judgement. There are few issues of fact or medical opinion on which we differ and he agrees (with the benefit of hindsight) with the crucial statement on which, perhaps the judgement hinges:

**'I believe most physicians would have done these in a patient with a prosthetic valve, raised ESR and CRP, low grade intermittent fever, weight loss and anaemia (unexplained after gastroscopy), despite some alternative explanations for some of these'.**

As stated in my original commentary, this was a conclusion I reached despite trying fervently to do so without hindsight bias.

I accept the comments he makes on the reasons why, individually, many of these factors were minimally present or non-specific, although I would contend that the presence of a raised ESR and/or CRP in anaemia are usually indicators of an acute or chronic disease process responsible for the anaemia. The judgemental question is whether the presence of all of these factors reached a sufficient threshold to raise

the question of an underlying infective process possibly involving the prosthetic valve and require investigation for this with a simple test – namely blood culture.

While it is possible, as [Dr C] states, that blood cultures at this time could have been negative, retrospectively I feel most physicians would consider that the probability of cultures at that time being positive would have been high. Such a result would thus in all probability have led to earlier treatment and reduced pain and suffering by the patient. **The omission therefore had probable adverse clinical consequences, however culpable or otherwise it was.**

As stated in my original opinion and reinforced by the further comments from [Dr C], there were a number of distractions and mitigating factors that deflected clinical attention from the eventual diagnosis and led to the omission of the relevant test at the time in question. Most clinicians would have had similar experiences in spite of conscientious clinical practice. Nevertheless, there was a potentially avoidable temporary adverse outcome.”

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## **Code of Health and Disability Services Consumers’ Rights**

The following Right in the Code of Health and Disability Services Consumers’ Rights is applicable to this complaint:

### *RIGHT 4*

#### *Right to Services of an Appropriate Standard*

*(4) Every consumer has the right to have services provided in a manner that minimises the potential harm to, and optimises the quality of life of, that consumer.*

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## Opinion: Breach — Dr C

Under Right 4(4) of the Code of Health and Disability Services Consumer's Rights (the Code), Mr A had the right to have services provided in a manner that minimised potential harm and optimised his quality of life. My consultant physician/cardiology expert Dr Stewart Mann advised that although there were mitigating circumstances and other potential contributors to Mr A's symptoms and findings, "[Dr C] and staff at [the first public hospital] should have conducted the necessary investigations (including blood cultures) before [Mr A's] discharge in late March. This was, on the face of it, a moderately serious error." Accordingly, for the reasons that follow, I consider that Dr C breached Right 4(4) of the Code.

Mr A was admitted to the first public hospital under Dr C on 11 March 2004 with abdominal pain and anaemia. Mr A, who had a history of heart valve surgery, reported that, in addition to his abdominal pain and anaemia, he had melaena, weight loss and continuing back pain. Dr C considered the cause of Mr A's symptoms to be gastrointestinal bleeding caused by his use of Vioxx and aspirin. During his admission, Mr A experienced occasional mild pyrexia and had a raised ESR. Dr Mann confirmed that it was appropriate for Dr C to reach a primary diagnosis of gastrointestinal bleeding caused by the use of analgesics and warfarin. He advised:

"[A]part from the raised ESR, there [was] nothing specific to raise suspicion of an infective process. Attribution of a raised ESR to a normal mechanical valve replacement is inappropriate but this is a non-specific test and, in the absence of any other supportive evidence, does not point specifically to a likely septicaemia."

This led to further investigations. A gastroscopy on 17 March was negative and the MRI on 22 March showed no evidence of inflammation. However, the radiologist reviewing the MRI raised the question of an alternative focus of infection. Dr Mann advised me that he felt it was appropriate for Mr A to be discharged and that Dr C intended to follow-up with repeat blood tests, ESR, additional inflammatory markers, B<sub>12</sub> and folic acid levels as part of later outpatient assessments.

In his expert advice, Dr Mann advised that although it cannot be proven, even in retrospect, that Mr A had endocarditis, once the results of his MRI and gastroscopy were known, especially in light of the radiologist's comment, there should have been a "low threshold for doing blood cultures in a patient with a prosthetic valve". He advised:

"I believe most physicians would have done these in a patient with a prosthetic valve, raised ESR and CRP, low grade intermittent fever, weight loss and anaemia (unexplained after gastroscopy), despite some alternative explanations for some of these. ... This would have potentially brought forward the diagnosis and treatment

of the septicaemia and reduced the period of the patient's distress and discomfort, although there would probably have been no difference in the long-term outcome."

Infectious diseases specialist Dr Chambers advised ACC that MRI scans usually have a sensitivity of about 95% if the infection has been present for more than two weeks, and changes become more obvious with time. In this case the MRI scan did not show changes despite three months of symptoms, which was unusual. However, despite this, "there was convincing evidence that an inflammatory process was present and that infection would be the likely cause. The evidence includes repeated observations that Mr A had an intermittently elevated body temperature, and elevated ESR, unexplained weight loss, and there was an unexplained anaemia that is a common accompaniment of long standing infections." The infectious diseases specialist advised ACC that melaena, in particular, suggests the existence of a severe condition and should be investigated thoroughly.

Dr Mann advised that, following the MRI scan, Dr C did not follow up with the vigour that would be expected in a patient with an ongoing potentially serious systemic infection and disabling symptoms. Dr Mann commented:

"A serious condition, for which there were clues, was not investigated with all the appropriate vigour resulting in a 3 week delay in diagnosis from the time this should have been considered."

In response to the provisional opinion, Dr C stated that, with the benefit of hindsight, he agrees that blood cultures should be taken in a patient with a prosthetic valve, raised CRP, low-grade intermittent fever, weight loss and anaemia. However, Dr C explained that, at the time, he was strongly reassured by a reasonable explanation for the symptoms and the negative MRI scan. In particular, Dr C noted that the weight loss was attributable to Mr A's chronic pain, the anaemia was explained by upper gastrointestinal bleeding, and the modest elevation of ESR, CRP and temperature was explained by Mr A's pain, associated poor nutrition, general debility, GI bleeding and anaemia. Dr C noted that repeated tests for white blood cell count did not support the presence of infection, and Mr A's temperature improved after three days of monitoring.

Dr C agreed with Dr Mann's statement that blood cultures should have been conducted prior to Mr A's discharge in March, "if we could go back in time with the knowledge we have now". However, he is of the opinion that blood cultures may not have revealed anything of concern because there was "no evidence of infection present".

Dr Mann reviewed Dr C's response to the provisional opinion and commented that while this was a very understandable error of omission, his earlier advice remained unchanged. Dr Mann explained that he reached those conclusions despite "ferverly"



trying to do so without hindsight bias. He stated: “The judgemental question is whether the presence of all of these factors reached a sufficient threshold to raise the question of an underlying infective process possibly involving the prosthetic valve and require investigation for this with a simple test – namely blood culture.” He conceded that, as Dr C states, the blood cultures at this time could have been negative, but he is of the opinion that the probability was high that they would have been positive and would have led to earlier treatment and reduced pain and suffering for Mr A.

While I agree that there were a number of distractions and mitigating factors that deflected clinical attention, and that many clinicians would have had similar experiences in spite of conscientious clinical practice, I accept Dr Mann’s advice that Dr C did not carry out investigations into the cause of Mr A’s condition with sufficient vigour before he was discharged on 27 March 2004. Despite the misleading MRI result, there were other clues that an infection was present and, as the physician in charge of Mr A’s case, Dr C had the responsibility to direct the team in the appropriate investigation of Mr A’s ongoing symptoms.

Accordingly, in my opinion, Dr C’s management of Mr A from 11 to 27 March 2004 did not minimise potential harm to Mr A and resulted in a three-week delay in diagnosis. Dr C therefore breached Right 4(4) of the Code.

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### **Opinion: No Breach — Dr B**

Mr and Mrs A complained that Dr B assumed that the cause of Mr A’s back pain was mechanical and that his rights were breached when Dr B neglected to consider any other differential diagnosis. Dr B was part of a team of medical staff from a variety of specialties who were involved in the assessment and treatment of Mr A’s deteriorating condition over a three-month period. My orthopaedic advisor, Dr Tregonning, commented that this case was a complex presentation of probable bacterial endocarditis (although this was not confirmed) and possible low-grade spondylodiscitis, but for both these diagnoses, the presentation was not classical. Dr Tregonning advised that Dr B’s management of Mr A’s condition was appropriate in the circumstances. Accordingly, in my view, Dr B provided Mr A with treatment with reasonable care and skill. The reasons for my decision are set out below:

On 10 February 2004, when Mr A’s recent episode of atrial fibrillation (which had required admission to the first public hospital) had resolved, he was transferred to the second public hospital orthopaedic unit under the care of Dr B for assessment of his ongoing back pain. Mr A’s spinal X-rays were repeated on his admission to the second public hospital, and confirmed the previous X-ray report of multiple areas of degeneration throughout his spine. In light of Mr A’s work-related injury four weeks

earlier, and in the absence of any obvious infective focus, Dr B concluded that Mr A's symptoms were mechanical in origin.

Mr A was treated with analgesia and anti-inflammatory medication and encouraged to mobilise. His ESR on 10 February was noted to be elevated (68mm/hr) but by 12 February, although still elevated, it had dropped to 40mm/hr. He also had intermittent mild pyrexia. On 12 February Mr A was seen by Dr B, who noted that although there had been some improvement in Mr A's back pain, the team would continue to assess him.

Dr B reviewed Mr A on 14 February, noted that he was still not walking well, advised that the bed-rest continue, and said that he would review him after the weekend. Mr and Mrs A informed Dr B of their concerns about the proposed plan to discharge Mr A on 16 February, and Dr B agreed to Mr A staying a further two days. He was then discharged to return to see Dr B at the Outpatient Clinic in four weeks.

Orthopaedic specialist Dr Taine advised ACC that in light Mr A's documented improvement during his February admission, the diagnosis at that time of mechanical back pain was reasonable, which concurs with the advice provided by my independent orthopaedic surgeon, Dr Tregonning. Dr Tregonning advised that Mr A's clinical progress during that admission was not consistent with an "overt infective lesion of the spine". He said that Dr B's management, treatment plan and follow-up for Mr A on his first admission was satisfactory, and his communication with Mr A was adequate.

On 1 March Mrs A contacted Dr B to ask if he could see Mr A earlier than previously planned, as the blood results received that day showed that Mr A's haemoglobin levels had dropped. Dr B saw Mr A at the Outpatient Clinic on 4 March and informed him that he would arrange his admission to the second public hospital for an MRI to exclude any occult pathology. Dr Tregonning noted that there was no record in the clinical notes at this time of Mr A's considerable weight loss, or the findings of the spinal examination. However, Dr B had discussed with Mr and Mrs A the need to investigate further to rule out any occult pathology that might have accounted for the persisting back pain. It was therefore, according to Dr Tregonning, "entirely appropriate" that Dr B arrange admission to the second public hospital for Mr A for further investigations, in particular the MRI and consultation with a haematologist and a gastroenterologist.

Dr Tregonning stated that Dr B responded appropriately to concerns about Mr A's weight loss and his condition not improving, by bringing the follow-up appointment forward by two weeks. He stated:

"[Dr B] seemed to appreciate the need for further investigations given the ongoing back pain, weight loss and decreasing haemoglobin level and in particular the need to rule out occult pathology. I note that there was no specific mention of infection made at this time."

However, before this could take place, Mr A was admitted to the first public hospital by his GP on 11 March for an urgent assessment of the cause of his falling haemoglobin. Mr A was under the care of Dr C from 19 March 2004. The assessment included a gastroscopy, performed on 18 March, which was unremarkable, and an MRI on 22 March. Dr B met with Mr A at the first public hospital on 24 March to discuss the results of the MRI, which showed only degenerative changes corresponding to the X-ray findings. Dr B arranged a follow-up appointment for Mr A to discuss these degenerative changes, but did not see him again.

I note that my consultant physician, Dr Mann, advised that once the results of Mr A's gastroscopy and MRI were known, especially in light of the radiologist's comment, other sources of inflammation should have been considered by Dr C and staff at the first public hospital. By contrast, Dr Tregonning advised that generally MRI examinations for infection at this stage are a very reliable investigation. He said that it appears that Dr B was "strongly influenced" by the MRI report. Dr Tregonning stated that given these findings it was not "totally inappropriate" for Dr B to continue to treat Mr A conservatively. He advised:

"I emphasise that this case is a complex presentation of probable bacterial endocarditis (although no vegetations were demonstrated), and possible low grade spondylodiscitis. However, for both these diagnoses, the presentation was not classical. ...

It is also possible that the low back pain was a component of bacterial endocarditis. It is my impression that it is not widely known in orthopaedic circles that bacterial endocarditis can cause low back pain in its own right.

I also point out that the diagnosis of spondylodiscitis, even in more straightforward cases, is frequently delayed up to 1–2 months. In this case despite repeated plain X-rays and MRI there was no conclusive evidence of spondylodiscitis.

Finally it is my opinion that, whilst it is unfortunate that an infective diagnosis was not made earlier (despite it being looked for), [Dr B's] management of this case could be considered to be appropriate in the circumstances."

I am conscious that Dr B and Dr C were reviewing the MRI scan from different perspectives. As an orthopaedic surgeon, Dr B was reviewing the MRI scan to consider spinal function and to identify the reasons for Mr A's back pain. Given that the MRI scan revealed degenerative changes but no infection, I accept my expert's opinion that it was reasonable for Dr B to treat the degenerative changes conservatively in the first instance.

Dr C, on the other hand, was reviewing the MRI scan to consider whether it revealed the cause of Mr A's abdominal pain and anaemia. Dr Mann has advised that in the context of other medical evidence of a likely infection, such as repeated observations of elevated body temperature, elevated ESR, unexplained weight loss, a history of melaena and unexplained anaemia, the radiologist's query about other causes of inflammation should have prompted further investigations.

Accordingly, in my opinion, Dr B provided appropriate care in compliance with the Code.

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### **Opinion: No Breach — The District Health Board**

#### *The second public hospital— February/March 2004*

Mr A complained about the co-ordination of care he received from the District Health Board. He stated that he was not treated "holistically" and the importance of involving his family in his care was disregarded. Mr A stated that he was not informed of the introduction of gentamicin into his treatment regime, and that none of the significant side effects were discussed with him. The management of Mr A's deteriorating condition between February and May 2004 was complex as he presented with cardiac, gastrointestinal and spinal problems. During this period he had six admissions to three different hospitals, and also transferred between the hospitals for specialised radiological examinations. The District Health Board had a duty of care to Mr A to ensure quality and continuity of services.

Mr A's first admission, on 7 February 2004, to the first public hospital was for suspected atrial fibrillation. Following an assessment in the Emergency Department, where atrial fibrillation was confirmed, he was admitted to the medical ward and commenced on an intravenous digoxin infusion and his condition monitored.

Dr Mann noted that Mr A was given a digoxin infusion for over an hour on 7 February, and he reverted to sinus rhythm on the afternoon of 8 February. Dr Mann stated that treatment for atrial fibrillation with digoxin is "perhaps a little 'old fashioned' but perfectly reasonable".

Mr A's painful back (which manifested a month earlier) was still causing him problems. He complained that on 9 February (although his atrial fibrillation had resolved) he was unable to sit or weight-bear because of his back pain, but the first public hospital staff informed him that he was to be discharged home. He had understood that instead of going home he was to be transferred to the second public hospital orthopaedic unit. Mr A stated that the transfer had been initiated by his GP, and the only reason that he was transferred to the second public hospital was because of intervention by his wife.

Mr A stated that he was discriminated against because an assumption was made that his back pain was mechanical and related to his occupation.

However, this does not appear to be the case, as the record shows that on 9 February, the physician who was responsible for Mr A's care, Dr I, ordered blood tests to check Mr A's ESR, and noted his plan to transfer Mr A to an orthopaedic ward for further assessment of his spine. Mr A was transferred to the second public hospital by ambulance on 10 February.

Dr Mann said that the dominant symptom Mr A was experiencing at that time was his back pain, which was so severe that inpatient referral was arranged for transfer to the second public hospital orthopaedic unit for further management. Dr Mann advised that it was reasonable for Mr A to be treated for his atrial fibrillation in the first public hospital, and that the admission was brief and focused not only on his cardiac problem but also on his back pain. Dr Mann stated that Mr A's history strongly suggested mechanical back pain, and there was nothing to "arouse suspicion of septicaemia at this stage".

In my view, the treatment Mr A received at the first public hospital in February was reasonable. His atrial fibrillation was controlled. His continuing back problem was recognised and the medical staff appropriately arranged his transfer to an orthopaedic unit. In my opinion there is no evidence that the first public hospital staff were discriminatory in their attitude to Mr A.

Mr A was readmitted to the first public hospital on 11 March 2004 for investigation of weight loss and melaena. He continued to have problems with back pain. Mr A had been offered a bed on 9 March but declined to be admitted. However, when his condition deteriorated further two days later, he was admitted. During this admission he was under the care of physician Dr C.

Mr A stated that he was seriously ill when admitted to the first public hospital, and complained that staff at the hospital "just passed it off as a mechanical back, thus putting my life at risk and my recovery back months".

Dr Mann noted that Mr A's readmission to the first public hospital was planned along with the referral for MRI. Mr A was now anaemic and gave a history of central abdominal pain and melaena. He was referred to gastroenterologist Dr K, at the second public hospital, and had a gastroscopy performed on 17 March, the result of which was negative. An MRI of his spine conducted on 22 March provided no conclusive evidence of inflammation although there was some focal pathology, and the radiologist raised the question of an alternative focus of infection.

The District Health Board's Quality and Risk Manager, Ms H, stated that the system at the DHB supports MRI scan requests being acted on immediately, and that bed

allocation is managed with clinical input according to the urgency of the required treatment. All relevant clinical information is available electronically, via the CIS (Clinical Information System) as soon as it is entered, to provide the clinicians with timely information. There is no indication that there was an undue delay in investigations requests being processed in a timely manner (although the time frames were unacceptable to Mrs A) and it appears that the results were forwarded promptly to the clinicians.

Dr Mann advised that gastrointestinal bleeding secondary to the use of analgesics and warfarin was a logical primary diagnosis, and the disparity between the apparently clear history of melaena and negative gastroscopy is “curious”. He said that Mr A was still being followed up by orthopaedic surgeon Dr B, and the team investigating Mr A’s gastrointestinal symptoms would have assumed that the back pain was being supervised by Dr B.

As already discussed, before Mr A was discharged on 27 March, Dr C and the medical staff at the first public hospital should have conducted the necessary investigations to identify the reason for the signs that were indicating an infective focus. The prime responsibility in leading the management of Mr A’s assessment and treatment lay with Dr C, and there is no evidence that the delay in the appropriate investigations being conducted was due to lack of co-ordination of services. The first public hospital responded appropriately in admitting Mr A and providing the investigative procedures ordered by the clinicians. Additionally, an occupational therapist and physiotherapist were involved in his ongoing care and the planning for his discharge. Accordingly, in my opinion, the first public hospital provided Mr A with appropriate services and reasonable treatment and care in the circumstances.

*The second public hospital— February to May 2004*

Mr A was seen by Dr B approximately four weeks after the onset of his back pain. He had a history of work-related trauma to his lower back dating from that time, and a background of similar episodes. Mr A’s diabetes was assessed and blood tests taken. Plain X-rays taken at the second public hospital showed degenerative changes and, as there was no other obvious cause for his back pain, a diagnosis of exacerbation of mechanical lower back pain, without neurological involvement was made.

As previously discussed, Dr Tregonning advised that there was satisfactory management and appropriate follow-up arranged for Mr A on his first admission to the first public hospital. He also noted that there was adequate communication with Mr A.

Ms H advised that while there may have been pressure on beds the day that Mr A was booked into the second public hospital for his MRI in March 2004, there are systems and processes to ensure that the clinical issues are considered in the provision of services being provided. There was another occasion, on 19 April 2004, when the unavailability of beds at the first public hospital resulted in a stressful period for Mr and Mrs A before he was admitted, later that day, to the second public hospital.

However, generally all requests for provision of services for Mr A were appropriately actioned.

Dr Mann noted that Mr A was admitted to the second public hospital by his GP on 19 April 2004, under the care of physician Dr L, for investigation of possible infective endocarditis. Blood cultures confirmed the diagnosis and the infective organism. Radiological examinations did not detect any valve lesions, therefore the spine remained the possible focus for infection. Mr A was administered the antibiotic Augmentin, intravenously for six days and orally for three days. Further examinations in the form of a TOE and a colonoscopy were performed to identify the cause of Mr A's symptoms, but no cause was found. Mr A was discharged on 30 April.

On 6 May Mr A was readmitted to the second public hospital under Dr K's surgical team. A gastroscopy was performed, which again was normal. Within the next three days, further blood studies isolated the original causative organism, Streptococcus B, and on 10 May Mr A was recommenced on intravenous Augmentin. The following day Mr A was transferred to the medical team, and his treatment and care were discussed with a cardiologist. Mr A was informed of the findings, and the treatment plan, including the proposal to transfer him to the city hospital for further management, was discussed with him. Mr A was transferred to the city hospital on 12 May 2004.

Dr Mann stated:

“I am a little surprised by the conclusion in the April admission that septicaemia (or ‘bacteraemia’ — organisms detected in the blood not causing illness) in a man with a prosthetic heart valve did not need to be treated from the outset in the usual intensive way for endocarditis with several weeks of high-dose intravenous antibiotics. However, this was a considered decision made in discussion with a cardiologist and did not lead to unwarranted delay in formal treatment. Augmentin was a reasonable choice of antibiotic prior to sensitivities becoming available and, having started it and reached the determination for a limited course, it was reasonable to complete it with this antibiotic.”

The infectious diseases specialist advised ACC that it is unlikely that the short course of Augmentin in any way compromised Mr A's subsequent treatment, and that once the diagnosis of endocarditis was made he would have been given six weeks of penicillin and gentamicin irrespective of the preceding short course of Augmentin.

Dr Mann advised:

“With recurrence of the infection later, the formal choice, dosage and monitoring of long-term antibiotic therapy would normally be done in collaboration with an infectious disease consultant. Starting the patient on high-dose intravenous penicillin and Gentamicin before transfer was, however, quite appropriate.”

He went on to say that detailed discussion of antibiotic choices with the patient would not usually be undertaken at this stage although some outline of the treatment plan should be given. Dr Mann noted that, even in retrospect, although there was clearly a septicaemia with a focus of infection that was resistant to a conventional course of broad-spectrum antibiotics, the diagnosis of endocarditis cannot be made with certainty.

Dr Mann advised that once the septicaemia was identified there was no “deficient or clearly incorrect treatment”. He agreed with Dr Chambers that the commencement of the penicillin and gentamicin in the second public hospital was “completely conventional”, but noted that the long term consequences for Mr A of the gentamicin therapy were “deeply unfortunate”. Dr Mann stated:

“I believe that responsibility for keeping [Mr A] informed of the risks from continuing gentamicin therapy lies much more with [the city hospital] given the relative duration of this treatment in the two institutions.”

Mr A was kept informed of the decisions regarding his management, including the two short courses of antibiotic therapy commenced by the second public hospital in April and May 2004, which it appears were critical to his survival when he became severely unwell. Long-term antibiotic therapy was later undertaken at [the city hospital]. I accept the advice of my expert that the gentamicin therapy at the second public hospital was relatively short and that the responsibility for keeping Mr A informed about the risks from continuing gentamicin therapy lies more with the city hospital.

It appears that there was wide consultation with a variety of specialists in the search for the cause of Mr A’s symptoms. In my view, the staff at the first and second hospitals made every endeavour to provide continuity of quality care to Mr A, under unusual and difficult circumstances, and to keep him informed of all aspects relating to his care. Accordingly, in my opinion, the District Health Board did not breach the Code.

#### *Vicarious liability*

Under section 72(2) of the Health and Disability Commissioner Act 1994, employers are vicariously liable for ensuring that employees comply with the Code. Under section 72(5) it is a defence for an employing authority to prove that it took such steps as were reasonably practicable to prevent the employee from doing or omitting to do the things that breached the Code.

As previously mentioned, the management of Mr A’s deteriorating condition was complex. He was assessed and managed by orthopaedic, cardiac and gastrology teams in two different hospitals, and in between admissions was being monitored by his general practitioner. Health care delivery in a hospital setting encompasses complex and often overlapping processes. At or between any of these points in the delivery of



care, delayed or neglected actions, technical or judgmental errors or communication failures may result in potential or actual adverse consequences for the patient.

It appears that Dr B and Dr C communicated appropriately with other medical staff in their search for an answer for the cause of Mr A's problems, but the numerous "distractors", described by the experts, contributed to the delay in the diagnosis of Mr A's septicaemia.

Dr C and Dr B were employed by the District Health Board. As an employer, the District Health Board is potentially vicariously liable for Dr C's breach of the Code. Dr C's decision not to follow up the possibility of infection as queried by the radiologist was a clinical one. In my view, there was no apparent systems failure that contributed to this situation, and I am satisfied that Dr C's omissions were beyond the scope of what the District Health Board, as an employer, could have prevented at a systems and management level, and are not matters for which it could reasonably be held liable.

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### **Other comment**

*Cole's Medical Practice in New Zealand* (Medical Council of New Zealand, 2001) states that the keeping of a proper medical record is "an important part of a good doctor-patient relationship" and is a tool for management and communicating with other health professionals, and a primary tool for the continuity of care.

Dr Tregonning commented that Dr B's recording of his consultations with Mr A was at times deficient, and noted a number of omissions. Dr Tregonning stated that when Mr A was initially seen by Dr B at the second public hospital on 10 February his records did not include the injury to Mr A's foot or the findings of the general examination or his lower back assessment. On 4 March Dr B's record of his assessment of Mr A did not include his clinical findings. There was no documentation or acknowledgement of Mr A's weight loss, which was considerable by this stage, and there was no documentation of the findings of the spinal examination. Dr Tregonning noted that Dr B informed Mr and Mrs A of the need for further investigations to rule out occult pathology, but in his letter of 26 March to Dr F he did not detail his clinical findings, the elevated ESR, Mr A's weight loss of two stone or the investigations into his anaemia.

It is important for clinicians to document in such a way that another doctor reassessing the patient would be able to make an adequate comparison between the previous and presenting clinical pictures. Although I appreciate that Dr B was focused on Mr A's spinal problems, the other potential contributors to Mr A's symptoms should have been clearly identified so that other clinicians involved in his care had the opportunity to evaluate the significance of these issues. I encourage Dr B to reflect on Dr Tregonning's criticism of his record-keeping.

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## **Recommendation**

I recommend that Dr C:

- Apologise in writing to Mr A for his breach of the Code. The apology is to be sent to the Commissioner's Office and will be forwarded to Mr A.
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## **Follow-up actions**

- A copy of this report will be sent to the Medical Council of New Zealand, the Royal Australasian College of Physicians, and the Royal Australasian College of Surgeons.
- A copy of this report, with identifying features removed, will be placed on the Health and Disability Commissioner website, [www.hdc.org.nz](http://www.hdc.org.nz), for educational purposes.