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LETTER FROM THE EDITOR

Dear readers:

Welcome back to the first edition of *ReFlections* for 2013. As always, we hope to present for your reading pleasure topics that are both interesting and relevant to your jobs in insurance medicine and underwriting. This edition has two objectives. The first, covered in two separate articles, is to discuss and review concepts relating to the vascular system. I have written the first article that deals with coronary artery disease and endothelial health. For our second article in this edition, we are fortunate to have a guest writer. Dr. Dave Rengachary is a general neurologist in St. Louis. His prior experience in the Primary Stroke Center of the Missouri Baptist Medical Center has given him considerable experience in the management of this disease. We are very pleased that he is willing to share his insights with us.

The second focus of this edition of *ReFlections* has to do with RGA's ongoing interest in Electronic Medical Records. Sue Wehrman continues to provide us with updates on EMRs and enlightens us on what these records will mean to us in the future of underwriting.

I hope you enjoy the contents of *ReFlections*, and we welcome any comments that you may have.

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CONCEPTS IN CORONARY ARTERY DISEASE

By J. Carl Holowaty M.D., D.B.I.M.

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Coronary artery disease (CAD) underwriting is a cornerstone of medical underwriting. As a major cause of death claims in the life insurance industry, it is vital that risk assessment for this impairment is as accurate as possible, especially in a highly competitive environment.

Fortunately a great many predictive tests as well as diagnostic tests are currently readily available, both clinically and for the underwriting process. Insurance laboratory blood tests such as lipid profiles and NT-ProBNP provide valuable insights into the probability that CAD may be present, and other insurance tests such as electrocardiograms provide additional insight into cardiac risk. In cases where investigations for cardiac symptoms such as chest pain have been conducted, the available underwriting information often includes perfusion studies, stress ECHOCardiograms and angiogram reports.

In spite of all this possible evidence to help diagnose and risk stratify CAD, there are still problematic cases. One such instance is a situation where

an applicant has typical chest pain, but diagnostic testing, including angiograms are negative. What is the outcome of such cases? Is the angiogram truly a 'gold standard' that can completely rule in or rule out CAD? Where does such an applicant fit into a risk assessment profile?

Another important issue to consider is the fact that, in spite of all the available predictive and diagnostic tests, CAD deaths still occur all too frequently in applicants who have favorable test results. Perhaps there are other risk factors or tests that can provide more insight and guidance into an individual's risk of death from CAD.

In a clinical study entitled "Low Diagnostic Yield of Elective Coronary Angiography" by R. Patel et al. (N Engl J Med 362;10 March 11, 2010), the authors concluded that "In this study, slightly more than one-third of patients without known disease who underwent elective cardiac catheterization had obstructive coronary artery disease." While they were illustrating what they felt to be a low diagnostic yield for angiography, it might be more important to consider that a significant number of these types of individuals without known disease could in fact be insurance candidates, and should they have had favorable insurance screening results, they might indeed qualify for the best rates of insurance.

Digging a bit further into the data from this study shows that, initially, almost 2 million applicants from the American College of Cardiology National Cardiovascular Data Registry between 2004 and 2008 were considered. Any individuals with histories of prior myocardial infarction, percutaneous intervention, coronary artery by-pass graft, cardiac transplantation, acute cardiac events, or other indications for diagnostic catheterization were excluded. This left a total of almost 400,000 individuals in the study, with a median age of 61. Of these:

- 53% were males, 47% were females
- 27% had diabetes
- 70% had hypertension
- All of the patients had elective catheterization
- 89% also had non-invasive testing

The results showed that 38% had obstructive lesions, i.e. > 70% obstruction in any epicardial vessel or > 50% of the left main.

Of these patients with obstructive lesions:

- 47% had 1-vessel disease
- 30.5% had 2-vessel disease
- 22.5% had 3-vessel disease

As one might expect, the rates of obstruction were highest in:

- Males
- Older people
- Diabetics
- Former users of tobacco
- Hypertensive people
- People with dyslipidemia
- People with peripheral vascular disease
- Those with positive non-invasive tests
- Those with higher Framingham scores
- Those with typical cardiac pain

Further evaluation of the studied population showed the distribution of population and the results according to their Framingham risk score.

- Of the population studied:
 - 29% were low risk
 - 55% were intermediate risk
 - 16% were high risk
- Of the group found to have obstructive disease:
 - 13.5% were low risk
 - 59.4% were intermediate risk
 - 27.1% were high risk
- Of the group found to have no obstructive disease:
 - 38.6% were low risk
 - 52.4% were intermediate risk
 - 13.5% were high risk

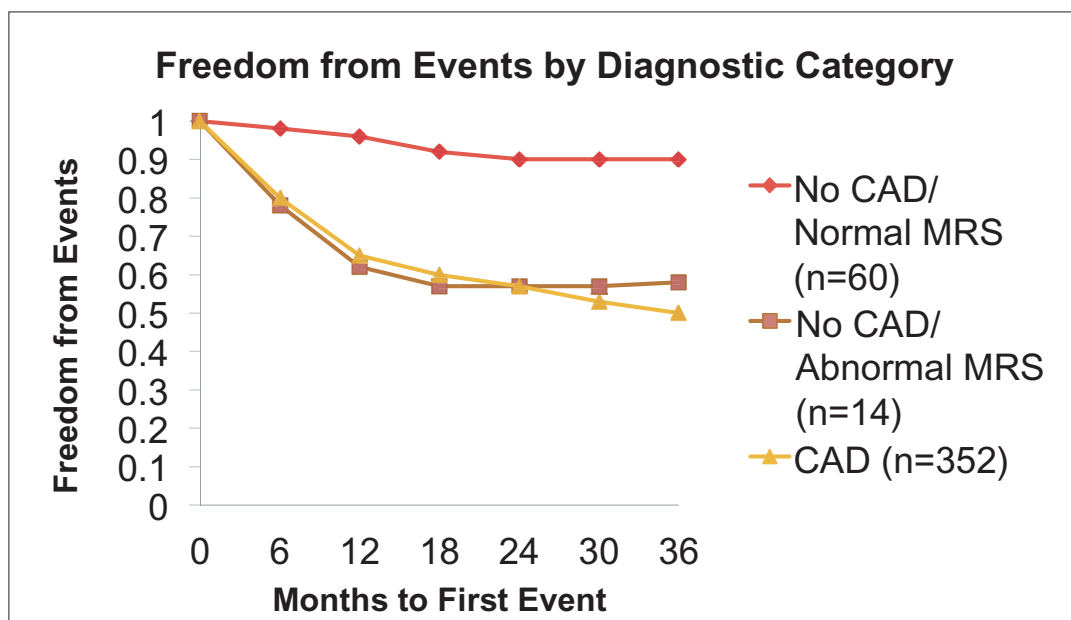
This study suggest that, while the Framingham criteria, upon which much of our risk selection for Preferred products is based are generally accurate, there are clearly individuals within the low-risk category that may not belong in Preferred risk pools, since at least some of them have obstructive CAD. The use of angiograms to risk-stratify this group in an insurance population is of course both completely impractical as well as ill-advised by any measure. Alternative evaluations may be possible however. This will be discussed further later in this article.

A second study, entitled “Prognosis in Women with Myocardial Ischemia in the Absence of Obstructive Coronary Disease.” by B.D. Johnson et al. (Circulation 2004; 109:2993-2999) evaluated women with signs and symptoms of myocardial ischemia in the absence of CAD. They found that 50% of women with typical chest pain do not have CAD as determined by angiography, whereas only 17% of men with typical chest pain do not have CAD by angiography. This condition has been referred to as cardiac Syndrome X, and it is a prognostic challenge for both clinicians and underwriters.

In this study, phosphorus-31 nuclear magnetic resonance spectroscopy (MRS) was used to monitor changes in energy consumption in the myocardium during stress. Evidence of reduced high-energy phosphates during stress is suggestive of myocardial ischemia. The myocardium of the tested subjects was scanned both before and after stress and then compared.

- Three groups of women were evaluated (all had angiography):
 - Women with no CAD and normal MRS
 - Women with no CAD and abnormal MRS
 - Women with CAD

Cardiovascular events were measured over the three-year study period. These events included cardiovascular death, myocardial infarction, cerebrovascular accidents, unstable angina, and other vascular events including peripheral arterial thrombosis. The following graph illustrates the ‘freedom from event’ rates in each of these three groups.



The ‘freedom from event’ rate in those women with no CAD on angiography, but with an abnormal MRS, is very similar to those with proven CAD on angiograms. Fortunately, in those subjects with no CAD but with abnormal MRS, the events were not as severe as those with proven CAD. There were no deaths in this group, with most of the events being unstable angina. Nevertheless, in this situation, the lack of angiographic evidence of obstructive lesions in the presence of typical chest pain should not be used as a lack of consideration for possible additional mortality over a longer period of time.

The authors of the study concluded that cardiac tests such as EKGs, myocardial perfusion testing, ECHOCardiograms and angiography all have limited utility due to technical artifacts particular to women.

They further concluded that MRS testing in women is useful in displaying myocardial ischemia, but probably most useful in the evaluation of anterior wall ischemia, and that it can predict the likelihood of future events such as unstable angina in a population of women with typical chest pain and no known CAD.

A third study, entitled “Stable Angina with no Obstructive Coronary Artery Disease is Associated with Increased Risks of Major Adverse Cardiovascular Events”, by L. Jespersen et al. (European Heart Journal 2012 33;734-744) evaluated 11,000 Danish patients with stable angina between 2000 and 2009. The study group included 4711 women and 6512 men who had undergone angiography. The studied population excluded those with a prior myocardial infarction, PCI or bypass procedure. The study considered results in both men and women. The criteria for ‘obstructive’ CAD were:

- No obstruction – 0% stenosis in all coronary arteries
- Diffuse non-obstructive CAD – 1 - 49% obstruction in any epicardial coronary artery
- Obstructive CAD – > 50% obstruction in any epicardial coronary artery

The results showed:

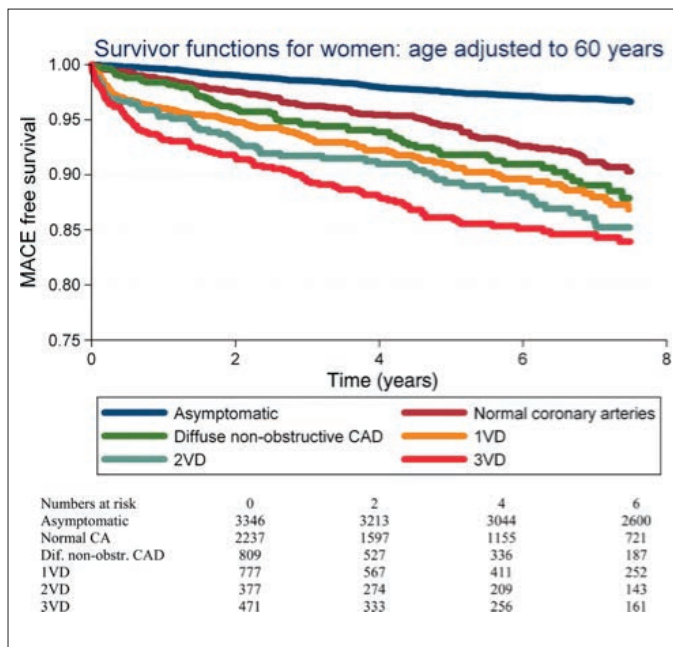
- Women
 - 48% of women with angina had normal angiograms
 - 17% had diffuse non-obstructive CAD
 - On average, women were 2.4 - 4.3 years older than men with the same degree of obstructive disease
- Men
 - 19% of men had normal angiograms
 - 14% had diffuse non-obstructive CAD

The study outcomes were cardiovascular mortality, MI, heart failure and stroke (termed MACE).

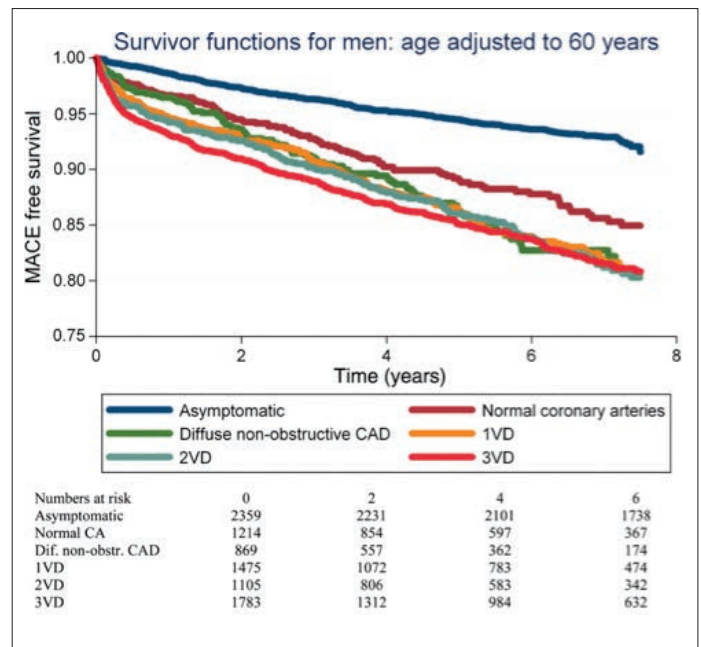
A graphic illustration of major adverse cardiovascular event-free survivor functions for men and women is shown in the two graphs below:

Major adverse cardiovascular event-free survivor functions for men and women

Women



Men



Jespersen L et al. Eur Heart J 2012;33:734-744

The authors of the study concluded that both men and women with stable angina pectoris or diffuse non-obstructive CAD have increased mortality risk. Those with normal arteries had 50% greater risk of MACE than the reference population. Those with diffuse non-obstructive CAD had an 82% higher risk of MACE. They also indicated that the increased risk is approximately the same for men and women.

These three studies, among other things, may serve to illustrate that a normal or near-normal angiogram in the presence of typical chest pain does not rule out all adverse CAD risk. This raises the question of why people have cardiac pain. This can be broken into several broad categories:

- Mechanism in obstructive CAD
 - Coronary artery blood supply is limited by atherosclerotic plaque
 - Coronary micro-vessels dilate in a compensatory fashion
 - Pain receptors in the myocardium react to ischemia
- Possible explanation in non-obstructive CAD
 - Epicardial arteries are normal (accounting for normal angiographic appearance)
 - Inappropriately increased vascular tone of the coronary micro-vessels leads to myocardial hypoperfusion and stimulation of the pain receptors in the myocardium
- Another possible explanation
 - Epicardial arteries are normal
 - The tone of coronary micro-vessels is normal
 - The sensitivity of pain receptors in the myocardium is increased, leading to pain typical of ischemia

Another issue to consider is whether the cardiac pain originates external to the epicardial vessels or the micro-vessels. Some non-CAD causes of cardiac pain are listed below.

- Aortic stenosis
- Hypertrophic cardiomyopathy
- Peri-myocarditis
- Rhythm disturbances
- Heart failure

Having delineated some of the potential risks of giving too much credit for normal angiogram results, it is also important to consider other factors that can help

with risk stratification for CAD. These factors could potentially either improve or worsen the traditional assignment of coronary risk based on tests such as ECHOCardiograms, perfusion studies or angiograms.

The first consideration is the role of the endothelium in coronary artery health. Endothelial dysfunction has been termed “the ultimate risk of the risk factors” by the Mayo Clinic. The endothelium is a mono-layer of cells that line the interior surface of all blood and lymphatic vessels. This layer of cells lines the entire circulatory system, including the capillaries, arteries, great vessels, and heart (where it is termed the endocardium).

The healthy endothelium provides many important functions in the body. It provides part of the mechanism that controls the flow of fluids through vessel walls. This function is highly specialized in the blood vessels of the glomeruli within the kidneys. The endothelium also plays a role in hemostasis, neutrophil recruitment, the immune system and neo-angiogenesis.

Endothelial cells are active in prevention of blood clotting with blood vessels. They do this by producing heparan sulfate, which activates anti-thrombin to inhibit the coagulation cascade, platelet aggregation, and leukocyte adhesion. *Dysfunction* of the endothelial layer is important in the formation of atheromatous plaque and the sequelae of plaque rupture.

Perhaps even more important is the ability of the endothelium to control blood vessel ‘tone’, i.e., the normal expansion and contraction of the blood vessels during the cardiac cycle as well as during events that put stress on parameters such as blood volume and blood pressure. The function of vasodilation is controlled through the endothelium by their production of nitric oxide, which mediates the response of the microvessels of the myocardium to relax in the presence of acetylcholine.

Another important role of the healthy endothelium in regard to CAD is its ability to prevent smooth muscle cell proliferation in arteries. Many of the functions of the endothelium are controlled and coordinated by autocrine and paracrine substances that are produced by the individual cells.

When the endothelium is not functioning properly, it can obviously affect all of the above noted functions. Endothelial dysfunction is a systemic pathological state of the endothelium. Among other things, it represents an imbalance between vasodilating

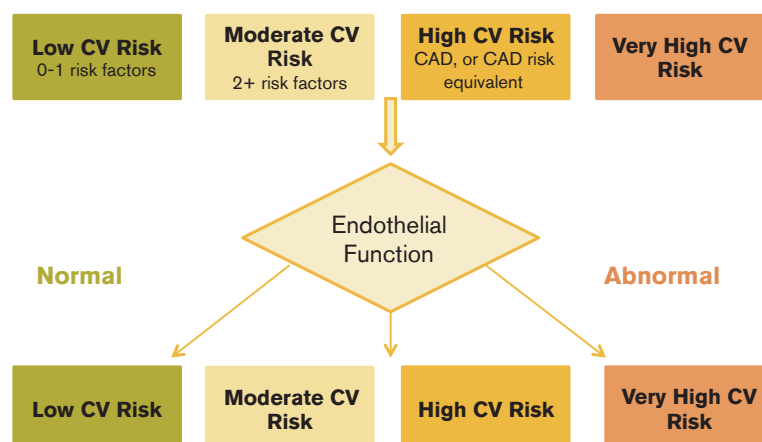
and vasoconstricting substances that control the tone of the endothelium. It is associated with hypertension, hypercholesterolemia, diabetes, septic shock, Bechet's disease and chronic renal failure. It is part of the physiological process that leads to coronary artery disease and other atherosclerotic conditions. It is thought to be caused by:

- Smoking
- Air pollution
- Hypertension
- Diabetes (specifically hyperinsulinemia)
- Hypercholesterolemia
- Obesity (particularly visceral adiposity)
- Diet
- Mental stress

Physiologically, endothelial dysfunction is characterized by a pro-inflammatory and pro-thrombotic state, as well as reduced vasodilation. This state is very consistent with what we know about coronary artery disease. The systemic manifestations of endothelial dysfunction include such entities as stroke/TIAs, sleep apnea, dementia, renal failure, myocardial infarction, metabolic syndrome, erectile dysfunction, and claudication.

Clearly, endothelial dysfunction is important to recognize and treat in order to prevent the onset and progress of its associated conditions. Fortunately, there are some relatively simple test procedures that can shed some light on its existence. One non-invasive method to evaluate endothelial dysfunction is the use of brachial blood flow measurement to assess Flow Mediated Dilation (FMD) of arteries. This technique assesses endothelium-dependent response to shear stress. This is accomplished with the use of a sphygmomanometer, which can be positioned on the arm, either above or below the elbow. A B-mode ultrasound is then used to measure the diameter of the artery in the arm both proximal and distal to the cuff, both before and after inflation. If the endothelium is functioning normally, inflation of the cuff should result in a dilation of the artery distal to the cuff. Failure to do so is suggestive of endothelial dysfunction. The reason the measurement of endothelial function is important is that it may be possible to use the results of the testing to re-classify cardiovascular risk. This has been suggested by L. Lind et al. in the study entitled "Endothelial Function in Resistance and Conduit Arteries and 5-Year Risk of Cardiovascular Disease (Circulation 2011 Apr 12;123(14):1545-51), as well as J. Yeboah et al. in a study entitled "Predictive Value of Brachial Flow-Mediated Dilation for Incident Cardiovascular Events in a Population Based Adults Free of Cardiovascular Disease at Baseline: The Multi-Ethnic Study of Atherosclerosis" (Circulation 2008; 118:A-30). The diagram below illustrates this potential re-classification of risk. While there may be other techniques developed to assess FMD, this use of brachial flow measurement has shown some value in a simple non-invasive test.

Endothelial Function Can Reclassify the Risk of the Patients



Ultimately of course, having detected endothelial dysfunction, it is important to treat it effectively and return the endothelium to a healthy state. Fortunately, there are already several effective treatments available. Some of the treatments include smoking cessation, weight reduction, exercise, lipid control, treatment of hypertension with ACE inhibitors, ARBs and calcium channel blockers, the use of statins, and omega-3 fatty acids in their natural form.

The final concept for discussion in this article is that of Fractional Flow Reserve (FFR). This is a technique used in cardiac catheterization to measure pressure differences across areas of coronary artery stenosis. This is very useful in determining if a stenotic obstruction is actually causing myocardial ischemia. This measurement is done during standard angiography by including a sensor at the tip of the catheter. This sensor measures blood pressure, temperature and blood flow. It is best performed during maximal blood flow in the coronary arteries. While it is not possible to stimulate this maximal blood flow by exercise, it can be simulated by the use of medications such as adenosine or papaverine. Measurements of blood pressure, temperature and flow are taken when the catheter tip is slowly pulled back through an area of apparent obstruction and data is compared from areas distal and proximal to the obstruction. This data is expressed as an absolute number that compares the pressure distal to the obstruction to that proximal to the obstruction. The numbers used to express FFR represent the approximate percentage of loss of blood flow across any area of stenosis. For example, an FFR of 0.5 represents a 50% drop in blood pressure. Studies have shown that an FFR of > 0.8 indicates that the obstruction is not causing significant loss in blood flow or pressure across the obstruction, whereas an FFR of < 0.5 is indicative of significant obstruction.

Some of the advantages of FFR are:

- It can be very useful to determine if there is a significant degree of collateral circulation around an area of tight stenosis
- Standard angiography can either under-estimate or over-estimate the degree of obstructive coronary disease, but FFR does not
- FFR can be useful to decide whether or not a stenting procedure is likely to improve coronary artery blood flow (and potentially relieve symptoms)
- FFR may reduce the number of stents needed, as well as the associated cost and morbidity of the procedures

- FFR does not increase the duration or risk of the angiography procedure

The main drawback of the FFR is that while it substantially enhances the value of angiography, it does not provide any information about plaque stability.

Several clinical studies have validated the use of FFR. The DEFER study (2005) showed that, when the FFR was > 0.75 , there was no survival advantage to performing a stenting procedure. The risk of cardiac death or MI when the FFR was > 0.75 was $< 1\%$ per year. Stenting did not improve this risk.

The FAME study (Fractional Flow Reserve vs. Angiography for Guiding Percutaneous Coronary Intervention) was published in 2009. It evaluated whether the addition of FFR measurements to standard angiography can improve outcome. It concluded that routine measurement of FFR in patients with multi-vessel coronary artery disease who are undergoing PCI with drug-eluting stents significantly reduces the rate of the composite end-point of death, non-fatal myocardial infarction, and repeat vascularization at one year. The avoidance of unnecessary stents was felt to reduce the mortality and morbidity related to those devices.

Hopefully, the routine adaptation of FFR in angiography reports will prove useful for underwriters when assessing the associated risk from either single-vessel or multi-vessel coronary artery disease. ■

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STROKE EPIDEMIOLOGY: GLOBAL VARIANCE, SECULAR AND EMERGING TRENDS

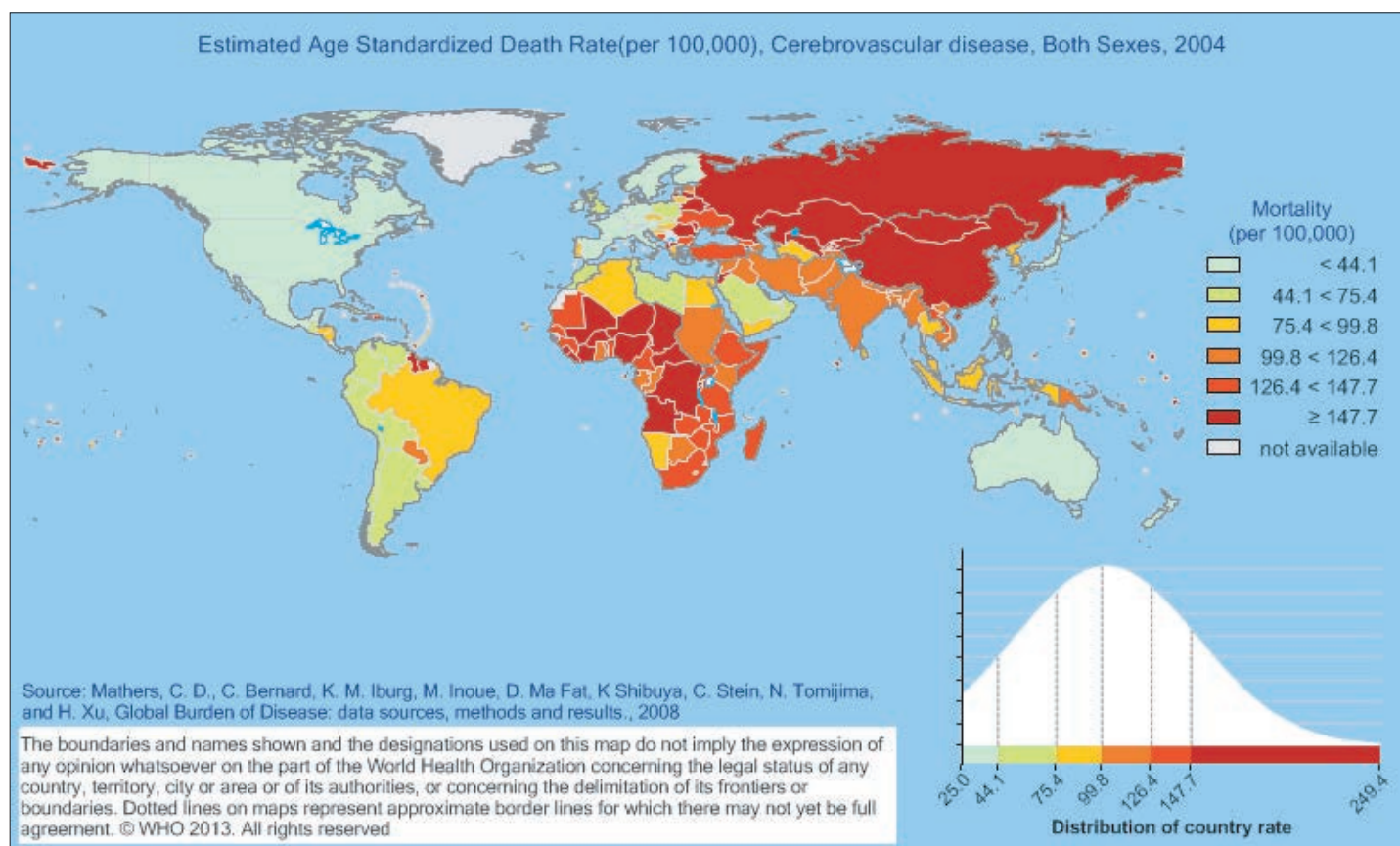
By **Dave Rengachary M.D.**

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Roughly 5.5 million deaths occur from stroke on a worldwide basis each year, making cerebrovascular disease the second-leading cause of death. In the United States, one stroke occurs every 40 seconds.¹ Mortality rates, however, tell only a small part of the story, as 80% of patients survive a first stroke, with roughly \$70 billion in annual health expenditures in the United States alone. Stroke and other neuropsychiatric disorders far outpace all other body systems in terms of years lived with a disability.²

Understanding trends in the morbidity and mortality of stroke will only be of increasing importance to the underwriter. Cerebrovascular disease is the sixth-leading cause of disease burden, and the WHO estimates that by 2030, it will jump to number four.

Figure 1 WHO world map showing estimated mortality from Cerebrovascular Disease based on 2004 data¹²



Source: Mathers C.D. et al. Global Burden of Disease: data, sources, methods and results, 2008

Predicting trends in stroke morbidity and mortality has been a daunting task given wide regional variance (Figure 1) that does not necessarily conform to expected cardiovascular trends and equally disparate capabilities in measurement and collection of stroke data.³ However, given that cerebrovascular disease is increasingly recognized as a treatable, but perhaps more importantly, preventable, phenomenon, a summary of trends in stroke epidemiology is in order.

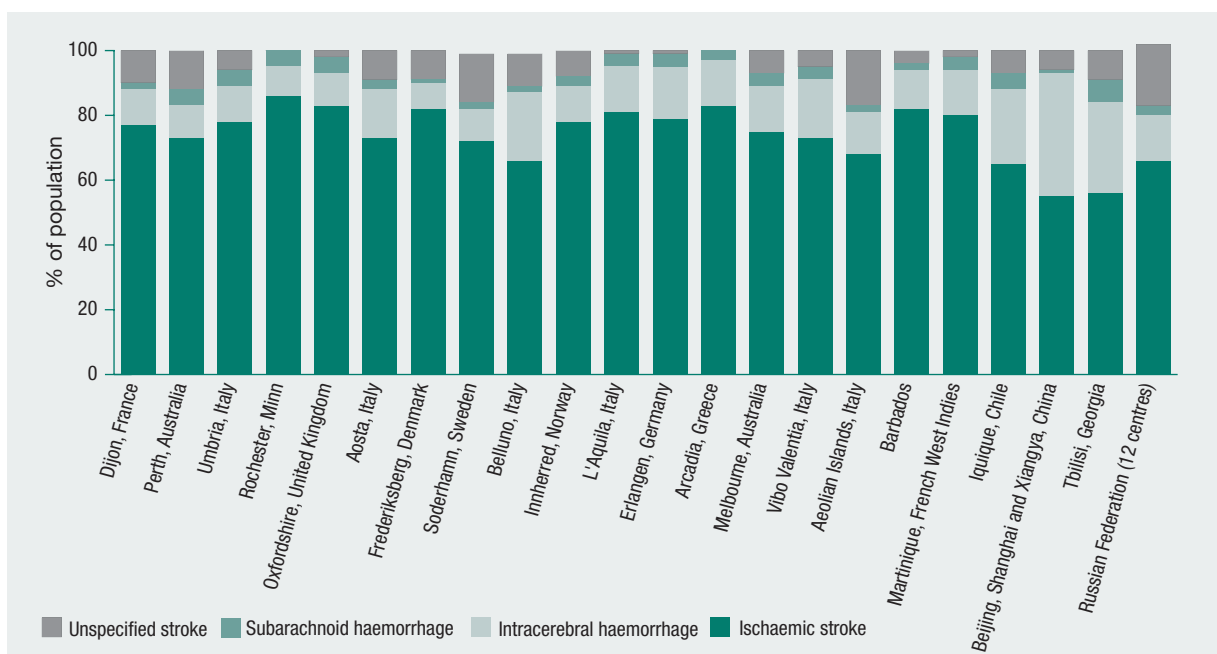
Definitions

It is first necessary to define a few terms. The term "stroke" refers to any disruption of blood supply to the brain producing a measurable neurologic deficit lasting more than 24 hours. Strokes are categorized as either ischemic, resulting from the propagation of a clot or occlusion of an artery from plaque, or *hemorrhagic*, referring to strokes that occur from rupture of an artery. *Subarachnoid hemorrhage* is a term reserved for hemorrhagic strokes that arise from the rupture of an aneurysm. Simply dividing strokes into subtypes provides the first opportunity to highlight the complex nature of stroke epidemiology. Significant regional variation is seen in various stroke subtypes (Figure 2). In the U.S., roughly 85% of strokes are ischemic, whereas 10-15% are hemorrhagic and 5% subarachnoid.¹ Studies have generally found a greater proportion of hemorrhagic strokes in Asia (20-30%), with higher smoking and blood pressure rates (and less access to blood pressure medications) theorized to explain the difference. A review of secular trends in stroke mortality from 1932 to 1999 in the U.K. found that the rate of mortality from ischemic stroke tended to parallel rates of coronary heart disease, while hemorrhagic strokes produced independent rates. Of note, overall mortality from hemorrhagic stroke has declined steadily while the rate of ischemic stroke peaked in the 1970s with a subsequent steady fall. A systematic review of 56 population-based studies from 1970 to 2008 analyzing worldwide stroke incidence found a clear decrease in the overall incidence of ischemic and hemorrhagic stroke, while the rates of subarachnoid hemorrhage remained steady.⁴ It becomes clear that, in terms of epidemiology, stroke subtypes should be thought of as entirely separate entities. The importance of the distinction is quickly seen in the different case fatality rates: 42% for intracerebral hemorrhage, 32% for subarachnoid hemorrhage, and 18% for ischemic stroke.⁵

Income Status

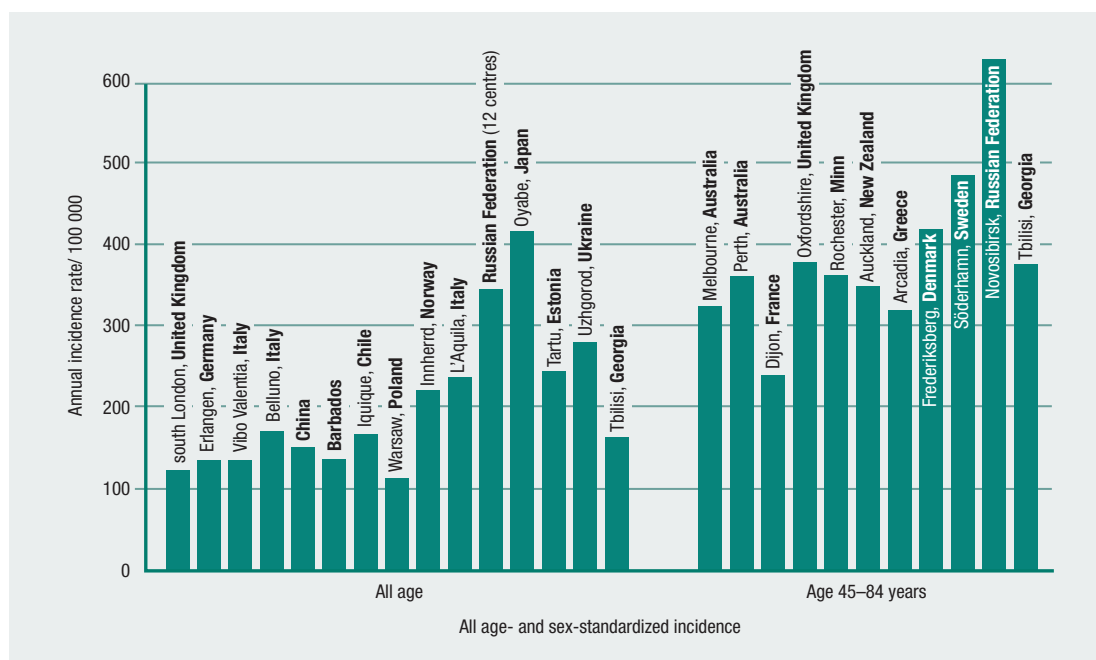
Unsurprisingly, there have been divergent secular trends in the incidence of stroke based on income status. Perhaps more sobering is the degree of divergence (Figure 3). In 2001, the WHO estimated that 85% of fatal strokes occurred in low- or middle-income countries. Global burden studies have concluded that such countries have over seven times rate of disability-adjusted life years (DALYs) than their high-income counterparts.³ From 1970 to 2008, the incidence of stroke in high-income countries has decreased by 42% but increased by more than 100% in low- to middle-income countries. For the first time, during the final study period (2000-2008) the incidence of stroke in lower-income countries exceeded those of higher-income countries, and by over 20%. Of the 3051 epidemiologic studies pooled for review, only 56 population-based studies met minimum inclusion criteria for completeness of data, highlighting the challenges of standardizing data sets.

Figure 2 Frequency of stroke subtypes in different populations¹³



Source: http://www.who.int/mental_health/neurology/neurodisolen/index.html

Figure 3 Regional Variance in Stroke Incidence¹³

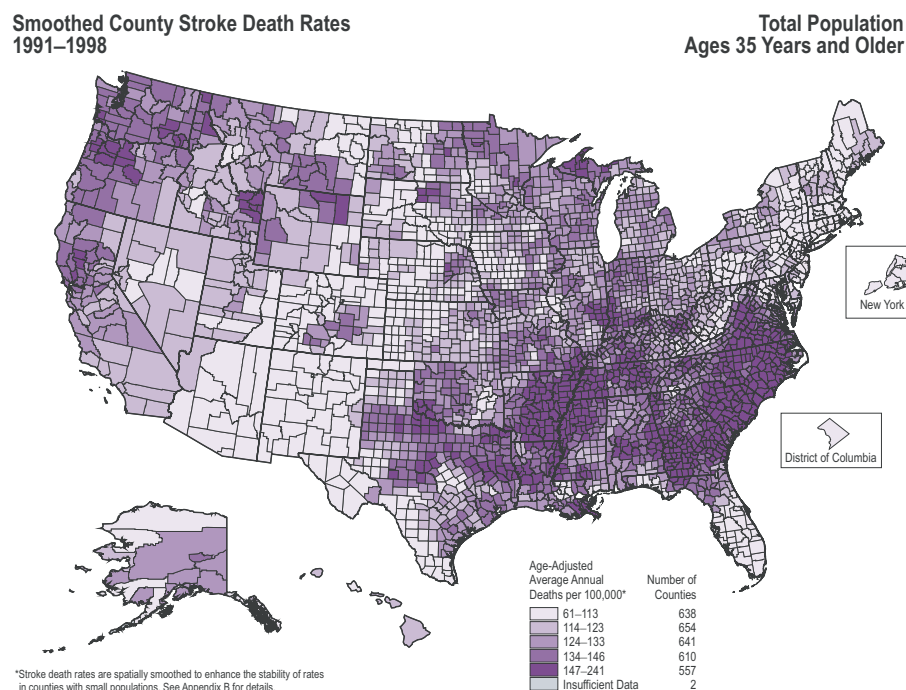


Source: http://www.who.int/mental_health/neurology/neurodiso/en/index.html

Geography

Nowhere is the importance of geographic influence on the mortality of stroke clearer than in the United States. The "stroke belt" refers to the high incidence of stroke in the Mississippi Valley and Southeastern United States and has been described by the CDC since 1962 (Figure 4).

Figure 4 Age-adjusted average (annual) deaths per 100,000¹¹



Source: Casper ML et al. *Atlas of Stroke Mortality: Centers for Disease Control and Prevention, 2003*

Unfortunately, the disparity has changed little in 50 years.⁶ Diabetes, hypertension, smoking, diet, and poor access to healthcare resources have been identified as potential contributing factors. Although ethnicity had long been assumed to play an important role, there is clearly more to the story. As an example, the stroke mortality rate among African Americans living in North Carolina is three times higher than those living in New York.⁷ In addition, Caucasians living in the stroke belt clearly have higher rates of stroke than age-adjusted counterparts elsewhere in the country.⁸

The WHO Global Burden of Disease project represented a comprehensive and complex effort to compare data from 112 registration systems across 94 countries.⁹ Stroke mortality, incidence, prevalence, and disability were all estimated. Relatively wide variance was found: There was a ten-fold variance in age- and sex-adjusted mortality rates when comparing the most- and least-affected countries. Western Europe and North America tended to be least-affected, while Russia, Eastern Europe, central Africa, and north Asia had the highest rates for stroke mortality. As would be expected, if not adjusted for age, overall mortality rates were actually higher in high-income countries secondary to longer life expectancies. Prevalence of traditional cardiovascular risk factor profiles (diabetes, hypertension, BMI, hyperlipidemia) tended to be less favorable in higher-income countries and actually served as relatively poor predictors of stroke mortality.

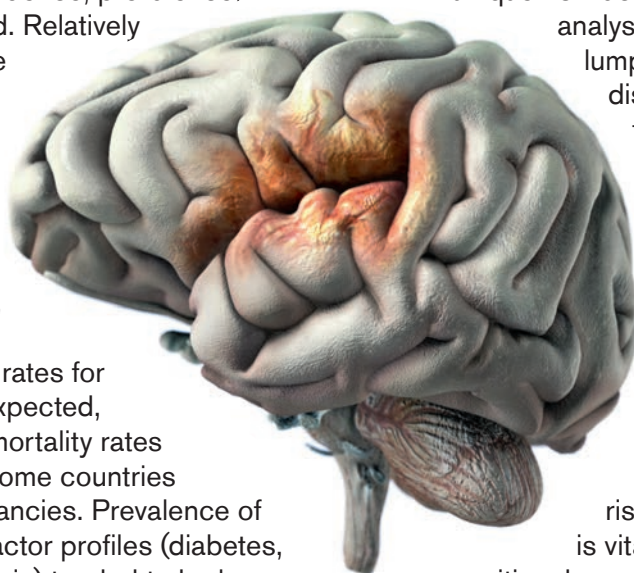
Gender

A review of 59 incidence studies across 19 countries by Appelros et al.¹⁰ found a somewhat higher-than-expected difference in stroke rates (33% higher incidence rate in men compared to the traditionally accepted 25-30% figure) as well as other findings suggesting a greater influence of gender than had been previously appreciated. Men also tend to have strokes at an earlier age (68.6 years for men and 72.9 for women).

Again it was clear that different subtypes of stroke behaved differently. As expected, ischemic and hemorrhagic stroke rates were higher in men. The rate of subarachnoid hemorrhage in women was higher; however, this did not reach statistical significance. Women also had a higher rate of cardioembolic strokes (strokes presumed to have arisen from the heart). It was clear, however, that although less common, strokes in women tended to be more severe, with higher case fatality rates (1.25 times higher than men).

Summary

From an underwriter's perspective, stroke should not be thought of as a single disease state but, instead, several separate disease entities, each of which entails unique risk factors, mortality, and morbidity analysis. Worse yet would be to lump stroke into "cardiovascular disease", as in many cases stroke trends can run independently of traditionally accepted vascular risk factors. It will be of critical importance both from an underwriting and public policy perspective to prepare for epidemic rates of stroke in low- and middle-income countries. "The continued implementation of population based and high risk stroke preventative strategies is vitally important to maintain the positive decrease in stroke incidence in high-income countries, and the immediate launch of stroke prevention programs, particularly blood pressure control and smoking cessation interventions, at the population and individual level, together with improved access to primary health care in low to middle income countries is needed. The time to decide whether or not stroke should be on governmental agendas in low- to middle-income countries has passed. Now is the time for action."²



References

1. Truelsen T, Begg S, Mathers C. The global burden of cerebrovascular disease. http://www.who.int/healthinfo/statistics/bod_cerebrovasculardiseasesstroke.pdf
2. Menken M, Munsat TL, Toole JF. The Global Burden of Disease Study: Implications for Neurology. *Arch Neurol*. 2000; 57:418-420
3. Mathers CD, Lopez AD, Murray CJL. The Burden of Disease and Mortality by Condition: Data, Methods, and Results for 2001. In: Lopez AD, Mathers CD, Ezzati M, et al., editors. *Global Burden of Disease and Risk Factors*. Washington (DC): World Bank; 2006.
4. Feigin V.L., Lawes C.M., Bennett D.A., Barker-Collo S.L., Parag V. Worldwide stroke incidence and early case fatality reported in 56 population-based studies: a systematic review (2009). *The Lancet Neurology*, 8(4), pp. 355-369.
5. Feigin V.L., Lawes C.M.M., Bennett D.A., Anderson C.S. Stroke epidemiology: A review of population-based studies of incidence, prevalence, and case-fatality in the late 20th century (2003). *Lancet Neurology*, 2(1), pp. 43-53.
6. Glymour MM, et al. "Birth and adult residence in the Stroke Belt independently predict stroke mortality". *Neurology*. 2009; 73:1858-65.
7. Morris DL, Shroeder EB. *Stroke Epidemiology*. Foundation for Education and Research in Neurological Emergencies. <http://www.uic.edu/com/ferne/pdf/strokeepi0501.pdf>
8. Stroke Belt Initiative: Project Accomplishments and Lessons Learned, National Heart, Lung, and Blood Institute, National Institutes of Health; reports on a conference that occurred in 1996.
9. Johnston S.C., Mendis S., Mathers C.D. Global variation in stroke burden and mortality: estimates from monitoring, surveillance, and modelling (2009). *The Lancet Neurology*, 8(4), pp. 345-354.
10. Appelros P, Stegmayr B, Terent A. Sex Differences in Stroke Epidemiology: A Systematic Review (2009). *Stroke*, 40, pp. 1082-1090.
11. Casper ML, Barnett E, Williams GI Jr., Halverson JA, Braham VE, Greenlund KJ. *Atlas of Stroke Mortality: Racial, Ethnic, and Geographic Disparities in the United States*. Atlanta, GA: Department of Health and Human Services, Centers for Disease Control and Prevention, 2003.
12. Mathers C.D., C. Bernard, K.M. Iburg, M. Inoue, D Ma Fat, K. Shibuya, C. Stein, N. Tomijima, and X. Hu. *Global Burden of Disease: data, sources, methods and results*, 2008.
13. *Neurological Disorders Public Health Challenges*. World Health Organization, Geneva, Switzerland 2006. http://www.who.int/mental_health/neurology/neurodiso/en/index.html.



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WHAT ARE ELECTRONIC DENTAL RECORDS?

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An Electronic Dental Record (EDR) contains everything traditionally stored in the paper chart, converted to digital format:

- Patient demographics
- Medical and dental histories, treatment plans, notes and updates
- Conversations about proposed treatment, risks and benefits of treatment, and alternatives
- Charts and study models
- Prescriptions, laboratory orders (including results), radiographs
- Consent forms, waivers, authorizations, referral letters and consultations, and other correspondence

EDR is a key technology that can improve the quality and efficiency of healthcare delivery. The benefits cited for adoption of technology in clinical medicine – quality improvement, outcomes improvement, patient safety, process efficiencies, cost reduction, and coordination of care – hold equally true in dentistry; however, adoption has been slow despite forecasted benefits. The dental profession has been reluctant to adopt; they are concerned they may be replacing an imperfect paper-based process with a cumbersome and largely untested electronic one. Therefore, medical and dental records have evolved differently and separately over time. As a result:

- There is poor communication among medical and dental providers;
- Data is duplicated and often inconsistent between medical and dental records;
- Structural barriers exist that make it difficult to coordinate medical/dental care.

Dentistry has been developing computerized standards since the early 1990s through the American Dental Association (ADA) National Standards Committee. It has set out a number of specifications and technical reports to ensure confidentiality, interoperability, and sound data architecture. At least nine different code sets have been identified for inclusion in the EDR.



Among them are SNODENT (Systemized Nomenclature of Dentistry); the vocabulary designed for electronic health and dental records. The ADA is charged with making SNODENT interoperable with the rest of the electronic health record. Currently, 38% of practices have digital patient records and another 34% are in the process of converting. Only 29% do not have electronic patient records.

Why are dental records important to overall patient health?

As EDRs and EHRs become integrated, dentists may find EHRs contain valuable data on their patients' medical conditions, current prescription medicines and potential drug interactions. Research has shown that more than 120 systemic diseases originate in the oral cavity. Oral disease has been associated with nutritional compromise, cancer, xerostomia (dry mouth), pneumonia, bacteremia, emphysema, brain abscess, heart problems, diabetes, surgery complications and mortality. Gum disease has been linked to premature birth, and infected dental tissues may cause periodontal disease that may in turn affect the neck, eyes, and brain. Oral disease increases risk factors for chronic diseases such as cardiovascular and cerebrovascular diseases, diabetes mellitus and respiratory disease (Rudman, W., Hart-Hester, S., Jones, W., Caputo, N., & Madison, M., 2010). For these reasons, dental information is valuable to overall patient care.

Dental records also have a forensic application known as Forensic Odontology which is the art and science of dentistry to resolve matters pertaining to the law. Applications of dental forensics include identification of human remains, assessment of bite marks, and the use of dental materials in the examination of evidence.

Technology systems currently available to dentists:

- **Electronic Dental Records (EDRs):** document medical and dental history, clinical examinations, periodontal screenings, problems and priorities, referral, release (and consent), insurance and prescription data.
- **Dental Practice Management (DPM) software:** provides several functions, including appointment scheduling, billing, accounting and reporting.
- **Digital Radiography:** these items instantly acquire and store images where they can be manipulated, viewed and transferred without using film.

How are EDRs regulated?

A few dental practice acts or regulations issued by U.S. state boards of dentistry specify requirements for dental records. However, most patient record-keeping requirements that apply to health care professionals are generic. In terms of the Health Insurance Portability and Accountability Act, dentists are considered “Covered Entities” under the Privacy and Security regulations.

Does the Certification Commission for Health Information Technology certify dental technology?

Yes – but finding a certified dental EMR is particularly challenging for dentists, as there are a limited number of certified products. Further, many of the requirements drafted for Meaningful Use do not specifically apply to oral health providers. Other challenges include:

- Absence of diagnostic, therapeutic, or decision support applications appropriate for dentists;
- Difficulty in meeting the minimum eligibility thresholds for Medicaid and cost to implement (the majority of dentists are solo practices); and
- Lack of proven interoperability between medical and dental records.

If providers meet the applicable MU requirements (15 core objectives plus five additional measures), they are eligible to receive up to \$63,750 for the adoption and meaningful use of electronic health records. ■

References

Charangowda, B. K. (2010). Dental Records: An overview. *Journal of Forensic Dental Sciences*.
Dykstra, B. (2012). *The economics of the digital dental record: Can you afford not to make the switch?* Dental Economics.

Frey, J. (2010). *Digital Dental Records on the Rise among Dentists*. Dental Practice Marketing and Management Blo.g

Rudman, W., Hart-Hester, S., Jones, W., Caputo, N., & Madison, M. (2010). *Integrating Medical and Dental Records: A New Frontier in Health Information Management*. AHIMA HIM Body of Knowledge.

Thyvalikakath, T. (2012). *HITECH Act and Dentists' Meaningful Use of Electronic Health Records*. Dental Informatics Blog.

U.S. Department of Health and Human Services. (2012). *What certified products are available for dentistry?* Health Information Technology and Quality Improvement.



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Susan L. Wehrman, Vice President, Electronic Health Record Initiatives, heads RGA's newly created Electronic Health Record (EHR) Initiatives area. This function conducts in-depth research and analysis of this evolving segment and monitors all activity in the U.S. and around the world, with the objectives of positioning RGA as an industry thought leader and better assisting clients with EHR-related issues.

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