COMMONWEALTH OF MASSACHUSETTS

Middlesex, ss. **Division of Administrative Law Appeals**

**Board of Registration in Medicine**,

Petitioner,

v. Docket No. RM-19-0282

**Mohan Govindan, M.D.**

Respondent

**Appearance for Petitioner:**

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**Administrative Magistrate:**

Kenneth J. Forton, Esq.

**RECOMMENDED DECISION**

On February 22, 2018, Petitioner Board of Registration in Medicine issued a Statement of Allegations ordering Respondent Mohan Govindan, M.D. to show cause why he should not be disciplined for practicing medicine in violation of law, regulations, or good and accepted medical practice during his interventional cardiology treatment of three patients: Patients A, B, and C. On that same date, the Board referred the matter to the Division of Administrative Law Appeals and issued an order to impound identities and use pseudonyms for the patients. Also on that day, Dr. Govindan entered into a non-disciplinary Voluntary Agreement for Practice Restrictions under which he would not perform any interventional cardiology procedures at any facility. Dr. Govindan filed his Answer to the Statement of Allegations on April 27, 2018.

A series of status conferences followed throughout 2018, including some discovery motion practice. On January 11, 2019, I held a telephone status conference to discuss the state of discovery. Thereafter, the parties requested and produced various medical records and other documents. I ordered the parties to file a Joint Status Report on September 25, 2019. They submitted the report on October 23, 2019; it included each party’s expert disclosure.

On October 21, 2020, the Board filed a motion to correct scrivener’s errors regarding certain dates in its Statement of Allegations. I allowed this motion on October 22, 2020. On October 29, 2020, the parties filed a Joint Pre-Hearing Memorandum, a Statement of Undisputed and Disputed Material Facts, and their agreed upon exhibits. I have marked it “A” for identification.

I held a Webex video hearing on November 10 and 12, 2020. The hearing was digitally recorded. I admitted ten exhibits into evidence at the hearing, including three diagrams. (Exs. 1-10.) Three witnesses testified: Dr. Govindan, Frederick Resnic, M.D. (Dr. Govindan’s expert), and Christopher Lawson, M.D. (Board’s expert).

The record closed on February 2, 2021 with the submission of post-hearing briefs.

**FINDINGS OF FACT**

Based on the testimony and documentary evidence submitted, I make the following findings of fact:

1. Mohan Govindan, M.D., graduated from medical school in India in 1973. He did post-graduate medical training in England, including six years of cardiology training, from 1981 to 1987. He received his Member of the Royal College of Physicians certification. (Govindan I: 56-58; Stipulation.)[[1]](#footnote-1)
2. Dr. Govindan performed his medical residency at St. Mary’s Hospital in Rochester, New York, from 1987 to 1990. He did his fellowship in cardiovascular disease at the University of Arkansas for Medical Sciences from 1990 to 1993. He completed his interventional cardiology fellowship at the Deborah Heart and Lung Center in New Jersey in 1994. Dr. Govindan is board-certified in internal medicine and cardiology. He has been licensed to practice medicine in Massachusetts since 2005. (Govindan I: 58-59; Stipulation.)
3. One of Dr. Govindan’s main job functions is performing cardiac catheterizations. Throughout his medical career, he has performed approximately 10,000 catheterizations. (Govindan I: 46, 59.)
4. Dr. Govindan currently works at the Lowell General Hospital Main Campus and Lowell General Hospital Saints Campus. Prior to the Board’s disciplinary action, he had clinical privileges to perform interventional cardiology procedures at the catheterization lab of Lahey Hospital and Medical Center (“Lahey”) in Burlington, MA**.** Dr. Govindan was not employed directly by Lahey Hospital. (Govindan I: 16; Resnic II: 13, 72.)

***Expert Witnesses***

1. Frederic Resnic, M.D. graduated from Mount Sinai Medical School in 1994, and performed his medical residency and interventional cardiology training at Brigham and Women’s Hospital from 1994 to 2001. He has been licensed to practice medicine in Massachusetts since 2000. Dr. Resnic is board-certified in cardiovascular disease and interventional cardiology. He was Director of the Brigham and Women’s Hospital Cardiac Catheterization Laboratory from 2005 to 2012. (Resnic II: 5-7.)
2. Dr. Resnic is currently Chairman of Cardiovascular Medicine at Lahey. A main part of Dr. Resnic’s job is to supervise 40 cardiologists, including 7 interventional cardiologists. He also maintains a clinical practice as an interventional cardiologist and works in the catheterization laboratory. He spends 25 to 40 percent of his time on direct patient care. Dr. Resnic is a member of the Research Grant Oversight Committee at Lahey and is a member of the Massachusetts Department of Public Health’s Invasive Cardiology Services Advisory Committee. (Resnic II: 6-7, 72.)
3. Dr. Resnic has known Dr. Govindan since 2012, when Dr. Resnic first joined Lahey. Dr. Resnic has never been Dr. Govindan’s boss or direct supervisor. Dr. Govindan and Dr. Resnic have a professional relationship. They do not have a social relationship. Between 2012 and 2016, Dr. Resnic observed Dr. Govindan’s performance on several occasions while he performed interventional cardiology procedures at Lahey. Dr. Resnic was not directly responsible for monitoring Dr. Govindan’s cardiology procedures. The head of Lahey’s catheterization lab is “responsible for ensuring that all cardiologist[s] who maintain privileges at Lahey” deliver care that “meets the requirements and standards for the hospital which are compliant with [the] Department of Public Health.” (Resnic II: 14-16, 72-74.)
4. Dr. Resnic reviewed the medical records of Patients A, B, and C. He provided his opinion based on these records and his personal observations of Dr. Govindan’s procedures performed at Lahey’s facilities from 2012 to 2016. (Resnic II: 18, 47, 52.)
5. Christopher Lawson, M.D., graduated from Georgetown Medical School in 2005. He performed his medical residency at New England/Tufts Medical Center, and a four-year cardiology fellowship, including two years of interventional cardiology, at St. Elizabeth’s Medical Center. Dr. Lawson completed his fellowship in 2012 and has been practicing as an interventional cardiologist since then. He is licensed to practice medicine in Massachusetts. He is board-certified in internal medicine, cardiovascular disease, and interventional cardiology. (Ex. 5; Lawson I: 143-144.)
6. Dr. Lawson spends the majority of his practice on direct patient care. He has worked as an interventional cardiologist at Norwood Hospital since November 2014. From July 2012 to October 2014, he worked as an interventional cardiologist at Wentworth-Douglas Hospital. (Ex. 5; Lawson I: 146-147.)
7. Dr. Lawson received the “Outstanding Cardiology Fellow” award in June 2011. He co-authored an article on “Stent Thrombosis Aspiration Thrombectomy” in 2005. (Ex. 5.)
8. Dr. Lawson reviewed the medical records of Patients A, B, and C and provided his opinion based only on these records. (Lawson I: 148.)

***Interventional Cardiology***

1. Interventional cardiologists diagnose cardiac conditions through *nonsurgical* interventions, usually cardiac catheterizations, hemodynamic testing, and percutaneous coronary intervention procedures, which include angioplasties and stenting. Conditions that commonly call for cardiac catheterizations are coronary artery and vascular disease, cholesterol plaques in the arteries, and other diseases of artery blockages, such as heart attacks. (Govindan I: 15-16; Lawson I: 146, 150; Resnic II: 12.)
2. “Operator” is a common term for an interventional cardiologist performing procedures, including catheterizations. (Lawson I: 210.)
3. The left main coronary artery splits into two branches: the circumflex artery and the left anterior descending artery. The left anterior descending artery further branches into diagonal arteries. The left side of the heart also contains the left ventricle, aorta, and blood vessels that supply blood to the heart muscles. The right side of the heart supplies blood back to the lungs. (Govindan I: 132-133; Lawson I: 61, 132-133, 173).
4. A catheter is a hollow tube through which the cardiologist may place a guidewire, a balloon catheter, a stent, or other coronary intervention equipment. Catheterizations have different purposes. A main purpose of a left heart catheterization is to measure the left ventricle’s pressure. Right heart catheterization is a procedure used to determine the cardiac output of the aorta. (Govindan I: 66; Lawson I: 149-150, 159.)
5. There are several types of catheters. A pigtail catheter is coiled at the tail end of the catheter. An interventional cardiologist generally chooses a pigtail catheter because it is blunt and thus minimizes the risk of perforating an artery. (Govindan I: 79-80.)
6. A coronary angiogram consists of “taking pictures of the coronary arteries using an IV [intravenous] dye injected through a catheter with x-ray imaging.” (Lawson I: 150.)
7. A guidewire is a metallic wire, akin to a rail, that guides the catheter to the designated area of the heart. A flow wire is a “bigger catheter through which equipment can be pas[sed].” The operator measures the patient’s pressure difference while the flow wire is in place and records a ratio that determines the level of aortic stenosis. (Govindan I: 67; Lawson I: 166.)
8. Aortic stenosis is the narrowing of an aortic valve that “reduces flow from one side of the valve to the other” and results in the heart working harder to “move the same amount of blood around the body.” The normal area of a cross-section of an aortic valve is 2 to 4 square centimeters. A valve area of 0.75 to 2 square centimeters is indicative of mild to moderate stenosis. Severe stenosis is defined as a valve area that is less than 0.75 square centimeters. A blockage of an artery of 70% or more is “significant.” Other diagnostic criteria of severity of stenosis are systolic heart murmurs, pressure gradients, and carotid pulse delays.[[2]](#footnote-2) (Govindan I: 89-90, 119; Lawson I: 152; Resnic II: 45-46.)
9. A stent is a wire tube that is placed in the heart’s artery or vessel wall. It functions as a type of scaffolding. A drug-eluting stent “delivers the drug that’s on the surface of the stent” and leaks out a drug that “encourages certain cells to multiply and coat the stent.” The drug inhibits the re-narrowing of the artery and prevents more scar tissue from forming in the artery. Predilatation, also called predilation, is the process of first using a balloon to open up the artery blockage *before* placing the actual stent in the artery. The process of predilating the balloon provides the operator with a sense of the size of the stent prior to the final placement of the stent. (Resnic II: 61-62, 86-89.).
10. A percutaneous cardiac intervention (“PCI”) is a procedure by which the operator blows “up a balloon to open up an artery” or places a stent to keep the artery open at the desired diameter. Angioplasty is the medical term for blowing up a balloon in the heart. (Lawson: 150.)
11. Thrombosis is theformation of a clot. A clot is a “collection of blood cells which have coalesced and formed a solid mass factor, which is called a thrombus.” (Govindan I: 121.)
12. A pressure gradient is the difference in pressure between the lower pressure in the aorta and the higher pressure in the left ventricle. The pressure gradient is measured in units of millimeters of mercury, abbreviated “mm Hg.” The pressure gradient indicates the presence and severity of aortic stenosis. (Lawson I: 155, 158; Resnic II: 23.)
13. There are multiple ways to obtain the heart’s pressure gradient. The peak-to-peak gradient is obtained by using the middle of each of the peak pressure readings on the left side and the readings on the right side. This gradient compares one single heartbeat to another single heartbeat. A *peak-to-peak* gradient of greater than 50 is commonly accepted as showing severe aortic stenosis. A *mean* gradient is obtained by taking the difference between the average (or mean) of the beats of the left ventricle and the average of the beats of the aorta. A *mean* gradient greater than 40 is commonly accepted as showing severe aortic stenosis. (Lawson I: 97; Resnic II: 23-26.)
14. An electrocardiogram (ECG or EKG) is a test measuring the heart’s electrical activity.[[3]](#footnote-3)
15. An echocardiogram is a form of non-invasive cardiac testing, that outlines the heart’s movement, by using ultrasound placed on the patient’s chest, which then reads out pictures of the patient’s heart valves and chambers.[[4]](#footnote-4) (Govindan I: 60.)
16. A coronary angiography is a process by which iodine containing contrast is “selectively injected into the coronary arteries to take pictures to assess for severity or any narrowings or abnormalities of the coronary arteries.” A coronary angiography is a standard procedure for interventional cardiologists evaluating for a suspicion of severe aortic stenosis in the absence of diagnostic symptoms during a physical examination. (Resnic II: 10-11, 39.)
17. A murmur is a flow disturbance over an area of the aortic valve that typically creates “turbulence in the blood, a vibration in the structures of the body” that “generates an audible signal.” Murmurs are graded on a scale of 1 to 6 based on intensity of sound. For instance, a Grade 1 systolic murmur is barely audible. A grade 2 murmur is “an audible but a soft murmur” and is one that “some might hear” and “some might not.” Murmurs are detected through various means, including auscultation, which is the process of listening to the patient’s heart with a stethoscope during a physical examination. The S1 and S2 sounds are the initial systolic sounds of the heart. The S2 sound is “comprised of the closing of the aortic valve, followed by the closing of pulmonic valve.” A Grade 2 systolic ejection murmur (“SEM”) is “heard with aortic stenosis.” (Lawson I: 150-151, 188-190, 199; Resnic II: 45.)
18. An edge perforation, also called a “small tear,” or “dissection,” is a tear at the edge of an artery and consists of the edge being “stretched between the stent scaffolding” and the artery. A full thickness artery perforation, also called a “grade 3” or “type 3” perforation, results in blood flowing freely outside the artery. With a full thickness perforation, the blood then rapidly fills the pericardium (the sac surrounding the heart) and the heart pumps much more quickly because the small space around the heart gets “squeezed” by the large amount of blood rushing out. (Govindan I: 54, Lawson I: 175-176, 180-181, Resnic II: 64.)
19. A tamponade is the procedure of blowing up a stent balloon in the area where there is a lack of blood flow to the artery, so that “no blood can leak out.” The tamponade gives the operator time to conduct other interventions “to try and overcome the problem that has been created.” Pericardiocentesis is the draining of blood from the “space between the heart and the pericardium.” (Govindan I: 137, Lawson I: 177.)

***Standard of Care in Interventional Cardiology***

1. The standard of care for an interventional cardiologist preparing a cardiac catheterization is to “aspirate it appropriately to make sure there is no clot and flush it with saline and prepare it ahead of time before introducing it into the coronary arteries.” Aspirating the catheter is sucking back the catheter to make sure it is flowing freely. (Lawson I: 168; Resnic II: 52.)
2. In 2014 and 2015, the standard of care for calculating a pressure gradient was *not* the peak-to-peak gradient. The standard of care in calculating the pressure gradient is to take the mean gradient, not the peak gradient. (Lawson I: 202, 207; Resnic II: 35-37.)
3. An interventional cardiologist should recognize severe aortic stenosis when the patient presents with a combination of the following symptoms: (a) an abnormal S2 sound and systolic ejection murmurs, (b) mean pressure gradients of 40 mm Hg or higher, (c) carotid pulse delays, and (d) clinical symptoms of shortness of breath and chest pain without other causes. A finding of a systolic ejection murmur, by itself, does not demonstrate that the patient has severe aortic stenosis. (Lawson I: 200, 224; Resnic II: 33, 45-47.)

***Patient A***

1. On September 17, 2014, Patient A saw Dr. Govindan upon recommendation for catheterization by Dr. David Malins, a cardiology partner of Dr. Govindan’s. Patient A was 57 years old. He had a history of recurring chest pain, and recent EKG changes with acute intermittent chest pain. (Ex. 1: 399, 407; Govindan I: 34-35, 41, 70-71.)[[5]](#footnote-5)
2. During the appointment, Dr. Govindan performed a physical examination of Patient A, which included palpation, percussion, and auscultation. He heard no murmurs during this exam. (Ex. 1: 252; Govindan I: 70-71.)
3. That day, Dr. Govindan performed a diagnostic catheterization due to Patient A’s unstable symptoms of worsening chest pain together with abnormal EKG findings. Dr. Govindan inserted a pigtail catheter into the patient’s aorta, through the aortic valve, and into the left ventricle. Dr. Govindan was able to pass the pigtail catheter through the left ventricle in 1.5 minutes. (Govindan I: 35-36, 78.)
4. In his approximately thirty years of experience, Dr. Govindan has never crossed a completely stenosed artery with a pigtail catheter in 90 seconds. Dr. Lawson agreed that reaching the left ventricle with a pigtail catheter in 90 seconds in a patient with severe aortic stenosis “would be rare.” This amount of time would be unexpectedly fast in a patient with “critical aortic stenosis” because it is “very hard to cross [a] stenotic calcified valve that’s not opening well.” (Govindan I: 85; Lawson I: 208-209; Resnic II: 42-43.)
5. During the catheterization, Dr. Govindan found significant disease in Patient A’s left anterior descending coronary artery and diagonal branch artery. He did not identify a murmur. Patient A’s left anterior descending coronary artery exhibited “eccentric stenosis proximally, tapering towards the origin of the first diagonal branch around 85% reduction in luminal diameter compared to the larger proximate vessel.” Dr. Govindan noted that the artery’s ostium (opening) was “mildly compromised with a 50% to 60% stenosis.” (Ex. 1: 408-409, 516-519, 523-533; Govindan I: 35-36; Lawson I: 185-186.)
6. At that point, Dr. Govindan decided to perform a percutaneous intervention because of the patient’s left anterior descending artery stenosis, which he believed was causing the patient’s chest pain. (Ex. 1: 409.)
7. During the procedure, Dr. Govindan measured the pressure of the left ventricle and the aorta simultaneously, using hemodynamic tracing. Tracing results demonstrated a pressure gradient. The degree of the gradient was in the range of 24.6 to 28.9 mm Hg. This gradient range correlates with “moderate aortic stenosis,” and is inconsistent with severe aortic stenosis. (Govindan I: 95, 103; Resnic II: 24-25, 32.)
8. Dr. Govindan’s procedure note stated there was “no gradient across [the] aortic valve.” He admits that a pressure gradient was present and that his documentation of “no gradient” was incorrect. (Ex. 1: 407-408; Govindan I: 107.)
9. An aortic valve that is approximately 1 square centimeter is within the range of moderate stenosis and should prompt an interventional cardiologist to conduct additional testing to determine the degree of stenosis. (Ex. 1: 507; Govindan I: 89-90.)
10. An interventional cardiologist who observes a pressure gradient should conduct a follow-up echocardiogram for the purposes of “further quantification of the degree of stenosis.” “[G]etting an echocardiogram at some point to understand more details about the anatomy and the sort of physical presentation of the valve itself” is helpful. However, the “gold standard” for detecting a pressure gradient is to defer to the “invasive measurement of the catheterization,” in part because “there are multiple reasons why an echocardiogram could be erroneous.” (Lawson I: 226; Resnic II: 35-37.)
11. On September 18, 2014, Patient A was seen by cardiologist Dr. Jerold Weiner, who conducted a physical examination. Dr. Weiner documented “no murmur.” (Ex. 1: 398; Lawson I: 194-195, 198.)
12. On October 1, 2014, Dr. Govindan saw Patient A for evaluation following his stent placement. He conducted an EKG and an echocardiogram during the visit. He noted that the patient’s symptoms were “rather atypical at present” and “not suggestive of stent thrombosis.” (Ex. 1: 529-530.)
13. On November 13, 2014, Patient A’s cardiac rehabilitation providers referred him to diagnostic cardiologist Dr. Kirk MacNaught due to continued left-sided chest pain. In addition, the patient reported a “new burning chest pain” after “he rushed out of the house to get to [the] car.”He was then “kicked out of cardiac rehab due to elevated blood pressure (212/78).” Upon physical examination including auscultation, Dr. MacNaught noted that Patient A had a Grade 2 systolic ejection murmur (“SEM”) with normal S1 and S2 sounds. (Ex. 1: 166-168; Govindan I: 109-110; Lawson II: 190, 200.)
14. On November 14, 2014, Dr. MacNaught performed a diagnostic catheterization of Patient A. The patient’s stents were in good shape. He documented the patient as having “*known* severe bicuspid aortic valve stenosis.” Dr. MacNaught referred the patient for “surgery for [aortic] valve replacement and aortic root repair.” His recommendation made no mention of severe aortic stenosis as an indication for the patient’s valve replacement. There are no diagnostic tests or other medical evidence other than Dr. MacNaught’s note that the patient was suffering from, or was diagnosed with, severe aortic stenosis. (Ex. 1: 210-213; Lawson I: 160-161; Resnic II: 46.) (emphasis added.)
15. A murmur indicative of aortic stenosis often takes years to develop and would be highly unlikely to develop in two months. Normal S1 and S2 sounds are inconsistent with severe aortic stenosis. With severe aortic stenosis, the S2 sound is “usually obliterated,” which means it would not be heard at all or would be merged with the “pulmonic S2.” A louder systolic murmur is suggestive of severe aortic stenosis. (Lawson I: 155-156, 190-192; Resnic II: 45-47.).
16. Dr. MacNaught’s notes did not explain a *new* diagnosis of severe aortic stenosis. He did not conduct or order an echocardiogram for the patient. (Ex. 1: 166, 409; Lawson I: 199, 201; Resnic II: 46-47.)

***Patient B***

1. On June 2, 2015, Patient B was admitted to Lowell General Hospital for chest pain, low blood pressure, diaphoresis (sweating) and EKG changes. At the time, the patient was 69 years old and had anemia and diabetes; both are cardiac risk factors. Anemia increases the risk of excess bleeding. That day, Patient B was referred to Dr. Govindan based on symptoms of “acute coronary syndrome” and concern for ischemia and artery stenosis. Dr. Govindan decided to perform a diagnostic catheterization and angiogram. (Ex. 2: 2624; Govindan I: 117-119; Lawson I: 162-163, 169.)
2. Preparing patients for catheterizations has become a nearly automated process for Dr. Govindan because he has performed several thousands of these procedures over his career. He makes sure to “flush a sheet repeatedly before” doing anything and then “flush these catheters. . . aspirate them . . . push saline through them” to attempt to “avoid any clot.” If a clot forms, Dr. Govindan knows to “dislodge the clot even if it’s going to get inside of the body” to redirect the clot to another part of the body where the least harm would occur. (Govindan I: 45-46.)
3. During the diagnostic catheterization, Dr. Govindan noted Patient B had circumflex artery stenosis of more than 70% caused by a lesion. Because of this condition, the location of the lesion, and the patient’s anemia-related vulnerabilities, Dr. Govindan decided to perform a percutaneous intervention, which involved stent placement. After discussion with the team, including Dr. MacNaught, Dr. Govindan decided to use a bare-metal stent instead of a drug-eluting stent. He made this choice because a bare metal stent was safer for the patient, who was anemic and suffered excessive bleeding. (Ex. 2: 2624-2626; Govindan I: 118-121.)
4. Prior to initiating the percutaneous intervention, Dr. Govindan administered 8,000 units of heparin to Patient B. Heparin is a blood thinner and anticoagulant. Heparin’s blood thinning effect is not instant and typically takes about “five to seven minutes” to take effect. (Ex. 2: 2625; Govindan I: 53, 122, 127, Resnic II: 52.)
5. During the percutaneous intervention, a thrombus (clot) formed in Patient B’s heart. The thrombus completely blocked blood flow of both the left anterior descending artery and the circumflex artery. Given that both “the left-anterior descending artery and the circumflex artery” were affected, the clot “had to arise from someplace other than those two arteries.” The catheter is a likely external source of a thrombus under such circumstances. (Govindan I: 51-52; Lawson I: 168-169.)
6. The formation of a thrombus is a “serious, rare but well-known complication of percutaneous coronary interventional procedures.” The occurrence of a thrombus during a percutaneous intervention is not, by itself, a violation of the standard of care. Even with “meticulous handling of the catheter, [and] the injection system” an interventional cardiologist may still inadvertently introduce a blood clot into the patient. Blood clots can also form when the blood thinner medication has not had enough time to act prior to the intervention. (Govindan I: 127, Resnic II: 47-49.)
7. Another way interventional cardiologists minimize the risk of clots is by monitoring the catheter’s pressure waveform before injecting it into the patient’s artery. Pressure waveform is a measurement of the pressure from the tip of the catheter to where “it’s being measured outside the body.” A clot would result in pressure concentrated at the tip of the catheter. (Resnic II: 51-52.)
8. A clot can form in a number of different locations during percutaneous intervention. The greatest risk of clot formation is inside the catheter lumen itself. Clots may also form outside the catheter lumen or inside the vessel where the guidewire is located. The clot can get pulled into the catheter’s tip, stick to it, and then move to the inside of the catheter once the catheter is placed into the coronary artery. The clot may be tiny or “a completely occlusive clot” that occupies “the entire lumen central channel of the catheter.” (Lawson I: 168-169; Resnic II: 54-56.)
9. Dr. Govindan is known to be meticulous in conducting interventional procedures. In the course of preparing catheters, he would flush the catheters repeatedly, aspirate the catheters, and push saline through them. Taking these actions is within the standard of care in preparing catheters to minimize the risk of clot formation. (Resnic II: 47-49, 52.)
10. Patient B had “a number of medical issues” which “potentially could make someone more prone to clotting.” These medical risk factors included diabetes and anemia, which causes low blood counts. (Lawson I: 169-170.)
11. On June 4, 2015, Dr. James Waters, a cardiologist at Lowell General Hospital, performed another diagnostic left heart catheterization of Patient B. He also performed a coronary angiography. The indications for performing this catheterization were the patient’s complication of thrombosis, cardiac shock, and emergent intubation during his June 2, 2015 catheterization. Dr. Waters documented that despite these complications, the patient “did well” and his left anterior descending artery disease was “treated medically.” (Ex. 2: 2627-2628; Govindan I: 130.)

***Patient C***

1. Patient C first saw Dr. Govindan in June 2014. At the time, the patient was 66 years old and had diabetes, hypertension, and dyslipidemia with episodes of dizziness, nausea, and chest pain. (Ex. 3: 304-306.)
2. On June 18, 2014, Dr. Govindan performed a diagnostic left heart cardiac catheterization and angiography of Patient C. The catheterization revealed 60 to 70 percent stenosis of the patient’s left anterior descending artery. To address this, Dr. Govindan decided to perform a percutaneous intervention, using stent placement to reopen the narrowed artery. (Ex. 3: 304-306; Govindan I: 132-133.)
3. Stent size represents the diameter of the stent when the stent’s balloon is inflated to a nominal pressure. The stent manufacturer’s chart provides the diameter of the stent within 0.1 millimeter or less of tolerance. When the operator inflates the stent, it is “upsized” between 0.5 millimeters and 1.0 millimeters. If at first the stent’s balloon is too small, the operator can use a larger balloon to inflate the stent more and remain within the manufacturer’s upsizing range. The standard practice in interventional cardiology is to estimate the size of the artery and “try to pick a stent that’s 1.1 times that” size. (Lawson I: 219-221; Resnic II: 85.)
4. Selecting a stent size is a judgment call. An interventional cardiologist faces “significant trade-offs” in choosing the stent size for a patient, whose “coronary arteries don’t adhere to th[e] convention” of 0.5-millimeter increments. (Lawson I: 212; Resnic II: 58.)
5. The risk of using a stent that is too small is that the stent will not touch “the vessel wall or if there’s too much of a space between the stent and the vessel wall, there’s a risk that the stent won’t . . . heal into the vessel wall.” If this occurs, then the patient is more likely to develop a blood clot in the stent, which can be deadly. Another risk of an under-sized stent is “late re-stenosis, which is a morbid but less often mortal outcome.” (Lawson I: 217-218; Resnic II: 60.)
6. When Dr. Govindan first started the percutaneous procedure, he placed a 2.5 mm balloon in Patient C’s diagonal branch ostium and a 3.0 mm predilatation balloon in the proximal segment of the left anterior descending artery. At that point, the proximal segment looked underexpanded. Because of this, Dr. Govindan decided to use the next larger stent, which was 3.5 mm. (Ex. 3: 306-307; Govindan I: 136; Resnic II: 58-60.)
7. Dr. Govindan’s choice to use the 3.5 mm balloon stent was “within the standard of care, especially in light of the location of this lesion” in the left anterior descending artery. More “than 90 percent of adult males” have proximal left anterior descending arteries that are larger than 3.5 mm. Dr. Govindan’s choice of stent size demonstrated a reasonable judgment, taking into consideration the risks of under-sized stents versus over-sized stents (discussed below) and Patient C’s circumstances. (Lawson I: 216; Resnic II: 58-60.)
8. During the balloon inflation to 3.5 mm diameter, Patient C complained of chest pain. Dr. Govindan noted that the “distended balloon did appear disproportionately larger than the vessel.” An angiogram revealed a type 3 artery perforation. Dr. Govindan and his team immediately proceeded with a cardiac tamponade,pericardiocentesis, blood transfusion, and the placement of two covered stents. After this, the patient’s perforation was sealed. After observing the patient for another 20 minutes, Dr. Govindan successfully completed placement of a 2.5 mm stent of the diagonal branch artery and a 3.0 mm stent of the proximal segment of the artery. (Ex. 3: 306-307; Govindan I: 137.)
9. A full thickness artery perforation, also known as a “Type 3” perforation, is a known, life-threatening complication of percutaneous coronary intervention. It is life-threatening because it creates a rapid drop in the patient’s blood pressure, which can lead to shock and ultimately death, if not treated immediately. (Lawson I: 175-176, Resnic II: 67.)
10. Dr. Govindan’s use of the 3.5 mm stent may have contributed to Patient C’s perforation. An artery has a limit when expanded by a balloon. It will usually adapt if the balloon stent is slightly oversized. If the balloon stent is “moderately to severely oversized” then the patient could experience a small tear known as an edge perforation. A full-thickness perforation is “surprising” and is not typically seenas a result of using oversized stents. (Ex. 5 at 6; Lawson I: 179-180, 211; Resnic II: 64-67.)
11. A more common and expected reason for a full thickness artery perforation is the presence of calcium deposits on the walls of the artery that break off when the stent is expanded. There is no explicit mention of calcium deposits or calcification in Patient C’s medical records. Dr. Govindan did note that the patient had plaque in two areas of the *right* coronary artery. Artery “plaque” may include calcium. An interventional cardiologist using the standard imaging technology in 2014 could sometimes miss calcium deposits inside arteries. (*See generally* Ex. 3; Resnic II: 59, 65-66.)

**CONCLUSION AND RECOMMENDATION**

The Board has not proven by a preponderance of the evidence that Dr. Govindan provided substandard care to Patients A, B, and C. Therefore, he should not be disciplined for practicing medicine in violation of law, regulations, or good and accepted medical practice.

***Statutory Basis for Discipline***

In Massachusetts, the Board is authorized to discipline physicians who have treated patients in violation of law, regulations, or good and accepted medical practice. G.L. c. 112, § 5; 243 CMR 1.01(2). A physician who practices medicine with “gross incompetence, or with gross negligence on a particular occasion or negligence on repeated occasions” is subject to discipline. G.L. c. 112, § 5; 243 CMR 1.03(5)(a)(3). Negligence occurs when a physician “fail[s] to meet generally accepted standards of care within the medical community.” *Bd. of Registration in Medicine v. Osei-Tutu*, Docket No. RM-07-64 (DALA, Jul. 8, 2008; adopted by Board, Feb. 25, 2009).

***Standard of Care***

All physicians must meet the standard of care, which is “the degree of care and skill of the average qualified practitioner, taking into account the advances in the profession.” *Brune v. Belinkoff*, 354 Mass. 102, 109 (1968). The standard of care is the level of care and skill that physicians in the same specialty commonly possess. *Palandijan v. Foster*, 446 Mass. 100, 104 (2006) (citing *Brune*); *McCarthy v. Boston City Hospital*, 358 Mass. 639, 643 (1971) (citing *Brune*). Evidence that other physicians may have treated a patient differently does not alone prove negligence, unless such treatment falls below the average competent professional judgment for physicians of the relevant specialty at the time in question. *See Grassis v. Retik*, 25 Mass. App. Ct. 595, 602 (1988) (citing *Brune*). Physicians may be required to choose one treatment from other medically appropriate alternatives that fall “within a reasonable range of medical judgment, taking into account the particular patient and circumstances.” *Barrette v. Hight*, 353 Mass. 268, 276 (1967).

***Witness Credibility***

Both the Board’s expert and Dr. Govindan’s expert are well-qualified to testify to the standard of care in interventional cardiology based upon their experience practicing in the field.  The Board contends that Dr. Resnic’s opinion should be given little weight because he is biased, based on his professional and financial interests, in opining that Dr. Govindan provided good medical care. Specifically, the Board presumes that because Dr. Resnic is Chair of Lahey’s cardiology division where Dr. Govindan has clinical privileges, he is motivated to testify in Dr. Govindan’s favor to avoid potential liability.

The Board cites two cases in support of its assertions. In the first one, a medical malpractice insurance case, the plaintiff requested the insurance records of three respondent-experts regarding their history of receiving payments from the malpractice insurer. *Thomas v. Sarfaty*, Case No. 9901361B, Middlesex Super. Ct., 13 Mass. L. Rptr. 17 (2001). The reason for the document request was to discern whether bias existed. The court ruled that the Plaintiff could request discovery materials on an expert witness’s financial interests to impeach the witness for potential bias. In the second case, the Board of Registration in Medicine found that a doctor’s expert witness was biased because the doctor and witness had a longstanding friendship, and the witness’s opinion was plainly contrary to the unrebutted evidence against the doctor. *Mtr. of Norman*, Case No. 89-16-SU (Bd. of Registration in Medicine, Jun. 6, 1990).

Here, Dr. Resnic and Dr. Govindan have a professional relationship. They do not socialize or refer to one another as “friends.” Dr. Govindan does not work for Dr. Resnic, and the Board has not proven that either doctor received payments for each other’s medical services. It is inevitable that Dr. Resnic has an interest in cardiologists practicing within the standard of care when they are exercising privileges at Lahey. To the extent that Dr. Resnic may have an appearance of bias, based upon a professional relationship with Dr. Govindan at Lahey, his testimony for each patient demonstrates that any such potential bias has not detracted from the quality and credibility of his opinion.I will determine the weight of Dr. Resnic’s testimony based on his explanation for each patient at issue, rather than give no weight to his testimony at all. *See Assessors of Pittsfield*, 329 Mass. 359, 361 (1952) (witness’s bias “goes only to his credibility, and is not a reason for exclusion of his testimony.”)

***Patient A***

The Board alleges that Dr. Govindan violated the standard of care in his treatment of Patient A by failing to diagnose severe aortic stenosis on two occasions and by failing to follow up on the diagnosis appropriately.

The medical records indicate that Dr. Govindan diagnosed aortic stenosis. The dispute is regarding only the *severity* of stenosis in light of the patient’s signs and symptoms. An interventional cardiologist should consider severe aortic stenosis based upon the following factors: (1) a mean pressure gradient of 40 mm Hg or above, (2) a systolic murmur, especially at S2, (3) carotid pulse delays, *and* (4) clinical symptoms of chest pain and shortness of breath.

The Board alleges that Dr. Govindan missed his first opportunity to diagnose severe aortic stenosis by not documenting Patient A’s heart murmur during auscultation on September 17, 2014. The murmur the Board refers to is a Grade 2 systolic ejection murmur. The experts and Dr. Govindan agree that a Grade 2 murmur is one that “some might hear” and “some might not.” Dr. Weiner, a cardiologist who physically examined Patient A the day after Dr. Govindan’s examination, documented no murmurs. Two months later, a third cardiologist, Dr. MacNaught, noted the patient had a Grade 2 systolic ejection murmur during auscultation. The Board contends that because Dr. MacNaught heard Patient A’s murmur, Dr. Govindan’s assessment of “no murmurs” two months earlier was below the standard of care. I conclude that in not discerning the patient’s murmur, Dr. Govindan did not violate the standard of care, given that some cardiologists can hear a Grade 2 murmur and others cannot.

The Board further argues that Dr. Govindan’s failure to detect a Grade 2 murmur during his physical examination of Patient A resulted in a missed diagnosis of severe aortic stenosis. I find that the Board did not present sufficient evidence that the ultimate diagnosis of a Grade 2 systolic ejection murmur was more likely than not diagnostic of severe aortic stenosis. This kind of murmur is just one of many factors that *may* be attributed to severe aortic stenosis. As of September 17, 2014, there was no other medical evidence besides a murmur that would have prompted the average interventional cardiologist to evaluate the patient for severe aortic stenosis.

The Board also alleges that Dr. Govindan provided substandard care because he did not accurately document Patient A’s pressure gradient during his diagnostic catheterization onSeptember 17, 2014. Dr. Govindan concedes that he made an error in documenting no pressure gradient in his catheterization procedure note.

Another part of the procedure record reflects that Patient A did have a pressure gradient. Dr. Lawson initially calculated a *peak-to-peak* gradient of 40 mm Hg. Later in his testimony, Dr. Lawson conceded that the standard of care for calculating a pressure gradient in 2014 was to compare the *mean* pressure readings of the left ventricle and the aorta. A mean gradient of 40 mm Hg and above indicates severe stenosis. Using individual pressure readings captured during the catheterization and applying the mean gradient calculation, Dr. Resnic and Dr. Lawson both estimated the patient’s mean pressure gradient as less than 40 mm Hg. This does not support a diagnosis of severe aortic stenosis.

Based upon the estimates that both expert witnesses made, even if Dr. Govindan had noted Patient A’s pressure gradient accurately, the value would be diagnostic for *moderate* aortic stenosis, not severe stenosis. Therefore, while Dr. Govindan made a documentation error in writing “no gradient,” the Board has not shown that this error was a violation of the standard of care by reason of missing a diagnosis of severe aortic stenosis.

There is, overall, a lack of evidence that Dr. Govindan missed a diagnosis of severe aortic stenosis. Patient A had some factors indicating aortic stenosis and Dr. Govindan noted the presence of stenosis in his catheterization procedure note. On the other side of the ledger, however, Dr. Lawson testified that it was highly unlikely that Dr. Govindan could have passed the pigtail catheter through Patient A’s left ventricle in 90 seconds if Patient A was severely stenosed. Generally, the narrower an artery is, the more time it takes to pass a pigtail catheter through the artery. The record does not reflect carotid pulse delays. Therefore, aside from chest pain and what was likely a Grade 2 systolic ejection murmur, there is no evidence that the patient had severe aortic stenosis when he saw Dr. Govindan in September 2014.

The Board also alleges that Dr. Govindan should have ordered a follow-up echocardiogram to examine the potential causes of Patient A’s chest pain and the pressure gradient. Dr. Govindan conducted an echocardiogram of this patient on October 1, 2014, about two weeks after the catheterization revealed a pressure gradient. The patient continued to experience chest pain during cardiac rehabilitation and was referred to Dr. MacNaught for further workup in November 2014. Dr. MacNaught performed a diagnostic catheterization. While Dr. MacNaught noted the patient’s “*known* severe bicuspid aortic valve stenosis,” he did not explain the connection between this diagnosis and the patient’s symptoms or the results of his diagnostic catheterizations. He did not discuss the results of the echocardiogram performed by Dr. Govindan, and did not order a follow-up echocardiogram.

The Board also pointed to the fact that upon Dr. MacNaught’s recommendation, the patient underwent a valve replacement, alleging the surgery was a consequence of Dr. Govindan’s failure to diagnose severe aortic stenosis. However, when asked to identify any of Dr. MacNaught’s references to diagnosing severe aortic stenosis, Dr. Lawson admitted there were none. (Lawson I: 200-201.)

However, the issue before me is not simply whether or not Patient A was diagnosed with severe aortic stenosis, but whether Dr. Govindan’s actions or omissions violated the standard of care to diagnose this condition. A single medical record notation of “known” severe stenosis does not explain how Dr. Govindan’s conduct violated the standard of care expected of the average interventional cardiologist in diagnosing severe aortic stenosis.

For the reasons stated, I find that the Board did not prove by a preponderance of the evidence that Dr. Govindan provided substandard care to Patient A by failing to diagnose severe aortic stenosis.

***Patient B***

The Board alleges that Dr. Govindan violated the standard of care in his treatment of Patient B by introducing a blood clot into the artery during his cardiac intervention procedure.

Patient B’s primary cardiologist referred him to Dr. Govindan for a diagnostic angiography, which revealed artery stenosis. Dr. Govindan proceeded with a percutaneous intervention, using a guidewire catheter to place a stent in the narrowed artery. There is no dispute as to two facts: (1) in the course of Dr. Govindan’s intervention, a blood clot formed in Patient B’s artery; and (2) Dr. Govindan addressed the clot so that the patient’s condition was stabilized and then completed the percutaneous intervention. Both experts testified that the standard of care required two critical steps prior to placing the catheter in the artery: (1) administer an anticoagulant to the patient prior to the procedure, and (2) aspirate and flush the catheter with saline to minimize risk of a clot forming in the catheter.

There is no dispute that Dr. Govindan took the first step – he administered an anticoagulant to the patient prior to the intervention. In terms of cleaning the catheter, Dr. Govindan testified that this process became automated after performing several thousand catheter-based interventions in his career. Dr. Resnic characterized Dr. Govindan as meticulous. He also testified that the records show that the catheter’s pre-injection waveform was *not* indicative of an occlusive blood clot. (Resnic II: 51-52.) One would expect a lack of waveform if an occlusive clot formed in the catheter before Dr. Govindan inserted it into the artery. *Id.* Additionally, Patient B had anemia and diabetes. The experts agreed that both conditions increased the likelihood of Patient B developing a blood clot.

Dr. Govindan argues that Dr. Lawson’s “surmise” that the clot formed from inadequate catheter preparation fails the preponderance of the evidence standard. (Lawson I: 210). “Surmise” is a synonym for guess or conjecture.[[6]](#footnote-6) Dr. Govindan is correct that when an expert’s opinion is expressed as a guess, conjecture, or speculation, that opinion fails to meet the preponderance of the evidence threshold. *Toubiana v. Priestly*, 402 Mass. 84, 91, 520 N.E.2d 1307 (1988) (expert’s word “foreseeable” was a “mere guess or conjecture” and thus given no evidentiary weight); *King’s Case*, 352 Mass. 488, 491-92, 225 N.E.2d 900 (1967) (“mere speculation or a guess from subordinate facts” was *not* preponderance of the evidence); *see* *also* *Commonwealth v. Kobrin*, 72 Mass. App. Ct. 589, 893 N.E.2d 384 (2008) (“conjecture or surmise” insufficient when determining the truth of a material fact.)

The fact that a blood clot formed during acatheterization, by itself, is not a violation of the standard of care. Without debating whether Dr. Lawson’s opinion was conjecture or speculation, he made an educated guess that the only plausible explanation for the formation of a blood clot was that Dr. Govindan failed to properly prepare the catheter. He did not consider other possible explanations for the formation of the clot. This explanation appears to assume negligence because of the occurrence of a bad outcome. It closely tracks the tort doctrine of *res ipsa loquitur* (“the thing speaks for itself”). That theory alone, even if it were applicable in Board discipline cases, only serves to establish a prima facie case, not prove the case by a preponderance of the evidence. *See Enrich v. Windmere Corp.*, 416 Mass. 83, 88 (1993) (“Res ipsa loquitur does not overcome the lack of evidence of the defendant’s negligence.”) Thus, Dr. Lawson’s opinion that the clot must have been caused by Dr. Govindan’s negligence is not reliable, and therefore I give it no weight.

For the reasons stated, the Board did not prove by a preponderance of the evidence that Dr. Govindan violated the standard of care by negligently causing a blood clot to form during the cardiac intervention of Patient B.

***Patient C***

The Board alleges that Dr. Govindan did not meet the standard of care in his treatment of Patient C by using an oversized stent during a percutaneous intervention that allegedly caused a full thickness artery perforation.

Patient C was referred to Dr. Govindan for severe narrowing of a long segment of his left anterior descending artery. To treat the stenosis, Dr. Govindan performed a percutaneous intervention, involving balloon stenting. He started with a 3.0 mm stent, which appeared too small for the artery. Then, Dr. Govindan used the balloon to inflate the stent to the next available size of 3.5 mm. Immediately thereafter, the patient had a full thickness artery perforation, which is the deepest type of artery tear. Dr. Govindan took emergency measures that saved the patient’s life, and then placed a 3.0 mm covered stent in the artery successfully.

The standard of carefor an interventional cardiologist choosing a stent size is to first estimate the size of the artery by imaging it, and then insert a balloon device to predilate the stent at a slightly lower diameter than the estimated artery size. The cardiologist next uses the balloon to gradually upsize the stent if it appears too small for the artery. Choosing and adjusting the stent size is a judgment call. The risks of using an undersized stent range from formation of a potentially fatal blood clot forming inside the stent to “late restenosis, which is a morbid but less often mortal outcome.” (Resnic II: 60). An oversized stent carries the risk of causing a small artery tear, and in rare cases it can cause a full thickness tear. (Lawson I: 179-180, 211; Resnic II: 64-67).

The issue that remains is whether the decision to use a 3.5 millimeter stent was a reasonable judgment call under the circumstances, including the location of the affected artery and the patient’s comorbidities. The procedure notes show that the 3.5 mm stent was oversized for Patient C’s artery by at most 0.5 millimeters, because it was between the inflation from 3.0 mm to 3.5 mm that the tear occurred. Dr. Resnic explained that a patient’s artery does not fall neatly within 0.5 millimeter increments, and arteries adjust slightly, but the adjustment has a limit.

While a full thickness tear can be caused by an oversized stent, such a sudden and severe tear is more likely due to sharp calcium deposits dislodging and piercing the artery wall. Patient C’s medical records do not document “calcification” or “calcium deposits” in the arteries, and there was no visible calcium in the pre-intervention cardiac imaging films. However, in 2014, when Dr. Govindan performed the intervention, the absence of calcium in imaging films did not preclude the possibility that the artery contained calcium deposits. (Resnic II: 65-66.)

Dr. Lawson believed the oversized stent contributed to the artery perforation, and classified the use of an oversized stent as a “high probability” of Dr. Govindan’s substandard care. (Lawson I: 176, 183-184.) Again, Dr. Govindan makes a detailed argument based on Dr. Lawson’s use of the words “surmise” and “probability,” and argues that these words diminish the evidentiary weight of Dr. Lawson’s opinion. For the purpose of determining whether testimony is reliable, the words themselves are not as critical as the substance of Dr. Lawson’s opinion and the context in which he expressed it. (See discussion regarding Patient B, above supra.)

Here, Dr. Lawson concluded that Dr. Govindan’s use of an oversized stent was substandard care, based solely on the occurrence of an artery tear right after the stent’s inflation to 3.5 mm. However, the artery tear may have been unrelated to the stent placement. There is no evidence that the stent caused the tear, even if it occurred right after the stent was inflated. While the tear was a severe and rare complication that can be caused by an oversized stent, Dr. Lawson’s reasoning does not establish that Dr. Govindan’s choice of stent size was contrary to acceptable practice for the average interventional cardiologist. Rather, Dr. Govindan chose a stent that he reasonable believed would be the most effective, and that choice was within the standard of care.

Therefore, the Board did not prove by a preponderance of the evidence that Dr. Govindan provided substandard care to Patient C.

**CONCLUSION**

Based on the evidence presented at the hearing, the Board did not prove by a preponderance of the evidence that Dr. Govindan was negligent in his care of Patients A, B, and C. While the complications that arose in the cases of Patient B and Patient C were life-threatening and rare, the mere occurrence of these events did not prove negligence on Dr. Govindan’s part. Therefore, I recommend that the Board not impose discipline on Dr. Govindan.

DIVISION OF ADMINISTRATIVE LAW APPEALS

Signed by Kenneth J. Forton

Kenneth J. Forton

Administrative Magistrate

DATED: April 28, 2021

1. Citations to the hearing transcripts will use the following format: [Name of Witness] [Transcript Volume]: [Page Numbers]. [↑](#footnote-ref-1)
2. Carotid pulse is felt on either side of the front of one’s neck below the jaw. https://medlineplus.gov/ency/imagepages/19386.htm. [↑](#footnote-ref-2)
3. American Heart Association, https://www.heart.org/en/health-topics/heart-attack/diagnosing-a-heart-attack/electrocardiogram-ecg-or-ekg. An EKG consists of placing “up to 12 sensors (electrodes)” on the patient’s chest, arms, and legs, with wires connected to a monitor that produces heart electrical signals on the computer readout. MayoClinic, https://www.mayoclinic.org/tests-procedures/ekg/about/pac-20384983. [↑](#footnote-ref-3)
4. Cleveland Clinic, https://my.clevelandclinic.org/health/diagnostics/16947-echocardiogram#:~:text=An%20echocardiogram%20(echo)%20is%20a,pumping%20action%20of%20the%20heart. [↑](#footnote-ref-4)
5. Citations to the medical records will be cited by the Petitioners’ Bates numbers omitting the placeholder zeroes that precede each Bates number. For instance, “000260” is cited as “260.” Exhibit 1 comprises medical records for Patient A. Exhibit 2 comprises medical records for Patient B. Exhibit 3 comprises medical records for Patient C. Thus, medical citations will be: [Ex. Number: Bates Page Numbers]. [↑](#footnote-ref-5)
6. Merriam-Webster Thesaurus, *Surmise*, https://www.merriam-webster.com/ thesaurus/surmise. [↑](#footnote-ref-6)