

NOTICE OF PUBLICATION BAN

In the College of Physicians and Surgeons of Ontario and Dr. Albert Ross Deep, this is notice that the Discipline Committee ordered that no person shall publish or broadcast the identity of the patients or any information that could disclose the identity of the patients under subsection 45(3) of the Health Professions Procedural Code (the “Code”), which is Schedule 2 to the *Regulated Health Professions Act, 1991*, S.O. 1991, c. 18, as amended.

Subsection 93(1) of the *Code*, which is concerned with failure to comply with these orders, reads:

Every person who contravenes an order made under section 45 or 47 is guilty of an offence and on conviction is liable to a fine of not more than \$10,000 for a first offence and not more than \$20,000 for a subsequent offence.

Indexed as: Deep (Re)

**THE DISCIPLINE COMMITTEE OF THE COLLEGE
OF PHYSICIANS AND SURGEONS OF ONTARIO**

IN THE MATTER OF a Hearing directed
by the Executive Committee of the College of Physicians
and Surgeons of Ontario, pursuant to Section 36(1)
of the **Health Professions Procedural Code**,
being Schedule 2 to the
Regulated Health Professions Act, 1991,
S.O. 1991, c.18, as amended

B E T W E E N:

THE COLLEGE OF PHYSICIANS AND SURGEONS OF ONTARIO

- and -

DR. ALBERT ROSS DEEP

PANEL MEMBERS:

**DR. M. GABEL
R. PRATT
DR. N. DE
J. DHAWAN
DR. T. MORIARITY**

Hearing Dates: April 30- May 4, 15-18, June 18-20, September 4-7,
10 & 11, 2007
Decision Date: April 29, 2008
Written Reasons Date: April 29, 2008

PUBLICATION BAN

DECISION AND REASONS FOR DECISION

The Discipline Committee of the College of Physicians and Surgeons of Ontario (the “Committee”) heard this matter at Toronto on April 30 to May 4, May 15 to May 18, June 18 to June 20, September 4 to September 7 and September 10 and 11, 2007. At the conclusion of the oral hearing, the Committee provided for additional time for the filing by Dr. Deep of written material, and allowed the College a right of reply. After the conclusion of this process, the Committee reserved its decision.

THE ALLEGATIONS

The Notice of Hearing alleged that Dr. Deep committed an act of professional misconduct:

1. under paragraph 27.21 of Ontario Regulation 448/80 and paragraph 29.22 of Ontario Regulation 548/90 made under the *Health Disciplines Act*, R.S.O. 1980 and paragraph 1(1)2 of Ontario Regulation 856/93 made under the *Medicine Act*, 1991, in that he failed to maintain the standard of practice of the profession.

The Notice of Hearing also alleged that Dr. Deep is incompetent as defined by subsection 52(1) of the Health Professions Procedural Code (the “Code”), which is schedule 2 to the *Regulated Health Professions Act, 1991*, subsection 60(4) of the *Health Disciplines Act*, R.S.O. 1980, c.196, and subsection 61(4) *Health Disciplines Act* R.S.O. 1990 c. H.4 and in that his care of patients displayed a lack of knowledge, skill or judgment or disregard for the welfare of his patients of a nature or to an extent that demonstrates that he is unfit to continue practise or that his practice should be restricted.

RESPONSE TO THE ALLEGATIONS

Dr. Deep denied the allegations set out in the Notice of Hearing.

RESERVE OF THE DECISION

The Committee reserved its decision at the end of the evidentiary phase of the hearing. We were aware that due deliberation on the allegations and the reasons that would support the decision would require a considerable amount of time, having regard to the complexity of the case and the extent of the evidence. The evidence consisted of testimony that spanned 18 hearing days. The Committee was presented with medical records that occupied six banker boxes and consisted of thousands of pages per patient, often double sided and in no easily apparent order, as we have noted in the reasons below. As well, there was a 204 page expert report, and Dr. Deep's response to it, including three volumes of additional submissions concerning the records. Additionally, more than 50 complex journal articles and reports were submitted among the 70 exhibits, many articles comprising a single exhibit. We would also note, as described below, that based on the final argument procedure that the Committee put into place to protect Dr. Deep's rights, there was an additional delay beyond the conclusion of the formal hearing to permit Dr. Deep the opportunity to file further responding material in response to the College's written submission. The Committee applied itself assiduously to review all of the evidence and the conflicting testimony on the interpretation of the evidence, and realized that an extended time frame for deliberation would be necessary to do justice to the many issues that had to be determined on the extensive hearing record.

EVIDENCE

Overview

The College presented the following evidence in support of the allegations:

- a. Oral evidence of expert witness, Dr. Z
- b. Expert report of Dr. Z (Exhibit 4)
- c. Written report "For Dr. Deep, General Questions" (Exhibit 8)
- d. Photocopied charts of 25 patients
- e. OHIP records of the patients (Exhibit 3)
- f. Multiple exhibits comprised of published literature on the areas under examination

- g. Policies of the CPSO addressing the area of medical records (Exhibits 63 and 64)
- h. Other exhibits addressing the issues the Committee had to decide

Dr. Deep presented the following evidence in defence of the allegations:

- a. Oral testimony as a fact witness
- b. Oral testimony as an expert witness
- c. Written expert evidence and report (Exhibit 5)
- d. Report entitled “Report of Albert R. Deep, M.D., F.R.C.P.” (to the Executive Committee of the College of Physicians and Surgeons of Ontario, Volumes I, II, and III (Exhibits 6A, B and C)
- e. 32 articles and reports bound as “Provided by Dr. Deep” (Exhibit 45)
- f. Additional articles from medical literature and reports (multiple exhibits)

Dr. Deep was self-represented. Prior to the hearing, Dr. Deep received a letter detailing the procedures in a discipline hearing from the Hearing Office of The College of Physicians and Surgeons of Ontario. The Chair took care to explain, throughout the proceedings, the procedures of the Committee, Dr. Deep’s rights and obligations, and the roles of the Committee and independent legal counsel (“ILC”).

At the commencement of the hearing, the Chair detailed Dr. Deep’s rights and responsibilities, as well as the Discipline Committee procedures. Dr. Deep received a copy of this presentation.

The Chair informed Dr. Deep that he would be advised and guided as to procedure, but the Committee could not advise him as to strategy and the content of his presentation. That said, during the hearing, the Committee was sensitive to Dr. Deep’s rights at all times.

At the conclusion of the College's evidentiary presentation, Dr. Deep informed the Committee that he would not be presenting oral evidence in his defence. The Chair informed Dr. Deep that this was a serious decision for him to be taking, and the Committee would not accept this as his final decision. The Committee advised him to consider the matter overnight, to consult counsel and others as he felt necessary, and it would hear his decision the following day. Dr. Deep reconsidered the matter and chose to present evidence in his defence.

The College, in summation, presented a written outline as part of its concluding arguments, as an aid to the Committee to follow the oral submission. Dr. Deep objected, stating he had only received notification at the conclusion of evidence that the College might utilize a written supportive document. He also objected to written material being part of the oral argument. The Committee ruled that it is a usual and accepted procedure in CPSO discipline hearings for the College and the defence to present written outlines of their submissions. The Committee is aware that these outlines are not evidence. Dr. Deep had the opportunity to do the same, especially as he was informed that the College might well be doing so. Notwithstanding that ruling, the Committee decided that, in the interests of fairness to an unrepresented physician, it would alter its procedure to allow Dr. Deep, if he chose, to submit his own written supportive document within seven days following the conclusion of the hearing, with the College having the right to reply in writing within seven additional days. At the conclusion of Dr. Deep's oral argument, he stated that he would not present such a document, as his oral presentation ought to be sufficient. The Committee acknowledged this decision, but refused to close this avenue and still gave Dr. Deep the seven days to reconsider his decision. Dr. Deep did reverse his stated position and submitted, within the required time, a written document in support of his position at the hearing. The College declined to respond to this document, as was its right under the Committee's ruling. This added an additional time period before the Committee would be in a position to deliberate on the totality of the evidence and the submissions of the parties.

While Dr. Deep was self-represented, the Committee is of the opinion that, with the guidance of the Chair and the assistance of the ILC, Dr. Deep was accorded assistance and latitude as a self-represented physician. As well, Dr. Deep informed the Committee that he had legal representation but freely chose to dismiss legal counsel assisting him, and that he had represented himself in other legal venues.

Evidentiary considerations

In making a decision concerning the allegations, the evidence for each patient was considered, with emphasis on the areas noted below. A more complete analysis of certain specific areas was undertaken. The standard of proof utilized by the Committee was the *Bernstein* standard of the balance of probabilities based on clear, cogent and convincing evidence. We considered it imperative to analyze the evidence in light of the standard of practice in each of the areas under review at the time that the patients in question were under Dr. Deep's care.

The following areas of concern were identified:

1. The status and state of the equipment used in office diagnosis.
2. Dr. Deep's medical records, their physical state and content including:
 - a. Evidence of communication with other physicians and medical teams;
 - b. Assessment, treatment plans and medication records;
 - c. Appropriate office based diagnostic testing;
 - d. Communication with patients regarding treatment and advice;
 - e. Appropriate documentation of psychotherapy;
 - f. Arrangement and content of the charts;
 - g. Acknowledgement of laboratory reports and test results; and
 - h. Ability of the chart to provide evidence of proper care of the patient, including the information needed to meet the standard of practice.

3. The use of anti-arrhythmic medications, especially in patients with structural heart disease or other high risk factors, with specific emphasis on the medication, Propafenone.
4. The pattern of prescription of Vioxx following the addition of “black box” warnings.
5. The diagnosis and treatment of hypertension.
6. The diagnosis, assessment, monitoring and follow-up of renal function.
7. The appropriateness of clinical examinations and laboratory tests performed prior to instituting treatment.
8. The appropriate, timely and acceptable monitoring of patients’ medical status and of instituted therapies.
9. The treatment and monitoring of lipid abnormalities.
10. The use and monitoring of anticoagulants.
11. The monitoring of potassium levels and of kidney function.
12. The monitoring and diagnosis of glucose metabolism.
13. If Dr. Deep assumed the multiple roles of a consulting cardiologist, internist and family physician, did he meet the standards of practice and was he competent.

Some of these issues apply to all the charts reviewed and are a background to an overview of the basic mechanics of Dr. Deep’s practice. This applies to the areas of medical records, psychotherapy, office administrative procedures and office equipment.

In other areas, it is possible to utilize the evidence concerning a specific patient when examining a specific practice pattern.

The Committee chose, in the interest of clarifying the issues and making a decision concerning the allegations, to proceed in the following manner. A number of the areas listed above are presented as individual topics with evidence from patients that are most applicable to that issue. We chose to evaluate in depth those specific issues, as they represent discrete areas of concern that allowed the Committee to evaluate Dr. Deep's practice. We follow this with a presentation of each patient whose charts were the subject of this hearing.

We chose to present the Committee's conclusions concerning the allegations in each area and with each patient.

The Evidence

The College called Dr. David Z as an expert witness. Dr. Deep testified as an expert witness in his own defence, but called no other witnesses.

Dr. Z

The College called Dr. Z as their expert witness. His education and experience was presented to the Committee (Exhibit 7). Dr. Z is an Associate Professor of Medicine at an urban medical school and health science centre in Ontario. He chairs a Pharmacy and Therapeutics Committee, and is Medical Director of a Cardiac Care Unit of in an Ontario Hospital. He is a peer reviewer of articles for thirteen journals and organizations. He has extensive teaching duties with medical residents and cardiology fellows. He worked in the cardiac catheterization laboratory for 12 years. As one of his self-assumed duties, he extensively reviews and summarizes medical literature that forms part of a database for use by other physicians at his centre. He routinely reviews and reports on up to 300 ECG's per day, as well as reviewing other invasive and none invasive cardiac procedure reports. Dr. Z's clinical experience is extensive and current. He attends a cardiac clinic and is on call for cardiac issues on a regular basis. He interacts with a large number of internists and cardiologists, and teaches medical students from their first year, and

trainees at all other levels of experience and training. Dr. Deep questioned his experience in clinical cardiology, and Dr. Z gave evidence that he sees in his clinical practice and consultation the full spectrum of disease symptoms potentially suggestive of cardiac abnormalities, including patients with manifestations of coronary artery disease, acute chest pain, unstable angina, heart failure, and rhythm disorders. While on call, he sees 50-60 new patients, as well as eight to ten patients a week on his clinic day.

The Committee reviewed Dr. Z's qualifications and experience, and took note of his answers relating to his experience when being cross-examined by Dr. Deep. The Committee accepted Dr. Z as an expert in cardiology and internal medicine, qualified to give opinion evidence.

In considering credibility and the weight to give to his evidence at the end of his testimony, we note the following. Dr. Z testified in direct examination for more than five days and in cross-examination for six days. His testimony was forthright, and he answered questions with reasoned opinions based on his experience and articles he presented to support his opinions. He had created an Excel database for keeping track of the myriad of data from the charts that was otherwise difficult to find or to make sense of chronologically. He utilized standards and guidelines applicable to the time in forming his opinions. During his testimony, he would admit to mistakes in his report, and the Committee found these were few in number and minor with respect to his forming opinions. He testified that he had hoped to meet with Dr. Deep to clarify issues in the reviewed charts, and made arrangements multiple times to do so, by various physical and electronic means, but these meetings were canceled by Dr. Deep. He therefore compiled a list of questions (Exhibit 8), but never received a reply from Dr. Deep. There were times when Dr. Z expressed his exasperation during the lengthy cross-examination, but these were few in number and the Committee was of the opinion that they were within acceptable limits. Dr. Z was reluctant to discuss the fees he received for his report, but acknowledged billing and receiving an interim amount. The Committee considered that the hourly figure presented was not excessive. In cross-examination, Dr. Z denied allegations leveled by Dr. Deep that he was paid to slant his report, or that a provincial grant for millions of dollars to his health centre by the provincial government was a

reward for conclusions in his report concerning Dr. Deep. It should be noted that no evidence was presented by Dr. Deep to support this or other such allegations.

Dr. Z reviewed each of the charts in depth. His preparation was impeccable, and he was able to bring clarity to areas of chaos within the charts concerning the sections under examination. He presented papers, and evaluated them clearly, in support of his conclusions. He readily pointed out where evidence was inconclusive, or had changed over the years. His explanations were lucid. As his standard, he presented consensus reports and guidelines that related to the years under consideration for each patient. He used guidelines, standards of practice and research based on contemporaneous evidence to support his opinion. He stated the standard of practice and his opinion on competency was based on his expectations of the level of knowledge and practice of newly graduating cardiologists who would be practicing in the community.

The Committee was impressed by his demeanour, knowledge of his subject academically and clinically, and his ability to elucidate the expected clinical behavior of a cardiologist, internist or medical trainee. This extended, in the Committee's opinion, to basic expectations of any physician practising medicine in Ontario at the relevant time and the present time. We find Dr. Z to be a credible witness. The Committee put great weight on his opinions and the material he presented to support it. (His report and oral evidence are collectively described as Dr. Z's testimony).

Dr. Albert R. Deep

Dr. Deep testified in his own defence. He also put himself forward as an expert in cardiology and internal medicine. The College did not challenge this qualification, and the Committee therefore proceeded on this basis. Dr. Deep graduated from Queen's University in 1959, and subsequently did graduate training for six years at the Montreal General Hospital. He did two years of further training in cardiology at Massachusetts's General Hospital. In Toronto, he was on staff at Toronto Western Hospital from 1967 to 1968, and at Toronto General Hospital from 1969 to 1971. He has been in private solo practice since that time, and has not been associated with any hospital in Toronto since the aforementioned dates. He stated in his "Expert evidence and report" that he has been

in the practice of cardiology “with some internal medicine” for 40 years. He states he is up to date in the Maintenance of Competence program of the Royal College of Physicians and Surgeons of Canada.

Dr. Deep relied heavily on a preponderance of non-peer reviewed material and his own judgment, experience and memory to support his positions concerning the areas of practice considered in the evidence. The Committee found that his arguments concerning Dr. Z’s evidence were replete with unjustified ad hominem attacks on Dr. Z, self-referential logic, and non-verifiable suppositions and speculations as to non-charted facts about his patients. He presented no independent evidence to support his position as to the standards of practice of peers. He appeared to practice in isolation.

Dr. Deep, as an expert, is not independent, and has a vested interest in the outcome. The Committee gave little weight to his expert opinions, finding them based on suspect material, most often without evidentiary support, and relying for his status as an expert on his experiences and accomplishments prior to 1971.

In testifying in his own defence, he presented material concerning the patient or area of practice under consideration, which will be evaluated individually below. In general, under cross-examination, his suppositions were presented as facts and were not supported by reliable written evidence. He did not call any viva voce evidence to support his claims. We were asked to accept facts that should or ought to have been documented in the medical record, or were speculative assumptions. His evidence would change when he was confronted with factual material that contradicted his original stance. While he was not required to bring witnesses, he would ascribe comments and views to patients and physicians, not supported by any material. He would state the standard in Toronto, but brought forward no supportive evidence or indication of contact with either community or hospital-based physicians or cardiologists. We found him as a witness to be unreliable, inconsistent and lacking in internal or external consistency. His credibility was low, best illustrated by his tendency to evoke unsubstantiated events in his patients’ lives when presented with evidence that cast doubt on the course of therapy listed in the chart. Further reasons for this conclusion will be elucidated in the review of the evidence.

Medical Records

The basis for the opinions of the witnesses, and the information relied on by the Committee, relate directly to the information present in the medical charts of the 25 patients entered as exhibits. The Committee, who heard evidence as to their content, reviewed these charts. Twenty of these records were chosen by the College investigators, with the remainder chosen by Dr. Deep.

Organization

The charts were large and disorganized. Some charts were over 1500 pages long (patient A -1651 pages, patient B -1502 pages), and while many of these pages were taken up by Electrocardiogram tracings, Dr. Z stated that he “had never seen anything like it.” He testified that none of the 25 charts reviewed were organized systematically or chronologically. With many patient encounters recorded on more than one page, the subsequent page(s) could be found hundreds of pages apart. Laboratory reports were not filed chronologically and were randomly interspersed in the charts. Patient names or other consistent identifiers were found on less than 1% of pages. Multiple Electrocardiograms (ECG’s) did not contain patient identifiers or indications of leads used. Dr. Z found examples of chart entries that did not correspond with the particular patient. He found that the chaotic organization as noted above made it difficult to find pertinent information, and that it sometimes took him over half a day to find specific materials or entries. In Dr. Z’s view, it was impossible to get a timely or reasonable overview of the patient’s medical status in the vast majority of the reviewed charts. Up-to-date medicine lists were most often not available. Notes and laboratory reports from outside sources did contain patient names. While the first page of an initial encounter did have identifying data and a birth date, this was not usually the first entry in the chart.

Dr. Deep testified that his charts were seized by the Ministry of Health in 1998 and were returned to him in this condition. He stated that, as a solo practitioner without secretarial employees, he could not rearrange these charts but he could find what he needed and had memory of pertinent material. Dr. Z gave evidence that the charts reviewed after 2000 were in the same state as those prior to the seizure date. In his report to the Executive Committee (Exhibit 6A, B and C), Dr. Deep presented additional or excerpted chart

entries. The Committee noted that there was the same lack of chronology or identifiers in these chart entries subsequently amassed by Dr. Deep. For example, in Exhibit 6C (patient A, Tab 16, page 4), there is an undated referral letter followed by lab reports dated consecutively 2006, 2004, 2004, ECG May 2006, Laboratory March 2006, July 1997, May 2005, June 2005, consult letter January 2006, and June 2001, Laboratory June 1997, Laboratory September 2004, undated note on shopping list paper indicating different drug substitution, undated patient medication profile from a drug store, consultation note September 2006, Laboratory July 2006, ECG August 2006, Laboratory February 2006, EEG March 2006, Laboratory April 2003, and so on.

Dr. Z's concerns also included the organization of content for each recorded patient visit. There was a lack of presenting complaints, and he was unable to ascertain the reasons for many visits. There were notes jotted in margins, scribbles, lines, arrows, circles and boxes with additional information. While Dr. Z noted that the style of handwriting appeared to him to be different for some of these additions, no evidence was adduced concerning this and the Committee makes no comment on this aspect of the charts.

Additionally, Dr. Z commented that there were no initials or other markings on lab reports and other outside assessments to indicate that they were reviewed, or any notational system to indicate need for follow-up of abnormal results.

Dr. Z testified that it is important for information in the chart to be readily ascertainable to other physicians if care is needed due to death or illness of the treating doctor. As well, the charts must allow the original physician to know the continuing status of the patient so as not to miss necessary information.

Dr. Deep testified that he saw the chart as personally belonging to him and for his own use; he could find information in a few minutes and he relied on his memory and knowledge of the patients. Dr. Deep stated that as he utilized no office staff, opened mail and did all the filing, he would have seen all laboratory data by necessity. He also insisted that, since he was healthy, there would be no need for any other doctor to use his charts and, if he were retiring, he would produce summaries. On cross-examination, Dr. Deep was unable to find information requested that ought to be present in his charts. He stated

it might well be in the original rather than the photocopied charts, but did not take the offered opportunity to bring in those charts. As well, on cross-examination he was unable to remember situations concerning his patients, and replied that the information requested was either unimportant, or that he could not be expected to remember everything.

CPSO Policy #5-05 Medical Records (Exhibit 63), and A Guide to Current Medical Record-Keeping Practices (Exhibit 64), were introduced in evidence. The former states, “The primary use of these records is for the treating physician and other health professionals to ascertain the patient’s medical history...” Other major principles concern the role of medical records in continuity and quality of care. The policy states that the medical record must “tell the story” of the patient. As well, it indicates, “The record is not just a personal memory aid for the individual physician who creates it. It must allow other health care providers to read quickly and understand the patient’s past and current health concerns.” The policy states, “All patient related documentation must be dated.” The policy continues, noting, “The first step in taking a patient’s medical history is to clarify and verify the patient’s reason for the visit.”

The policy goes on to state “Multidisciplinary care is a fact of life in our health care system and the medical record serves as the conduit of information shared between health professionals.”

Dr. Z testified that the poor medical records resulted in missed diagnoses, particularly those based on abnormal laboratory results. The specific issues related to thyroid function, renal failure, anticoagulant use, hyperkalemia (elevated potassium levels in the blood), and elevated glucose levels.

The Committee concludes that the physical state of the medical records made the retrieval and knowledge of information pertaining to patients with long-term complex problems and multiple treatments difficult for Dr. Deep and extremely difficult for any other health professional. It is the Committee’s finding on the state of Dr. Deep’s records that it would not be possible for Dr. Deep or any physician to find vital information or to know important parameters necessary for adequate patient care and safety. We find that the physical state of the charts does not meet the standard of practice, could bring patient’s

lives into danger, and shows a disregard for the welfare of his patients. Dr. Deep's reasoning that the state of the charts was due to his lack of clerical help is not an acceptable explanation, and could have been easily remedied. He demonstrated in testimony that his memory was not a substitute for adequate records.

Communication with Other Physicians

Dr. Z testified and noted in his written report that "he was shocked that Dr. Deep rarely initiated correspondence with other physicians after a patient encounter. He rarely copied laboratory and other test results to other physicians involved in the care of the patient," and "it is clear from Dr. Deep's own notes that some of the patient's family doctors did not even know that Dr. Deep was involved in the patient's care." He noted that "[t]here were other instances where Dr. Deep might have considered discussion with colleagues; for example, Dr. Deep started anticoagulation with coumadin without notification of the family physician, changed medications initiated by a neurologist at a stroke clinic where the patient was under continuing care, made changes to the medical therapy and organized cardiac investigations on a patient who had been cared for in-hospital with an acute myocardial infarction by a cardiologist who was following the patient, failed to clarify why a patient is taking two different alpha-blockers, one prescribed by Dr. Deep and the other by a Urologist, and made changes to psychotropic therapy on a patient being followed by both a family doctor and a psychiatrist." As an example of the above, Dr. Deep altered the medications prescribed by a neurologist caring for patient D's stroke. There was no communication about doing this. Dr. Z stated that communication is paramount in patient care, and needs to be timely as well.

Dr. Deep, in answer to these observations, stated that he was capable of treating patients as a family physician, internist and cardiologist. His view was that, if the family physician wanted to communicate about his therapy, they could do so and, unless there was a formal consultation request, he had no obligation to communicate with other physicians. In the case where he changed medications from the neurological clinic, the patient was not seeing the neurologist for a while, and could tell the neurologist at a subsequent visit what medication changes had occurred. In the case of the psychotropic medication, the patient was seeing the psychiatrist for a different reason and only one

time, and in the myocardial infarction follow-up, the patient was not able to get an appointment soon enough and wanted Dr. Deep to take over his management. Dr. Deep stated that patients asked him to take over their full care and that there was therefore no need to communicate with other physicians.

In reference to the lack of consultation notes on patient E, patient D and patient F, Dr. Deep indicated he “can’t be certain these were not dictated, typed up by [himself] and just not on file.” He suggested that, in transfer from home to office, they may not have made it to his files, or consultation notes may not have been done because Dr. Y, a local physician, may have transferred care of these patients to him (as opposed to asking for a consultation) and therefore no note was due. We found no notations in the chart to indicate that this was the case, nor was any evidence brought forward to support this contention.

Dr. Deep justified his communication patterns not only by placing the responsibility to communicate with him on other physicians, but on the basis that his patients were “intelligent or highly intelligent” and he could rely on their previous histories as they stated them. The patients could communicate medication changes or other results to their other caretakers. He said this was a factor in his communication patterns, as well as his taking at face value patients’ reports of diagnosis and treatment. Yet, in cross-examination concerning patient Dr. X, where Dr. Z had questioned the lack of follow-up of his self-reported history of diabetes, Dr. Deep stated that he was not required to take the word of patients as to their diagnosis. The Committee finds the disparity between Dr. Deep’s explanation of his communication patterns and its failure to apply to Dr. X (Dr. Deep did not follow-up on his reported diagnosis even though he characterized Dr. X as highly intelligent and as the referring physician for his wife, Mrs. F, with whom he discussed her treatment) as lacking in truth and negating Dr. Deep’s insistence that he believed that the patients could be responsible for self-reporting their own illness, as well as communicating with other physicians.

The Committee is of the opinion that, for this purpose, leaving the question of the appropriateness of the treatment changes made by Dr. Deep for individual patient

analysis, the lack of communication with other physicians could cause harm to the patients, interfere with the continuity of care, and does not meet the standard of practice. Dr. Deep's lack of insight into the need to communicate with other health professionals and his insistence that his own therapeutic maneuvers require no communication with other physicians are indicators of poor judgment and are likely to cause harm to patients under his care.

Office Based Diagnostic Testing

Dr. Z raised three related issues in reference to the office based testing performed by Dr. Deep: the quality of the ECG tracings reviewed in the charts; the appropriateness of the frequency of the ECG's; and, the performance of a test, systolic time intervals, by Dr. Deep. He also commented on the lack of calibration of blood pressure cuffs as well as the old and new ECG machines.

Dr. Z, on reviewing the hundreds of tracings in the 25 charts, concluded that most were not diagnostic due to lack of calibration and missing leads. It was impossible to follow tracings due to their lack of quality and wandering baselines. They were also lacking patient identities and labeling of the leads. In most cases, the requisite 12 leads were not present. The Committee noted examples in all charts, and agreed with Dr. Z's assessment of their poor quality, lack of patient identifiers, and the impossibility of making a reliable interpretation. ECG's were done on most, if not all, patient visits. Dr. Z stated this was excessive, without cause, and unnecessary as a routine, especially with the disproportionate number of follow-up visits for conditions that did not warrant monthly or more frequent visits. Dr. Deep stated that, while he could interpret these ECG's, another forum had criticized these tracings and he had purchased a new machine. While Dr. Z did not see any of these new tracings in his review of charts, the tracings themselves from the new machine, as exhibited in tracings submitted by Dr. Deep, were of diagnostic quality. Dr. Deep denied that calibration was necessary on his old machine, and continued to insist that, even without patient identifiers, he had filed them and knew to whom they belonged. He was of the opinion that they were of diagnostic use, and the reason for the new machine was to please another forum's objections. On cross-examination, Dr. Deep was presented with an ECG (volume 1B, page 20). Dr. Deep

ascribed the poor quality to the CPSO's lack of proper photocopying. He was offered the opportunity to bring in the original if this was true, but did not do so.

Dr. Z was of the opinion that, even if the ECG's had been of diagnostic quality, there was no clinical reason to repeat them as often as Dr. Deep did. Dr. Deep stated that there can always be silent changes in heart condition that can only be picked up by doing an ECG.

Dr. Z was of the opinion that systolic time intervals were an antiquated technique with little clinical use; while it could be measured, it is not currently done as it has no clinical value. Dr. Deep defended its use quoting an article published in July 1975 (Exhibit 60) which referred to the use of this measurement in chronic hypertension. At that time, it was the opinion of the authors that the systolic time interval "may be useful in evaluating hypertensive patients."

It was Dr. Z's evidence that the standard of practice for a consulting cardiologist, at the present time and at all times in question, is to have ECG machines and blood pressure machines calibrated routinely. Dr. Deep was of the opinion that the machines were self-calibrating, that his blood pressure cuffs agreed with each other and, while Dr. Z could easily get machines calibrated at a university centre, such was not the case in Toronto.

The Committee accepts the evidence of Dr. Z on the deficient quality of the ECG tracings, and finds that an overwhelming number of tracings were not of diagnostic quality. This could bring danger to patients treated or not treated based on these tracings. For example, in the case of patient A, Dr. Z pointed out that ECG interpretation was wrong. Dr. Deep states "interpolated VEDs" (otherwise known as PVC's (premature ventricular contractions) or VPB's (ventricular premature beats)) when they are not interpolations at all. He mistakes "poor R wave progression" for anterior wall myocardial infarction, when there are many reasons for it rather than that one possibility. The Committee believes that these tracings did not meet the standard of practice when they were done, and could result in adverse patient outcomes. We were not convinced that Dr. Deep's explanation for the frequency of the tests, that they might possibly reveal an abnormality, was plausible. We accept that probability rather than possibility governs medical decisions, especially around testing, and Dr. Deep has not understood this

concept. Dr. Deep stated that silent ischemia does occur in cardiac patients and can only be discovered by an ECG at each visit. This logic is in itself irrefutable, but, if taken at face value, would require an ECG by every doctor with every patient at every visit. The Committee finds that the frequency of ECG's and Dr. Deep's explanation for them shows a lack of diagnostic acumen and judgment.

The use of systolic time intervals appears to be unnecessary and, while they might well have met the standard of practice in the 1960s and 1970s, we concur with Dr. Z that they are no longer necessary. Our concern is that in using this as a measure of left ventricular function, Dr. Deep might well not perform tests of greater use and specificity.

Clinical Assessment of Patients' Cardiac Status

Dr. Z stated that the cases he reviewed were "garden variety cardiology" and that in evaluating diagnoses and care he used the standard of what he would expect of a physician successfully completing his cardiology training and preparing to be a community cardiologist.

By that standard, Dr. Z considered Dr. Deep's clinical assessment to be below that expected of a cardiologist and, therefore, put patients at danger. Often his diagnosis was later shown to be inaccurate by objective testing. He noted that patient G was clinically diagnosed with aortic stenosis when tests done much later showed no clinically significant disease. Patient H was clinically diagnosed with aortic stenosis and was later objectively shown to lack any clinically significant disorder of this valve. Patient F did have aortic stenosis, but was inadequately followed-up. There was a clinical diagnosis of mitral regurgitation in patient I, patient J, patient K, and patient L, and these were objectively later proved to be of no clinical significance. Patient I was treated for 18 years for angina. Dr. Z noted that he did not have the disease and could have had a definitive diagnosis if an early stress test or coronary angiography had been done at an appropriate time.

Dr. Z noted that a diagnosis was often based solely on a history of past treatment and without proper testing to ascertain the cause of the symptom.

Dr. Deep did not do Holter monitor testing when indicated. He testified that it would be too inconvenient for his patients to get this test, or that they were too busy to do so.

Dr. Deep treated patients for what he considered life-threatening arrhythmias based on his insufficient ECG's. Dr. Z was of the view that Holter monitors would have shown conclusively that these arrhythmias were not life threatening and, in doing that test, could have spared patients being placed on medications.

Dr. Z strongly disputed Dr. Deep's statements that patients could commonly have a normal stress test, echocardiogram, isotope study and still have substantial coronary artery disease that needed treatment.

Dr. Deep suggested that true coronary artery disease was difficult to assess without an IVUS (intravascular ultrasound) test. It was Dr. Deep's contention that, if you did an IVUS on the patients diagnosed as having possible coronary artery disease based on family history, it would find the pathology. Dr. Z explained the process of using this tool in selected cases and selected areas of a coronary artery, but stated that this concept of disease was theoretical at best, and was an inappropriate excuse for Dr. Deep not doing the accepted and routine diagnostic tests before initiating treatment.

The Committee accepts the evidence of Dr. Z that, in the area of the overall clinical assessment and follow-up of patients, Dr. Deep practised below the standard expected of a community cardiologist. As Dr. Deep continues to demonstrate a lack of understanding of the noted deficiencies, the Committee finds that he is incompetent at the present time.

Psychotherapy

Dr. Deep billed for psychotherapy on most patient visits (OHIP records, Exhibit 3). Dr. Z stated that no cardiologist he knew did this, and that counseling (as differentiated from psychotherapy) about the illness was part of the assessment and treatment included in the fee schedule for a specialist consultation or follow-up. He also testified that there were no notes to show that psychotherapy had taken place, or what the content was. These sessions ranged from 46 minutes to one hour and 16 minutes. The charts did record the

time, as required by OHIP. Dr. Deep's rationale was that patients with cardiac problems always have an emotional problem and that treating them with "talk therapy" was a necessary and beneficial requirement. He testified that his training to do this stemmed from his experience talking with people. On cross-examination, he agreed that his patients were not informed he was doing psychotherapy with them as it would, in his opinion, negate its effectiveness. They did not need to give permission or know they were receiving it for it to be effective. The content included talking about their problems – such as business worries and financial or home problems. Dr. Deep reviewed his understanding of the Ministry of Health's definition of psychotherapy for OHIP purposes and stated it includes dealing with such issues as "difficulties in the work place"; he added that providing this is a service for patients and remuneration for it is at "G.P. rates" and not what a cardiologist could make per hour. He stated that he could earn more money doing ECG's, et cetera, and that patients do not need to be advised they are entering into psychotherapy. It helps them with any problems they may have. He saw no reason to inform any family practitioner of the patient's possible psychological distress. He explained the lack of notes as being due to a court case in which a patient record was exposed in the press. He was concerned with patient privacy thereafter. He stated that he used to make notes prior to this case, but produced no charts to confirm this. There was, therefore, no record of the content of psychotherapy encounters in the charts.

The Committee highlights three instances of particular concern related to psychotherapy. In the case of Dr. X, Dr. Deep defended the lack of physical examinations, deemed necessary by Dr. Z, on the basis that the patient was in a rush and there was no time to perform the expected examination. Yet, it was noted that on the same days lacking physicals, he billed for psychotherapy, content unrecorded. In the case of patient A, the patient was noted to have an abnormal heart rate, which was evaluated by Dr. Deep as an emergency, and the patient was sent by taxi to the hospital where he was admitted to the cardiac care unit for seven days. On that same day, where the chart indicated an emergent situation with immediate transport (while we question the mode of transport in this case, rather than the expected ambulance with monitoring, this is another issue), there was a billing for two units of psychotherapy, content unrecorded. In the third case of patient B,

Dr. Deep commented on her paranoid ideation and that, on one occasion, she was delusional. No specific diagnosis or differential diagnosis was established, yet psychotherapy was performed. There was no neurological examination, no referral to a psychiatrist or psychogeriatrician. In his testimony, Dr. Deep said the patient felt discriminated against as a French Canadian, and redefined the meaning of the diagnosis of paranoid ideation as an “inappropriate belief in discrimination” and that she improved with “talk” therapy.

The Committee concludes that, if psychotherapy was performed, the lack of any charting to substantiate it, as well as the inappropriate usage of this therapy in the aforementioned cases, brought patients into danger. With respect to this modality, the allegation of not meeting the standards of the profession and of incompetence are proven.

Anti-Arrhythmic Treatment

General Description of Issue

The issue at hand is the use of anti-arrhythmic drugs, especially Propafenone. While other anti-arrhythmic medications were used by Dr. Deep, Propafenone best illustrates the issues raised in the allegations. Propafenone is an anti-arrhythmic agent possessing class 1C properties in the modified electrophysiological classification of Vaughan-Williams. It has a direct stabilizing action on myocardial cell membranes.

However, studies in the late 1980s showed that, in patients with ischemic heart disease and prior myocardial infarction (MI or heart attack) who had ventricular arrhythmias with none to mild symptoms, anti-arrhythmic drugs suppressed the arrhythmias but increased the overall risk of death. Dr. Z was emphatic that this class of medications should generally be avoided or used very cautiously in any patient with structural heart disease. The risks of drug induced serious arrhythmia with a high risk of death must be considered.

Dr. Z reviewed the literature to support his expert opinion. The CAST study “Mortality and Morbidity in patients receiving Encainide, Flecainide, or Placebo,” New England

Journal of Medicine, March 1991 (Exhibit 13), demonstrated that the use of these two class IC agents to treat asymptomatic or mildly symptomatic ventricular dysfunction after MI carries an excessive risk of mortality. The conclusions from the study read: “there was an excess of deaths due to arrhythmia and deaths due to shock after acute recurrent myocardial infarction in patients treated with the two agents.”

The CASH Study (Exhibit 14) was initiated because several recent studies provided evidence that class I drugs are less effective than class III in patients with ventricular arrhythmias, most likely because of pro-arrhythmic effects. In this study, the salient fact was “[a]ssignment (of patients) to Propafenone was discontinued on the request of the Safety monitoring board after an interim analysis conducted on 58 patients showed a 61% higher mortality rate than in 61 Implantable Cardiac pacemaker patients.”

The MUSTT trial (Exhibit 15), while looking at the role of programmable ventricular stimulation, noted that “[m]ortality was greatest in the few patients receiving Propafenone. The sample size was small and this result was not statistically significant, but was confirmatory of previous data on the risks of Propafenone.”

Dr. Z introduced a meta-analysis of multiple anti-arrhythmic trials (“A Meta-analysis of Antiarrhythmic Drug Trials,” Exhibit 16). He indicated that prevention of sudden death by means of anti-arrhythmic drug therapy has been a much sought after goal in cardiology for a generation. In the 1980s, it became clear that asymptomatic ventricular arrhythmia was a predictor of poor outcomes in survivors of MI. It became common to use drugs that suppressed arrhythmia, based on the hypothesis that asymptomatic arrhythmia was a trigger for episodes of ventricular fibrillation and that suppression of the trigger would reduce the risk of this adverse outcome. This meta-analysis concluded that there is important evidence of an increased risk of death with all class I drugs with these patients.

It must be noted that Dr. Z relied on meta-analysis studies. He testified that this was a modern and well-accepted statistical way of looking at multiple studies in a single area. Dr. Deep disputed the reliability of meta-analysis and rejected the results of these studies. In his cross-examination, he appeared unaware of how these studies were assembled.

The Compendium of Pharmaceuticals (Exhibit 17) states that Rythmol (Propafenone) is an anti-arrhythmic agent that possesses class 1C properties, and continues:

“[c]ontraindications are severe or uncontrolled congestive heart failure, cardiogenic shock, sinoatrial, atrioventricular, and intraventricular disorders of impulse conduction and sinus node dysfunction in the absence of an artificial pacemaker, severe bradycardia and various other heart disorders. Propafenone is contraindicated with wide QRS complex.”

Warnings include mortality as per the CAST trial noted above. It was noted that Propafenone has pro-arrhythmic effects: Propafenone may cause new or worsen existing arrhythmias. Such pro-arrhythmic effects range from an increase in frequency of PVC's, to the development of more severe ventricular tachycardia, ventricular fibrillation or Torsades de Pointes. “It is therefore essential that each patient administered propafenone be evaluated clinically and electrocardiographically prior to, and during therapy to determine whether the response to propafenone supports continued treatment”.

In a peer reviewed paper, “Inpatient versus outpatient antiarrhythmic drug (AAD) initiation” (Exhibit 30), it was noted that there is no simple answer to the question of proper venue for treatment initiation; however, the recommendation from this paper is that patients for whom AAD is considered and who have structural heart disease, are best treated in hospital to start or increase AAD therapy.

In the study “Effect of prophylactic amiodarone on mortality after acute myocardial infarction” (Exhibit 40) it was noted that, during the past decade, randomized trials have investigated the ability of several anti-arrhythmic agents to reduce premature death in patients at high risk of arrhythmia. Apart from beta-blockers, no other agent has

conclusively shown to reduce mortality. Indeed there have been clear increases in mortality with some class 1 and class 3 agents. Amiodarone, however, has several anti-arrhythmic actions and unique properties. The conclusion of this review is that prophylactic Amiodarone reduces the rate of arrhythmic sudden death in high-risk patients with recent MI or CHF and this effect results in an overall reduction of 13% in total mortality.

In a study entitled “Proarrhythmia” (Exhibit 52), pro-arrhythmia is defined as the provocation of a new arrhythmia or the aggravation of a pre-existing one during therapy with a drug at doses below those considered to be toxic. This article focused on the mechanisms for anti-arrhythmic drugs causing pro-arrhythmia. Propafenone is listed as one of the drugs with potential to cause pro-arrhythmia.

In “Placebo-Controlled Evaluations of Propafenone for Atrial Tachyarrhythmias” (Exhibit 53), it is noted again that Propafenone is a class 1C anti-arrhythmic drug. It supports the contention that Propafenone has a very acceptable side-effect profile when used in the treatment of SVA’s (supraventricular arrhythmias or atrial arrhythmias), particularly when used in selected patients without structural heart disease.

Dr. Deep introduced the study “Limitations to antiarrhythmic drug use in patients with atrial fibrillation” (Exhibit 46), which noted that maintenance of sinus rhythm by the use of anti-arrhythmic drugs is often the initial therapy for atrial fibrillation (AF), although recent trials have cast doubt on whether rhythm control should be used routinely with patients with AF.

It noted that “[i]n spite of these contraindications, a high percentage of physicians (30 to 40%) still prescribed these drugs.” The interpretation of the authors is that there is no effective treatment in these situations, so less than appropriate drugs are being used. It should be noted that this study dealt with atrial arrhythmias rather than the ventricular arrhythmias that Dr. Deep was treating in the preponderance of the cases reviewed.

Dr. Deep also introduced studies in his book of documents, the great majority of which were not peer reviewed or were drug company supported summaries and opinions which he stated supported his use of Propafenone in his patients. In regard to these studies and reports, Dr. Z introduced a paper on “Outcomes on clinical trial funded by for-profit and not-for-profit organizations (Exhibit 55). It concluded, “[r]ecent cardiovascular trials funded by for-profit organizations are more likely to report positive findings than trials funded by not-for-profit organizations.”

Dr. Z’s General Comments on the Use of Propafenone by Dr. Deep

Studies in the 1980s showed that the use of anti-arrhythmic drugs in patients with ischemic heart disease and prior MI who had ventricular arrhythmias and no to mild symptoms, suppressed the arrhythmias but increased the overall risk of death. These medications are generally avoided or used very cautiously in any patient with structural heart disease and the risks of serious arrhythmias must be considered. Dr. Z believed that, in all six patients noted below, the risk of harm was greater than any possible benefit. In two patients, the drug was contraindicated. There was little documented discussion of the risks with the patients. Dr. Deep made many of the diagnoses for treatment with Propafenone based on his office ECG, most of which, as noted above, were uninterpretable, or on clinical auscultation alone. He never considered starting the patients on medication in an in-patient setting. There was neither a baseline established nor systematic monitoring for possible adverse symptoms. He gave little concern to potential drug interaction.

Dr. Deep’s general comments on the use of Propafenone

Dr. Deep testified that he used Propafenone with patients where, in his judgment, the benefit of treatment clearly outweighed the risk. It was indicated and used in patients with documented symptomatic ventricular arrhythmia when the symptoms were of sufficient severity to require treatment. Whereas the incidence of pro-arrhythmia is more significant in patients treated for ventricular tachycardia or fibrillation, the incidence of pro-arrhythmia in patients with less severe or benign arrhythmias, which includes patients with an increase in frequency of PVC’s, was 1.6%. He emphasized that the CAST trial did not include Propafenone, but admitted that the preponderance of opinion of most

cardiologists today supported the CAST trial as a standard, and because Propafenone is a class 1C agent, it can be included. It should be noted, as Dr. Z commented, that no study would ever be done on Propafenone alone as there is so much data linking it to class 1C and to increased deaths (as noted in the studies cited above), that it would be unethical to do so. Dr. Deep stated Amiodarone has a much greater incidence of pro-arrhythmic effect; however, the exhibits reviewed do not support this. He stated that, contrary to the evidence of Dr. Z, either Amiodarone or Propafenone can be safely started in an outpatient setting, except for sustained ventricular tachycardia, which none of these patients had. He stated that there is evidence that Propafenone is both efficacious and safe in the treatment of paroxysmal atrial fibrillation, although the supporting study noted above also emphasized its use in patients without structural heart disease. Dr. Deep stated that the world of cardiology was not changed by the CAST study and that 38% of physicians would treat their patients with Propafenone notwithstanding structural heart disease. If a patient has bigeminal rhythm, you do not wait 24 or 48 hours for a monitor study or you risk the patient dying. Regarding the CAST trial, Dr. Deep stated that drugs other than Propafenone were included, that trial patients had fewer ventricular extra beats than his patients did, and that the study was seriously flawed and is not generally accepted. He stated that Propafenone was the only anti-arrhythmic drug that did not prolong the QT interval.

In cross-examination of Dr. Z, Dr. Deep suggested that pharmaceutical company publications are as good as peer reviewed, consensus, or meta-analysis studies, and attempted to discredit the basic studies noted above. Dr. Z's testimony continued to support, to the Committee's satisfaction, his, and the cardiology communities', acceptance of the validity of these studies. The Committee does not accept the evidence of Dr. Deep.

Patient Review concerning Propafenone

Patient H

Dr. Deep began the use Propafenone in 1993 for what he, in testimony, described as "symptomatic" ventricular ectopic depolarizations, which were recorded on his office

ECG. Dr. Z was concerned that he found no documentation of an arrhythmia or association of an arrhythmia with symptoms, no Holter monitor report and no discussion of risks, all necessary as Dr. Deep believed that the patient had angina pectoris. The use of Propafenone in this case was questionable given CAST study results. In addition, the treatment was initiated on an outpatient basis, and no specific ECG monitoring for pro-arrhythmia was noted for a patient who had been previously diagnosed with structural heart disease by Dr. Deep.

Dr. Z stated that the use of a potentially toxic drug to suppress what was described as symptomatic (although there was no evidence of this) premature ventricular beats, a benign problem, showed a lack of knowledge and judgment, and there was no follow-up care. Dr. Deep later increased the dose, showing further serious lack of knowledge and judgment.

Dr. Deep stopped the drug Amiodarone that patient H had been taking, and immediately started Propafenone. Dr. Z's concerns were that Amiodarone has a very long half-life, thus increasing the risk of the patient being on Propafenone, which was also contraindicated. If one were to start the drug on the basis that the benefits outweighed the risk, then the treatment should have been initiated in a hospital, and followed closely with proper ECG's.

In general, the diagnosis and decision to treat ventricular arrhythmias should be preceded by a complete workup, including a Holter Monitor (a 24 hour ECG), to determine if the arrhythmia is significant and if the subjective symptoms have any relationship to the objective findings. This was not done with this patient or with others where it was obviously indicated in order to confirm a diagnosis or to properly diagnose and treat a patient.

Dr. Deep stated that he did not do these types of studies because of the inconvenience to the patient of having to travel to a different site for the tests, or because the patient refused as they were busy, rushed or could not make the time available. Dr. Z opined,

and we agree, that this is not an issue in downtown Toronto and, in any case, the patients were traveling to his office. As well, even those who he stated were too busy or rushed had psychotherapy of 30 minutes or more recorded in their chart during visits when diagnostic tests were appropriate to order. The Committee does not consider Dr. Deep's explanation valid or meaningful.

Dr. Z stated that alternate treatments should have been considered and were available in Toronto during the time period under consideration.

Dr. Deep testified that the CAST trial did not include Propafenone, and that Amiodarone has a much greater incidence of pro-arrhythmic effect. He stated either Amiodarone or Propafenone could be safely started as an outpatient, except for sustained ventricular tachycardia which this patient did not have. Dr. Deep was of the opinion that three other cardiologists seeing patient H allowed for the continuation of Propafenone.

The Committee notes that it had no testimony from other cardiologists on using or continuing this medication. The Committee accepts the conclusions of the CAST trial as being conclusive as they apply to this patient.

Dr. Deep does not view Propafenone as a drug with significant risks. We believe the evidence contradicts this. He started the drug while the patient was on another anti-arrhythmic, and did not demonstrate appropriate concern about monitoring or starting treatment in an inpatient environment. He began treatment based on his interpretation of her ECG's, which are of poor quality and uninterpretable. There is no workup of the symptoms to relate them to the supposed ECG finding as would be expected and, according to Dr. Z, they were misdiagnosed to begin with.

Patient M

This patient was a 76-year-old (in 1997) whom Dr. Deep diagnosed as having coronary artery disease with angina pectoris. In July 2000, he prescribed Propafenone for "symptomatic" premature atrial contractions and/or angina. Dr. Z felt the patient was

clearly asymptomatic to begin with, yet the drug was used for a patient with structural heart disease, given the known risks of pro-arrhythmia. Dr. Deep stated that, in his judgment, the atrial contractions were symptomatic, but there were no investigations to relate this to any cardiac abnormality in need of treatment.

The Committee finds that, notwithstanding Dr. Deep's belief that the atrial abnormalities were symptomatic, this patient was placed on a potentially dangerous drug without valid indication, proper investigation or follow-up.

Patient K

Patient K was 58-years-old when first seen by Dr. Deep in 1997. He diagnosed the patient as having rheumatic heart disease and mitral regurgitation. He prescribed Propafenone, Sektal (a Beta Blocker), Avapro, and a statin, while stopping the Sotalol that she was previously taking. He stated that she had a history of atrial fibrillation and symptomatic arrhythmia so severe that they interfered with her sleep. He believes his dosage was sufficiently low so there was little risk.

Dr. Z was concerned that Dr. Deep prescribed Propafenone without a diagnosis of a specific arrhythmia. As well, he could see no reason to stop her Sotalol, which could have controlled her arrhythmia with much less risk than a medication like Propafenone. Dr. Deep later increased the dosage. Based on Dr. Deep's diagnosis, she had structural heart disease as well. Dr. Deep based his treatment choice on the patient telling him that she previously had atrial fibrillation, without communicating with previous physicians. However, patients should be evaluated clinically and electrocardiographically prior to treatment with Propafenone (Exhibit 17). As in the previous patients, while Propafenone can be an appropriate drug for treating symptomatic atrial fibrillation, it is contraindicated in patients with structural heart disease (Exhibit 53).

Dr. Z concluded that the care was below standard in that Dr. Deep administered a potentially harmful drug without full diagnosis or communication with other physicians, and with little follow-up. The Committee accepts the evidence of Dr. Z.

Patient N

Patient N was first seen in 1995 at 70-years-old. Propafenone was started in 2002 based on Dr. Deep's observation of numerous supra ventricular ectopics beats and ventricular extra-systolic depolarizations. He postulates that an increase in Paxil, a SSRI antidepressant, may have been the cause and prescribed Propafenone. In 2004, Dr. Deep increased the dosage, based on a Holter monitor that showed 4603 supra ventricular ectopics beats. He stated that she had numerous atrial premature beats and a bigeminal rhythm on occasions with ventricular extra beats, and Propafenone was proper treatment. He stated that he discontinued the treatment in 2005 since she had a recurrence of bigeminal rhythm even with Propafenone. He opined that the widening of the QRS complex seen on an ECG was probably due to the development of a left bundle branch block (LBBB). He did not see any evidence of Propafenone toxicity.

Dr. Z's concerns included the poor quality ECG on which the diagnosis was based, and the prescribing of Propafenone for asymptomatic extra beats in a patient Dr. Deep believed to have structural heart disease. Propafenone use demonstrated a serious lack of judgment. There was widening of the QRS complex and, contrary to Dr. Deep's explanation, the LBBB pattern did not disappear or reverse with discontinuance of Propafenone, which would negate Dr. Deep's explanation for the widening of the QRS complex. He also did not monitor the patient adequately or stop the medication at an appropriate earlier time when the QRS widening was first noted. In fact, he did not stop the medication until a year later. Dr. Z concluded that this was evidence of Propafenone toxicity with a serious risk of pro-arrhythmia and the Propafenone should have been stopped immediately. He concluded that this reflected a very serious lack of knowledge and was the first time he observed a cardiologist do such a thing. Dr. Z was of the opinion that it was only luck that this patient did not sustain a serious, possibly fatal, ventricular arrhythmia.

The Committee accepts the explanation of Dr. Z and concludes that in the treatment of patient N, Dr. Deep showed a very serious lack of knowledge, skill, and judgment that placed the patient in danger.

Patient A

Dr. Deep first saw this patient, aged 61, in June 1997. He had recently undergone bypass surgery and was diabetic. Dr. Z's review of the laboratory results showed renal failure (see below for discussion on the differences of opinion on diagnosing renal failure). In January 1998, Dr. Deep started him on Propafenone. He based the use of the medication on a physical examination in which he noted numerous ectopic beats plus occasional bigeminal rhythm. The patient went on and off Propafenone over the next four years. Dr. Deep stated that anti-arrhythmic therapy to suppress symptomatic ventricular extra beats in patients with severe CAD with bigeminal and trigeminal rhythm is indicated. He used the Compendium of Pharmaceutical Drugs (Exhibit 17) in support of this treatment and an article (Exhibit 46). He stated that the extra beats were symptomatic. On review of the chart, Dr. Z was unable to find any cardiac symptoms that indicated the use of Propafenone. Dr. Z commented that Dr. Deep was treating asymptomatic premature ventricular beats, which he believed were based on misinterpreted ECG's. Dr. Z continued to point out that the literature clearly states that treating asymptomatic premature ventricular beats with anti-arrhythmic drugs in patients who are post-myocardial infarction is harmful. Patient A had ischemic heart disease (prior MI and coronary bypass) and structural heart disease based on the ECG of July 1997, which showed mild left ventricular systolic dysfunction and mild to moderate mitral regurgitation. Other concerns were starting Propafenone in an outpatient setting, lack of follow-up, dosage changes without ECG follow-up, initial prescribing based solely on the use of auscultation, and the use of Propafenone with inadequate monitoring when the patient had renal failure.

Dr. Z was further concerned with using Propafenone in combination with Indapamide, particularly when the nephrologist was not aware that the patient was taking Propafenone. Indapamide is known to cause QT interval prolongation and a potentially fatal arrhythmia

called Torsades de Pointes. The concomitant use of Propafenone and Indapamide is potentially dangerous and calls for either a reassessment of the need for Propafenone or, at minimum, more vigilant ECG monitoring.

Dr. Deep commented that Dr. Z's reliance on a 1989 study lacks an "air of reality." He also stated that the arrhythmias were identified by ECG and not based solely on stethoscope. He opined that Dr. Z's knowledge of the lack of QT prolongation with Propafenone is deficient.

The Committee accepts the testimony of Dr. Z. The patient was inappropriately treated with Propafenone as there was clearly structural heart disease, as well as a lack of significant ECG abnormalities in the tracings that could be utilized. The use of this medication falls below the standard of care. Dr. Deep's continued belief as to the use of this medication and his refusal to acknowledge the preponderance of evidence showing this drug was not used in a safe manner indicates that the allegation of incompetence is proved as well.

Patient O

This patient first saw Dr. Deep in 1985. There was a history of prior myocardial infarction, left ventricular systolic dysfunction, and bypass surgery. Dr. Deep first prescribed Propafenone in 1993. In 2001, in response to a question from Dr. W, a cardiologist, he stated that the patient never had atrial fibrillation but was on Propafenone for multiple symptomatic ventricular ectopic depolarizations. Dr. W stopped the Propafenone at his next visit. Dr. Z commented that, in reviewing the charts, there was no evidence of palpitations, no Holter monitor was done, and a stress test done in November 1992 showed no evidence of arrhythmia. A patient with ischemic heart disease and moderately impaired left ventricular systolic dysfunction should not be on Propafenone based on the previously noted criteria. Patient O was also known to have a complete right bundle branch block (RBBB) on his resting ECG since 1986, another reason that Propafenone was contraindicated. There was a lack of baseline studies and inadequate ongoing monitoring. Dr. Z's summary and conclusions were that this patient

should never have been on Propafenone. Given the history, the drug was absolutely contraindicated.

Dr. Deep states that Propafenone has a much lesser pro-arrhythmic effect than most of the drugs used in treatment of arrhythmias. Irrespective of the CAST study and other trials, which in his opinion may or may not be valid, untreated multiple VEDs in patients who have a very compromised coronary circulation have a negative effect on cardiac function and predispose them to ventricular tachycardia or ventricular fibrillation. He also stated that Propafenone, in small safe doses, prevented the VED's with no untoward effect. RBBB is a much less severe conduction defect than LBBB and it is unreasonable for a hospital physician to state that Propafenone should only be commenced in a hospital setting.

The Committee notes that evidence does not support Dr. Deep's contention that Propafenone has a less pro-arrhythmic effect than other drugs (Exhibit. 52). Dr. Deep stated that he would bring evidence showing that Propafenone works differently than other class 3 drugs, but valid evidence was never presented to the Committee. Notwithstanding Dr. Deep's contention as to the correctness of his judgment to use the medication in this case, there are still the serious issues of lack of proper diagnostic testing, documentation and follow-up. Dr. Deep, in his defence, does not believe any of the evidence presented by Dr. Z. The only contrary evidence presented is Exhibit 46, which shows that other physicians were still using the drug in the period 1991 to 1996. That paper does not suggest that Propafenone is safe, nor does it state the situations where it was used; it only states that new drugs should be introduced to deal with arrhythmic problems.

Conclusions Regarding Anti-Arrhythmic Treatment

1. The evidence of Dr. Z, as supported by Exhibits 13, 14, 15, 16, 17 and 53, is that Propafenone should be used only in situations where the well-documented risks are outweighed by the potential benefits. Propafenone is indicated for the treatment of documented life-threatening ventricular arrhythmias, such as sustained ventricular tachycardia. It may also be used for the treatment of patients with *documented*

symptomatic ventricular arrhythmias when the symptoms are of sufficient severity to require treatment (Exhibit 17). The same exhibit clearly states that the drug is contraindicated for people with prior heart disease (as documented in Exhibits 13 through 16). Dr. Deep discounted all of the evidence that Dr. Z presented. He stated that he believes the CAST study is flawed and, since Propafenone was not involved, it should be discounted. He did not comment on the other exhibits. He relied on Exhibit 46, which suggests that other physicians, at the time this study was done, were still prescribing Propafenone even where there was structural heart disease. The article does not support the use of Propafenone in these situations; it only says that some physicians were still using Propafenone in contraindicated situations and that other remedies were needed. Dr. Deep also continually stated that he would provide evidence that Propafenone is less toxic than other anti-arrhythmics but the evidence presented does not support that position. Dr. Deep's presentation of Exhibit 46 showed all anti-arrhythmics have potential harmful side effects.

Based on the overriding majority of evidence, we conclude that Propafenone should not be used where there is structural heart disease. Any use of this drug should have valid reasons for considering its use as compared to other possible treatments. If the drug is used, proper diagnostic testing must be done, there should be an awareness of other medications that might potentiate its adverse and life threatening effects, and there must be proper follow-up. None of these criteria were met by Dr. Deep.

2. The second major area of difference in the expert testimony is determining whether there was a symptomatic arrhythmia for which Propafenone is indicated. In each of the six patients that Dr. Deep treated with Propafenone, he relied on the patient's word, his stethoscope, or his in-office ECG machine to determine the arrhythmia. Dr. Z reviewed charts for each of these patients and found no significant arrhythmias. Dr. Deep demonstrated an insufficient level of care. Exhibit 17 clearly states that each patient who is to be administered Propafenone should be evaluated clinically and electrocardiographically prior to treatment. Dr. Deep's ECG machine was known to be inferior - ECGs were incomplete, unlabeled and below any acceptable standard as

evidenced by Dr. Z's testimony. Dr. Deep later replaced this machine and, while stating that he had no difficulty understanding the ECG's produced, utilized the new machine to show that he now does proper ECG's. Logic would dictate that he had to be aware or should have been aware of the state of tracings coming from his ECG, and that basing clinical decisions on these tracings was poor judgment and showed a lack of concern for the safety of patients under his care.

3. Because of Propafenone's potentially life-threatening adverse effects, the Compendium (Exhibit 17) states that it is essential that each patient administered Propafenone be evaluated clinically and electrocardiographically during therapy to determine whether the response to Propafenone supports continued treatment. There is no evidence that Dr. Deep provided ongoing monitoring.
4. Evidence was presented that patients with structural heart disease should be initially treated in a hospital setting (Exhibit 30). While the severity of illness of some of these patients may be questioned because Dr. Deep did not do a full clinical evaluation, at least some of them clearly did have a significant past history and should have been very closely monitored at onset. Dr. Deep disregarded the need for this level of care.
5. The Committee concludes that in the use of this medication, Dr. Deep falls below the standard of care and demonstrates incompetence.

Renal Function and Electrolytes

On review of the charts, Dr. Z raised the issues as to whether Dr. Deep properly managed clinical situations where renal failure was a real or potential issue, as well as whether he properly and safely monitored the measurement of serum electrolytes, especially potassium. The interaction of disease, the medications used to treat it, and the effects on organ systems such as the kidney are a prime area of potential harm to patients. The monitoring and recognition of potentially dangerous effects of medication and the progression of kidney disease is a major issue in the treatment of cardiac related problems and other illnesses.

Dr. Z testified that the main areas indicative of the level of practice in this category are measurement and recognition of renal failure, and the effects of medication and renal failure on the level of potassium in the blood. These issues are a concern in any patient, but especially those with diabetes, hypertension, or other causes of extrinsic or intrinsic kidney disease.

Dr. Z noted that the use of various medications for hypertension causes a 5% risk of renal failure, and a 10% risk of hyperkalemia (a rise in serum potassium above the normal range) in the first year of therapy. The risk of these effects rises with diabetes, chronic renal failure, or with the addition of beta-blocker medications and NSAIDs in combination with other medications.

Dr. Z explained that creatinine is a substance measured in the blood. It is a product of muscle breakdown and increases in the blood as the kidneys fail. It is a measure of kidney function. Many medications are secreted by the kidneys and require dosage adjustments to prevent toxicity. Many cardiac drugs have an effect on the kidneys and electrolytes as regulated by the kidneys. In six patients, serum creatinine measurements were not actively pursued, investigated or noted by Dr. Deep. These patients are: patient M, patient P, patient O, patient A, Dr. X and patient Q.

Dr. Z stated that the standard of practice is that potassium and creatinine should be measured at baseline, at one week and two to four weeks after initiation of therapy with medications that increase the risk of these changes. He noted that many of the patients had no baseline measurements and one patient had no electrolytes measured at all.

Dr. Z noted that in the 15 people placed on one or two antihypertensive medications (known as ARB's and/or ACE inhibitors), elevation of serum potassium was noted in three, which was in line with the known risk of elevation. Dr. Deep disagreed, insisting the risk of elevation was four in 1000. (Exhibit 23).

The patients noted to have become hyperkalemic were patient P, patient O, and Dr. X. Patient A had borderline elevation.

When using Aldactone (a medication known to potentially raise potassium levels) in combination with ACE inhibitors or ARB's, the chance of hyperkalemia increases and requires vigilant monitoring. Dr. Deep prescribed this medication combination to patient O and patient N. Dr. Z stated that hyperkalemia is seen in as many as 30% of patients on the above combinations, and occurred in patient O.

In Dr. Z's opinion, the hyperkalemia seen in these three patients was inadequately managed. All three cases related to inappropriate monitoring and had elevated potassium. Dr. Deep made no notations on the clinical record or lab reports acknowledging these abnormal results. One was recognized months later. Eventually changes were made, but usually months after the test. In addition, electrolytes were not measured each time the creatinine level was measured.

Dr. Deep did not appear to react to increases in serum creatinine in the six patients reviewed (patients M, P, O, A, Dr. X and Q). Dr. Z discussed the measurement of renal function. He pointed out that aside from the creatinine, the accepted way to measure and follow renal status is the eGFR (MDRD). This test is derived from the creatinine measurement and other known parameters and is routinely reported on all laboratory results. Dr. Z pointed out that elevations in this measurement were ignored and not treated or dealt with. Dr. Deep disagreed that this measurement has any validity. He cited the original paper describing this methodology and stated its accuracy was restricted to the groups tested in that original paper and did not apply to other groups. Dr. Z pointed out that further papers show the test is a sensitive and accurate predictor of renal function in the vast majority of patient groups, and that the original paper rightly confined its analysis of results to the group it tested. Dr. Deep's objections could not be used as a way to invalidate the test and the experience garnered over the years since the original paper.

Dr. Deep gave evidence concerning articles reviewed on the internet stating that the test is unreliable. We saw no reputable articles that would lead to this conclusion.

Dr. Deep continued to ignore the mass of accumulated evidence as to the validity of this measurement in justifying his reliance on creatinine levels alone in the monitoring of his patients.

The Committee is convinced by the preponderance of evidence and the common standard that this measurement is a valid predictor, and attention to values outside the normal range would be expected of any physician. Dr. Deep continues to believe that, in his sole judgment, this value can be ignored. The Committee is of the opinion that this does not meet the professional standard, shows poor judgment and places patients at risk.

In patient A's case, Dr. Deep prescribed Propafenone and Lozide multiple times. Dr. V was following patient A in the nephrology clinic at a Toronto hospital. It is clear from Dr. V's communications to Dr. Deep that she was unaware he was taking Propafenone. Dr. Deep did not correspond with Dr. V. In his defence, he stated that patient A was intelligent, had a drug list, and would have communicated with the physicians he was seeing at the nephrology clinic. As noted in discussion on anti-arrhythmics, Lozide is known to prolong the QT interval and contribute, especially in combination with Propafenone, to a potential fatal arrhythmia called Torsades de Pointes.

Dr. Z noted hyperkalemia in three patients: P, O, and Dr. X. Except for patient O, whose elevation was noted months after being reported at which time medication changes were made, there was no notation on the clinical record of their laboratory reports showing elevation, or any action taken. Dr. Deep stated he reacted to the result, and that obviously the patients were well at this time.

Dr. Z noted on cross-examination that potassium elevation can result in sudden death, and the fact that the patients did not die is not evidence of proper treatment. Dr. Z added that poor record-keeping might well have been the cause of not reacting in an acceptable

manner. We agree, as it is obvious that Dr. Deep is aware of the issues associated with an elevation of potassium.

The Committee is of the opinion on the evidence accepted by it that the care of these patients in this regard fell below the expected professional standard, and Dr. Deep's insistence that there were no problems in his non-response to the abnormal results of laboratory tests leads the Committee to conclude that his lack of judgment is such that incompetence is established.

Lipid Metabolism and Treatment

The treatment of lipid abnormalities is an integral part of medical care and especially central in the practice of preventive medicine and cardiology. It is well known that a low level of high density cholesterol and a high level of low density cholesterol are major predictors of heart and blood vessel disease. One of the treatments available is lipid lowering medication. Instituting treatment with these medications commits the patient to long term medication use with the attendant possibility of side effects, as well as associated financial costs. Dyslipidemia is one of many risk factors for developing coronary artery disease. It is, however, a risk factor that can be modified with lifestyle changes and medications. Over the years, guidelines have been established to help physicians rationalize lipid management and provide consistent care based on certain criteria. The guidelines have changed with time as new information, based on research, has come along. As compared to guidelines from the past, current guidelines promote a more aggressive approach to dyslipidemia management in those patients at high risk of coronary artery disease and a less aggressive approach for those at low risk. The guidelines recommend using the Framingham Risk Score to calculate cardiovascular risk as part of an initial patient assessment. This score helps physicians determine what the best course of management might be for each individual patient based on their projected risks of developing coronary heart disease. The guidelines therefore look for best practices in estimating the risk for patients based on multiple factors, including blood lipid measurements. They recommend, at the time each of the guidelines were published, the best known information on the best treatment for a statistically positive outcome with

the least likelihood of negative short and long term effects. All the guidelines emphasize the need for lifestyle change as the first line of treatment, including rigorous dietary modifications and exercise. The use of these latter modalities often requires the cooperation of other health care workers, such as dietitians and exercise prescribers.

Dr. Z testified that, in relation to management of dyslipidemia, there were deficiencies in Dr. Deep's care of patients as a cardiologist and internist. There was a lack of measurement of blood lipids and other chemistries before starting a program of drug intervention, diet and exercise were not discussed, follow-up was inadequate, medication dosages were excessive, lipid levels achieved were often excessive considering the risk levels of the patients, and guidelines were not followed for instituting or monitoring lipid levels. Dr. Z utilized the guidelines in effect at the time that each patient was being treated. In his estimation, while guidelines are a summary of best practices based on the accumulated data, they are meant to guide. It is important for the physician to indicate why he is choosing a different path, to inform the patient of the reasons for the choice, and the risks and benefits attendant on doing so. The patients below were reviewed from this perspective.

Dr. Deep informed the Committee that he used his best judgment and that the guidelines were often behind the facts. He stated that he expected in the future all patients would be treated as individuals based on their individual genetic makeup, and he was ahead of the curve in utilizing data that would point to this in making decisions as to when and how to treat. He also felt there was no need to subject his patients to an additional dietary consultation as he prescribed the DASH (Dietary Approaches to Stop Hypertension) diet and, in any case, it would have been inconvenient for the patients to travel to a dietitian. The Committee found no documentation of this in the patient's records. He also indicated that often patients came to him with hyperlipidemia and he saw no reason to do additional tests before treating, as they were self-reporting the situation. He also considered family history to be of greater importance than acknowledged in the guidelines, and a better guide to treatment. As well, due to lifestyle considerations, his patients were unable to attempt exercise or had failed at it prior to his instituting treatment.

Patient H

Dr. Deep first saw this patient in June of 1989. It is unclear, from the medical record, how or why she came to be a patient.

In August 2005, he prescribed Lipitor, 10 mg, every one to two days. At the time, she was 67-years-old and her medical record listed the following diagnoses: hypertension, mitral regurgitation and aortic stenosis.

Lab values, from June, 2005, indicated that her LDL (so called “bad cholesterol”) was 2.93 mmol/L and her TC:HDL-C ratio (total cholesterol to HDL ratio) was 2.83.

Dr. Z testified that Dr. Deep:

1. Failed to document the indication for use of Lipitor in this patient.
2. Failed to recommend a trial of diet and exercise prior to initiating pharmacological therapy.
3. Failed to estimate this patient’s cardiovascular risk according to the guidelines of the day (Recommendations for the management of dyslipidemia and the prevention of CVD: 2003 update).
4. Failed to discuss the goals of pharmacological therapy and set target lipid levels.
5. Failed to consider other statins that may have been reduced the potential for a drug interaction with Propafenone (another drug prescribed by Dr. Deep).

In response to the evidence presented, Dr. Deep testified that an aortic atheroma, detected on an echocardiogram done in August, 1998, constituted a stronger indication to normalize patient H’s lipids. He stated that he did not wait for guidelines to tell him to

normalize serum cholesterol in a patient with substantial cardiac problems; this was just common sense and known in the 1960s before guidelines kept coming and changing.

The echocardiogram, referred to in Dr. Deep's testimony, reported patient H to have mild mitral valve regurgitation, a mild aortic atheroma and no aortic stenosis.

The Committee finds Dr. Z's testimony was clear, cogent and convincing. Dr. Deep's testimony does not change the Committee's conclusions, as it does not answer the facts noted by Dr. Z. We would note that the echocardiogram results referred to by Dr. Deep do not support his argument regarding his treatment choice.

Patient J

It is not clear from the medical record when or why Dr. Deep first saw this patient. It may have been as early as 1997.

In October of 2001, Dr. Deep prescribed Pravachol, 20 mg at bedtime, for this patient. He was 53 at that time and had the following list of diagnoses: mitral regurgitation, chronic anxiety, GERD (reflux) and hemorrhoids.

Lab values from September, 2001 indicated that he had an LDL of 3.95 mmol/L and a TC:HDL-C ratio of 4.23.

Dr. Z testified that Dr. Deep:

1. Failed to document an examination or visit on the day Pravachol was prescribed.
2. Failed to estimate the patient's cardiovascular risk according to the guidelines of the day (Recommendations for the management of dyslipidemia, 2000). This patient's cardiovascular risk could be calculated to be low and he had lipid values already at target for an individual at moderate risk, without instituting pharmacological therapy.

3. Failed to discuss the goals of pharmacological therapy and set target lipid levels.
4. Failed to establish adequate follow-up or initiate monitoring for potential toxicity related to use of this medication.

In response to the evidence presented, Dr. Deep testified that he had treated patient J's mother for many years. She had multiple risk factors for heart disease and died in her late eighties. With patient J's family history, Dr. Deep felt it was important to treat his hyperlipidemia. Dr. Deep stated that he tried diet and exercise for this patient but his lipids remained high. He added that he did not know if he had documented this in his file but knew, from his recollection of patient J's treatment, that this was the case. He stated further that he did not adhere robotically to guidelines.

The Committee notes Dr. Deep's contention that the patient's family history was a factor in the treatment instituted. While clinical judgment is a valued parameter, this patient, even factoring in the mother's history, was at target levels before treatment. As well, the lack of documentation noted by Dr. Z and the lack of any indication or any chart notation of use of diet and exercise, such as referral to a dietician, leads us to conclude that these treatments were, on the balance of probabilities, not done.

Patient M

Dr. Deep first saw this patient in July of 1997. It is not clear if another physician referred him.

In February 2004, Dr. Deep prescribed Lipitor for this patient. He was 83 at that time and had the following list of diagnoses: history of pulmonary embolus, emphysema, coronary artery disease with angina, osteoarthritis and clubbing.

The most recent lab values in the chart were from July 1999, and indicated an LDL of 2.46 mmol/L and a total cholesterol level of 3.94 mmol/L. No lipid levels appeared in the medical chart from 1999 to 2004.

Dr. Z testified that Dr. Deep:

1. Failed to adequately and appropriately establish this patient's cardiovascular risk.
2. Failed to assess this patient's lipid status prior to prescribing the Lipitor.
3. Failed to discuss target lipid levels and set goals for pharmacological therapy.

Dr. Deep did not specifically reply to these concerns in his oral testimony. The Committee accepts Dr. Z's testimony and concludes that Dr. Deep did not meet the standards of the profession in his treatment of this patient.

Patient R

In May 2004, at her initial visit, Dr. Deep prescribed Lipitor, 20 mg, at bedtime, for this patient. She was 50-years-old at the time and had the following list of diagnoses: hypertension, obesity, hyperlipidemia, probable mild "NOCAD," sinusitis, cervical disk disease and varicose veins.

No lab work for lipid levels appeared in her medical record until September 2004, and no lab work was done prior to initiating pharmacological therapy.

In September 2004, her LDL was 2.35 mmol/L and her TC:HDL-C ratio was 3.71. In February 2005 her LDL was 2.79 mmol/L and her TC:HDL-C ratio was 3.67.

In January 2005, Dr. Deep documented prescribing L-Thyroxine 50 ug to help this patient with her weight reduction and cholesterol. From the laboratory values in the medical record regarding her thyroid status, a diagnosis of hypothyroidism was not clearly established.

Dr. Z testified that Dr. Deep:

1. Failed to appropriately assess this patient's lipid levels prior to prescribing a lipid-lowering agent.
2. Failed to recommend a trial of diet and exercise prior to initiating pharmacological therapy.
3. Failed to establish the patient's cardiovascular risk according to the guideline of the day (Recommendations for the management of dyslipidemia and the prevention of CVD: 2003 update). This patient was already at target lipid levels for an individual who could be calculated to be low risk.
4. Failed to discuss the goals of pharmacological therapy and set target lipid levels.
5. Failed to establish a diagnosis of hypothyroidism (a known secondary cause of hypercholesterolemia) and, if so established, failed to adequately treat this disorder and reassess lipid status prior to starting Lipitor.

In response to the evidence presented, Dr. Deep stated that he relied on tests she had done in Austria. He added that her brother had suffered a myocardial infarction at age 47 and that her father had died of a ruptured thoracic aortic aneurysm. She was obese and could not lose weight despite his prescribing a DASH diet for her. He stated she should have her cholesterol normalized despite the guidelines and he did not see the advantage of waiting for the patient to lose weight before instituting therapy. He acknowledged that the effect of lipid lowering on the vasculature might not be seen for 12 to 24 months. He felt her treatment had been highly successful.

Of note, no copies of lab testing done in Austria or specific reference to such testing was found in patient R's chart.

Based on the evidence, the Committee is of the opinion that the treatment of this patient fell below the standard. Dr. Deep's response was not supported by evidence that ought to

have been available, especially as he states he relied upon it. We are unconvinced that Dr. Deep made an effort to treat the patient with diet, exercise and lifestyle changes either before or as a part of her treatment, nor was there the expected communication between Dr. Deep and other physicians caring for her.

Patient F

In August 2000, Dr. Deep prescribed Lipitor, 20 mg at bedtime, for this patient at her initial visit. She was 65-years-old at the time and had the following list of diagnoses: hypertension, sleep apnea, Pott's disease, rheumatic fever, aortic stenosis, Sjogren's syndrome, obesity and coronary artery disease with "mild MI."

The first lab work reported in the medical record was done in December of 2000. Dr. Z testified that, other than performing blood pressure measurements, Dr. Deep did not examine this patient's cardiovascular system until 2004. In addition, he testified that the diagnosis of coronary artery disease and previous myocardial infarction ("mild MI") were not established diagnoses but based on Dr. Deep's misinterpretation of a nuclear medicine stress test done in July of 2000. No further objective testing was done until after 2001.

Dr. Z concluded that Dr. Deep:

1. Failed to perform adequate and appropriate lab work to establish the patient's lipid levels prior to prescribing Lipitor.
2. Failed to establish a clear indication for use of Lipitor.
3. Failed to recommend a trial of diet and exercise prior to initiating pharmacological therapy.
4. Failed to initiate a referral to a dietician despite the patient's diagnosis of obesity and abnormal fasting blood glucose levels reported in her medical record.

5. Failed to discuss the goals of pharmacological therapy or establish target lipid levels.

In response to the evidence presented, Dr. Deep stated that patient F was very obese, had attempted a diet, and could not exercise because of her obesity. He told the Committee that he had, of course, discussed treating her lipids with diet and exercise but that this was out of the question. He said she had voluntarily discontinued taking her cholesterol medications and, on those occasions, her cholesterol levels became elevated. When she was on the medication, her cholesterol was well controlled. He added that he put all of his patients on DASH diets because this diet included measures to control cholesterol. He stated further that her obesity, aortic stenosis and angina would indicate the need for statin control of her cholesterol and that she would certainly have plaques in her arteries not shown on her radioisotope studies. He added that her husband, a physician, was very happy with her treatment and brought her back frequently.

No documentation of a trial DASH diet was found in the medical record.

The Committee concludes that Dr. Deep's replies do not answer the concerns noted by Dr. Z. Dr. Deep answers valid concerns, such as the misinterpretation of the radioisotope study, with speculative assumptions of what might be present in the face of evidence to the contrary. Dr. Z's concerns are valid and reflect Dr. Deep's failure to meet the standards expected of a cardiologist and his disregard for the welfare of the patient.

Patient D

In September, 2000, Dr. Deep prescribed Pravachol, 10 mg, for this patient at her initial visit. She was 70-years-old at the time and had the following list of diagnoses: stroke, mitral valve prolapse, hyperlipidemia and anxiety.

The first lab tests done were performed in September, 2000 and indicated a total cholesterol of 5.85 mmol/L, an LDL-C of 3.25 mmol/L and a TC:HDL-C ratio of 3.47. In mid September, 2000, Dr. Deep increased her dose of Pravachol to 20 mg.

Dr. Z testified that Dr. Deep:

1. Failed to assess this patient's lipid levels prior to prescribing a statin.
2. Failed to establish an indication for prescribing this medication for this patient. In the year 2000, there was no scientific evidence to support the use of a statin in hyperlipidemia to prevent a future stroke.
3. Failed to discuss the goals of pharmacological therapy or target lipid levels according to the guideline of the day (The Canadian Consensus Conference on Cholesterol: Final Report).
4. Failed to recommend an adequate trial of diet and exercise prior to initiating pharmacological therapy according to the guideline of the day.
5. Failed to assess her initial response to the 10 mg dose of Pravachol prior to increasing the dose to 20 mg.

In response to the evidence presented, Dr. Deep stated that patient D was a nurse, knew her lipids were high and was on statins when she came to his office. He said that clearly, he would continue her statins at that visit and he, in fact, increased her dose.

Dr. Deep referred to an excerpt from "Cardiology Scientific Update", dated June 2006. This is a review publication put out by St. Michael's Hospital Department of Cardiology. Dr. Deep commented that the ASTEROID Trial had demonstrated that, by IVUS (intravascular ultrasound), regression of atherosclerosis could be seen in individuals taking a lipid medication known as Rosuvastatin.

Dr. Deep initially said that patient D's lipid management indicated statin therapy because she had a history of stroke. On referring to the publication above, he indicated that

patient D had had angina.

Dr. Deep also referred to the SPARCL Trial (High-Dose Atorvastatin after Stroke or Transient Ischemic Attack), published in the New England Journal of Medicine, August 10, 2006. He stated that this article supported his treatment of patient D's lipids because she had suffered a stroke and had TIA's (transient ischemic attacks). He said this article concluded that in patients with recent stroke or TIA, the incidence of future stroke was reduced by using Atorvastatin. There was a slight increased risk of hemorrhagic stroke in patients with known coronary artery disease. He said it would have been improper not to treat her lipids with statins.

Dr. Z stated that the benefit of lowering lipids in decreasing the risk of future stroke was not known until after this trial was published in August of 2006. Dr. Deep treated her lipids with statin therapy in 2000.

The Committee concludes that, even with supporting evidence in 2006 that a specific statin is of use in reducing future stroke, albeit with an increase in hemorrhagic stroke in those with coronary artery disease, Dr. Deep had no way of knowing this in 2000. In any case, he did not properly monitor her medication, nor was he using the medication noted in the study. We find his explanation an ex post facto argument that does not excuse his mismanagement of her condition, and find that he did not meet the standard expected of a cardiologist.

Patient K

Dr. Deep first saw this patient in March of 1997. At the time she was 58-years-old and had the following diagnoses listed: hypertension, "RHD with MR", atrial fibrillation, symptomatic ectopic depolarizations and anxiety.

She had blood work done in April, 1997 that showed a total cholesterol of 5.69 mmol/L and a triglyceride level of 0.83 mmol/L. In mid April, 1997, blood work was done again and indicated a total cholesterol level of 6.12, a triglyceride level of 0.86, an LDL of 4.12

and an HDL-C of 1.61 mmol/L.

While it was not clear from the medical record when Dr. Deep initiated therapy with Pravachol 20 mg, it appears to have been prescribed in late November, 1997.

Subsequent lipid levels done in October, 1997 showed a total cholesterol of 5.29, a triglyceride level of 1.06, an LDL-C of 3.49 and an HDL-C of 1.32 mmol/L.

Dr. Z testified that Dr. Deep:

1. Failed to establish an indication for prescribing a statin for this patient.
2. Failed to establish an appropriate target lipid level according to the guidelines of the day.

We heard no evidence that would contradict the above concerns, and it is the opinion of the Committee that the treatment of this patient by Dr. Deep demonstrates a serious lack of knowledge, skill and judgment and constitutes care below the standard of practice.

Patient S

It is not clear from the medical record when Dr. Deep first saw this patient. The earliest notes date to May 1984, but there is lab work in the file dating back to 1982. In 1984, patient S was 40-years-old and had the following list of diagnoses: coronary artery disease, anterior wall myocardial infarction, mitral regurgitation, aortic stenosis, hypertension, hyperbetalipoproteinemia and homonymous hemianopsia.

Dr. Deep prescribed Pravastatin for patient S. He did have established hyperlipidemia. Lab work done in 2003 indicated that his LDL-C on the statin was 1.88 mmol/L. In August of 2005, the Pravastatin was discontinued because of medication-related side effects and the patient was prescribed Lipidil Supra.

Of note, in 1996, the patient had a nuclear medicine stress test done and there was no evidence of ischemia or prior myocardial infarction at that time.

Dr. Z expressed concern that Dr. Deep:

1. Failed to discuss the goals of pharmacological therapy and establish appropriate target lipid levels. In 2003, the guideline of the day did not recommend a target LDL of <2 mmol/L even in a high risk patient.
2. Failed to acknowledge that the 1996 negative stress test suggested a more reasonable target LDL for this patient may have been <4 , indicating less aggressive statin therapy.

In discussing the management of patient S's lipids Dr. Deep referred to a one page excerpt from a publication entitled "The Biology that Underlies Atherosclerotic Disease," by Dr. Peter Libby. The date or source of this publication could not be identified; this publication did not appear to be an article from a peer-reviewed journal.

Dr. Deep quoted Dr. Libby in saying that: "angiography offers a poor basis for therapeutic decisions" and "minor plaques cannot be appreciated by angiography". Dr. Deep testified that this concept was important in his treatment of patient S. He added that this patient had a normal angiogram, a strong family history and his lipids were elevated. He said he had elected to stabilize any plaque that might be in existence. He indicated that, in his view, patient S did not need a coronary arteriogram but needed treatment of his lipids.

While this patient did have established hyperlipidemia, the Committee found that Dr. Deep's explanation lacked a credible grounding. As well, the results of the tests showing the long held misdiagnosis further causes the Committee to find that Dr. Deep demonstrated a lack of knowledge, skill and judgment and that his care of the patient was below the standard of practice.

Patient T

Dr. Deep first saw this patient in September 2000. At that time, she was 79-years-old and had the following list of diagnoses: bronchitis, coronary artery disease with angina pectoris, osteoarthritis, “? past hypertension” and anxiety. Her medical record dated November 15, 2000, states: “add Pravachol.” In late April, 2003 another note states: “Pravachol”.

Non-fasting lab values from September, 2000 indicated that her total cholesterol value was 5.78 and her triglyceride level was 1.38 mmol/L.

Dr. Z testified that Dr. Deep:

1. Failed to clearly identify if this medication had been prescribed for this patient and if so, what dose she was prescribed and whether or not she was taking it.
2. Failed to perform a repeat fasting lipid profile after subsequent abnormal non-fasting values were reported.
3. Failed to adequately and appropriately address potentially modifiable cardiovascular risk factors in this patient including her hypertension and smoking.
4. Failed to recommend, according to the guidelines of the day, a trial of diet and exercise to lower her lipids.

In discussing the management of patient T’s lipids, Dr. Deep referred to an article published in The New England Journal of Medicine, entitled “Optimal Medical Therapy with or without PCI for Stable Coronary Disease,” dated April 12, 2007 (PCI is another term for angioplasty). He quoted the study conclusion that: “as an initial management strategy in patients with stable coronary artery disease PCI did not reduce the risk of death, myocardial infarction or other major cardiovascular events when added to optimal medical therapy.”

Dr. Deep stated that he did not send patient T for perfusion studies because she had arthritis and had difficulty getting out of bed. This would not have improved her management and he opted for conservative therapy that included aspirin, beta-blockers and statins.

We find that Dr. Deep's testimony did not address the valid concerns expressed by Dr. Z. Not performing perfusion studies for the stated reasons does not remove any obligation to properly manage the dyslipidemia aspects of this case. We find that the treatment of this patient demonstrates a serious lack of knowledge, skill and judgment and constitutes care below the standard of practice.

Patient U

From the medical record, it appears Dr. Deep first saw this patient in June 1997. The following are listed as her diagnoses: mitral regurgitation, "BEH" ("? benign essential hypertension"), obesity, varicose veins, capillary fragility and bereavement depression.

Lab tests were done in June, 1997 and indicate a total cholesterol of 7.28, an LDL-C of 5.24 and an HDL-C of 1.20 mmol/L.

Dr. Deep prescribed Pravachol, 20 mg daily, for her in July, 1997. From 1997 to 2003, the medical record indicates that he made many changes to her lipid-lowering medication. The type and dose of medication was changed multiple times with no clear documentation as to the reasons for these changes. In 1998, it appears as though she may have been taking both Pravachol and Lipitor.

In 2005 her LDL was 2.3 mmol/L and her TC:HDL-C ratio was 2.9.

While this patient had an established diagnosis of hyperlipidemia, Dr. Z noted that Dr. Deep:

1. Failed to recommend an adequate trial of diet and exercise according to the guideline of the day, prior to initiating pharmacotherapy (The Canadian Consensus Conference on Cholesterol: Final Report).
2. Failed to clearly document the changes made to lipid medication regimens and why those changes were made.
3. Failed to adjust the patient's target lipid levels in accordance with the changes in guidelines over the course of her care.

Dr. Deep stated that, in his estimation, she was a high-risk patient because her sister and mother had strokes. He thought aggressive lipid therapy was indicated for her.

We find that the concerns expressed by Dr. Z are valid, and worrisome as to the concern for the welfare of this patient. Dr. Deep's explanation boils down to judgment trumping all other timely guidelines and consensus, as well as progress of management within these paradigms of care.

It is the opinion of the Committee that this demonstrates a serious lack of knowledge, skill and judgment and constitutes care below the standard of practice.

Patient AA

Dr. Deep first saw this patient in March, 2005. She was 72-years-old and had the following list of diagnoses: angina pectoris "probably secondary to coronary artery disease", S.A.D., "C.D.D." and "r/o pred. factors."

Dr. Deep prescribed Lipitor for her at the initial visit. In the summer of 2005, she was switched from Lipitor to Lipidil Supra.

In October 2005, her AST and ALT (tests for liver enzyme level) had increased to four to six times normal. The letter referring this patient to Dr. Deep indicated that she had elevations in her liver tests in 1989.

Dr. Z testified that Dr. Deep:

1. Failed to assess her lipid status prior to prescribing pharmacological therapy.
2. Failed to recommend an adequate trial of diet and exercise prior to initiating lipid-lowering drug therapy.
3. Failed to establish her cardiovascular risk according to the guidelines of the day.
4. Failed to discuss the goals of pharmacological therapy and set target lipid levels.
5. Failed to acknowledge, on the lab report or in the clinical record, a significant elevation in liver function tests following initiation of lipid-lowering medication.

In response to the evidence presented, Dr. Deep testified that repeat liver tests were done and were normal. He stated that the elevation in her liver function tests could be explained on the basis of such factors as wine consumption and fatty liver.

We find that Dr. Deep's explanation is superficial and after the fact speculation presented as fact. The Committee accepts the opinion of Dr. Z.

It is the opinion of the Committee that the treatment of this patient demonstrates a serious lack of knowledge, skill and judgment and constitutes care below the standard of practice.

Patient A

Dr. Deep first saw patient A in June, 1997. He was 61-years-old and had the following list of diagnoses: CABG (May 16/97), acute "diaphragmatic MI," remote ASMI, mitral regurgitation, diabetes mellitus and renal/ureteric calculi. From this record, it is clear that he had undergone coronary bypass surgery the month prior to his first visit.

Dr. Deep prescribed Pravachol, 20 mg daily, at the initial assessment. Lab testing was done in June, 1997 and indicates a total cholesterol of 4.92, triglyceride level of 0.59, LCL-C of 2.94 and an HDL-C of 1.71 mmol/L.

In Dr. Z's opinion, Dr. Deep:

1. Failed to assess the patient's lipid status prior to prescribing pharmacotherapy.
2. Failed to recommend a trial of diet and exercise according to the guidelines of the time.
3. Failed to repeat the lipid levels in two months time to insure that the initial levels were not related to the acute effect of bypass surgery.
4. Failed to establish target levels of LDL-C for this patient.

Dr. Deep did not address this evaluation and opinion in any way the Committee could assess. The Committee accepts the opinion of Dr. Z.

It is the finding of the Committee that Dr. Deep's treatment of this patient as described above demonstrates a lack of knowledge, skill and judgment and constitutes care below the standard of practice.

Patient B

Dr. Deep first saw this 65-year-old female in December, 1997. She had apparently been on Lipitor and was not taking her medication. At the initial visit, Dr. Deep prescribed Lipitor, 20 mg daily. Lab testing was done in January 1998.

Dr. Z testified that Dr. Deep:

1. Failed to establish baseline lipid levels prior to prescribing pharmacotherapy.

2. Failed to assess her cardiovascular risk.
3. Failed to recommend a trial of diet and exercise as per the guidelines of the day.
4. Failed to establish treatment goals.

In response, Dr. Deep stated that initially patient B's main problem was hypertension. A DASH diet and exercise program was tried. He said her blood pressure normalized eventually due to understanding her stresses and strains and with lifestyle adjustments. Following this, the major problem became her hyperlipidemia, as her cholesterol was very high.

We can find no evidence in the chart to support Dr. Deep's contention that lifestyle modification was tried. No use of other resources, if his efforts were insufficient, was noted. If he considered hyperlipidemia her main problem, his lack of instituting proper treatment protocols did not address this concern.

It is the finding of the Committee that Dr. Deep's treatment of this patient as described above demonstrates a serious lack of knowledge, skill and judgment and constitutes care below the standard of practice.

Patient N

Dr. Deep first saw this 70-year-old woman in August, 1995. Her diagnoses were listed as follows: hypertension, coronary artery disease with unstable angina, hyperlipidemia, obesity and bereavement depression.

At the initial visit, Dr. Deep discontinued all of her cardiac medications and prescribed a number of new medications including Pravachol, 20 mg daily.

Laboratory testing was done in August, 1995 and indicated her total cholesterol was 5.50,

triglycerides were 2.09, LDL-C 3.07 and her HDL-C 1.48 mmol/L. Her CK (an enzyme that can be used to measure cardiac muscle status) was mildly elevated but this was not acknowledged on two subsequent visits in August of 1995.

Dr. Deep discontinued statins on several occasions secondary to complaints of muscle weakness. He prescribed Lipitor in July 2004. No recent lipid levels were found in the patient's medical record. CK was again mildly elevated in May 2004 but no subsequent CK levels were found in the clinical record. An AST was not done until October of 2005.

Dr. Z testified that Dr. Deep:

1. Failed to perform a baseline assessment of her lipid status to establish the diagnosis prior to prescribing pharmacotherapy.
2. Failed to recommend a trial of diet and exercise prior to initiating pharmacotherapy according to the guidelines of the day.
3. Failed to acknowledge, on the lab report or in the clinical record, an elevated CK on two occasions.
4. Failed to institute adequate and appropriate monitoring of her CK and liver tests after initiating statin therapy to look for potential drug-related toxicities.

We heard no reply evidence that addressed the issues Dr. Z raised in a direct and meaningful manner and we accept Dr. Z's testimony.

It is the finding of the Committee that Dr. Deep's treatment of this patient as described above demonstrates a serious lack of knowledge, skill and judgment and constitutes care below the standard of practice.

Dr. X

Dr. Deep first saw this 73-year-old man in October, 2000. His diagnoses were listed as follows: hypertension with microalbuminuria, NIDDM, psoriasis, diverticulosis, gastritis, obesity and anxiety.

Dr. Deep prescribed Lipitor, 20 mg, a medication he was already taking.

Laboratory tests were recorded with no date as to when they were done. Lipid levels were not included. Subsequent lab testing was done in October, 2000 and indicated a total cholesterol of 4.71, triglyceride level of 3.46, LDL-C of 2.13 and an HDL-C of 0.99 mmol/L.

Dr. Deep discontinued Lipitor at either a September, 2001 or October, 2001 appointment and prescribed Lipidil Supra in its place. The date is not certain from the chart nor is the reason for the change in medication clearly identified. In December, 2001, he prescribed Lipitor again. In November 2004, he increased the dose of Lipitor to 40 mg daily. The patient's creatinine level had increased over time to 206 in October 2005.

Dr. Z testified that Dr. Deep:

1. Failed to discuss target lipid levels.
2. Failed to recognize that a reduced dose of Lipidil Supra should have been considered at the time the initial prescription was given, considering the patient's impaired renal function.
3. Failed to re-evaluate the dose or need for Lipidil Supra as the patient's creatinine became increasingly elevated.

We heard no reply evidence that addressed the issues Dr. Z raised in a direct and meaningful manner.

We accept the expert opinion of Dr. Z, and find that Dr. Deep did not meet the expected professional standard of practice in the care and treatment of this patient.

Patient L

Dr. Deep first saw this 40-year-old woman in December of 1997. No diagnosis was noted in the chart at that time.

The medical record does not provide a clear progression of events. It appears that Dr. Deep prescribed Lipitor in December, 1998. Several notes mention use of a statin and one undated entry states “D/C Pravachol – liver.” “Lipitor 10” is recorded in some notes from the spring and summer of 1999, and a note from October 1999 states “considering Pravachol Q2days.”

Lab testing done in December of 1998 indicated a total cholesterol of 6.1, triglycerides of 0.94, HDL-C of 1.51 and LDL-C of 4.16 mmol/L. From the clinical record it appears that she continued to take a statin at least until November of 2005. Lab tests from September of 2005 showed a total cholesterol of 3.7, LDL-C of 1.8, HDL-C of 1.6 mmol/L and a TC:HDL-C ratio of 2.3.

Dr. Z testified that Dr. Deep:

1. Failed to recommend a trial of diet and exercise prior to initiating pharmacotherapy.
2. Failed to discuss the goals of pharmacotherapy and set target lipid levels.
3. Failed to discuss the risks of statin use.
4. Failed to adjust target lipid levels based on cardiovascular risk and changes to guidelines based on evolving research over time.

5. Failed to chart in a fashion that would allow another health care worker to follow the treatment progression and rationale.

We heard no reply evidence that addressed the issues Dr. Z raised in a direct and meaningful manner.

It is the finding of the Committee that Dr. Deep's treatment of this patient described above demonstrates a serious lack of knowledge, skill and judgment and constitutes care below the standard of practice.

Patient BB

Dr. Deep first saw this 58-year-old man in June, 2005. The following list of diagnoses was present in the chart: "LSDD," osteoarthritis, remote pelvic fracture, remote renal calculi and obesity.

At a subsequent visit two weeks later, Dr. Deep provided a weight reduction diet to the patient. In July, 2005, lab testing indicated a total cholesterol of 5.91, LDL of 3.79, HDL of 1.16 mmol/L and a TC:HDL of 5.09. Late in July, 2005, Dr. Deep prescribed Lipitor, 10 mg daily, for the patient. Lab testing in September 2005 showed his LDL to be 2.44 and his TC:HDL ratio to be 3.79

Dr. Z testified that Dr. Deep:

1. Failed to estimate the patient's cardiovascular risk according to the guidelines of the day. Calculating the patient's cardiovascular risk assessment to target him at low risk, he would be at target lipid levels without pharmacotherapy.
2. Failed to recommend an adequate trial of diet and exercise prior to initiating drug therapy.
3. Failed to discuss goals of pharmacotherapy and set target lipid levels.

4. Failed to identify a plan for monitoring potential drug-related toxicities.

We heard no reply evidence that addressed the issues raised by Dr. Z in a direct and meaningful manner.

It is the finding of the Committee that Dr. Deep's treatment of this patient described above demonstrates a serious lack of knowledge, skill and judgment and constitutes care below the standard of practice.

Patient Q

Dr. Deep first saw this 71-year-old man in August, 2002. He had recently been discharged from hospital following an acute myocardial infarction. His diagnoses were listed as: coronary artery disease with recent "AWMI with apical involvement," hyperlipidemia and anxiety regarding BP.

In September 2006, lab work showed his total cholesterol to be 4.04, triglycerides 1.53, HDL 0.82, LDL 2.52 mmol/L and TC:HDL ratio 4.93. Dr. Deep treated him with lipid-lowering medication.

Dr. Z testified that Dr. Deep:

1. Failed to set appropriate lipid target levels for this patient despite the fact that he had established coronary artery disease, a history of myocardial infarction and was at high risk for future cardiovascular events.

The Committee noted that, as in the case of patient A, Dr. Deep failed to get baseline lipids and justified not doing this based on an extensive cardiac family history; because of this history, the patient should have levels of LDL.C reduced to <2 mg. Dr. Z noted that the guidelines at that time were not that low. Dr. Deep testified he was "ahead of the curve" in going for blood levels that low.

Dr. Z testified there was no doubt that this patient required therapy for hyperlipidemia; however, the difficulty with Dr. Deep's justification for his treatment goals is that guidelines are set not only by considering best known data. The level is set to produce what is considered the best outcome versus the risks of using the medications. No medications are without risk and the guidelines, relying on evidence-based medicine, are set to produce the best outcome with the fewest side effects and expense. While a physician's judgment may go outside these guidelines, it must be based on facts more than on beliefs, and an ex post facto justification (the lowering of the target levels in later guidelines) does not justify reaching outside the guidelines without knowledge of the possible negative effects and informing the patient of the risks and benefits of using a particular medication. It behooves a practitioner who does this to closely follow the course of the patient with correct tests (in this case, repeat lipid profile, liver function tests, CK, electrolytes), to act on results, and to be very wary of the effect of other drugs proposed and utilized.

It is the finding of the Committee that Dr. Deep's treatment of patient Q demonstrates a serious lack of knowledge, skill and judgment and constitutes incompetence as well as care below the standard of practice.

Patient CC

Dr. Deep first saw patient CC in 1988. At the time, he was 30-years-old and had the following diagnoses: pharyngitis, duodenal ulcer, trauma right foot, "coronary diathesis" and costochondritis. He was seen intermittently until 1999 and then not again until September 2005. The following are his listed diagnoses in 2005: hypertension, obesity, "coronary diathesis" and remote duodenal ulcer.

Dr. Z stated he was not certain what "coronary diathesis" referred to, but thought Dr. Deep might have meant a concern about coronary artery disease.

Nuclear medicine stress testing in 1994 and 2005 demonstrated no indication of

myocardial ischemia.

In October 2005, lab testing indicated he had a fasting blood sugar of 7.4 (elevated). This finding was not acknowledged by Dr. Deep on the lab report or identified as a concern regarding diabetes in the medical record. It appeared, from the chart, that the blood sugar was not repeated soon after. A triglyceride level was reported as 2.78 mmol/L (elevated) and Dr. Deep increased this patient's Crestor dose. At the time, patient CC had an LDL-C at target at 1.9 mmol/L.

No note was made in the medical record that this patient may have metabolic syndrome. There is no record of a waist circumference measurement. There is no documentation to suggest that this patient was advised, in response to the elevated triglyceride, to avoid alcohol or lose weight. There was no record of referral to a dietician and no apparent link was made between the elevated triglyceride and the elevated blood sugar.

Dr. Z was concerned that Dr. Deep had not identified that this patient was likely diabetic, that his elevated triglycerides were likely related to the elevated blood sugars, and that he had not advised lifestyle modifications as a trial prior to pharmacotherapy.

Dr. Z also expressed concern that Dr. Deep failed to document that this patient had metabolic syndrome, which had many implications for his cardiovascular management. Dr. Deep failed to recognize that this patient needed to have further investigation to establish a diagnosis of diabetes and that the elevated triglyceride may have been related to the elevated blood sugar. He also failed to advise this patient to avoid alcohol and lose weight, and failed to refer him to a dietician for further lifestyle modification prior to increasing his lipid medication.

In response to the evidence presented, Dr. Deep referred to the "Recommendations for the management of dyslipidemia and the prevention of cardiovascular disease: Summary of the 2003 Update" published in the Canadian Medical Association Journal (2003). He stated that he wanted to use this article to indicate that he was aware of the criteria for

metabolic syndrome. He agreed that the patient had this diagnosis and had all of the criteria, with the exception that his LDL was high and he had impaired fasting blood glucose and not diabetes in his opinion.

Dr. Deep stated that he did not write a diagnosis of metabolic syndrome in the file because it would in no way alter his management and had no clinical therapeutic implications. As a cardiologist, he would aggressively treat all of this.

The Committee accepted the testimony of Dr. Z and finds that Dr. Deep demonstrates a serious lack of knowledge, skill and judgment and that his treatment of this patient as described above constitutes care below the standard of practice.

Conclusions regarding Lipid Metabolism and Treatment

Taking into consideration the testimony of Dr. Z and Dr. Deep, a review of the patients' medical records and additional written material provided by Dr. Deep, not limited to but including patient lab testing and investigations, the Committee concluded the following:

1. Investigation of hyperlipidemia

Dr. Deep failed to adequately and appropriately investigate hyperlipidemia. In many instances, he failed to perform baseline lipid levels prior to initiating pharmacotherapy and failed to assess cardiovascular risk factors. In doing so, he failed to establish a clear indication for pharmacotherapy in many of these patients.

2. Treatment

Dr. Deep failed to establish a clear treatment plan for hyperlipidemia and failed to discuss therapeutic goals and set appropriate target lipid levels. He failed in most instances to recommend a trial of diet and exercise as part of, or as an antecedent to, pharmacotherapy. He did not refer patients who may have benefited from a dietician's advice. In at least one instance, he failed to address potentially modifiable cardiovascular risk factors, such as smoking, as part of the treatment plan. He failed to discuss the risks of statin therapy and failed to make adjustments in medications over time as new

information became available regarding the management of hyperlipidemia.

3. Follow-up

Dr. Deep failed to perform comprehensive and regular monitoring of patients on lipid pharmacotherapy for potential drug-related toxicities. When lab testing was done, there were multiple instances where he failed to acknowledge and act on abnormal findings.

4. Documentation

Dr. Deep failed to provide clear documentation as to type and dose of prescribed medication, when and why changes to medications were made, side effect profiles and patient compliance.

5. Drug interactions

Dr. Deep failed to consider the potential for drug interactions when prescribing statins for some of the patients.

6. Co-morbid conditions

Dr. Deep failed to consider co-morbidity such as hypothyroidism and renal impairment prior to initiating lipid pharmacotherapy.

7. Consideration of guidelines

Dr. Deep did not consider the guidelines on the management of hyperlipidemia in terms of assessing cardiovascular risk, considering non-pharmacologic therapies or setting target lipid levels.

The Committee accepts the opinion of Dr. Z. It is the finding of the Committee that in Dr. Deep's care of patients there is a pervasive pattern of disregard for diagnosis, evaluation, treatment and follow-up that exposed patients to potential harm with no apparent benefit. There was over-treatment of hyperlipidemia when there was no clear indication to do so; because of this, financial costs were incurred and there was the potential for exposure to drug-related toxicities. There was under-treatment that exposed patients to an increased

risk of cardiovascular events. Full disclosure of the risks of lipid pharmacotherapy should have occurred, but did not, with all of the patients, as well as a frank discussion about why the guidelines of the day were not taken into consideration.

Dr. Deep displayed a serious lack of knowledge, skill and judgment and did not meet the standard of care expected of a reasonable cardiologist or internist in his care of patients.

GLUCOSE METABOLISM

Diabetes Mellitus is a significant disorder. Knowledge and proper management of this disease is central to the practice of internal medicine and its subspecialties, including cardiology. Diabetes is a serious condition that affects multiple body systems and has a myriad of implications and complications associated with it. Dr. Z testified concerning Dr. Deep's management of patients with blood sugar abnormalities. He testified that there are guidelines available to identify and deal with abnormalities in blood glucose. These guidelines have been available for years and are well known and accepted in the medical community. Dr. Z provided two articles that outlined the recommended steps in dealing with abnormal blood sugar levels: "Definition, Classification and Diagnosis of Diabetes and Other Dysglycemic Categories" (Clinical Practice Guidelines, 2003, Exhibit 11) and "Screening and Prevention" (Clinical Practice Guidelines, 2003, Exhibit 12). He added that these two exhibits would constitute clinical practice guidelines. Dr. Z stated that his opinion of Dr. Deep's practice are not solely based on these guidelines but also on the expertise of the endocrinologists he works with in his centre who deal with diabetes routinely.

There were two patients with diabetes documented by Dr. Deep: Patient B and Dr. X. Dr. Z identified nine patients as having abnormal blood glucose levels that required further evaluation: patient I, patient F, patient D, patient S, patient Q, patient AA, patient O, patient CC and patient N. In these patients, Dr. Deep measured either, or a combination of, random blood sugar, fasting blood sugars and on occasion the glycated hemoglobin (A1C) test. While Dr. Deep may have responded in some of these cases, he did not do so according to the guidelines. Examples cited by Dr. Z and noted by the Committee include:

1. Patient B. Dr. Z testified that diabetes is not listed as a problem in this patient's chart prior to October, 2000. There were abnormal results reported as early as May 26, 1999 but no treatment was initiated until June 2004. Published guidelines of 1998 indicated the proper way to follow-up on diabetes but these were not followed by Dr. Deep.

2. Dr. X. This patient stated he had diabetes when first seen by Dr. Deep. Dr. Deep testified that he had no reason to believe this because Dr. X was a generalist physician (other testimony of Dr. Deep indicated he was a retired pediatric nephrologist); however, Dr. Deep testified that in other patients he did believe their unsupported histories based on their "intelligence level." Dr. Z pointed out there was a blood sugar level of 7.1, outside the normal range, but there was no comment on this level or any follow-up. Dr. Deep indicated that he was aware of this level, but was of the belief that this, along with other measurements, indicated impaired fasting blood sugar, not diabetes. Dr. Deep also presented normal levels of HA1C as indicative of a non-diabetic state. Dr. Z noted that this test is utilized to follow patients on treatment and is a measure of glucose control over time, but is not used as a diagnostic test. No glucose tolerance test was done. Dr. Deep testified that, in his judgment, this was not necessary; it was also too difficult and time consuming for this patient (as well as other patients) to get this test.

Dr. Deep presented multiple normal results of glucose tests in the patients identified by Dr. Z as having suspicious results in need of more sensitive follow-up testing. He considers follow-up unnecessary for blood sugar levels below the level diagnostic for diabetes and blood sugar levels above normal and below the laboratory reported abnormal level can be ignored.

Dr. Z concluded that Dr. Deep's care was insufficient. The medical record contained no indication of appropriate management and follow-up of abnormal glucose metabolism markers. His management and approach to hyperglycemia was not according to published guidelines and there was no explanation in the charts for the deviation from these consensus documents. He used HA1C as a diagnostic test, where its use is to follow

results of treatment strategies. He did not do follow-up glucose levels with further diagnostic testing, such as the glucose tolerance test.

The Committee was concerned that there was no acknowledgment of the Canadian Diabetic guidelines and the need to document and treat early signs of glucose metabolism abnormalities appropriately. Dr. Deep appears to either not be aware of these guidelines, or to not believe in their validity versus his clinical judgment. As noted in the case of Dr. X, there appears to be a disconnect in this case around diabetes, between the known abnormalities and the varieties of responses to the diagnostic signals. The final defence of Dr. Deep as to his treatment choices appears to us to be “clinical judgment and experience” above diagnostic tests and guidelines. As in other clinical areas, he believes some patients’ information concerning their illnesses and not others, with no basis for this choice or with a basis that has no factual foundation.

While no patient can be demonstrated to have died from the lack of proper follow-up in the cases noted and the time frame observed, it is apparent that the possible optimal treatment of long term preventable complications has been missed to the detriment of patients. The Committee accepts the evidence of Dr. Z and concludes that Dr. Deep did not meet the standards of practice in the diagnosis and treatment of glucose abnormalities. He did not pay attention to abnormal results, misused and misinterpreted the utility of available tests, did not appreciate the relationship of dysglycemia to concurrent heart disease, and demonstrated a lack of knowledge and judgment in this area that constitutes falling below the standards of practice of the profession and meets the criteria defining incompetence.

USE OF VIOXX AND NON-STEROIDAL ANTI-INFLAMMATORY AGENTS

These agents, especially Vioxx, were widely used in the treatment of arthritis and other pain syndromes. In April 2002, Health Canada issued warnings (Exhibit 29) of increased risk of heart attacks in patients with pre-existing cardiac abnormalities. Further evidence was introduced (Exhibit 47) that by 2002 it was widely known that these agents were not to be used as first line therapy, especially in patients with pre-existing cardiac disease.

Dr. Z testified that Dr. Deep used this agent after the warnings in nine patients: patient F, patient K, patient O, patient H, Dr. X, patient I, patient M, patient R and patient N. According to the charts, Dr. Deep believed all of these patients to have intrinsic heart disease. Dr. Z noted that there is no evidence of discussion with the patients concerning the risks attendant in the choice of this medication. There was also lack of monitoring of this medication's potential interaction with other medications, including renal effects and hyperkalemia.

Dr. Deep was unable to recall what he told patients about this drug, but stated that he did not need to chart each of these discussions. He stated the risk was low at the time he prescribed this medication. He testified that none of his patients had any ill effects and he did not feel obligated to discuss the risks with his patients, as he really did not believe that patients were competent to make decisions about their own treatment. Dr. Deep also testified that the manufacturers of Vioxx had yet to lose a case in court concerning adverse effects of Vioxx.

While not in the same category as Vioxx, Dr. Z noted misuse of other anti-inflammatory agents. With patient S, Dr. Deep discussed the advisability of using ASA (aspirin) at levels of up to 650 mg daily. Dr. Z stated there was absolutely no evidence that doses larger than 75-325 had any benefit on prevention of adverse cardiovascular events, and ASA has its own possible side effects. Dr. Deep stated that there was evidence that coated low dose ASA was useless and larger doses were reasonable. He did not support this with scientifically valid evidence.

Based on his lack of notation and memory, and his assertion that patients were not competent to make therapeutic decisions, we find that Dr. Deep did not inform patients of the risks and dangers in using this medication, thus placing the patients at risk. This constitutes a failure to meet the professional standards of practice expected of a cardiologist.

THE PATIENTS

Patient A

The patient first saw Dr. Deep in June, 1997 at age 61. He had bypass surgery in May, 1997. There is no indication as to how he was referred. He had a 56-year history of diabetes. There were multiple diagnoses, including a note of “borderline (increased) cholesterol.” While the reasons are not clear to Dr. Z, Dr. Deep added an anti-lipid medication and Quinapril (an ACE inhibitor) to patient A’s regimen. Dr. V, a nephrologist at a Toronto hospital, was following him because of diabetic nephropathy.

Dr. Z’s concerns were:

1. ECG’s contained in the patient’s charts were not 12 lead ECG’s, were lacking lead identification, had no indication of calibration or paper speed, were not labeled with the patient’s name, had significant baseline wander and electrical interference. They were not of diagnostic quality. In addition, on the vast majority of visits, these were done without being medically necessary. (Dr. Z noted that this was true for almost all of the ECG’s he looked at in all of the patient records. Quality of the ECG’s seemed to deteriorate over time.)
2. Frequency of visits for this patient (monthly for eight years) without observable medical manipulation of his treatment. He was quite stable, attended cardiac rehabilitation, and saw a nephrologist every six months.
3. Dr. Deep prescribed Pravachol, a lipid-lowering medication, for this patient with no baseline assessment of lipid levels. There was no trial of diet or exercise suggested to this patient prior to initiating medication. This approach did not follow the guidelines of the time and would not have been standard practice. The guidelines recommended a trial of diet and exercise for six months. Cholesterol levels should have been repeated in two months time to allow for the acute effects of recent cardiac bypass surgery. No doubt, he would have needed therapy for hyperlipidemia, but he was not properly assessed, and not monitored to the level recommended at the time.

4. Propafenone, an anti-arrhythmic drug, was prescribed in January, 1998. He had asymptomatic extra heartbeats and structural heart disease (previous MI and CABG). A note specifically states there were no symptoms of chest pain or palpitations. On a physical, “Numerous ectopic beats plus occasional bigeminal rhythm” was noted. Dr. Deep stopped Propafenone in April, 1998, and reinstituted it a year later. It is not entirely clear from the chart how long he took this medication. In April, 2001, there were no specific cardiac complaints but use of the drug resumed. By November, the patient had stopped the medications, and the March 2002 chart states “Use propafenone only if VEDs symptomatic. Reassure.” Propafenone was again started in April 2002 because of “VEDs q3rd beat resume Rythmol(Propafenone) BID.”

Dr. Z stated that this was a very serious concern. Asymptomatic patients should never be treated. There was no indication for Propafenone use in this patient and, in fact, there is incontrovertible evidence that anyone with structural heart disease receiving this type of drug is exposed to a risk of sudden death. Furthermore, Dr. Deep started the medication based on a clinical exam and not on diagnostic testing (no Holter monitor testing was used). Propafenone was started in the outpatient setting. It remains unclear if this is a safe thing to do. In addition, given patient A’s renal insufficiency there was an increased potential for drug toxicity.

Propafenone was contraindicated in this patient. Dr. Z stated that Dr. Deep displayed a very serious lack of knowledge and judgment in prescribing Propafenone, and that there was concern for the patient’s safety.

5. In prescribing other medications, such as Lozide, which have a pro-arrhythmic potential, Dr. Deep displayed a lack of knowledge and judgment. This exposed the patient to increased risk of pro-arrhythmia and sudden death. There was similar concern with Dr. Deep’s use of antibiotics such as Ciprofloxacin and Levoflox, which are associated with QT prolongation in and of themselves and have the potential to exacerbate the pro-arrhythmic effects of Propafenone.

6. Dr. Deep failed to communicate with this patient's nephrologist regarding the use of Propafenone and Lozide - a combination that could have a significant impact on renal function. He failed to communicate with the patient's urologist regarding medications patient A was taking, and the patient ended up taking two alpha-blocking agents concurrently (Cardura prescribed by Dr. Deep and Flomax prescribed by the urologist).
7. As a cardiologist, Dr. Deep should have known how to calculate mean arterial BP accurately.
8. Dr. Deep failed to adequately monitor patient A's renal function and electrolytes after he was started on an ACE inhibitor, and on an ongoing basis.
9. Dr. Deep switched this patient back and forth between Lozide and Chlorthalidone, or had him taking both medications with no apparent rational.
10. Dr. Deep undertook approximately 80 minutes of psychotherapy prior to sending the patient to the emergency department for an injury to his tibia he deemed serious enough to require emergent treatment. There was no notation regarding the issues discussed at this psychotherapy session.
11. Dr. Deep failed to recognize severe renal failure in this patient. Given the medications patient A had been prescribed, there was an increased potential for toxicity with his reduced renal function.

Dr. Deep testified as follows. For this patient to be seen yearly or by a family doctor would have been "a death sentence." He performed monthly ECG's as diabetics "frequently" have silent ischemia, and he needed frequent adjustment of his medications for hypertension. A family physician would not be able to adequately supervise and treat him. He started statins immediately without baseline or non-drug maneuvers knowing he had type 1 diabetes, a bypass graft and severe hypertension. He was a high-risk patient

and LDL-cholesterol should be treated down to 2.0 or less, rather than what the guidelines of the time recommended. He used Propafenone to treat severe symptomatic ventricular extra beats and bigeminal and trigeminal rhythm on occasion. He considered that the benefits outweighed the risks. He testified that he did not get Holter monitor tests, as it was inconvenient for patients to get this examination in Toronto.

He stated that Dr. Z is wrong about use of antibiotics that prolong the QT interval, and he used this antibiotic (Ciprofloxacin) to treat a foot ulcer. He claims it was the only antibiotic the infection was sensitive to. There is no record of antibiotic sensitivities in the chart. Dr. Deep stated there was no need to follow the nephrologist's recommendation concerning medications, and the patient was intelligent enough to keep the nephrologist informed without the necessity of physician communication. The patient was regulating his own insulin and if he followed Dr. Deep's suggestion to "lighten up" on his insulin dose, he would discuss this with Dr. ZZ (family physician) at the next visit without necessity of Dr. Deep's input.

The Committee found Dr. Z's opinion to be persuasive. We agree that even if there was a reason to use Propafenone in this case, there was no proper diagnosis, follow-up or appreciation of the dangers involved. We reject Dr. Deep's explanations that the lack of a Holter monitor was due to difficulty in procuring this test in Toronto as being specious and untrue.

The pattern of care noted in this case leads the Committee to conclude that Dr. Deep did not meet the standards of the profession and shows incompetence in his management of this patient.

Patient B

Dr. Deep first saw this 65-year-old female in December, 1997. She had apparently been on Lipitor and was not taking her medication. At the initial visit she was prescribed Lipitor 20 mg daily, and Sectral (a beta-blocker agent) 100 mg (1/2 to 1 pill daily). Laboratory testing was later ordered in January of 1998.

Dr. Z testified to the following concerns:

1. No clear indication for the use of Sectral was identified in this patient's chart.
2. There were an excessive number of visits (four volumes of charts) for hyperlipidemia. Dr. Z could find no rationale for this.
3. The frequency of office-based diagnostic testing (ECG's, apex cardiograms, phono cardiograms, STI's) with no indication, as there were no ongoing cardiac problems.
4. Dr. Deep prescribed a statin at the first visit prior to having lipid levels done and without an adequate trial of lifestyle changes as per the guidelines of the times. No cardiovascular risk assessment based on the standards of the time was done on this or any of the patients to determine the need for medication to lower lipids levels.
5. Failure to follow through on tests suggested or mentioned in Dr. Deep's notes. This may suggest that Dr. Deep's office was lacking a system to make sure tests were ordered and done.
6. There was no evidence of a problem list in this chart at the expected and appropriate times. The first list noted was in June, 2004. In addition to hyperlipidemia, it also mentions M.R. (Mitral Regurgitation), anxiety, osteoporosis and borderline thyroid (arrow down).
7. Diabetes was not listed as a problem in this patient's chart prior to October, 2000. The medical record contained no indication of appropriate management and follow-up of diabetes. Dr. Deep's care was insufficient. There were abnormal results reported as early as May, 1999 but no treatment was initiated until June 2004. There was no follow-up to abnormal results and no additional testing. Guidelines published in 1998 indicated the proper way to follow-up on abnormal glucose tests and to treat the illness. This was

never done. Dr. Z saw the fact that Dr. Deep did not undertake ASA therapy as a primary preventative measure for this patient, as an additional serious omission.

8. Dr. Deep diagnosed mitral regurgitation but there was no follow-up to establish the diagnosis, determine the etiology or develop a management plan. No echocardiogram was done. Dr. Z stated that he would expect trainees to do a more thorough evaluation than Dr. Deep did. He added that Dr. Deep's diagnosis was based on a cursory and incomplete examination. He did not do an adequate baseline assessment or arrange appropriate follow-up. This showed a very serious lack of knowledge, skill and judgment. Dr. Z testified that the patient did not have mitral regurgitation, yet she was given antibiotic prophylaxis based on this diagnosis, which was unnecessary.

Dr. Deep submitted the results of an echocardiogram showing a normal mitral valve in order to demonstrate the lack of need for further intervention or testing. Dr. Z said that this should have been done in February 1999, soon after the diagnosis was made. The Committee noted that the test was ordered and performed in January, 2006, long after the dates of the chart review.

9. Dr. Deep testified that he acted as her family physician in this situation and that he has the knowledge to do vaginal exams. He treated the condition he found to make it easier for the patient. There was, however, no evidence of lab testing concerning the vaginal discharge. Furthermore, there is no documentation to support him acting as her family physician or performing the expected preventative care for a woman of her age.

10. Dr. Deep failed to respond, or demonstrated a delayed response, to abnormal thyroid tests. Tests were elevated starting in May 1998. It was not clear from the record if she was being treated and, if so, why the TSH remained elevated. Not only are thyroid abnormalities problematic in and of themselves, but they can have an impact on lipid disorders and diabetes.

11. Dr. Deep's charts comment on paranoid ideation and that on one occasion she was

delusional. The chart established no specific diagnosis, yet psychotherapy was performed. There was no neurological examination or referral to a psychiatrist or psychogeriatrician. In testimony, Dr. Deep said she felt discriminated against as a French Canadian, and redefined his charting on paranoid ideation as meaning an inappropriate belief in discrimination. He testified that she improved on talk therapy. As in other patients, there is no charting of psychotherapy.

Dr. Deep submitted evidence dating from 1994 and 1997 not in the charts given to Dr. Z. He stated that the patient was not compliant with following his recommendations for hypoglycemic agents and deferred treatment for mild hypothyroidism replacement therapy for two years. There was no record to support this contention.

The Committee, in evaluating the treatment of this patient, accepts Dr. Z's opinion that her care was below the standard expected of the profession and demonstrates a serious lack of judgment and disregard for the welfare of the patient. Dr. Deep's explanations for his performance lacked credibility and were not believed by the Committee.

Patient BB

Dr. Deep first saw this 58-year-old man in June, 2005. The following list of diagnoses was present in the chart: "LSDD", osteoarthritis, remote pelvic fracture, remote renal calculi and obesity.

Dr. Z had the following concerns:

1. The frequency of visits – five visits over six months that, given no identified cardiac problem requiring ongoing care - seems excessive.
2. Dr. Deep prescribed Lipitor to a patient with no cardiac complaints, within one month of a trial of diet and, in doing so, failed to meet the guidelines of the time. Counseling regarding lifestyle was recorded in the chart on the first visit, but no details of what was discussed could be found. The patient was already at target levels when drug therapy

began. There was no assessment of possible secondary causes of hyperlipidemia and assessment of cardiac risk was below the standard expected.

3. The ECG reported as normal by Dr. Deep had significant electrical interference, was not a 12 lead ECG and was poor quality and non-diagnostic.

Dr. Deep testified that the patient was overweight and failed at the trial diet because he was an executive and dined out frequently. While there is no notation of dietary advice, Dr. Deep testified he gives all patients the DASH diet for weight loss. No notation of an exercise trial was noted.

The Committee is of the opinion that Dr. Z's concerns are substantiated in the evidence. While this patient's file does not present the same level of concern noted in many of the other reviewed charts, we find that Dr. Deep did not meet standards of professional practice. Dr. Deep testified that he provided lipid-lowering medication for this patient immediately because of his work life and inability to exercise or follow a diet regimen, but these statements were not supported by the records and the Committee doubted their veracity.

Patient P

Dr. Deep first saw this patient at age 79.

Dr. Z, in his testimony, expressed the following concerns:

1. The patient's cardiovascular examination was below the level expected of a cardiologist. Blood pressure should have been measured in both arms, the description of the murmur found is lacking in detail and did not help to differentiate the cause of the murmur, there was no description of heart sounds, and no maneuvers to change the patient's position and help diagnose the murmur were documented. Her blood pressure was registered as 130/60.

2. It is unclear how the diagnosis of coronary artery disease was made. This is not a trivial diagnosis and, once made, appropriate management needs to be initiated (such as daily ASA use, modification of risk factors, et cetera). Dr. Deep's explained that he made the diagnosis based on plaque seen in her proximal aorta, which is sufficient for a diagnosis of coronary artery disease.

3. Medication lists were not present in more than 3% of visits. The first recording of medications was in August 1998, but with no indication of who ordered and who tracked each medication. This was important as she was being followed at a regional centre for refractory anemia.

4. The frequency of visits. There were more than 100 visits in eight years. While she may have been receiving B12 injections, her bone marrow does not indicate a deficiency. She was given iron, folate and B12 by Dr. Deep, yet there was no clear indication to do so, nor any communication with, her hematologist.

5. Dr. Deep discontinued this patient's Zoloft with no evidence of cardiac reasons to do so, with no communication with the prescribing physician and with no evidence of discussion between Dr. Deep and the patient about why it should be discontinued.

6. Dr. Deep failed to recognize moderate renal failure in this patient. A clinician might not do anything about this, but needs to be aware, monitor renal function and watch closely the types of drugs and doses being used. Dr. Deep prescribed NSAIDS, ACE inhibitors and Digoxin and ARB's without regard for her renal failure.

7. Dr. Deep prescribed Digoxin for this patient with no indication to do so. Furthermore, the dose he prescribed was too high. His rationale (rapid heart rate, CAD and a loud aortic murmur) for doing so makes no sense. He did not even record a heart rate at the visit when he prescribed the Digoxin. Dr. Z questioned the development of Digoxin toxicity in this patient. Concern of toxicity should have arisen when she had some diarrhea. He stated that it would have been appropriate to stop the drug and do a

blood level.

8. Dr. Deep failed to adequately monitor the renal function and potassium in this and many other patients taking ACEI's and ARB's. This is a serious concern, particularly in patients with impaired renal function.

9. Dr. Deep failed to recognize hyperkalemia, a potentially lethal problem. He failed to respond to elevations in potassium when blood work showed these abnormalities.

10. Dr. Deep prescribed the NSAID, Arthrotec, for this patient. NSAIDS are known to increase the risk of renal failure and hyperkalemia, especially in combination with other drugs such as ACE inhibitors or ARB's. The patient's potassium went up following this prescription, and Dr. Deep did not respond to this. Alternative treatments should have been considered and the risks discussed with the patient. If an alternative was not suitable, blood work should have been done within one week.

11. Patient P was on a high dose of L-troxine given her age and size. Doses are usually adjusted based on laboratory work. This did not seem to be the case.

12. Dr. Deep diagnosed this patient with mild to moderate aortic stenosis. Dr. Z was concerned that Dr. Deep had described her murmur as grade 5 out of 6. This would suggest critical stenosis, requiring confirmatory testing which was not done.

Furthermore, the grading of the murmur changed over time - it went from 3/6 to 5/6 over a seven month period which, while unlikely, would require investigation which was not initiated.

Dr. Deep testified that her blood creatinine was normal, and this was not changed by the results of her eGFR (glomerular filtration rate). He denied there was any indication of Digoxin toxicity, and testified that the diarrhea was a result of another cause and resolved spontaneously. He does not believe that the diarrhea alone required any suspicion of Digoxin toxicity. He stated that he prescribed Zoloft for her depression and in summation

added that he “presumes that I stopped it.”

The Committee accepts the evidence of Dr. Z that, in each of these areas, there is a pervasive lack of awareness of clinical factors that substantiate the allegation that Dr. Deep did not meet the standard of practice of the profession and demonstrates incompetence.

Patient Q

The patient first saw Dr. Deep in 2002 as a 71-year-old. He had a family doctor, Dr. YY.

Dr. Z testified to the following concerns:

1. The patient was being followed by a cardiologist, Dr. XX, and by his family physician. He was in a cardiac rehabilitation program following a myocardial infarction (MI) when Dr. Deep began to see him. He did not need a second cardiologist.
2. Dr. Deep indicated in his records that he planned to repeat the patient’s angiogram and cardiolute study when these had already been done in hospital. Dr. Z stated that it did not make sense to expose the patient to the risks of these tests given the information was already there.
3. Dr. Deep recommended the use of low-dose Coumadin with the patient’s ASA post MI. There is no scientific evidence to support the addition of low-dose Coumadin. The addition of subtherapeutic Coumadin adds no benefit and increases the risk of bleeding. Furthermore, Dr. Deep noted in his chart that the patient was not taking ASA. This exposed the patient to an increased risk of vascular accident, MI and death in a very vulnerable period following an MI. Dr. Z stated that Dr. Deep should have known this and described this as “bread and butter” cardiology. There was no discussion with Dr. XX, the cardiologist.
4. There were elevations of fasting blood glucose levels. It was important to establish a

diagnosis of diabetes and further investigations should have been done. This is particularly important in a patient who has had a heart attack, and where a diagnosis of diabetes would add a significant additional risk for future heart events.

5. Dr. Deep did not appear to know that marginal increases in creatinine might still indicate mild to moderate renal impairment. This should have been a flag for future consideration.

6. Patient Q's lipids were not aggressively treated and he was likely at the highest risk for future heart events.

Dr. Deep testified that Dr. XX did not follow the patient and that he received necessary information from a secretary at the hospital by phone. There was no reason to make contact with the hospital cardiologist. He stated that, in his opinion, low-level anticoagulation along with ASA had a beneficial result.

Dr. Z testified that there is no evidence to support the low-level anticoagulation along with the ASA. As well, Dr. Deep continued to use creatinine alone as a measurement of renal function, denying any utility to the accepted eGFR.

The Committee accepts the evidence given by Dr. Z. Dr. Deep's explanations for lack of communication with the hospital, his refusal to accept the validity of the eGFR measurement when it differs from his diagnosis and his treatment of patient Q post MI do not, in any way, explain the deficiencies noted by Dr. Z. These deficiencies exposed the patient to harm and substantiated the allegations of practicing below the standard of practice of the profession and incompetence.

Patient J

Dr. Deep first saw this patient in November 1997. There were no specific complaints noted in the chart. Diagnoses were listed as "mitral regurgitation, chronic anxiety and GERD."

Dr. Z's concerns were:

1. Patient J was labeled with a diagnosis of mitral regurgitation, which he did not have. (Echocardiogram, Exhibit 2, Volume 6, page 229). Dr. Z stated that a basic premise in medical care is not to label a patient with a disease they do not have as it will impact their life in multiple ways. He stated that making this diagnosis was basic cardiology. While no cardiac disease was confirmed, the patient continued to visit a cardiologist and have ECGs and systolic time intervals without cause.
2. While he was asymptomatic from a cardiac standpoint, his cholesterol was treated based on blood levels alone and he was prescribed Pravachol. No risk assessment was done and there was no indication of target levels being decided on. This patient was actually in a low risk category and was already at a level lower than the guidelines recommended. Dr. Z was of the opinion that, relying on this patient's family history alone to institute treatment and considering the risks of medication, treatment was not indicated.

Dr. Deep testified that because he had a family history of coronary artery disease, he should receive treatment, and that he relied on his judgment and common sense to institute treatment.

In his evidence, Dr. Deep stated that since the echocardiogram showed a "trace of Mitral Regurgitation," he therefore had this disorder. Dr. Z noted that a report of trace regurgitation is considered physiologic or normal and not a diagnosis of Mitral regurgitation. Such a finding cannot be used to justify the diagnosis. He also pointed out that on other occasions, Dr. Deep used trace reports to argue that someone does not have mitral stenosis. Dr. Deep stated that the clinically-heard murmur was clear to him, and that echocardiology does not accurately assess the severity of mitral murmurs. He agreed in cross-examination that lipid guidelines may suggest non-treatment, but future guidelines may be different with gene determination testing and, in any case, he used his

clinical judgment in beginning treatment. He also stated that Dr. Z made false accusations concerning possible liver toxicity as liver function tests were on file.

The Committee was concerned with the unsupported diagnosis of mitral regurgitation and the negative defence that Dr. Deep could hear the abnormality even if it was not seen on an echocardiogram, because they cannot be relied on in any case. Combined with his evidence that the echocardiogram supported his diagnosis, we find the logic unsupportable and self-serving. The treatment of hyperlipidemia is not an act solely of an individual physician's judgment and cannot be based on suppositions as to the way the future will justify his choices.

We conclude on the evidence before us that the allegations of failure to maintain standards of practice and incompetence are proven in respect to this patient.

Dr. X

This 73-year-old male became a patient of Dr. Deep in October, 2000. He was diagnosed with hypertension with microalbuminuria, NIDDM (non-insulin dependent diabetes melitus), psoriasis, diverticulosis, gastritis, obesity and anxiety.

Dr. Z testified to the following concerns:

1. Dr. Deep did not note low platelet counts on two occasions as a diagnosis or issue.
2. At the initial visit, Dr. Deep indicated he was thinking about increasing the dose of Lipitor. No lipid levels were done. Combinations of Lipidil and Lipitor were used, and it was unclear as to the indication.
3. Dr. Deep failed to appreciate that Dr. X had renal impairment and the severity of his renal failure. He should have been referred to a nephrologist. This was not listed as a medical problem in his chart.

4. A combination of ACE Inhibitors and ARB was used, increasing the risk of hyperkalemia. Lack of monitoring was a concern, especially with diabetes.
5. Vioxx was prescribed for Dr. X. Vioxx with ASA increases the risk of GI bleed. Vioxx may actually counteract the benefit of ASA, and it can negate the effect of blood pressure medications and potentiate the effect of ACEI's and ARB's on potassium and creatinine. It took Dr. Deep six months to discontinue the Vioxx after there was a marked change in Dr. X's renal function.
6. Dr. Deep failed to recognize that Dr. X was hyperkalemic. Frequent monitoring should have taken place. There was the potential for a fatal complication because Dr. Deep did not re-measure his potassium and had no idea in which direction the potassium was going.
7. Dr. Deep ignored the Canadian guidelines on the management of diabetes. He stated that although Dr. X informed him of his diabetic state, he had no reason to believe him, as he was just a "General Practitioner." In fact, he was a pediatric nephrologist. Dr. Deep testified that he measured glycated hemoglobin and this was normal. Dr. Z pointed out that this test does not mean that someone is cured of his diabetes, but is used as a measure of control in someone being treated for diabetes.
8. Dr. Deep suggested that a nephrologist, Dr. WW, was following the patient but there was no evidence of this in the file and no correspondence from Dr. Deep to Dr. WW.
9. Dr. Deep's assessments were not complete and lacked physical examinations that were an integral component of the assessment type charged to OHIP. Dr. Deep stated this was due to the patient being in a rush and only wanting his blood pressure done, yet the Committee notes there was time for psychotherapy at each visit.
10. There were no psychotherapy notes. Dr. Deep testified that he decided not to elaborate on the therapy for confidentiality reasons.

Dr. Deep believed that based on his HbA1C, the patient is not a diabetic but has impaired fasting glucose. As well, he did not have hyperkalemia, as the dangerous level is greater than six. Dr. Z reiterated his diagnosis based not on a single standard but on the standard of each laboratory; based on levels compared to each laboratory normal, he did have hyperkalemia.

The Committee accepts the testimony of Dr. Z and finds that Dr. Deep failed to maintain the standard of practice of the profession and is incompetent.

Patient F

Dr. Deep first saw this 65-year-old woman in August 2000. She had the following list of diagnoses: hypertension, sleep apnea, Pott's disease, rheumatic fever, aortic stenosis, Sjogren's syndrome, obesity and coronary artery disease with "mild MI."

Dr. Z listed the following areas of concern in his testimony:

1. Dr. Deep did not send a consultation note to the referring physician, Dr. VV.
2. It is uncertain how the diagnosis of coronary artery disease (CAD) was made.
3. Dr. Deep prescribed Lipitor, thus initiating drug therapy without doing prior blood tests and without first establishing the diagnosis.
4. Vioxx was prescribed to a patient Dr. Deep believed had CAD despite the April 2002 Health Canada warning about increased cardiovascular risks with this drug. This risk should have been discussed with the patient and possible alternatives explored.
5. Dr. Deep displayed a lack of knowledge and judgment regarding her blood sugar management and did not adhere to the guidelines of the day.

6. There was absolutely no indication for the use of Digoxin in this patient.
7. Dr. Deep failed to recognize that, based on valve size (from her echocardiogram), her aortic stenosis was moderate and not mild. There was no evidence that Dr. Deep discussed SBE (subacute bacterial endocarditis) prophylaxis with her. Dr. Z stated that this is basic cardiology. SBE is a very serious and potentially life-threatening condition. This was not discussed with the patient until two years later.
8. Follow-up of her aortic stenosis was inadequate. Echocardiograms should have been done every two to three years.
9. Dr. Deep seemed to lack knowledge of the triggers indicating when to perform surgery in aortic stenosis.
10. There was no cardiovascular system examination on an occasion when this patient had a dental abscess and root canal. One would want to make sure there were no apparent changes in cardiac status given her risk of SBE with aortic stenosis (a valvular bacterial infection possible from a dental abscess).
11. Dr. Deep continued to prescribe NSAIDS for this patient. She was on blood pressure lowering medications and NSAIDS can negate the effects of these medications.
12. Dr. Deep's clinical assessment of her aortic stenosis was inadequate. Notations concerning her cardiac murmur suggested that it appeared and disappeared. It was unclear why this should be the case. If it did disappear, this would be cause for concern and may indicate change in heart strength and warranted further investigation.

Dr. Deep stated that her husband, whom he was also treating, was the referral source and was able to communicate any changes in her condition to other physicians, including other specialists involved in her care. He stated that he believes that her husband likely informed him of her history of hyperlipidemia before he began treatment. Yet Dr. Deep,

at another juncture, described her husband as unreliable to give his own history of diabetes despite the fact he was a physician.

The Committee noted that, while Dr. Deep states that her husband was the referral source, there was a letter from Dr. VV in her chart. The Committee is convinced by Dr. Z's description of her cardiac status and the lack of proper diagnosis, follow-up and treatment. The use of Vioxx was inadvisable and, at the least, should have been done with full disclosure of risks and benefits to the patient, as well as discussion of alternatives.

The Committee concludes that, with respect to the care of this patient, the allegations of failure to maintain the standard of practice of the profession and incompetence are proven.

Patient CC

Dr. Deep first saw this patient in 1988 at the age of 30 for chest pains. His cardiac related diagnosis was "Cor (sic coronary) diathesis." In 1998, there was a further diagnosis of "cor art diathesis with previous angina pectoris."

Dr. Z's concerns were:

1. Dr. Deep did not recognize that this patient was a diabetic and did not treat him as such. He increased his dose of Crestor to deal with the elevation of triglycerides, but these were most likely elevated because of the abnormal blood sugars. The best course would have been to deal with the blood sugar levels. He failed to recognize the association between elevated blood sugars and elevated triglycerides.
2. Dr. Deep displayed a lack of knowledge in suggesting that a normal HbA1C precludes the diagnosis of diabetes. This test is used as a measure of control and is not a sufficient test for ruling out diabetes. This is basic knowledge for every physician and not just cardiologists.

3. Nuclear medicine tests in 1994 and 1998 showed absence of myocardial ischemia, belying the diagnosis of coronary illness.

The Committee considers Dr. Z's descriptions and conclusions to be valid markers of the treatment of this patient and finds the deficiencies support the allegations of failure to maintain the standard of practice of the profession and incompetence.

Patient O

A self-referred patient first seen in 1985, he had a history of prior myocardial infarcts, left ventricular systolic dysfunction, and multi-vessel coronary bypass surgery. He also had hypertension, hyperlipidemia and was obese.

Dr. Z, in his testimony, expressed the following concerns:

1. Dr. Deep prescribed Propafenone despite the fact the patient had structural cardiac disease and impaired heart function. Dr. Deep treated him for extra ventricular beats and exposed this patient to an increased risk of death. Dr. Z stated that the information regarding Propafenone and its risks is basic knowledge known since 1988. This patient had a conduction abnormality (right BBB) contraindicating the use of this drug. The fact that Dr. Deep prescribed it for him is a very serious concern. Furthermore, the fact that Dr. Deep discontinued this patient's Amiodarone in favour of Propafenone suggested a disregard or lack of knowledge regarding the relative risks of these two drugs. Dr. Z commented that while Amiodarone has many side effects and requires vigilant monitoring, it reduces arrhythmic death and has an overall reduction on mortality of 13%.
2. Dr. Deep failed to recognize that this patient's renal impairment was moderate and not mild. In addition, he failed to adjust the dose of medications accordingly.
3. The patient developed hyperkalemia, a serious and potentially life-threatening problem and Dr. Deep failed to act, in a timely fashion, on elevations of this patient's potassium. Dr. Deep failed to monitor for the potentially adverse effects of drugs this

patient was taking. He appeared to lack an awareness of additive adverse effects of these drugs.

4. It is unclear why Dr. Deep changed the patient's anti-hypertensive medications from an ACE inhibitor to the ARB, Avapro. Again, the patient needed monitoring for either of these drugs.

5. Dr. Deep prescribed Aldactone for this patient. Studies have shown that this drug lowered mortality in patients with heart impairment but increased their risk of hyperkalemia. Subsequent publications showed an increased risk of hospitalization and death once this drug was used more frequently. The concern here is not Dr. Deep's use of the drug, but the lack of close and careful monitoring.

6. It was not clear why Digoxin was added to this patient's regimen. He was not symptomatic from a cardiac standpoint. Dr. Deep should have chosen a more appropriate dose of Digoxin and should have been aware of the patient's renal function and monitored it if he felt this medication was indicated.

7. Patient O developed Digoxin toxicity. Dr. Deep stated that he left a message on patient O's voicemail, but the dose was not decreased until this patient saw another cardiologist. Dr. Z stated that it is likely that this patient had Digoxin toxicity for many months. This represents a failure to monitor and a failure to respond to abnormal lab results.

8. As with other patients, there is a concern regarding this patient's elevated blood glucose levels. Dr. Deep failed to delineate the problem.

9. Dr. Deep prescribed Vioxx for this patient without regard for the potential cardiovascular risks and without discussing these risks with the patient.

10. Dr. Deep discontinued the patient's ASA and Plavix post stent placement and put him

on Coumadin. In doing so, he failed to provide this patient with what was at that time, and is currently, accepted as the best therapy post stent. He exposed this patient to an increased risk of bleeding and possible clot formation.

Dr. Deep testified that the elevation of potassium was “slight” and that he left a message for him concerning this. In relation to the Warfaren utilization, he testified that he used subtherapeutic levels deliberately and patients were instructed to use ASA simultaneously. He stated there was literature support for this, but the papers provided did not support this contention.

Dr. Deep defended his use of Vioxx as follows: during the patient’s admission, the hospital would not have placed a cardiac stent had they felt his congestive heart failure was related to his Vioxx use.

The Committee accepts Dr. Z’s evidence of patient O’s care and its deficiencies, and finds that it placed patient O’s in life threatening danger and was below the expected standard of practice of the profession, as well as incompetent in that it showed a serious lack of knowledge and judgment on the part of Dr. Deep.

Patient D

This 70-year-old woman’s first visit was in September , 2000. She initially presented with a history of a stroke in August, 2000. Dr. Y possibly referred her, although this is not clear in the chart. Dr. UU at a Toronto hospital was following her at a stroke clinic. Her listed diagnoses were: Recent CVA, PMV (Prolapse Mitral valve), hyperlipidemia, anxiety and HH (the latter initials were not clarified).

Dr. Z’s testified about the following concerns:

1. ECG’s in this patient’s chart were incomplete and were of very poor quality.
2. Dr. Deep prescribed Pravachol, a statin, for her at a time when it was not known to

prevent recurrent strokes. There was no discussion of appropriate target levels and the goal of treatment was not outlined in the chart.

3. Dr. Deep increased her dose of ASA. She was on ASA and Plavix. There is no evidence that increasing the dose beyond 325 mg provides any benefit, but it does increase the risk of bleeding.
4. Dr. Deep prescribed Quinipril, which did not and does not have the same proven benefit as Ramipril. Dr. Z's concern was not choice of drug but the failure to monitor because of the side effect profile of this drug.
5. Dr. Deep failed to fully and completely investigate and delineate the etiology of this patient's chest pain.
6. Dr. Deep did not send a consult note to the referring physician (while there was question of whether or not there was a referring physician, it was noted that the visit was billed to OHIP as a Consult-Internal medicine). In addition, he did not communicate his concerns regarding patient D's cardiac status to Dr. Y, a gynecologist she was referred to for surgery.
7. Medications were again prescribed for coronary artery disease without properly establishing the diagnosis.
8. There were concerns similar to other patients regarding her elevated blood glucose levels. Dr. Deep failed to further delineate this problem.
9. Dr. Deep stopped the patient's Plavix, (an anti-clotting agent) prescribed by Dr. UU at the stroke clinic, because of easy bruising. There was no communication with the clinic or Dr. UU concerning this, and Dr. Deep did not seek advice as to the recommended course of action.

Dr. Deep testified that patient D was already on statins when she came to his office the first time and it was logical to continue the medications before getting laboratory tests. He also testified that the patient could not get an early appointment to the stroke clinic. As an internist, he could use his judgment to stop the medication and the patient was intelligent enough to inform the clinic on her next visit.

The Committee accepts that the original prescription for statin medication was reasonable, but is concerned over the choice of medication and the lack of a plan to delineate the proper dosage and goals of treatment.

We accept the opinion of Dr. Z concerning the deficiencies in Dr. Deep's care of the patient described above and find that they constitute proof of the allegations that he failed to maintain the standard of practice of the profession and that he is incompetent.

Patient G

This 59-year-old man came to Dr. Deep in June 2005. There was no specific medical complaint or indication of how he was referred. The diagnoses in the patient's chart included Coronary Diathesis (not a term usually used by cardiologists), borderline hypertension, systolic ejection murmur (aortic stenosis, mitral regurgitation?) and alternating right bundle branch block.

Dr. Z testified about the following concerns:

1. It was unclear why the patient was seeing a cardiologist on a monthly basis, without any significant ongoing cardiac related disease. He had a normal nuclear medical stress test and an essentially normal echocardiogram. He did not have aortic stenosis. The RBBB is likely rate related based on the result of the stress test and does not require intensive follow-up.
2. There was no evidence of a documented psychiatric disorder necessitating five visits with psychotherapy.

3. There was no evidence of performance of systolic time intervals, although OHIP was billed. In any case, there was no rationale for repeating this test in any patient and surely not for this patient on a monthly basis when he was known to have normal left ventricular function.

Dr. Deep defends his diagnosis of Coronary Diathesis as used by “eminent cardiologists” as referring to genetic factors. He states he did psychotherapy as the patient had labile hypertension with “white coat effect” and he needed to provide insight into his anxiety.

He denied Dr. Z’s contention that a normal nuclear medicine test indicates absence of cardiac disease, and that changes in the wall of the coronary blood vessels, with normal perfusion studies, are critically important and require treatment.

The Committee notes that this patient lacked any significant cardiac disease. Dr. Deep’s justification of silent cardiac disease was not proved in this patient; no tests were undertaken to assess this possibility. The Committee notes that the concept itself lends unsupported justification to label any patient as having cardiac disease given the psycho-social and medical implications of applying such a label. This is below the standard expected of a cardiologist and could cause significant harm to patients.

The Committee accepts the evidence of Dr. Z and finds that Dr. Deep’s treatment of this patient was below the standard of professional practice and demonstrates incompetence.

Patient EE

Dr. Deep first saw this 76-year-old woman in October, 2005, although the date listed on the chart is 2006. While the chart was difficult to interpret as notations relating to this visit were on the last page of the chart, they appear to indicate blood pressures of 168/65 and 180/75 and a notation that blood pressure “tends to be low.” On this initial visit, she was started on Avapro 150 mg daily for hypertension.

Dr. Z's concerns were as follows:

1. Dr. Deep started blood pressure medications at the first visit without looking for secondary causes of hypertension or performing repeat blood pressure measurements. He did not follow, or explain his lack of following, the Canadian guidelines for hypertension management applicable to that time frame.
2. There was no prior family history of hypertension, the physical history lacked adequate details of the blood pressure measurements, and there was no performance of an adequate physical examination, including performance of fundoscopy.
3. Dr. Z did not find the expected adequate laboratory assessment associated with a making a diagnosis of hypertension. There were no clinical features of a hypertensive emergency that required urgent treatment before testing. This patient had had recent surgery and her pain may have been impacting her blood pressure. Monitoring is key.
4. The choice of medication (if even needed) was not unreasonable, but there are stronger scientifically based choices for treatment of systolic hypertension in the elderly such as thiazide diuretics and others.

Dr. Deep testified that he relied on her historical data even though she was unclear and inaccurate about some of her past medical findings. He stated that, during the period subsequent to his suspension from practice, he received a letter from an emergency room that confirmed her diagnosis of hypertension.

The Committee does not dispute the possible diagnosis of hypertension. What is concerning is the lack of a proper history, physical examination and appropriate work-up that, based on the accepted testimony of Dr. Z, is expected of a cardiologist. Initiating treatment while relying on a history of an admittedly unreliable patient, showed errors in knowledge and judgment and could bring the life of a patient into danger. The Committee is of the opinion that Dr. Deep's treatment of this patient was below the standard of

professional practice and demonstrates incompetence.

Patient L

Dr. Deep first saw this 40-year-old woman in December, 1997. Dr. Deep stated in testimony that her physician referred her, although there was no consult letter or communication with this physician. Dr. Z found the initial workup to be scanty and incomplete. Although she had no specific cardiac complaints, she was followed every one or two months for eight years.

Dr. Z had the following concerns:

1. Dr. Deep prescribed Lipitor for this patient. Her lipid levels were already at target levels and her ten-year risk of coronary artery disease was low at 3%. Her levels reached by treatment were far below that required.
2. Dr. Deep started her on Provera (a progesterone hormone) in July 2000. She was apparently seeing a gynecologist at the time. There is no proper history and physical examination prior to starting this medication. This is not something a cardiologist usually does, as monitoring is required.
3. Dr. Deep failed to communicate to the patient's family physician a concern regarding finding a "cervical node" (lymph node in her neck) described as "smooth size of small olive." No further mention or follow-up of this is noted.
4. Dr. Deep diagnosed this patient with mitral regurgitation, a diagnosis she does not have. Nine months later, there is a notation that the mitral regurgitation is "mild." On a later echocardiogram, "trace physiologic mitral regurgitation" was reported. This is within normal limits. Many labs do not, in fact, even report this because it is not significant. This is something that should be obvious to a cardiologist.
5. On two occasions, syncope and "lightheaded lately" were listed. The only diagnosis

relating to these symptoms were “Stress induced anxiety attacks, discussed.” No proper work-up or differential diagnosis of this potentially dangerous symptom was noted.

6. Following initiation of therapy for hypertension, there was no baseline blood work or proper monitoring of the possible complications of the drugs utilized.

7. It seemed apparent from the chart that Dr. Deep was prescribing blood pressure medication for patient L’s mother, whom he had not seen. Patients on antihypertensive medication require follow-up.

Dr. Deep testified he knew her mitral regurgitation was clinically insignificant and, as a skilled cardiologist, could hear trace and mild murmurs and intentionally did not order an ultrasound. He stated he did order an ultrasound later because of Dr. Z’s report. He stated she did have a weight reduction trial that was successful for short periods and then reversed due to her employment and studies.

He stated he does not “slavishly” follow guidelines and that although her risk of coronary artery disease was 3%, for her it was 100%.

He stated that Dr. Y originally started the hormonal replacement, and he just repeated prescriptions.

He stated that he treated her mother due to what he considered an improper treatment stemming from an emergency room visit.

The Committee accepts the testimony of Dr. Z that the care of this patient was superficial, and exposed her to potential danger. There was lack of proper monitoring, and a reliance on a self-proclaimed ability to assess murmurs that Dr. Z stated was not possible. Even if these murmurs could be assessed as stated by Dr. Deep, the lack of confirmation after making the diagnosis, and only referring to it as mild nine months later, fell below the expected standard of practice. We find the undocumented explanations for later ordering

an ultrasound and for the mother's prescriptions not credible. The Committee concludes the allegations that the treatment of patient L was below the standard of professional practice are proved and demonstrates incompetence.

Patient E

Referred by her gynecologist in September, 1999 concerning an abnormal ECG, Dr. Deep diagnosed her with "hypertension, and Left Bundle branch block, r/o Coronary artery disease, anxiety, Irritable Bowel Syndrome, and Cardiomyopathy, probable."

There were no consultation notes to Dr. Y. Dr. Deep stated he did not do the note in 1999 because Dr. Y had retired. Exhibit 65 indicates that he did not retire until 2003. On cross-examination, Dr. Deep stated that he must have done a note but that it was in "another dormant file." Dr. Deep did not enter this additional file into evidence.

Dr. Z's concerns are as follows:

1. Dr. Deep diagnosed this patient with cardiomyopathy. He failed to further delineate this problem by investigating to find an underlying cause of the cardiomyopathy. Dr. Z considered this a major deficiency.
2. Dr. Deep prescribed an ARB for this patient. At the time, there was no evidence for the use of ARB's with cardiomyopathy and the patient should have been prescribed an ACEI first.
3. There was deficient monitoring of blood chemistry. In the six years of charts that he reviewed, there was only one record of electrolytes measured.
4. The patient exhibited resistant hypertension with normal renal function. Dr. Deep should have investigated for other causes of hypertension, such as renal artery abnormalities.
5. There was lack of an appropriate and complete assessment when the patient

complained of light-headedness in February 2000.

6. When this patient moved and Dr. Deep sent a summary of her cardiac care, he did not mention his thought that this patient had a cardiomyopathy to the new physician. This is a serious omission. Dr. Z used the term “boggled his mind” in noting this omission.

Dr. Deep stated she was referred by another reliable patient who was able to give information for her; furthermore, he argued that she was seen in hospital and prescribed a thiazide which supported the diagnosis of hypertension when he first saw her.

Dr. Deep was of the opinion that her cardiomyopathy was obviously hypertensive in origin. As well, he stated that her hypertension was well controlled and disputes that it was resistant.

With respect to his failure to list cardiomyopathy as a problem in the medical summary given to the patient when she moved, Dr. Deep stated this document was merely an “information document” and it was not addressed to a specific physician. He stated that a diligent family physician could have called him and, as he stated on cross-examination, he questioned why Dr. Z would “alarm” the family physician with this information.

The Committee is convinced on the evidence before it, especially concerning the cardiomyopathy, that the condition was not properly delineated, that changes in left ventricular function were not properly followed, that no communication occurred to a new physician regarding this possible major problem, and that Dr. Deep’s explanation of this omission lacks credibility. It is expected that a note for use by any physician would mention this diagnosis, and the expectation that a new family physician should contact him concerning an unmentioned diagnosis is farfetched and not in the realm of any specialist’s expectation. While there is a dispute as to the presence of “resistant hypertension,” the fact that Dr. Deep added a fourth anti-hypertensive to her regimen would indicate to the Committee that the hypertension was not responsive to treatment. We therefore agree that further studies to delineate other treatable causes of hypertension

should have been pursued. We find the care displayed a serious lack of judgment, fell below the standard of practice of the profession and demonstrates incompetence.

Patient K

Dr. Deep first saw this 58-year-old woman in March 1997. She was on a beta-blocker medication (Sotalol), apparently for atrial fibrillation. The recorded diagnoses were “Rheumatic heart disease with Mitral regurgitation, symptomatic ectopic repolarizations, and past history of atrial fibrillation, benign hypertension, Anxiety, and rule out Hyperlipidemia and Coronary Heart Disease.” For the latter, the chart notes “imponderable.”

Dr. Z testified to the following concerns:

1. There is no objective evidence of atrial fibrillation in this patient’s chart. If a patient came to a cardiologist with this historical diagnosis, you would expect him or her to obtain this information from the preceding physician. Dr. Deep’s evidence is that he did not obtain the chart because the event was more than 15 years ago. When he was shown that it occurred eight years prior, Dr. Deep suggested that he did not need the record. Dr. Deep stopped the medication she was on and substituted Propafenone at an excessive prescribed dose, and later prescribed it on a “when necessary” basis when the patient was exposed to cold weather. The latter form of dosing was unheard of by Dr. Z and clinically had no justification. There was no consideration of why she was on her original medication, or why one would substitute a contraindicated medication. A proper investigation was not done, although there was a Holter monitor report from December 1996 that was reported by the laboratory as a “normal Holter Monitor recording.” There is no indication of any correspondence between the patient’s symptoms of extra systoles and the Holter monitor report.
2. Dr. Deep failed to discuss treatment options for atrial fibrillation with this patient - rate control and Coumadin versus rhythm control. He did not discuss the risks of Propafenone. There was some potential danger in switching the patient from Sotalol to

Propafenone with no wash out period between the medications. There was no urgency to start Propafenone after discontinuing Sotalol as she was in normal sinus rhythm at the time. As well, Dr. Deep increased the dosage of Propafenone via a phone conversation without proper examination or diagnosis.

3. In November, 2001, Dr. Deep mentions anxiety concerning skipped beats when outside in cold weather and therefore instructs the use of Propafenone two or three times a day when out in the cold. Dr. Z found this to be an “unbelievable recommendation for such a potentially dangerous anti-arrhythmic medication.” Dr. Z considered that this sequence of events (treating a non-documented arrhythmia with potentially toxic medications) showed a very serious lack of knowledge, skill and judgment.

4. She was treated for hypertension due to symptoms that Dr. Deep felt were related to this diagnosis. A transient ischemic episode was mentioned but considered less probable. At that time and with that diagnosis, there was not a complete physical and neurological examination before starting medication. Four days later, with a blood pressure of 134/80 (normal range), her dose of medication was doubled. An adjustment upward of anti-hypertensive medications after four days when the blood pressure was at target levels was not indicated.

5. The patient was at target levels for the dates that cholesterol was measured, and there was no rationale for the prescription of Pravachol, or any explanation given in the record. There was no recorded discussion of the pros and cons of therapy with this anti-lipid agent.

6. The echocardiogram showed mild to moderate mitral regurgitation. This should have been noted and followed, especially in a patient with atrial fibrillation where there is an increased potential for atrial dilatation. There was no follow-up of a changing diagnostic picture. Although Dr. Deep diagnosed the mitral disorder by auscultation (listening with a stethoscope), there was no notation of any change in the auscultory findings with the change in the echocardiogram.

7. Dr. Deep noted possible coronary artery disease but failed to delineate this further. She had a cardiolute stress test done but, when this did not help in the diagnosis, no further investigations were done. Over six years, no laboratory delineation of this diagnosis was performed. In a 2004 letter to a Dr. TT, a surgeon she was referred to for an adnexal mass, the diagnosis of coronary artery disease or the results of the stress test were not mentioned or provided to this physician.

8. Dr. Z described Dr. Deep's preoperative assessment as "woefully lacking." The cardiac concerns had not been sorted out and he suggested to the general surgeon that the surgeon arrange an angiogram at the time of her surgery. A diagnosis of coronary artery disease, and thus her risk of undergoing a general anesthetic, should have been established prior to surgery.

9. Dr. Deep prescribed Vioxx for this patient despite ample evidence and a Health Canada advisory that preceded the prescription. He believed this patient had coronary artery disease and thus exposed her to an increased risk of the cardiovascular problems associated with the drug. He did not discuss these risks with the patient.

10. Dr. Z was concerned about potential boundary violations in Dr. Deep seeing her at his country residence. As there was no mention of others being there, it would appear to him a serious lack of judgment.

Dr. Deep testified that he judged the dose of Sotalol she was taking on presentation was low and switched her. He is of the opinion that the Sotalol and Propafenone had a low potential for fatal heart arrhythmia, and that combining the two was not an issue. He stated that she did not have a family physician when she first came to him and that, as an intelligent patient, he could believe her diagnosis. He stated he did not need her previous medical records.

He stated that the medical visits at his country home were to save her trips to Toronto,

and her husband was present during the examination.

The patient deferred tests that he was to order so she could holiday down south and because they were superseded by need for chemotherapy for cancer of the ovary that he diagnosed on a clinical basis and referred her for treatment.

We accept Dr. Deep's explanation for the visits to his vacation home, that proposed cardiac evaluations were deferred due to her diagnosis of cancer, and that no boundary violation occurred.

The Committee accepts Dr. Z's evaluation of the limitations in Dr. Deep's handling of the cardiological aspects of care. The lack of proper diagnosis, followed by treatment change for a diagnosis that was not established and use of a more dangerous medication with no specific reason, cannot be justified. The continued lack of communication and absence of proper follow-up lead the Committee to conclude that the care of this patient was below the standard of practice of the profession and demonstrates incompetence.

Patient AA

Dr. Deep first saw the patient at age 72. She was referred by her family physician because of atypical chest pain, palpitations and a fluctuating blood pressure. Diagnoses listed were angina pectoris probably secondary to coronary artery disease, seasonal affective disorder and non-obstructive coronary artery disease (NOCAD).

Dr. Z testified to the following concerns:

1. A consultation note was not sent to her family physician for five months, and there were discrepancies between the chart and the consultation note.
2. Having made a provisional diagnosis of coronary artery disease, there were no investigations at the time to establish the diagnosis. The diagnosis of NOCAD could not be made from the history and, in any case, it was not a diagnosis but a group of possible

diseases that need delineation. According to a stress test that was performed subsequently, she did not have coronary artery disease.

3. Regarding his prescription of lipid lowering agents, Dr. Deep should have tried lifestyle changes before initiating drug therapy. In addition, he missed elevations in her liver function tests. These could have been exacerbated by medications.

4. Dr. Deep failed to further investigate elevated fasting blood glucose levels in this patient. There was no proper follow-up according to accepted guidelines.

5. The ECG's were of poor, non-diagnostic quality, with no identification of leads, making it impossible to verify the abnormalities that Dr. Deep commented were there.

6. Dr. Deep provided psychotherapy while the patient had a family physician and was seeing a psychiatrist. No coordination between Dr. Deep and these other physicians was noted in the chart. Dr. Z could find no evidence of physical examinations in April, 2006 or August, 2006 appointments as part of the medical assessment. Dr. Deep testified that she may have had another "pressing engagement" that day, but the OHIP records show that Dr. Deep billed for time based psychotherapy on both of those days.

7. Dr. Deep billed for systolic time intervals but no evidence of their performance was noted in the chart. Dr. Z was of the opinion that, even if performed and not noted, they could not be justified on a patient without valvular heart disease or left ventricular systolic dysfunction.

Dr. Deep considered that Dr. Z demonstrated incompetence in stating that the stress test ruled out coronary heart disease. He stated that 60% obstruction was required for a positive test. He denied missing the rise in liver enzymes but does not consider them important and ascribed them to a transient cause. He denied that the level of glucose was indicative of possible diabetes, stating it is "impaired glucose function, and no further follow-up required." He said that the patient may have been in a hurry on the dates no

physical examination was done but that he did discuss her cardiac condition with her. He stated that the patient chose to discuss personal matters with him, which explained the psychotherapy.

He testified that, if systolic time intervals were billed, they were performed.

The Committee can find no documentation supporting Dr. Deep's contentions, and concludes that the explanations are ex post facto. Dr. Z's critique of the care is both externally and internally consistent and was accepted by the Committee. We find that the professional care of the patient was below the expected standard and shows disregard for the welfare of the patient to the extent that it demonstrates incompetence.

Patient U

This patient first seen in June 1997 with a complaint of easy bruisability. Her listed diagnoses were mitral regurgitation, benign essential hypertension and obesity. There was dyslipidemia.

Dr. Z's concerns were:

1. Based on the record, she did not have mitral regurgitation, as noted on an echocardiogram done in June, 1997 as well as other tests.
2. Dr. Deep put her on female hormone therapy (Premarin and Provera); the reasons for this were unclear and there was no discussion with the patient regarding risks and benefits of hormone replacement therapy noted, nor was there any communication with her family physician. While this would have been acceptable in 1997 for treating her hyperlipidemia, if that was the purpose, it should have been tried for six months with a remeasurement of her lipids before switching to a statin-type medication. She did not have a proper trial of exercise and diet as recommended by the accepted guidelines of the time. There were also complaints of headache that were not followed-up adequately.

3. There was no evidence in the chart that physical examinations were performed on ten visits for which assessments that included physical examination as part of their protocol were billed.

4. In discussing her lipid lowering medications, it is difficult to follow the changes and the reasoning for the changes in these prescriptions. She was being treated to levels below that required by the guidelines of the day.

Dr. Deep testified that she did have mitral regurgitation that he could hear on auscultation, as he can hear murmurs well. Since her laboratory test showed trace regurgitation, he felt his point was made. He also testified that the patient stated that often she did not want a complete examination or an ECG and he worked with her preference.

His choice of treatment for lipid abnormality was based on genetic factors in her family history, and because she had tried diet and exercise in the past. He used his best judgment and did not wait for hope as “hope is not an effective therapeutic strategy,” and he is not bound by guidelines. He stated that all the levels and medications used in treatment could be found in the chart, even if not kept on a specific page.

The Committee found that Dr. Deep’s explanation that he did not do physical examinations on the ten days noted due to the patient’s choice, contradicts his general approach, i.e., in the patient doctor relationship, he knows best. This is noted in his performing psychotherapy without informing patients that he is doing so and his testimony stating that he, and not patients, determines treatment. As he charged OHIP for an examination that included physical examination, it would seem that there is a contradiction - either he made a mistake in billing, did not record a physical he did, or did not do a physical yet charged for it. In any case, we find Dr. Deep’s explanation to be internally inconsistent with the facts of this patient’s record or the totality of his practice and testimony.

During testimony, the Committee heard from Dr. Z that the finding of trace regurgitation

on echocardiograms was insignificant and did not justify a diagnosis and the sequelae from such a diagnosis. Throughout his testimony, Dr. Deep justified his auscultory diagnosis based on reports of trace regurgitation. This patient's diagnosis cannot be justified by Dr. Deep, and it was made and carried forward to the detriment of the patient. It also calls into question Dr. Deep's appreciation of his own skill and judgment.

The treatment of this patient's lipids brings once more to the fore the question of treatment beyond the known consensus goals of the time. We can appreciate Dr. Deep's explanation that he is ahead of the curve and that family genetic factors were a central issue for him. This position does not take into account the possible side effects and morbidity associated with drug use, a not insignificant risk, nor the long term unknown risk of the lower levels in causing future mortality. That this was, at the time, an uncertainty, rather than a quantifiable risk, we question if Dr. Deep should have been doing this without fulsome discussion with the patient as to why he was of the opinion to treat outside the consensus parameters. We have Dr. Deep's testimony that he did so, but no notations or other evidence is available to confirm this testimony.

While it may be possible to find laboratory results somewhere in the chart and correlate them with the treatment proposed, as Dr. Z did in some circumstances, we believe the extreme difficulty in doing so would bring the patient into danger. It would be impossible to easily ascertain information from the chart, especially if another physician were to have to take care of the patient. As well, although Dr. Deep tells us he placed the patient on the DASH diet and exercise program, which failed (or had a past history of failure) and therefore necessitated drug treatment, he never referred the patient either to a dietician or for exercise prescription. His explanation, that this was too inconvenient or time consuming for patients, or they were too busy, is seen by the Committee as generally without merit based on the patients' availability for psychotherapy, and on his location in the city of Toronto.

We find, based on Dr. Z's testimony and analysis of the evidence, that Dr. Deep failed to maintain the standard of practice of the profession and is incompetent in his care of this

patient.

Patient I

This patient was first seen in April, 1986 when he was 49-years-old. The cardiac diagnosis, based on a history of two episodes of tightness in chest, was coronary artery disease with Angina Pectoris. There were non-cardiac diagnoses of “Peptic Ulcer r/o GBD (? Unknown acronym) r/o CDD (cervical disc disease,) and Bronchitis and Sinusitis.”

Treatment began immediately with the addition of Atenolol for Angina Pectoris. Three months later the diagnoses also included hyperlipidemia, coronary artery disease (CAD) with ventricular premature beats and mitral regurgitation.

Dr. Z expressed the following concerns:

1. Dr. Deep diagnosed mitral regurgitation on clinical assessment but the patient did not have this problem. He failed to establish the diagnosis and failed to plan appropriate follow-up. There is mention that an echocardiogram was planned in 1995 and 2000, but was not done until September 2004. He disregarded a patient’s concerns about this diagnosis. If Dr. Deep believed this patient to have mitral valve regurgitation, he should have discussed antibiotic prophylaxis for SBE with him, which he did not. His management of this patient fell below the standard and he displayed a serious lack of knowledge, skill and judgment in the workup of the cardiac status of this patient.
2. It is clear from the chart that the patient was anxious about his cardiac status and numerous hours were spent in individual psychotherapy. This anxiety could have been averted by a more thorough assessment in 1986.
3. Dr. Deep diagnosed this patient with CAD and angina. The history is scanty and insufficient and below what is expected of a cardiologist. It is not possible to know if symptoms are typical, atypical or non-anginal chest pain. He failed to establish the

diagnosis and assess prognosis. A stress test should have been performed after the initial assessment. Investigation was delayed for six years. By the late 1980s, the patient was on large doses of a beta-blocker, calcium blocker and a nitrate and had ongoing symptoms. In 1994, there was the addition of Isordil at 60 mg tid, a large starting dose, in the face of a note that says “no chest pain.” The indication for this nitrate drug was not clear, nor has it ever been shown to have a cardiac protective effect. In March, 1991, the chart notes that stress testing and thallium were considered and not done “as they would not change management; therefore patient spared the inconvenience.”

The patient was treated without an established diagnosis and with multiple medications for an extensive length of time. Dr. Z commented, “I fail to see how clarification of the diagnosis is any less convenient than taking unnecessary medications for many, many years, not to mention the implication of the diagnosis itself.”

Tests were done in 1991 when the patient expressed concern. Dr. Z interpreted the tests as showing evidence against hemodynamically significant coronary artery disease, and that patient I most likely had non-cardiac chest pain. Despite this, Dr. Deep continued to treat him for coronary artery disease with angina. When a stress thallium test was performed in 1996, he did not develop chest pain, and there was no finding beyond borderline significant results. In 2004, a Cardiolyte stress test was done without symptom development.

Dr. Z stated that all the test results showed strong evidence against the probability of a hemodynamically significant coronary heart disease. His conclusion was that “I doubt that many cardiologists would be satisfied simply treating a young man for 18 years as if he had coronary artery disease when the diagnosis was in doubt.”

4. There was no clear rationale for using either an ACE inhibitor or an ARB antihypertensive drug as neither is anti-anginal and the patient had no indication for the medications’ use. Laboratory monitoring of these drugs was inadequate and below the standard of care. As well, Dr. Deep used Mexetil, an anti-arrhythmic drug that would be

contraindicated due to increased chance of death (CAST trial) in a patient who had structural cardiac abnormalities, which Dr. Deep believed the patient had. The indications for starting and stopping the medication were not clear, and it should have only been used in life threatening arrhythmias, none of which is noted in the chart.

5. Dr. Deep believed this patient to have heart disease and yet he prescribed Vioxx despite the known cardiovascular risks with no discussion of those risks with the patient. In his report, Dr. Z interpreted the chart as saying seven to ten Vioxx per day had been prescribed for this patient. Dr. Deep stated this was a prescription for seven to ten Vioxx total, for him to use as needed. Dr. Z apologized for this misinterpretation. Aside from the issue of how one prescribes dispensing seven to ten tablets, the concern remains as to the advisability of using the medication in the face of the cardiac diagnosis made by Dr. Deep.

6. He failed to explore high normal fasting blood sugar levels. According to the Canadian guideline at the time, testing with an oral glucose tolerance test should be considered in individuals with fasting glucose of 5.7 - 6.9 in order to identify individuals with impaired glucose tolerance or diabetes mellitus. Three levels between 2002 and 2004 met this criterion. By 2005, there was a normal level. Elevated glucose levels that are below the threshold for diabetes but above normal do have clinical consequences.

7. By October, 2005, many of the medications were finally stopped, although he was still being prescribed Atenolol on every two days and “also on days when systolic blood pressure greater than 140.” There is no medical justification for using a beta-blocker agent in this fashion, and shows lack of knowledge.

8. The concern about this patient is “very serious,” as he was seen for 18 years without resolution of any of the issues that should have been sorted out at the beginning.

Dr. Deep testified in writing and orally that he believes that the mitral regurgitation was missed on cardiac echogram. He rejected that the patient was treated incorrectly for 18

years, and stated that the history indicates he was treated for hypertension which diminished on his retirement. Dr. Deep stated that non-invasive tests cannot rule out substantial coronary artery disease. In his summary, he stated “Angina Pectoris is a symptom which can only be ascertained properly by the cardiologist or physician who takes a history. It is incredibly insulting for a proven incompetent alleged expert from a second tier academic centre to suggest that a competent well trained cardiologist does not diagnose angina appropriately.”

He stated the patient had no indication to have a coronary arteriogram performed, as he was asymptomatic on competent treatment, and alleged bias by Dr. Z.

Dr. Deep stated that the ACE inhibitor and ARB were properly prescribed, but gives no reasons beyond that.

He stated that you must treat the disease you do not see on coronary arteriography. He stated that a review of older files showed that he had left ventricular hypertrophy, and had a positive ECG exercise study and ventricular extra beats at one out of every fourth beat clinically. None of these tests was entered as evidence, and apparently preceded the treatment evaluated by Dr. Z. They do not address the treatment given during the 18-year period under review.

Dr. Deep stated that the patient had a random blood sugar of 7.0 and a fasting of 5.0 so there was not a metabolic abnormality.

As well, in his opinion, Atenolol is indicated for treatment of his angina and hypertension.

The Committee accepts the testimony of Dr. Z. Dr. Deep’s assertions of pre-existing disease are undocumented and he did not answer the question of why, based on the tests performed, he still insisted on the diagnosis of coronary artery disease. His explanation concerning the mitral regurgitation is unsupported and appears to have no basis in fact.

He continued to hold to the diagnosis of angina and mitral regurgitation notwithstanding the evidence presented.

He justified the use of antihypertensive medication on pre-existing hypertension that ceased on retirement. We find this explanation to be without merit.

The lack of follow-up of high normal blood sugars, while seemingly a less dangerous situation than any of the other issues, indicates a lack of knowledge concerning the pathogenesis and sequelae of blood sugar abnormalities.

We find that the treatment of this patient fell below the expected standard and exhibited a deficient level of knowledge, judgment and care so as to bring the patient into danger. The allegations against Dr. Deep of failure to maintain the standard of practice of the profession and incompetence are established on the evidence.

Patient N

Dr. Deep first saw this patient in August, 1995 at age 70. She presented with a history of hypertension.

Dr. Z testified that his concerns included the following:

1. Dr. Deep discontinued this patient's three cardiac medications at the first visit and started her on five new ones all at once. It would have been impossible to determine which one was causing problems if the patient developed side effects. Her problem should have been further delineated first.
2. ASA and Coumadin were used in combination. There is no evidence of benefit but incontrovertible evidence of an increased serious risk of bleeding.
3. Dr. Deep discontinued this patient's psychotropic medications without communicating with her physician. There is no evidence of a psychiatric examination or

diagnosis. This is atypical of a cardiologist's practice. Generally, a specialist would communicate concerns to the family physician and offer some advice regarding the safest drugs to use.

4. Dr. Deep prescribed Pravachol for control of dyslipidemia. His approach to management was rushed and not in keeping with published guidelines. There was not adequate monitoring for possible drug related toxicities.
5. There were elevated fasting blood glucose levels. This is the same set of concerns as noted in previous discussions of patients dealing with blood sugar levels.
6. Dr. Deep prescribed Quinipril. There is no role for this medication in unstable angina.
7. Vioxx was prescribed in June 2002 and September 2003, when it appears she was being treated as if she had coronary artery disease with angina. The Health Canada advisory was published in April 2002, instructing that caution should be used in patients with a diagnosis of ischemic heart disease. There was no record in the chart of a discussion concerning risk and benefits.
8. In treating hypertension, there was not proper monitoring following use of an ACE inhibitor, after adding Aldosterone and increasing the dose of the ACE inhibitor.
9. Detrol (Tolterodine) a medication for an overactive bladder, should not have been used, as there was no diagnosis that indicates its use. This medication is known to prolong the QT interval, which, in combination with the Propafenone she was on at the time, makes it considerably more dangerous.
10. By far the most concerning decision for this patient was the use of Propafenone, which Dr. Z described as "shocking." He stated that he was unable to identify any cardiac symptomatology. On all of the ECG's performed, it was impossible to make a diagnosis. Dr. Deep decided to use Propafenone based on what he said were extra atrial and

ventricular beats. She was on the medication for 18 months and experienced toxicity as demonstrated by ECG's on the record. In April, 2004, an ECG does not show a conduction abnormality. In May, 2004, it shows QRS widening and a right bundle branch block due to the Propafenone. In October, 2005, Dr. Deep recognized this QRS widening and discontinued the Propafenone. In November 2005, the ECG no longer showed the RBBB. She was asymptomatic thereafter, which indicates that Dr. Deep was responding to innocuous extra beats with inappropriate and unnecessary medication. Dr. Z stated that, in his opinion, the patient had Propafenone toxicity and it took Dr. Deep 18 months to respond to this. As Dr. Deep thought this patient had ischemic heart disease, Dr. Z cannot understand why Propafenone would be used for something that is asymptomatic and expose her to such serious risks. This was a life threatening error and, on cross-examination, Dr. Deep stated she "was lucky to be alive." Dr. Deep insisted this patient's RBBB was not due to Propafenone toxicity. Dr. Z felt that, if this were true, then she had an alternating bundle branch block, which was a prime indication to notify her concerning the need for a pacemaker.

Dr. Deep commented that this patient traveled to Egypt and Connecticut, and that her brother in Egypt and sister in Connecticut, both physicians, were content with his care. He stated that the combination of Aspirin and Coumadin is superior to each medication alone in preventing acute cardiac syndromes or death following myocardial infarction. He stated that at the dose he selected, the INR (indication of proper level of Coumadin) was below a therapeutic level in any case.

He defended his stopping of the Paxil she was on for her psychiatric diagnosis on the basis that it had known cardiac side effects, but that she resumed it notwithstanding the side effects.

He believed her blood sugar levels being below diabetic levels sufficiently preclude any dysglycemia. He disputed Dr. Z's characterization of his lipid treatment, as she had her cholesterol levels done outside the country. As well, she had a family history of cardiac illness.

He justified his use of Propafenone on the basis that she had extra ventricular beats, and that he discontinued it not because of toxicity, but because it was not controlling her symptoms. He disputed that the ECG's showed toxicity and feels that the drug is useful where the benefits outweigh the risks.

He considered that he used Vioxx with caution. He testified that he considered its dangers untrue, as lawsuits have been decided in Merck's favour and present black box warnings on drugs apply to all NSAIDS and COX inhibitors. Dr. Deep testified that he could not recall if he had any discussions concerning the medication with the patient, and testified it was irrelevant since all NSAIDS were the same and he exercised his judgment and discretion in using it.

Dr. Deep did not agree with Dr. Z's comments regarding Detrol as he believes that the combination affects different parts of the ECG and therefore does not increase danger of pro-arrhythmic effect.

In considering the difference of opinion concerning the anticoagulant usage, we accept Dr. Z's opinion, as it is supported by experience and literature. Dr. Deep, while stating his combination is superior, also hedges by saying the levels of Coumadin were below therapeutic levels so there at least would not be an untoward bleeding episode. We believe he cannot have it both ways and, in any case, he presented no valid support for his position.

While we do not dispute that Dr. Deep could or should recommend stopping the Paxil due to his perception of possible cardiac side effects, he did not notify other physicians involved with her care, nor did he offer her a different medication and, in fact, she remained on it.

The use of Propafenone was unnecessary, the wrong choice of medication and there was a lack of appropriate vigilance and follow-up.

We conclude that the treatment of this patient by Dr. Deep was inappropriate from diagnosis to evaluation to treatment, as well as in his failure to recognize the extreme danger that he put this patient's life in. Dr. Deep continues to lack appreciation of his use of anti-arrhythmic agents, the lack of diagnosis, and the misuse of medications in this patient. We find the treatment of this patient to fall below the standard of care of the profession. It brings Dr. Deep's knowledge and judgment into question. As well, there is a serious disregard for the welfare of the patient inherent in this level of care. Incompetence is established.

Patient R

Dr. Deep first saw this 50-year-old woman in May, 2004. Her listed diagnoses were hypertension, obesity, hyperlipidemia and probable mild non-occlusive coronary artery disease. She was also diagnosed as having sinusitis, cervical disc disease and varicose veins.

Dr. Z expressed concern in his testimony about the following:

1. Dr. Deep prescribed Lipitor, 20 mg. With reference to the recommendations in 2003 for risk evaluation and treatment of dyslipidemia, and without medication, her risk of coronary artery disease was low and she was already at target levels even before proceeding with an adequate trial of diet and exercise. The levels she reached with pharmacotherapy were those for someone evaluated by the standards as high risk, and exposed her, for no useful reason, to the possible side effects of this therapy. There was no laboratory work relating to the hyperlipidemia until four months after starting therapy. No regimen of diet or exercise was found in chart.
2. He prescribed Vioxx, 25 mg daily, at the first visit without adequate assessment of her cardiovascular risk. He prescribed Vioxx in May 2004, after the Health Canada advisory of April 2002, to a patient in whom he had diagnosed structural heart disease. There was no discussion of the risks with the patient. It should be noted that Dr. Z could

not find any support for this diagnosis of Non Obstructive Coronary Artery Disease, but if Dr. Deep were treating her with that diagnosis then the use of Vioxx, as noted, was inappropriate.

3. Treating the patient's hypertension with Avapro as it was prescribed on an "as needed" basis; her blood pressure was already at target levels at the first visit. He did not properly follow-up with assessment of her electrolytes and renal function; the first measurement concerning renal function was four months later, and no electrolyte values could be found in the chart. As well, the patient was taking an NSAID medication that can interact with the ARB (Avapro) leading to renal and electrolyte abnormalities.

4. In the treatment of her thyroid condition, there were no notations of any correspondence with the physician in Austria who was alleged to be treating and testing her as well. Dr. Deep alleged during cross-examination of Dr. Z that the thyroid medication was prescribed in Austria. There were no notations in the chart to support this contention. He prescribed thyroid medication to help with her weight reduction and cholesterol, without any indication she was hypothyroid. The values reported from the Austrian physician were in normal range and apparently not treated by this physician. There were results of tests and differential diagnoses that were not clarified or entertained by Dr. Deep prior to instituting treatment. In August 2005, Dr. Deep noted blood test results that were hyperthyroid, yet he did not change the dose of thyroid medication, exposing her to the risks and harm of iatrogenic hyperthyroidism. In addition, if she did have mild hyperthyroidism, then along with her recorded headaches, the possibility of a central cause (pituitary) existed and there was no investigation noted, nor any recorded neurological examination.

Dr. Deep stated that the original lipid tests were done in Austria and he had no reason to doubt them. She had a family history of cardiac disease in her father and brother. He is of the belief that Avapro does not change serum electrolytes or renal function. He stated that he provided her with the DASH diet and she could not lose weight although physically active. He did not communicate with the Austrian physicians, but had no

reason to doubt their results.

The Committee was convinced by Dr. Z's testimony and report that this patient was improperly treated and exposed to unnecessary risks. There was improper follow-up, lack of communication with treating physicians, improper medication use, and a lack of proper diagnosis. The treatment of this patient does not meet the standards of the profession and establishes incompetence as defined in the statute.

Patient T

This patient was first seen in September 2000 at age 79. While the chart was difficult to interpret, it appears that she was seeing a Dr. SS as her family physician, that there was a question of increased heart size and that she had a Holter monitor test and was on Nitroderm patch. She was diagnosed as having bronchitis and coronary artery disease with angina pectoris, osteoarthritis and anxiety. The patient did mention that she had a history of increased heart size.

Dr. Z's testified to the following concerns:

1. There was no contact with the family physician concerning past history or results of his consultation, nor was there a follow-up on an echocardiogram that he mentioned on the first visit he would do. It was noted on a chest radiograph that there was "mild ventricular enlargement." There was never a follow-up of this issue.
2. The diagnosis of coronary artery disease with angina was never dealt with by a proper history or physical examination.
3. He stopped the anti-hypertensive drug she was taking, and prescribed in its place Altace, 5 mg daily. Because of poor organization of the chart, missing medication lists and prescriptions, it was difficult to follow her changing anti-hypertensive medications. The combinations of medications utilized along with the use of NSAID's, as well as the lack of proper follow-up of essential laboratory data, exposed the patient to risk and was

below the standard of practice, illustrating poor judgment and knowledge.

4. In October 2000, Dr. Deep stopped the beta-blocker Sotalol, reasons unspecified. He did an ECG in October, 2000 that was of extremely poor quality and “essentially non-diagnostic.” Dr. Z disagreed with Dr. Deep’s interpretation that the ECG showed sinus bradycardia with interpolated nodal beats. The Sotalol was added back with a note that it was done to “mainly decrease HR.” Dr. Z commented that this drug has a serious pro-arrhythmic effect especially with women using diuretics. The indication for which it was used is unacceptable, and no cause for the diagnosed tachycardia was determined.

Dr. Z, in his oral testimony, wanted to emphasize that it would have been a simple matter to sort out this patient’s concern regarding an enlarged heart by doing an echocardiogram. This was not done.

5. Drug treatment for dyslipidemia began on what Dr. Z surmised as a November, 2000 appointment, although the chart notation has no name or date. Dr. Z was concerned that the medication use was based on a non-fasting sample, that no modification was attempted in weight, exercise, smoking cessation, and that no referral to a dietician was made. No repeat fasting level was done before initiation of therapy.

6. In May, 2001, she complained of fatigue, and was found to have a heart rate of 30-34 beats per minute. The situation was felt to be of such severity that she was sent by taxi to hospital where she was admitted to the cardiac care unit. Dr. Z notes that on that day, no ECG was performed, although Dr. Deep stated on numerous times that he did ECG’s on all visits as cardiac illness can be silent. On this occasion, when indicated, it was not found. The sending of the patient to the hospital for assessment was reasonable; what was unacceptable is the OHIP notation of two units (51 minutes) of psychotherapy with no notation of content. Working on the basis that such psychotherapy was performed, it would seem to delay the evaluation of her dangerously low heart rate for an hour and put the patient’s life in danger.

7. The use of Diazepam in December, 2002 when the patient was already on a same class medication. In January 2004, he added more sedation of Ativan (lorazepam), 2 mg, at bedtime. It was unclear to Dr. Z why Dr. Deep prescribed these medications when she had a family physician and no psychiatric diagnosis was present. Dr. Z was not able to find chart verification that the patient was actually seen and examined when the Ativan was prescribed.

8. The patient was hospitalized with acute bronchitis in December 2004, and the hospital recommendation sent to Dr. Deep by letter was for her to have repeat pulmonary function tests. There is no evidence that this letter was reviewed or that the tests were ever performed.

Dr. Deep disagreed with her initial blood pressure noted by Dr. Z, and testified that she gave a classical history of angina. He stated she was referred by a friend and clearly instructed him to not contact Dr. SS, whose treatment Dr. Deep felt was inadequate. He stated that he could assume her cardiac size would regress with adequate hypertension control. Because of her arthritis, he would not send her for ultrasound tests or radioisotope perfusion studies. He felt the patient was accustomed to use of benzodiazepenes based on his recollection and had situational problems that required it. He felt that repeat pulmonary function tests would not alter his treatment and that she was a smoker who would not quit. He states in his final summary that she was sent to hospital with an ECG by ambulance, but makes no notation concerning the psychotherapy.

The Committee concludes that Dr. Z's assessment of the chart and treatment was an accurate reflection of Dr. Deep's care. Dr. Deep's explanations were specious, did not reflect the chart, added unverifiable patient information and directions, and relied on ideas not reflective of reality. That the patient would have been discomforted by having to proceed for tests, when she could be seen frequently at his office in Toronto and traveled to Florida, is seen by the Committee as unbelievable excuses to explain away proper adherence to the expected standards of a cardiologist in a metropolitan area. The Committee concludes that, in the cited areas of concern, Dr. Deep's treatment and

evaluation were below the standards of the profession and demonstrate incompetence.

Patient H

This patient was first seen in June, 1989, at 51 years of age. Her diagnoses included hypertension, mitral regurgitation with aortic stenosis, mild, probably on rheumatic basis.

Dr. Z testified to the following concerns:

1. The chart organization made it difficult to be clear as to sequence. For example, there was no note for a June, 1989 visit, but a typed note dated in July, 1989 was found in the chart referring to the June visit.
2. Propafenone was apparently prescribed for this patient for symptomatic ventricular ectopic depolarizations as noted in a letter dated in August, 1993. He could find no documentation of an arrhythmia in the chart. PVC's are benign and treatment with Propafenone is excessive and exposes the patient to possibilities of pro-arrhythmia and death. Again, Dr. Deep believed this patient had structural heart disease and should never have prescribed this medication in the first place. Please refer to the extensive discussion of the Propafenone issue in the section on anti-arrhythmic drugs. In summary, there was a lack of diagnosis, misuse of this drug in someone with her diagnosis, lack of proper monitoring, and no Holter monitor performed at the time the medication was prescribed.
3. Biaxin, an antibiotic, was prescribed while she was on Propafenone. Biaxin is, in and of itself, a pro-arrhythmic drug. Dr. Z expressed concern with regard to the additive effects of pro-arrhythmic drugs.
4. A diagnosis of aortic stenosis was made on a clinical basis. Dr. Deep describes an aortic stenosis murmur up until 1993, but then later describes a holosystolic murmur. In a letter to a Dr. RR, he commented on a possible etiology due to viral endocarditis, a diagnosis a cardiologist ought not to consider as the most likely differential diagnosis. He stated that he did not do further laboratory follow-up as he considered her aortic stenosis

to be clinically insignificant. Dr. Z found the changing clinical findings and scanty description of physical signs suggested “a lack of clinical skills.”

5. Dr. Deep prescribed Vioxx, believing the patient had structural heart disease, after the warnings concerning Vioxx in this situation were in place. He had diagnosed her with angina and there should have been an informed discussion with her regarding the potential risks of using this drug.

6. A discussion concerning the use of Coumadin should have occurred in 2002. It was not discussed with her again until 2005, after Dr. QQ, a hospital cardiologist, raised the issue. Dr. Deep executed a document he called “a release” for her to sign in June of 2005, attesting that she had refused the medication.

7. Dr. Deep did a gynecological examination, not an expected procedure for a cardiologist.

8. As noted in the section on dyslipidemia treatment, she received medication without assessment of her risk factors.

Dr. Deep stated that clearly Coumadin was discussed, albeit at a latter date. Dr. Deep said he discussed this one month before he received Dr. QQ’s consultation note. He changed the drug that was prescribed for her atrial fibrillation due to the side effects. Dr. Deep testified that the Propafenone did her no harm and RBBB is often associated with mitral valve disease. The RBBB could not be attributed to the Propafenone she received. He disagreed that the CAST study relied on by Dr. Z had any merit in influencing his use of Propafenone. He noted that the diagnosis of angina was historically accurate.

Dr. Deep stated that, as an internist, he could do pelvic examinations and it saved her having to go for another appointment to get the medication she needed. He also mentioned he was functioning as her family doctor.

Dr. Deep considered the use of Vioxx appropriate. He considered that the patient had a short occurrence of atrial fibrillation and he successfully treated it. Dr. Z was wrong to attribute it to anti-arrhythmic medication. He stated that Biaxin does not cause an arrhythmia called Torsades de Pointes.

The Committee acknowledges that as an internist Dr. Deep can do pelvic examinations, although the Committee had concern that there was no one else in attendance in his office setting. We also question that, if he is functioning, as he insists, as the family doctor, there are commonly held expectations of preventative care in a post-menopausal patient that include proper diagnosis of a pelvic symptom, pap smears, breast examinations, bone densities and other standards of practice that the physician members of the Committee have knowledge of. However, the Committee was not presented with expert evidence as to the standards of practice of a family physician in this area and, while concerned, made no finding as to his performance in the role of family physician.

The Committee notes that Dr. Deep's explanation concerning his lack of use of Coumadin or his choice of Biaxin and Propafenone does not answer the criticisms documented by Dr. Z. He used as evidence his interpretation of a study that 38% of doctors would use this type of anti-arrhythmic drug in contraindicated circumstances.

The Committee is of the opinion that the use of the anti-arrhythmic medication Propafenone was not indicated, and that its use and the subsequent management and lack of proper monitoring and addition of other medications that increased its possible side effects, was below the standards of the profession and demonstrates incompetence. The explanations of Dr. Deep, when taken against the overwhelming evidence presented by Dr. Z in this area, as well as the management of her other conditions, confirms the conclusion of Dr. Z that "the use of a potentially toxic medication to suppress apparently symptomatic (although no evidence to support this claim) premature ventricular beats, which is a benign problem, shows a lack of knowledge and judgment."

Patient S

According to written notes, this patient was 40-years-old when he started seeing Dr. Deep in 1984, although there was mention in the chart of a laboratory report in 1982. His diagnostic list in 1985 was coronary artery disease (CAD) with anterior wall myocardial infarction (AWMI), mitral regurgitation and aortic stenosis. There was a hyperlipidemia issue, hypertension, and transient homonymous hemianopsia (a transient loss of vision of part of the visual field).

In considering the record, Dr. Z had the following comments and concerns:

1. The major concern in this patient was that Dr. Deep diagnosed three conditions he did not have – myocardial infarction, aortic stenosis and mitral regurgitation.
2. His blood sugars results were borderline, with one high random sugar that was not followed-up for five months. There was a borderline upper limit normal sugar in 2005. No follow-up was noted. The Canadian Diabetes Association Clinical Practice Guidelines suggest a formal glucose tolerance test.
3. For a decade, he was seen every one to three months for hypertension and hyperlipidemia; there is a real question of the need for specialist consultation this often.
4. There was no notation about how diagnoses were made, or about information given to patient concerning these diagnoses.
5. The myocardial infarct diagnosis may have been based on an ECG, which was not only misinterpreted as to what kind of MI he had, but also in and of itself does not prove an MI. The findings may have been in keeping with a normal variant.
6. Given that he was 40-years-old at the time of diagnosis, the implications for lifestyle, insurance and such are significant and tests should have been done to delineate if such a diagnosis was present. There was no notation of any discussion of Dr. Deep's findings

and the implications in this young man believing he had coronary artery disease and a prior MI. When tests were finally done in 1996, they were normal and showed no evidence of either a prior MI or ischemia.

7. He discussed with the patient the advisability of using ASA at levels of up to 650 mg daily. There is no evidence that doses larger than 75-325 had any benefit on cardiovascular events, and ASA has its own possible side effects.

8. His hyperlipidemia treatment included medication to bring him far below the value required by guidelines, especially as his risk was moderate, not high. He was too aggressively treated with doses larger than necessary. In fact, in August, 2005, the medication was changed because of side effects attributed to the original medication.

9. Dr. Deep diagnosed aortic and mitral valve disease. Neither was properly defined clinically and the descriptions of the physical finding were below the standard expected of a cardiologist. As well, there are multiple potential causes for these findings. If there was a concern regarding valve disease, there was a need for delineation, follow-up and use of antibiotic prophylaxis.

10. The majority of the ECG's did not include a patient name or identifier, as well as being below an acceptable standard.

11. In 2004, the patient described visual symptoms. A diagnosis of "Prob TIA or temporary spasm arteriole" was made. There was a lack of a full neurological examination, nor was an examination of the interior of the eye performed. A referral was made to an ophthalmologist, who suggested multiple diagnostic tests. The tests are mentioned in the chart notes, but Dr. Z was unable to find any evidence that the tests were performed. In summary, there was no adequate history, physical examination or diagnostic testing, and considering the multiple diagnoses entertained by Dr. Deep, there was no adequate differential diagnosis of the causes possible.

Dr. Deep testified that he treated the hyperlipidemia aggressively because of family history and was not a “slave” to guidelines. He denied that monthly visits were excessive and stated that he was free to see patients as frequently as he deemed necessary. He felt that Dr. Z’s adherence to guidelines and less frequent visits was accompanied by large mortality rates.

He stated that, while Dr. Z recommended a coronary arteriogram, in his clinical judgment it was not necessary. He stated that there was only “one spurious” increased glucose.

Dr. Deep disputed the evidence related to aspirin doses, stating that low dose aspirin, if buffered, is ineffective.

He introduced a coronary arteriogram performed January 2007, while he was not in practice, which was essentially normal. He stated that this was evidence that he treated the patient correctly.

The Committee accepts the complete analysis of the evidence presented by Dr. Z. We are most concerned that this patient was labeled with a set of diagnoses that were unnecessary, and that follow-up of symptoms and diagnoses were not made at appropriate times. While there is no question the patient survived, the treatment was below the standard of the profession, and demonstrates incompetence. Dr. Deep based his defence on justifying his treatment on the end finding of survival. We are of the opinion that a competent Ontario cardiologist would have not made the original diagnoses, and if he had, he would have followed-up with the proper tests. As well, a competent cardiologist would not have over-treated hyperlipidemia, relied on inadequate ECG’s and subjected the patient to numerous unnecessary visits. With the risk factors present, he or she would have followed-up abnormal sugar results and responded to neurological symptoms with proper diagnosis and follow-up.

The Committee finds that the allegations of failure to meet the standards of practice of the profession and incompetence are proven with respect to the care of this patient by Dr.

Deep.

Patient M

This patient was 76-years-old in 1997 when first seen. He was diagnosed as having coronary artery disease with angina pectoris.

Dr. Z testified to the following concerns:

1. A stress test and lipid levels were not done for two years in a patient where there was a concern regarding possible coronary artery disease.
2. Following an examination that revealed occasional extra beats of no clinical significance, Dr. Deep prescribed Propafenone to a patient whom he believed had structural heart disease. The use of this drug was potentially dangerous in this man.
3. The use of the antibiotic Biaxin, prescribed while the patient was on Propafenone, was contraindicated.
4. This patient had moderate renal failure but this was not recognized or monitored.
5. As noted in other patients, he prescribed Vioxx despite warnings of its dangers, especially to those with structural heart disease.
6. A 76-year-old male with a strong likelihood of coronary artery disease was not aggressively treated for lipid levels.
7. There were serious omissions regarding his preoperative assessment for the patient's hip surgery. Dr. Deep made no mention of patient M's history of pulmonary embolus (hip surgery has associated with it one of the highest risks for clot formation), and chronic renal failure was not mentioned. Valve abnormalities were not mentioned and his history of chest pain was not mentioned.

8. Communication with his family physician did not reflect the diagnosis or medication he was using.

9. Medications such as Propafenone were discontinued in May 2005, with no related note as to why they were necessary in the first place, or why they were discontinued if they were necessary for the treatment of angina and abnormal rhythms.

Dr. Deep denied the need for a Holter monitor study before instituting treatment with Propafenone based on the degree of frequency he saw of ventricular extra beats; the patient's life would have been in jeopardy if Propafenone was not used. He stated that, despite the contraindications for the use of Propafenone in patients with structural heart disease, if the physician believes the benefits outweigh the risks, it can be used. He cited one non-peer reviewed article that he stated supports this contention. He stated he was aware of the renal failure, and that the last test was normal. He outlined his disbelief in the determination of the eGFR as an accurate determiner of renal status. He added that coronary arteriography misses 99% of coronary disease and that diseases not seen must be treated.

The Committee accepts the analysis of the chart as presented by Dr. Z. It illustrates that Dr. Deep placed this patient in danger by treating without a proper diagnosis and by lack of attention to obvious problems such as renal failure. There was a lack of communication and misleading communication with his family physician, as well as with hospital physicians. Dr. Deep's defence follows what the Committee considers his continuing logical fallacy: there are illnesses present that are not noted by the extensive available armamentarium of diagnostic modalities, and therefore treatment of these non-diagnosed illnesses is mandatory. He also relies solely on his clinical judgment, uninfluenced by the preponderance of accumulated peer reviewed evidence, or by the use of judgment based on a clear, cogent and documented basis. Judgment and intuition are not the same. The Committee concludes that Dr. Deep's treatment of this patient was below the standard expected of a cardiologist in Ontario and his testimony demonstrates

continuing present incompetence.

Decision Summary

The Committee evaluated the material presented as exhibits, the testimony of the College's expert Dr. Z, and the testimony of Dr. Deep. We have evaluated the evidence and made our decision on the *Bernstein* standard of balance of probabilities and that proof of the allegations must be based on clear, cogent and convincing evidence. We were aware, in evaluating evidence in a hearing where the lack of clinical competence of a long time practicing physician is the basis of the allegations, that a standard of proof higher than mere balance of probability is to be observed.

While proof of the allegations does not require that all areas under question meet the standard, and it is the totality of the evidence that is to be evaluated, we evaluated each patient and each of the major areas that underlies the allegations, and presented our conclusions in each of these areas.

As noted, we placed great weight on Dr. Z's testimony, written and oral. This testimony remained solid under extensive cross-examination. The written evidence submitted by Dr. Z required him to attempt to make sense of very chaotic records. In a sense, he did a service to these patients and Dr. Deep by organizing the records in a coherent and searchable way. It also allowed him, and Dr. Deep if he so wished, to see the areas of Dr. Deep's care that were below standard, questionable, or unexplained. We emphasize that we are well aware that Dr. Z applied standards and practices of the day, rather than any later evolved standards. We accept his ability to delineate the standards expected of a physician in general and a community cardiologist in particular, based on his extensive clinical experience, his training of medical students, trainees, and community cardiologists. He made efforts to meet with Dr. Deep to get further explanation of his findings before reaching final conclusions. Dr. Deep refused this offer. We are aware Dr. Deep had no obligation to meet with Dr. Z, but it is indicative of an attitude that was the basis of his defence against Dr. Z's findings and conclusions. The basis of this defence was an ad hominem attack on Dr. Z, accusing him of malfeasance, financial motivations,

deficient clinical experience and judgment, and allegations that his family responsibilities made it impossible for him to be a competent cardiologist. We reject these allegations as baseless, and useless in evaluating the conclusions of Dr. Z.

We were cognizant of Dr. Deep's training and accomplishments during and directly after his training. We are also aware that following this period in the 1960s and early 1970s, Dr. Deep had limited contact with other physicians. He testified that he has continued to meet the continuing educational requirements of the Royal College, but presented no evidence to allow us to conclude other than what is apparent - he practiced in isolation. It was also apparent from his testimony that he did not see the need for patients to be included in the decision making process, yet he would state undocumented refusals of the patients when confronted with inadequacies of his treatment. In evaluating his testimony, we found it self-referential, relying on undocumented suppositions and on chronologies that were out of order and did not tell the narrative of the patient at the times of Dr. Z's critiques. He relied on logic that worked on the infinite possibility of some condition being present, rather than on probability and diagnosis. His ultimate defence was his experience and considered judgment. We are very aware of the role these play in medical practice, yet judgment needs to be based on a solid foundation. In medical practice, this is evidence, consensus, consultation, patients' needs, honesty, and the ability to describe a basis for judgment. It is not sufficient for a physician to say he thinks and believes something without appropriate support for that belief. Dr. Deep's lack of communication within the profession, and his failure to inform his patients of the reasons and choices for his decisions, bring danger to his patients and make the work of other physicians treating them that much more difficult.

While Dr. Deep stated that the charts were for his use only and that he had a good memory of his patients, their chaotic condition was a factor in the lack of proper diagnostic and prognostic care. The excessive number of obviously substandard ECG's, denied by Dr. Deep in the face of flagrant obvious deficiencies, were interpreted by the Committee as showing lack of clinical acumen, poor judgment and, we must say, demonstrated a blindness to something that was simply sloppy and detrimental to

patients. Care of patients based on substandard ECG's is dangerous. That Dr. Deep still defends these ECG's after he finally started using a different machine did not foretell a positive future in the care of his patients.

Clinically, we have looked in depth at the allegations in reference to the use of anti-arrhythmic agents, specifically Propafenone. Based on the evidence and our analysis of it, we find Dr. Deep disregarded basic literature on the indications and dangers of this medication, ignored the proper way to follow patients on this medication, and withheld information on the use of this drug from other physicians. He placed patients in danger. We are of the opinion that the evidence of Dr. Deep's practice in this area in and of itself establishes failures to meet the standard of care of the profession and incompetence.

Dr. Deep's unwillingness to bring his attention to the area of renal failure and its effects on his use of medications, and his refusal to see any utility in the standard everyday use of information such as the eGFR results, points to a blindness to accepted data and a self-serving justification for his lack of proper patient care. This too, establishes his failure to meet the standard of care and incompetence.

The lack of psychotherapy notes (with the justification that this was to protect patient privacy), its billing even in physical emergency situations, and the admission that most of the patients were not aware they were receiving psychotherapy, lead us to conclude that, if performed, he did not meet the records standards testified to by Dr. Z and as noted in CPSO policy. On the balance of probability, we find that psychotherapy was not performed. We cannot judge Dr. Deep's competency in this area as we have no records to evaluate. We received no evidence of any training or continued education in psychotherapy, leading us to be concerned as to the adequacy of any psychotherapy that might have been performed. Experience with people is not the only training required. We were not asked to make a finding on the volume of billed and unrecorded psychotherapy and, while troubled by this aspect, make no finding. However, the considerable amount of time charged for psychotherapy services, while at the same time not doing appropriate tests for recognized cardiac symptoms, is further evidence of his lack of judgment.

The areas of management of lipid abnormalities and diabetes similarly show a disregard for the evidence, consensus and standards of the profession. While deviation from the guidelines is always possible, there should be demonstrable reasons to do so. Ignoring them under the guise of superior personal knowledge have, in the reviewed cases, confirmed that Dr. Deep failed to maintain the standard of practice of the profession and seriously disregarded the welfare of his patients, in a way and to an extent that demonstrates incompetence.

In reviewing the care of each patient, an overriding pattern of serious deficiency is established.

In evaluating in detail and in totality the clinical issues discussed, we note that these issues represent serious and dangerous distortions and misrepresentations of knowledge and judgment that put at risk the health and lives of patients. The serious misrepresentations and omissions reviewed are major. When taken individually and as a pattern of disregard for the care of patients, the Committee is convinced that Dr. Deep is incompetent. His belief in his own infallibility, untempered by the opinion and evidence of his peers, is a perversion of the practice of medicine.

FINDINGS

In conclusion, the Committee finds it has been proved that:

1. Dr. Deep has committed an act of professional misconduct under paragraph 27.21 of Ontario Regulation 448/80 and paragraph 29.22 of Ontario Regulation 548/90 made under the *Health Disciplines Act*, R.S.O. 1980 and paragraph 1(1)2 of Ontario Regulation 856/93 made under the *Medicine Act, 1991*, in that he failed to maintain the standard of practice of the profession.
2. Dr. Deep is incompetent as defined by subsection 52(1) of the Code, which is schedule 2 to the *Regulated Health Professions Act, 1991*, subsection 60(4) of the

Health Disciplines Act, R.S.O. 1980, c.196, and subsection 61(4) *Health Disciplines Act* R.S.O. 1990 c. H.4 and in that his care of patients displayed a lack of knowledge, skill or judgment or disregard for the welfare of his patients of a nature or to an extent that demonstrates that he is unfit to continue practise or that his practice should be restricted.

The Committee requests that the Hearings Office schedule a penalty hearing pertaining to the findings made.

NOTICE OF PUBLICATION BAN

In the College of Physicians and Surgeons of Ontario and Dr. Albert Ross Deep, this is notice that the Discipline Committee ordered that no person shall publish or broadcast the identity of the patients or any information that could disclose the identity of the patients under subsection 45(3) of the Health Professions Procedural Code (the “Code”), which is Schedule 2 to the *Regulated Health Professions Act, 1991*, S.O. 1991, c. 18, as amended.

Subsection 93(1) of the *Code*, which is concerned with failure to comply with these orders, reads:

Every person who contravenes an order made under section 45 or 47 is guilty of an offence and on conviction is liable to a fine of not more than \$10,000 for a first offence and not more than \$20,000 for a subsequent offence.

Indexed as: Deep (Re)

**THE DISCIPLINE COMMITTEE OF THE COLLEGE
OF PHYSICIANS AND SURGEONS OF ONTARIO**

IN THE MATTER OF a Hearing directed
by the Executive Committee of the College of Physicians
and Surgeons of Ontario, pursuant to Section 36(1)
of the **Health Professions Procedural Code**,
being Schedule 2 to the
Regulated Health Professions Act, 1991,
S.O. 1991, c.18, as amended

B E T W E E N:

THE COLLEGE OF PHYSICIANS AND SURGEONS OF ONTARIO

- and -

DR. ALBERT ROSS DEEP

PANEL MEMBERS:

**DR. M. GABEL
R. PRATT
DR. N. DE
J. DHAWAN
DR. T. MORIARITY**

Penalty Hearing Dates: June 2, 2008
Penalty Decision Date: July 30, 2008
Written Penalty Reasons Date: July 30, 2008

PUBLICATION BAN

DECISION AND REASONS FOR DECISION ON PENALTY AND COSTS

On April 29, 2008, the Committee delivered its written decision setting out its finding that Dr. Deep committed an act of professional misconduct under paragraph 27.21 of Ontario Regulation 448/80 and paragraph 29.22 of Ontario Regulation 548/90 made under the *Health Disciplines Act*, R.S.O. 1980 and paragraph 1(1)2 of Ontario Regulation 856/93 made under the *Medicine Act, 1991*. The Committee found that Dr. Deep failed to maintain the standard of practice of the profession. The Committee also found that Dr. Deep is incompetent as defined by subsection 52(1) of the Code, in that his care of patients displayed a lack of knowledge, skill or judgment or disregard for the welfare of his patients of a nature or to an extent that demonstrates that he is unfit to continue practice or that his practice should be restricted.

The Committee heard evidence and submissions on penalty on June 2, 2008, and reserved its decision.

EVIDENCE AND SUBMISSIONS ON PENALTY

The Committee heard submissions related to penalty from College counsel and evidence and submissions from Dr. Deep representing himself.

Prior to hearing submissions, the Chair of the panel informed Dr. Deep, as a self-represented physician, of the procedures to be followed at the penalty hearing and the material that can be presented to assist the Committee to determine the penalty flowing from their decision. It was also entered into the record that Dr. Deep had received, in advance of the penalty hearing, a letter from the Hearings Office enclosing a memorandum that informed him of these procedures.

College counsel submitted that the proper penalty would be revocation of Dr. Deep's certificate of registration. She supported this request by quoting from and summarizing from the College's perspective the main points enunciated in the decision on finding. She pointed to the findings that Dr. Deep fell below the standard in relation to: his medical records; his lack of communication with physicians and other health care workers; his deficient office based diagnostic testing; the frequency and quality of ECGs; his clinical

assessment and follow up; misdiagnosis; his use of anti-arrhythmic medications; his failure to appreciate the role and means of dealing with renal function and electrolytes; the diagnosis and treatment of problems of lipid metabolism and glucose metabolism; and, his prescribing of Vioxx. College counsel noted that Dr. Deep fell below the standard in all 25 patient charts that were reviewed. She noted that the College had not asked for a finding concerning Dr. Deep's provision of psychotherapy and, therefore, there should be no aspect of the penalty related to his care in this area. College counsel referred to Dr. Deep's lack of insight into and understanding of the deficiencies found by the Committee and their import, and she highlighted the finding of incompetence.

College counsel submitted that there were no mitigating factors in relation to penalty, but there were aggravating factors, such as Dr. Deep's lack of insight and absence of any remorse. She submitted that there was no sense of a willingness to be educated as Dr. Deep was convinced that he was always right. He showed a serious and profound lack of skill, knowledge and judgment that brought patients into danger. She submitted that public protection could be assured only by revocation of Dr. Deep's certificate of registration.

Dr. Deep submitted that he was disappointed by the decision and that it was a miscarriage of justice. He was of the opinion that the College had misconstrued his self-confidence as arrogance.

Although the Chair informed Dr. Deep repeatedly that the Committee does not revisit its decision on finding during the penalty phase of the hearing, Dr. Deep stated that, in relation to his lack of communication with other physicians, his training was highly advanced, he was up to date and he had no need to consult. He referred back to being a consultant during his residency days. He stated he had no difficulty in reading his own ECGs.

Dr. Deep stated that he was up to date in his MoCOMP and that he had just taken a course on Cardiology for the Practitioner for a continuing medication education (CME) credit of 5.5 hours. He stated that he would be taking other courses and submitted a page

from a journal or magazine, which listed future courses. Some of the courses were underlined.

Dr. Deep stated that College counsel was misleading the Committee with its “alleged expert”, Dr. Z. Dr. Deep stated that the Committee should take into consideration that it may have been completely wrong and, therefore, should not remove his professional status. He informed the Committee that he had nothing to be remorseful about, as he is competent and responsible. He stated all this was, “simply a disagreement between two allegedly competent experts” and, therefore, the Committee ought not penalize one member. He reiterated that the College expert gave “false evidence” and was “loose with the truth under oath.” He contested College counsel statements that he always knows best and was infallible, since no one claims infallibility and “some have higher IQ’s.” In Dr. Deep’s opinion, none of the patients had life threatening illnesses, all were doing extremely well, and none of them had “significant mistreatment”. He informed the Committee of his Notice of Appeal and that the Committee should consider if there was a reasonable apprehension of bias. He stated that he intended to consult police concerning libelous denunciations. He also informed us that this was not a threat.

DECISION AND REASONS ON PENALTY

The Committee considered the submissions of College counsel and Dr. Deep, as well as the CME documents submitted by Dr. Deep. The Committee also had regard to the detailed findings and conclusions in our Decision and Reasons for Decision.

The Committee is aware that the purpose of penalty is to protect the public, express the professions abhorrence of the impugned behavior, uphold the honour and integrity of the profession and the public’s confidence in self regulation, deter the physician and others from the same type of behavior, and to encourage rehabilitation to the extent possible.

We reiterate our final conclusion in the Decision and Reasons for Decision on finding: “In reviewing the care of each patient, an overriding pattern of serious deficiency is established. In evaluating in detail and in totality the clinical issues discussed, we note

that these issues represent serious and dangerous distortions and misrepresentations of knowledge and judgment that put at risk the health and lives of patients. The serious misrepresentations and omissions reviewed are major. When taken individually and as a pattern of disregard for the care of patients, the Committee is convinced that Dr. Deep is incompetent. His belief in his own infallibility, untempered by the opinion and evidence of his peers, is a perversion of the practice of medicine.”

We need not repeat the extent of the lack of knowledge, skill and judgment in each clinical and practice area reviewed and the specific deficiencies noted in regard to each patient. We reiterate that they were extensive and pervasive. Dr. Deep demonstrates a lack of understanding of this, continuing in the penalty hearing to make ad hominem attacks on the College expert, as well as on other representatives of the College. He insists on his own superior competence, again referring to his performance as a resident and fellow over 30 years ago. He presents no convincing evidence of a desire to learn from his mistakes. We were shown one certificate of attendance at a non-specialist day in cardiology, and a page from an unknown source with courses underlined that we were asked to believe he was going to take. We find no mitigating circumstances that would influence our decision as to penalty. There are no signs of remorse, of insight, or of any meaningful desire for remediation. The totality of his rejection of the decision and the support of his past acts demonstrates a propensity to engage in the proscribed conduct in the future.

In arriving at the appropriate penalty, we did not consider issues relating to psychotherapy that are noted in our decision on finding. We also paid no attention to any statements concerning questions of appeal, denunciations to police, or the content of the attempts to re-argue the facts of the case.

Having regard to our findings and having considered the evidence and submissions on penalty, the Committee can find no other way to perform its duty to protect the public than to direct the Registrar to revoke forthwith the certificate of registration of Dr. Albert Ross Deep.

SUBMISSIONS ON COSTS

The College requested that the Committee order costs of \$55,500 pursuant to section 53.1 of the Code. This was said to be calculated on the basis of eighteen days of hearing, twelve days at the rate of \$2,500 and six days at \$3,600, and the penalty hearing day at \$3,600. Counsel for the College summarized these numbers as the amounts that College Council has mandated should be requested for costs on a per-diem basis. It was emphasized that this in no way met the true costs of the College for a discipline proceeding, being less than even the costs of the Committee, Independent Legal Counsel, and the court reporter alone. In this case the College did not ask for costs related to the investigation or expert evidence.

Dr. Deep submitted that no costs should be ordered.

College counsel submitted that this was an appropriate case for costs under section 53.1(3) of the Code. In support, College counsel referenced *Freedman v. Royal College of Dental Surgeons of Ontario*, where the Divisional Court upheld an award of \$85,000 in costs to the Dental College. College counsel submitted Dr. Deep's case was analogous in multiple areas. College counsel submitted that Dr. Deep's defence was to attack Dr. Z, the College's expert, and this attack was both personal and lacked any medical foundation. Dr. Deep called himself as an expert and did not call an independent expert. His testimony was partisan, unhelpful, and not credible. The duration of the hearing was extended by his attitude throughout of not noting or acknowledging any error when warranted, and by his prolonged cross-examination of Dr. Z and his own protracted testimony, both of which were repetitious and overly lengthy. As well, the lack of organization of his charts extended the time required of Dr. Z, as well as that of the Committee, to review, understand and interpret these written records. College counsel submitted these factors as well as the analogous details of *Freedman* to be supportive of the appropriateness of costs being awarded.

Dr. Deep repeated his beliefs concerning the findings themselves and submitted oral testimony concerning his financial condition, which included various bank, tax, MRC, OHIP troubles and debts, which he is challenging legally. He stated that these legal challenges entail high legal costs. He stated that if the Committee was unable to retract its decision, it ought to waive costs, as a “difference of professional opinion ought not to penalize one member.” He stated that it was the College’s expert’s false statement and his looseness with the truth under oath (an opinion rejected by the Committee), that was the cause of the extended hearing, and he denied it was his fault. He submitted that *Freedman* does not apply, as he was a dentist. As well, he stated he was “victimized” by 17 months of being unable to practice. On cross-examination, he was asked about his financial situation in that he owned two homes. He replied that this was true, but that they were heavily mortgaged.

DECISION AND REASONS ON COSTS

The Committee considered the submissions and the one case submitted for consideration.

We first considered whether this was an appropriate case for costs. We concluded that it was an appropriate case. The allegations of professional misconduct and incompetence were proved. A major factor in the time for the hearing was the condition of the patients’ charts, which took untold hours for the expert to review and extensive time for the Committee to review. That the state of these charts contributed to the danger to Dr. Deep’s patients was enunciated in the decision.

In oral evidence, Dr. Z was as concise as possible considering the charts he had to review and make sense of, but the cross examination by Dr. Deep was rambling, repetitious, disorganized and appeared directed more to confuse than clarify. This was true as well of Dr. Deep’s direct testimony.

The Committee is well aware that the prospect of costs being awarded should not inhibit a physician from making full answer and defence but, in this case, the defence was not full, but was misleading and lacked credibility. It was Dr. Deep's choice to dismiss counsel and to defend himself. He had the right to make that choice, but in representing himself he had a responsibility to act reasonably at the hearing. The rules and procedure were explained to him. Even in this penalty phase, he was advised repeatedly as to the process, and that it was not a venue to re-examine the basis of the findings. He remained intent on doing so, and again, preponderantly by ad hominem attacks on the College expert, and by willfully ignoring the analysis and conclusions set out in the Committee's decision.

Dr. Deep's refusal to act reasonably and professionally during the hearing was taken into account.

We would agree with what the Discipline Committee stated in *Freedman* and emphasize its application to this case: "However his attitude, and his behavior, which included his refusal to acknowledge at any time throughout this entire process that he had made an error or that the clinical result was not acceptable, contributed to the overall length of the hearing."

All of these factors support that this Committee should make an award of costs to the College.

THE QUANTUM OF THE COSTS ORDER

When considering the appropriateness of the quantum of costs to be awarded, the Committee was aware of the impact of such an unnecessarily protracted hearing on the profession as a whole. While the profession bears a cost for the privilege of self-regulating, the profession should not have to bear the full costs of unnecessarily extended hearings and a non-credible defence. The factual allegations that were proved were all

within Dr. Deep's capability of changing, and should have been known by a prudent and reasonable member of the profession.

The Committee is aware that the quantum of costs requested by the College involves a predetermined amount calculated on a per diem basis. The requested per diem does not begin to cover the true cost of the daily hearing, much less the non-proximate costs such as investigation, legal staff, and expert evidence.

The Committee noted that the per diem amount requested was changed during the hearing from \$2,500 to \$3,600. We felt that the per diem that should apply is the per diem that was utilized at the start of the hearing, and we decided that Dr. Deep should not bear the increase in the per diem that took place during the hearing. The Committee is exercising its discretion to order that Dr. Deep pay costs on the basis of the nineteen days of hearing at the original rate of \$2,500 per diem. Therefore, we order Dr. Deep to pay costs to the College in the amount of \$47,500. These costs are to be paid within 30 days of this order.

If Dr. Deep wishes, we are prepared to receive and consider written submissions from him as to the timing and terms of this payment. If such submissions are delivered by Dr. Deep, we require the College's reply be delivered within seven days of receipt of his submission. We will then issue a final order as to the timing and terms of payment. If Dr. Deep does not deliver written submissions asking for a variation of the timing or terms of payment, then our order as stated below will stand.

ORDER

The Discipline Committee orders and directs that:

1. The Registrar revoke the certificate of registration of Dr. Albert Ross Deep effective immediately.

2. Dr. Deep pay to the College costs in the amount of \$47,500, to be paid within 30 days of the date of this Order, unless the Committee has made an order following submissions that varies the date or terms of payment.