Transthoracic resistance in human defibrillation. Influence of body weight, chest size, serial shocks, paddle size and paddle contact pressure.

R E Kerber, J Grayzel, R Hoyt, M Marcus and J Kennedy

Circulation. 1981;63:676-682
doi: 10.1161/01.CIR.63.3.676
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0009-7322. Online ISSN: 1524-4539

Transthoracic Resistance in Human Defibrillation

Influence of Body Weight, Chest Size, Serial Shocks, Paddle Size and Paddle Contact Pressure

RICHARD E. KERBER, M.D., JOSEPH GRAYZEL, M.D., ROBERT HOYT, B.S., MELVIN MARCUS, M.D., AND JEFFREY KENNEDY, B.S.

SUMMARY **Successful defibrillation depends on delivery of adequate electrical current to the heart; one of the major determinants of current flow is transthoracic resistance (TTR). To study the factors influencing TTR, we prospectively collected data from 44 patients undergoing emergency defibrillation. Shocks of 94–450 J delivered energy were administered from specially calibrated Datascope defibrillators that displayed peak current flow, thereby permitting determination of TTR. Shocks were applied from standard (8.5-cm diameter) or large (13 cm) paddles placed anteriorly and laterally. First-shock TTR ranged from 15–143 Ω. There was a weak correlation between TTR and body weight ($r = 0.45, p < 0.05$) and a stronger correlation between TTR and chest width ($r = 0.80, p < 0.01$). Twenty-three patients who were defibrillated using standard 8.5-cm paddles had a mean TTR of 67 ± 36 Ω (± SD), whereas 21 patients who received shocks using paddle pairs with at least one large (13 cm) paddle had a 21% lower TTR of 53 ± 24 Ω ($p = 0.05$, unpaired t test). Ten patients received first and second shocks at the same energy level; TTR declined only 8%, from 52 ± 19 to 48 ± 16 Ω ($p < 0.01$, paired t test). In closed-chest dogs, shocks were administered using a spring apparatus that regulated paddle contact pressure against the thorax. Firmer contact pressure caused TTR to decrease 25%, from 48 ± 22 to 36 ± 17 Ω ($p < 0.01$, paired t test). Thus, human TTR varies widely and is related most closely to chest size. TTR declines only slightly with a second shock at the same energy level. More substantial reductions in TTR and increases in current flow can be achieved by using large paddles and applying firm paddle contact pressure.**

THE ELECTRICAL DOSE required for human defibrillation remains controversial.[1] Although dose is usually quantified by the delivered energy, it is the electrical current flow between the paddles that actually depolarizes a critical amount of myocardium and terminates ventricular fibrillation.[2] Current flow is determined not only by the energy selected, but also by the transthoracic resistance (TTR). In a patient with unusually high TTR, current flow might be inadequate for defibrillation. It would be important to reduce transthoracic resistance if possible. Animal studies have suggested that TTR can be reduced by using large defibrillator paddles and a low-resistance interface between paddles and skin.[3, 4] However, neither the range nor the determinants of TTR have been adequately evaluated in human defibrillation.

In patients undergoing emergency defibrillation, we undertook a prospective investigation of several potentially important factors influencing TTR: body weight, chest size, chest wall thickness, paddle size and the effects of repeated shocks of the same energy level. Another possible determinant of TTR, paddle contact pressure, was studied in shocks applied to animals.

From the Cardiovascular Center and the Department of Internal Medicine, University of Iowa Hospital, Iowa City, Iowa.

Supported in part by NHLBI grant HL-014388 and by a grant from the Datascope Corporation.

Dr. Grayzel's address: 262 Fountain Road, Englewood, New Jersey.

Address for correspondence: Richard E. Kerber, M.D., Department of Medicine, University of Iowa Hospital, Iowa City, Iowa 52242.

Received August 26, 1979; revision accepted July 23, 1980.
Circulation 63, No. 3, 1981.

Methods

All defibrillations were performed using Datascope MD2J damped sinusoidal wave form defibrillators. In this defibrillator, when an energy level is selected the energy that will be delivered into a 50-Ω resistance is displayed; if any charge leaks off, the display indicates the decline. Thus, at the moment the defibrillator was fired, the exact amount of delivered energy was displayed and recorded. After discharge, the peak current (in amperes) that flowed between the paddles was displayed and recorded.

To permit calculation of TTR, each defibrillator was charged to energy settings ranging from 75–460 J, and at each energy level was fired into dummy resistances ranging from 15–150 Ω. The resultant peak current flow for each firing was noted and current vs resistance calibration curves were plotted for each energy level (fig. 1). Thus, knowing the defibrillator used, the energy displayed before firing and the current that resulted permitted us to determine a patient's TTR from each defibrillator's calibration curve.

To evaluate the effect of paddle size on TTR, we equipped, at random, some defibrillators with two standard 8.5-cm-diameter paddles and others with one standard 8.5-cm and one specially constructed 13-cm-diameter paddle, and yet others with two 13-cm paddles. Paddles were coated with Hewlett-Packard Redux paste, a low-resistance interface between paddle and skin,[3] and placed so that the anterior (positive) paddle was centered over the upper right parasternal area and the lateral (negative) paddle was over the cardiac apex. When paddle pairs of unequal size were used, the smaller paddle was always placed over the

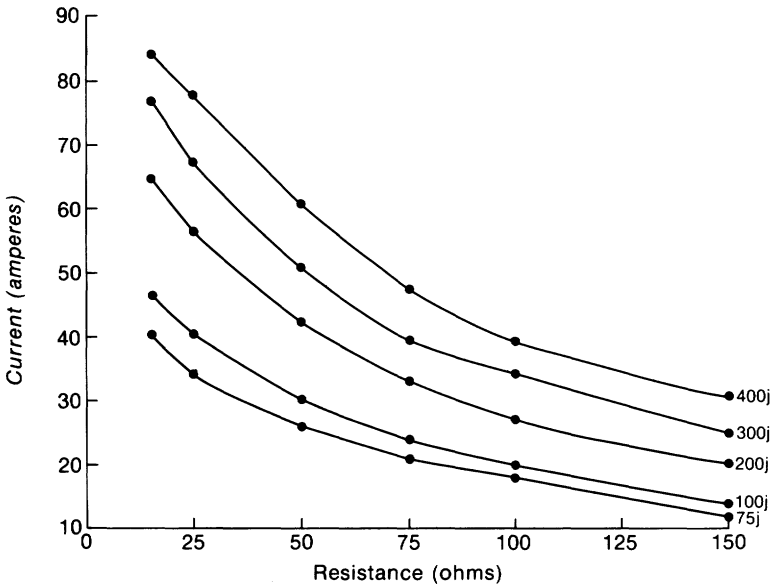

FIGURE 1. *Typical calibration curves for the Datascope MD2J defibrillator, showing peak current flow vs transthoracic resistance at various energy levels. This family of curves was constructed for each defibrillator used by firing the defibrillator into dummy resistances. Knowing the energy displayed before firing and the resultant peak current allowed determination of the patient's transthoracic resistance from such curves.*

cardiac apex. Anteroposterior paddle placement was not used in any patient.

Using a protocol approved by the Human Research Committee of the University of Iowa, we prospectively collected data on 44 patients undergoing emergency defibrillation. Body weights ranged from 20–159 kg (mean 72 kg). The clinical diagnoses of the patients varied widely. Half the patients were known to have cardiac disease: chronic ischemic heart disease (10 patients), acute myocardial infarction (eight patients), valvular heart disease (three patients) and congestive cardiomyopathy (one patient). Fourteen patients had primarily noncardiac disorders, including sepsis (three patients), severe diabetes (two patients), pulmonary embolism (two patients), cerebral vascular accident (two patients), renal failure (one patient), lymphoma (one patient), metastatic carcinoma (one patient), severe lung disease with CO_2 retention (one patient), and chronic osteomyelitis (one patient). In eight patients defibrillated on or shortly after admission to the hospital, insufficient information was available to establish a diagnosis. Physicians administering shocks were advised to select an initial energy dose of 2 J/kg body weight, which would result in an initial shock of 150–200 J for the average adult, a dose shown by others to be effective in most patients.[1] If the first shock failed to defibrillate, the shock was repeated using a dose of 4 J/kg, then 6 J/kg. This protocol was followed in most cases; some patients received several shocks at the same energy dose, which ranged from 100–400 J. In all cases, delivered energy was displayed before firing, and peak current after firing was recorded and used to calculate TTR.

The effects of paddle contact pressure were studied in closed-chest dogs anaesthetized with chloralose-urethane and ventilated mechanically. Contact pressure was assessed in four dogs by designing a paddle-holding apparatus that enabled the operator to adjust the tension of a spring scale connecting the paddle levers and thereby to select paddle contact pressure against the thorax. We estimated light contact pressure with hand-held paddles to be the equivalent of 10 N of tension in the paddle-holding apparatus. Firm pressure was estimated to be equivalent to 50 N of tension, equivalent to a fivefold increase in effective contact pressure. Values of peak current obtained at these tensions were similar to currents obtained in preliminary animal studies that used lightly and firmly applied hand-held paddles. The paddles were coated with Redux paste, mounted in the holding apparatus and applied to a shaved chest. In additional studies in three of these dogs, no paste was used and bare paddles were applied to the shaved skin. Shocks were synchronized to the R wave of the ECG in these dogs and delivered when the lungs were at peak inspiration. Light and firm pressure shocks were given in random order using 8.5-cm or 13-cm paddles and a 20- or 40-J energy dose.

We reasoned that TTR might be related to the physical separation between the paddles, and that this would in turn be related to chest width (i.e., lateral chest diameter), as anterolateral paddle placement was used, and possibly also to the thickness of the chest wall tissues. Therefore, we reviewed chest x-rays, which were available in 29 patients. On the posteroanterior films, we measured the maximal chest width between outermost skin folds. From the lateral chest x-rays (available in 20 patients), we measured the distance between the anterior skin and the manubrium-sternum junction (i.e., anterior chest wall thickness in the region where the anterior paddle was placed).

Statistical Analysis

Linear regression analysis was performed to determine the relationship between first-shock TTR and body weight and that between TTR and the chest x-ray measurements. To determine the effects of pad-

TABLE 1. *Correlations Between Transthoracic Resistance and Its Potential Determinants*

Determinant	Paddle size	n	r	p	Slope	Intercept
Body weight	All sizes	44	0.28	NS	—	—
	Standard*	21	0 45	< 0.05	0.57	29
	Large†	23	0.15	NS	—	—
Chest width	All sizes	29	0.52	< 0.05	3.4	−71
	Standard	14	0.80	< 0.01	5.5	−131
	Large	15	0.51	< 0.05	3.4	−84
Chest wall thickness	All sizes	20	0.45	< 0.05	11.4	25
	Standard	8	0.27	NS	—	—
	Large	12	0.50	< 0.05	14	17

*Two 8.5-cm-diameter paddles.
†Two 13-cm-diameter paddles or one 13-cm and one 8.5-cm paddle.

dle size on TTR, we used an unpaired t test to compare the group shocked with two 8.5-cm paddles and the group shocked with one or two 13.5-cm paddles. We used the paired t test to compare the effect of repeated same-energy shocks in patients and the effects of variable paddle contact pressure on TTR in dogs. All results are reported as mean ± SD.

Results

Range of TTR and Correlation with Potential Determinants (table 1)

The mean first-shock TTR for the whole group ranged from 15–143 Ω (mean 60 ± 31 Ω).

TTR and Body Weight

There was no correlation between TTR and body weight for the group as a whole ($r = 0.28, p$ = NS) or for subgroups of patients who received first shocks at the same energy level. Because TTR is affected by paddle size (see below), we also performed linear regression analysis of subgroups of patients who received shocks from standard paddles only and from one or two large paddles. There was a significant but weak correlation between TTR and body weight ($r = 0.45, p < 0.05$) for the group of patients shocked with two standard-size paddles. Three of 21 patients who received shocks from standard paddles weighed more than 90 kg, as did six of 23 patients who received shocks from large paddles (p = NS).

TTR and Chest Size

Comparison of the TTR values of the entire group with the chest x-ray measurements showed statistically significant but weak correlations between TTR and chest width ($r = 0.52, p < 0.05$) and chest wall thickness ($r = 0.45, p < 0.05$). Separating the patients into two groups based on paddle size substantially improved the correlation with chest width ($r = 0.80$, $p < 0.01$) in patients shocked with two standard paddles (fig. 2).

TTR and Paddle Size

Figure 3 shows first-shock delivered energy and TTR using two standard (8.5 cm) paddles (n = 21) compared with that using one or two large (13 cm) paddles (n = 23). The mean delivered energy received by the two groups was virtually identical: 226 ± 94 J (standard paddles) vs 230 ± 104 J (one or two large paddles). However, the transthoracic resistance of the patients receiving shocks from one or two large paddles was 21% lower: 67 ± 36 Ω (range 16–143 Ω) using standard paddles vs 53 ± 24 Ω (range 15–132 Ω) using large paddles ($p = 0.05$).

We also subdivided the large-paddle group into patients who received shocks from one large and one

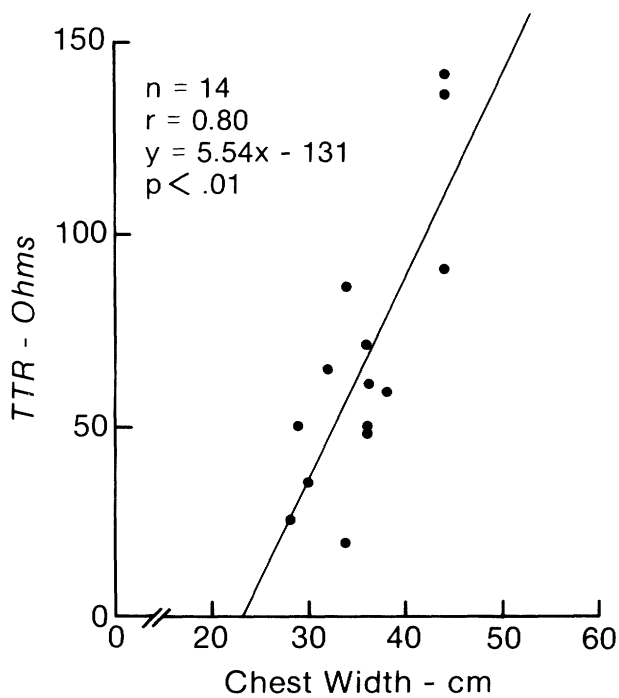

FIGURE 2. *Transthoracic resistance (TTR) vs chest width. A good correlation between these two variables is present for patients shocked with standard size paddles.*

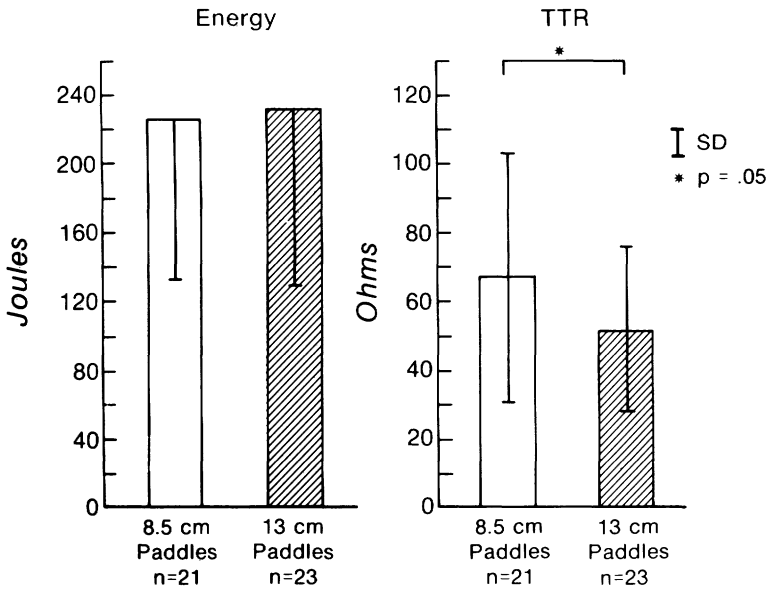

FIGURE 3. *Effect of paddle size on transthoracic resistance (TTR). Both groups received shocks of similar energy levels, but the patients shocked with one or two large paddles had significantly lower TTR (paired t test). 8.5 cm paddles = two standard size paddles; 13 cm paddles = two large paddles or one large and one standard paddle.*

standard paddle (nine patients) and those who were shocked with two large paddles (14 patients). The mean delivered energy of the one large/one standard paddle group was 199 ± 88 J vs 255 ± 109 J for the two large paddle group (p = NS). The corresponding TTRs were 47 ± 19 Ω vs 56 ± 26 Ω (p = NS).

Effect of Repeated Same-energy Shocks on TTR

Ten patients received their first two shocks at the same energy level, which ranged from 100–400 J. The mean energy for both shocks was 235 ± 91 J. Figure 4 plots the TTR and peak current of these two shocks. TTR declined only 8%, from 52 ± 19 to 48 ± 16 Ω ($p < 0.01$); peak current increased only 4%, from 46 ± 16 to 48 ± 16 A ($p < 0.01$). TTR showed no decrease and therefore current flow showed no increase with the second shock in three of these 10 patients.

Effect of Paddle Contact Pressure on TTR

Shocks using light and firm paddle pressures (in random order) were delivered to nonfibrillating dogs using 8.5-cm paddles coated with Redux paste and a 40-J energy dose (2 J/kg body weight). Light paddle contact pressure resulted in a TTR of 48 ± 22 Ω whereas with firm contact pressure TTR was 25% lower, 36 ± 17 Ω ($p < 0.01$) (fig. 5). Peak current flow was 21 ± 8 A with low contact pressure and 23 ± 6 A with firm pressure ($p < 0.01$), a 10% increase (fig. 5). Using large (13 cm) paddles and a lower energy dose (20 J), low contact pressure resulted in a TTR of 42 ± 4 Ω and a peak current flow of 15 ± 1 A. Firm contact pressure resulted in a TTR of 29 ± 1 Ω ($p < 0.01$), a 31% lower value, and a peak current flow of 18 ± 0 A ($p < 0.01$), a 16% increase. Similar decreases in TTR and increases in peak current occurred with firm pressure even when bare paddles were applied to the shaved skin. With 8.5-cm paddles and a 40-J energy dose, TTR decreased from 95 ± 15 to 60 ± 6 Ω ($p < 0.05$) as contact pressure was increased from light to firm. Peak current flow increased from 14 ± 1 to 18 ± 1 A ($p < 0.05$).

Discussion

The main findings of this investigation are (1) the range of TTR in humans is very wide; (2) TTR is weakly related to body weight and more strongly to chest width; (3) TTR is lowered and current flow in-

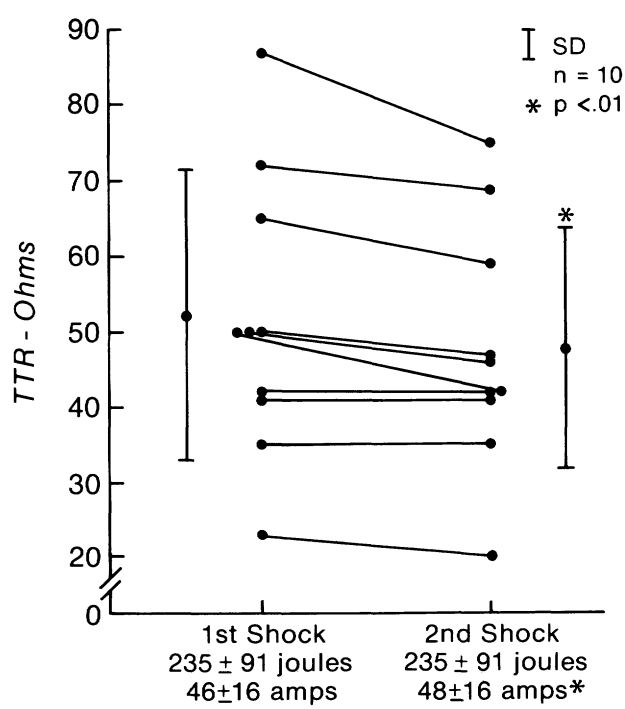

FIGURE 4. *Effect of same-energy shocks on transthoracic resistance (TTR). A second shock at the same energy level resulted in a significant but small decline in TTR and an increase in peak current flow.*

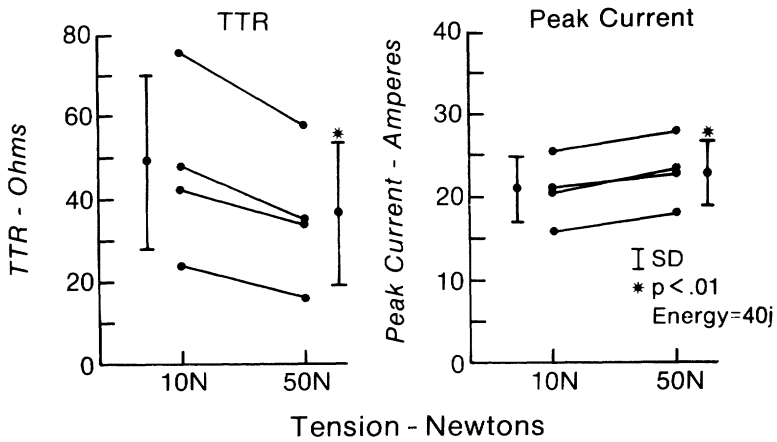

FIGURE 5. *Effects of paddle contact pressure in dogs. Firmer paddle contact pressure against the thorax (higher tension in the spring apparatus) resulted in a significant decrease in transthoracic resistance (TTR) and an increase in peak current flow.*

creased by using paddles larger than those generally manufactured; (4) TTR is lowered and current flow increased by applying paddles firmly to the chest; (5) although TTR is lower during a second shock of the same energy as the first, this decline and the resultant increase in current flow are very small and of questionable clinical significance.

Tacker and co-workers, in animal and human studies,[5-7] found that the energy and current necessary to defibrillate were directly related to body weight and that heavy subjects required higher energies. These investigators suggested that presently available defibrillators may provide inadequate energy and current to defibrillate some heavy patients, and called for the construction of more powerful units. Other workers have vigorously disagreed, finding that present defibrillators are adequate to defibrillate virtually all patients, including heavy ones.[8-10] Assuming that some heavier patients may need higher energies to defibrillate, one possible mechanism consistent with the studies of Tacker et al.,[5-7] is that heavy subjects have higher TTR. Our data show that TTR is weakly related to body weight. However, TTR is more clearly related to chest width, a relationship also noted by Ewy et al.,[11] who studied patients undergoing elective cardioversion with anteroposterior paddles and found a similar relationship ($r = 0.82$) between TTR and anteroposterior chest diameter. If the energy selected is low and therefore marginal for defibrillation,[8] a high TRR might result in inadequate current flow and failure to defibrillate a heavy, big-chested subject.

Although the threshold current for human defibrillation has not been established, we have noted successful defibrillation with peak current flow as low as 0.21 A/kg body weight.[12] Patton and Pantridge[13] found that the mean current required to defibrillate was 0.35 A/kg. Using the latter figure, a 100-kg subject would require a peak current flow of 35 A to defibrillate. Figure 1 shows that a defibrillator capable of delivering 400 J would generate more than 35 A of current across the chest if the transthoracic resistance were less than 130 Ω, which was the case in 41 of our 44 patients. Three patients in our study had TTR greater than 130 Ω; their body weights were 80, 90 and 159 kg. The presumptive current requirements for defibrillation in these three patients, using a threshold of 0.35 A/kg, would be 28 A, 32 A and 56 A. A 400-J defibrillator would generate adequate current to defibrillate the first two of these heavy patients, but would fall short of the current necessary to defibrillate the heaviest patient, who had a TTR of 137 Ω. This theoretical analysis is in ageeement with published data[8-10] indicating that in most patients the widely available 400-J maximal energy defibrillators are adequate, but it suggests that in an occasional very large patient, the current flow from such defibrillators may be insufficient. Because TTR can be decreased by use of large paddles and firm contact pressure, such maneuvers might be of critical importance in a very large patient with high TTR.

In a preliminary communication of ours in 1978,[14] the relationship between TTR and body weight failed to achieve statistical significance. That report was based on 23 patients and is superseded by the expanded number of 44 patients we report now, where the relationships between TTR and body weight and TTR and chest size proved to be statistically significant.

A transthoracic resistance of 50 Ω is assumed when reporting the delivered energy of shocks.[15] Although this figure is useful for standardization of defibrillators, it is a great oversimplification if used to estimate the anticipated current flow, as the range of TTR we encountered varied eightfold, from 15–143 Ω, and averaged 67 Ω for 8.5-cm paddles. Because even the best relationship between TTR and its determinants was only $r = 0.80$ (TTR vs chest width), it is very difficult to estimate accurately how much current will actually flow from the first shock.

Previous studies comparing 13-cm paddles with 8-cm paddles in shocks applied to nonfibrillating anaesthetized dogs, as well as studies of elective cardioversion in humans, showed a lower TTR with large paddles.[4, 16] Our study extends these observations to patients undergoing emergency defibrillation. At any given energy level, use of larger paddles will lower TTR, increase current flow and improve the likelihood of achieving defibrillation. This would be especially important in cases where the current flow is marginal for successful defibrillation, perhaps because

of high TTR. The less concentrated current path resulting from use of large paddles probably also reduces the likelihood of causing myocardial necrosis at higher energy levels.[17] However, overly large paddles could result in a substantial portion of the total current flow traversing extracardiac paths within the thorax, missing the heart and thereby reducing the proportion of current available for defibrillation.[18] Animal studies in our laboratory have shown that *intracardiac* current in 20-kg dogs is increased by using 13-cm rather than 8.5-cm paddles.[19] Moreover, Thomas et al.[20] found that 12.8-cm paddles were more effective than 8-cm paddles in canine defibrillation. Because the human heart is larger than the dog heart, it seems probable that 13-cm paddles would result in increased intracardiac current and improved defibrillation success in humans also. Although this study does not establish the ideal paddle size in humans, it suggests that paddles larger than those presently manufactured should be used.

Although firm paddle contact pressure is advised in defibrillation,[21] an experimental basis for this recommendation has been lacking. This study shows that firm pressure is indeed beneficial because it reduces TTR and increases current flow. It appears that a substantial proportion of total TTR is at the paddle-skin interface. Firm mechanical contact pressure probably reduces TTR by increasing the number of low-resistance electrical contact points between the paddle surface and the skin. A more uniform dispersion of the electrode paste may also occur with higher contact pressure, but firm contact pressure reduced TTR even when bare paddles were used.

Factors of paddle size and paddle contact pressure appear to be additive in reducing TTR. In the same dogs, standard-size paddles applied with light contact pressure yielded a mean TTR of 48 Ω, whereas large paddles applied firmly reduced TTR to 29 Ω. Thus, increasing both paddle size and contact pressure resulted in a combined TTR decline of 40%.

Studies in experimental animals[22, 23] and in patients undergoing elective cardioversion[24] suggested that TTR decreases with repeated shocks at the same energy level. This phenomenon was most evident between the first and second shocks. Chambers et al.[24] suggested that this may explain why electrical conversion from ventricular fibrillation can occur after an initial failure at the same energy level. Although we confirmed that TTR in emergency defibrillation does decrease with a second same-energy shock, the magnitude of this decline was small, and the resultant increase in peak current flow was only 4%. This increment is unlikely to be meaningful in the clinical setting. A substantial increase in current flow can be obtained more reliably and quickly by selecting a higher energy for a second defibrillation attempt. For example, in the 10 patients who received a second shock at the same energy level as the first (235 ± 91 J), the current increased only 4%, from 46 ± 16 to 48 ± 16 A ($p < 0.01$). In contrast, in another nine patients, the delivered energy was increased by 100 J for a second shock, from 231 ± 52 to 329 ± 54 J. This resulted in a 25% increase in current flow, from 50 ± 14 to 62 ± 14 A ($p < 0.001$, paired t test).

In this report, we included data from shocks whether or not the shocks were successful in terminating ventricular fibrillation. Defibrillation success is influenced by many factors. In addition to adequate current passing through the heart, other factors that have been proposed as influencing defibrillation success include the duration of ventricular fibrillation,[8, 25] metabolic abnormalities[25] and the cardiac diagnosis and state of the myocardium.[8, 10, 26] Thus, although reducing TTR and increasing current flow should improve the chances of successful defibrillation, this single factor is only one of several determinants.

We conclude that in human defibrillation TTR varies widely and is best related to chest size. TTR can be substantially reduced and peak current flow increased during defibrillation by using large paddles and firm paddle contact pressure. These maneuvers will maximize current flow from presently available defibrillators. However, repeating an initially unsuccessful shock at the same energy level causes only minimal changes in TTR and peak current. Therefore, an initially unsuccessful shock should be quickly followed by a second shock at a higher energy level to increase current flow substantially and avoid delays in achieving defibrillation.

References

1. Lown B, Crampton RS, DeSilva RA, Gascho J: The energy for ventricular defibrillation — too little or too much? N Engl J Med **298:** 1252, 1978
2. Zipes DP, Fischer J, King RM, Nicoll A de B, Jolly WW: Termination of ventricular fibrillation in dogs by depolarizing a critical amount of myocardium. Am J Cardiol **36:** 37, 1975
3. Ewy GA, Tapen D: Impedance to transthoracic direct current discharge: a model for testing interface material. Med Instrum **12:** 47, 1978
4. Ewy G: Defibrillator paddle electrodes. *In* Proceedings of the Cardiac Defibrillation Conference, West Lafayette, Indiana, Purdue University, 1975, pp 39–44
5. Tacker WA, Galioto TM, Giuliani E, Geddes LA, McNamara G: Energy dosage for human trans-chest electrical ventricular defibrillation. N Engl J Med **210:** 214, 1974
6. Geddes LA, Tacker WA, Rosborough JP, Moore AG, Cabler PS: Electrical dose for ventricular defibrillation of large and small subjects using precordial electrodes. J Clin Invest **53:** 310, 1974
7. Collins RE, Giuliani ER, Tacker WA, Geddes LA: Transthoracic ventricular defibrillation: success and body weight. (abstr) Med Instrum **12:** 53, 1978
8. Campbell NPS, Webb SW, Adgey AAJ, Pantridge JF: Transthoracic ventricular defibrillation in adults. Br Med J **2:** 1379, 1977
9. Gascho JA, Crampton RS, Sipes JN, Cherwek ML, Hunter FP, O'Brien WM: Energy levels and patient weight in ventricular defibrillation. JAMA **242:** 1380, 1979
10. Gascho JA, Crampton RS, Cherwek ML, Sipes JN, Hunter FP, O'Brien WM: Determinants of ventricular defibrillation in adults. Circulation **60:** 231, 1979
11. Ewy GA, Ewy MD, Silverman J: Determinants of human transthoracic resistance to direct current discharge. (abstr) Circulation **46** (suppl II): II-150, 1972
12. Kerber RE, Grayzel J, Gascho J, Kennedy J, Hoyt R: What are the current requirements for human defibrillation? (abstr) Med Instrum **14:** 56, 1980
13. Patton JN, Pantridge JF: Current required for ventricular

defibrillation. Br Med J **1:** 513, 1979
14. Kerber R, Hoyt R: Human transthoracic resistance in emergency defibrillation: range and relationship to body weight. (abstr) Circulation **57** (suppl II): II-204, 1978
15. Standards for Cardiopulmonary Resuscitation and Emergency Cardiac Care. JAMA **227:** 837, 1974
16. Connell PN, Ewy GA, Dahl CF, Ewy MD: Transthoracic impedance to defibrillator discharge. Effect of electrode size and electrode-chest wall interface. J Electrocardiol **6:** 313, 1973
17. Dahl CF, Ewy GA, Warner ED, Thomas ED: Myocardial necrosis from direct current countershock. Effect of paddle electrode size and time interval between discharges. Circulation **50:** 956, 1974
18. Ewy GA: Effectiveness of direct current defibrillation: role of paddle electrode size: II. (letter) Am Heart J **93:** 674, 1977
19. Hoyt R, Grayzel J, Kerber R: Intracardiac current in defibrillation. Effect of paddle size. (abstr) Circulation **59** (suppl II): II-192, 1979
20. Thomas ED, Ewy GA, Dahl CF, Ewy MD: Effectiveness of direct current defibrillation: role of paddle electrode size. Am Heart J **93:** 463, 1977
21. Parker MR: Defibrillation and synchronized cardioversion. *In* Advanced Cardiac Life Support. Dallas, American Heart Association, 1975
22. Geddes LA, Tacker WA, Cabler DP, Chapman K, Rivera BS, Idder H: Decrease in transthoracic resistance during successive ventricular defibrillation trials. Med Instrum **9:** 179, 1975
23. Dahl CF, Ewy GA, Ewy MD, Thomas ED: Transthoracic impedance to direct current discharge: effect of repeated countershocks. Med Instrum **10:** 151, 1976
24. Chambers W, Miles R, Stratbucker R: Human chest resistance during successive countershocks. (abstr) Circulation **56** (suppl III): III-183, 1977
25. Kerber RE, Sarnat W: Factors influencing the success of ventricular defibrillation. Circulation **60:** 226, 1979
26. Tacker WA, Geddes LA, Cabler PS, Moore AG: Electrical threshold for defibrillation of canine ventricles following myocardial infarction. Am Heart J **88:** 476, 1974

Oral Prostaglandin E_2 in Ductus-dependent Pulmonary Circulation

ERIC D. SILOVE, M.D., J. YASHU COE, M.B., M. F. SHIU, M.D., JANE D. BRUNT, B.SC., ANTONY J. F. PAGE, M.B., SHYAM P. SINGH, M.B., AND MURRAY D. MITCHELL, M.A., D. PHIL.

SUMMARY **Prostaglandin E_2 (PGE_2) was administered orally, in doses of 12–65 μg/kg at intervals of 1–4 hours, to 12 neonates in whom the pulmonary circulation depended on patency of the ductus arteriosus. After an oral dose, both oxygen saturation (Sa_{O_2}) and plasma PGE_2 concentration increased consistently within 15–30 minutes, reaching values comparable to those during i.v. infusions. Treatment continued for 5 days to 4 months. In eight infants, PGE_2 withdrawal resulted in a decrease of Sa_{O_2}, from a mean of 75 ± 7% to 57 ± 10% (± SD).**

The ductus remained responsive for long periods — in four infants, for over 3 months. Consequently, surgery could be delayed until the infants and their pulmonary arteries had grown. Side effects during oral therapy were similar to those during i.v. infusion but were less severe in this series. The effectiveness and simplicity of oral PGE_2 administration have advantages over i.v. administration, especially for long-term treatment.

INFUSIONS of the E-type prostaglandins are widely used to maintain patency of the ductus arteriosus in neonates with severely reduced pulmonary blood flow.[1-7] Therapy usually continues for hours or days; the longest reported course of i.v. therapy has been 29 days in one infant.[7] We have briefly described the efficacy of long-term oral prostaglandin E_2 (PGE_2)[8-10] and now report our experience of oral therapy in 12 patients. In particular, we tested (1) whether oral PGE_2 consistently maintained ductus patency; (2) whether oral PGE_2 could easily be substituted for i.v. therapy; (3) the requirements of dosage and frequency of administration; and (4) whether the ductus remained PGE_2-dependent after a period of months.

Patients and Methods

This study was approved by the Research Ethical Committees of both the Children's Hospital and the Central Birmingham Health District. Informed parental consent was obtained in each case.

Twelve infants with severely diminished pulmonary blood flow were treated with oral PGE_2. Their mean weight was 2.90 kg. The clinical features are given in table 1. In patients 2, 3, 4, 6, 11 and 12, the surgeons considered the pulmonary arteries, as shown by angiography, to be too small to attempt a shunt operation. We hoped that prolonged treatment would encourage growth. In patients 1 and 7, PGE_2 therapy was restarted after failure of a palliative operation.

From the Department of Pediatric Cardiology, Children's Hospital, Ladywood Middleway, Birmingham, and Nuffield Department of Obstetrics and Gynaecology, University of Oxford, John Radcliffe Hospital, Oxford, England.

Presented in part at the 52nd Scientific Sessions of the American Heart Association, Anaheim, California, November 1979.

Dr. Mitchell is a Medical Research Council Senior Research Fellow.

Address for correspondence: Dr. E. D. Silove, Children's Hospital, Lady Middleway, Birmingham B16 8ET, England.

Received October 9, 1979; revision accepted July 11, 1980.
Circulation 63, No. 3, 1981.