EXHIBIT BL

CARDIOLOGY

Dr Hariharan Raju

MQ Health
MACQUARIE UNIVERSITY
HEALTH SCIENCES CENTRE

Date: 18 April 2019

Dr Stuart Gray
Cardillo Gray Partners
PO BOX 409
NEWCASTLE NSW 2300

Dear Dr Gray

Re: Ms Kathleen FOLBIGG DOB: 14-6-1967 (ID: 367553)

I reviewed this 51-year-old lady again with respect to her family history and symptoms at MQ Health Cardiology today.

Since my original consultation at Silver Water Women's Correctional Centre on 20 February 2019 (as detailed in prior correspondence dated 21 February 2019), I have now performed further investigations.

I note she had an ECG undertaken on 24 March 2003 which shows sinus rhythm of rate 50 beats per minute with normal P and QRS morphology, borderline QT (460 ms) with notched inferior T-waves and small U-wave seen both in the precordium and inferior leads. I am uncertain as to the relevance of this. A subsequent ECG undertaken on 24 December 2018 was entirely normal as was her echocardiogram on 22 February 2019.

Today I proceeded to perform postural ECG and exercise provocation (recorded under MRN 54321).

Her resting ECG was entirely within normal limits with sinus rhythm of rate 67 beats per minute, with normal P and QRS morphology, normal PR and QT interval, no pathological changes of repolarisation. A modified ECG (precordial leads cranially displaced with V1 and V2 into the second, third and fourth intercostal spaces) showed no evidence of a Brugada phenotype. Immediately following standing she had appropriate QT adaptation, with no absolute QT prolongation.

She managed 12.30 minutes of a Bruce protocol treadmill exercise provocation test, reflecting an excellent workload of 12.7METS and achieving peak heart rate of 173 beats per minute (102% maximal age predicted) which represented her peak exercise capacity. She had a moderate hypertensive response during exercise (baseline 110/80 mmHg, peak 200/80 mmHg) with a precipitous drop immediately post exercise (130/80 mmHg). During exercise she had an appropriate chronotropic and dromotropic response with multiple episodes of likely artefact during exercise but possible isolated ectopics of LVOT origin. There were no exertional or post exertional sustained or non-sustained arrhythmias and no pathological changes of repolarisation during the test; non-specific upsloping ST depression is seen at peak exertion of maximal 0.01mV. There were no ectopics beyond 10 seconds of recovery and although her blood pressure fell very early post exercise her heart rate remained 119 at four minutes post exercise. At that point, her absolute QT was 320 ms which corrects to 451 (Bazett). In summary, this is an unremarkable study,

apart from the presence of possible isolated LVOT ectopy at excellent workload.

She had a 24-hour ambulatory Holter monitor fitted at the same time. I requested that she be allowed to perform normal activities in the correctional facility while wearing this monitor, although prior arrangements had been made for prison clinic observation. The results of this will be available to me in the coming days.

I also took the opportunity to discuss a further episode of syncope with her. In her youth (around age 12) she describes losing consciousness after a swimming event. She believes she swam 25 metres, completed the race and her next recollection is recovering consciousness on the side of the pool. She does not recall any other events but it is clear that she was helped out of the pool on that occasion.

The post exertional (rather than peak exertional) nature of her syncopal episodes, together with multiple episodes being recorded with a prolonged period of recovery are consistent with reflex or neurocardiogenic aetiology. This is in keeping with her post exercise significant drop in blood pressure which is seen commonly in trained individuals; certainly her excellent exercise capacity of 12.7 METs is consistent with training.

It was also helpful to review the autopsy reports from her four children. All had normal cardiac post-mortem findings, although I note the description of lymphocytic infiltration associated with myocyte necrosis in Laura. Non-specific histologic changes such as lymphocyte infiltration are associated with sudden unexplained death (SUD) in the context of primary arrhythmia syndromes and are not necessarily diagnostic of myocardial disease as the primary cause of death. However, the association with surrounding necrosis is suggestive of myocarditis.

With respect to further investigation of Kathleen, my opinion is that the likely yield of cardiac pathology will be negligible. Given her as yet recurrent unexplained syncope, prolonged heart rhythm monitoring with implantable loop recorder may be prudent if her 24-hour ambulatory Holter monitor fails to document any pathological arrhythmia. This offers potential for symptom-rhythm correlation (or absence thereof). Additional investigations may be performed if clinical suspicion of cardiogenetic disease changes in the future.

For example, cardiac MRI may very rarely establish a diagnosis of subtle cardiomyopathy in the presence of normal ECG and echocardiography. Drug provocation testing with ajmaline and/or epinephrine may unmask concealed arrhythmia syndromes. However, her family history is very unusual for familial Brugada syndrome; childhood onset of fatal events in children, even in sleep, is very rare in Brugada syndrome. Additionally, most forms of CPVT and LQT are rarely associated with death in sleep. Moreover, the role of epinephrine testing remains controversial in the diagnosis of CPVT and LQT; in the absence of other clinical indicators of the diagnosis, I am not certain that it will offer any additional diagnostic yield.

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I have shared the results of investigations performed today with Prof Skinner in line with your and the patient's request.

Addendum 24 April 2019:
24 hour Holter monitor showed: Sinus rhythm throughout with appropriate spread in heart rate histogram. Very rare (<0.01%) late-coupled (>400ms) ventricular ectopy, with axis consistent with outflow tract origin. Very rare (<0.01%) atrial ectopy. No sustained or non-sustained arrhythmia. No significant ventricular pauses. No symptomatic patient activations. Summary: normal Holter monitor in absence of patient symptoms.

In summary, following comprehensive non-invasive evaluation, Kathleen has no phenotypic evidence of either cardiomyopathy or primary arrhythmia syndrome. The only abnormality detected is the presence of possible exertional ventricular ectopy which is consistent with an idiopathic focus and likely of no clinical relevance. The borderline repolarisations changes seen on her resting ECG in 2003 are also not diagnostic of pathology. Her multiple syncopal episodes are likely to be of reflex aetiology, which is benign. As previously stated, identification of a cardiogenetic cause of sudden death in Kathleen's children warrants similar comprehensive evaluation of their father.

Yours sincerely,

Electronically Approved by
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