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George Gore, Esq.
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Re: Estate of Michael Carrick vs. Cleveland Clinic Foundation et al.

Dear Mr. Gore:

I have reviewed the documents which you sent to me regarding the above-captioned case. These include the medical records of Michael Carrick from Dr. Riley, Lakewood Hospital, and the Cleveland Clinic, as well as depositions by Drs. Riley, Bralliar, Broughan, Heyka, and Nakamoto. As we discussed over the telephone and during our meeting in January 1991, I believe the care provided to Mr. Carrick by the Cleveland Clinic and its staff was consistent with accepted standards of care, and that there should be no cause for action against these defendants. The medical history is, indeed, a complicated one. Mr. Carrick suffered a number of unfortunate complications of his disease, but I do not believe that any of these were the result of any acts of negligence on the part of the Cleveland Clinic or its staff. There remains some controversy as to the exact nature of some of these complications, including the etiology of Mr. Carrick's renal disease itself. I will begin by attempting to sort out some of these controversial issues, then I will address the questions which you posed to me in your letter of December 7, 1990. (1)

Prior to his admission to the Cleveland Clinic, Mr. Carrick had been cared for by Dr. Robert Riley for hypertension, gout and progressive renal insufficiency. The etiology of this renal insufficiency is not clear from Dr. Riley's medical records nor from his deposition. It was presumed to be some form of chronic interstitial nephritis, compounded by hypertension. The exact nature of interstitial nephritis was never documented by renal biopsy nor attributed to any specific etiologic agent. It was noted that Mr. Carrick did have one shrunken kidney, although it is not clear whether this represented atrophy secondary to renal vascular disease or a congenital abnormality. Mr. Carrick did have difficult-to-control hypertension, and so the possibility of renal vascular disease had been considered by Dr. Riley, but never pursued with angiographic studies. Renal artery stenosis can lead to progressive renal insufficiency on the basis of decreased blood flow to the kidney; in some of these cases, control of the blood pressure may actually compound the renal dysfunction by further compromising renal blood flow. Patients with congenital dysplastic or hypoplastic kidneys will often develop disease later in life in the contralateral kidney, usually presenting with protein in the urine, then progressing to end-stage renal disease over five to ten years. No mention is made of the presence or absence of protein in Mr. Carrick's urine, although it is clear that Mr. Carrick did not have heavy protein loss in his urine as his serum albumin remained normal. (2) (3) ?

Mr. Carrick was being treated by Dr. Riley for gout on the basis of recurrent acute arthritis in the setting of an elevated uric acid level.

However, the only way to make a definitive diagnosis of acute gouty arthritis is to demonstrate uric acid crystals in the fluid of an affected joint. I find no evidence that this was ever done in Mr. Carrick. Moreover, I suspect that as Mr. Carrick's "gout" became more and more refractory to therapy in 1988 and 1989, he was actually suffering from pseudogout, the deposition of calcium pyrophosphate crystals in the joints. Pseudogout, as the name suggests, can mimic gout, and is often seen in the context of chronic renal failure, especially among patients whose serum phosphorous levels are markedly elevated as was Mr. Carrick's. I can understand Dr. Riley's frustration in being unable to control Mr. Carrick's "gouty" symptoms using conventional anti-gout therapy such as allopurinol, indomethacin, and colchicine. In such a context, Dr. Riley's resorting to the use of prednisone to control what must have been an extremely severe joint pain experience by Mr. Carrick is understandable, although somewhat unusual. The use of prednisone in 1989 almost certainly contributed to the marked elevation of Mr. Carrick's BUN out of proportion to his serum creatinine and may have also contributed to the elevation of CPK which was noted at Lakewood Hospital. But more significantly, the failure of Mr. Carrick's diffuse bone and joint pain to respond to therapy as radical as prednisone underscores the advanced nature of his bone and joint disease and indicates that parathyroidectomy was the only therapeutic alternative.

Whether or not Mr. Carrick's intermittent treatment with nonsteroidal anti-inflammatory agents such as Indocin and Naprosyn might have contributed to his chronic renal failure is unclear. It is known that continuous use of these agents may lead to a chronic interstitial nephritis with progressive renal insufficiency. However, it appears from Dr. Riley's records and deposition that Mr. Carrick was treated only intermittently with these agents, which is not generally associated with progressive renal damage. That is not to say that these agents cannot produce a form of acute renal damage, either through an adverse hemodynamic change within the kidney or through a form of acute interstitial nephritis. Both of these forms of acute renal injury are generally reversed upon discontinuation of the drug, so it is difficult for me to state that Dr. Riley's prescription of these agents was a proximate cause of Mr. Carrick's progressive renal failure.

Mr. Carrick was transferred from Lakewood Hospital to the Cleveland Clinic on March 28, 1989 in terrible shape. His musculoskeletal pain was so severe that it required parenteral narcotics for control. X-rays showed metastatic calcification throughout many muscle planes of the body as well as marked renal osteodystrophy. His parathyroid hormone level was spectacularly elevated at 1650, so there was no question that parathyroidectomy was indicated. His serum phosphorous was elevated at 8.4 with a serum calcium of 7.7. Since the calcium-phosphorous product of greater than 60 is felt to contribute to metastatic calcifications, it was felt to be imperative that his serum phosphorous be reduced with a phosphate binder. The use of an aluminum containing phosphate binder would be preferred to a calcium containing phosphate binder at this point, as the use of the latter would raise the serum calcium and might not decrease the product prior to reducing the phosphorous level. Therefore, it was appropriate to choose Dialume 2 tabs. tid as the phosphate binder, in an attempt to reduce the serum phosphorous level. Once the phosphorous was reduced, then it would be appropriate to raise the serum calcium using calcium supplements as well as Vitamin D. However, it should be pointed out that all medical therapy, including dialysis, use of phosphate binders, calcium supplements, Vitamin D, or whatever, would not have had a significant impact on Mr. Carrick's painful and debilitating osteodystrophy. The only way to turn off the markedly elevated parathyroid hormone level that was actively consuming his bones was to perform a parathyroidectomy and, appropriately, Mr. Carrick was scheduled for readmission for such a procedure.

Mr. Carrick was readmitted to the Cleveland Clinic on April 10, 1989 for total parathyroidectomy with autotransplant of the right upper and lower

glands as described by the operative note. Preoperatively, his lungs were clear and his physical exam revealed no signs of fluid overload or congestive heart failure. Preoperative labs were remarkable for a serum sodium of 121, potassium 5.5, bicarbonate 12, BUN 98, and creatinine 6.2. Calcium and phosphorous were 8.1 and 9.3, respectively, with a markedly elevated product, further underscoring the failure of medical therapy to control Mr. Carrick's hyperparathyroidism, osteodystrophy, or metastatic calcifications. I consulted with our own transplant surgeon, Dr. James Schulak, regarding whether Mr. Carrick's preoperative chemistries would have constituted a contraindication to surgery or would have required preoperative dialysis treatment. Dr. Schulak, who performs the majority of parathyroidectomies among renal failure patients at our hospital, said that azotemia (a high BUN) itself would not be a contraindication to parathyroidectomy. Even if one were to argue that a high BUN would contribute to a platelet defect and an increased risk of bleeding, hemostasis is generally easy to achieve in a parathyroidectomy as the surgical site is small and not highly vascular. Dr. Schulak also did not feel that a bowel prep would be indicated for a renal failure patient undergoing parathyroidectomy as the procedure is not generally associated with postoperative ileus. The decision to perform dialysis in such a setting is a medical one, dictated by any complications which the patient may be experiencing as a result of the renal failure, not by the prospect of surgery. As Mr. Carrick was not suffering any clear complications of his renal failure prior to his parathyroidectomy that could be expected to be immediately reversed by dialysis, the decision not to perform dialysis preoperatively is justified.

Postoperatively, the patient continued to complain of severe pain secondary to his polyarticular arthritis. A rheumatology consult was obtained and aspiration of the right knee joint revealed calcium crystals, consistent with a diagnosis of pseudogout, not gout. The patient continued to be treated conservatively for his renal failure during the first four days of his hospitalization, which is understandable since most patients with a serum creatinine of 6.2 have significant residual renal function and do not require dialysis. The decreased serum sodium can be treated with fluid restriction, the increased serum potassium can be treated with dietary potassium restriction and diuretics, and a decreased bicarbonate level can be treated with bicarbonate supplements. However, on April 14, it became clear that the patient's renal function was continuing to decline, and he was begun on hemodialysis therapy. It was also noted on that date that he had developed right lower lobe pneumonia, and appropriate antibiotic therapy was initiated. I do not believe that the failure to dialyze Mr. Carrick before April 14 was a proximate cause of his development of pneumonia. Patients with advanced renal failure are known to have decreased leukocyte function and to be at higher risk for the development of pyogenic infections. Some European studies in patients with acute renal failure have suggested that more intensive dialysis may improve mortality by decreasing the risk of infection. However, I am aware of no clinical studies that have demonstrated convincingly what early or aggressive dialysis for the sole purpose of improving host defenses significantly decreases the incidence of infection or improves mortality. Therefore, Mr. Carrick's physicians cannot be faulted for failing to dialyze Mr. Carrick earlier for this reason.

On April 15, 1989, Mr. Carrick developed acute dilatation of the colon requiring an emergency decompressive colonoscopy. It has been alleged by the plaintiff's expert witness that the Dialume which Mr. Carrick was receiving as well as the failure to perform a bowel prep preoperatively contributed to the development of the colonic dilatation. Indeed, Dialume is associated with constipation and often requires the concomitant use of stool softeners and cathartics. However, to implicate Dialume would also require that the patient had developed obstipation as a cause of his colonic dilatation, and this was not found to be the case. Patients with a number of chronic illnesses, including renal failure, have been known to develop a nonobstructive colonic

dilatation called Olgilvy's syndrome, sometimes following a surgical procedure. This is unrelated to the use of Dialume, bowel preps, or even the nature of the surgery, cannot be predicted, and cannot be prevented. When it does occur, it is corrected as in Mr. Carrick's case with a decompressive colonoscopy.

Mr. Carrick's condition continued to decline as he developed pseudomonas sepsis and respiratory failure requiring mechanical ventilation. On April 23, 1989, he was found to have a pathological femur fracture which is unquestionably related to his underlying renal osteodystrophy, and not the consequence of any other care which he received at the Cleveland Clinic. Ultimately, the patient developed multi-system failure and expired on May 17, 1989. I can understand the anger and frustration experienced by Mr. Carrick's family following this extremely unfortunate outcome, but I can find no evidence that any failure to adhere to a standard of care by the Cleveland Clinic or its staff was a proximate cause of any of the complications which Mr. Carrick experienced and which ultimately led to his death.

In the above context, the answers to the questions which you posed to me in your letter of December 7, 1990 should be evident:

1) Dr. Gorbaty, expert witness for the plaintiff, states that standard medical treatment, including dialysis, probably would have reversed Mr. Carrick's disease process in 6 to 12 months. The facts of the case simply do not support this. Mr. Carrick's renal osteodystrophy and metastatic calcification was so severe that he required narcotic analgesics for pain control. He was totally debilitated by his disease. His parathyroid hormone level was so high as to almost be out of measurable range and was clearly contributing to the rapid destruction of Mr. Carrick's remaining bones. Medical therapy, if initiated early in the course of Mr. Carrick's renal disease, perhaps when his serum creatinine was around 2, might have prevented the severe hyperparathyroidism and the renal osteodystrophy. However, at the time Mr. Carrick presented in the Cleveland Clinic, his bones were in extremis. He could no longer afford the luxury of waiting to see whether medical therapy would work. Appropriate medical therapy might have slowed down the further progression of his osteodystrophy and metastatic calcification, but to state that it would have reversed it is ludicrous. Mr. Carrick clearly needed the parathyroidectomy which he received.

2) Dr. Gorbaty claims that Mr. Carrick should have been dialyzed preoperatively because his BUN in excess of 100 placed him at greater risk for bleeding. First of all, since Mr. Carrick did not bleed perioperatively, to state that the failure to dialyze him preoperatively was the proximate cause of any complication which he experienced is simply not supported by the facts. Furthermore, as I mentioned above, Dr. James Schulak disagrees with Dr. Gorbaty's contention and states that he will routinely operate on patients with a BUN greater than 100, especially if the surgery such as a parathyroidectomy is not a particularly bloody one. Finally, the correlation between the bleeding time, the single best in vivo indicator of platelet function, and the BUN in renal disease patients has classically been poor. Patients with comparable levels of platelet dysfunction due to other reasons, such as aspirin therapy, are generally not considered contraindicated for surgery. Does Dr. Gorbaty suggest that any patient who has received aspirin and who has a bleeding time similar to that which might be encountered in a renal failure patient be forced to wait one week for the aspirin to wear off before undergoing elective surgery?

3) Dr. Gorbaty alleges that the postoperative ileus that Mr. Carrick experienced was due to the Dialume which he had been receiving and that Mr. Carrick should have undergone a bowel prep prior to the parathyroidectomy. Dialume is associated with constipation, but there is no evidence that Mr. Carrick's postoperative ileus was in any way related to obstruction by fecal

material. The Dialume was indicated medically for treatment of Mr. Carrick's hyperphosphatemia. Dr. Gorbaty stated that Mr. Carrick should have been given adequate trial of medical therapy before being subjected to parathyroidectomy. If Dr. Gorbaty would not allow for Dialume to be used because of its association with constipation, what would he have used for treatment of the hyperphosphatemia? It seems that Dr. Gorbaty would like to condemn the Cleveland Clinic and its staff for treating Mr. Carrick surgically or medically. Dr. James Schulak states that it is almost unheard of to have a patient undergo a bowel prep prior to a parathyroidectomy as such a procedure is really associated with postoperative ileus. When bowel preps are performed preoperatively, they are done so to prevent obstructive ileus related to decreased bowel motility. There is no evidence that Mr. Carrick had an obstructive ileus, therefore whether or not he received a bowel prep prior to his parathyroidectomy is moot.

4) Dr. Gorbaty makes the preposterous statement that Mr. Carrick's postoperative ileus was due to obstruction by "rocks" composed of phosphate binders. There is absolutely no evidence in the medical record to support this allegation.

5) Dr. Gorbaty states that the failure to dialyze Mr. Carrick preoperatively may have contributed to his postoperative pneumonia. This is a very difficult allegation to prove or disprove. Indeed, there have been some studies that have suggested that patients with acute renal failure may have a lower incidence of infections and improved mortality when intensively dialyzed. However, this is a completely different patient population from those with chronic renal failure, and extrapolations may not be valid. I am familiar with no clinical studies in patients with chronic renal failure that suggest that early or more aggressive dialysis decreases the incidence of infectious complications. Mr. Carrick had a number of medical conditions which may have predisposed him to developing pneumonia aside from his renal failure. He was in severe pain secondary to his osteodystrophy and metastatic calcifications and may not have been able to take appropriate deep breaths or coughs. He was also being medicated with narcotic analgesics for his pain, which would have also decreased his cough reflex and his ability to expectorate respiratory secretions. I believe that these factors contributed to his development of pneumonia much more than the fact that he was not dialyzed preoperatively. Therefore, the decision not to dialyze Mr. Carrick preoperatively was not a proximate cause of his development of pneumonia.

6) Dr. Gorbaty has suggested the hip fracture which occurred postoperatively was contributed to by the aluminum which Mr. Carrick was receiving in the form of Dialume. First of all, this is an outrageous suggestion as it takes several years of aluminum therapy to accumulate sufficient aluminum within the bone to place the bone at risk for fracture. Secondly, checking an aluminum level would have no meaning in the setting of Dialume therapy of relatively short duration. Thirdly, Dr. Gorbaty would again like to have his cake and eat it too. He condemns the Cleveland Clinic and its physicians for not giving Mr. Carrick an adequate trial of medical therapy in the form of aluminum containing phosphate binders even though this is the only appropriate method of reducing Mr. Carrick's serum phosphorous and calcium-phosphate product in an attempt to decrease his metastatic calcifications and suppress his secondary parathyroidism.

7) Dr. Gorbaty states that Mr. Carrick suffered a hearing loss which was almost certainly caused by administration of aminoglycoside antibiotics. Mr. Carrick had a life-threatening pseudomonas septicemia, the treatment for which was aminoglycoside antibiotics. Aminoglycosides are the most effective antibiotic agents available against pseudomonas infections. Occasionally, physicians will avoid their use in patients with chronic renal insufficiency as the nephrotoxicity of these agents may further compromise renal function. Mr. Carrick was already receiving chronic dialysis treatment and, therefore,

the effect of aminoglycoside antibiotics on his renal function was not a great consideration. To withhold aminoglycoside antibiotics in the setting of the pseudomonas septicemia because of the effect these agents might have on his hearing would be a most inappropriate assessment of priorities, and would be tantamount to malpractice. Mr. Carrick's physicians appropriately chose aminoglycoside antibiotics in the setting of a life-threatening pseudomonas infection and, even if it could be proven that these agents had an adverse effect on Mr. Carrick's hearing, their use would still be the standard of care.

I apologize for my delay in sending you this written response to the issues you have raised in connection with this case, and I hope that my opinions will be useful in the defense of your clients. If I can be of any further service, please do not hesitate to call.

Sincerely yours,

Jay B. Wish, M.D.
Associate Professor of Medicine

JBW:nm