UNIVERSITY OF ALBERTA

AN EVALUATION OF A DECISION TREE FOR CLINICAL PRACTICE

BY

FLORENCE MELCHIOR-MACDOUGALL ©

A THESIS SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

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October 15, 1992

Dear Florence McDougall:

Thank you for your interest in the Chronic Wound Management Decision Tree. I will give you permission for one time use of the tree as a decision tool for your Master's Thesis.

I would appreciate a summary of the results of your research and would welcome any suggestions you may have for tool improvement.

Sincerely

Cheryl L. Knight

Cheryl L. Knight, RN, MN
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January 4, 1993
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Florence McDugal
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Dear Florence,

I regret to inform you that I cannot locate the negatives for the photos which you asked for. You may, however, reproduce the photos if you wish. You may wish try photocopying or having a professional photographer retake the photos from the copies you have.

You have my permission to copy the photos and replicate the study, A Comparison Of A Decision Tree With Other Wound Care Decision Support Tools: Effects On Accuracy And Level Of Confidence. If I can be of further assistance please let me know. I'll try to be more prompt in answering next time.

Sincerely,

Terry Willey

Terry Willey, RN MN CETN

UNIVERSITY OF ALBERTA

RELEASE FORM

NAME OF AUTHOR: Florence Melchior-MacDougall

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(Signed) F MacDougall

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DATE: April 22, 1993

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THE UNDERSIGNED CERTIFY THAT THEY HAVE READ, AND RECOMMEND TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH FOR ACCEPTANCE, A THESIS ENTITLED "AN EVALUATION OF A DECISION TREE FOR CLINICAL PRACTICE" SUBMITTED BY FLORENCE MELCHIOR-MACDOUGALL IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF NURSING.

Dr. Janice Lander(Supervisor)

Dr. Janet Kerr

Dr. Geoff Taylor

DATE: April 16, 1993

To my son, Logan, my sister, Marie and

my brother, Michael

ABSTRACT

Nurses routinely make complex clinical decisions under conditions of uncertainty. They collect large amounts of unmanageable data in the process of making clinical decisions. To assist nurses in collecting and organizing data and making complex clinical decisions, some nursing scholars recommend decision support systems. A decision tree is a support system that leads the nurse from general to specific assessments and ultimately a decision choice or outcome. A decision tree was examined, in this study, for its utility in promoting accuracy in decision making for chronic wound management among home care nurses. It was found that home care nurses who used the decision tree made better decisions about staging and product choices for chronic wounds. More research is necessary to discover if chronic wound management decision trees translate into improved patient outcomes.

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Decision making is the most crucial clinical function that the professional nurse performs (Aspinall, 1976; Casebeer, 1991; Hughes & Young, 1990; Jones, 1988; Wurzbach, 1991). Uncertainly is inherent in clinical decision making because the nurse has few instruments of uncertainty (eg. thermometer, sphygmomanometer) to collect data from which decisions can be made. Using perceptual processes, the nurse must make inferences from multiple, uncertain patient cues about the intangible condition of the patient and the correct decisions for patient care. Therefore, arriving at a clinical decision is a cognitively complex task undertaken by the nurse in an uncertain environment (Carnevali, 1984; Hammond, 1966; Jones, 1988).

The decision making process incorporates a number of steps wherein incoming information is assimilated, interpreted and appraised. In nursing, the steps in the decision making process include decisions about (a) what cues are relevant to the patient situation, (b) what the cues mean (ie. diagnosis) and (c) what course of action will lead to the desired patient outcome (King, 1991; Prescott, Dennis, & Jacox, 1987). The nursing process is the outline that nurses have used to define the steps of the decision making process (Grier, 1984; Tanner, 1983).

Research has demonstrated that nurses collect large amounts of data, which may not always be relevant, before making a clinical decision (Hammond, 1966; Grier, 1984). These large, unmanageable data sets hinder quick decision making and can lead to less than optimal decisions (Brennan, 1990; King, 1991). Benner (1984) studied the decision making process used by expert nurses and found that expert nurses were able to immediately grasp the whole allowing for decisions that were automatic and intuitive. She demonstrated that expert nurses, in a familiar environment, were able to fluidly and efficiently make complex clinical decisions without conscious analysis. However, in an unfamiliar environment or when the patient situation was not evolving as exected, the expert nurse needed to revert to a more analytical mode of decision making. Hughes and Young (1990) found that consistency of decision making decreased when the complexity and uncertainty of the patient situation increased. They concluded that decision support systems are beneficial when there are clinically complex decisions to be made.

Decision theory employs models of mathematical probability to describe or prescribe the decision making process. In application, decision theory is most often used to recommend how decisions should be made; decisions are made by (a) weighing assessments in order to arrive at a diagnosis and (b) choosing an intervention that has the highest probability of a favourable outcome (Le Breck, 1989; Elstein et al., 1983; Grier, 1976; Tanner, 1986).

Decision analysis, first described by Raiffa (1968), is an approach to decision making that has been derived from decision theory. Using decision analysis, complex diagnostic tasks are broken down into smaller more manageable components that can be individually analyzed. The tool of decision analysis, a decision tree, prescribes logical steps that lead from the general to the specific describing all possible choices and outcomes (Greep & Siezenis, 1989; Lanza & Bently, 1991; Raiffa, 1968; Skewchuk & Francis, 1988; Tanner, 1983).

Decision trees employ multiplicative corroboration in that one facet of the situation is established before the decision maker (in this case, a nurse) moves on to another. Decision trees begin with a binary choice of assessment at the top, left-hand corner of the page. Each subsequent binary choice (or node) leads the nurse along a path that eventually leads to a course of action or an outcome. A decision tree, therefore, directs the nurse in collecting and organizing relevant data in order to make assessments and plan nursing care (Coleman, 1989; Jones, 1988; Skewchuk & Francis, 1988). Because the nurse must exercise subjective judgement at each node, decision trees do not substitute for the knowledge and expertise of the nurse, but instead enhance and augment the nurse's decision making skills and strategies (Brennan, 1990; O'Leary, 1992; Ryan, 1985).

Although not widely studied in nursing, results indicate that decision trees are useful for nursing practice. Research has demonstrated that they improve accuracy of decision making, and therefore decision trees may improve patient care. Aspinall (1979) studied the utility of decision trees in improving accuracy of diagnoses among nurses. She found that the nurses who used a decision tree were more accurate at making a diagnosis about a disease state (cirrhosis of the liver) than those who did not. Willey and Swords (1991) compared the utility of chronic wound care decision trees with other chronic would care decision support tools. They found that nurses using decision trees and narrative decision support made more accurate diagnoses than nurses using other or no decision support tools. Willey and Swords also found that nurses who used decision trees to make decisions about chronic wound care management were more confident in the decisions that they made. Recently, other decision making tools for clinical nursing care have been introduced. In 1992, Mantle used a decision tree to successfully manage constipation among clients in an extended care unit. In an intensive care unit, Van Den Berg and Visinski (1992) used a decision tree to help nurses decide when to draw arterial blood gases from patients. They found that when the decision tree was used on the unit, less arterial blood gases were drawn than when it was not.

Because chronic wound care is complex and embodies multiple variables, a decision tree may be useful in reducing the complexity of chronic wound care management to more simple rules of procedure. A summary of the literature on chronic wounds will be presented here (the full review is located in Appendix A).

There is a body of knowledge that nurses can use to formulate standards for assessing and treating chronic wounds. The treatment of chronic wounds was revolutionized in the early 1960's with Winter's (1962) research on moist environments for healing. Prior to that time, wound care and dressings had not changed much since the 1600's (Alvarez, Rozint, & Wiseman, 1989). Winter's work was corroborated and advanced by later studies (ALvarez, Mertz, & Eaglestein, 1983; Bothwell & Rovee, 1971; Hinman & Maibach, 1963; Katz, McGinley, & Leyden, 1986; Mertz & Eaglestein, 1984; Rovee, Kurowsky, & Labun, 1972: Winter & Scales, 1963). The past 30 years have seen an explosion in the number of dressings and products available to ensure moist, occlusive environments for chronic wound healing. In the 1960's transparent adhesive films were introduced, whereas in the 1980's hydrocolloids, hydrogels, polyurethane foams, and absorptive dressings were developed and marketed.

In addition to advances in dressings and dressing products for chronic wounds, the assessment of chronic wounds has been standardized. The International Association for Enterostomal Therapy (1987) and the National Pressure Ulcer Advisory Panel (1989) have adapted Shea's (1975) categorization of pressure ulcers to encompass all chronic wounds. A Stage I wound appears as an acute inflammation without a break in the skin. A Stage II wound is characterized by partial thickness epidermal or dermal loss. The ulcer is shallow with a painful, pink, moist base. Stage III wounds involve full thickness skin loss with damage or necrosis of subcutaneous tissue. The ulcer is deep, but does not extend to underlying fascia. There may be exudate with or without infection, necrotic tissue and sinus tract formation. A Stage IV wound is characterized by full thickness skin loss with extensive damage to underlying fascia, and possibly tendons, bones and joints. The ulcer presents as a deep crater and may include necrotic tissue or eschar, tracking and tunnelling and exudate with or without infection. The base is usually not painful. Although there are only four stages to describe a chronic wound, a fifth stage ("unable to assess wound in present state") is added by practitioners to encompass wounds that have large amounts of concealing exudate or non-viable tissue.

The extent of information which is available and the complexity of the clinical problem lead to inadequate and inconsistent chronic wound management. Because of difficulty managing wounds in a home care program, a decision tree was developed. In this study, the utility of this chronic wound decision tree was examined for its efficacy in promoting accuracy of decision making in the assessment and dressing product choice among home care nurses. It was postulated that the results form this study would replicate the findings of Willey and Swords (1991). However, the decision tree used by Willey and Swords was not to be used in this study because the Edmonton Board of Health Home Care Program has wound care protocols that are different from the protocols used by Willey and Swords. The results from this study add to nursing knowledge on clinical decision making in chronic wound management.

METHOD

Sample and Setting

All of the home care nurses n=94 from the Edmonton Board of Health Home Care Program were asked to participate in this study. These nurses are responsible for making home visits to patients with various health problems. Home care nurses provide a variety of services (eg. dressing changes, pre-loading of insulin syringes and health education). The goal of the home care program is to keep people in the community and in their homes.

Instruments

The chronic wound management decision tree, which was used for this study, was formulated by Knight (1992) (see Appendix B). This decision tree has been reviewed by a panel of experts from across North America and has been deemed an accurate representation of current chronic wound management practice.

The wound assessment materials (see Appendix C) consisted of: (a) photographs of three patients with chronic wounds, (b) brief descriptions of the patient's state of health and (c) questions about stage of wound and appropriate dressings. Lists of dressing products were not included with the wound assessment materials because Willey and Swords (1991) found that the use of a dressing list did not effect accuracy of decision making on product choice. The photographs that were used in this study are the same photographs used by Willey and Swords (1991), and they were used with their permission. In a first phase of Willey and Swords study, eight photographs representing the five stages of chronic wounds were distributed to a panel of experts who were asked to identify a dressing that would be the most effective for maximizing and hastening wound healing for each wound. From these photographs, three were selected that represented a Stage I, Stage III, and a Stage V wound, and it is these three photographs that were used in this study.

Procedure

At the time that this study took place, all home care nurses were gathered together for an educational session on chronic wound management. Prior to the educational session, an introductory letter was sent to all the home care offices inviting the nurses to participate in this study. On arrival at the auditorium where the session was to take place, the home care nurses were randomly assigned to control or experimental group by random sorting of research materials. They were given these materials when they entered the auditorium and then were shown where to sit. The control group sat at the front of the auditorium while the experimental group sat at the back. Included with the research materials was an information sheet (see Appendix D), which informed the home care nurses that they could choose to participate or not participate and that their responses would be confidential and anonymous.

After everyone was seated, the researcher asked both groups to complete the biographical questionnaire (see Appendix E) which was included with the research materials. When the home care nurses had completed the questionnaire they were asked to place it in its separate envelope and pass it to the end of their row for collection.

Then, the nurses were asked to complete a test which focused on the nurse's ability to accurately assess and choose a dressing product for the three chronic wounds. From the photograph and the information given, the nurses were asked to stage each chronic wound using the standards accepted by the Edmonton Board of Health Home Care Program which reflect the standards set by the National Pressure Ulcer Advisory Board Panel and the International Association for Enterostomal Therapy. Descriptions of the stages were included in the test. Secondly, the nurses were asked to choose an appropriate dressing product for each of the three chronic wounds. The experimental group was provided with the chronic wound management decision tree and brief instructions. The control group were treated identically to the experimental group except that they were not provided with the decision tree. The nurses were allowed 20 minutes to complete the test. Included in the test was a question on the number of chronic wounds cared for each week by the participating home care nurse.

In the second phase of the study, the photographs and cases were assessed by five local practitioners to confirm the accuracy of responses in staging and product choices. They were asked to rate the product choices in the same manner as described by Willey and Swords (1991). They rated each product choice as: (a) absolutely contraindicated, (b) inappropriate, (c) neither appropriate or inappropriate, (d) appropriate or (e) most appropriate. The practitioners were also asked to stage each of the three chronic wounds according to the descriptions included in the test given to the home care nurses.

Design

An after-only experimental design was chosen for this study. The independent variable was the presence or absence of the decision tree. Accuracy of responses to the test questions was the dependent variable. The hypotheses of the study were:

1. Home care nurses who use a chronic wound management decision tree will make a greater number of accurate assessments of wound stage than home care nurses who do not use the decision tree.
2. Home care nurses who use a chronic wound management decision tree will make a greater number of accurate dressing choices than home care nurses who do not use the decision tree.

It was assumed that the two groups would be comparable on biographical variables because of random assignment.

RESULTS

Sample Characteristics

A total of 94 home care nurses were in attendance for the educational session, and all were asked to and did participate in this study. Forty-seven nurses were randomly assigned to each group. The biographical characteristics of the sample are described by group in Table 1. Overall, home care nurses reported a mean of 5.6 (SD 4.3) years of nursing experience in home care and a mean of 15.5 (SD 8.7) years of total nursing experience. The mean number of wounds cared for in a week by the home care nurses was 1.7 (SD 1.7).

Types of nursing education was not significantly different for the two groups (chi-square analysis). Amount of nursing experience in home care and total amount of nursing experience were also not significantly different for group (t-test).

Accuracy of Decision Making

Staging.

Subject's accuracy for staging of wounds was assessed separately for each of the three cases. The results, broken down by case and group, are presented in Table 2. For the Stage I Wound presented in the

Table 1. Characteristics of Sample by Group[1]

Variable	Control (n=47)	Group Experimental (n=47)	All Subjects (n=94)
1. Education			
Diploma	7(14.9%)	5(10.6%)	12(12.7%)
Basic Degree	16(34%)	18(38.3%)	34(36.2%)
Post-RN Degree	18(38.3%)	19(40.4%)	37(39.4%)
Other	6(12.8%)	5(10.6%)	11(11.7%)
2. Amount of Nursing Experience in Home Care (in years)			
Range	0-24.9	.08-20	0-24.9
Mean	5.4	5.8	5.6
SD	4.6	4.1	4.4
3. Total Amount of Nursing Experience (in years)			
Range	2-33	2-40	2-40
Mean	15.2	15.7	15.5
SD	9.0	8.4	8.7
4. Number of Chronic Wounds Cared for in a Week			
Range	0.6	0-6	0-6
Mean	1.6	1.7	1.7
SD	1.7	1.8	1.7

[1]Groups not significantly different for these characteristics.

Table 2. Frequency of Accurate Staging Choices for the Three Cases

	Case 1		Case 2[1]		Case 3		Total
	Control	Exp.	Control	Exp.	Control	Exp.	
Accurate	19[2] (.40)	27 (.57)	16 (.34)	33 (.70)	7 (.15)	3 (.06)	105
Inaccurate	28 (.60)	20 (.43)	31 (.66)	14 (.30)	40 (.85)	44 (.94)	177
Total	47	47	47	47	47	47	282

1 Chi-square=10.91 df=1 p<.001
Phi-coefficient=.36 p<.001

2 Proportions in parentheses

first case, 19 nurses in the control group and 27 nurses in the experimental group accurately staged the wound. The chi-square statistic used to compare the two groups was not significant.

For the Stage III Wound presented in the second case, 16 nurses in the control group and 33 nurses in the experimental group accurately staged the wound. This resulted in a significant difference between the two groups (Chi-square=10.91 df=1 $p<.001$). There were more nurses in the experimental group and fewer in the control group who accurately staged the wound than were expected.

The wound presented in the third case was covered with non-viable tissue and ought to be staged in that condition. Seven nurses in the control group and three nurses in the experimental group were accurate. The chi-square statistic comparing the two groups did not reach significance.

Overall, the rate of accurate staging was 48/94 and 49/94 for Case 1 and 2 respectively. For Case 3, the accuracy rate was 10/94.

Product Choice.

When the product choices made by the nurses in this study were assessed by expert local practitioners, there was general agreement about adequacy of the products named for the three cases. Using the assessment of the local practitioners and a further review of the relevant literature about the products, the subject's responses were then rated and placed into one of the following six categories: (a) no response or "I don't know", (b) absolutely contraindicated, (c) inappropriate, (d) appropriate or (f) most appropriate (see Table 3). Because 9 of the 18 cells had less than the required frequencies for statistical analyses, the categories were collapsed into two categories: accurate and inaccurate product choices. Accurate product choices included those choices that were appropriate or most appropriate. Inaccurate product choices included all other options.

Accuracy of product choice was analyzed separately for each of the three cases. Significant group differences were found only for Case 1 (Chi-square=5.20 df=1 $p<.05$). The frequency of an accurate choice was significantly greater than expected for the experimental group and less than expected for the control group.

Table 3. Frequency of Product Choices for the Three Cases

		Case 1[1]		Case 2		Case 3		Total
		Control	Exp.	Control	Exp.	Control	Exp.	
Acc	Most approp	4	19	0	4	5	11	43
	Appropriate	16	13	18	18	23	15	103
	Total	20[2]	32	18	22	28	26	146
		(.42)	(.68)	(.38)	(.47)	(.60)	(.55)	
Inacc	Neither	20	14	17	23	3	9	86
	Inapprop	3	1	7	1	11	10	33
	Abs. Contra	1	0	1	0	0	0	2
	No response	3	0	4	1	5	2	15
	Total	27	15	29	25	19	21	136
		(.58)	(.32)	(.62)	(.53)	(.40)	(.45)	
Total		47	47	47	47	47	47	282

1 Chi-square=5.20 df=1 p<.05; Phi-coefficient=.26 p<.01
2 Proportions in parentheses

Comparison of Total Number of Accurate Decisions

Scores were assigned for each accurate staging and product selection decision. Nurses could achieve scores from 0-6 (see Table 4). Mean total score was 2.7 (SD 1.3). The two treatment groups were compared using analysis of variance (ANOVA). The test revealed a significant difference between the two groups ($F=8.7$ $df=1,92$ $p<.01$). The nurses in the experimental group made a significantly greater number of accurate decisions (mean 3.1 SD 1.3) when compared to the nurses in the control group (mean 2.3 SD 1.2).

When the number of wounds cared for per week was correlated with total scores, the correlation was near zero and non-significant ($r=.02$).

DISCUSSION

The results of this study indicate that decision trees are useful in helping nurses to make complex clinical decisions. The use of a decision tree for chronic wound management led to better decisions about staging and product choice among home care nurses in this study. However, the decision tree was not absolutely effective in guiding decision making as some

Table 4. Measures of Central Tendency for Total Accuracy Score by Group

	Group	
	Control	Experimental
Range	0-4	1-5
Mean	2.3	3.1
SD	1.2	1.3
Confidence Interval for Mean	1.96-2.66	2.69-3.44

F-ratio=8.7 df=1,92 $p<.01$

nurses made errors even when employing it.

There are several explanations for the failure of the decision tree to result in error-free decision making. One is the lack of thorough instruction on the use of the decision tree. This was the first time that the participating nurses had been exposed to the decision tree and instructions given to them on its use were very brief. Thus, errors they made about staging of wound and product choice may have been due to lack of familiarity with the tree.

Another explanation that is plausible is that the participants were asked to make decisions based on photographs of wounds. Thus, their view of the wounds was rather limited. The decision tree may more accurately guide decision making when it is used in an actual clinical situation. The control group, of course, might also have improved decision making when faced with an authentic wound.

The third and most plausible explanation is that nurses did not have the necessary knowledge and assessment skills to use the decision tree appropriately. Decision trees cannot be a substitute for knowledge and skill; they are an adjunct to basic skills. The nurses in this study were attending an educational session on chronic wound management to obtain basic skill in chronic wound management. Their need for knowledge was supported by the findings of this study because they had difficulty assessing and staging chronic wounds.

The type of staging errors that were made by the nurses were the same for the first two cases. The nurses tended to overstage the wound, that is, assess it as a Stage II instead of a Stage I and a Stage IV instead of a Stage III Wound. For the chronic wound in Case 3, most nurses assessed the wound as if it was not covered by non-viable tissue. Because the stage "unable to stage wound in present state" was self-explanatory, this reflected a lack of knowledge about chronic wounds.

For the chronic wound in Case 2, a significantly greater number of nurses in the experimental group accurately staged the wound. But when product choices for the wound were compared, no significant differences were found between the two groups. Most nurses who inaccurately staged the wound assessed it as a Stage IV Wound. Because the accurate product choices for a minimally draining Stage III and Stage IV Wound are similar, this may account for the large number of accurate product choices despite the number of inaccurate staging choices.

In Case 3, only 10 of the nurses accurately staged the wound, yet there were more accurate product choices for this case than either of the other two. Although the nurses could not accurately stage the wound, they were cognizant that the non-viable tissue had to be removed before healing could occur. This was evident by the product choices that they made.

The experience of caring for chronic wounds did not necessarily improve accuracy of decision making. Home care nurses who had experience with chronic wounds were no more accurate than their inexperienced counterparts. Apparently, in this field, experience alone does not translate into accurate decisions. Therefore, knowledge as well as experience is necessary for accurate decision making.

This chronic wound management decision tree was found to be useful in a controlled setting, but whether or not its use will translate into improved patient outcomes cannot be accurately determined from this study. Further study in the clinical field is necessary before conclusions can be made about the efficacy of decision trees in improving patient outcomes. As well, nurses from home care were used as subjects. Whether or not the same results would be obtained with hospital-based nurses cannot be ascertained from these data. However, Willey and Swords (1991) used nurses from hospitals in their study and their results were similar to results from this study.

The use of photographs, minimal patient information and the group setting differs from that which would be found in a real clinical situation. The nurses were allowed a specific period of time to complete the questionnaires and were not allowed to ask questions about the cases or confer with their colleagues. In a clinical situation, the nurse could ask questions of the patient and perhaps discuss the case with other nurses. Because the situation was different from the clinical situation, then nurses may have been making decisions under duress which may have hampered their decision making skills.

Multiple analyses were conducted with staging and product choice data. Although chi-square analysis detected a difference on only two out of six decisions, an ANOVA on overall score revealed a significant difference between the two groups. Because of multiple comparisons (staging score, product score and total score), there was an increased chance of making a Type I error in the analyses of these data (Cook & Campbell, 1979). However, when a new significant F-ratio was computed, taking the change in alpha into consideration, a significant difference was still detected between the means of the two groups ($p<.01$).

The differences in mean total scores between the two groups was close to one accurate decision out of six possible accurate decisions. Although this may not appear to be a big difference on first appraisal, it is a significant difference considering that this was the first time that these nurses had used the decision tree. In addition, one more accurate decision means one less inaccurate decison, and this is certainly of importance to the clients that the home care nurses serve. Besides the increased benefits to the clients that home care nurses serve, the use of a decision tree may also be cost effective. From the data, it was apparent that nurses who were using the decision tree chose products that would hasten healing, thus ultimately requiring fewer dressing changes. When nurses made incorrect choices, the products they chose were often more expensive than appropriate ones.

This decision tree was found to be effective overall in guiding nursing decisions in this study which was conducted before an educational sesssion was held. It would be interesting to determine the value of the decison tree with and without an educational session.

The question about whether decision trees ought to be used in other fields of nursing arises. Some forms of decision trees have already been used in triage and in making management decisions, and have been found useful in those fields. Because decision trees require that the nurse has a knowledge base, it stands to reason that a decision tree must also be based on a foundation of knowledge. Clinical fields that have been well researched and require complex decision making are likely candidates for decision trees.

Regarding the matter of who should use a decision tree, a nurse who has a working, but not expert knowledge of the field would find a decision tree helpful. Use of a decision tree may also serve as a reminder of educational sessions that were attended.

As was found in other studies, this study reaffirmed the notion that decision trees can onlly guide the choices of the nurse; the nurse must be cognizant about how to use the decision tree and must have a basic understanding of the principles of the clinical field in question. Decision trees cannot be substituted for a sound knowledge base, however, decision trees can be valuable as expert systems for nurses who need to make complex clinical decisions under uncertain conditions.

References

Alvarez, O. M., Mertz, P. M., & Eaglestein, E. W. (1983).The effect of occlusive dressings on collagensynthesis and re-epithelization in superficial wounds. Journal of Surgical Research, 35, 142-148.

Alvarez, O., Rozint, J., & Wiseman, D. (1989). Moist environments for healing: Matching dressing to the wound. Wounds: A Compendium of Clinical Research and Practice, Premiere issue, 35-51.

Aspinall, M. J. (1976). Nursing diagnosis---the weak link. Nursing Outlook, 24, 433-437.

Aspinall, M. J. (1979). Use of a decision tree to improve accuracy of diagnoisis. Nursing Research, 28(3), 182-185.

Benner, P. (1984). From novice to expert: Excellence and power in clinical nursing practice. Menlo Park, Ca: Addision-Wesley.

Bothwell, J. W., & Rovee, D. T. (1971). The effect of dressings on the repair of cutaneous wounds in humans. In K. J. Harkiss (Ed.), Surgical dressings and wound healing (pp. 78-97). London: Crosby Lockwood.

Brennan, P. (1990). Decision support systems in nursing: An overview. In J. Ozbolt, D. Vanderwal, & K. Hannah (Eds.), Decision support systems in nursing (pp. 13-14). Toronto: Mosby.

Carnevali, D. L. (1989). The diagnostic reasoning process. In D. L. Carnevali, P. H. Mitchell, N. F. Woods, & C. A. Tanner (Eds.), Diagnostic reasoning in nursing (pp. 25-56). Philadelphia: Lippincott.

Casebeer, L. (1991). Fostering decision making in nursing. Journal of Nursing Staff Development, 7(6), 271-274.

Coleman, R. L. (1991). The use of decision analyses in quality assessment. Quality Review Bulletin, 12, 383-

391.

Cook, T. D., & Campbell, D. T. (1979). Quasi-experimentation: Design & analysis issues for field settings. Boston: Houghton Mifflin.

Elstein, A. S., Holmes, M. M., Ravitch, M. M., Rovner, D. R., Holzman, G. B., & Rothert, M. L. (1983). Medical decisions in perpective: Applied research in cognitive psychology. Perspectives in Biology and Medicine, 26, 486-499.

Grier, M. R. (1976). Decision making about patient care. Nursing Research, 25, 105-110.

Grier, M. R. (1984). Information processing in nursing practice. In H. H. Werley & J. J. Fitzpatrick (Eds.), Annual review of nursing research (Vol. II) (pp. 265-287). New York: Springer.

Greep, J. M., & Siezenis, L. M. L. C. (1989). Methods of decision analysis: Protocols, decision trees and algorithms in medicine. World Journal of Surgery, 13, 240-244.

Hammond, K. (1966). Clinical inference in nursing. Nursing Research, 15(1), 27-38.

Hinman, C. D., & Maibach, H. I. (1963). Effect of air exposure and occlusion on experimental skin wounds. Nature, 200, 377-378.

Hughes, K. K., & Young, W. B. (1990). The relationship between task complexity and decision making consistency. Research in Nursing and Health, 3(3), 189-197.

International Association for Enterostomal Therapy. (1987). Standards of care: Dermal wound, pressure sores. Irvine, CA: Author.

Katz, S., McGinley, K., & Leydon, L. L. (1986). Semi-permeable occlusive dressings: Effects on growth of pathogenic bacteria and re-epithelialization of superficial wouns. Archives of Dermatology, 122, 58-62.

King, W. (1991). Information technology and the management of preventive services. In P. Marr, R. Axford & S. Newbold (Eds.), Nursing informatics'91. Proceedings of the post conference on health care technology: Implications for change (pp. 120-124). New York: Springer-Verley.

Knight, C. (1992). Chronic wound management decision tree. Unpublished manuscript. Good Samaritan Auxiliary Hospital, Edmonton, AB.

Jones, J. A. (1988). Clinical reasoning in nursing. Journal of Advanced Nursing, 13, 185-192.

Lanza, M. L., & Bantly, A. (1991). Decision analysis: A method to improve quality of care for nursing practice. Journal of Nursing Care Quality, 6(1), 60-72.

Le Breck, D. B. (1986). Clinical judgement: A comparison of theoretical perspectives. In W. L. Holzemer (Ed.), Review of research in nursing education (Vol. II) (pp. 33-55). New York: NLN

Mantle, J. (1992). Research and serendipitous secondary findings. The Canadian Nurse, 88(1), 15-18.

Mertz, P. M., & Eaglestein, W. H. (1984). The effect of semi-occlusive dressings on the microbial population in superficial wounds. Archives of Surgery, 119, 287-289.

National Pressure Ulcer Advisory Panel. (1989). Pressure ulcer prevalence, cost and risk assessment: Consensus development conference statement. Decubitus, 2(2), 24-28.

O'Leary, M. A. (1992). Information and its influence on decision making. AAOHN Journal, 40(1), 42-43.

Prescott, P. A., Dennis, K., & Jacox, A. (1987). Clinical decision making of staff nurses. Image: Journal of Nursing Scholarship, 19(2), 56-62.

Raiffa, H. (1968). Decision analysis: Introductory lectures on choices under uncertainty. Don Mills, OT:

Addison-Wesley.

Rovee, O. T., Kurowsky, C. A., & Labun, J. (1972). Local wound environment and epidermal healing. Archives of Dermatology, 106, 330-334.

Ryan, S. (1985). An expert system for nursing practice: Clinical decision support. Computers in Nursing, 3(2), 77-84.

Shea, J. D. (1975). Pressure sores classification and management. Clinical Orthopedic and Related Research, 112, 89-100.

Skewchuk, R. M., & Francis, K. T. (1988). Principles of clinical decision making--an introduction to decision analysis: A special communication. Physical Therapy, 68(3), 357-359.

Tanner, C. A. (1983). Research on clinical judgement. In W. L. Holzemer (Ed.), Review of research in nursing education (pp. 1-32). Thorofare, NJ: Slack.

Van Den Berg, R., & Visinski, P. (1992). Decision trees in ICU. The Canadian Nurse, 88(1), 28-29.

Willey, T. L., & Swords, L. (1991). A comparison of a decision tree with other wound care decision support tools: Effects on accuracy and level of confidence. Unpublished master's thesis. Nell Hodgson Woodruff School of Nursing, Emory University, Georgia.

Winter, G. D. (1962). Formation of the scab and the rate of epithelialization of superficial wounds in the skin of the young domestic pig. Nature, 193, 293-294.

Winter, G. D., & Scales, J. T. (1963). Effects of air drying and dressings on the surface of a wound. Nature, 197, 91-92.

Wurzbach, M. E. (1991). Judgement under conditions of uncertainty. Nursing Forum, 26(3), 27-34.

APPENDIX A

Chronic Wounds: A Review of Current Nursing Care

Abstract

Chronic wounds occur in 4-23% of all patients and represent a major fiscal burden on health care. In this paper, current knowledge about chronic wounds and practice related to their care are discussed. Chronic wounds heal like acute wounds, but healing is often prolonged or delayed because of the compromised condition of the patient. In this paper, the three phases of healing (inflammatory, reconstructive, and remodelling) are presented and described in detail. Unlike acute wounds, chronic wounds are the result of an underlying problem such as vascular irregularities, autoimmune diseases, hematologic abnormalities, infections and diabetes. Whatever the underlying problem, chronic wounds are caused by tissue ischemia which deprives cells of necessary oxygen and nutrients and results in tissue necrosis. Practitioners use the extent of tissue necrosis to stage wounds from a level of I to IV with a Stage IV Wound representing the most tissue necrosis. Nursing care for patients with chronic wounds includes attention to both intrinsic (infection, nutrition, incontinence, edema, and immobility) and extrinsic (pressure, shear, friction,

and moisture) factors that influence the patient as a whole. In the past 30 years, local care of chronic wounds has changed from dry to moist environments for wound healing. Several categories of dressing products are available to facilitate moist healing, manage exudate, and debride necrotic tissue. These categories include traditional gauze, transparent adhesive, hydrocolloid wafer, polyurethane foam, hydrogel, and absorptive dressings. Other treatments for local care include surgical options, hyperbaric oxygen, growth factors, and electrical stimulation. Nurses require an understanding of wound healing and the etiology of chronic wounds in order to provide systemic and local nursing care that promotes and maximizes chronic wound healing.

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Chronic Wounds: A Review of Current Nursing Care

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Chronic wounds are problematic and frustrating for patients who suffer with them and for the nurses who provide care for those patients. As our population ages and the incidence of chronic disease increases, the incidence of chronic wounds also increases (Bergman, 1986; Kynes, 1986; National Pressure Ulcer Advisory Panel, 1989). The incidence of chronic wounds in North America has been reported as 4-14% for hospitalized patients, 8.7-19% for home care patients, and 12-23% for patients in long term care institutions (Kelly & Mobily, 1991). Brandeis, Norris, Nash, & Lipsitz (1990) found that 17.4% of patients who were admitted from home to long term care institutions had chronic wounds. Although the pain, suffering, and humiliation of living with a chronic wound cannot be measured, estimates of fiscal costs for one chronic wound ranges from $4,000.00 to $40,000.00 (U.S.) (Brandeis et al, 1990). In this paper, current knowledge and practices in the sphere of chronic wound care will be presented. As well, nursing assessment will be discussed.

Physiology of Healing

Human beings heal by two different processes: regeneration and scar formation. Healing by regeneration occurs when lost tissue is replaced by the same type of tissue. Partial thickness wounds (only skin involved) heal by regeneration. Scar formation involves the production of connective tissue to fill in a defect left by the loss of tissue. Full thickness wounds which involve skin, subcutaneous tissues and even muscle or bone must heal by scar formation and it is that process which will be described here (David, 1985; Doughty, 1990; Doughty, 1992).

The healing process is usually divided into stages or phases, but in reality it is a continuous process of overlapping, small events that ultimately lead to a closed wound (Cooper, 1990; Needham, 1952; Westaby, 1985). Westaby (1985) divides healing into four stages: traumatic inflammation, destructive, proliferation and maturation. Others divide healing into three phases: **inflammatory** (exudative/lag/defence and demolition), **reconstructive** (proliferative/ fibroblastic /connective/incremental) and **remodeling** (differential/resorptive/plateau) (Anderson, 1985; Cooper, 1990; Hardy, 1989; Kurzuk-Howard, Simpson & Palmieri, 1985; Schurmann, 1982). In this paper, three phases of healing will be presented.

Inflammatory Phase

Inflammation, a necessary part of healing, is a process that occurs in response to factors released by damaged tissues and blood vessels. It begins within minutes following injury and may last up to 5 days. Following injury, whole blood carrying fibrin and platelets enters the wound. The blood coagulates forming a fibrin clot preventing further fluid loss and protecting the wound from the outside environment. Simultaneously, fibrin clots are also formed in surrounding lymphatic vessels sealing off the wound from the rest of the body and preventing the spread of infection. The platelets release growth factors which attract cells and materials necessary for wound healing. After blood enters the wound, vasoconstriction lasting 5-10 minutes occurs preventing further blood loss. Vasodilation, stimulated by the release of histamine and serotonin from injured tissues and vessels, follows vasoconstriction and is responsible for the heat, redness, pain and swelling of inflammation. Vasodilation and chemical factors causing increased vessel permeability immediately allow for increased amounts of plasma proteins, antibodies, complement, water and electrolytes to be brought to the injured tissue. These materials are necessary for repairing the wound (David, 1985; Doughty, 1992; Hardy, 1989; Schurmann, 1982; Westaby, 1985).

Within 6 hours following injury white blood cells, attracted by chemical mediators released by injured tissues, arrive at the injury site. White blood cells carry out the process of phagocytosis--attacking and engulfing bacteria and removing necrotic tissue. The first white blood cells seen at the injury site are polymorphonuclear granulocytes. Their numbers quickly decline if no infection is present and they are replaced by monocytes. The monocytes modify becoming macrophages and remain at the injury site throughout the healing process apparently directing wound repair. Hydrolytic enzymes within the macrophages make them effective and efficient removers of cellular debris. From this digestion process, amino acids and sugars are made available for wound repair (Cooper, 1990; Doughty, 1992; Irwin, 1981, Schurmann, 1982; Westaby, 1985).

Macrophages are also instrumental in the process of neovascularization. They secrete an angiogenesis factor which stimulates the growth of endothelial cells on patent vessel ends. These growing vessels come in contact with other growing vessels forming capillary loops and hence neovascularization is achieved (Alvarerz, Rozint, & Wiseman, 1989; Anderson, 1985; Hardy, 1985; Schurmann, 1982)

Exudate from surroundi[illegible] lood vessels keeps the wound bed moist allowing for [illegible] promoting cell movement and mitosis. About 12 hours after injury, epithelial cells at the wound edges begin to divide and migrate across the wound. The new epithelial cells burrow down beneath the fibrin clot, dissolving the clot as they advance in order to maintain contact with the wound bed; As the epithelial cells advance across the wound they meet other epithelial cells and migration ceases. Thus, a new epithelial covering is provided for the wound (Alvarez et al., 1989; Hardy, 1985; Irwin, 1981; Schurmann, 1982).

The inflammatory phase ends when the wound bed has been cleared of bacteria and cellular debris. A chronic inflammatory response, due to local infection or necrotic debris, inhibits and retards wound healing by not allowing for the condition necessary (a clean wound bed) for the reconstructive phase to take place (Alvarez et al., 1989; Doughty, 1992).

Reconstructive Phase

The reconstructive phase begins 4-5 days following injury and lasts 2-4 weeks. This phase is dominated by the fibroblastic and myofibroblastic cells that migrate to the wound site in response to chemotactic factors. The fibroblasts synthesize collagen which will replace lost connective tissue. New collagen fibres are arranged in compact bundles and provide strength for the healing wound. Myofibroblasts containing contractile proteins attach at the wound margins and are instrumental in the process of wound contraction. Through wound contraction, the size of the area to be healed and the resulting scar is reduced. Epithelialization and neovascularization continue throughout the reconstructive phase providing a blood supply and a protective covering for the healing wound (Doughty, 1992; Hardy, 1985; Schurmann, 1982).

The reconstructive phase ends when lost connective tissue is replaced by granulation tissue and the wound has been resurfaced through epithelialization. Wound healing can be delayed in the reconstructive phase as a result of chronic inflammation in the inflammatory phase. Chronic inflammation results in an over production of collagen. Increased amounts of collagen interfere with circulation and the migration of healthy cells (David, 1985).

Remodeling Phase

The remodeling phase begins after the reconstructive phase has ended and may span a period of several weeks to several years. Although the wound appears healed after the reconstructive phase has ended, the scar is bulky and it lacks strength. During remodeling, the collagen fibres which make up scar tissue are rearranged in an attempt to approximate the structure and function of the scar tissue to the surrounding tissue. Scar tissue in muscle is modified to mimic muscle tissue and so on. Collagen fibres that were laid down compactly, but haphazardly during the reconstructive phase are now rewoven, through collagen lysis and collagen synthesis, into an organized, compact pattern that mimics the organization of the surrounding tissue. Contraction of the wound continues as the size and shape of the scar contracts over time with centripetal movement of the surrounding skin (David, 1985; Doughty, 1992; Hardy, 1985; Irwin, 1981; Schurmann, 1982; Westaby, 1985). Remodeling can be affected by a prolonged reconstructive phase. Increased collagen production in the reconstructive phase leads to a hypertrophic or painful and unsightly scar that will not reduce in size (David, 1985).

The above healing process is a well coordinated series events in healthy patients with acute wounds. Patients with chronic wounds are usually compromised by ill-health at the outset and may stall in one or more phases (David, 1985; Doughty, 1992). As well, it must be noted that the healing process in humans is not perfect; injured tissue never completely returns to normal. Injured tissue never regains more than 70-80% of its pre-injured strength and therefore is susceptible to further injury (Hardy, 1985; Jackson & Rovee, 1988).

Etiology of Chronic Wounds

Chronic wounds, which include pressure ulcers, venous ulcers and arterial ulcers, are the result of prolonged ischemia to tissues. Tissues that are deprived of oxygen and nutrients for an extended period of time become necrotic and a chronic wound is the visible result of that necrosis (Alvarez et al., 1989; Kosiak, 1991; Marvel, Rodheaver & Xakillis, 1992; Rolstad, 1991). Unlike acute wounds which are the result of surgery or trauma in an otherwise healthy individual, chronic wounds are the result of an underlying problem. Conditions that predispose a patient to the development of a chronic wound include "...pressure, vascular irregularities, burns, diabetes, autoimmune diseases, hematologic abnormalities, infections and reactions to chemical and radiotherapeutic agents" (Alvarez et al., 1989, p.36).

Pressure Ulcers

A pressure ulcer (pressure sore, decubitus ulcer, bed sore) occurs over bony prominences and is the result of localized tissue ischemia and necrosis (Kosiak, 1959; Kosiak, 1991). Commonly, pressure ulcers occur below the waist over the bony prominences of the sacrum, ischium, trochanter, malleolus and heel, but they can also occur over the elbows, occiput, knees and ears (Barton & Barton, 1981; Bryant, 1992; Ek &

Bowman, 1982; Longe, 1986, Torrance, 1983). Pressure, shear, friction and moisture have been identified as the principle causal factors in pressure ulcer formation (Alvarez, 1991; Barton & Barton, 1981; Longe, 1986,; Torrance, 1983).

Pressure

Prolonged pressure has been implicated as the primary factor in pressure ulcer formation. Pressure and shearing forces were identified by Groth (1942, cited in Kerr, Stinson, Bay, Thurston, & Leatt, 1980) as causal to deep pressure ulcers while friction was more likely to result in more superficial ulcers. Kosiak (1959) reported that normal capillary pressure ranges from 13 to 32 mm Hg. When pressure exceeding capillary pressure is applied to tissue, capillary flow is blocked and ischemia ensues. Kosiak found that as little as 60 mm Hg of pressure applied over a one hour period caused pathological changes in tissue. He found that relieving pressure at regular 5 minute intervals almost completely reversed the effects of pressure even at high level (240 mm Hg). Kosiak also found that evidence of tissue necrosis caused by unrelieved pressure does not manifest itself until at least 3 days following the incident.

Body weight or pressure in immobile and bedridden patients is not evenly distributed, but is concentrated at particular points such as bony prominences (Bryant, 1992; Longe, 1986; Torrance, 1983). Pressure at these points exceeds the amount of pressure needed to occlude capillary blood flow. These bony prominences usually have little subcutaneous tissue to cushion the effects of the extra pressure and this compounds the problem. Injury from prolonged pressure can involve many layers of tissue. Skin, superficial fascia, deep fascia, muscle and even bone can be injured (Bryant, 1992; Torrance, 1983).

Shear

Shearing forces stretch and bend blood vessels reducing the amount of pressure necessary for occlusion to occur (Alvarez, 1991; Kosiak, 1991). Shearing forces (Forward Slide Phenomenon) are created when a patient is sitting up in bed at an elevation of more than 30 degrees. The sacrum is exposed to increased pressure as body weight is redistributed and concentrated over the sacrum. At the same time, body weight slides toward the foot of the bed. Friction (resistance) holds the sacral skin in contact with the bed causing tension between the superficial and deeper tissues. In other words, the deeper tissues slide, but the superficial tissues do not. This is the force or tension that causes blood vessels to stretch and bend (Alvarez, 1989; Torrance, 1983).

Friction

Friction, the rubbing and chafing of skin against a resistive surface such as bed linens, can cause damage to superficial tissues and often occurs as a result of improper positioning and the use of restraints. Superficial abrasions and skin tears are the visible evidence of injury due to friction (Alvarez, 1991; Torrance, 1983). Friction (resistance) increases susceptibility to ulceration from pressure. Dinsdale (1973) demonstrated that 240 mm Hg of pressure was necessary to cause a pressure ulcer, but in combination with friction, ulcers were formed with levels as low as 45 mm Hg.

Moisture

Incontinence and perspiration lead to moisture that increases friction between the skin and the bed or chair surface. This friction can cause superficial skin breakdown as noted above. If moisture is excessive, friction decreases, but maceration of the skin develops. Macerated skin is weakened in two ways: (a) the barrier (stratum corneum) becomes more permeable to noxious substances and (b) the bonds between epidermal cells lose strength. Thus, the skin is susceptible to infection and superficial injury. Candida skin infections and skin tears often occur with macerated skin (Alveraz, 1991; Longe, 1986; Torrance, 1983).

Venous Ulcers

Venous ulcers, also the result of ischemia, usually occur on the lower legs and ankles. Venous hypertension related to faulty valves, gravity, and underlying pathologies leads to edema resulting in increased pressure on arterial blood vessels. Blood flow to tissues is impeded, ischemia occurs and tissue death follows. The ensuing ulcer is not unlike the pressure ulcer in appearance, but the surrounding skin is often discoloured, edematous and cyanotic. The ulcer, which is usually painless unless infected, has an irregular border with a shallow crater. Exudate is almost always present and varies from small to large amounts (Fowler, 1982; International Association for Enterostomal Therapy, 1988; Lanyan, VanNieuwenhuyzen & Wearing, 1987; Ryan, 1985; Venn, 1987; Zink, Rousseau, & Holloway, 1992).

Arterial Ulcers

Arterial ulcers usually occur around the foot and toe area and are the result of ischemia caused by arterial insufficiency. Signs of arterial insufficiency include absent distal limb pulses, pallor of the extremity on elevation and cyanosis of the extremity in the dependent position. Arteriosclerosis or "hardening and narrowing of the arteries" is caused by patchy, lipoidal degeneration of the intima, calcification of arterial muscles and endothelial thickening of the arterial walls. As arteriosclerotic disease progresses, plaques form on the arterial walls and eventually occlude blood flow. The obstruction of oxygen and nutrients to the tissues leads to tissue necrosis. Arterial ulcers often appear on the feet following trauma or pressure from a shoe or cast. Diabetics who suffer from small vessel arterial disease and diabetic neuropathy are especially prone to arterial ulcers. The arterial ulcer like other ulcers appears as an area of tissue loss, but unlike venous ulcers the surrounding skin is not discoloured (Fowler, 1982; International Association for Enterostomal Therapy, 1988; Venn, 1987; Zink, Rousseau, & Holloway, 1992).

Assessment of Risk

Correctly identifying those patients who are at risk for developing chronic wounds allows the nurse to employ an appropriate prevention program on an individual basis. Deciding when and where expending time and energy on prevention will be the most useful is the goal of most risk assessment tools (Braden & Bryant, 1990). Assessment tools have been developed to identify those who are at risk for pressure ulcer formation. The risk of developing venous or arterial ulcers is directly related to the presence of the underlying disease pathology. Assessment tools addressing the risk of leg ulcer formation are not available in the literature.

Nurses working in almost all fields of nursing require and appreciate assessment tools that identify patients at risk for pressure ulcer formation. Dimant and Francis (1989) and Rolstad (1991) claim that an assessment tool should be included in all chronic wound management programs. Furthermore, systemic rather than local risk assessment is mandatory for early prevention.

The Norton Scale, introduced in Britain by Doreen Norton in 1962 (cited Norton, 1975), was the first assessment tool developed. The scale addressed five risk factors: physical condition, mental condition, activity, mobility and incontinence. A score of 14 or less out of a possible 20 indicated risk for pressure ulcer formation. The Norton Scale has served as the prototype for all subsequent scales including Gosnell's (1973) Scale and Braden's (Bergstrom, Braden, Lagussa, & Holman, 1987) Scale (Braden & Bryant, 1990).

Gosnell (1973) developed a risk assessment tool not unlike Norton's Scale and tested it in nursing practice. The scale included mental status, continence, mobility, activity and nutrition. Scores ranging from 5-20 are possible, 5 being at the least risk and 20 the most. Gosnell found that a score of 11 or less was indicative of high risk for pressure ulcer formation. An assessment of skin status including skin appearance, tone, and sensation is included in the tool, but it is not included in the numerical score.

Bergstrom et al. (1987) found that nurses were engaged in unnecessary preventative care because existing scales over predicted the risk of pressure ulcer formation. Braden (Bergstrom et al., 1987) developed another scale for predicting pressure ulcer formation. Braden's Scale has 6 subscales: sensory perception, skin moisture, activity, mobility, friction and shear and nutritional status. The Braden Scale takes less than one minute to complete (Braden & Bryant, 1990) and has been found to be 100% sensitive and 88% specific (Bergstrom et al., 1987). Research has demonstrated that the Braden Scale also over predicts, but less than the Norton (1962, cited in Norton, 1975) or Gosnell (1973) Scales (Braden & Bryant, 1990).

In the past 10 years, research has led to a better understanding of risk factors and the incidence of pressure ulcer formation. Ek and Bowman (1982) in a study of the characteristics of patients with pressure ulcers found that those who were at most risk for developing pressure ulcers were (a) women, (b) over the age of 65, (c) incontinent and (d) immobile. Pajk, Craven, Cameron-Barry, Shipps and Bennen (1986) confirmed the findings of Ek and Bowman (1982) in that there is a relationship between age, gender and skin breakdown. Pajk et al. (1986) also found that a low rating on 5 risk factors derived from the Norton and Gosnell Scales (mental status, continence, mobility, activity and nutrition) were indicative of increased risk for pressure ulcer formation. Marvel, Rodheaver and Xakillis (1992) report that successful pressure ulcer prediction rests on four factors: mobility/activity deficit, moisture/incontinence, nutritional deficit and altered level of consciousness.

Assessment of Chronic Wounds

Assessment and staging of chronic wounds provide important information for selection of wound care and wound care dressings and products (Rolstad, 1991). Guttman (1955) developed a three stage assessment system which was based on increasing degrees of tissue damage. The first stage was characterized by redness which disappeared on removal of pressure. The second stage involved damage to superficial tissues. The third stage of damage was characterized by damage to deep tissue layers, extending to muscle and even bone.

Shea (1975) categorized pressure ulcers into four stages. The International Association for Enterostomal Therapy (1987) and The National Pressure Ulcer Advisory Panel (198) have adapted these four stages to include all chronic wounds.

Stage I Wound

A Stage I wound appears as an acute inflammation without a break in the skin, when pressure is released, redness does not disappear. The wound blanches and is warm and firm to touch.

Stage II Wound

A Stage II Wound is characterized by partial thickness epidermal or dermal loss. The ulcer is shallow with a painful, pink, moist base. The edges of the ulcer are distinct, and the surrounding skin is reddened.

Stage III Wound

Stage III wounds involve full thickness skin loss with damage or necrosis of subcutaneous tissue. The ulcer is deep, but does not extend to underlying fascia. There may be exudate with or without infection, necrotic tissue and sinus tract formation. The wound is usually not painful.

Stage IV Wound

A Stage IV wound is characterized by full thickness skin loss with extensive damage to underlying fascia and sometimes to tendons, bones and joints as well. The ulcer presents as a deep crater and may include necrotic tissue or eschar, tracking and tunnelling and exudate with or without infection. The base is usually not painful.

Besides staging, Fowler and Goupil (1984) recommend that wounds be assessed for location, size, drainage, odour and undermining. Green and Katz (1991) report that wound inspection should include the following points: size, skin colour, pain at site, pulse, temperature, location, odour, drainage and condition of surrounding skin. Rolstad (1991) states that wounds should be assessed for location, presence of epithelialization and/or granulation tissue, pain, edema, exudate (character, volume, and odour), undermining or sinus tracts, necrotic tissue (type and amount) and condition of surrounding skin. Alvarez (1991) has also outlined specific areas of inspection which include: necrotic tissue, erythema. edema, induration, wound margins, wound depth, exudate and granulation tissue.

Recently, establishing the colour of a wound (pink/red, yellow and black) has been used as a quick, easy method of assessing chronic wounds. Moriarty (1988) has determined that staging a wound does not reflect progress, but the colour of the wound indicates more clearly the phase of healing. A pink/red wound has been revascularized and is ready for healing. A yellow wound indicates infection--frequent cleansing and debriding are required. A black wound indicates necrotic tissue which must be removed before proper healing can take place. Ryan (1985) and Lanyon et al. (1987) demonstrated that the above colour system could also be used to manage leg ulcers. Using colours as a guide, venous and arterial leg ulcers were appropriately assessed, cleansed and dressed.

Because chronic wounds heal slowly, assessing them for healing is problematic. The assessments described above are subjective rather than objective. Measuring wound size quantitatively has been proposed as a means of assessing healing objectively. Several methods of measurement have been proposed; some are simple and some are complex.

Manual measurements of width, length and depth are used frequently to measure ulcer area and volume. A decrease in area or volume indicates wound healing (Trelease, 1988; VanRijswijk & Cuzzel, 1991). Thomas and Wysocki (1990) studied this method of wound measurement and determined it was subject to inconsistencies. If the wound is on a skin surface that is curved, measurements differ depending on the position of the patient. Another method for measuring wounds using length, depth and width has been developed. Kundin (1989) developed a gauge for measuring the area of a surface lesion and the volume of a crater wound. The Kundin device is comprised of X, Y, and Z axes. The X axis corresponds to North and South, the Y axis to East and West, and the Z axis measures depth. Formulas for ascertaining area and volume are included with the tool. Thomas and Wysocki (1991) found that the Kundin device underestimated wound area and volume.

Photographs are frequently used as a method of tracking wound healing, but photographs are two dimensional and hence cannot reveal the tri-dimensionality of wound surfaces (Thomas and Wysocki, 1991). Bulstrode, Goode, and Scott (1986) report that stereophotogrammetry is an accurate method that allows for measurement in three dimensions. Unfortunately, the method is costly and time consuming which does not lend stereophotogrammetry useful in clinical practice.

The third method of wound measurement is acetate tracing. Acetate is placed over the wound and the margins are traced with an indelible marking pen (Anthony, 1984; Bohannon & Pfaller, 1983; Thomas & Wysocki, 1990). Measurements can be taken from the tracing using (a) length x width, (b) by plotting the area using graph paper and then counting the squares or (c) with an image analysis system. Thomas and Wysocki (1990) found that acetate tracing with the use of an image analysis system is the most accurate method for measuring wound area.

Nursing Care

Nursing care for patients with chronic wounds must be both systemic and local. A holistic care plan that addresses the patient's total environment (internal and external) will more likely lead to successful wound healing than a plan that focuses only on the wound.

Systemic Care

Multiple underlying systemic factors, both intrinsic and extrinsic need to be addressed for chronic wounds to heal. A thorough assessment including an assessment of risk factors will assist the nurse in identifying intrinsic and extrinsic factors that may retard or promote healing.

Intrinsic factors

Intrinsic factors are related to the patient's general physical and mental condition. That is, their degree of mobility, nutritional status, age, presence of pathologies (diabetes, arterial insufficiency, autoimmune diseases, neurological disorders, etc.), mental status, cardiovascular system, presence of local or systemic infection and degree of continence. Intrinsic wound healing retardant factors such as disorders), infection, inadequate nutrition, incontinence, edema and immobility need to be corrected (when possible) for healing to take place. All of the above, except uncontrolled diseases, will be discussed in this paper as part of systemic nursing care. Other intrinsic factors such as age, gender, some neurological disorders (eg. quadraplegic) and sometimes mental status are not treatable, but need to be considered as part of an individualized program (Bryant, Shannon, Pieper, Braden & Morris, 1992; David, 1985; Fellin, 1984; Kurzuk-Howard et al., 1985).

Infection.

Besides local infection of the chronic wound, which will be discussed under local care, systemic infections are of serious concern. A systemic infection further compromises the ability of a patient to heal a chronic wound. If fever is present, basal metabolic rate is increased which increases the demand for oxygen further depriving ischemic tissues of oxygen. As well, infection increases the need for nutrients and when this is compounded with existing inadequate nutrition, the patient's body defenses and reserves are additionally weakened (Bryant et al., 1992; Torrance, 1983).

A bacterial infection, often of the lungs, urinary tract or abdomen, is indicated if the patient presents with a temperature of 37.5 degrees Centigrade or higher and a high white blood count (greater than 14,000). The absence of fewer and an immune response does not necessarily exclude the possibility of infection--

especially in the elderly. An elderly patient who is deteriorating from baseline status should be investigated for likely infections. Appropriate antibiotics and supportive care should be administered immediately (Alvarez, Rozint, & Wiseman, 1989; Kosiak, 1991).

Nutrition.

Inadequate nutrition is often a contributory factor in retarded wound healing. Inadequate intake is often cited as the cause, but immobility and illness compound the problem (Alvarez et al., 1989; Kosiak, 1991; Rubin, 1988). In the elderly, inadequate intake is often associated with the loss of social interaction. Aloneness, loneliness and grieving, which are often a part of aging, diminish the pleasure of meal time resulting in loss of appetite and apathy toward food. The effects of inadequate nutrition are anemia, hypoproteinemia, hypocalcemia and a suppressed immune system (Fellin, 1984; Kosiak, 1991; Morley, 1990; Rolstad, 1991). Supplements of protein (80-100 gm/day) and Vitamin C (1000 mg/day) are recommended to ensure adequate nutritional support for the healing wound. The efficacy of zinc supplements in promoting wound healing has been studied, but has not been found to be beneficial (Alvarez et al., 1989). Colburn (1990) states that adequate hydration is also of importance and a fluid intake of 2000 mL/day is recommended if it is not contraindicated by other pathologies. If the patient is unable to orally maintain adequate nutrition and hydration, nasogastric feeding, gastrostomy tube feeding or parental nutrition is indicated (Longe, 1986; Kosiak, 1991; Rolstad, 1991; Westaby, 1985).

Obesity retards wound healing because obese patients suffer from poor circulation leading to a hypoxic wound. This is additionally compounded by poor mobility and problems with excessive moisture. Adequate nutritional support with a goal of weight reduction is the desired outcome of this scenario (David, 1985; Rolstad, 1991).

Incontinence.

The moisture and contamination of urinary and fecal incontinence seriously complicate wound healing. Keeping the incontinent patient clean and dry is the goal of nursing care (Norton, 1975; Barton & Barton, 1981). Often, urinary incontinence can be cured or improved (Alvarez et al., 1989). An assessment of bladder function and pattern can assist in the development of an individualized nursing care plan for toileting (Alvarez et al., 1989; Barton & Barton, 1981; Torrance. 1983). The use of condom, indwelling and suprapubic catheters often lead to infection which further compromises wound healing (Torrance, 1983). Absorbent diapers and pads provide comfort and convenience, but they can promote skin maceration and ultimately ulcer formation. They can also be humiliating to a patient whose self-esteem is already compromised by incontinence. Absorbent pads and diapers should be used in conjunction with frequent washings with mild soap and water and moisture barrier cremes such as petrolatum and zinc oxide (Alvarez, 1989; Barton & Barton, 1981; Norton, 1975).

Fecal incontinence is often the result of fecal impaction. Once fecal impaction has been treated (with enemas and manual removal), a bowel training program with emphasis on bulk forming materials and regular toileting can be instituted (Barton & Barton, 1981).

Edema.

Edema increases the distance that oxygen and nutrients have to travel from the capillaries to the cell and hence inhibits healing by denying the wound oxygen and nutrients (Kosiak, 1991). Edema is most often due to underlying pathologies such as renal and cardiovascular disorders which need to be treated before edema will be decreased. Dependent edema related to other causes most often occurs in the sacral area. For example, the individual with a protein deficiency will demonstrate edema as a result of decreased osmotic pressure in the blood vessels allowing fluid to leak into the interstitial spaces (Colburn, 1990; Fellin, 1984; Venn, 1987; Kosiak, 1991). Edema that occurs in conjunction with venular dysfunction can be treated with exercise, support bandages, TEDS and elevating the legs (Lanyon et al., 1987; Ryan, 1985). Careful handling and cleansing of edematous skin is also an important aspect of nursing care (Venn, 1987).

Immobility.

Immobility and prolonged bedrest have deleterious effects on body metabolism (Kosiak, 1991). Rubin (1988) reports that patients who are confined to bed have decreased plasma and calcium levels, secrete less gastric juice, have decreased blood flow through their calves and demonstrate glucose intolerance. This patient is also susceptible to infections because of a decrease in leukocyte function and number (Kosiak, 1991).

Externally, skin changes take place. Because the skin is not used to weight bearing and the patient cannot freely make postural changes, it is exposed to prolonged pressure over bony prominences. As well, perspiration cannot freely evaporate because air flow is restricted around an immobile patient. These conditions add up to pressure and moisture over skin and tissues unused to such treatment, and the result is pressure ulcer formation (Longe, 1986; Rubin, 1988).

To combat the effects of bedrest and immobility, nursing care needs to include daily ambulation (if possible), frequent position changes (lateral positions at 30 degrees oblique angles), range of motion exercises and the avoidance of sedation and restraining devices. In addition to relieving pressure, physical activity increases blood flow to the healing wound (Alvarez, 1991; Green & Katz, 1991; Kosiak, 1991). Something should also be said about the psychosocial benefits of physical activity which can influence feelings of well-being and hence promote wound healing.

Extrinsic Factors

Nursing care has the greatest impact on the external environment or the extrinsic factors to which a patient is exposed. It is a misconception that nursing care alone can prevent chronic wound formation (Allman, 1989; Braden & Bryant, 1990; Kerr, Stinson, & Shannon, 1981; Sebern, 1986), but it can modulate contributing factors. Extrinsic factors include pressure, shear, friction and moisture (Makelbust, 1987: Norton, 1975).

Pressure and shear.

Because pressure is the single most contributory cause of pressure and venous ulcers, relieving pressure is of paramount importance for nursing care. To relieve pressure for patients at high risk or suffering from pressure ulcers, frequent repositioning has been the treatment of choice. Nursing clinicians agree that q1-2h position changes that alternate between the back and right and left 30 degree oblique angle, lateral positions are the most useful for relieving prolonged pressure. Positioning at sharper angles (eg. 90 degree side-lying) puts pressure on other bony prominences such as the trochanter (Braden & Bryant, 1990; Colburn, 1990). The 30 degree rule also applies to sitting up in bed. Sitting up in bed relieves pressure at some points, but at higher elevations shearing forces occlude blood flow to tissues already exposed to pressure. A slight elevation as a position change will achieve the same ends and place the patient at less risk (Braden & Bryant, 1990). Keeping the bed linens free of wrinkles and crumbs is also important in counteracting points of pressure (Norton, 1975).

Increasing blood flow to skin and tissues exposed to pressure through massaging is no longer considered appropriate nursing care. Rubbing can cause further damage to already ischemic or damages tissues (Braden & Bryant, 1990; Kerr et al., 1980; Morley, 1990).

There are special devices and mattresses available for reducing pressure on the skin and tissues of the immobile patient. Comfort devices such as air rings can cause damage and should not be used. These devices occlude circulation up new points of pressure and further decrease circulation to compromised tissues (Bryant et al., 1992). Mattress overlays of air or water are useful for patients who are at some risk for pressure ulcer formation. Mattress replacements (Made of polyurethane foam) reduce pressure and are used for those at moderate risk. Specialty beds (usually rented) are filled with air or fluid that alternate areas of pressure. They are used for patient with large ulcers or who have had surgical closure of an ulcer (Colburn, 1990).

As mentioned earlier, relieving pressure in patient with venous ulcers is dependent on reducing edema. In addition, points of external pressure such as a catheter tubing pressing against a leg, too tight bed sheets or edematous legs resting on bare wheelchair supports must be avoided. The use of heel pads, beneficial cushioning and protective devices and bed cradles can be beneficial (Venn, 1987).

Friction and moisture.

Norton (1975) states that friction often occurs from fast and unskilful lifting up in bed and from the careless or rough giving of a bedpan. Gentleness is certainly in order when nursing patients at risk for chronic wound formation. The use of lifting devices and sheets can help to make lifting easier and less damaging to the skin of the patient. Because the perspiration of the immobile patient cannot evaporate, care must be taken to ensure that bed linens are kept dry to guard against maceration. As mentioned earlier, the incontinent patient must also be kept clean and dry.

Local Care

Local care of a chronic wound includes assessment (discussed earlier), cleansing, debriding and dressing. A thorough assessment at each dressing change will guide the nurses' choice of wound care and wound care products.

Cleansing

Ideally, cleansing removes bacteria, necrotic tissue, foreign bodies and purulent exudate from the healing wound. This protects the wound from contaminates and allows the nurse to visualize the wound base and make nursing care decisions based upon that assessment (David, 1985; Doughty, 1992; Longe, 1986).

In the past, topical antiseptics have been used to clean wounds. These substances (eg. 1% povidone-iodine, 0.25% acetic acid, 3% hydrogen-peroxide, 0.5%

sodium hypochlorite) have been found to be cytotoxic and actually delay wound healing (Thomason, 1989). Soaps and astringents are also harmful to healing tissues. They remove skin oils causing cracks, chapping and disruption of normal skin pH hampering the protective normal acid mantle (Longe, 1986). Rodheaver(1988) cautions that wounds should be treated gently with consideration of the philosophy: "Don't put in a wound what you wouldn't put in your own eye" (p. 61). Rodheaver reports that in his studies of wound care, wounds cleansed with normal saline resulted in lower infection rates than those cleansed with 1% povidone-iodine. Practitioners agree that the best cleansing agents are isotonic solutions such as Normal Saline and Ringers Lactate. These solutions are not cytotoxic, and in addition they moisturize the wound promoting epithelialization and capillary migration (Allman, 1989; Alvarez, 1989; Nursing, 1991; Rodheaver, 1988; Thomason, 1989).

The clean or non-infected wound requires gentle flushing with Normal Saline or Ringers Lactate. The use of a piston syringe without a needle is an effective method of flushing that does not damage new tissue. The use of hydrotherapy or high-pressure irrigation is contraindicated for clean wounds because it can damage new tissue and remove substances in the wound bed that are necessary for healing (Doughty, 1992).

Infected or necrotic wounds require thorough irrigation to remove necrotic debris and bacteria. Doughty (1991) recommends using a 35 mL syringe with a 19 gauge needle to flush the wound with Normal Saline or Ringers Lactate. This method effectively removes bacteria, but minimizes damage to new tissue. Wounds that are heavily contaminated with bacteria and have large amounts of exudate may be flushed with an antiseptic. Doughty (1992) notes that some practitioners advocate that antiseptics should never be used because they hinder the body's innate defenses against infection. To loosen and remove large amounts of necrotic tissue, hydrotherapy may be used with caution. Extensive use of hydrotherapy may cause maceration of surrounding tissue (David, 1985; Doughty, 1992).

Alvarez (1991) reports that his research has demonstrated that topical antibiotics used to combat local wound infection speed healing simply by providing a moist environment. He recommends that topical antibiotics should only be used on visibly infected wounds and their use be restricted to 10-15 days because extended use can lead to resistant bacteria.

Debridement

Necrotic tissue in a wound can cause chronic inflammation and can serve as an ideal environment for bacterial proliferation. When necrotic tissue cannot be removed through cleansing, it must be removed through mechanical, chemical or autolytic debridement (Alvarez, 1991; Doughty, 1992; Longe, 1986). Doughty (1992) cautions not to remove the dry eschar in wounds exposed to chronic ischemia. The dry eschar acts as a barrier to external contaminants and bacteria and if removed, the already compromised patient would be open to overwhelming infection. She recommends applying topical antibiotic solutions to the dry eschar and covering the wound with dry gauze dressing.

Mechanical debridement.

There are several methods of mechanical debridement; the method chosen depends on the amount of necrotic tissue and the condition of the patient. For chronic wounds with large amounts of necrotic tissue, surgical debridement under anaesthetic may be required. For minor amounts of necrotic tissue, debridement may be accomplished by wet-to-dry gauze, scalpel or CO_2 laser (Alvarez, 1991; Kosiak, 1991).

To remove minor amounts of necrotic tissue, wet-to-dry gauze is often the nursing treatment of choice. The chronic wound is packed with sterile gauze soaked in normal saline and then covered with dry gauze. When the dressing is removed (usually every 4 hours), necrotic tissue is also removed (Nursing, 1991; VanRijswijk & Cuzzel, 1991). Because this method can be painful and may dry out or damage healing tissue, Fellin (1984) and Braden and Bryant (1990) recommend that wet-to-moist gauze be used. The gauze is removed before it is dry thereby preventing drying and damaging of healing tissue.

Debridement by scalpel can be performed by a physician or a nurse when minor amounts of necrotic tissue are to be removed. Debridement by scalpel can be very painful for the patient restricting the amount of necrotic tissue that can be removed in one treatment. Following debridement, the wound is flushed with Normal Saline and minor bleeding is managed with pressure and/or silver nitrate sticks (Doughty, 1992).

CO_2 lasers are used by physicians to quickly vaporize necrotic tissue. CO_2 lasers do not require anaesthetic and they are painless for the patient. In addition, the laser provides instantaneous hemostasis and sterilization and stimulates platelets to release growth factors (Braden & Bryant, 1990; Doughty, 1992).

Chemical debridement.

Chemical debriding agents in the form of enzymatic ointments, solutions powders and sprays are commercially available. In practice, the efficacy of these agents is questionable (Kosiak, 1991; Longe, 1986). Longe (1986) recommends that chemical debriding agents be used in conjunction with other debridement modalities. In a wound care update, Nursing (1991) does not recommend the use of enzymatic preparations for effective wound debridement.

Autolytic debridement.

Autolytic debridement is becoming more popular with practitioners. Through autolysis, necrotic tissue is broken down by the body's own white blood cells. This is accomplished through the use of occlusive, moisture-retentive dressings such as transparent films or hydrocolloids. The moist wound surface promotes rehydration of the necrotic or avascular tissue allowing white blood cells to migrate to the area. The white blood cells and their digestive enzymes debride the necrotic tissue through phagocytosis (Doughty, 1992; Fellin, 1984; VanRijswijk & Cuzzel, 1991). Fellin (1984) reports that the use of hydrocolloid dressings to autolytically debride necrotic tissue saves in nursing time and dressing costs, and promotes healing.

Dressings

Dressing care for all wounds was revolutionized in the early 1960's with Winter's (1962) research on moist environments for healing. Prior to that time, wound care and dressings had not changed since the 1600's. With the idea that all wounds need to breathe, wounds were covered with dry, absorbent, non-occlusive dressings (Alvarez et al., 1989; Hutchinson & McGuckin, 1990). Winter's (1962) work was corroborated and advanced by later studies (Alterescu, 1983; Alvarez, Mertz, & Eaglestein, 1983; Bothwell & Rovee, 1971; Hinman & Maibach, 1963; Kurzuk-Howard et al., 1985;

Mertz & Eaglestein, 1984; Rovee, Kurowsky, & Labon, 1972; Saydak, 1990: Winter & Scales, 1963). It is now accepted that wound healing is promoted when the wound surface is kept moist, and this is achieved through occlusive dressing products which seal off the wound from the outside environment.

Not only do occlusive dressings promote healing, but they are also cost efficient. Fellin (1984), Miller (1988, cited in Hutchinson & McGuckin, 1990), and Seburn (1986) demonstrated that when occlusive dressings were compared to traditional wet-to-dry gauze dressings, there were substantial cost savings. Because occlusive dressings can be left in place for several days (or longer), less dressing supplies and nursing time are required for wound care, and this results in cost savings (Fellin, 1984; Longe, 1986).

In addition to speedier healing and cost efficiency, wounds dressed with occlusive dressings demonstrate lower infection rates than wounds dressed with non-occlusive dressings. In their compilation of 75 studies on wound infection rates, Hutchinson & McGuckin (1990) found an infection rate of 2.6% and 7.1% respectively for wounds dressed with occlusive and non-occlusive dressings.

Scales and Winter (1963) stated that the ideal dressing should absorb fluid and exudate, protect the wound from injury, reduce the risk of infection and not adhere to the wound surface. Carr and Lalagos (1990) agree with this ideal, but their requirements are more exacting. They stated that the ideal dressing should "... maintain a clean wound base, avoid dehydration and maintain body temperature, protect surrounding skin, remove necrotic tissue, minimize bacterial growth, promote formation of granulation tissue, facilitate re-epithelialization, manage exudate, minimize disruption of the wound bed, control odour, prevent maceration, relieve pain, facilitate application of medication, stay in place apply/remove easily and be cost efficient" (p. 39). There is no dressing product available today that meets all of the above requirements. Dressing products that are available fall into several categories: traditional gauze, transparent adhesive, hydrocolloid, polyurethane foam, hydrogel and absorption dressings (Alvarez et al., 1989; Doughty, 1992).

Traditional gauze dressings.

Gauze dressings have traditionally been dry allowing wound surfaces and deep tissues to dry out, but they can be modified by soaking the gauze in Normal Saline to create a moist environment. This requires frequent dressing changes or frequent re-moistening. A gauze dressing is used correctly when the wound is packed lightly (to prevent occlusion of blood flow) with moistened fine gauze mesh or ribbons. The wet gauze is kept away from surrounding skin to prevent maceration. If the dressing is allowed to dry out, even of purposes of debridement, tissues will be injured when the gauze is removed. If kept moist, gauze dressings can facilitate autolysis, absorb exudate and fill sinus tracts. Gauze dressings can be used in combination with transparent adhesive dressings providing both packing and occlusiveness (Doughty, 1992; Myers, 1982: Torrance, 1983).

Transparent adhesive dressings.

Transparent adhesive dressings (eg. Tegaderm [3M Health Care], Bioclusive [Johnson & Johnson], Op-site {Smith & Nephew]) were the first dressings introduced that facilitated occlusive wound healing. The dressings are made of a semi-permeable polyurethane film with an adhesive border that attaches to surrounding skin. They are permeable to gases (eg. O_2 and H_2O vapour), but are impermeable to liquids and bacteria (Conforti, 1989; Doughty, 1992; Rolstad, 1991).

Maintenance of wound hydration, facilitation of autolytic debridement, wound protection from injury, friction and contaminants, pain control and insulation are some of the advantages of transparent adhesive dressings. Because water vapour is allowed to pass through the film, the dressing also protects the surrounding skin from maceration. At the same time, oxygen diffuses into the wound bed and supplies some oxygen to proliferating granulation tissue. The dressing also allows for visual examination of the wound without dressing removal. Transparent adhesive dressings do not absorb exudate. When exudate is present, the dressing can either be removed and replaced or the exudate can be aspirated out of the wound bed with a needle and syringe (the needle site is patched with an adhesive dressing). Transparent adhesive dressings do not fill dead space and therefore are not recommended for Stage III or IV ulcers (Alvarez et al., 1989; David, 1985; Doughty, 1982; Hutchinson & McGuckin, 1990; Rolstad, 1991; Turner, 1985).

Hydrocolloid wafer dressings.

Hydrocolloid wafer dressings incorporate hydrophillic particles (eg. guar, karaya, carboxymethyl cellulose, gelatin, pectin) with an adhesive matrix. The hydrophillic particles absorb wound exudate by capillarity. The outer surface of the dressing is water repellant. Hydrocolloid wafers (eg. DuoDerm [ConvaTec], Comfeel [Coloplast] are occlusive and they have the added advantage of conforming to and sealing difficult to dress areas such as the sacrum, heel and trochanter. In addition to absorbing wound exudate, they facilitate autolytic debridement, provide pain control and protect the wound from bacterial invasion, injury, friction and temperature variations. When hydrocolloid wafers are removed there is often a characteristic odour and a yellowish gel which may be mistaken for infection. The odour and gel are actually from dressing decomposition--a result of colloid particle swelling in the adhesive mass. Hydrocolloid wafers are recommended for Stage II and III wounds, but they are not recommended for Stage I, Stage IV, dry, infected or heavily exuding wounds. Unless used with additional packing, hydrocolloid wafer dressings are not recommended for wounds with sinus tracts, depth or with dead space (Alvarez et al., 1989; Braden & Bryant, 1990; Conforti, 1989; Doughty, 1992; Rolstad, 1991).

Polyurethane foam dressings.

Polyurethane foam dressings (eg. Synthaderm [Derma-Lok], Lyofoam [Acme], Silastic [Dow] are absorbent like hydrocolloid wafers, but they are not adhesive. To seal the foam to the wound, a secondary dressing such as a transparent adhesive dressing must be applied as a covering. In addition to sealing the wound, the secondary dressing should provide a bacterial barrier and prevent the foam dressing from drying out. If allowed to dry out, the foam can injure tissue when it is removed. Polyurethane foam dressings absorb exudate, facilitate autolysis, promote hydration of the wound bed and protect the wound from injury, friction and temperature variations. If exudate is too heavy, the foam dressing may promote maceration of the wound and surrounding skin. Polyurethane foam dressings are indicated for moderately exuding Stage II, III and IV wounds. Also, they are appropriate dressings to use for infected wounds. They are not recommended for Stage I or dry wounds (Alvaraz et al., 1989; Doughty, 1992; Rolstad, 1991).

Hydrogels.

Hydrogels contain large amounts of water (80-99%) trapped in a lattice made of a cross-linked polymer. Hydrogels (eg. Geliperm [Geistlich], Intrasite Gel [Smith & Nephew], Vigilon [Bard]) conform to wound surfaces, facilitate a moist wound surface and promote capillary migration, epithelialization and autolytic debridement. As well, hydrogels fill dead space and control pain through their cooling properties. Because hydrogel dressings are non-adherent, they can be removed without tissue trauma. Like polyurethane foam dressings, secondary dressings are required to secure them in place, prevent drying out and to provide occlusiveness and a bacterial barrier. Hydrogels are available in two forms: sheet and granulate. The sheets are recommended for Stage II or III wounds and the granulates for Stage III or IV wounds. Hydrogels do not have a large absorptive capacity and therefore are not indicated for wounds with moderate to large amounts of exudate. They are not recommended for wounds that are infected or have deep undermining or tunnelling (Alvarez et al., 1989; Braden & Bryant, 1990; Doughty, 1992, Eagelstein, Mertz, & Falanga, 1987; Rolstad, 1991).

Absorptive dressings.

Absorptive dressing products are available as beads (eg. Debrisan [Pharmacia}, DuoDerm granules [ConvaTec], pastes (eg. Comfeel Paste [Coloplast], Envisan Paste [Marion Labs]) and as calcium alginate dressings (eg. Sorbsan [Dow], Kaltostat [Calgon]). Absorptive dressings are indicated for Stage II, III or IV wounds with moderate to large amounts of exudate and/or necrosis. They are not recommended for Stage I, minimally exuding or infected wounds. The beads and pastes are not recommended for deep, tunnelling wounds (Alvarez et al., 1989; Doughty, 1992; Fowler & Papen, 1991; Rolstad, 1991).

Absorptive beads and pastes are composed of colloidal, hydrophillic particles such as polysaccharide starches and natural copolymers. The particles are super absorbent and can absorb up to 100 times their weight in fluid. Absorptive beads and pastes can be used to fill dead space and absorb excess fluid. They must be used in conjunction with other dressings such as transparent adhesive films or hydrocolloids. In addition to absorbing exudate, the beads and pastes facilitate moist wound healing, autolysis and may control odour. If beads are used, care must be taken to remove all the beads with each dressing change to prevent the formation of granuloma. If allowed to dry out, beads and pastes may adhere to both necrotic and granulating tissue causing damage to healthy tissue when they are removed. The pastes are easier to remove than the beads and are less likely to injure granulating tissue on removal (Alvarez et al., 1989; David, 1985; Doughty, 1992; Rolstad, 1991).

Absorptive dressings composed of calcium alginate (a naturally occurring polymer derived from brown seaweed) form a gel during absorption. Calcium alginate dressings are conformable, but non-occlusive requiring an occlusive, secondary dressing. The dressing is removed when it is fully saturated and still moist; they cause little trauma on removal. Calcium alginate dressings control odour and cause minimal maceration of surrounding skin (Doughty, 1991; Fowler & Papen, 1991; Rolstad, 1991).

Other Treatment Modalities

The care and treatment of chronic wounds described under local care are the most widely used methods, but there are other treatments that are gaining credibility as viable treatment options. These treatments include the use of hyperbaric oxygen, growth factors and electrical stimulation. As well, there are surgical options for non-healing, chronic wounds. An understanding of surgical options and newly developed treatments will assist the nurse in assessing and providing nursing care for patients with chronic wounds.

Surgical Options

Surgical options include skin grafts, tissue flaps and tissue expansion. Skin grafts for chronic wounds are usually split-thickness grafts from donor site transplanted to the wound bed. Because chronic wounds often involve deep tissue loss, and skin grafts do not replace tissue, skin grafts are not commonly used as a treatment option (Bryant et al., 1992; Kosiak, 1991; Torrance, 1983).

The most common procedure for surgically closing a chronic wound is by tissue flap. Rigorous surgical debridement of necrotic and/or avascular tissue is followed by advancement/rotation/transposition of a fasciocutaneous or musculocutaneous flap from an adjacent tissue to the wound bed. Regardless of method, the tissue flap is partially detached from its original position and then re-attached over the chronic wound. In this way, the flap retains its circulatory support and fills in the defect left by lost tissue (Bryant et al., 1992; Torrance, 1983).

A less common surgical procedure is tissue expansion. When tissue flaps cannot be performed because of the lack of adjacent tissue to the wound, hollow silistac sacs are surgically placed into the subcutaneous or submuscular tissue next to the wound. After placement, sterile fluid is injected into the sac resulting in growth and expansion of the overlying subcutaneous and/or submuscular tissues. A tissue flap is performed when enough tissue has been formed to fill the wound (Bryant et al., 1992).

Hyperbaric Oxygen

Hyperbaric oxygen (HBO) treatments involve periodic administrations of 100% oxygen at two or three times normal atmospheric pressure. Theoretically, the treatment is effective for promoting wound healing because it addresses the common underlying wound healing because it addresses the common underlying cause of chronic wounds--ischemia. Davis, Buckley and Barr (1988) reported a 70% success rate when they used HBO treatment on chronic wounds. Several beneficial effects of HBO have been cited. First, hyperoxygenation of tissues occurs because the oxygen is delivered under pressure. Although hemoglobin molecules are restricted in the amount of oxygen they can carry, oxygen under pressure can be forced into the blood and plasma improving tissue perfusion in ischemia areas. Secondly, HBO functions as a vasoconstrictor (less ischemia--less vessel dilation) which reduces edema in compromised tissue. Thirdly, when PO_2 is increased fibroblasts increase collagen production forming a supporting matrix for capillary angiogenesis as new capillary beds are formed, oxygenation for cell mitosis and ultimately wound healing is provided. Lastly, HBO increases bacterial killing by white blood cells, decreases the ability of some bacteria to produce toxins and increase the ability of antibiotics to kill bacteria (Davis et al., 1988; Meiroff, Junen, Mitchell, & Palmer, 1992).

Growth Factors

Increased knowledge about wound healing has led to the identification of multiple growth factors. Growth factors are proteins that affect target cells and have many functions. They act in concert to heal wounds and control fetal development, but they have also been implicated in the stimulation of cancer cells.

Therapeutic growth factors are synthesized from original genes through molecular biology. They are applied daily to open wounds and covered with a non-adherent dressing. Thus far, several growth factors have been synthesized and studied for their effects on wound healing, and the results are positive (Braden & Bryant, 1990; Confer, 1992; Knighton, Ciresi, Figel, & Butler, 1986; Ratafia, 1988). Growth factors that are becoming commercially available include (a) transforming growth factor-beta which can attract inflammatory cells in some situations and stimulate proliferation of fibroblasts in others, (b) platelet derived growth factor which causes white blood cells to secrete enzymes and structural proteins, increases fibroblastic proliferation and stimulates chemotaxis, (c) fibroblast growth factor which causes proliferation of fibroblasts, muscle cells, pericytes and nerve cells and (d) epidermal growth factor which targets epidermal cells, glial cells, smooth muscle cells and fibroblasts stimulating them to proliferate (Confer, 1992).

Electrical Stimulation

In the past 10 years, electrical stimulation has been tried as a healing stimulant for wounds, but studies have failed to demonstrate a definitive therapeutic effect. Problematic in using electrical stimulation as a wound healing treatment is the uncertainty about the kind of current (direct or pulsed), the wave length and the amount of voltage that should be used (Beverly & Coombs, 1985; Braden & Bryant, 1990; Frantz, 1992; Kosiak, 1992).

Conclusion

A chronic wound, whether it be a pressure, venous or arterial ulcer is the visible result of tissue ischemia, death and necrosis. Nursing care of the patient with a chronic wound begins with a thorough assessment and includes treatment of both systemic and local factors. Systemic care should address both intrinsic and extrinsic factors that can promote or retard wound healing. Local care of a chronic wound includes assessment, debriding and dressing. Other treatments for chronic wounds include surgical options and the newly introduced treatments of hyperbaric oxygen, growth factors and electrical stimulation. An understanding of wound healing, the etiology of chronic wounds and the significance of risk factors will assist the nurse in providing appropriate care for the patient who is at risk for developing or who has a chronic wound.

References

Allman, R. (1989). Pressure ulcers among the elderly. New England Journal of Medicine, 320, 850-853.

Alvarez, O. (1991). Pressure ulcers: Critical considerations in prevention and management. Clinical Materials, 8, 209-222.

Alvarez O., Mertz, P., & Eaglestein, W. (1983). The effect of occlusive dressings on collagen synteasis and re-epithelialization in superficial wounds. Journal of Surgical Research, 35, 142-148.

Alvarez, O., Rozint, J., & Wiseman, D. (1989). Moist environments for healing: Matching the dressing to the wound. Wounds: A Compendium of Clinical Research and Practice, Premiere issue, 35-51.

Anderson, M. (1985). Wound care. The Canadian Nurse, 81(8), 34-37.

Anthony D. (1984). Measuring pressure sores accurately. Nursing Times, 80(36), 33-35.

Barton, A., & Barton, M. (1981). The management and prevention of pressure sores. London, UK: Faber and Faber.

Bergman, R. (1986). Nursing in a changing world. International Nursing Review, 33, 110-116.

Bergstrom, N., Braden, B., Laguzza, A., & Holman, V. (1987). The Braden Scale for predicting pressure sore risk. Nursing Research, 36 205-210.

Beverly M., & Coombs, R. (1985). The management of compound fractures. In S. Westaby (Ed.), Wound Care (pp. 121-130). London, Uk: William Heinemann Medical Books.

Bohannon, R., & Pfaller, B. (1983). Documentation of wound surface area from tracings of wound perimeters. Physical Therapy, 63, 1622-1624.

Bothwell, J., & Rovee, D. (1971), The effect of dressings on the repair of cutaneous wounds in humans. In K.J. Harkiss (Ed.), Surgical dressings and wound healing (pp. 78-97). London, UK: Crosby Lockwood.

Braden, B., & Bryant, R. (1990). Innovations to prevent and treat pressure ulcers. Geriatric Nursing, 11(4), 182-186.

Brandeis, G., Norris, J., Nash, J., & Lipsitz, L. (1990). The epidemiology and natural history of pressure ulcers in elderly nursing home residents. Journal of the American Medical Association, 264 2905-2909.

Bryant, R. (1992), Skin pathology. In R. Bryant (Ed.), Acute and chronic wounds: Nursing Management (pp. 10-30). Toronto:Mosby.

Bryant, R., Shannon, M., Pieper, B., Braden, B., & Morris, D. (1992). Pressure ulcers. In R. Bryant (Ed.), Acute and chronic wounds: Nursing Management (pp. 105-163). Toronto: Mosby.

Bulstrode, C., Goode, A., & Scott, P. (1986). Stereophotogrammetry for measuring rates of cutaneous healing: A comparison with conventional techniques. Clinical Sciences, 71, 437-443.

Carr, R., & Lalagos, D. (1990). Clinical evaluation of a polymeric membrane dressing in the treatment of pressure ulcer. Decubitus, 3(3), 38-40, 42.

Colburn, L. (1990). Pressure ulcers: How to recognize and care for patients at risk. Nursing, 20(12), 60-63.

Confer, D. (1992). Molecular regulation. In R. Bryant (Ed.), Acute and chronic wounds: Nursing management (pp. 302-307).Toronto: Moseby.

Conforti, C. (1989). Pressure sores: Dressed for healing. Nursing, 19(3), 58-61.

Cooper, D. (1990). Optimizing wound healing. Nursing Clinics of North America, 25(1), 165-180.

Cuzzel, J. (1990). Clues: Bruised, torn skin. American Journal of Nursing, 90(3), 16, 18.

David, J. (1985). Wound management: A comprehensive guide to dressing and healing. Nursing Handbook Series. London, UK: Martin Dunitz.

Davis, J., Buckley, C., & Barr, O.(1988). Compromised soft tissue wounds: Correction of wound hypoxia. In J. Davis & T. Hunt (Eds.), Problem wounds: The role of oxygen (pp. 143-152). New York: Elseviev.

Dimant, J., & Francis, M. (1989). Pressure sore prevention and management. Journal of gerentological Nursing, 14(8), 18-25.

Dinsdale, S. (1973). Decubitus ulcers in swine: Light and electron microscope study of pathogenesis. Archives of Physical Medicine and Rehabilitation, 54, 51-51.

Doughty, D. (1990). The process of wound healing: A nursing perspective. Progressions: Development in Ostomy and Wound Care, 2(1), 3-12.

Doughty, D. (1992). Principles of wound healing and management. In R. bryant (Ed.), Acute and chronic wounds: Nursing management (pp. 31-68). Toronto: Mosby.

Eaglestein, W., Mertz, P., & Falanaga, V. (1987). Occlusive dressings. American Family Physician, 35(3), 211-216.

Ek, A., & Bowman, G. (1982). A descriptive study of pressure sores and the characteristics of patients. Journal of Advanced Nursing, 7, 51-57.

Fellin, R. (1984). Managing decubitus ulcers. Nursing Management, 15(2), 29-30.

Fowler, E. (1982). Pressure sores: A deadly nuisance. Journal of Gerentological Nursing, 8, 680-685.

Fowler, E., & Goupil, D. (1984). Comparison of the wet-to-dry dressing and copolymer starch in the management of debrided pressure sores. Journal of Enterostomal Therapy, 11(1), 22-25.

Fowler, E., & Papen, J. (1991). Evaluation of an alginate dressing for pressure ulcers. Decubitus, 4(3), 47-48, 50, 52-53.

Frantz, R. (1992). Electrical stimulation. In R. Bryant (Ed.), Acute and chronic wounds: Nursing management (pp. 308-311). Toronto: Mosby.

Gosnell, D. (1973). An asessment tool to identify pressure sores. Nursing research, 22(1), 55-59.

Green, E., & Katz, J. (1991). Practice guidelines for management of pressures ulcers. Decubitus, 4(1), 36-42.

Guttman, L. (1955). The problem of treatment of pressure sores in patients with spinal paraplegia. British Journal of Plastic surgery, 8, 196-213.

Hardy, M. (1989). The biology of scar formation. Physical Therapy, 69, 1014-1023.

Hinman, C., & Maibach, H. (1963). Effect of air exposure and occlusion on experimental skin wounds. Nature, 200, 377-378.

Hutchinson, J., & McGuckin, M. (1990). Occlusive dressings: A microbiological and clinical review. American Journal of Infection, 18(4), 257-258.

International Association for Enterostomal Therapy. (1987). Standards of care: Dermal wound, pressure sores. Irvine, CA: Author.

International Association for Enterostomal Therapy (Standards Committee). (1988). Dermal wounds: Leg ulcers. Journal of Enterostomal Therapy, 15(3), 102-117.

Irwin, T. (1981). Wound healing: Principles and practice. New York: Chapman & Hall.

Jackson, D., & Rovee, D. (1988). Current concepts in wound healing: Research and theory. Journal of Enterostomal Therapy, 15(3), 133-137.

Kelly, L., & Mobily, P. (1991). Iatrogenesis in the elderly: Impaired skin integrity. Journal of Gerentological Nursing, 17(9), 24-29.

Kerr, J., Stinson, S., Bay, K., Thurston, N., & Leatt, P. (1980). Pressure sores: Nurses' knowledge, attitudes and clinical judgement--a preliminary investigation. Edmonton, AB: Faculty of Nursing, University of Alberta.

Kerr, J., Stinson, S., & Shannon, M. (1981). Pressure sores: Distinguishing fact from fiction. The Canadian Nurse, 77(7), 23-28.

Knighton, D., Ciresi, K., Fiegel, F., Austin, L., & Butler, E. (1986). Classification and treatment of chronic nonhealing wounds: Successful treatment with autologous platelet-derived wound healing factors. Annals of Surgery, Sept., 322-330.

Kosiak, M. (1959). Etiology and pathology of ischemic ulcers. Archives of Physical Medicine and Rehabilitation, 40, 61-69.

Kosiak, M. (1991). Prevention and rehabilitation of pressure ulcers. Decubitus, 4(2), 60-68.

Kundin, J. (1989). A new way to size up a wound. American Journal of Nursing, 89, 206-207.

Kurzuk-Howard, G., Simpson, L., & Palmieri, A. (1985). Decubitus ulcer care: A comparative study. Western Journal of Nursing Research, 7(1), 58-79.

Kynes, P. (1986). A new perspective on pressure sore prevention. Journal of Enterosomal Therapy, 13(2), 42-43.

Lanyon, S., VanNieuwenhuyzen, J., & Wearing, J. (1987). Leg ulcers: Frustration or challenge. The Canadian Nurse, 83(6), 33-36.

Longe, R. (1986). Current concepts in clinical therapeutics: Pressure sores. Clinical Pharmacy, 5, 669-681.

Makelbust, J. (1987). Pressure ulcers: Etiology and prevention. Nursing Clinics of North America, 22, 359-377.

Marvel, E., Rodheaver, G., & Xakillis, G. (1992). Pressure ulcers in adults: Prediction and prevention. Decubitus 5(3), 26-30.

Mertz, P., & Eaglestein, W. (1984). The effect of semi-occlusive dressings on the microbial population in superficial wounds. Archives of Surgery, 119, 287-289.

Moriarty, M. (1988). How color can clarify wound care. RN, Sept, 51.

Morley, M. (1990). Managing decubitus ulcers. The Canadian Nurse, 8(9), 19-20.

Myers, J. (1982). Wound healing and the use of modern surgical dressing. Pharmaceutical Journal, 229, 103-104.

National Pressure Ulcer Advisory Panel. (1989). Pressure ulcer prevalence, cost and risk assessment: Consensus development conference statement. Decubitus, 2(2), 24-28.

Needham, A. (1952). Regeneration and wound healing. London, UK: Methuen.

Nemiroff, P., Junen, A., Mitchell, S., & Palmer, J. (1992). Hyperbaric oxygen. In R. Bryant (Ed.). Acute and chronic wounds: Nursing management (pp. 311-314). Toronto: Moseby.

Norton, C. (1975). Research and the problem of pressure sores. Nursing mirror, Feb. 13, 65-67.

_____, (1991). Wound care update. Nursing, 21(4), 47-50.

Pajk, M., Craven, G., Cameron-Barry, J., Shipps, T., &

Bennen, N. (1986). Investigating the problem of pressure sores. Journal of Gerentological Nursing, 12(7), 11-16.

Ratafia, M. (1988). Growth factors for wound healing: Summary of a survey of authorities. Ostomuy/Wound Management, 20, 20-24.

Rodheaver, G. (1988). Controversies in topical wound management. Ostomy/Wound Management, 20, 58-68.

Rolstad, B. (1991). Treatment objectives in chronic wound care. Home Healthcare Nurse, 9(6), 38-44.

Rovee, O., Kurowsky, C., & Labun, J. (1972). Local wound environment and epidermal healing. Archives of dermatology, 106, 330-334.

Rubin, M. (1988). The physiology of bed rest. American Journal of Nursing, 88(1), 50-56.

Ryan, T. (1985). Current management of leg ulcers. Drugs, 30, 461-468.

Saydak, S. (1990). A pilot test of two methods for the treatment of pressure ulcers. Journal of Enterostomal Therapy, 17(3), 139-142.

Scales, J., & Winter, G. (1963). The adhesion of wound dressings: An experimental study. In D. Sloane (Ed.), Wound healing: Proceedings of a symposium held on 12-13 November, 1959 (pp. 54-61). New York: Pergamon Press.

Schurmann, D. (1982). The nature of wound healing. AORN Journal, 35, 1068-1077.

Sebern, M. (1986). Pressure ulcer managemant in home health care: Efficacy and cost effectiveness of moisture vapour permeable dressings. Archives of Physical Medicine and Rehabilitation, 67, 726-729.

Shea, J. (1975). Pressure sore classification and management. Clinical Orthopedic and Related Research, 112, 89-100.

Thomas, A., & Wysocki, A. (1990). The healing wound: A comparison of three clinically useful methods of measurement. Decubitus, 3(1), 18-25.

Thomason, S. (1989). Front-line antispectics: Gentler wound care is often smarter wound care. Geriatric Nursing, September/October, 235-236.

Torrance, C. (1983). Pressure sores: Aetiology, treatment, and prevention. London, UK: Croom Helm.

Trelease, C. (1983). Developing standards for wound care. Ostomy/Wound Management, 20, 46-50.

Turner, T. (1985). Which dressing and why? In S. Westaby (Ed.), Wound care (pp. 58-69). London, UK: William Heinemann Medical Books.

Venn, G. (1987). Leg ulcers: Pointers to management. Nursing Times, Mar. 25, 49, 51-52.

VanRijswijk, L., & Cuzzel, J. (1991). Managing full-thickness wounds. American Journal of Nursing, 91(6), 18, 22.

Westaby, S. (1985). Fundamentals of wound healing. In S. Westaby (Ed.), Wound care (pp. 11-21). London, UK: William Heinmann Medical Books.

Winter, G. (1962). Formation of the scab and the rate ofepithelialization of superficial wound in the skin of the young domestic pig. Nature, 193, 293-294.

Winter, G., & Scales, J. (1963). Effects of air drying and dressings on the surface of a wound. Nature, 197, 91-92.

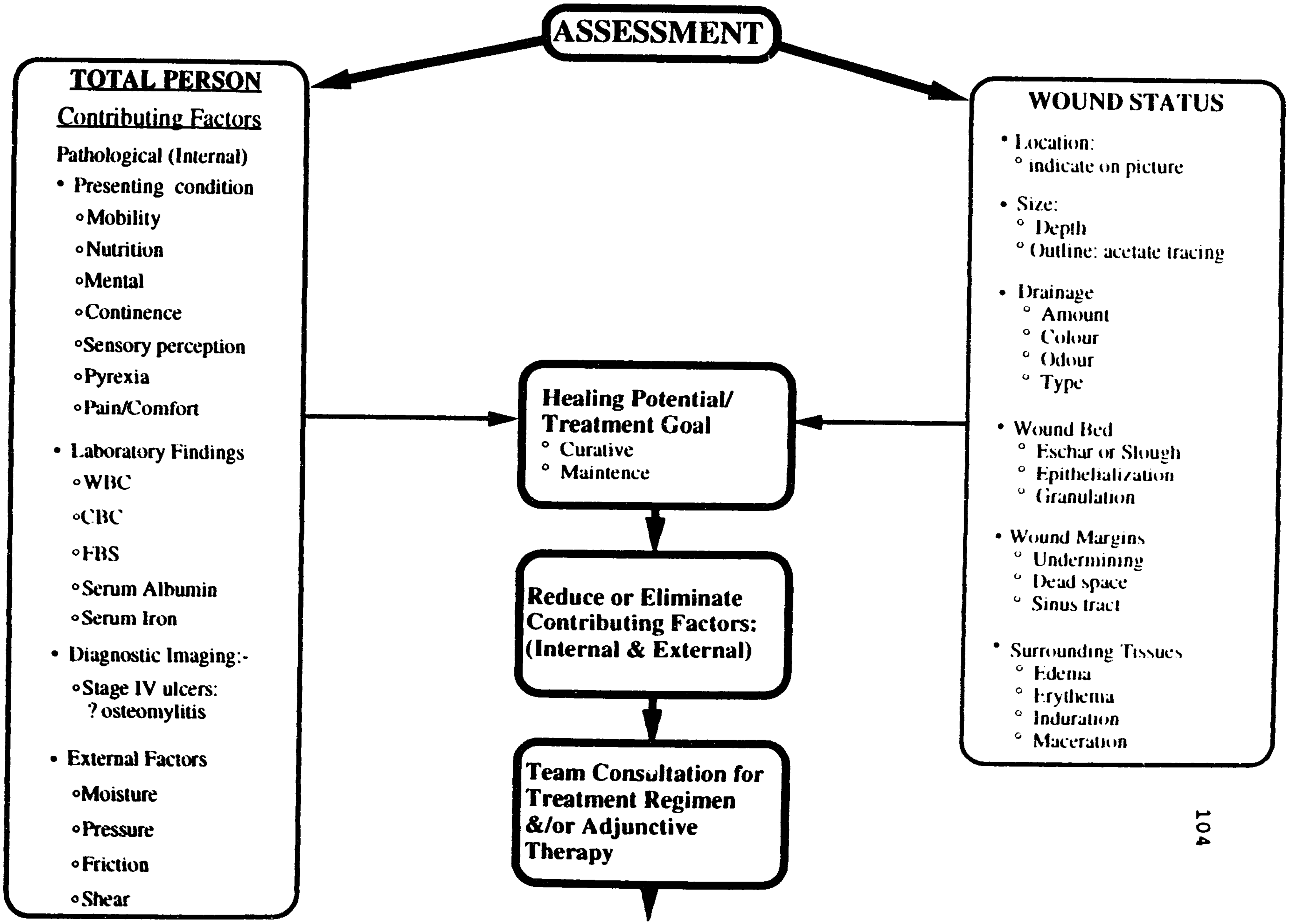
APPENDIX B

Chronic Wound Management Decision Tree

How to Follow Decision Tree

Page 1	Guides you through factors to consider in your assessement of the total person and the wound.
Page 2	Further assessment and beginning treatment options for Infection and Necrosis.
Page 3&4	Guides you through your final assessment of the wound now that you have addressed infection and removed necrotic tissue.

CHRONIC WOUND MANAGEMENT TOOL



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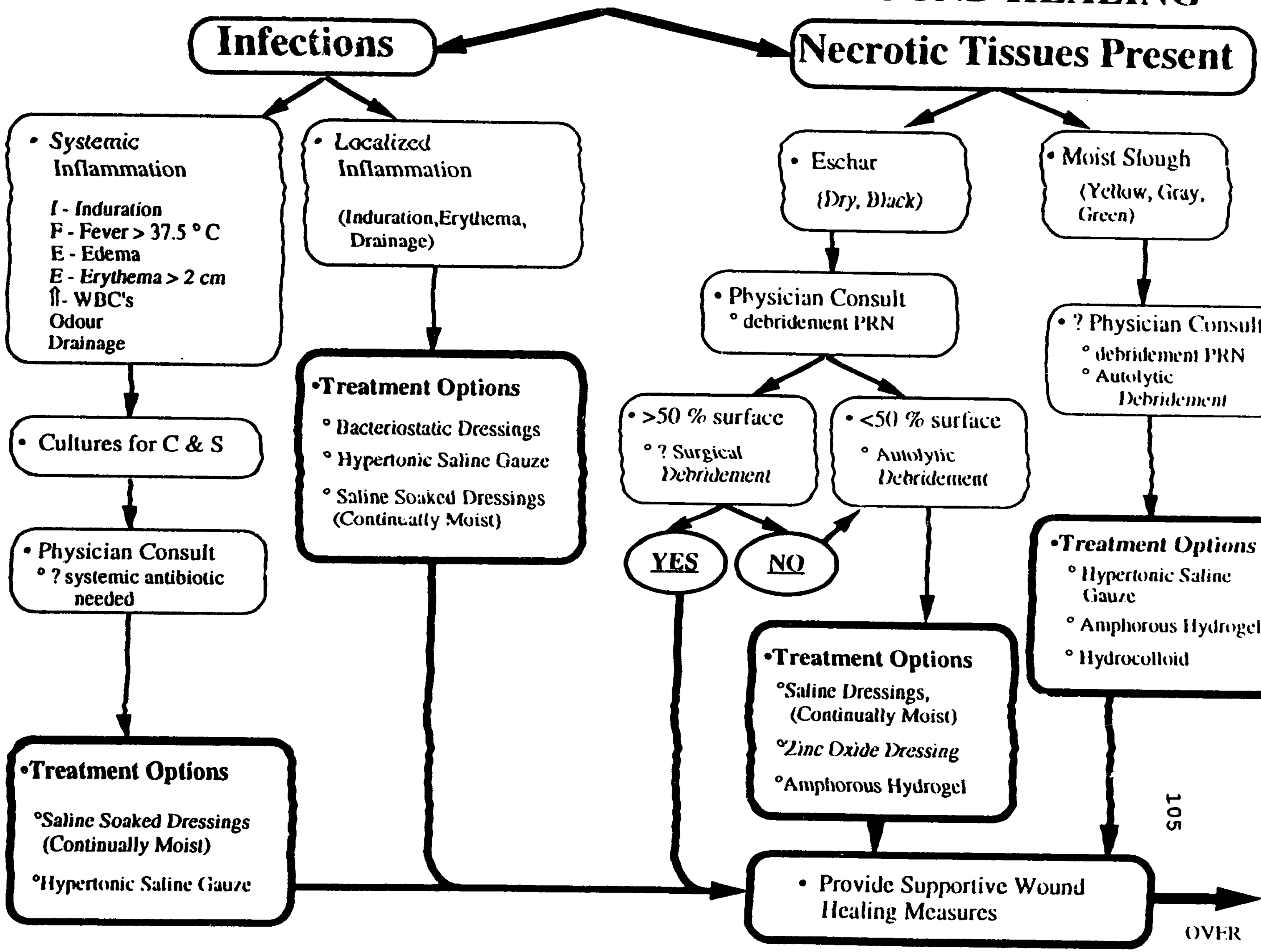
ELIMINATE FACTORS WHICH DELAY WOUND HEALING



WOUND HEALING ENVIRONMENTS

- Moist Wound Surface
- Manage Exudate
- Wound Temperature (Warm)
- Eliminate Dead Space
- Free of Clinical Infection
- Remove Necrotic Tissue
- Control Contamination

STAGE I: Reddened skin with unbroken surface; skin does not blanch with pressure.

- **Treatment Options**
  - Barrier and/or Moisture Lotions
  - Non-adherent Dressings
  - Transparent/Thin Hydrocolloid Wafer
  - Transparent Film Dressing

STAGE III: Full thickness skin loss with damage or necrosis *extending into subcutaneous* tissues.

- **Physician Consult**
  - ? Debridement with undermining wound edges

Drainage Amount

Scant / Minimal

- **Treatment Options**
  - Amphorous Hydrogel Wafer
  - Hydrocolloid Wafer/Paste
  - Saline Soaked Dressings (Continually Moist)

Moderate

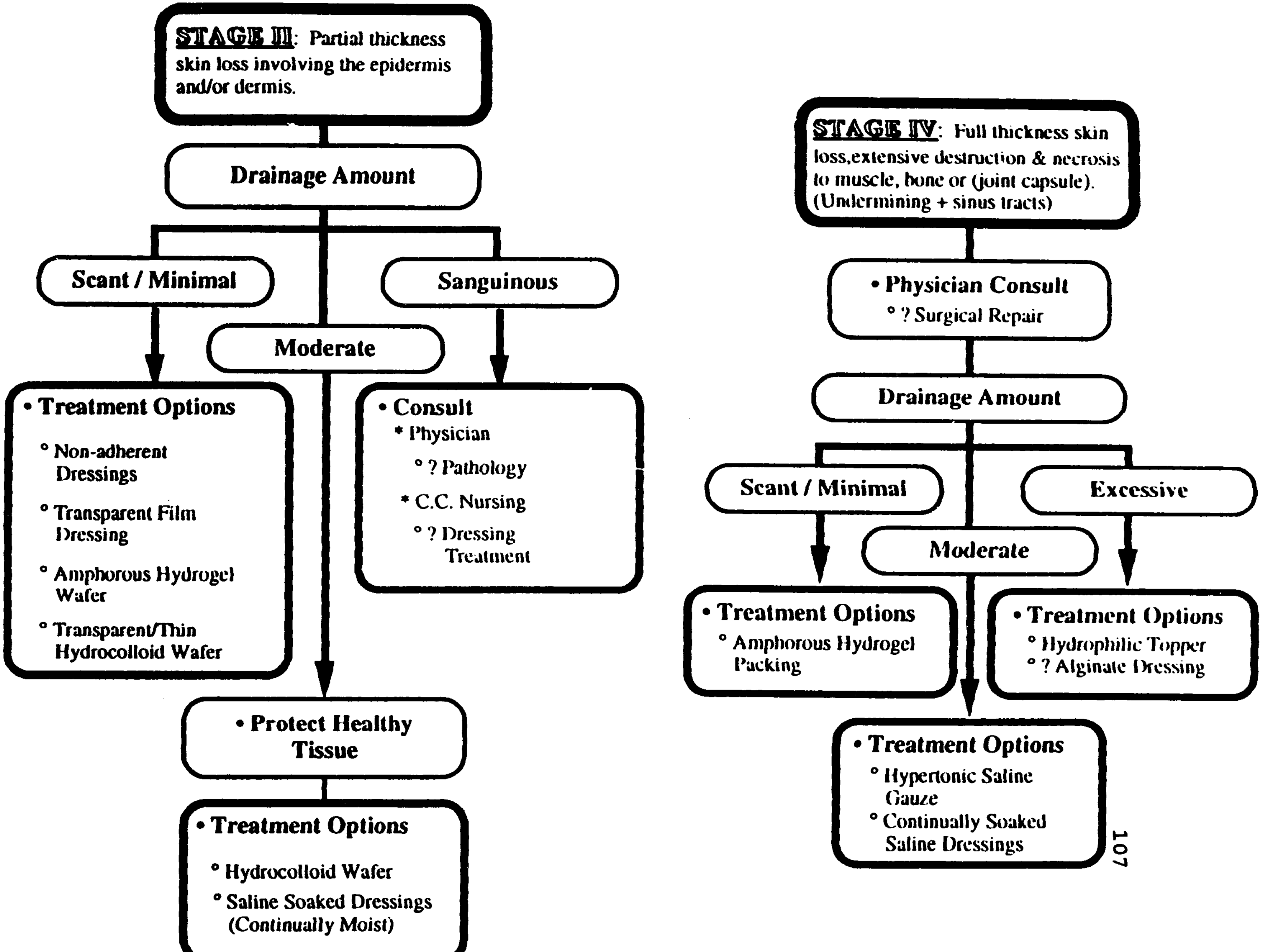
- **Treatment Options**
  - Hydrocolloid Wafer/Paste
  - Saline Soaked Dressings (Continually Moist)
  - Hypertonic Saline Gauze

Excessive

- **Treatment Options**
  - Hydrophilic Packing
  - Saline Soaked Dressings (Continually Moist) & Hydrophilic Topper
  - Hydrocolloid Wafer/Paste
  - ? Alginate Dressing

Sanguinous

- **Consult**
  - Physician
    - ? Pathology
  - C.C. Nursing
    - ? Dressing Treatment



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APPENDIX C

Cases and Questionnaires

1. **Before proceeding,** please answer the following question.

   In one week, for how many home care clients do you provide chronic wound care? (approximately)

---

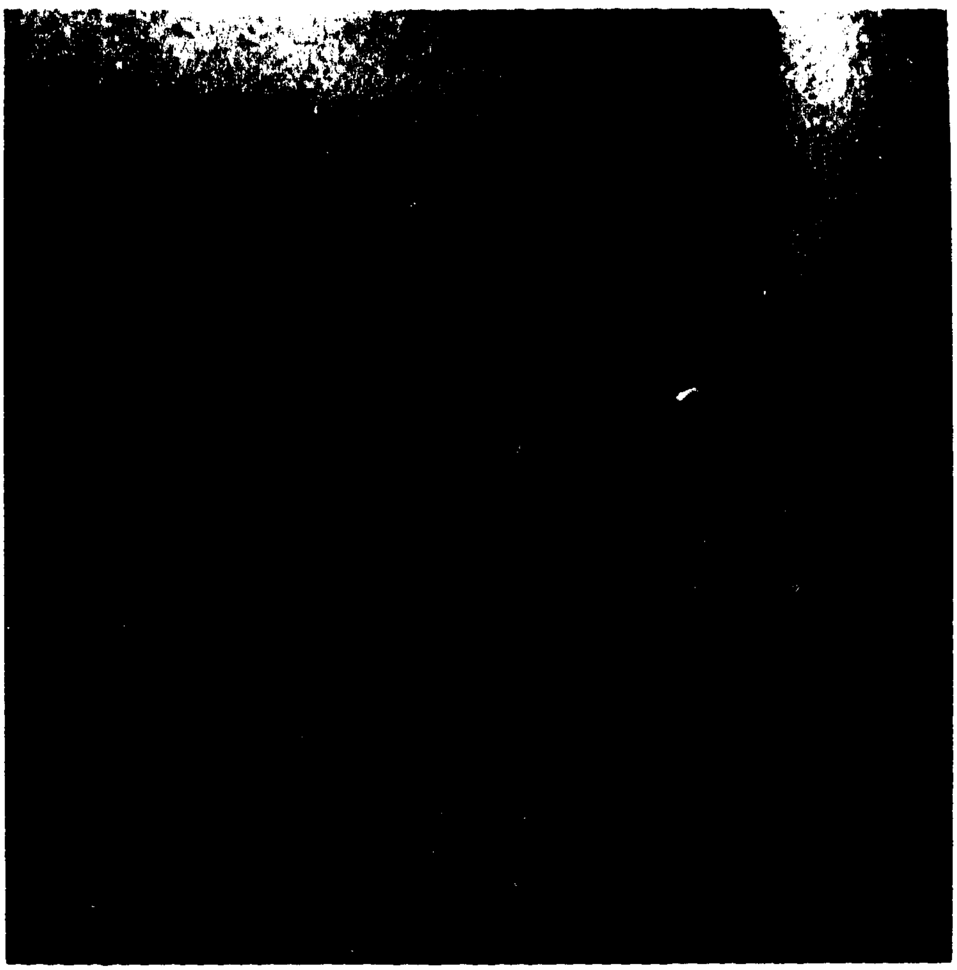
2. **Now,** proceed to the three case histories in the following pages.

CASE 1

<u>Patient history</u> - 57 year old male who had coronary artery bypass surgery within the past week.

<u>Description and location of wound</u> - fluid filled blisters on buttocks near coccyx surrounded by mild redness, otherwise this patient's skin is intact. Reddened area with blisters measures approximately 6 cm in length and 2 cm in width, shape is linear.

<u>Photograph of wound</u>

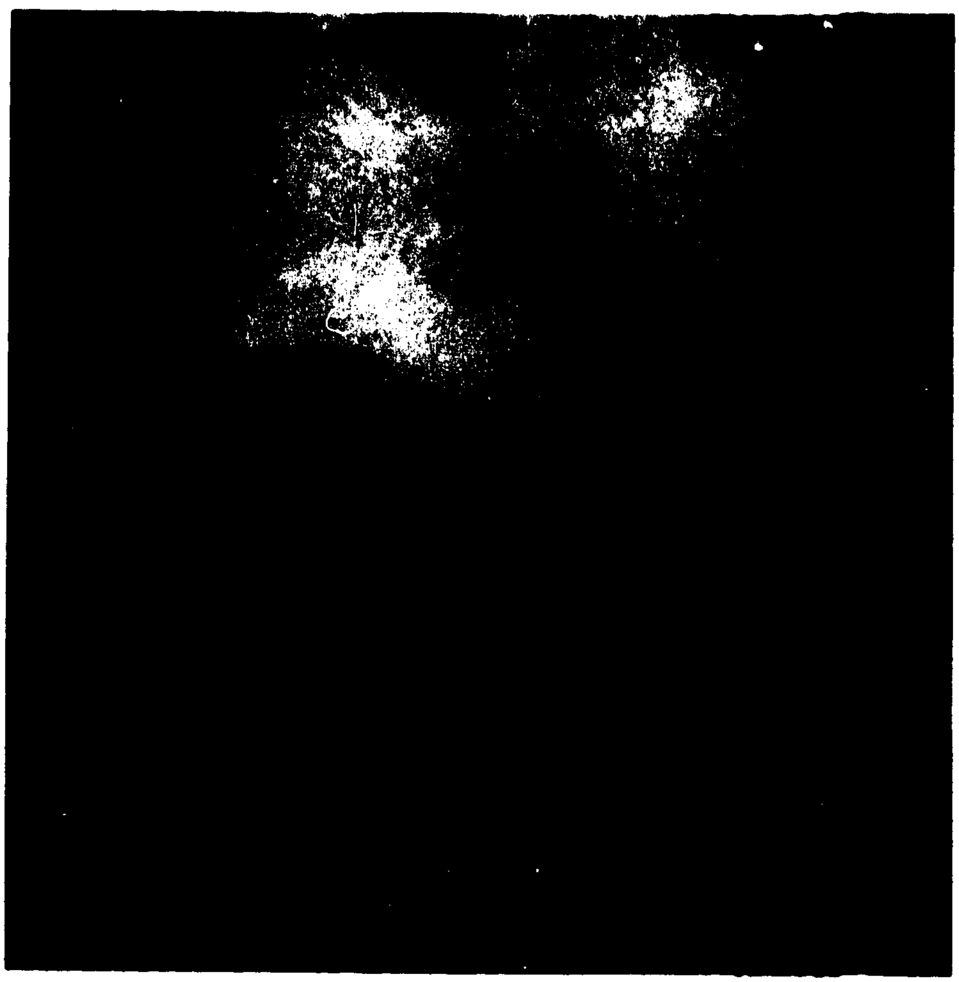


CASE 2

Patient history - a 23 year old male quadraplegic who was hospitalized for pneumonia.

Description and location of wound - the width of the wound is 8 cm, length is 5 cm, shape is oval. The wound is clean and has no undermining or tunneling. The surrounding skin is soft with no induration, redness, edema or heat. There is no purulent drainage from the wound. The amount of drainage or exudate is minimal. The wound is located over the right buttock.

Photograph of wound



CASE 3

Patient history - 73 year old male hospitalized following mitral valve replacement and multiple vessel coronary bypass surgery.

Description and location of wound - on inner aspect of the right lower extremity just above the ankle. There is no undermining, tracking or tunneling. The wound depth is 3 mm, length is 5 cm, with is 3 cm, shape is oval. The wound is covered with a thick, yellow fibrous tissue. There is minimal amount of drainage or exudate.

Photograph of wound

QUESTIONNAIRE FOR CASES 1 - 3
Control Group

1. Assessment of Wound Depth:

Using the picture in the case history, select the <u>one</u> **WOUND DEPTH** which best describes the wound in **CASE HISTORY ()**.

____Stage I (redness)
____Stage II (loss of superficial skin layer only)
____Stage III (tissue loss extending into subcutaneous tissue)
____Stage IV (tissue loss extending to muscle and/or bone)
____Unable to determine stage of wound in present state

2. Choice of Wound Dressing:

Using your knowledge and experience in wound care, state below the specific type of dressing that you feel would be the <u>**Most Appropriate**</u> for promoting maximum healing in the shortest amount of time for the wound in the **CASE HISTORY ()**.

____

____

____

QUESTIONNAIRE FOR CASES 1 - 3
Experimental Group

1. Assessment of Wound Depth:

Using the picture in the case history and the decision tree to help make your selection, select the <u>one</u> WOUND DEPTH which best describes the wound in **CASE HISTORY** ().

____Stage I (redness)
____Stage II (loss of superficial skin layer only)
____Stage III (tissue loss extending into subcutaneous tissue)
____Stage IV (tissue loss extending to muscle and/or bone)
____unable to determine stage of wound in present state

2. Choice of Wound Dressing:

Using the decision tree as a reference, identify the specific type of dressing that would be the **<u>Most Appropriate</u>** for promoting maximum healing in the shortest amount of time for the wound in **CASE HISTORY** ().

____

____

____

APPENDIX D

Information Sheet

1. PLEASE, DO <u>NOT</u> OPEN ENVELOPES

2. READ THIS PAGE NOW

These materials are part of a study called "Clinical Decision Making in Chronic Wound Management".

This study is being do

Florence MacD
Master of Nursing Candidate
University of Alberta
Phone: Leave message at 455-1955 or 492-6317

The purpose is to examine the use of different materials in making decisions about wound management.

You will be shown three pictures of wounds and given information about the cases. You will be asked how you would care for each wound. The materials which have been provided for you to make a decision will vary; you will be randomly assigned to type of material. You will be able to see all of the materials during the educational session on wound management which follows this study.

You will be asked to provide some information about yourself, such as education and nursing experience. You will not be asked for your name.

This study will take about 30 minutes.

You do not have to participate in the study if you do not wish. No one will know that you have not participated unless you choose to tell them. Simply hand in the uncompleted materials at the same time as everyone else.

If there are questions you prefer not to answer, just leave them out.

Your answers to the questions will not be shown to anyone. Your responses are confidential and anonymous. Only grouped or aggregate information will be made available to you, the other staff and and Clinical Coordinators of Home Care.

If you have any questions after the study, please call Florence MacDougall at the above phone number or Janice Lander (thesis supervisor) at the Faculty of Nursing (492-6317).

PLEASE, WAIT FOR INSTRUCTIONS BEFORE PROCEEDING

APPENDIX E

Biographical Questionnaire

BIOGRAPHICAL QUESTIONNAIRE

1. Education: Please indicate your level of education:

    ____Diploma in Nursing
    ____Basic Degree in Nursing
    ____Post-RN Degree in Nursing
    ____Other (please specify below)

    ____

2. Amount of nursing experience in home care: Please indicate in number of years and months.

    ____

3. Total amount of nursing experince: Please indicate in number of years and months.

    ____