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Presidential Address

A letter to Helen

John W. Kirklin, M.D., *Birmingham, Ala.*

Dr. Sloan, thank you for your warm introduction. Fellows of the Association and guests: Thank *you* for the privilege of being your President this year and for allowing me to preside over this Fifty-ninth Annual Meeting of The American Association for Thoracic Surgery. You must know that the Presidency is all honor and privilege, including the unique one of getting a place on the program without even turning in an abstract! But the real work is done by others.

In thinking about this address, I've dreamed all the dreams of other presidents of this Association. I've dreamed of entertaining you as Danny Kaye might, of inspiring you as John Kennedy might, of teaching you as Peter Medawar might, of charming you as Denton Cooley might, even of intimidating and overwhelming you as the Pittsburgh Steelers might. But dreams are for the dreamer, and not often are translatable into something palatable let alone useful for so esteemed an audience as you.

I thought I might simply read to you a letter which I wrote one day this past winter as I sat in my home in Birmingham, Alabama, trying to translate some vague thoughts into this address.

First, I must explain about Helen. She really exists and is part of the most important association to which I belong, and that is my family. My wife, Peggy, has with her love sustained me throughout our lives together. Two sons, John and Jim, one a lawyer in New York City and one a cardiac surgical resident in Boston, and a daughter complete our wonderful family. Helen is our daughter and she now shares her life with her husband, Dr. Robert Ray, a member of the faculty of the Department of English of the University of Florida in Gainesville. When there's time she plays her violin in chamber music groups and tennis with her husband and occasionally even with us.

Birminghan, Ala.
February, 1979

Dear Helen,

I am taking a respite from the task of trying to write a Presidential Address by writing to you instead, for I'm discouraged by my lack of progress.

The Presidential Address is to be presented before The American Association for Thoracic Surgery, and it has taken me back in memories to the early years when I went as a resident to those meetings. When I was with Dr. Robert Gross in Boston in 1948, it was easy to do what we did, which was to make great lists of reparative operations for congenital and acquired heart disease and notes as to how they might be done. When I joined the surgical staff of the Mayo Clinic in 1950, a dream seemed to come true each day, as we sailed with

From the University of Alabama Medical Center, Birmingham, Ala. 35294.

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all the confidence of youth into closed cardiac surgery. Late in 1951 we began to work with extracorporeal circulation and pump oxygenators in the laboratory—much to the amusement of the many visitors who made Rochester, Minnesota, one of their stopping places. In 1953 Dr. John Gibbon in Philadelphia, Pennsylvania, did what I believe was the first successful intracardiac operation with cardiopulmonary bypass using a pump oxygenator. However, this event had little impact on the surgical world, in large part because his subsequent four patients all died of an assortment of complex problems. But in the spring of 1954, just 25 years ago, Walt Lillehei and Richard Varco, at the University of Minnesota, for sure made an impact when they began successfully to do open-heart operations in infants and children with cardiopulmonary bypass using a pump and, believe it or not, another human being as the oxygenator. Walt Lillehei, with Dick Varco, really began this modern era of cardiac surgery with cardiopulmonary bypass, and I hope he will be at the meeting in Boston. He always was and still is a great *hero* of mine, because of his enormous ability and warm friendship. It's some cruel trick of fate that there is no operation called the Lillehei operation; yet he was one of cardiac surgery's greatest innovators and did scores of "first time" operations. [Dear colleagues: May I depart from my text to ask this great and pioneering cardiac surgeon to stand to your applause. Walt Lillehei, may we see you?] In early 1955 we began open-heart surgery at the Mayo Clinic with a pump oxygenator. It was exciting to us, and I'm sure to Walt Lillehei and Richard Varco in Minneapolis, that for a year or so the only places in the world regularly doing open-heart surgery with cardiopulmonary bypass were two towns in Minnesota, 90 miles apart. In those days almost every other operation we did was a new and "first time" one, and we were all so busy that usually we didn't take the time to lay our claims to priority in publications.

Much of the excitement in those early days was generated by the wonderful young people who came to learn cardiac surgery. This is still true today. Our methods for helping these people learn cardiac surgery have continuously evolved in as real a way, as has surgery itself. We have learned that the essence of the educational experience in cardiovascular surgery is the resident's realization that every step of the preoperative evaluation, of the decision-making process, of the operation itself including literally the various "moves" of the surgeon during the procedure, and of the postoperative care must be rational, focused, direct, and simple. While learning this disciplined approach, the resident must also learn to think about alternatives to his plans and about the times when he must improvise in a light-footed way to meet some unexpected development. The most critical and important part of the cardiovascular surgical resident's learning is *in the operating room,* and this is where we try to be certain that he learns well and completely the thousand and one little details of operative cardiac surgery. This means endless hours of scrubbing with them, of searching for their wasted motions and helping them to get rid of them, of increasing their perception of tissue planes and insisting that they use them in their dissection, of ensuring that they keep the operation moving forward, and so on, and so on, and so on.

Well, these young people in training all over the world do not remain residents for long, and soon they become players on the stage of cardiac surgery. They are better trained than we who have preceded them, and they have the energy and quickness of youth. Soon they begin, quite appropriately, to challenge the more senior among us. We have some advantages from the experiences that our years of surgery have given us. If we have good fortune and good health, our technical skills can be maintained and actually improved through many years of surgery. Indeed, we must make improvements, because not only do our younger colleagues challenge us, but also cardiac surgery is testing us every day as is life itself. This was eloquently stated by Charles Evans Hughes, once Chief Justice of the Supreme Court. He said: "One of the most important lessons of life is, that success must continually be won, and is never finally achieved. . . . Every day puts at risk all that has been gained—the greater the achievement the more serious is the risk of loss. It is not worthwhile to talk of the end of the period—for you are always at the beginning of a new one. . . ."

After years of cardiac surgery and many tests and challenges from different operations, from patients, from colleagues, from difficult scientific and operational problems, and even more after too many deaths through the years that could not then be prevented, we gradually tend to become a little weary and in some small sense infinitely sad because of life's inevitabilities. You know, Helen, this was in some ways the theme of a film we all saw together years ago, called *The Gunfighter,* with Gregory Peck. You may remember that in the opening scene, the gunfighter is seen in the distance, obviously on a very long ride. Soon we saw that he was tired, very tired, and a little sad. In the gunfight in the saloon that followed, he obviously was wearied by the necessary shooting of his violent young challenger. You'll remember that this scene in the film had its humor and its tragedy, and in a

Table I. *Primary operations for aortic valve replacement, 1975 to July, 1978 (n = 657)*

Category	*No.*	*Hospital deaths*		
		No.	*%*	*70% CL*
AVR alone, primary	403	7	1.7%	1.1%-2.7% } p = 0.08
AVR + CABG	191	8	4.2%	2.7%-6.3% }
AVR + RAA	53	3	6%	2%-11%
AVR + CABG + RAA	10	2	20%	7%-41%
Total	657	20	3.0%	2.4%-3.9%
			p = 0.004	

Legend: CL, Confidence limits. AVR, Aortic valve replacement. CABG, Coronary artery bypass grafting. RAA, Resection of aortic aneurysm.

tongue-in-cheek way I'd suggest it has its parallels in modern cardiac surgery. In the film, the gunfighter and the young punk shot it out in a lonely bar far out West. But in a figurative sense, at least, the location could have been cardiac surgery, and the old gunfighter could have been any of several senior cardiac surgeons, challenged as to their quickness and skill by one of the wonderful new generation of cardiac surgeons. Of course, in our world of cardiac surgery, the challenge of the young is not to a gunfight. At its best, it is the challenge to maintain our surgical skills and competence, to refine continually our knowledge, to generate new knowledge—and the challenges and our response to them often result in greater excellence by both the challenged and the challenger. In the end, of course, youth will prevail.

The challenges come mostly from cardiac surgery itself. They have made it necessary for us through the years to find new and better ways of doing most things. Helen, let me give you a glimpse of one way of doing this, and that is through the identification of the things which in toto determine the success or failure of our operations, things which we call *incremental risk factors*. Once we have identified these for a given operation at a given point in time, we can approach decision-making for individual patients more precisely. Then we also can plan *research* directed at understanding the underlying mechanisms and at developing ways of neutralizing these incremental risk factors and decreasing the risk of the operation in the future.

Let's look at some data concerning *aortic valve replacement*, as it was done in Alabama from 1975 to July 1978. This is an interesting period because part way through it we changed from protecting the heart by simple cold ischemic arrest to cold cardioplegia. During this time period 840 patients underwent aortic valve replacement, either alone or in combination with some other procedure exclusive of mitral valve replacement.

Table II. *Primary operations for aortic valve replacement, 1975 to July, 1978 (n = 657)*

Causes of hospital death	*No.*	*Percent of total cases*	*70% CL*
Cardiac death	12	1.8	1.3%-2.5%
Hemorrhage	4	0.61	0.31%-1.1%
Infection	3	0.45	0.20%-0.92%
Pulmonary embolism	1	0.15	0.02%-0.52%
Total	20	3.0	2.4%-3.9%

Legend: CL, confidence limits.

Let's look in detail at the 657 patients in whom the operation was a primary one (Table I).

As a first step, let's look at what we judge retrospectively to be the causes of death (Table II). Twelve were from cardiac causes, four from massive hemorrhage, three from infection, and one from pulmonary embolism. We have *analyzed* the incremental risk factors underlying *each* of these causes of hospital death, and the findings are all fascinating. However, let's look at the analysis of just one of them, death from cardiac causes.

We will consider first the patients in whom simple cold ischemic arrest was used. We analyzed all the preoperative, intraoperative, and postoperative variables, 25 in all, that might be *incremental risk factors* as regards death from cardiac causes. These included dyspnea, orthopnea, syncope, heart failure, left ventricular size, bypass time, and so on. The operations included associated coronary bypass grafting or resection of the ascending aorta. Only four of the 25 turned out to be statistically significant as determinants of hospital death from cardiac causes (Table III). We found that these variables, *not concomitant coronary artery bypass grafting or resection of ascending aorta, were the basic incremental risk factors* in these patients.

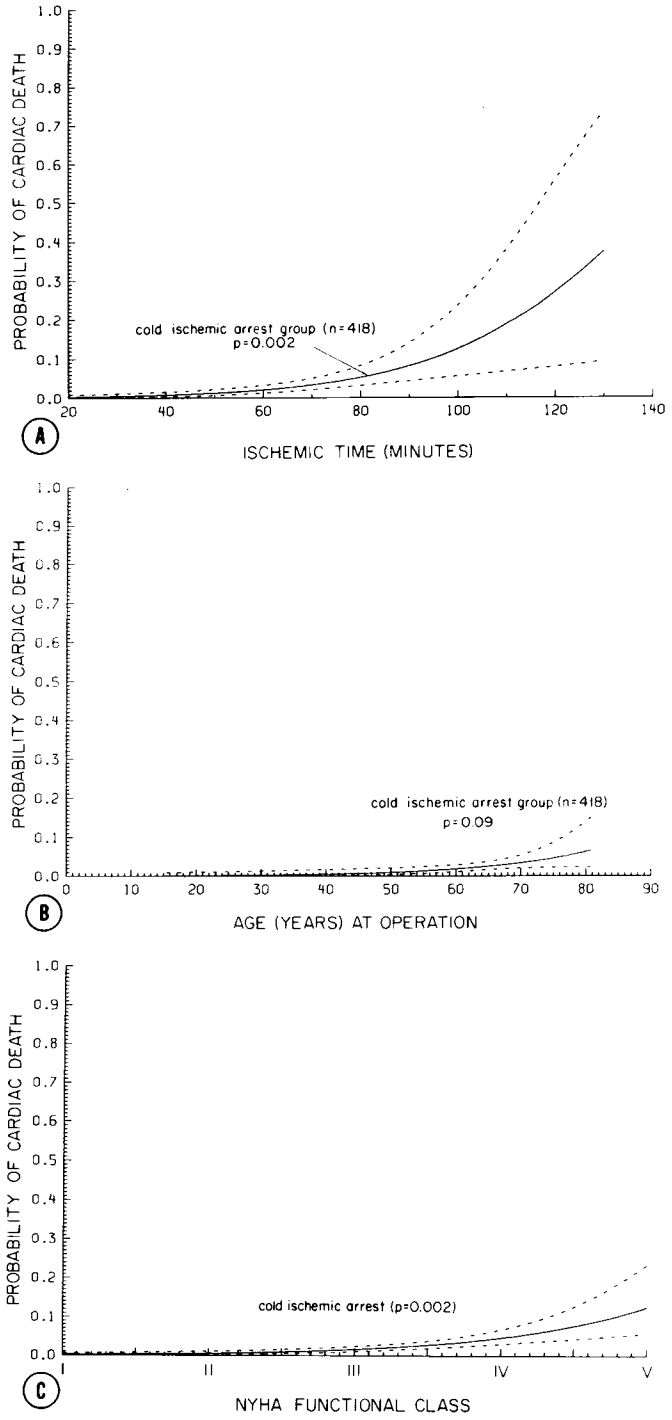

Fig. 1. Nomograms of individual parametric analysis of incremental risk factors for hospital death from cardiac causes after isolated or combined aortic valve replacement (UAB, 1975 to July, 1978) using cold ischemic arrest. The *solid line* is the point estimate, and the *dotted lines* enclose the 70% confidence limits. The equations and their statistics are available upon request for this and the other figures.

A, Ischemic (aortic cross-clamp) time. When several ischemic periods were used, the sum of the individual times was used. *B*, Age of the patient at operation. *C*, New York Heart Association (N.Y.H.A.) functional class, treated as a continuous variable. Class V was used for patients whose operations were done as emergencies because of acute and severe hemodynamic deterioration (usually from acute bacterial endocarditis).

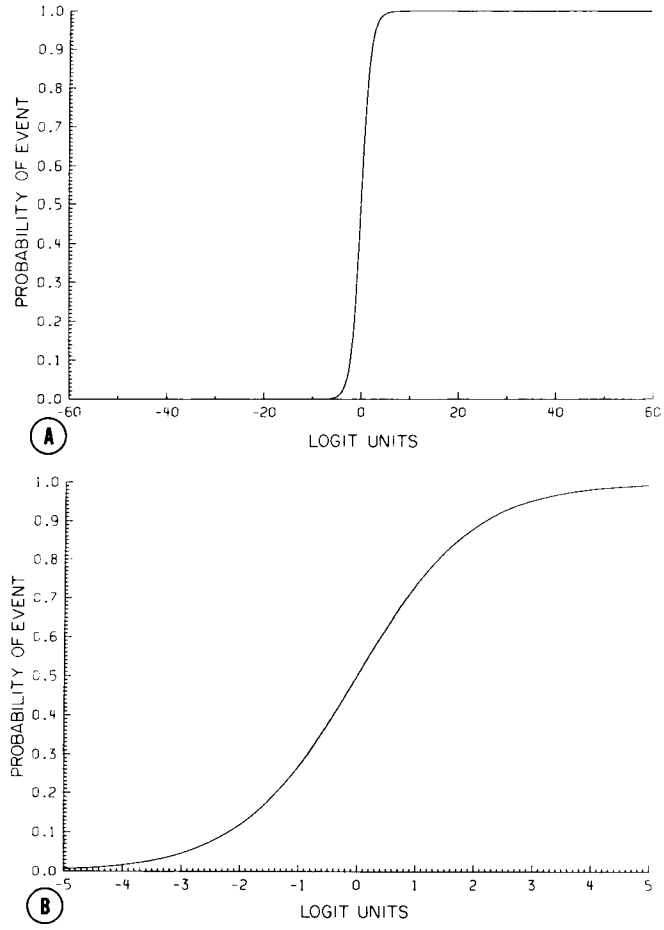

Fig. 2. Nomograms of the logistic equation. *A*, The nomogram is presented with ±60 logit units along the *horizontal axis*. The equation and the nomogram indicate a specific probability for each specific total logit unit. *B*, Nomogram of the same equation, portrayed with ±5 logit units along the *horizontal axis*. The same specific probability is denoted by each specific total logit unit, and the relation between the two as portrayed in the nomogram only seems to be different.

We can grasp the implications of this by looking at nomograms, or graphic displays, of the *individual* effect of each of these *incremental risk factors*. We see verified what most cardiac surgeons have intuitively believed, that after 45 to 60 minutes the risk of keeping the aorta cross-clamped rises steeply with the method of cold ischemia (Fig. 1, *A*). We see that in patients older than 65 to 70 years, older age becomes an incremental risk factor of importance (Fig. 1, *B*). The preoperative state of the patient, as expressed in the New York Heart Association (N.Y.H.A.) classification, with Class V used to denote operations done as an emergency because of acute deterioration of the hemodynamic state, also is a significant determinant of the risk of operation (Fig. 1, *C*).

Let me introduce you to some concepts developed years ago by Dr. Joe Berkson at the Mayo Clinic, who first introduced me to statistics in 1946, before you were born, Helen. Berkson, and other statisticians, observed these probability phenomena in many areas, including the response of animals to drugs in various doses, red cell hemolysis, and enzymatic reactions. No doubt many other events, such as the behavior of populations, fracture of aircraft wings, and so forth, are also basically probability events. What Berkson did was to realize that the phenomena which he studied could be described by a *logistic equation*. We believe this equation is also a generally applicable way of expressing *incremental risk factors* in cardiac surgery:

$$\ln(P/1 - P) = Z = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \ldots + \beta_k x_k \quad (1)$$

where ln = natural logarithm, P = probability of an event, and $\beta_0 + \beta_1 x_1 + \ldots$ = the logit unit.

To visualize this equation, we can look at a nomo-

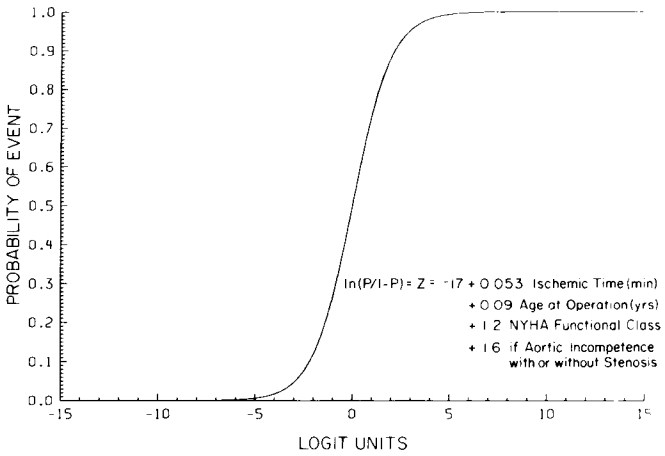

Fig. 3. Nomogram of the logistic relation and the logistic equation for the probability of event (cardiac hospital death after isolated or combined aortic valve replacement with cold ischemic arrest). The intercept, or starting point on the *horizontal axis* of logit units is −17. Each coefficient times its individual incremental risk factor describes a logit unit to be added. The resultant total logit units yield a specific probability of hospital death from cardiac causes (on the *vertical axis*). The 70% confidence limits (one standard deviation) of the probability can be calculated.

Table III. *Isolated and combined aortic valve replacement (1975 to July, 1978): Primary operation, cold ischemic arrest (n = 418); multivariate analysis of the determinants of cardiac deaths (n = 8)*

*Incremental risk factors**	*Coefficient†*	*p Value*
Ischemic time (min)	0.053 ± 0.0182	p = 0.004
Age of operation (yr)	0.09 ± 0.046	p = 0.05
N.Y.H.A. Functional Class (I to V)	1.2 ± 0.58	p = 0.05
Presence of aortic incompetence with or without stenosis	1.6 ± 0.89	p = 0.07

Legend: N.Y.H.A., New York Heart Association.
*Twenty-one other factors not significant (p > 0.20).
†Intercept = −17 ± 4.1 (p = 0.00003).

gram of it (Fig. 2, *A*). The probability of an event, lying along the vertical axis, could be the probability of hospital death from cardiac causes after isolated or combined aortic valve replacement. The logit units on the horizontal axis, as they become more negative and move to the left, approach a value indicating a probability of hospital death of zero, and as they become positive and move to the right, they approach a value indicating a probability of hospital death of 100%. We can all visualize circumstances in an elderly patient in advanced heart failure, for example, in whom some event during the operation such as the need for a second aortic cross-clamp is "the straw that breaks the camel's back." In other words, the patient is already sitting on the logit plot nomogram so close to the vertical upswing that the smallest additional incremental risk steeply increases the probability of hospital death. If we look at a smaller part of the whole scale of logit units (Fig. 2, *B*), the shape of the curve only *seems* to be different. As I have said, Helen, these relations probably pertain in all biological systems and perhaps in physical and sociological ones as well. The trick, of course, is to obtain quantitative information with which appropriate analyses and transformations can be made. Thereby, we can understand the nature of the problem and, after that, through research can modify in a favorable way the probability of the event we are considering.

Let's now go back to aortic valve replacement, without or with concomitant coronary artery bypass grafting or resection of the ascending aorta. The multivariate analysis can be expressed in a logistic equation incorporating the determinants of hospital death from cardiac causes and their coefficients, and viewed against the background of the nomogram of the logistic equation (Fig. 3). We note that the intercept is −17. Thus in this operation we start far to the left on the logit scale, in a very safe area. However, when we add many, many minutes of ischemic time, or many years of age, or N.Y.H.A. Class IV, we move so far to the right that we enter the area where the risk rises steeply.

Let's now look at the patients operated upon in the latter part of our experience, in whom cold cardioplegia

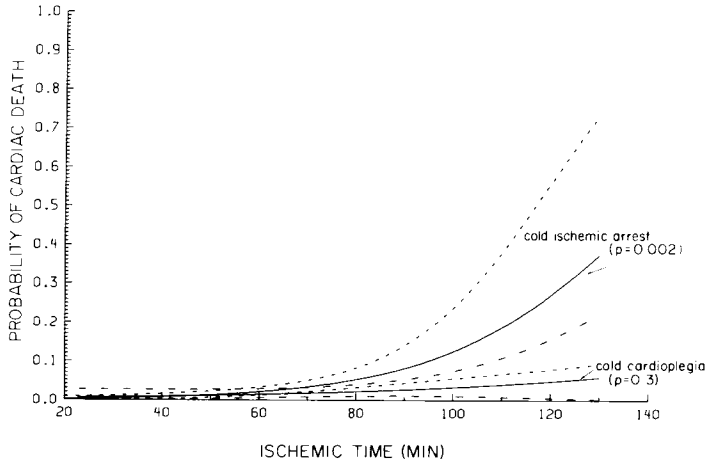

Fig. 4. Nomogram of the effect of ischemic time, a single variable, on the probability of hospital death from cardiac causes after isolated and combined aortic valve replacement (UAB, 1975 to July, 1978) when cold ischemic arrest was used and when cold cardioplegia was used. The presentation is as in Fig. 2. The p values are for the effect from the single variable analysis. Although the 70% confidence limits are overlapping, by multivariate analysis ischemic time is a significant ($p = 0.004$) incremental risk when cold ischemia is used, but not ($p > 0.20$) when cold cardioplegia is used.

was used as the method for myocardial preservation. Again, we have made a multivariate analysis and this time *only one factor*, The N.Y.H.A. class, is significant. Age of the patient, aortic cross-clamp time, and aortic incompetence no longer are significant factors. Thus the research that developed this new method of myocardial preservation has, in fact, neutralized three factors which once were incremental risk factors after aortic valve replacement. This becomes rather dramatic if we look just at aortic cross-clamp time. With cold cardioplegia, up to the longest cross-clamp time of 125 minutes, there was *no* effect of the longer periods of myocardial ischemia on the probability of hospital death from cardiac causes (Fig. 4). However, the job is not done, for there does remain an effect of the preoperative status of the patient. Thus we must focus our efforts on determining why we continue to have an increased risk, even with cold cardioplegia, from N.Y.H.A. Classes IV and V. To do this, we need to know more about the biochemistry and histopathology of myocardial failure. We must develop additional methods for neutralizing and reversing these factors so as to make these operations still safer in the future.

Now, Helen, let me show you just one more example. You know that Sir Brian Barratt-Boyes, our honored guest at the meeting, made a great contribution when he demonstrated the advantages of primary intracardiac repair in infancy for many types of congenital heart disease. Subsequently, many others have achieved remarkably good results, particularly in certain defects. However, in spite of these results, our analyses of our own experiences and those of others around the world lead us to believe that the *required intraoperative support techniques* for open intracardiac surgery are in and of themselves *incremental risk factors* which become particularly important in the very young.

Remember Walt Lillehei's early spectacular results in young infants, in whom he used controlled cross-circulation and another human being as an oxygenator and blood purifier 25 years ago, in the earliest days of cardiac surgery. It has taken many, many years and lots of innovation and effort to duplicate let alone beat those early results in infants now that we use *pump oxygenators*. Recall also that so-called closed operations, *without* the use of a heart-lung machine, are very safely done in young infants. For example, with Eduardo Arciniegas we have looked at the relation of age to the probability of hospital death after shunting operations for the tetralogy of Fallot. Very young age increased the risk only a little. We now believe that even this may not be from the young age per se, but rather from the fact that with the techniques used some very small babies did not ultimately have well-functioning shunts. Thus we have the idea that very young age per se is not an important incremental risk factor when we do not have to use cardiopulmonary bypass, or profound hypothermia and total circulatory arrest, and so forth.

You can imagine how important it is to know whether the special support techniques for *open in-*

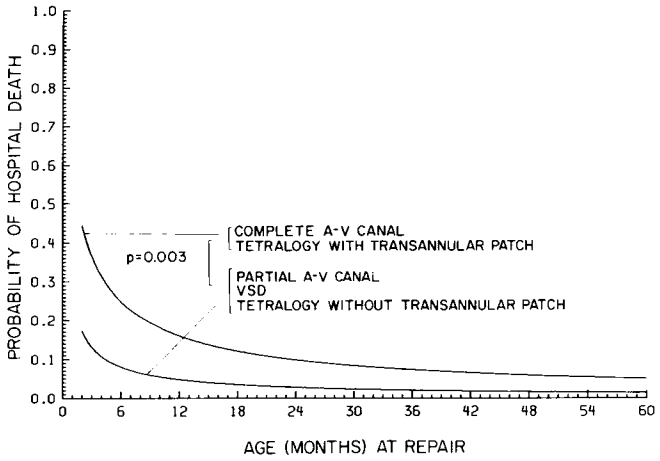

Fig. 5. Nomogram of the parametric analysis of the relation of age to mortality in the two groups of patients with congenital heart disease. (These series date back to 1972 and do not necessarily reflect current experience.)

Table IV. *Congenital heart disease: Study groups for incremental risk factors*

Category	*Source*	*No.*	*Hospital deaths*	
			No.	*%*
Single large VSD	UAB 1972-1978	114	4	3.6
Partial AV canal	UAB 1972-1979	111	1	0.9
Complete AV canal	UAB 1975-1978	39	8	21
Classical tetralogy of Fallot (without transannular patch)	UAB 1972-1978	117	4	3.5
Classical tetralogy of Fallot (with transannular patch)	UAB 1972-1978	74	10	14
Shunting operations for tetralogy	Detroit-UAB 1967-1978	146	6	4.1
Total anomalous pulmonary venous connection	UAB 1975-1978	17	2	12
Total		618	35	5.7

Legend: VSD, Ventricular septal defect. AV, Atrioventricular. UAB, University of Alabama at Birmingham.

tracardiac operations are incremental risk factors in infants and young children. If they are, we must work intensely to identify mechanisms and devise ways of neutralizing the incremental risks. Let's begin by examining, *in one group,* certain types of congenital heart disease that we have in the past analyzed in detail. Currently, hospital mortality rates may be lower, but that is not the point. We want to look for various *incremental risk factors,* particularly young age, in a more powerful way than if we looked at the individual groups with their smaller number of patients. Of course, we do this by the same multivariate analysis technique with the logistic equation (Table IV).

With this, we see that *young age,* in the period studied, was a highly significant incremental risk in open-heart surgery, with a p value of 0.00002, in the *patients* with *ventricular septal defect, (VSD),* indicated by the intercept value, *partial atrioventricular (AV) canal, tetralogy without transannular patch, complete AV canal,* and *tetralogy with transannular patch* (Table V). As we have already seen, the patients with closed shunting operations behaved differently, as did those with total anomalous pulmonary venous connection, for reasons we shall not discuss here. Very interestingly, in the first five groups, the slopes of the curves relating age to probability of hospital death were the same during this period (Fig. 5). Further, at any given age, the risks of repair of VSD, partial AV canal, and classical tetralogy of Fallot without a transannular patch were pretty much the same. Complete AV canal was an incremental risk, we think primarily because of the technical challenges inherent in transforming a somewhat incompetent common AV valve into competent mitral and tricuspid valves. This illustrates one of the purposes of such analyses, for having come to this opinion we have concentrated during the operations on obtaining competent AV valves with methods that will assure good healing. We believe this is producing better results. Classical tetralogy repaired in infancy *with* a transannular patch was also an incremental risk of about the same magnitude as complete AV canal, we believe due in large part, but perhaps not completely so, to the detrimental effect of the transannular patch. Perhaps this incremental risk also can be neutralized, as Castaneda's work suggests.

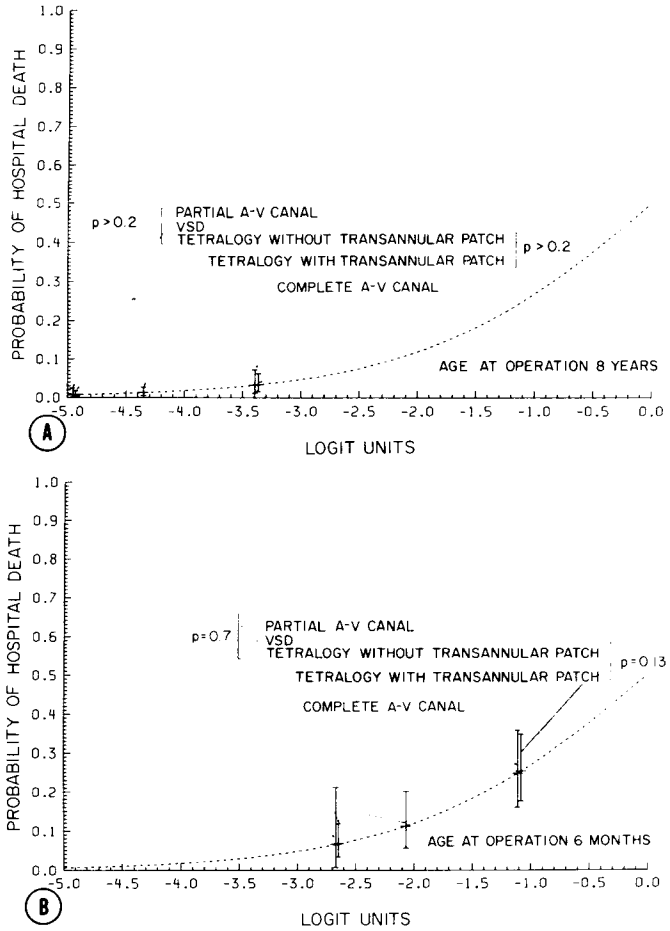

Fig. 6. The point estimates of risk are on the *dotted line* of the basic logistic relation, and the bars represent the 70% confidence limits or one standard deviation. *A*, Differences between the two groups of congenital heart disease are not apparent at an older age (8 years for example), being "masked" by the effect of older age in moving risk well to the left of the area of upswing of the risk lines. *B*, Young age, using 6 months of age as an example, in the series analyzed brings risk closer to the area of upswing of the risk line and thus brings out a difference in risk of complete repair between the two groups. (These series date back to 1972 and do not necessarily reflect current experience). *VSD*, Ventricular septal defect. *A-V*, Atrioventricular.

Now, let's look at the logit units for this experience on the basic nomogram of the logistic equation. Let's study five children (Fig. 6, *A*), each of whom is 8 years old, each with a different malformation, and ask about the probability of hospital death after repair in each during the period studied. The older age of 8 years is going to take all of these patients relatively far to the left on the logit curve and thus away from the point where the probability of hospital death rises steeply. The risk of repair in the patient with a large single VSD at this age was nearly zero during this period, and that of repair of partial AV canal and of tetralogy without transannular patch was not significantly different from it. When we add the incremental risk of a transannular patch in the tetralogy, we still do not get to the steep part of the curve. Thus at this age the risk was not significantly different from the very low risk of repairing tetralogy without a transannular patch.

Now, let's look at the risk of hospital death during the time period of the study in five infants, *6 months old,* undergoing primary repair of the same malformations during this study period (Fig. 6, *B*). The risks of repair of single large VSD, of a partial AV canal, or of a tetralogy without use of a transannular patch were a little higher than at age 8 years, but they were not significantly different one from the other. However, the young age of 6 months brought them more to the right on the logit scale and thus closer to the point where "a straw could break the camel's back." Thus when the incremental risk of transannular patching is added in

Table V. *Congenital heart disease: Study groups for incremental risk factors*

Category	*Coefficient ± S.D.*	*p Value for incremental coefficient of lesion*	*p Value for age-risk slope different from VSD*
ln (age)	−0.82 ± 0.194	p = 0.00002	
Partial AV canal	0 ± 1.2	p = 1.0	0.5
Classical tetralogy (without patch)	0.6 ± 0.76	p = 0.4	0.5
Complete AV canal	1.5 ± 0.68	p = 0.02	0.8
Classical tetralogy (with patch)	1.6 ± 0.64	p = 0.01	0.7
Shunting operations		Slope of curve different	0.07
Total anomalous pulmonary venous connection		Slope of curve different	0.20

Legend: Intercept −1.2 ± 0.64 (p = 0.07) = ventricular septal defect effect.

Table VI. *Possible mechanisms for incremental risks of cardiopulmonary bypass*

Production of vasoactive polypeptides and other "toxins" from denaturation of proteins such as complement (in the oxygenator)
Hypoxic capillary damage (from nonpulsatile flow, profound hypothermia, or total circulatory arrest)
Renal damage (from "toxins," hemoglobin and myoglobin, nonpulsatile flow, circulatory arrest)

the tetralogy, then the probability of hospital death becomes greater (that is, p value = 0.13) than in tetralogy repaired without a transannular patch. The risk was also higher in the 6-month-old patient requiring repair of complete AV canal. We could say that young age unmasks the incremental risks of these two situations, or that older age neutralizes them.

These are data just from one institution, and you might argue that they are *very different* in another institution. Well, we think not. We believe that the results are reasonably representative of the truth *during that time period* in most institutions. We have had the privilege of analyzing large series from several different and well known institutions around the world, and the results are remarkably similar. In fact, when we combine their data with ours, we get larger numbers and thus can draw conclusions more reliably. Doing this, the p value for the difference between transannular patch and no transannular patch in the tetralogy in 6-month-old infants during that time period becomes 0.003, of course highly significant.

Again, one of the purposes of such analyses is to point the way to the research which should be done to neutralize this incremental effect. We begin by asking about the mechanisms within the very young which make them more susceptible to the damaging effects of the support systems for open-heart surgery. Or we could ask what it is about these support techniques, whether they be cardiopulmonary bypass or profound hypothermia and total circulatory arrest, that produces the incremental risk that is unmasked by young age. As regards the latter, we could list a number of things, most of which deserve further intense study and research directed toward their understanding and prevention (Table VI). When this has been successfully done, the age-risk curve should then become flat, just as it is for shunting operations, and the results of intracardiac repair in infants should become still better. Our current experience indicates that this may already have occurred in the case of VSD and no doubt in some institutions in other malformations as well.

Well, Helen I am trying to think about conveying some of these ideas in the Presidential Address. If I do so, I *must* make people understand that these analyses I've been telling you about, the research I've been alluding to, the new knowledge, all of this is for naught if the surgeon cannot translate it into an effective operation. That is the exciting reality about cardiac surgery. The quiet, concentrated, efficient operating room is something beautiful. It is truly the place and the moment which most importantly determines the results of cardiac surgery.

The really quite important advances in many, many aspects of cardiac surgery have changed remarkably the cardiac surgery intensive care units. In earlier times, even 5 years ago, the scene was often one of intense treatment and intervention. Now, it is very different. Its atmosphere is quiet, intense, but warm and friendly. Critical situations still exist, but they can be met *without* frenzy, and yet with dispatch and organized intensity. Indeed, it is in the cardiovascular surgery intensive care unit that we reap the enormous and irreplaceable human rewards for all of our study and research, for all of the science and surgery, and for the

Fig. 7.

hours of postoperative care, as we watch our patients large and small return to their families and to a healthy life ahead (Fig. 7).

Well, Helen, as usual I have gone on much too long. I thank you for reading through this one-sided discussion. It has been a help to me, and I hope out of it I can piece together something for Boston in April.

Love,
Dad

I extend my deep appreciation to all who made the work described and the address itself possible. They were cited individually in the address. Special mention must be made again of the contributions of Eugene H. Blackstone, M.D., Ms. Sandy O'Brien and my cardiovascular surgical colleagues at the University of Alabama Medical Center, Drs. Robert B. Karp, Nicholas T. Kouchoukos, Albert D. Pacifico, and George L. Zorn.

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