The Use of Expected Value as an Aid to Decisions Regarding Anticoagulation in Patients With Atrial Fibrillation

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Background: The method described provides a rational means for determining whether to institute chronic anticoagulation to prevent stroke in patients with chronic atrial fibrillation under a variety of clinical circumstances.

Summary of Comment: The concept of expected value is used in conjunction with data from clinical studies to define the net value of anticoagulation to the patient. A full year of anticoagulation is warranted in patients with recent stroke or transient ischemic attack thought to be due to cardiogenic embolism who feel that stroke is a very serious event with nearly as much disvalue as death. If stroke has a lesser degree of negative value to the patient, or it is uncertain whether the stroke was in a large-vessel distribution, or it is uncertain whether a large-vessel distribution stroke was due to cardiogenic embolism, 6 months or less of anticoagulation may be warranted. Indefinite anticoagulation is justifiable in most patients with chronic atrial fibrillation without a history of stroke or transient ischemic attack but may be contraindicated in certain patients at extremely low risk for embolism and in patients who place a low value on stroke relative to death and who have a modest increase in risk for fatal hemorrhage.

Conclusions: The method described provides a means readily usable by clinicians to make anticoagulation decisions in patients with chronic atrial fibrillation that will address risk-benefit tradeoffs with somewhat greater precision than current approaches. (Stroke. 1993;24:2128-2134.)

KEY WORDS • anticoagulants • atrial fibrillation • embolism

There are a number of circumstances involving patients at risk for stroke due to cerebrovascular disease or cardiogenic embolism in which the potential risks and benefits of chronic anticoagulation are sufficiently close as to render clinical decision making quite difficult. In part this reflects the fact that the risks and benefits of anticoagulation have not yet been completely defined at the population level, an issue that can only be addressed by further study. However, in part it reflects uncertainties intrinsic to individual patients. Dealing with these uncertainties in a rational, quantitative fashion will be the focus of this article. Specifically, I will discuss anticoagulation in patients with stroke or transient ischemic attack and chronic atrial fibrillation in whom the relation of the rhythm abnormality to the ischemic event is uncertain or in whom the nature of the ischemic event itself is uncertain (ie, microvascular or large-vessel distribution). I will also discuss anticoagulation of patients with atrial fibrillation in the absence of stroke or transient ischemic attack. Although a quantitative approach is used, many uncertainties remain, and the results should be used as a guide to decision making and not applied in a formulaic fashion.

The general approach to be used makes use of the concept of expected value, which is equal to the product of the actual value of the end point and the likelihood of the end point. Thus, the expected value of a 99:1 prospect at the horse races that pays $100 to win is (0.01)($100)=$1. The concept of expected value provides a way of arriving at the value of an uncertain end point in a rational fashion. The actual value of the end point in the context of this article is to be determined by the patient (see “Appendix”). The likelihood of the end point can be ascertained from existing data. Anticoagulation decisions in patients at risk for cardiogenic embolic stroke have been discussed previously in a decision analytic framework, but the method used failed to include an explicit assessment of the value system of the patient, something repeatedly shown to be very important; it also involved cumbersome calculations requiring the use of a decision-making computer program. The method used in the present article is intended to be easily usable by clinicians with access only to the data included herein.

Stroke or Transient Ischemic Attack

Probable Cardiogenic Embolism

In the patient with a recent stroke due to atrial fibrillation, the 1-year likelihood of stroke recurrence is approximately 20%. By the end of 1 year, the stroke rate asymptotically approaches 5%/y. The expected
disvalue for the first year of no treatment at all, assuming the stroke was definitely due to cardiogenic embolism, is 0.20 \( V_\delta \), where \( V_\delta \) is the value of recurrent stroke as judged by the patient. Data from four major multicenter prospective randomized studies have indicated that stroke occurrence in chronic atrial fibrillation can be reduced by approximately 65% with chronic warfarin administration.\(^8\)\(^\text{--}\)\(^11\) There are few studies on the value of chronic anticoagulation in preventing stroke recurrence,\(^5\)\(^,\)\(^12\) none of a quality that meets present-day standards. However, these data suggest that until controlled studies are completed, it is reasonable to assume that anticoagulation is just as effective in preventing stroke recurrence as stroke occurrence. The expected 1-year positive (prophylactic) value of anticoagulation in the patient under discussion would then be (0.65)(0.20)\( V_\delta \), or 0.13\( V_\delta \).

The major risks of anticoagulation are systemic and intracranial hemorrhage. I will assume that because stroke is such an important event, the only other event that bears serious consideration in an anticoagulation decision is death. That is, I will assume that discomfort and transient reductions in quality of life related to nonfatal systemic hemorrhages are, compared with stroke, relatively unimportant and can reasonably be ignored in our decision analysis.

Data on risk of fatal hemorrhage in chronically anticoagulated stroke patients are quite limited, but they suggest the risk is quite high, approximately 4\%\( \text{y} \).\(^12\)\(^,\)\(^13\) Two thirds of fatalities are due to intracranial hemorrhage.\(^12\)\(^,\)\(^13\) These figures are derived in good part from prospective randomized studies of chronic anticoagulation of stroke patients conducted 30 or more years ago, using anticoagulation regimens of varying intensities. The patients were more likely to have inadequately controlled blood pressure than today (a known risk factor for intracerebral hemorrhage), but other reasons for excessive risk of intracranial hemorrhage are not immediately apparent. Most data suggest that unlike systemic hemorrhage, which is overwhelmingly associated with excessive anticoagulation (when it is not due to a potentially hemorrhagic lesion, such as a peptic ulcer), intracranial hemorrhage bears relatively little relation to degree of anticoagulation\(^13\)\(^,\)\(^15\) (but see Reference 16). Thus, the defining and relatively irreducible risk to be reckoned with in chronic anticoagulation is that of intracranial hemorrhage. Assuming a fatal hemorrhage rate of 4\%\( \text{y} \), the 1-year expected negative value of chronic anticoagulation attributable to death in a stroke patient is 0.04\( V_\delta \), where \( V_\delta \) is the disvalue of death as judged by the patient (Table 1). As many as one third of intracranial hemorrhages during anticoagulation are not fatal,\(^14\)\(^,\)\(^15\) and most of these nonfatal hemorrhages are associated with lasting residua equivalent to those of ischemic stroke. Including these residua increases the expected negative value of anticoagulation by 8\% (0.33\times0.25) to 27\% (0.33\times0.8), depending on whether the patient attaches modest (0.25\( V_\delta \)) or high (0.8\( V_\delta \)) negative value to stroke, respectively (see below). Because of the uncertainty surrounding the 4\%\( \text{y} \) fatal hemorrhage figure for anticoagulated stroke patients, calculations have also been made assuming a rate of 2\%\( \text{y} \) (Table 2).

For the patient with stroke definitely due to cardiogenic embolism, the 1-year expected positive value of chronic anticoagulation, 0.13\( V_\delta \), is clearly considerably higher than the 1-year expected negative value of chronic anticoagulation, 0.043\( V_\delta \) to 0.05\( V_\delta \), as long as the patient feels that stroke is a very serious event with a negative value close to that of death. However, these values are close enough that an explicit assessment of the patient’s values is warranted. This can often be done in a relatively informal manner, but in many instances a formal standard gamble discussion will be necessary (see “Appendix”). In my experience with standard gamble discussions in this context, patients who place a high value on intact intellectual function and physical independence will often conclude that stroke has 80\% of the disvalue of death; at the other extreme, patients may conclude that stroke has as little as 25\% of the disvalue of death. Assuming the 25\% figure, the expected positive value (EV\(+\)) of anticoagulation would be (0.13)(0.25)\( V_\delta \)=0.033\( V_\delta \), and the expected negative value (EV\(\text{--}\)) of anticoagulation would be 0.04\( V_\delta \)+0.33\( V_\delta \)=0.043\( V_\delta \) (taking into account the stroke morbidity attributable to the one third of intracranial hemorrhages that are not fatal). A full year of anticoagulation would have a negative net value. Assuming the 80\% figure, the expected positive value of anticoagulation would be (0.13)(0.8)\( V_\delta \)=0.104\( V_\delta \), the expected negative value would be 0.04\( V_\delta \)+0.33\( V_\delta \)=0.05\( V_\delta \), and the net value of a full year of anticoagulation would be positive. If explicit assessment of the patient’s values indicates that stroke has less than 35\% (0.04 + (0.35)(0.33)(0.04)/0.13) of the disvalue of death, then the net expected value of anticoagulation for 1 year becomes negative. In this circumstance, it would make sense to anticoagulate for only 6 months, reducing the risk of fatal hemorrhage to approximately 2\% (EV\(\text{--}\) = 0.02\( V_\delta \)) but covering approximately 75\%\(^2\) of the 1-year risk of embolic stroke [EV\(+\) = (0.75)(0.2)(0.65) (0.3)\( V_\delta \)=0.029\( V_\delta \)] (Table 1). This assumes of course that the risk of fatal hemorrhage is evenly distributed over time; limited data suggest that it is.\(^13\)

Possible Cardiac or Artery-to-Artery Thromboembolism

It is often difficult to determine in a stroke patient with atrial fibrillation whether the embolus came from the heart or from a site of atheromatous disease in the neck or the aorta. Carotid duplex or angiographic evidence of carotid thrombus in situ or, to a lesser extent, ulceration; a stuttering, waxing and waning, progressive course\(^17\) (but see Reference 18); and multiple events in a single vascular territory, particularly the posterior circulation, favor artery-to-artery thromboembolism. Evidence of multiple events in different cerebral arterial distributions; stroke in conjunction with hema-
turia (presumably reflecting renal embolism\(^19\)); and hemorrhagic infarction\(^10\) favor cardiogenic embolism.

In the final analysis, however, in an elderly patient with a single cerebrovascular event and carotid stenosis without in situ thrombus, it is generally not possible to determine the origin of the cerebral embolus with certainty. Instead, this risk must be calculated on the basis of probabilities. In patients with chronic atrial fibrillation, the annual risk of stroke attributable to the atrial fibrillation is 3.5\%.\(^2\) I will assume that the annual stroke risk in the distribution of a particular carotid is 45\% of this, which is the fraction of total cerebral blood
TABLE 1. Summary of Expected Values of Anticoagulation for Patients With Atrial Fibrillation

<table>
<thead>
<tr>
<th>Recent Stroke or TIA</th>
<th>History of Stroke/TIA ≥ 1 Year Prior</th>
<th>1-Year Probability of Stroke</th>
<th>1-Year Probability of Fatal Hemorrhage</th>
<th>1-Year Expected Positive Value</th>
<th>1-Year Expected Negative Value</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite cardiogenic embolic (P[stroke is cardiogenic]=1)</td>
<td>None</td>
<td>0.20</td>
<td>0.04</td>
<td>0.80V₀</td>
<td>0.25V₀</td>
<td>0.30V₀</td>
</tr>
<tr>
<td>Large-vessel distribution ipsilateral to ≥80% stenosis (P[stroke is cardiogenic]=.34)</td>
<td>None</td>
<td>0.20*</td>
<td>0.04</td>
<td>0.80V₀</td>
<td>0.25V₀</td>
<td>0.035V₀</td>
</tr>
<tr>
<td>Large-vessel vs lacunar stroke; normal carotids (P[stroke is cardiogenic]=.5)</td>
<td>None</td>
<td>0.20</td>
<td>0.04</td>
<td>0.80V₀</td>
<td>0.25V₀</td>
<td>0.052V₀</td>
</tr>
<tr>
<td>Large-vessel vs lacunar stroke ipsilateral to ≥80% stenosis (P[stroke is cardiogenic]=.17)</td>
<td>None</td>
<td>0.20*</td>
<td>0.04</td>
<td>0.25V₀</td>
<td>0.005V₀</td>
<td>0.043V₀</td>
</tr>
<tr>
<td>None</td>
<td>None</td>
<td>0.05</td>
<td>0.005</td>
<td>0.80V₀</td>
<td>0.25V₀</td>
<td>0.026V₀</td>
</tr>
<tr>
<td>None; reduced risk for systemic embolism</td>
<td>None</td>
<td>0.02</td>
<td>0.005</td>
<td>0.80V₀</td>
<td>0.25V₀</td>
<td>0.010V₀</td>
</tr>
<tr>
<td>None</td>
<td>Present</td>
<td>0.05</td>
<td>0.04</td>
<td>0.25V₀</td>
<td>0.003V₀</td>
<td>0.0054V₀</td>
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TIA indicates transient ischemic attack; V₀, value to patient of stroke; V₀, value to patient of death. 1-year expected positive value = (1-year probability of stroke)\(\times\)probability that stroke is cardiogenic\(\times\)efficacy of warfarin\(\times\)V₀. 1-year expected negative value = (1-year probability of fatal hemorrhage)\(\times\)efficacy of warfarin - 85%; probability of nonfatal intracranial hemorrhage is assumed to be 0.33 that of fatal hemorrhages of all types.

*Assumes patients with >80% stenosis and atrial fibrillation are not at higher than usual risk for stroke.
†Assumes warfarin does not reduce risk of lacunar stroke or artery-to-artery thromboembolic stroke. 
‡Assumes risk of fatal hemorrhage is evenly distributed over time.

flow supplied by each carotid. In patients with asymptomatic carotid stenosis of greater than 80%, the annual risk of stroke without preceding transient ischemic attack may be as high as 4%.2,21-26 I will assume that three fourths of these strokes are artery-to-artery thromboembolic and ipsilateral to the stenosis.26 In a patient with both conditions who then experiences a large-artery distribution cerebral infarction in the territory of the stenotic vessel, the probability that the stroke was due to cardiogenic embolism is then \((3.5)(0.45)/[(3.5)(0.45)+(0.75)(4)]\)=0.34; this of course assumes that there are no other possible sources of embolism, a degree of oversimplification that is probably tolerable in the present context.27 In patients with lesser degrees of ipsilateral carotid disease, the likelihood of cardiogenic embolism will be higher; it presumably approaches 100% in the patient with normal vessels. With extreme carotid stenosis, the obstruction might actually prevent cardiogenic emboli from reaching the brain; no good data are available, but this consideration leads to the common-sense conclusion that a cardiac embolus is probably not the cause of the stroke in these circumstances and that consideration might be given to carotid endarterectomy.

Assuming a 34% likelihood of cardiogenic embolism, the 1-year positive expected value of anticoagulation in the patient with high-grade ipsilateral carotid stenosis would be \((0.34)(0.65)(0.20)V₀\) or 0.044V₀. This assumes that the 1-year stroke rate in such a patient is not greater than 20% and that chronic anticoagulation is of no value in preventing recurrent stroke due to artery-to-artery thromboembolism; neither hypothesis has been adequately tested. In this case, even if the patient valued stroke very negatively (eg. \(V₀=0.8V₀\)), the expected positive value of anticoagulation (\(EV^+= (0.8)(0.044)V₀=0.035V₀\)) would be less than the expected negative value of anticoagulation, 0.051V₀, and a full year of anticoagulation could not be justified.

**Possible Lacunar Infarction**

There is a general consensus that lacunar infarction is almost always due to intrinsic microvascular disease and is uncommonly embolic in origin.20-21 Thus, if a patient in chronic atrial fibrillation experiences a lacunar stroke, it is safe to say that the stroke was unlikely to be due to cardiogenic embolism, and the conditions of the clinical decision are those of the patient with chronic asymptomatic atrial fibrillation (see below). Unfortunately, situations frequently arise in which it is not possible to be sure whether the patient has had a lacunar or a large-vessel event. This is because of the fact that clinical discrimination between the two types of stroke is frequently inadequate, particularly when there is a mild neurological deficit, and the fact that computed tomography detects only 40% of lacunes and 70% of large-vessel infarcts29 and magnetic resonance imaging detects about 80% of lacunes30,31 but is probably not much better than computed tomography at detecting
TABLE 2. Analysis of the Effect of Different Estimates of Warfarin Efficacy and Risk in Patients With Prior Cerebrovascular Events

<table>
<thead>
<tr>
<th>Recent Stroke or TIA</th>
<th>Probability That Stroke Is Cardiogenic</th>
<th>1-Year Probability of Fatal Hemorrhage</th>
<th>Efficacy of Warfarin</th>
<th>1-Year Expected Positive Value</th>
<th>1-Year Expected Negative Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite cardiogenic embolic</td>
<td>1</td>
<td>0.20</td>
<td>0.04</td>
<td>0.65</td>
<td>0.80V₀</td>
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<tr>
<td>Large-vessel distribution ipsilateral to ≥80% stenosis</td>
<td>0.34</td>
<td>0.20</td>
<td>0.04</td>
<td>0.65</td>
<td>0.80V₀</td>
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<tr>
<td>Large-vessel vs lacunar stroke; normal carotids</td>
<td>0.50</td>
<td>0.20</td>
<td>0.04</td>
<td>0.65</td>
<td>0.80V₀</td>
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<tr>
<td>Chronic atrial fibrillation; old stroke</td>
<td>. .</td>
<td>0.05</td>
<td>0.02</td>
<td>0.65</td>
<td>0.80V₀</td>
</tr>
</tbody>
</table>

TIA indicates transient ischemic attack; V₀, value to patient of stroke; V₀, value to patient of death. 1-year expected positive value=(1-year probability of stroke)(probability that stroke is cardiogenic)(efficacy of warfarin)V₀. 1-year expected negative value=(1-year probability of fatal hemorrhage)V₀+(probability of nonfatal intracranial hemorrhage)V₀. Assumed efficacy of warfarin=65%; probability of nonfatal intracranial hemorrhage is assumed to be 0.33 that of fatal hemorrhages of all types.

In such cases, one might estimate the probability of a large-vessel event at 50%. The expected positive value of anticoagulation in a patient in these circumstances in whom there is no evidence of atheromatous cerebrovascular disease would then be (0.5)(0.65)(0.20)V₀=0.065V₀. If the patient attached a high negative value to stroke (eg, V₀=0.8V₀), the expected positive value of anticoagulation would be (0.065)(0.8)V₀=0.052V₀, the expected negative value 0.05V₀, and a full year of anticoagulation would be justifiable. However, for V₀ even slightly less than this, some curtailment of duration of anticoagulation would be indicated. I am assuming that the risk of fatal hemorrhage is the same in patients with lacunar infarction as it is in patients with large-vessel infarction; this is untested. If there is severe cerebrovascular disease with greater than 80% ipsilateral carotid stenosis, such that there is a probability of only 34% that the stroke, if large-vessel, is due to cardiogenic embolism, then the expected positive value of anticoagulation becomes (0.34)(0.065V₀)=0.022V₀, which will always be less than half the expected negative value of anticoagulation, 0.043V₀, indicating that the duration of anticoagulation should be less than 6 months in all such patients.

The risks of chronic anticoagulation can often be substantially mitigated by successful cardioversion, which allows early discontinuation of anticoagulants but requires long-term follow-up to detect expeditiously recurrent atrial fibrillation.

Because of the uncertainty as to whether warfarin is just as effective in preventing stroke recurrence as it is in
No Recent Embolism

The total annual stroke risk in patients with chronic atrial fibrillation of greater than 1-year duration and in patients with atrial fibrillation and a history of stroke more than 1 year prior is approximately 5%.8,9,10 Warfarin may reduce this by 65%.8,9,10 Thus, the expected positive value of warfarin is (0.65)(0.05)\(V_p=0.0325V_p\). If the disvalue to the patient of stroke is 0.8\(V_p\), the expected positive value of anticoagulation becomes (0.0325)(0.8)\(V_p=0.026V_p\). The annual rate of fatal hemorrhage in recent randomized studies of anticoagulation in patients with atrial fibrillation was 0.4%,8,9,10 In older studies of chronic anticoagulation of patients with ischemic heart disease it was 0.6%.13 I will assume a rate of 0.5% for this analysis. The expected negative value of warfarin would then be 0.0063\(V_p\), taking into account morbidity from nonfatal intracranial hemorrhage. The net expected value of anticoagulation for 1 year would then be (0.026−0.0063)\(V_p=0.0197V_p\), providing a strong rationale for chronic anticoagulation. At the other extreme, patients may consider stroke to have only 25% of the disvalue of death, yielding an expected positive value of anticoagulation of (0.25)(0.0325)\(V_p=0.008V_p\). This is only 1.48 times the expected negative value of anticoagulation (0.008/0.0054), and if the risk of fatal hemorrhage with anticoagulation were suspected to be much higher than usual, the net expected value of chronic anticoagulation would become negative. In the patient with a history of stroke, however remote, the annual risk of fatal hemorrhage, as noted above, may be 4%, thus contraindicating anticoagulation in all stroke patients after the first year (but see sensitivity analysis, Table 2). Further studies are necessary to refine our ability to predict fatal intracranial hemorrhage in anticoagulated patients.

I have assumed an annual stroke risk of 5% in patients with chronic atrial fibrillation without a history of stroke or transient ischemic attack in the preceding year. Recent studies by the Stroke Prevention in Atrial Fibrillation Investigators have identified several clinical and echocardiographic features that predict increased risk of stroke in patients with atrial fibrillation: a history of hypertension, previous thromboembolism, recent congestive heart failure, global left ventricular dysfunction, and left atrial size greater than 2.5 cm/m² by M-mode echocardiography.33,34 Annual stroke risk was 1% in patients with none of these risk factors, 6% in patients with 1 or 2 risk factors, and 18.6% in patients with 3 or more risk factors. Framingham Study data also suggest that the risk of stroke during the first year of chronic atrial fibrillation is 13%.35 Unfortunately, there are no data on the efficacy of warfarin in curbing these heightened stroke risks. However, these data do serve to identify a population of patients with chronic atrial fibrillation who are at such low risk of stroke that anticoagulation is not warranted. Other studies have identified patients younger than age 60 with lone atrial fibrillation (unassociated with clinical, electrocardiographic, or chest roentgenographic evidence of heart disease) and, less certainly, patients with paroxysmal atrial fibrillation as having risks of stroke too low to warrant anticoagulation.6,7

Conclusion

Many uncertainties remain regarding risks of stroke and hemorrhage in the circumstances discussed in this article. Both the institution and the withholding of anticoagulation may be associated with major morbidity or death. Nevertheless, clinicians are obliged to make a decision, and they strive to make a maximally rational one. My own bedside standard gamble discussions with patients suggest that the disvalue of stroke may vary between approximately 0.25\(V_p\) and 0.8\(V_p\), representing a threefold difference. Because of the closeness of benefits and risks of anticoagulation under many of the circumstances I have considered, this threefold difference is frequently sufficiently large to have a major impact on initiation or duration of anticoagulation (Table 1). The use of an expected value approach further permits the explicit handling of multiple sources of uncertainty in estimating likelihood of an outcome. Because of the uncertainties in risk estimates, the method I have discussed cannot be used in a doctrinaire or formulaic fashion but rather should yield some rough guidelines for treatment that will address risk-benefit tradeoffs with somewhat greater precision than current approaches. The advantages of the method and the impact of explicit assessment of patient values are likely to be felt even with somewhat different assessments of the various risks involved. When the difference between expected positive and negative values is small, the balance may be tipped by issues such as patient concerns regarding taking medication or discomfort and reduction in quality of life related to nonfatal systemic hemorrhage.

Appendix

Explicit ascertainment of the patient’s rating of the disvalue of stroke relative to death can be done using a standard gamble technique.36 The following is a model of the dialogue the physician might have with the patient:

You have experienced a stroke. Based on the tests we have conducted, we believe that stroke was caused by a fragment of blood clot that formed in your heart, broke off, and traveled to your brain. We know that under these circumstances, there is about a 1 in 5 chance that another clot will travel to your brain within the next year, possibly causing an even larger stroke. A medication is available, warfarin, that will reduce this risk of having another stroke, perhaps by as much as 2/3. However, warfarin can cause severe bleeding, including bleeding into the head, which can be fatal. Thus, we need to carefully weigh the benefits of warfarin against the risks. There is no uniformly correct decision in a situation like this. Rather, you and I need to work together to arrive at the correct decision for you. I know about the medical aspects of this decision. However, this is a decision involving a chance of having a stroke and a chance of dying. Therefore, I need to know how you feel about having a stroke and dying. In particular, I need to know how bad it would be for you to have another stroke. This is a hard question for anyone to answer. To help you answer, I am going to show you a make-believe situation and ask you how you would respond. This situation will undoubtedly make you feel uncomfortable, so just do your best. Please keep in mind that it is make-believe, designed to help me evaluate your feelings.

Pretend you are at a crossroads in life (show patient Figure). Imagine you have an illness that will certainly cause a stroke...
within the next few days (point to the bottom path). However, there is a radical treatment that could absolutely prevent you from having a stroke (point to upper fork of top path). Unfortunately, this treatment carries with it a risk of death (point to lower fork of top path). What is the minimum chance of a cure that you would have to have to accept the radical treatment? I'm going to put some numbers in here and I want you to tell me when it seems like it's a toss-up—the two paths seem about the same. (Write in 50 on both forks of upper path). Here, if you accept the treatment, you have a 50-50 chance of a cure—good health without a stroke—but also a 50-50 chance of dying. Over here (point to lower path), you will definitely have a stroke. Which path would you choose? Remember, these numbers do not apply directly to your situation; however, they will help me to advise you whether to take the radical treatment, which does involve accepting some risk of dying in order to prevent stroke. (Substitute in successively higher or lower numbers in 10% to 20% increments until the patient understands the problem and settles on a set of numbers. The percentages on the two upper branches must always add up to 100. The likelihood of death at which the patient is ambivalent about the radical treatment constitutes the best estimate of his valuation of stroke relative to death.)

This approach to estimating the disvalue of stroke makes some simplifying assumptions. First, it treats stroke as a permanent state, as opposed to an event followed by some degree of recovery and return to at least some aspects of normal life. Second, it treats all strokes as equal, when in fact the impact of minor and major stroke is obviously quite different. Third, it leaves to the patient the task of estimating the long-term personal ramifications of nonfatal events that occur during the treatment (or non-treatment) period. Other investigators have used Markov and related models to deal with these problems in various contexts.\textsuperscript{13,20,21,23,27,30,36,37} These models in essence calculate expected values for each of the remaining months of the patient's life, entering into the calculation the probability that the patient will experience a change in state, eg, from health to stroke, stroke to death, or health to death, in any particular month. Arbitrary disvalues are attached to the various states, eg, major stroke and minor stroke, and a discount factor is used to account for the fact that events that occur or states that exist at some time in the future have less value, or disvalue, than when they develop immediately. The net result is an estimate of the value of a particular strategy in chronic fibrillation. Stroke. 1990;21:4-13.

24. Moneta GL, Taylor DC, Nichols SC, Berglin RO, Zierler E, Kazmers A, Clowes AW, Strandness DE. Operative versus nonop-


The use of expected value as an aid to decisions regarding anticoagulation in patients with atrial fibrillation.

S E Nadeau

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