



If you have discovered material in AURA which is unlawful e.g. breaches copyright, (either yours or that of a third party) or any other law, including but not limited to those relating to patent, trademark, confidentiality, data protection, obscenity, defamation, libel, then please read our [Takedown Policy](#) and [contact the service](#) immediately

MATHEMATICAL MODELLING OF PATIENT RISKS IN A
HOSPITAL ENVIRONMENT, USING MULTIPLE REGRESSION ANALYSIS

A THESIS SUBMITTED TO
THE UNIVERSITY OF ASTON IN BIRMINGHAM
FOR THE AWARD OF THE DEGREE OF
DOCTOR OF PHILOSOPHY

by

BRIAN ARTHUR BIBBY B.Sc. (Hons).

JUNE, 1982

MATHEMATICAL MODELLING OF PATIENT RISKS IN A
HOSPITAL ENVIRONMENT, USING MULTIPLE REGRESSION ANALYSIS

Submitted by

BRIAN ARTHUR BIBBY

for the award of the degree of Doctor of Philosophy

June, 1982

S U M M A R Y

The aim of this research work was primarily to examine the relevance of patient parameters, ward structures, procedures and practices, in respect of the potential hazards of wound cross-infection and nasal colonisation with multiple resistant strains of Staphylococcus aureus, which it is thought might provide a useful indication of a patient's general susceptibility to wound infection.

Information from a large cross-sectional survey involving 12,000 patients from some 41 hospitals and 375 wards was collected over a five-year period from 1967-72, and its validity checked before any subsequent analysis was carried out. Many environmental factors and procedures which had previously been thought (but never conclusively proved) to have an influence on wound infection or nasal colonisation rates, were assessed, and subsequently dismissed as not being significant, provided that the standard of the current range of practices and procedures is maintained and not allowed to deteriorate.

Retrospective analysis revealed that the probability of wound infection was influenced by the patient's age, duration of pre-operative hospitalisation, sex, type of wound, presence and type of drain, number of patients in ward, and other special risk factors, whilst nasal colonisation was found to be influenced by the patient's age, total duration of hospitalisation, sex, antibiotics, proportion of occupied beds in the ward, average distance between bed centres and special risk factors.

A multi-variate regression analysis technique was used to develop statistical models, consisting of variable patient and environmental factors which were found to have a significant influence on the risks pertaining to wound infection and nasal colonisation.

A relationship between wound infection and nasal colonisation was then established and this led to the development of a more advanced model for predicting wound infections, taking advantage of the additional knowledge of the patient's state of nasal colonisation prior to operation.

NOSOCOMIAL INFECTION
CROSS-INFECTION
STAPHYLOCOCCUS AUREUS
STATISTICAL MODEL
EPIDEMIOLOGY

C O N T E N T S

SUMMARY

DECLARATION

viii

DEDICATION

ix

ACKNOWLEDGEMENTS

x

LIST OF TABLES

xi

LIST OF FIGURES

xiii

DEFINITIONS

xv

CHAPTER	1	INTRODUCTION	
	1.1	Historical background	1
	1.2	Cost of hospital-acquired infection for acute beds in England and Wales	4
	1.3	Breakdown of additional costs of infection	6
	1.4	Quantitative measurement of risks to patients	8
	1.5	Mathematical modelling of infection	12
CHAPTER	2	LITERATURE SURVEY	
	2.1	Relative predominance of <u>Staph. aureus</u>	15
	2.2	Age of patient	18
	2.3	Sex of patient	18
	2.4	Pre-operative hospitalisation	19
	2.5	Effect of prophylactic antibiotics	19

	2.6	Pre-operative wound preparation	21
	2.7	Length of incision and duration of operation	22
	2.8	Air conditioning and ventilation	23
	2.9	Drained wounds	23
	2.10	Special risks	24
	2.11	Transmission of infections to wounds	25
	2.11.1	Urinary catheters and intravenous devices	26
	2.11.2	Contaminated equipment	27
	2.11.3	Infected patient secretions or excretions	27
	2.11.4	Contaminated solutions	28
	2.11.5	Hand transmission	29
	2.11.6	Self-infection	29
	2.12	Isolation procedures	31
	2.13	Effect of wound infection on the duration of post-operative hospitalisation	32
	2.14	Communication and reporting of infections	32
	2.15	Infection control programmes	34
	2.16	Cost benefits of effective infection control	35
CHAPTER	3	IMPLICATIONS	
	3.1	Critique of the literature survey	37
	3.2	General conclusions derived from the literature survey	39
CHAPTER	4	ASSESSMENT of WARD PARAMETERS	

	4.1	The effect that varying ward parameters have on 'nasal colonisation rates' and 'wound infection rates' in a hospital environment	42
	4.2	Action taken - based on ward data summarised in Tables 1-6 (a and b)	47
CHAPTER	5	WARD STRUCTURES AND FACILITIES	
	5.1	Analysis of nasal results summarised in Tables 1a and 1b	50
	5.2	Analysis of wound results summarised in Tables 2a and 2b	58
CHAPTER	6	WARD PRACTICES I	
	6.1	Analysis of nasal results summarised in Tables 3a and 3b	64
	6.2	Analysis of wound results summarised in Tables 4a and 4b	69
CHAPTER	7	WARD PRACTICES II	
	7.1	Analysis of nasal results summarised in Tables 5a and 5b	73
	7.2	Analysis of wound results summarised in Tables 6a and 6b	80
CHAPTER	8	DISTRIBUTION OF THE PATIENT POPULATION	
	8.1	Age distribution and patients	86
	8.2	Relationship between age, drains and wound type	88
	8.3	Relationship between age and duration of hospitalisation	92

	8.4	Distribution of pre-operative hospitalisation	94
	8.5	Distribution of post-operative hospitalisation	96
	8.6	Distribution of total durations of hospitalisation	98
CHAPTER	9	DISTRIBUTION OF COLONISATION AND INFECTION RATES	
	9.1	Relationship between age and patient risks	104
	9.2	Relationship between duration of hospitalisation and patient risks	110
	9.3	Influence of antibiotics	117
	9.4	Distribution of different wound types	120
	9.5	Influence of operation duration and incision length	123
	9.6	Distribution of different drain types	125
	9.7	Influence of special risk factors	125
	9.8	Pure risk profiles	129
	9.9	Partitioned risk profiles	135
CHAPTER	10	STEPWISE REGRESSION PROCEDURE	
	10.1	Computational method for stepwise regression procedure	140
	10.2	Mathematical discussion for the stepwise regression procedure	143
CHAPTER	11	SUMMARY OF CALCULATIONS USED IN THE STEPWISE REGRESSION PROCEDURE	

	11.1	Selection of the key element	148
	11.2	Calculation of regression coefficients and standard deviations	150
	11.3	Calculation of the square of the multiple correlation coefficient	151
	11.4	Calculation of predicted values for the dependent variable and deviations between 'actual' and 'predicted' values	152
	11.5	Results derived from the stepwise regression procedure	152
	11.6	Distribution of predicted patient risks	159
CHAPTER	12	RELATIONSHIP BETWEEN NASAL COLONISATION AND WOUND INFECTION	
	12.1	Evaluation of interdependence	164
	12.2	Accuracy of the wound infection model	172
	12.3	Extent of staphylococcal wound infections	174
CHAPTER	13	CONCLUSIONS	179
CHAPTER	14	FUTURE WORK	183
<u>A P P E N D I C E S</u>			
APPENDIX	A	COST OF ANTIBIOTIC THERAPY	
	A1	Relative cost of five days oral antibiotic therapy with tablets/ capsules	186

APPENDIX B	WARD SURVEY RECORDS	
B1	Ward structures and facilities	187
B2	Ward practices I	193
B3	Ward practices II	198
APPENDIX C	PATIENT SURVEY RECORDS	
C1	Patient's information record	204
APPENDIX D	TYPE OF PATIENTS AND OPERATIONS IN THE SURVEY	
D1	Type of patients in the survey	207
D2	Type of operations in the survey	209
APPENDIX E	VALIDATION OF DATA	
E1	Listing of variables used in the processing, selection and validation of patient data for both 'nasal acquisition rate' and 'wound infection rate' models	212
E2	Flowchart for validation of each patient's nasal input data	216
E3	Flowchart for validation of each patient's wound input data	222
APPENDIX F	MULTIPLE REGRESSION ANALYSIS	
F1	Listing of variables used in the 'stepwise regression' program	228
F2	Flowchart for regression analysis program	232

REFERENCES

242

BIBLIOGRAPHY

256

DECLARATION

No part of the work described in this thesis has been submitted in support of an application for another degree or qualification of this or any other University or other institute of learning.

No part of the work described in this thesis has been done in collaboration with any other person.

B. A. Bibby

DEDICATED

TO

MY PARENTS

A C K N O W L E D G E M E N T S

Foremost, I would like to thank my Research Supervisor, Dr. R. A. Etheridge[†] for his invaluable advice, guidance and continued encouragement throughout the duration of this project.

Professor G. A. J. Ayliffe^{†*} and Mr. B. J. Collins^{*} are thanked for providing me with much valuable advice based on their vast wealth of experience in all aspects of medical microbiology, as are their colleagues from the Hospital Infection Research Laboratory, who carried out the original surveys and analysed the huge volume of bacteriological samples collected in the process.

I wish also to thank Professor R. H. Thornley[†] for affording me the opportunity to carry-out this research work in the Bio-Medical Engineering Unit of his department.

I gratefully acknowledge the financial support provided by the Science and Engineering Research Council, together with additional resources provided by the Medical Research Council.

Finally, I wish to thank Elizabeth Melding for her interest, patience and competent efforts in typing-up the original manuscript, and Barbara Homer for her additional expertise in preparing the final manuscript.

[†]Department of Production Technology and Production Management, University of Aston in Birmingham.

[†]Department of Medical Microbiology, University of Birmingham.

^{*}Hospital Infection Research Laboratory, Dudley Road Hospital, Birmingham.

L I S T O F T A B L E S

1a & 1b	Analysis of variable ward structures and facilities (with respect to the 'nasal acquisition' rate of resistant <u>Staph. aureus</u>)	51, 52
2a & 2b	Analysis of variable ward structures and facilities (with respect to patient 'wound infection' rates)	59, 60
3a & 3b	Anaylsis of variable ward practices I (with respect to the 'nasal acquisition' rate of resistant <u>Staph. aureus</u>)	65, 66
4a & 4b	Analysis of variable ward practices I (with respect to patient 'wound infection' rates)	70, 71
5a & 5b	Analysis of variable ward practices II (with respect to the 'nasal acquisition' rate of resistant <u>Staph. aureus</u>)	74, 75
6a & 6b	Analysis of variable ward practices II (with respect to patient 'wound infection' rates)	81, 82
7	Age distribution of patients for different TYPES of WOUND	90
8	Nasal colonisation rates for different AGE groups	106
9	Wound infection rates for different AGE groups	108
10	Nasal colonisation rates connected with different TOTAL durations of HOSPITALISATION	112
11	Wound infection rates connected with different durations of PRE-OPERATIVE HOSPITALISATION	116
12	Nasal colonisation rates associated with different courses of ANTIBIOTIC TREATMENT	119
13	Infection rates for different TYPES of WOUND	121
14	Nasal colonisation rates associated with different TYPES of WOUND	122
15	Wound infection rates associated with different operation duration-incision lengths	124

16	Wound infection rates associated with different TYPES of DRAIN	126
17	Nasal colonisation rates linked with different SPECIAL RISK FACTORS	127
18	Wound infection rates linked with different SPECIAL RISK FACTORS	128
19	Analysis of variance for the nasal colonisation model	153
20	Summary of statistical information on parameters relevant to nasal colonisation rates	155
21	Analysis of variance for the wound infection model	156
22	Summary of statistical information on parameters relevant to wound infection rates	158
23	Relationship between nasal colonisation and wound infection	166
24a	Influence of colonisation on wound infection	168
24b	Contingency table for actual wound infections	171
25	Contingency table for predicted wound infections	173
26	Breakdown of infected wounds	174
27	Analysis of variance for the wound-nasal model	176
28	Statistical information on parameters (including colonisation) relevant to wound infection rates	178

L I S T O F F I G U R E S

1	Age distribution for ALL patients	87
2	Age distribution for those patients having an operation wound	89
3	Cumulative distribution of patients having staphylococcal wounds	91
4	Age of patient versus total duration of hospitalisation	93
5	Cumulative distribution of pre-operative hospitalisation	95
6	Cumulative distribution of post-operative hospitalisation	97
7	Cumulative distribution of hospitalisation for ALL patients	99
8	Cumulative distribution of hospitalisation for different sexes of patient	101
9	Cumulative distribution of hospitalisation for operated patients	102
10	Age of patient versus rate of nasal colonisation	105
11	Age of patient versus rate of wound infection	107
12	Relationship between different types of wound infection and age	109
13	Total duration of hospitalisation versus rate of nasal colonisation	111
14	Influence of antibiotics on rates of nasal colonisation	114
15	Duration of pre-operative hospitalisation versus rate of wound infection	115
16	Relationship between different types of wound infection	118

17	Colonisation profile for all patients' noses	131
18	Infection profile for all patients' wounds	132
19	Infection profile for patients' wounds infected with staphylococci	133
20	Infection profile for patients' wounds	134
21	Colonisation profile for different types of patient	136
22	Colonisation profile for different types of patient wounds	137
23	Infection profile for different types of patient	138
24	Distribution of patients associated with nasal colonisation	160
25	Distribution of operated patients associated with nasal colonisation	161
26	Distribution of patients associated with wound infection	162
27	Overall relationship between nasal colonisation and wound infection	165

DEFINITIONS

- HOSPITAL-ACQUIRED INFECTION
(NOSOCOMIAL INFECTION) - An infection which occurs in a patient after admission to hospital, but one which was not present, or incubating, at the time of admission.
- COMMUNITY-ACQUIRED INFECTION - An infection which occurs before a patient is admitted to hospital but which may not become apparent for up to 72 hours after admission.
- STAPHYLOCOCCUS AUREUS - Coagulase-positive staphylococcus.
- NASAL COLONISATION - A patient is defined as colonised, if Staphylococcus aureus, which was resistant to at least the antibiotic tetracycline, could be recovered from nasal swabs.
- CLEAN WOUND - An operation where the gastrointestinal, respiratory, genital or urinary tracts, are not entered, and where inflammation is not encountered (e.g. hernia repair, pinning for traction).

CLEAN-CONTAMINATED WOUND

- A wound operation that transects one of the above systems, where bacterial contamination might occur, but without significant spillage (e.g. operations on stomach, gall bladder, vagina, appendicectomy en passant).

CONTAMINATED WOUND

- An operation where one of the above systems (which is known to be contaminated) is opened, or in the vicinity of apparent inflammatory reactions (e.g. operations on the colon, mouth or perforated appendix).

WOUND INFECTION

- Wounds are defined as being infected if the severity is considered to be greater than doubtful, i.e. mild, moderate or severe.

MILD INFECTION

- A superficial or small area of inflammation with only a minimal discharge.

MODERATE INFECTION

- Superficial inflammation covering more than one third of the wound with a serous exudate or small amount of purulent discharge, or a deeper

infection involving a smaller area which usually has a purulent discharge.

SEVERE INFECTION

- A deep wound infection which is purulent, and may or may not have sinuses, fistulae, widespread cellulitis or wound breakdown with an obvious inflammatory reaction and pus.

PREVALENCE

- Prevalence is the ratio of the number of cases (at any given time) to the population (at that time). Results from this type of survey tend to be weighted according to the number of days spent in hospital care by infected or colonised patients.

INCIDENCE

- Incidence rate gives the number of new cases of a disease per unit time, but no reference is made to the size of the population. More useful are relative incidence rates which use 'person years' as the denominator, e.g. relative incidence rate might be the number of new cases per 1,000 persons per year. Results from this

type of survey tend to be weighted according to the number of infected or colonised patients admitted or discharged.

ASEPSIS

- The term asepsis is taken to mean not only physical methods used to prevent contamination of wounds, but also to include prophylaxis by antiseptics and antibiotics.

C H A P T E R 1

INTRODUCTION

1.1. Historical background

Semmelweiss, in 1847, identified the hands of doctors and students as carriers of infection in puerperal sepsis, which he eventually reduced by insisting on handwashing with hypochlorite solution. Ironically, the day after Lister applied carbolic acid as an antiseptic to a compound fracture of young James Greenlees at Glasgow Royal Infirmary in 1865, Semmelweiss himself died of a wound infection (1, 1958).

When the first reports on antiseptics in surgery were published over 100 years ago by Joseph Lister (2, 1867), the clinical benefits soon became apparent. This gentle Quaker was responsible for, arguably, the greatest milestone in the history of surgery, because today, it is very difficult to visualise the misery and suffering caused by surgical operations subsequently resulting in 'hospital gangrene' from the pre-Listerian era. This work led to further advancements in 1882 by Neuber of Kiel, who was the first to break away from Lister's antiseptic wound irrigation in favour of aseptic surgery. He substituted saline irrigations, adapted caps and gowns, developed theatre furniture together with instruments that could be sterilized. Lister, however, did not accept the aseptic method and stated "...asepsis in this imperfect world is not to be trusted. Human carelessness and fallibility are common and it is safer to have an antiseptic" (3, 1949). Lister then continued to use carbolic acid to prevent access of bacteria to his patients' wounds and also to destroy those already present (4, 1970).

Lord Moynihan was reported as saying that two out of three patients used to die after opening the peritoneum, when he was a house surgeon in 1888. Brewer, writing in 1915, reported that 20 years earlier - when he joined the staff of the Roosevelt Hospital in New York, that septic infection followed as a result of some 40% of all clean operations. More recently, with the gradual introduction of dust-free operating theatres, isolation methods, and antibiotic therapy to combat bacterial infection, FOWLER (5, 1963) advocated that it was more evident that antisepsis must be used as the weapon of attack to supplement the main defence of asepsis.

As a result of these developments, many of the more severe infections disappeared at an early stage, but less severe wound infection has continued to be a cause for great concern. Although much time and effort have been devoted to reducing wound infection, new hazards are created with every medical advance that keeps sick patients alive and allows a continually expanding range of operations to be performed on an increasing pool of highly susceptible 'altered hosts'. This is particularly emphasised with forms of treatment that interfere with the body's natural resistance mechanisms, e.g. the use of cytotoxic and immunosuppressive drug therapy. Since the hospitalised patient is more often than not an 'altered host' with enhanced susceptibility to infection due either to their treatment, or indeed, an underlying disease, then we clearly need to strengthen the patient's own host-defence mechanism or provide protection from potentially harmful pathogens. Ignoring these basic requirements, will inevitably result in substantially increased infection risks.

As certain infections have been brought progressively under control with the aid of antibiotics, other infections have taken their place. ALTEMEIER et al (6, 1973) noted the development of superimposed or secondary infections developing during the course of antibiotic therapy, together with an increase in those infections which were caused by bacteria previously considered to have little or no virulence.

As WENZEL et al (7, 1976) quite rightly pointed out, it should be remembered that not all hospital-acquired infections can be prevented. Certain procedures carried out within hospitals are known to be associated with a potential risk of infection, but since they are essential to the patient for either diagnosis or therapeutic treatment, they have to be used.

As a first step towards reducing preventable infection, we may recall OSLER'S axiom which states that, "It is more important to know what sort of patient has the disease, than to know what sort of disease the patient has", so we must be able to recognise severely compromised patients and identify high risk procedures.

The concept of evaluating the extent of hospital-acquired infection (or as it is sometimes referred to, nosocomial infection) was first considered by KISLAK et al (8, 1964). In a later study by ADLER and SHULMAN (9, 1970), the authors suggested that prevalence surveys are indeed a simple and effective method for determining significant trends and problems related to nosocomial infections, but they also went on to emphasise the limitations of this type of survey, whilst highlighting the need for continuous ongoing surveys.

When all survey data has been collected and analysed, however, the problem of presentation still exists. Since the staff who could make best use of the results and take action upon them are not usually experts in the evaluation of experimental data, there is a definite need to present all arguments in a logical manner, supported by data which is unambiguous, clear and simple to understand.

1.2 Cost of hospital-acquired infection for acute beds in England and Wales (excluding children)

National Health Service (N.H.S.) statistics for the year 1977/78 put the average cost per in-patient day at £28.60p, whilst a cost basis of £54.89p per 'acute' bed was the figure in use at Dudley Road Hospital. It is presumed that infected patients will require additional days in hospital as a direct consequence of their being infected, and it is the cost of these additional days spent in hospital that are used as a basis for our costing. FREEMAN et al (10, 1979) propose that any patient acquiring an infection in hospital may expect his stay to be extended by an average of 13 days, but SCHECKLER (11, 1980) suggests that as few as 3 additional days would be spent in hospital. For our purposes of determining the MINIMUM additional cost of hospital-acquired infection, we shall use the lower figure of 3 days to yield the following summary of additional costs:-

Number of patients as taken from 1977/78 N.H.S. statistics

Total number of patients treated	3,970,397
Number of occupied beds	109,451

Average length of stay per patient	10.6 days
Number of patients with a hospital-acquired infection (using a presumed ** infection rate of 5%)	198,520

** These figures are based on a MINIMAL infection rate of 5% which is derived from previous CONTINUOUS surveys, whilst the current rate from CROSS-SECTIONAL surveys is around 9.2%.

The infections can be broken down as follows:-

Urinary tract infections (2.0%)	79,408
Wound infections (1.5%)	59,556
Respiratory tract infections (1.0%)	39,704
Other infections (0.5%)	19,852
	<hr/>
Total	198,520

Now, using the 5% infection rate involving the 198,520 patients receiving 3 extra days of in-patient treatment at £54.89p per day, we find that the cost is an additional £32,690,288 which is broken down as follows:-

Urinary tract infections	£13,076,115
Wound infections	9,807,086
Respiratory tract infections	6,538,058
Other infections	3,269,029
	<hr/>
Total	£32,690,288

It must, however, be remembered that the above figures only apply to acute hospital beds, which do, in fact, have the highest rate (with respect to hospital-acquired infections), yet only represent some 36% of total hospital beds. Bearing in mind (all infection rates, number of additional days spent in hospital, and the total cost of the additional time spent in hospital by infected patients - have all been set at the lowest levels) that the figures shown for cost, represent only one single item of additional cost, then it is apparent that there is great scope for utilising resources in a more cost effective manner.

1.3 Breakdown of additional costs of infection

i) Direct costs to N.H.S. -

Time - cost for each additional day spent in hospital.

Treatment - additional use of antibiotics, surgery and dressings.

Facilities - use of single rooms or other isolation facilities.

Services - use and/or additional use of medical, nursing, microbiological, Central Sterile Supplies Department (C.S.S.D.) and infection control services.

ii) Costs to patient or family -

Reduced activity - loss of income or reduced productivity.

Mental trauma - cost of visiting.

Pain, death - loss of service to family.

iii) Indirect cost to government -

Loss of taxable income.

Cost of sickness and/or supplementary benefits.

Cost of home help, community nursing services, etc.

iv) Cost of prevention (for consumables) -

Prophylactic antibiotics.

Antiseptics and disinfectants.

Dressings for clean wounds.

Disposable, re-sterilized or disinfected items.

v) Cost of prevention (capital and maintenance)

Autoclaves and other equipment for supplying heat.

Sterilization and disinfection.

Laundrying - uniforms and protective clothing.

Equipment decontamination, C.S.S.D. and Theatre Sterile Supplies
Department (T.S.S.D.).

Isolation - wards, cubicles, etc.

Ventilation - theatre, Intensive Therapy Unit (I.T.U.),
leukaemic units, etc.

Domestic service costs.

Laboratory costs - hoods, cabinets, etc.

Occupational health service, immunisation, records.

Infection control services.

NOTING: that prevention of infection may not be the only reason for providing the above equipment and services.

If one postulates that each infected patient requires only a single course of antibiotic treatment (even of the least expensive kind - see appendix A), it could well cost an additional £198,520 (based on prescription charges of £1 per item in January 1981). It is estimated that at least 10% of all infected patients are expected to be in receipt of SICKNESS or OTHER benefits which constitute direct costs to the Exchequer. Many other factors, including the use of community services after discharge, loss of taxable income, etc., could well be expected to put more direct costs in excess of £100,000,000 and could well be many times that figure.

Whilst the total cost of measures used to prevent infection is extremely difficult to calculate, it is almost certain that if all the many measures were costed, many would be found to be extremely expensive and possibly irrelevant, AYLIFFE and COLLINS (12, 1982); COLLINS (extensive personal communication).

1.4 Quantitative measurement of risks to patients

It has long been known that various factors have a significant influence on infection risk, but it has always been extremely difficult to

quantify that risk to the degree that it can be used as a realistic basis for the allocation of resources, particularly when we are unaware of the proportions by which each individual or combination of factors contributes to infection risks. Previous attempts to solve this problem have been incomplete, difficult to understand and virtually impossible to apply by medical and nursing staff.

Now, any method which enables relevant factors to be identified or RISK to be quantified, should ensure that a more logical and cost effective approach is made in respect of selecting priorities for allocating the scarce resources of finance, trained staff, specialist equipment, and facilities in order to utilise maximum effectiveness.

The direct costs of controlling and monitoring infection risks are largely confined to the cost of infection control nurses - since all other staff are primarily employed in some other capacity.

It would appear, from a recent joint survey by the Public Health Laboratory Service (P.H.L.S.) and N.H.S., that the number of Infection Control Nurses (I.C.N.'s) is equal to 161 full-time equivalents. Even if all these specialist nurses were paid the maximum salary for a nursing officer (approximately £7,000 per annum), then the total cost each year is only around £1,127,000. This is a very small amount of money when it is considered that one of the main tasks for an I.C.N. is to identify priorities at ward level, to evaluate, and to teach cost effective measures. Although research back-up is required, it is thought that this function could well reduce the present amount of money which is wasted on ineffective measures intended to reduce the risk of cross-infection within a hospital environment.

It can, of course, be argued that when costs are analysed in terms of day-to-day requirements, unrealistic overall costs will result simply because occupancy of the available beds is high. Despite this fact, potentially preventable hospital-acquired infections will undoubtedly lead to extended waiting lists, and the longer that people remain on waiting lists for treatment, and the longer they remain drawing on national resources, rather than contributing.

Some thorough cost evaluation of hospital-acquired infection is long overdue, and moreover, it seems likely that we are making an uneconomically low investment in its prevention.

Now a major contribution towards reducing hospital-acquired infections would be made possible if RISKS to patients could be quantified. It is with this aim borne in mind, that we turn to statistical regression modelling as a key tool for identifying, monitoring and indeed predicting infection risks within the patient population. However, this approach has to overcome a massive initial problem, since every patient is unique, and can be placed in many different surroundings with differing ward practices and indeed different environmental facilities - but with the advent of high-speed electronic computers, the previously impossible task of collating large complex data sets has been brought within manageable proportions.

Care should, however, be taken when using the regression models, with respect to automatically assuming that it is possible to predict one variable from prior knowledge of the other related variable parameters. In some cases, the resultant prediction would be quite

valid, whilst in most other situations the relationship between correlation and prediction will lead to fundamental errors in reasoning. Consider the case where just two parameters are involved, here we would generally (and wrongly) assume that because there is a relationship between the two variable parameters, that a change in one of the variables would automatically cause a change in the second. This phenomenon is particularly apparent when one variable precedes the other in time, i.e. there is a temporal relationship. Serious consideration should be given to the fact that the variables may not, in fact, be directly connected, but instead they may vary by virtue of a common LINK in the form of one (or more) additional variables.

To summarise, we may postulate that any analysis of correlation between variables is indeed a necessary, but not a sufficient, condition to establish that any relationship exists between those variables concerned, i.e. studies of correlation alone do not really allow any valid predictions to be made, with respect to that mechanism causing variations in any of the parameters.

LINK factors are often very difficult to isolate because they are usually hidden within a complex web of secondary and higher order interactions between two or more of the other variables.

To achieve the objectives set out prior to the development for each of the respective regression models (which were updated and expanded during the course of the exercise), however, we do not actually need to isolate or indeed identify any of the LINK factors, provided we are sure that all of the important ones are contained somewhere within the

respective models for monitoring either 'wound infection rates' or 'nasal colonisation rates' in the patient population.

1.5 Mathematical modelling of infection

A model is a mathematical representation of a particular 'real life' problem. The models are derived by a process of eliminating those variable parameters which prove to have no significant influence on either nasal colonisation or wound infection rates, respectively.

Use of mathematical models is often second nature to any person who has been trained in any scientific discipline requiring a high degree of numerate skill, but there is still a definite need to clarify exactly what a mathematical model is, together with a detailed explanation of its use and justification thereof. It must also be pointed out that a process of model building actually does exist, because this may obscure the fact that assumptions DO have to be made when one tries to mathematically simulate any real life situation. Modelling is not a precise subject and there is a definite need to maintain the scope for less definable creative skills. More often than not, such models are the only satisfactory means of solving a particular problem, despite the fact that they can be expensive to construct, but hopefully not off-putting, to the personnel that they were designed to aid.

The two models produced are derived from survey data collected from 41 hospitals in the West Midlands Region over a five-year period from 1967-72. Three-hundred-and-seventy-five wards were visited

over the five years, some on more than one occasion. The patient data collected includes microbiology from some 10,173 nasal swabs and 2,980 wounds, whilst survey information relating to ward structures and facilities, practices I, and practices II, was recorded on each visit.

The resultant models produced were constructed (independently of each other) by comparing ACTUAL wound infection and nasal colonisation rates for each individual factor with the respective EXPECTED rates derived from calculated probability distributions. These expected values take into account the various patient and ward parameters which have been found to significantly influence either rates of wound infection or nasal colonisation, and hence will change as a progressive series of modifications and refinements are made to each of the models. Now, provided that variations within any factor are considered to be both medically (or microbiologically) relevant AND statistically significant (i.e. there is a sufficient difference between OBSERVED and EXPECTED values, which are then subjected to 'goodness of fit' tests), then each of the factors conforming to BOTH of these requirements are carried forward for further consideration, as being a contributory element of that stepwise regression model which is specifically concerned with either 'patient nasal colonisation rates' or 'patient wound infection rates'.

Stepwise regression techniques were then used to give a numerical value for the effect of selected combinations of significant factors, which together form models for predicting 'nasal colonisation rates' and 'wound infection rates' for any patient population, within a hospital environment.

Of the two models produced, one uses the rate of nasal acquisition for tetracycline-resistant Staph. aureus, whilst the second model uses wound infection rates to evaluate the susceptibility of patients to post-operative wound infection.

On completion of all refinements to these two models, it is expected that they will be used to assist in achieving the following objectives:-

1. To make more valid comparisons between different locations or the same location at different times, whilst correcting for any changes with respect to the susceptibility of patient populations.
2. To differentiate between preventable and non-preventable infections associated with the various patient and ward parameters, which may each in turn be changed in order to minimise the risk of cross-infection.

CHAPTER 2

LITERATURE SURVEY

The literature survey can be divided into five categories to illustrate:-

- .. changing characteristics of Staphylococcus aureus, together with its gradual decline and replacement by Gram-negative bacilli.
- .. influence that patient parameters have, with respect to the nasal acquisition of Staph. aureus or susceptibility to wound infection.
- .. effect that variable ward parameters and practices may have on reducing the risks of cross-infection within the ward.
- .. vehicles by which pathogens are transmitted to infect a patient's wound.
- .. need for effective reporting of infections in order to establish better preventative measures.

2.1 Relative predominance of Staph. aureus

Penicillin-resistant strains of coagulase-positive haemolytic Staph. aureus recovered from hospitalised patients have increased since the introduction of penicillin in 1941 (13; 14; 15; 16). HOWE (17, 1954) suggests that resistance to penicillin, or other antibiotics including tetracycline, is roughly proportional to its use in a given area.

Between the years 1956-1959, it was noted that phage type 80/81 was the predominant strain of Staph. aureus responsible for nosocomial infections, BENNETT et al (18, 1959). At that time, it was reputed to be no ordinary staphylococcus, but endowed with an unusual ability to incite disease in man, and even several years later, COHEN et al (19, 1964) found that some 70% of hospital-acquired pathogenic staphylococci resulted in wound infections, with the very young, aged and debilitated patients being the most susceptible groups to serious staphylococcal infections.

Not only is the nose an important source of dissemination of Staph. aureus to the rest of the body and of suppurative disease, particularly in those patients with open wounds, EHRENKRANZ (20, 1964), but it is also thought to be a convenient and accurate index of the general carrier-state for coagulase-positive staphylococci, KNIGHT et al (21, 1958). LOEWENTHAL (22, 1962) found that nasal staphylococcal carriers were subject to a risk of wound infection which was numerically twice that for equivalent patients who were not colonised.

WILLIAMS et al (23, 1959) found that patients admitted to hospital as carriers of Staph. aureus were less liable to acquire 'hospital' strains than patients admitted as 'non-carriers', then again, three years later, WILLIAMS et al (24, 1962) reported that patients acquiring staphylococci in the nose during hospitalisation were five times more likely to suffer staphylococcal wound sepsis than patients who never acquired any staphylococci, whilst FARRER and MacLEOD (25, 1960), found that relative freedom from staphylococcal infection prior to their stay in hospital, did not reflect any state of immunity.

Patients whose wounds do become infected in hospital may expect their hospitalisation to be extended by 37.4 days for staphylococcal infections, and 25.2 days for those infections caused by Gram-negative bacilli, THORBURN et al (26, 1968). Clearly the need was not only recognised to control staphylococcal infection, but also to gain a better understanding of its behaviour in order that highly susceptible patients might be given greater protection. From 1964 to 1968, the isolation of Staph. aureus dropped by over 50% in hospital-acquired infections, BARRETT et al (27, 1968), highlighting a major breakthrough in the control of cross-infection by this particularly troublesome Gram-positive cocci. Various reports over the years have indicated the relative predominance of staphylococcal and Gram-negative infections. The PUBLIC HEALTH LABORATORY SERVICE (28, 1960) found 27% of all patients to be carriers of Staph. aureus, but four years later, the number of staphylococcal infections began to drop and a seasonal variation became apparent, THORNTON et al (29, 1964). By 1967, some 64.5% of all hospital-acquired infections were due to Gram-negative bacilli. Patients entering the hospital without an infection were found to have high counts of community-acquired Gram-positive organisms and very low counts of Gram-negative organisms, whereas, if a patient was infected on admission to the hospital, then counts of Gram-positive organisms were found to be low, whilst counts of hospital-acquired Gram-negative bacilli were found to be very much higher, MacNAMARA et al (30, 1967). In the early 1970's ADLER et al (31, 1971) cited Klebsiella pneumoniae as being the Gram-negative bacilli most frequently isolated during the course of their survey, whilst MOODY and BURKE (32, 1972) reported that Gram-negative bacilli still accounted for over 60% of all hospital-acquired

infections. Towards the end of the decade, AYLIFFE et al (33, 1979) reported that the prevalence of patients with multi-resistant strains of Staph. aureus in their noses still showed a progressive decline.

2.2 Age of patient

Basically, there are just two differing views held by researchers for the influence of a patient's age on his natural susceptibility. Increase in a patient's age results in a lowering of the body's natural resistance to infections, and so the patient's susceptibility to post-operative wound infection or nasal colonisation is increased, particularly in the elderly (24; 25; 28; 34; 35; 36; 37; 38). The opposite view is taken by others (4; 19; 39; 40; 41) who suggest that a patient's susceptibility to wound infection is statistically independent of age, even though SCHRECK and HOPPS (39, 1960) indicate a localised peak between the ages of 51 - 60 years, whilst STEINHAUER et al (41, 1967) highlight that the 10 - 19 years of age group appeared to be relatively immune to most infections.

2.3 Sex of patient

Two views again prevail, (25 and 42) suggest that males are subject to significantly more risks than females, whilst (19; 40; 41; 43) hold the view that from their studies, there are no detectable differences between the results derived in respect of the susceptibility to either nasal colonisation or wound infection for either sex of patient.

2.4 Pre-operative hospitalisation

Extensive periods of pre-operative hospitalisation may well be essential for diagnostic purposes, but during this time, the patient becomes increasingly contaminated by hospital bacteria, CRUSE (4, 1970). The patient may be host to organisms to which he has not developed a natural immunity, or perhaps to others which may be particularly resistant to antibiotics, but in either case, one mechanism by which the patient may develop a subsequent wound infection has been established, and this route will be further strengthened with every additional day of pre-operative stay in hospital.

ALTEMEIER (44, 1966) found that post-operative wound infection rates increased 4-fold if the duration of pre-operative stay exceeded 14 days. Other research has agreed that the longer a patient stays in hospital before an operation, the higher the probability of being host to a subsequent wound infection, because of a decrease in natural resistance (28; 34; 37; 45; 46).

2.5 Effect of prophylactic antibiotics

THOBURN et al (26, 1968) expressed the hope that antimicrobial therapy would eliminate severe diseases caused through infection, but problems with staphylococci developing a resistance to antibiotics were unfortunately not foreseen as being associated with indiscriminate use of prophylactic antibiotics, MINCHEW and CLUFF (36, 1961). Widespread use of antibiotics tends to eliminate susceptible strains of staphylococci and replaces them with resistant organisms derived

from other patients or from members of staff, but unfortunately, the patient is left as host to a reservoir of uncontrolled multi-resistant strains (17; 21; 45; 47; 48), so the tendency to use chemotherapy as a partial substitute for meticulous sterile techniques, must be avoided.

Fortunately, in recent years, the number of multi-resistant strains of Staph. aureus has reduced dramatically in this country so that they no longer represent a major threat to patients, AYLIFFE et al (33, 1979). COHEN et al (19, 1964) suggests that the use of prophylactic antibiotics does not prevent wound infection, but merely delays its appearance by an average of 2.0 days. Approximately one-third of all patients receive some form of antibiotics (9; 26; 49; 50; 51; 52), despite the hypothesis that the use of antibiotics prophylactically might cause more hospital-acquired infection than it prevents. KNIGHT and colleagues (47, 1956) were unable to find any relationship between carrier rates and the intensity with which antimicrobial drugs were used. More than a decade later, SCHECKLER and BENNETT (49, 1970) were still unable to find any link between nosocomial infection and the extent to which antibiotics were used, but HINTON and ORR (53, 1957) found that any increase or decrease in the rate of recovery of staphylococci resistant to a particular antibiotic, was directly proportional to the relative use of that antibiotic.

Although ADLER et al (31, 1971) reported that the rate of post-operative wound infections was reduced when antimicrobial agents were given to patients, FEINGOLD (54, 1970) indicated that existing preventative measures are often inadequate and antibiotic therapy is

frequently suboptimal and may get worse if more strains of multi-resistant organisms emerge in the future. Inappropriate use of antimicrobials must, therefore, be discouraged, DIXON (55, 1975).

A number of methods for controlling antibiotic-resistant staphylococci (including periodic withdrawal of certain drugs from use in hospitals to preserve their anti-staphylococcal effect) have yielded some measure of temporary success, but no clear solution has yet been advanced to the problem of chemotherapeutic management of serious infections caused by ANY antibiotic-resistant organisms, except by careful individualisation with intensive bacteriological control, FINLAND and JONES (56, 1956), although prophylaxis in the short term is now an established and effective method. Research aimed at strengthening host defences and protecting patients from pathogens must, therefore, be pursued together with better surveillance, control and more vigorous measures to minimise the risk of hospital-acquired infections.

2.6 Pre-operative wound preparation

Skin, in health, is heavily colonised with Gram-positive cocci and diphtheroid species, but these are uncommon causes of wound infection. In exceptional circumstances, however, if traumatised or diseased, the skin can become colonised with almost any common bacteria. ROBERTSON (57, 1958) found that patients having positive skin cultures prior to operation were associated with a post-operative wound infection rate which was five times that for comparable patients with negative cultures. Transient contamination with microbes will also occur if

the skin comes into contact with any contaminated sources such as fomites, but normally, these organisms will not survive on the skin for more than a few hours. CRUSE and FOORD (34, 1973), in their investigations, found that pre-operative preparation of the pre-operative site reduced the risk of wound infection, whilst shaving of the operation site had the opposite effect, and these results were confirmed by further study from CRUSE (58, 1975).

2.7 Length of incision and duration of operation

Longer operation durations are associated with an increased risk of infection (19; 34; 35; 45) and when combined with larger incisions, it was found that this constituted additional risks. CRUSE (4, 1970) suggests that the reasons for this, being that bacterial contamination of an incision increases with time, because cells are increasingly damaged by exposure to air as well as trauma from retractors, or because a patient's general resistance is very much lowered as a result of the extensive blood loss and shock which may be associated with longer operative procedures. ALTEMEIER and CULBERTSON (59, 1965) add a further refinement to postulate that wound infection is the unfavourable result of the equation.

$$\frac{\text{Dose of bacteria} \times \text{Virulence}}{\text{Resistance of patient}}$$

However, conflicting evidence came from DINEEN (40, 1961) who thought that the duration of operation might be considered important in the development of post-operative wound infection, but showed from his extensive study that this was not, in fact, the case.

2.8 Air conditioning and ventilation

SHOOTER et al (60, 1956) looked at the ventilation in the ward and theatre, to find that positive pressure ventilation, combined with avoidance of zones of still air over the operating table, appeared to lower the risk of wound infection. LOWBURY (61, 1954) in an excellent study on filtered air-conditioning and its effect on patients' burns, found that during the removal of burns dressing, counts of bacteria increased - but were reduced rapidly when the air-conditioning system was in operation. However, when the air-conditioning system was switched off, bacterial counts did not drop and were still present in the air when the next patient entered the dressing station for removal of dressings. Although the only organisms monitored were Pseudomonas aeruginosa, their removal from the air is representative of most of the organisms which cause wound infections, including Staph. aureus, which colonise the noses of some patients. BLOWERS (62, 1961) suggests that plenum ventilation with a high exchange rate, would reduce the number of airborne organisms, but highlights the prohibitive cost of installation in the hospital ward. Nevertheless, the role of airborne infection in theatres remains uncertain.

2.9 Drained wounds

In a report from the MEDICAL RESEARCH COUNCIL (37, 1968) it was emphasised that a drained wound carries a much higher risk of becoming infected than does a closed wound, which agreed with the views of many others (28; 34; 42; 63; 64). The report went on to recommend that, "Drainage should, therefore, only be used when there are

definite indications for it". Whilst commenting on the M.R.C. report DAVIDSON et al (35, 1971), highlighted the difficulties associated with determining just how long drainage should continue. Whilst admitting to lack of factual evidence for such a claim, they went on to state that the longer the period of time drainage was maintained, the greater the likelihood of infection alongside the tube, or indeed up the lumen. CRUSE (58, 1975) pointed out that if drains had to be used, then a system of closed wound suction drainage would minimise any additional risk of infection. On the other hand, in a somewhat controversial report, COHEN et al (19, 1964) maintained that there is no increased risk of infection from post-operative drains.

2.10 Special risks

There is no argument that a ward should be kept clean, but little agreement exists on how often and by what methods each part should be cleaned, WILLIAMS et al (65, 1966). Similarly, there is much disagreement within the medical profession as to whether or not certain 'special risk' factors have any significant influence on the rates of nasal colonisation and wound infection within the patient population. Diabetic patients admitted to hospital without any infection were at no greater risk of developing nosocomial infection than were comparable non-diabetic patients (19; 39). The opposite view, however, is taken by others (4; 25; 34; 44; 45; 46) who all suggest that the risk to these patients is substantially increased.

Obesity and malnutrition are other medical conditions on which there are slightly differing viewpoints. The MEDICAL RESEARCH COUNCIL

(37, 1968) published a report which included reference to the fact that obese patients were subject to an increased infection risk, whilst others (34; 44; 46) added malnutrition as a further factor which increased the patient's potential susceptibility to infection.

CRUSE and FOORD (34, 1973) found that the use of steroid therapy did not increase the risk of infection, and COHEN et al (19, 1964) added cancer and uraemia as further factors deemed not to have any detrimental effects with respect to wound infection. However, in his extensive studies, ALTEMEIER (44, 1966) found that there was a significant increase in risk, and went on to suggest that both cancer chemotherapy and the prolonged administration of immunosuppressive drugs, together with the use of extensive irradiation, may reduce the count of circulating leucocyte to such a critical level that the patient may become a drastically altered host, with very little or no resistance to nosocomial infections. With these problems borne in mind, we are compelled, as was EICKHOFF (66, 1975), to ask the question as to whether or not immunosuppressive techniques can be targeted more specifically in such a way that the patient's own defence mechanisms are left intact, without reducing the therapeutic value of this very useful method of treatment. ALTEMEIER (46, 1970) further highlights the fact that bacteria have been repeatedly shown to be opportunists which are quick to take advantage of our mistakes, inadequacies and lack of knowledge.

2.11 Transmission of infections to wounds

According to MATSEN (67, 1973) infections occurring in the hospital, arise essentially from just two sources; they are either endogenous

where they arise from flora which are resident on the patient (and under normal circumstances do not have any detrimental effect on the patient) or, they may be exogenous which means that they are transmitted from external sources to the patient. The common sources of infection being associated with:-

i) EXOGENOUS TRANSMISSION

- . Urinary catheters and intravenous devices
- . Contaminated equipment
- . Infected patient secretions or excretions
- . Contaminated solutions
- . Hand transmission

ii) ENDOGENOUS TRANSMISSION

- . Self infection

2.11.1 Urinary catheters and intravenous devices

Risks due to urinary tract infections and their causes are well documented, but unfortunately, recommendations are not always adhered to! These problems are particularly evident in high-risk debilitated and aged patients together with those patients who are receiving steroid or immunosuppressive therapy (6; 45; 66; 68). Suggestions were made by MATSEN (67, 1973) and WILLIAMS (69, 1970) that urinary tract infections were the most frequent type that were acquired in

hospital, with instrumentation being cited as the most common predisposing factor. In 95% or more of these cases, patients developing significant bacteria within 4 days of insertion of an indwelling urinary catheter, unless specific measures are taken to prevent infection. A more recent study by MEERS et al (70, 1981) reported that the prevalence of wound infections was numerically equal to three-fifths the number of recorded urinary infections.

2.11.2 Contaminated equipment

EICKHOFF (66, 1975) and SCHAFFNER (71, 1976) question the contamination that is sometimes found in supposedly sterile products and equipment which are often supplied commercially from outside the hospital, but this is a rare cause of infection.

2.11.3 Infected patient secretions or excretions

The human body is host to a vast range of microbes which may be present in very large numbers. For example - faeces may contain in excess of 100,000,000 per gram. The exact type and numbers required to cause infection in any individual site will depend on the state of health of the host and the external conditions prevailing at that time.

Urine in the bladder is normally sterile, but like all human secretions or excretions, will become contaminated from the skin when voided. It will be further contaminated when a patient is suffering

from an infection of the urinary tract where the range of organisms are broadly similar to those causing wound infections.

Organisms contained in normal skin secretions are not usually pathogenic, but secretions from wounds may contain large numbers of pathogens.

The large intestine is packed with an extremely wide range of organisms, all of which are potentially capable of causing wound infection, and this range may be further expanded during enteric infection.

The oral cavity may also be colonised with Staphylococcal and Streptococcal species and in certain cases of respiratory tract infection, both nasal and oral secretions can contain a wide range of pathogens, including Gram-positive cocci, Gram-negative bacilli and microbes.

2.11.4 Contaminated solutions

At the time when the patient undergoes an operation a MEDICAL RESEARCH COUNCIL report (37, 1968) reveals, that either transient or resident organisms on the skin may provide a reservoir of potential pathogens which may infect the patient's wound. In preparing the operation site, the goal must be to remove the maximum number of pathogens without causing any damage to the skin or introducing any new pathogens from disinfectant solutions which may not be entirely sterile or indeed from shaving brushes which may have been previously contaminated, AYLIFFE et al (72, 1965).

2.11.5 Hand transmission

WILLIAMS (69, 1970) emphasises the effectiveness of non-touch techniques, but when contact between nurses' hands and the patient is unavoidable, we must ask how clean are the nurses' hands? The importance of handwashing as a method of preventing the spread of infection is well known, STREETER (38, 1967) suggesting that to halt the transfer of organisms from one person to another, no single measure is quite so effective as frequent handwashing with hand disinfectant, using 70% alcohol generally being found to be effective, AYLIFFE et al (73, 1975), but on occasions large numbers of organisms were found to survive this technique. CASEWELL and PHILLIPS (74, 1977) reported that handwashing with chlorhexidine hand cleanser reliably gave reductions in hand counts for Klebsiella spp. of 98% - 100%.

In an experiment to evaluate the handwashing techniques of various grades of staff, TAYLOR (75; 76, 1978) found that no particular group of nurses (ranging from auxiliary grade to the highly qualified state registered nurse) washed their hands well, and that the time taken was often too brief. It should, therefore, be borne in mind that no amount of sterile supplies or environmental disinfection can protect the patient from cross-infection by ward and theatre staff with contaminated hands.

2.11.6 Self-infection

A detailed study of wound infections by LOEWENTHAL (22, 1962) showed that 'spray-on' plastic dressings reduced the rate of endogenous or

'self-infection' in the wards. ROUNTREE et al (43, 1960) and BASSETT et al (77, 1963) concluded that all evidence pointed to wards as the chief place of infection, with self-infection of the patients playing a secondary role. The authors also agree with LOEWENTHAL that the rate of infection was much lower in wounds sealed with 'norbecutane' or similar products, as opposed to those wounds with drainage tubes or covered with gauze pads. JEFFERY and SKLAROFF (1, 1958) several years earlier, thought that infections generally originated in the operating theatre with only a few infections being considered results of cross-infection in the wards. In the same year, DINEEN and PEARCE (78, 1958) classified all wound infections into just three groups:-

- those due to - . Breaks in aseptic technique
- . Host-parasite relationship
- . Persistent organisms

Additional work from MINCHEW and CLUFF (36, 1961) observed (on average) a 7-day time-lag between a patient's operation and the recognition of wound sepsis, suggesting that many post-operative wounds may become infected on the wards rather than in the operating theatre. However, it cannot be over-emphasised, that great care must be taken when interpreting these results because it is not really conclusive evidence that the infections did not have an incubation period of 7 days, after originating from the operating theatre. MacNAMARA et al (30, 1967) concluded that the modern hospital with its ever-advancing technology, offers many new vehicles by which potential pathogens may be transmitted to the highly susceptible, compromised host.

2.12 Isolation procedures

Infected patients must be effectively isolated in order to reduce/prevent cross-infection, DIXON (55, 1975). Patients should be isolated when they are suffering from disorders making them particularly susceptible to infection, or when being treated, for example, with immunosuppressive drugs, which increase the patient's susceptibility to infection. WILLIAMS and colleagues (24, 1962) optimistically proposed that efficient and effective isolation policies could reduce the risk of cross-infection by as much as 50%. They backed-up this proposal, citing results from their trial, which indicated that after six weeks in hospital, the rates of acquisition of Staph. aureus in an open ward was some 23%. as compared to 11% for patients in an isolation ward, whilst more recently AYLIFFE et al (79, 1971) found the incidence rate for colonisation of wounds with multiple resistant strains of Staph. aureus to be 2.2% in the open ward, as compared with an average of 3.4% in a selection of ventilated single-bed rooms.

Isolating carriers of certain organisms would be a valuable means of protecting other susceptible patients and so preventing the spread of infections, but PARKER et al (48, 1965) pointed out that this could well present many practical difficulties in those hospitals with few purpose-built isolation facilities and large undivided wards. It was further emphasised that isolation cubicles had not been found to have much effect on the acquisition of sensitive strains of staphylococci, but a considerably reduced rate of organisms resistant to two or more antibiotics was discovered.

2.13 Effect of wound infection on the duration of post-operative hospitalisation

Many medical research teams have attempted to estimate the number of additional days that a patient can expect to spend in hospital, as a direct consequence of post-operative wound infection, and estimates have varied from 6.5 days to 17.6 days (10; 11; 19; 22; 28; 34; 58; 80; 81; 82; 83; 84). In a paper by ROUNTREE et al (43, 1960) the authors found that the effective use of available beds in a surgical unit was reduced by approximately 5% which was made up of extended hospital stays ranging from 3 to 35 days, due to the acquisition of a staphylococcal wound infection. Another paper produced by the PUBLIC HEALTH LABORATORY SERVICE (28, 1960) put the excess duration of hospitalisation due to wound sepsis, in England and Wales, at somewhere in the region of 1,000,000 bed days per year (or about 3% of the total bed occupancy for acute hospital beds). The total excess cost was estimated to be about 0.5% of the total cost of the National Health Service or about £3,300,000 at 1960 prices!

2.14 Communication and reporting of infections

EICKHOFF (66, 1975) stresses the need for more effective communication to improve the dissemination and utilisation of knowledge, particularly that concerning antimicrobial agents. DIXON (55, 1975) proposes that education programmes for staff should be modernised to stress the risks associated with cross-infection, and to demonstrate the benefits of continuous surveillance programmes.

The first Infection Control Sister (I.C.S.) was appointed, on an experimental basis, at the Torbay Hospital in 1959, and on many occasions since, has the usefulness of an infection control sister as a key member of the hospital staff, been highlighted in respect of such additional duties as identifying hospital-acquired problems, patient risks, environmental hazards, teaching and so forth (65; 85; 86).

Reviews of infections ought to be carried out in order to distinguish which infections are nosocomial and which are community-acquired, in order that any hospital-acquired infections may be reduced to an absolute minimum in the light of experience gained, EICKHOFF (87, 1978).

In order to establish effective preventative measures, however, the problems of under-reporting in respect of hospital-acquired infections (41; 50; 51; 68; 88; 89; 90; 91) have to be overcome. In one particular case, MULLHOLLAND et al (90, 1974) illustrated the problem with a specific case where physicians at a particular hospital found only 1.3% of the patients had a hospital-acquired infection, whereas an infection control nurse employed at the hospital found 13.2% in the same patients - an incredible ten-fold increase! Suggestions as to why this situation should exist have included - differing definitions of infection, problems arising from publicity, and within the American Hospital Network further problems may arise from legal implications (88; 91; 92). MacPHERSON (91, 1968) proposes that differences in reported infection rates may occur because there is no uniform agreement in respect of what constitutes a hospital-acquired infection, and furthermore, some investigators confined their

studies to look primarily at staphylococcal infections, whilst others looked at a broader spectrum of infections. COLBECK (93, 1962) indicates that one man's wound infection may be another's sterile reaction to suture material, and even when objective criteria are assessed, personal judgements begin to creep into the assessment - for example, temperature elevation, since it is not confined specifically to infection.

Hence, many infection rates reported by various hospitals often do not represent differences in the actual incidence of infection, but instead, may merely reflect differences in the accuracy of respective reporting systems.

WENZEL et al (50, 1976) not only agreed that there is a need for an accurate surveillance system, but they go further by asking the question as to why no systematic survey has been carried out to assess the accuracy and time required for various reporting systems. WILLIAMS (69, 1970) goes further by indicating that many methods of surveillance have been tried in the past, but nobody has actually tried to measure any of the benefits arising as a direct result of surveillance itself. It must surely be less relevant to find a surveillance system which reveals how many patients have infected wounds in a given hospital, than it is to find one that is best for maintaining a high standard of aseptic alert, for example.

2.15 Infection control programmes

Particularly resistant strains of bacteria reside in the modern

hospital today, and consequently hospital-acquired infections are only likely to be controlled if hospital staff make conscientious efforts to identify potential risk factors together with misdirected practices, and to change them through the use of effective infection control programmes, (94; 95). BRADBEER et al (96, 1966) review the introduction of the Infection Control Nurse (I.C.N.) in respect of performing the duties of surveillance, prevention and control, whilst additionally charging her with the responsibility of setting-up and maintaining, the new lines of communication required to deal with the problems of cross-infection. However, despite the immense success of the infection control nurse, she still shares the fate of all workers in the field of preventative medicine - being little appreciated by clinical colleagues, because of the inherent difficulties associated with statistically quantifying the number of infections that may have been prevented as a direct or indirect result of that work performed by the infection control nurse.

WENZEL (94, 1970) looked further into the future, when computers may well open-up new horizons in the evaluation of hospital infections, and ultimately provide a wealth of information which it is hoped will be of great value in improving certain areas of infection control.

2.16 Cost benefits of effective infection control

BENNETT (97, 1978) suggests that preventative programmes related to hospital-acquired infections are only 25% effective, yet still estimates that 370 million dollars are saved each year, in the United States

alone, for the additional costs related to potential nosocomial infections which have been prevented. Wound sepsis is estimated to cost 7,000 dollars per patient by ALTEMEIER (46, 1970), which means that the total cost of post-operative wound-infection throughout America would cost a staggering 9.83 billion dollars, and this figure was calculated at 1967 prices! BARTLETT (98, 1974) estimates the excess cost to be 500,000,000 dollars per year, whilst SCHAFFNER (71, 1976) puts the cost at 1.5 billion dollars annually in America, for just the cost of the bed (but excluding any additional special treatments) for an excess period of hospitalisation averaging out to 7 days.

CHAPTER 3

IMPLICATIONS

3.1 Critique of the literature survey

EICKOFF (99, 1969) suggested that the changing character of nosocomial infections often results from the changing character of medical care. Throughout the literature survey it became increasingly apparent that the absolute rates of infection were a little meaningless in respect of trying to compare one author's results with another's. Vast differences in the methods of reporting, and indeed different definitions of infection, were all too evident. Some techniques involved collecting patient data on a continuous basis, whilst others prefer prevalence surveys, some use general infection rates, whilst others sub-divided into hospital-acquired and community-acquired. Many investigators have quoted overall rates for hospital-acquired infections, but few have been compelled to look at the inherent differences between the patient populations. It is not really valid to compare infection rates for various hospitals where different conditions prevail. For example, the age distribution of patients and an unspecified mix of clean, clean-contaminated, and contaminated wounds (7; 8; 11; 26; 27; 31; 32; 34; 43; 44; 50; 55; 58; 68; 83; 99; 100; 101; 102) will have a significant influence on wound infection rates, as would further sub-divisions into mild, moderate and severe infections.

It follows that overall wound-infection rates are primarily dependent on the degree of contamination associated with different

operations. Therefore, overall wound-infection rates are somewhat meaningless unless the degree of contamination and type of operation are indicated.

It has been suggested by HOWE (17, 1954) and CRUSE (58, 1975) that the clean wound infection rate, which is thought to be the most sensitive indication of surgical technique, should not be allowed to rise beyond 2%.

BRITT et al (101, 1976) made the observation that variations between survey results from different hospitals may well be accounted for by the fact that smaller hospitals tend to be associated with fewer critically ill patients, whilst more complicated procedures (with higher risks) are usually only carried out within larger hospitals. RHAME and SUDDERTH (103, 1981) further suggest that all surveys are biased towards longer-stay patients.

Fortunately, valuable information can be salvaged in respect of the relationship between wound infection and carriers of coagulase-positive staphylococci. ROUNTREE et al (43, 1960) found that 9% of patients who were carriers of Staphylococcus aureus on admission, eventually infected their own wounds. WILLIAMS et al (23, 1959) observed that 7.1% of carriers and 2.0% of non-carriers of staphylococci, eventually developed wound sepsis. These results imply that if we could only prevent the acquisition of coagulase-positive staphylococci in the nose or remove them from existing carriers, then perhaps the incidence of wound sepsis could be reduced. Nevertheless, it must be remembered that currently most infections are caused by Gram-negative bacilli which differs from the situation as it was in 1960.

3.2 General conclusions derived from the literature survey

From the extensive literature survey, it has become apparent that much uncertainty exists in respect of evaluating the extent to which individual factors contribute to the risks of nasal colonisation with resistant staphylococci or post-operative wound infection. Variations in methodology, together with small patient numbers and often unspecified (or restricted) operation categories and wound types, introduce even more confusion.

It is generally accepted that after patients enter the hospital, some become colonised with tetracycline-resistant Staph. aureus, whilst in the same environment other patients' noses remain free from colonisation. The reasons for this apparently random selection of patients who become colonised still remains unknown, and it can only be postulated (but not conclusively proved) that a patient's natural immunological resistance (even though it is not quantifiable) plays a substantial role in determining which patients ultimately become colonised with hospital-acquired antibiotic-resistant Staph. aureus.

From the information cited in the literature survey discussed in Chapter 2, it is impossible to pinpoint specific links between nasal colonisation and wound infection because of the difficulties associated with trying to separate interacting factors. A further complication arises when accurate records are not kept in respect of whether nasal colonisation occurred before, or after, operative procedures were performed. If nasal acquisition could be identified as being present sometime after the patient's operation, then valid relationships become even more difficult to establish.

Although it is difficult at this stage to draw any firm conclusions, it has been established that greater exposure to the ward environment results in an increased risk of the patient becoming colonised with antibiotic-resistant Staph. aureus, subject to differing levels of ward contamination. Colonisation of any patient site with antibiotic-resistant organisms during hospitalisation is undesirable, since colonised sites can form reservoirs of potentially infectious material which may be the subsequent cause of infections, or indeed contribute to the general level of organisms within the environment which are capable of causing infections.

Whilst information on routes, sources and mechanisms leading to colonisation must be potentially of great value, it is also considered possible that the factors influencing colonisation may enable us to gain a greater understanding of patients in respect of their susceptibility to microbial challenge, or to assist in quantifying immunological competence. This aspect has, however, not yet been fully developed, and will need to be carefully researched in the near future.

Although the relative importance of those factors affecting post-operative wound infection has not been adequately resolved, the parameters are a little more clearly defined, with classification of operation, drain type, and age of patient together with duration of pre-operative hospitalisation, varying combinations of immunosuppressive drugs and steroid therapy, all being found to have a significant influence on rates of wound infection within the hospitalised patient population.

Statistically, difficulties became apparent in determining whether more high risk patients were in receipt of antibiotic therapy, and

since it is very difficult to distinguish between cause and effect, further complications arose when a precise determination of the circumstances in which antibiotics were administered, could not be verified retrospectively.

C H A P T E R 4

ASSESSMENT OF WARD PARAMETERS

4.1 The effect that varying ward parameters have on 'nasal colonisation rates' and 'wound infection rates' in a hospital environment

Previous work (Ph.D. thesis by GOONATILAKE), (104, 1978) has considered the effect that different patient parameters have on:-

- a) the NASAL COLONISATION rate of patients, and,
- b) patient WOUND INFECTION rates.

To progress from this piece of work, one needs to consider the effects on both 'nasal colonisation rates' and 'wound infection rates' which can be attributed directly or indirectly to differing:-

- 1) ward STRUCTURES and FACILITIES
- 2) ward PRACTICES I
- 3) ward PRACTICES II

Now to compare variations within any of the above three groups, the technique adopted is to use 'goodness of fit' tests between OBSERVED and EXPECTED frequencies based on the quantity,

$$\chi^2_{\text{calc}} = \sum_{i=1}^k \frac{(O_i - E_i)^2}{E_i}$$

Where χ^2_{calc} is a value of the random variable X^2 , whose sampling distribution is approximated very closely by the chi-square distribution.

The symbols O_i and E_i represent the observed and expected frequencies, respectively, for the i^{th} cell.

If the OBSERVED and EXPECTED frequencies are fairly close together, then the value of χ_{calc}^2 will be small, in which case it will be reasonable to accept to hypothesis under which the EXPECTED frequencies were calculated, i.e. there is a non-significant (N/S) difference between OBSERVED and EXPECTED values. If, however, there is little agreement between the OBSERVED and EXPECTED frequencies, χ_{calc}^2 will be large, and hence the null hypothesis must be rejected, in favour of there being a significant (Sig) difference between what was actually OBSERVED and what might reasonably have been EXPECTED. The limit for acceptance (or rejection) is usually taken to be at the 5% level of significance, which can be found from chi-squared tables at $\chi_{\text{tab}}^2 = \chi_{0.05}^2$.

Application of this technique for our purposes, is best illustrated by considering the following example from differing 'ward structures and facilities':-

Consider the effect of varying 'the AVERAGE DISTANCE between BED CENTRES' on the 'NASAL ACQUISITION RATE of STAPHYLOCOCCUS AUREUS', for the patients in ANY ward.

If we propose the hypothesis, H_0 : that the proportion of colonised patients is INDEPENDENT of the average DISTANCE between bed centres, i.e. it is constant, and test this against the alternative hypothesis, H_1 : that the AVERAGE DISTANCE between bed centres does have a significant effect on the proportion of patients colonised in any ward.

Let, O_i be the observed frequency of colonised patients in group 'i', and, E_i be the expected frequency of colonised patients in group 'i', where E_i is calculated from the following formula:-

$$E_i = \frac{(\text{no. of patients in group 'i'}) \times (\text{total no. of colonised patients})}{(\text{total NUMBER of patients})}$$

Consider the specimen values following, which shows clearly how all the OBSERVED and EXPECTED frequencies are derived using the formulae above, and then used to calculate a value of chi-square, from:-

$$\chi^2_{\text{calc}} = \sum_{i=1}^5 \frac{(\text{Observed, } O_i - \text{Expected, } E_i)^2}{(\text{Expected, } E_i)}$$

average distance	<6'	6'-6'11"	7'-7'11"	8'-8'11"	>9'	totals
total in group	1303	3585	2600	2066	619	10173
OBSERVED number infected (O_i)	128 (9.82%)	390 (10.88%)	186 (7.15%)	141 (6.82%)	40 (6.46%)	885 (8.70%)
EXPECTED number infected (E_i)	113.35	311.88	226.19	179.73	53.85	885
$\frac{(O_i - E_i)^2}{E_i}$	1.89	19.57	7.14	8.35	3.56	40.51

Perform 'goodness of fit' tests

$$\chi^2 = \sum_{i=1}^5 \frac{(O_i - E_i)^2}{E_i} = 40.51$$

$$\chi^2_{0.05} (4 \text{ degrees of freedom}) = 9.488$$

[where degrees of freedom (d.f) = number of groups - 1]

Therefore, since the calculated value, 40.51 is GREATER than the tabulated value of 9.488, we must REJECT the hypothesis H_0 , in favour of the alternative hypothesis H_1 : that the AVERAGE DISTANCE between bed centres does have a significant effect on the proportion of patients colonised by Staph. aureus, in any ward.

The precise 'GROUPINGS' of patient frequencies, for each of the variable parameters considered, can be found in the following appendices -

- . Ward structures and facilities - Appendix B1
- . Ward practices I - Appendix B2
- . Ward practices II - Appendix B3

A SUMMARY of the results produced, in a similar manner to the example just considered, are shown in Tables 1a, 2a, 3a, 4a, 5a and 6a. Here we are actually testing to see if differences between any of the numerous groups contained within the various categories of ward structures, facilities, practices I and practices II, do, in actual fact, have a significant effect on either patients' 'nasal colonisation rates' or patients' 'wound infection rates', within the ward environment.

In the analysis, wound infection rates are derived from calculations based on the groups of patients whose wounds were described as being clinically infected (graded as mild, moderate or severe), but excluding those infections which were doubtful, together with certain categories of operations such as drainage of abscesses (because these were not really considered to constitute post-operative wounds).

The procedure was then repeated using a second set of EXPECTED frequencies. These take into account those patient parameters (from the extensive list shown in the patient's information record - Appendix C1) which were found to have a significant effect on either 'wound infection rates' or 'nasal colonisation rates', and a summary of the results produced when using the second set of expected frequencies, are shown in Tables 1b, 2b, 3b, 4b, 5b and 6b.

The patient parameters found to have a significant effect on 'nasal colonisation' rates (see STEPWISE REGRESSION procedure in Chapter 10 for detailed documentation of the computational method for determining precisely which factors are significant) are listed below:-

- . AGE of patient
- . SEX of patient
- . TOTAL DURATION of stay in hospital
- . ANTIBIOTIC treatment
- . SPECIAL RISK factors (including immunosuppressive drugs, steroids, etc.)

Whilst the patient parameters found to have a significant effect on 'wound infection rates' are:-

- . AGE of patient
- . SEX of patient

- . DURATION of pre-operative stay in hospital
- . CATEGORY of wound
- . TYPE of DRAIN
- . SPECIAL RISK factors (including immunosuppressive drugs, steroids, etc.)

4.2 Action taken - based on the ward data summarised in Tables 1 - 6 (a and b)

All entries which are indicated as being non-significant, can be dispensed with immediately on the grounds that there are no significant variations between any of the different categories contained within each of the groups that represent the many variable ward structures, facilities, practices I, and practices II that have been analysed.

For those variable ward structures, facilities, practices I, and practices II that have been calculated to have significant variations between the different categories contained within each of the respective groups, we need to devote a little more thought to assess the validity and implications surrounding these results and their ultimate use.

It was felt that the primary value of any mathematical models produced, would initially lie in the field of correcting wound infection and nasal colonisation rates for patient parameters (e.g. age, sex, length of stay in hospital, etc.), in order that changes in wound infection or nasal colonisation rates resulting directly (or indirectly) from variations in either the ward environment or procedures may be

detected. In addition, it is envisaged that the models will be used to correct for changes in patient populations and associated ward parameters in order that more valid comparisons may be made between data derived from different sources and under differing local conditions.

Much of the analysis in Tables 1 - 6 (a and b) has really been to assess the effects of many procedural and environmental factors with a view to eliminating them as being irrelevant with respect to having any influence on either wound infection rates or the rates pertaining to colonisation with tetracycline-resistant Staph. aureus, in the patient population. In choosing relevant modelling factors, it must be borne in mind that the final version of any mathematical models must be:-

- . simple
- . easy to use
- . well understood
- . acceptable to the surgeon and microbiologist, whilst not being subject to continual changes, otherwise much resistance would be experienced in trying to persuade staff to use them.

Hence, it was decided that the following criteria would be adopted for considering whether any particular parameter was relevant to a specific model:-

- 1) The factor should, in itself, be significant and should remain so when adjusted for other significant factors.

- 2) The apparent effect of all significant factors must be explainable by some known or postulated mechanism, i.e. its effect must be either medically or microbiologically justifiable.
- 3) The factor, in itself, must have its own significant effect, which must not be due entirely to its relationship with some other significant factors.
- 4) The factor, itself, must have been in use at the time of the survey, must still be in current use, and must be likely to remain so for the foreseeable future. This will eliminate bad practices that have already been changed, or those which are likely to be changed as a direct (or indirect) consequence of the results derived from the analysis contained within this thesis.
- 5) The distribution of wound infection rates or nasal colonisation rates within any particular group must follow some kind of trend rather than being randomly distributed.

If any particular factor conforms to all five of the criteria indicated above, then that factor is forwarded for further analysis by computerised stepwise regression programmes. These ultimately produce mathematical prediction models, which include all the significant and relevant factors for monitoring wound infection and nasal colonisation rates, respectively.

CHAPTER 5

WARD STRUCTURES AND FACILITIES

5.1 Analysis of nasal results summarised in Tables 1a and 1b

Consider the significant results with respect to the 'rate of nasal acquisition of tetracycline-resistant Staphylococcus aureus' as follows:-

Age of ward is considered to give a reasonably accurate reflection of the general age of that hospital to which it belongs (even taking into account new wards which may have been built on to older hospitals), since the architectural features designed into any new ward should incorporate the 'knowledge of the day' that was available in respect of building a hospital ward with a view to minimising CROSS-INFECTION risk, which was thought to be attributed directly or indirectly to particular ward structures or facilities. Over the period of time in question, however, progress was never consistent, as hospital building was very restricted during the war years and little money was spent during the transition period when the National Health Service took over responsibility for hospitals, in 1948. Progress, therefore, tended to be made in short bursts and for this reason there appears to be a somewhat discontinuous relationship between 'age of ward' and 'patient nasal colonisation rates'. Additionally, there is a tendency to utilise older hospitals for geriatric patients, whilst the newer hospital facilities are generally used for the treatment of high risk patients, such as 'intensive care' or 'premature baby units', etc. In the interim period of time between the completion of the survey in 1972 (which spanned a period of 5 years, from which the results of this thesis are derived) and the current time, a catching-up phase

Table 1a

Analysis of variable ward structures and facilities
(with respect to the 'nasal acquisition' rate of resistant Staph. aureus)

VARIABLE WARD STRUCTURE OR FACILITY	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Age of ward	40.59	5	11.07	Sig.
Position of ward	5.23	2	5.99	N/S
Sex of patients in ward	45.10	3	7.815	Sig.
Number of beds - main ward	42.41	6	12.59	Sig.
Height of ward	2.51	2	5.99	N/S
Floor area - main ward	9.74	3	7.815	Sig.
Division of ward	5.53	6	12.59	N/S
Number of occupied beds - main ward	39.28	6	12.59	Sig.
Proportion of beds occupied - main ward	32.28	5	11.07	Sig.
Average distance between bed centres	40.51	4	9.488	Sig.
Average floor area per bed **	16.23	6	12.59	Sig.
Average floor area per (occupied) bed **	27.86	6	12.59	Sig.
Number of bed spaces less than 2 metres	2.40	3	7.815	N/S
Light entering the ward	2.08	2	5.99	N/S
Type of floor - main ward	26.55	6	12.59	Sig.
Condition of floor - main ward	14.97	5	11.07	Sig.
Condition of walls - main ward	21.90	5	11.07	Sig.
Ventilation of wound dressing room	5.63	2	5.99	N/S
Appraisal of sterilizing or preparation room	16.13	4	9.488	Sig.
Appraisal of kitchen	15.69	4	9.488	Sig.
Location of sluice room relative to ward	4.91	2	5.99	N/S
Size of sluice room	6.81	4	9.488	N/S
Type of floor in sluice room	24.93	5	11.07	Sig.
General condition of sluice room	15.79	4	9.488	Sig.
Size and design of sluice room	21.43	3	7.815	Sig.

** these variable parameters have been derived from combining two of the other variables

Table 1b

Analysis of variable ward structures and facilities(with respect to the 'nasal acquisition' rate[†] of resistant Staph. aureus)

VARIABLE WARD STRUCTURE OR FACILITY	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Age of ward	17.89	5	11.07	Sig.
Position of ward	3.08	2	5.99	N/S
Sex of patients in ward	26.29	3	7.815	Sig.
Number of beds - main ward	18.36	6	12.59	Sig.
Height of ward	2.47	2	5.99	N/S
Floor area - main ward	6.25	3	7.815	N/S
Division of ward	5.76	6	12.59	N/S
Number of occupied beds - main ward	19.90	6	12.59	Sig.
Proportion of beds occupied - main ward	15.86	5	11.07	Sig.
Average distance between bed centres	29.51	4	9.488	Sig.
Average floor area per bed **	19.64	6	12.59	Sig.
Average floor area per (occupied) bed **	31.60	6	12.59	Sig.
Number of bed spaces less than 2 metres	13.40	3	7.815	Sig.
Light entering the ward	0.20	2	5.99	N/S
Type of floor - main ward	21.95	6	12.59	Sig.
Condition of floor - main ward	5.18	5	11.07	N/S
Condition of walls - main ward	8.25	5	11.07	N/S
Ventilation of wound dressing room	2.26	2	5.99	N/S
Appraisal of sterilizing or preparation room	8.98	4	9.488	N/S
Appraisal of kitchen	7.54	4	9.488	N/S
Location of sluice room relative to ward	4.41	2	5.99	N/S
Size of sluice room	3.34	4	9.488	N/S
Type of floor in sluice room	20.08	5	11.07	Sig.
General condition of sluice room	6.14	4	9.488	N/S
Size and design of sluice room	13.24	3	7.815	Sig.

** these variable parameters have been derived from combining two of the other variables

[†] modified to account for significant patient parameters

has occurred with respect to many of the 'geriatric wards', which have undergone extensive updating modifications. It is for a combination of these reasons (not only because 'age of ward' is too inconsistently linked with patient nasal colonisation rates, but also because it is interlinked with other significant factors, which are included for assessment in the regression model specifically concerned with modelling 'patient nasal colonisation rates'), that the variable representing 'age of ward' is excluded from any further calculations.

'Sex of patient in ward' appears to have a potentially significant effect on patients' nasal colonisation rates, but is rejected from further consideration as being a relevant part of that regression model (discussed in Chapter 11) which is specifically concerned with modelling 'patient nasal colonisation rates'. The main reason for exclusion being based on the fact that not only were very few mixed wards visited during the survey and information (that may have been useful) from those wards which did contain both male and female patients, was not recorded in respect of the exact 'proportional mix' of the different sexes, but the patient's sex has already been accounted for as a patient parameter.

'Number of beds in ward' and 'floor area of ward' (when considered independently of patient parameters) both appear to have a potentially significant effect on patient nasal colonisation rates. However, when these results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting nasal colonisation rates, only the variation between the 'number of beds in ward' remains significant whilst the differences in the 'floor area

of ward' become non-significant. This result appears to be logical since the 'floor area of ward' alone does not really constitute a cross-infection risk, whereas if it had been combined together with the 'number of beds in ward', this would give us a new factor representing one aspect of 'overcrowding' in the ward. Hence, for this reason they have been combined to give a new variable, 'average floor area per bed'. This also, proves to significantly affect the patient nasal colonisation rate, and when considered together with the other three significant factors (which represent different aspects of potential overcrowding), namely, 'average distance between bed centres', 'proportion of beds occupied - main ward' and 'number of occupied beds' (i.e. number of patients in ward), we have at our disposal FOUR parameters which together are thought to reflect WARD OVERCROWDING. However, these four factors may be reduced to just THREE, since the overcrowding aspect reflected by 'average floor area per bed' is implicitly contained within the other ward parameters representing 'average distance between bed centres', 'proportion of beds occupied - main ward' and 'number of occupied beds'. The conclusion that is drawn as a result of these particular three factors emerging as having a significant effect on nasal colonisation rates, is to confirm the theory that the total number of patients and relative degree of proximity in any single environment, namely the ward, are highly relevant factors with respect to influencing staphylococcal cross-infection risks. For example, if it is assumed that cross-infection with tetracycline-resistant nasal strains occurs via airborne routes, then high infection rates can be expected when there are a large number of patients in the same room, breathing the same air, or if beds are close together, since the concentration of organisms from a single infected patient will decrease, with distance, through dilution.

** Noting that the 'average floor area per (occupied) bed' has been omitted because we do not want to include the 'floor area of ward' twice over, since this would have a cumulative 'doubling-up' effect on that part of the increase in nasal colonisation rates, which can be attributed solely to variations in the 'floor area of ward'.

'Number of bed spaces less than 2 metres' appears to have a potentially significant effect on patients' nasal colonisation rates, but is rejected from further consideration as being part of the appropriate regression model, because this particular parameter is incorporated within the variable representing 'average distance between bed centres'.

'Type of floor - main ward' is most certainly linked to 'age of ward', because the type of flooring used in any ward has always been influenced by the development of building methods, costs, and availability of materials. Wooden block floors and terrazzo were popular in older buildings, but became too expensive, or were unavailable, during war periods. As time progressed, lino was replaced by vinyl tiles in the newer hospitals, whilst the newest ward floors make extensive use of welded sheet vinyl. Previous work carried out by the Hospital Infection Research Laboratory at Dudley Road Hospital has shown that, once settled on the floor, organisms represent only a small risk in respect of re-infecting patients unless they are redistributed by mechanical methods, e.g. sweeping with a broom. It is, however, of some interest to note one of the findings, that contaminated skin scales were more firmly attached to vinyl floors (by electrostatic bonding) than they were to terrazzo floors. It was, however, decided that the variable representing 'type of floor - main ward' appeared

to be so closely associated with other factors (already analysed) and since there was separate evidence that re-distribution of bacteria from the floor was unlikely to be significant, then we can exclude this parameter from further consideration as an independent factor significantly influencing patient nasal colonisation rates.

'Condition of floor-main ward' and 'condition of walls - main ward' (when considered independently of patient parameters), both appear to have a potentially significant effect on patient nasal colonisation rates. However, when these results are adjusted to take into account those patient parameters which are accepted as significantly affecting nasal colonisation rates both the variations in 'condition of floor - main ward' and 'condition of walls - main ward' become non-significant. This result appears to be logical, since both of the factors are probably associated with 'age of ward' and 'number of beds in ward', i.e. geriatric patients, for example, do not require nursing skills that involve a lot of technical expertise, hence, they tend to reside in the oldest wards of the older hospitals (which may well have developed from the 'work houses' of days gone by). It is these wards that are usually given a lower priority with respect to upgrading, as compared with high care areas, such as surgical and other acute wards which usually have upgrading modifications made to accommodate more sophisticated equipment such as electronic monitoring, piped-oxygen, suction, etc. Hence, for these reasons both 'condition of floor - main ward' and 'condition of walls - main ward' are excluded as independent factors involved in any further calculations.

'Appraisal of sterilizing or preparation room' and 'appraisal of kitchen' (when considered independently of patient parameters) both

appear to have a potentially significant effect on patient nasal colonisation rates, but when the results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting nasal colonisation rates, both 'appraisal of sterilizing or preparation room' together with 'appraisal of kitchen' prove to be non-significant. These results are accepted because patients do not enter the sterilizing or preparation room, nor do they have any access to food preparation areas; and food in itself is a very unlikely source of antibiotic-resistant Staph. aureus anyway. Therefore, these variables are excluded from any further consideration as being independent factors having any significant effect on patient colonisation rates, in favour of the alternative proposal that any variations are possibly linked to 'age of ward'.

'Type of floor in sluice room', 'general condition of sluice room', together with 'size and design of sluice room' (when considered independently of patient parameters) all appear to have a potentially significant effect on patient nasal colonisation rates. When these results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting nasal colonisation rates, we reveal some very misleading results. 'General condition of sluice room' proves to be non-significant whilst both 'type of floor in sluice room' along with 'size and design of sluice room' remain as potentially significant factors. The only explanation for this result, being that the latter two factors could well be linked with 'age of ward' and 'number of beds in ward'. However, 'type of floor in sluice room', 'general condition of sluice room', along with 'size and design of sluice room' are all rejected as being factors which are relevant to nasal colonisation, because it should always be

remembered that any contamination in the sluice room area would consist primarily of Gram-negative bacteria (which are transmitted only by 'direct contact'), and would not have any influence on tetracycline-resistant Staph aureus which are Gram-positive bacteria (which are more likely to be transmitted via airborne routes). Furthermore, it should be noted that the sluice room is not designated as a patient area, and so it is very unlikely to have any significant effect on patient staphylococcal cross-infection rates, when patients do not have any access to this particular restricted area. However, it is possible that inadequately cleaned equipment could be stored in the sluice room, and this might have a subsequent influence on cross-infection.

5.2 Analysis of wound results summarised in Tables 2a and 2b

Consider the significant results with respect to the 'rate of wound infections' as follows:-

'Age of ward' is accepted as having a non-significant effect on patient wound infection rates, because surgical wards cannot be considered typical of the entire range of wards. Geriatric and psychiatric patients, for example, infrequently require surgery, and tend to be located in the older wards, and are transferred to specialist surgical wards, only for operative procedures to be performed. Taken together, these have the net effect of making 'age of ward' a redundant factor, so it is rejected from any further consideration as being a relevant part of that regression model (discussed in Chapter 11) which is specifically concerned with modelling 'patient wound infection rates'.

Table 2a

Analysis of variable ward structures and facilities
(with respect to patient 'wound infection' rates)

VARIABLE WARD STRUCTURE OR FACILITY	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Age of ward	4.19	5	11.07	N/S
Position of ward	2.90	2	5.99	N/S
Sex of patients in ward	15.49	3	7.815	Sig.
Number of beds - main ward	18.92	6	12.59	Sig.
Height of ward	0.21	2	5.99	N/S
Floor area - main ward	5.26	3	7.815	N/S
Division of ward	6.44	6	12.59	N/S
Number of occupied beds - main ward	30.15	6	12.59	Sig.
Proportion of beds occupied - main ward	9.63	5	11.07	N/S
Average distance between bed centres	8.08	4	9.488	N/S
Average floor area per bed **	9.96	6	12.59	N/S
Average floor area per (occupied) bed **	8.02	6	12.59	N/S
Number of bed spaces less than 2 metres	7.90	3	7.815	Sig.
Light entering the ward	7.80	2	5.99	Sig.
Type of floor - main ward	22.78	6	12.59	Sig.
Condition of floor - main ward	8.35	5	11.07	N/S
Condition of walls - main ward	4.95	5	11.07	N/S
Ventilation of wound dressing room	13.42	2	5.99	Sig.
Appraisal of sterilizing or preparation room	2.85	4	9.488	N/S
Appraisal of kitchen	5.30	4	9.488	N/S
Location of sluice room relative to ward	10.11	2	5.99	Sig.
Size of sluice room	2.97	4	9.488	N/S
Type of floor in sluice room	14.36	5	11.07	Sig.
General condition of sluice room	0.65	4	9.488	N/S
Size and design of sluice room	1.84	3	7.815	N/S

** these variable parameters have been derived from combining two of the other variables

Table 2b

Analysis of variable ward structures and facilities

(with respect to patient 'wound infection' rates[†])

VARIABLE WARD STRUCTURE OR FACILITY	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Age of ward	3.91	5	11.07	N/S
Position of ward	3.81	2	5.99	N/S
Sex of patients in ward	4.20	3	7.815	N/S
Number of beds - main ward	11.24	6	12.59	N/S
Height of ward	1.16	2	5.99	N/S
Floor area - main ward	3.88	3	7.815	N/S
Division of ward	2.74	6	12.59	N/S
Number of occupied beds - main ward	14.05	6	12.59	Sig.
Proportion of beds occupied - main ward	2.90	5	11.07	N/S
Average distance between bed centres	8.94	4	9.488	N/S
Average floor area per bed **	8.13	6	12.59	N/S
Average floor area per (occupied) bed **	2.92	6	12.59	N/S
Number of bed spaces less than 2 metres	7.05	3	7.815	N/S
Light entering the ward	5.31	2	5.99	N/S
Type of floor - main ward	10.96	6	12.59	N/S
Condition of floor - main ward	9.45	5	11.07	N/S
Condition of walls - main ward	2.29	5	11.07	N/S
Ventilation of wound dressing room	5.58	2	5.99	N/S
Appraisal of sterilizing or preparation room	4.79	4	9.488	N/S
Appraisal of kitchen	3.64	4	9.488	N/S
Location of sluice room relative to ward	2.08	2	5.99	N/S
Size of sluice room	6.56	4	9.488	N/S
Type of floor in sluice room	7.66	5	11.07	N/S
General condition of sluice room	4.60	4	9.488	N/S
Size and design of sluice room	4.52	3	7.815	N/S

** these variable parameters have been derived from combining two of the other variables

† modified to account for significant patient parameters

'Sex of patients in ward' (when considered independently of patient parameters) appears to have a potentially significant effect on patient wound infection rates. However, when the results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting wound infection rates, then any variation between 'sex of patients in ward' becomes non-significant. This result in itself is accepted because not only were very few mixed wards visited during the survey and information (that may have been useful) from those wards which contained both male and female patients, was not recorded in respect of the exact 'proportional mix' of the different sexes, but sex has already been accounted for as a patient parameter.

'Number of beds - main ward' and 'number of bed spaces less than 2 metres' (when considered independently of patient parameters) appear to have a potentially significant effect on patient wound infection rates. When these results are adjusted, however, to take into account those variable patient parameters which are accepted as significantly affecting wound infection rates, then any variations within those factors respectively representing the 'number of beds - main ward' and the 'number of bed spaces less than 2 metres' become non-significant. These results appear to be logical, since the 'number of (occupied) beds - main ward' is a far more relevant factor (with respect to the transmission of infections from one patient's wound to another), because neither the 'number of beds - main ward' nor the 'number of bed spaces less than 2 metres' give any indication of how many beds remain empty in any given ward. Hence, these two aspects of overcrowding are excluded from any further calculations.

'Number of (occupied) beds - main ward' is accepted as having a potentially significant effect on patient wound infection rates, and so is retained for further assessment, in that regression model which is specifically concerned with modelling 'patient wound infection rates', because the 'number of (occupied) beds - main ward' is considered to give some index of a patient's potential exposure to any other patient(s) who may have an infected wound.

'Proportion of beds occupied - main ward', 'average floor area per bed', 'average floor area per (occupied) bed', and 'average distance between bed centres' are all accepted as not having any significant effect on patient wound infection rates, and so are excluded as aspects of overcrowding from any further calculations, because the actual 'number of (occupied) beds - main ward' establishes itself as a considerably more dominant factor, which has an overriding effect on all the other less important recessive factors.

If we review the results for:-

- . 'number of beds - main ward'
- . 'number of bed spaces less than 2 metres'
- . 'proportion of beds occupied - main ward'
- . 'average floor area per bed'
- . 'average floor area per (occupied) bed'
- . 'average distance between bed centres'

It can be seen that the above six aspects of overcrowding, which were thought to be directly or indirectly related to wound infection rates, are far less important than had been previously thought before the results were analysed from this extensive survey. It is apparent that the 'number of (occupied) beds - main ward' has a far more significant effect on patient wound infection rates, because it is considered to give an accurate reflection of any patient's potential risk, in respect of being exposed to other patients who may have infected wounds.

'Light entering the ward', 'type of floor - main ward', 'ventilation of wound dressing room', 'location of sluice room relative to ward', and 'type of floor in sluice room' (when considered independently of patient parameters) all appear to have a potentially significant effect on patient wound infection rates. However, when these results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting wound infection rates, then any variation within each of these five parameters becomes non-significant. These results are accepted because all the five parameters are so closely associated with other factors (already analysed) that we can exclude them as independent factors which have any influence on patient wound infection rates.

CHAPTER 6

WARD PRACTICES I

6.1 Analysis of nasal results summarised in Tables 3a and 3b

Consider the significant results with respect to the 'rate of nasal acquisition of tetracycline-resistant Staphylococcus aureus' as follows:-

'Total number of nursing staff', 'number of S.R.N. day staff' and 'number of S.R.N. night staff' (when considered independently of patient parameters), all appear to have a potentially significant effect on patient nasal colonisation rates. However, when these results are adjusted to take into account those patient parameters which are accepted as significantly affecting nasal colonisation rates then the 'total number of nursing staff' and the 'number of S.R.N. night staff' both become non-significant, whilst the 'number of S.R.N. day staff' remains significant. Any variation in the 'total number of nursing staff' is accepted as being non-significant because this factor is clearly related to the 'number of occupied beds - main ward' and 'type of patient' (which should only reflect the degree of nursing care required), whilst having no direct effect on the transmission of airborne Staph. aureus. The 'number of S.R.N. night staff' is accepted as being non-significant because this variable is more probably associated with the speciality of surgery and acute cases, where it is more likely that highly trained S.R.N. staff are available at night, than they would be for, say, a geriatric ward (where S.R.N.'s are not really a necessity when patients

Table 3a

Analysis of variable ward practices I

(with respect to the 'nasal acquisition' rate of resistant Staph. aureus)

VARIABLE WARD PRACTICES I	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Total number of nursing staff	11.91	5	11.07	Sig.
Number of S.R.N. day staff	18.30	1	3.84	Sig.
Number of S.R.N. night staff	5.08	1	3.84	Sig.
Number of S.E.N. day staff	3.81	2	5.99	N/S
Number of S.E.N. night staff	0.59	1	3.84	N/S
Average number of patients per S.R.N.	29.43	6	12.59	Sig.
Average number of patients per S.E.N.	26.76	6	12.59	Sig.
Average number of patients per member of the nursing staff	20.58	2	5.99	Sig.
Floors - method of wet cleaning (routine)	6.99	2	5.99	Sig.
Floors - method of dry cleaning (poor methods) **	2.12	4	9.488	N/S
Floors - vacuum with filter ** versus all others	26.04	1	3.84	Sig.
Floors - frequency of cleaning	1.40	2	5.99	N/S
Complete ward - frequency of cleaning	3.53	2	5.99	N/S
Lower wall - frequency of cleaning	2.52	2	5.99	N/S
Whole wall - frequency of routine cleaning	4.92	2	5.99	N/S
Whole wall - frequency of special cleaning	0.16	1	3.84	N/S

** Preliminary analysis of the results, indicates that vacuum cleaners with filters are far superior to any of the other methods of dry cleaning (which are deemed 'poor methods')

Table 3b

Analysis of variable ward practices I

(with respect to the 'nasal acquisition' rate[†] of resistant Staph. aureus)

VARIABLE WARD PRACTICES I	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Total number of nursing staff	0.07	5	11.07	N/S
Number of S.R.N. day staff	3.97	1	3.84	Sig.
Number of S.R.N. night staff	0.15	1	3.84	N/S
Number of S.E.N. day staff	3.57	2	5.99	N/S
Number of S.E.N. night staff	1.23	1	3.84	N/S
Average number of patients per S.R.N.	13.06	6	12.59	Sig.
Average number of patients per S.E.N.	13.11	6	12.59	Sig.
Average number of patients per member of the nursing staff	4.92	2	5.99	N/S
Floors - method of wet cleaning (routine)	2.12	2	5.99	N/S
Floors - method of dry cleaning (poor methods) **	9.64	4	9.488	Sig.
Floors - vacuum with filter ** versus all others	15.53	1	3.84	Sig.
Floors - frequency of cleaning	0.21	2	5.99	N/S
Complete ward - frequency of cleaning	3.34	2	5.99	N/S
Lower wall - frequency of cleaning	0.41	2	5.99	N/S
Whole wall - frequency of routine cleaning	1.82	2	5.99	N/S
Whole wall - frequency of special cleaning	1.72	1	3.84	N/S

** Preliminary analysis of the results, indicates that vacuum cleaners with filters are far superior to any of the other methods of dry cleaning (which are deemed 'poor methods')

† modified to account for significant patient parameters

are asleep). Hence, it has very little (if any) effect on patient nasal colonisation rates. The 'number of S.R.N. day staff' appears to have a significant effect on patient nasal colonisation rates, but when studied in greater detail, it becomes clear that there is no difference in the functions of S.E.N.'s and S.R.N.'s that can logically affect the nasal acquisition rate of Staph. aureus within the hospital environment. It may, however, be possible that the presence of a larger proportion of qualified staff is an indication of specialist wards which have an unusually high standard of care and a greater knowledge of infection risk, e.g. intensive care units.

Wards with small numbers of patients would also have the same effect of reducing the rates of nasal colonisation by lowering the patients' probability of coming into contact with any other colonised patients. Therefore, the 'number of S.R.N. day staff' together with the 'number of S.R.N. night staff' and the 'total number of nursing staff' are all rejected as being independent parameters having any influence on patient nasal colonisation rates, and so are excluded from further consideration as being a relevant part of that regression model which is specifically concerned with 'patient nasal colonisation rates'.

'Average number of patients per S.R.N.', 'average number of patients per S.E.N.' and 'average number of patients per member of the nursing staff' (when considered independently of patient parameters), all appear to have a potentially significant effect on patient nasal colonisation rates. However, when these results are adjusted to take into account those patient parameters which are accepted as significantly affecting



nasal colonisation rates, then only the differences in the 'average number of patients per S.R.N.' and 'average number of patients per S.E.N.' remain significant. Now, because staffing levels could only possibly have a very indirect influence on patient nasal colonisation rates insofar as higher numbers of staff (which is not dependent on the level of training or degree of nursing skill) would increase the total numbers (of patients and staff) in any given ward environment, each of these variable parameters representing 'average number of patients per S.R.N.', 'average number of patients per S.E.N.', together with 'average number of patients per member of the nursing staff' can be excluded from further consideration as being independent factors having any significant influence on patient nasal colonisation rates.

'Floors - method of dry cleaning (poor methods)', when adjusted to take into account those patient parameters which are accepted as significantly affecting nasal colonisation rates, appears itself to have some influence on the proportion of patients colonised with tetracycline-resistant Staph. aureus. However, more detailed analysis reveals that every type of dry cleaning within the variable representing 'floors - method of dry cleaning (poor methods)', proves to be inferior in comparison with 'vacuum cleaners with filters', hence this parameter is excluded from any further calculations in favour of creating a new variable (whose analysis is shown below) which represents 'floors - vacuum cleaner with filter versus all others'.

'Floors - vacuum cleaner with filter versus all others' proves to have a significant effect on patient nasal colonisation rates. When ward floors are cleaned with a vacuum cleaner which contains a filtration

system, this will actually remove Staph. aureus from the air, whereas all other methods (particularly brooms) have the effect of redistributing Staph. aureus organisms (which may have settled on the floor) back into the air. It is for this reason, that the use of brooms is now forbidden in patient areas and a British Standard specification has been introduced, making filters compulsory with respect to those vacuum cleaners intended for use in hospitals. Since the time when the survey was taken, the methods used for dry cleaning floors have become more standardised and so making any differences in the dry cleaning methods used for 'floors - vacuum cleaners with filters versus all others' a redundant factor. This variable, therefore, can be eliminated from any further consideration as being a relevant part of that regression model which is specifically concerned with modelling 'patient nasal colonisation rates'.

6.2 Analysis of wound results summarised in Tables 4a and 4b

The only factor which appears to have a potentially significant effect on wound infection rates (when considered independently of patient parameters) is 'whole wall - frequency of routine cleaning'. However, when the results are adjusted to take into account those patient parameters which are accepted as significantly affecting wound infection rates, then any variation within the factor representing 'whole wall - frequency of routine cleaning' becomes non-significant. This result is readily accepted because 'whole wall - frequency of routine cleaning' is not connected with any of the likely mechanisms which are known to have an influence on the rates of cross-infection for patients' wounds.

Table 4a

Analysis of variable ward practices I
(with respect to patient 'wound infection' rates)

VARIABLE WARD PRACTICES I	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Total number of nursing staff	9.72	5	11.07	N/S
Number of S.R.N. day staff	3.31	1	3.84	N/S
Number of S.R.N. night staff	0.95	1	3.84	N/S
Number of S.E.N. day staff	1.66	2	5.99	N/S
Number of S.E.N. night staff	0.06	1	3.84	N/S
Average number of patients per S.R.N.	7.23	6	12.59	N/S
Average number of patients per S.E.N.	6.99	6	12.59	N/S
Average number of patients per member of the nursing staff	2.13	2	5.99	N/S
Floors - method of wet cleaning (routine)	2.05	2	5.99	N/S
Floors - method of dry cleaning (poor methods) **	5.44	4	9.488	N/S
Floors - vacuum with filter ** versus all others	2.74	1	3.84	N/S
Floors - frequency of cleaning	3.41	2	5.99	N/S
Complete ward - frequency of cleaning	0.64	2	5.99	N/S
Lower wall - frequency of cleaning	2.30	2	5.99	N/S
Whole wall - frequency of routine cleaning	7.45	2	5.99	Sig.
Whole wall - frequency of special cleaning	0.76	1	3.84	N/S

** Preliminary analysis of the results, indicates that vacuum cleaners with filters are far superior to any of the other methods of dry cleaning (which are deemed 'poor methods')

Table 4b

Analysis of variable ward practices I
(with respect to patient 'wound infection' rates[†])

VARIABLE WARD PRACTICE I	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Total number of nursing staff	0.12	5	11.07	N/S
Number of S.R.N. day staff	0.00	1	3.84	N/S
Number of S.R.N. night staff	0.00	1	3.84	N/S
Number of S.E.N. day staff	0.97	2	5.99	N/S
Number of S.E.N. night staff	0.21	1	3.84	N/S
Average number of patients per S.R.N.	5.60	6	12.59	N/S
Average number of patients per S.E.N.	7.48	6	12.59	N/S
Average number of patients per member of the nursing staff	0.30	2	12.59	N/S
Floors - method of wet cleaning (routine)	2.87	2	5.99	N/S
Floors - method of dry cleaning (poor methods) **	5.54	4	9.488	N/S
Floors - vacuum with filter ** versus all others	2.43	1	3.84	N/S
Floors - frequency of cleaning	3.85	2	5.99	N/S
Complete ward - frequency of cleaning	1.23	2	5.99	N/S
Lower wall - frequency of cleaning	4.83	2	5.99	N/S
Whole wall - frequency of routine cleaning	4.64	2	5.99	N/S
Whole wall - frequency of special cleaning	1.11	1	3.84	N/S

** Preliminary analysis of the results, indicates that vacuum cleaners with filters are far superior to any other methods of dry cleaning (which are deemed 'poor methods')

[†] modified to account for significant patient parameters

All factors contained within the category of variable ward practices I (which were thought to have a potential effect on patient wound infection rates), are, therefore, eliminated from further consideration as being part of that regression model which is specifically concerned with modelling 'patient wound infection rates'.

CHAPTER 7

WARD PRACTICES II

7.1 Analysis of nasal results summarised in Tables 5a and 5b

Consider the significant results with respect to the 'rate of nasal acquisition of tetracycline-resistant Staphylococcus aureus' as follows:-

'Treatment of ward shaving razor' appears to have a potentially significant effect on patient nasal colonisation rates. This result in itself could have been anticipated if it was wound infection rates that were under discussion (because use of 'disposable razors' and 'razors not used' prove to be significantly better than all the other categories for 'treatments of ward shaving razor') but, since there is no direct mechanism by which this factor is likely to have any significant influence on nasal colonisation rates, it is excluded from any further calculations.

'Number of staff in dressing team' and 'dress of "dressing team"' (when considered independently of patient parameters) both appear to have a potentially significant effect on patient nasal colonisation rates. However, when the results are adjusted to take into account those variable patient parameters which are accepted as having a significant effect on nasal colonisation rates, then it is only the differences in 'dress of "dressing team"' that remain significant. A little deeper thought reveals that neither of these parameters can have any relevance in the building of a mathematical model for monitoring nasal colonisation rates, since both the 'number of staff in dressing team' and 'dress of "dressing team"' can only have a very

Table 5a

Analysis of variable ward practices II

(with respect to the 'nasal acquisition' rate of resistant Staph. aureus)

VARIABLE WARD PRACTICE II	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Schedule for pre-operative preparation	0.04	2	5.99	N/S
Method of pre-operative preparation	5.04	2	5.99	N/S
Treatment of ward shaving razor	13.06	5	11.07	Sig.
Location of main wound dressing site	0.96	2	5.99	N/S
Number of staff in dressing team	9.91	2	5.99	Sig.
Dress of 'dressing team'	52.75	4	9.488	Sig.
Are gloves used ?	2.03	2	5.99	N/S
Method of handwashing - general	13.95	2	5.99	Sig.
Method of handwashing - special	3.73	2	5.99	N/S
Method of handwashing - dressings	12.71	2	5.99	Sig.
Use of hand cream in wards	27.00	4	9.488	Sig.
Occasions when scrubbing is used	1.88	2	5.99	N/S
Treatment of nail brushes	19.60	2	5.99	Sig.
Hand cream - container	6.15	1	3.84	Sig.
Treatment of Cheatle's forceps	12.33	3	7.815	Sig.
Type of dressing - clean undrained wounds	10.11	3	7.815	Sig.
Type of dressing - drained wounds	0.29	3	7.815	N/S
Type of dressing - dirty or septic wounds	13.31	3	7.815	Sig.
Cleansing lotion used on clean wounds	9.09	4	9.488	N/S
Cleansing lotion used on dirty or septic wounds	11.53	4	9.488	Sig.
Appraisal of isolation facilities	29.06	2	5.99	Sig.
Isolation of wound infections	28.01	4	9.488	Sig.
Isolation of infections due to <u>Staph. aureus</u>	48.31	4	9.488	Sig.

Table 5b

Analysis of variable ward practices II

(with respect to the 'nasal acquisition' rate[†] of resistant Staph. aureus)

VARIABLE WARD PRACTICES II	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Schedule for pre-operative preparation	5.74	2	5.99	N/S
Method of pre-operative preparation	2.48	2	5.99	N/S
Treatment of ward shaving razor	15.35	5	11.07	Sig.
Location of main wound dressing site	3.64	2	5.99	N/S
Number of staff in dressing team	5.81	2	5.99	N/S
Dress of 'dressing team'	37.30	4	9.488	Sig.
Are gloves used?	2.58	2	5.99	N/S
Method of handwashing - general	5.47	2	5.99	N/S
Method of handwashing - special	2.66	2	5.99	N/S
Method of handwashing - dressings	3.18	2	5.99	N/S
Use of hand cream in wards	15.65	4	9.488	Sig.
Occasions when scrubbing is used	1.87	2	5.99	N/S
Treatment of nail brushes	8.83	2	5.99	Sig.
Hand cream - container	2.95	1	3.84	N/S
Treatment of Cheatle's forceps	10.51	3	7.815	Sig.
Type of dressing - clean undrained wounds	1.68	3	7.815	N/S
Type of dressing - drained wounds	1.01	3	7.815	N/S
Type of dressing - dirty or septic wounds	2.25	3	7.815	N/S
Cleansing lotion used on clean wounds	15.83	4	9.488	Sig.
Cleansing lotion used on dirty or septic wounds	9.15	4	9.488	N/S
Appraisal of isolation facilities	10.13	2	5.99	Sig.
Isolation of wound infections	8.32	4	9.488	N/S
Isolation of infections due to <u>Staph. aureus</u>	22.06	4	9.488	Sig.

[†] modified to account for significant patient parameters

transient effect on each patient because of the very small amount of time where possible contact can occur, and there could only be any possible effect on the small proportion of patients actually having dressed wounds. Inconsistencies become apparent when analysing the 'dress of "dressing team"' because 'no special dress' produces the lowest rates of colonisation whilst higher rates of colonisation result from wearing any form of special dress.

Presuming the source of Staphylococcus aureus was exhaled air, it would have been logical, if for example, the use of masks had reduced incidences of nasal colonisation, but from the results analysed this appeared not to be the case. There is, however, some suggestion that the Staph. aureus causing colonisation are derived from contaminated skin surrounding a colonised nose. Wearing a mask may well cause friction in this area, and so resulting in the dispersal of a greater number of contaminated skin scales. Therefore, the factors representing 'number of staff in dressing team' and 'dress of "dressing team"' are excluded from any further consideration as being relevant parts of that regression model which is specifically concerned with modelling 'patient nasal colonisation rates'.

'Method of handwashing - general' and 'method of handwashing - dressings' (when considered independently of patient parameters) both appear to have a potentially significant effect on patient nasal colonisation rates. However, when the results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting nasal colonisation rates, then any variations that do exist become non-significant. In fact, both 'method of

handwashing - general' and 'method of handwashing - dressings' may be eliminated from any further calculations for the same reasons - namely, that one of the products (hexachlorophane), which was extensively used at the time of the survey, is no longer in use because of its reputed toxicity - instead, chlorhexidine is increasingly used. Therefore, since the results are no longer valid with respect to those products which are currently in use, then the outdated results derived from these two variables are excluded from any further calculations.

'Use of hand cream in wards', 'treatment of nail brushes' and 'hand-cream container' (when considered independently of patient parameters) all appear to have a potentially significant effect on patient nasal colonisation rates. However, when the results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting nasal colonisation rates, only the variations in the 'use of hand cream in wards' and 'treatment of nail brushes' remain significant. There is thought to be no mechanism which can logically link any of the three factors with the transmission of tetracycline-resistant Staph. aureus, and furthermore, each of these parameters is now considered to be bad practice and so their use has been reduced (wherever possible). It is for a combination of these reasons, that the 'use of hand cream in wards', 'treatment of nail brushes' and 'hand-cream container' are all excluded from any other calculations, because these are not considered to be independent factors likely to have any significant influence on the current rates of nasal colonisation.

'Treatment of Cheatles forceps' appears to have a potentially significant effect on patient nasal colonisation rates. However, when the back-

ground behind the results is analysed, it is found that Cheate's forceps are now rarely used because of changing practices. Hence, this factor can be excluded from further consideration as being a relevant part of that regression model which is specially concerned with modelling 'patient nasal colonisation rates'.

'Type of dressing - clean undrained wounds' and 'type of dressing - dirty or septic wounds' (when considered independently of patient parameters), both appear to have a potentially significant effect on patient nasal colonisation rates, but when the results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting nasal colonisation rates, any variations in these two parameters become non-significant. Clearly, the question of dressings would only be relevant for those patients having wounds (which is approximately 31% of all patients from whom nasal swabs were analysed - this number being further subdivided into different types of wound), which has the effect of creating a biased sample for analysis. Add to this the fact that 'no dressing' is more likely to be used for healed wounds, whereas, infected wounds are more likely to be 'dressed' and one becomes aware that different dressings are not given an equivalent challenge because they are used in different situations. The type of dressing used for infected wounds is even less likely to be relevant (with respect to nasal colonisation) because a high proportion of infected wounds will be dressed, and so virtually eliminating the possibility of those patients having staphylococcal wound infections from transferring the organism from a wound to their nose - this being verified by the variable representing both 'type of dressing - clean undrained wounds' and 'type of dressing -

dirty or septic wounds' changing their status from being significant to non-significant, as modifications for patient parameters were taken into consideration. Therefore, it is for a combination of these reasons that we reject the factors representing 'type of dressing - clean undrained wounds' and 'type of dressing - dirty or septic wounds' from any further calculations, since no direct mechanism could be found to indicate that different types of dressing (for any kind of wound) had any influence on nasal colonisation rates.

'Cleansing lotion used on clean wounds' (when considered independently of patient parameters) is observed not to have any significant effect on patient nasal colonisation rates, whilst 'cleansing lotion used on dirty or septic wounds' does appear to significantly affect colonisation rates. However, when the results are adjusted to take into account those patient parameters which are accepted as significantly affecting nasal colonisation rates, the significance status for each of the two variables changes so that any differences in the 'cleansing lotion used on dirty or septic wounds' becomes non-significant whilst those for 'cleansing lotion used on clean wounds' appear to have a significant effect on those survey results derived for the rates of nasal colonisation in patients. A little deeper research reveals that cleansing lotions will not constitute a relevant part of that regression model which is specifically concerned with modelling 'patient nasal colonisation rates', because the results will only be applicable to that small group of patients who actually have a wound. Hence, the variables representing 'cleansing lotion used on clean wounds' and 'cleansing lotion used on dirty or septic wounds' are excluded from any further calculations.

'Appraisal of isolation facilities', 'isolation of wound infections', and 'isolation of infections due to Staph. aureus' (when considered independently of patient parameters) all appear to have a potentially significant effect on patient nasal colonisation rates. However, when the results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting nasal colonisation rates, then it is only the differences in 'appraisal of isolation facilities' and those of 'isolation of infections due to Staph. aureus' that remain significant. It is, of course, logical that if a patient with a staphylococcal infection (which represents some 35.4% of all wound infections) is isolated in adequate facilities, then this will reduce the risk of colonisation in the main ward by virtue of the fact that the infected patient would not come into direct contact with any others in the ward. 'Isolation of infections due to Staph. aureus', should be contained within the factor representing 'isolation of wound infections', but despite their apparent significance, since only a very small number of patients were actually isolated, both the factors representing 'isolation of infections due to Staph. aureus' and that representing 'isolation of wound infections', together with the variable indicating the 'appraisal of isolation facilities', are excluded from any further consideration as being of any relevance to that regression model which is specifically concerned with modelling 'patient nasal colonisation rates'.

7.2 Analysis of wound results summarised in Tables 6a and 6b

Consider the significant results with respect to the 'rate of wound infections' as follows:-

Table 6a

Analysis of variable ward practices II
(with respect to patient 'wound infection' rates)

VARIABLE WARD PRACTICE II	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Schedule for pre-operative preparation	3.72	2	5.99	N/S
Method of pre-operative preparation	8.77	2	5.99	Sig.
Treatment of ward shaving razor	10.38	5	11.07	N/S
Location of main wound dressing site	9.78	2	5.99	Sig.
Number of staff in dressing team	4.42	2	5.99	N/S
Dress of 'dressing team'	10.67	4	9.488	Sig.
Are gloves used?	1.76	2	5.99	N/S
Method of handwashing - general	1.58	2	5.99	N/S
Method of handwashing - special	3.21	2	5.99	N/S
Method of handwashing - dressings	0.74	2	5.99	N/S
Use of hand cream in wards	12.77	4	9.488	Sig.
Occasions when scrubbing is used	0.74	2	5.99	N/S
Treatment of nail brushes	7.34	2	5.99	Sig.
Hand cream - container	1.22	1	3.84	N/S
Treatment of Cheatle's forceps	2.46	3	7.815	N/S
Type of dressing - clean undrained wounds	2.50	3	7.815	N/S
Type of dressing - drained wounds	6.28	3	7.815	N/S
Type of dressing - dirty or septic wounds	0.24	3	7.815	N/S
Cleansing lotion used on clean wounds	14.66	4	9.488	Sig.
Cleansing lotion used on dirty or septic wounds	4.73	4	9.488	N/S
Appraisal of isolation facilities	0.32	2	5.99	N/S
Isolation of wound infections	6.89	4	9.488	N/S
Isolation of infections due to <u>Staph. aureus</u>	2.99	4	9.488	N/S

Table 6b

Analysis of variable ward practices II
(with respect to patient 'wound infection' rates[†])

VARIABLE WARD PRACTICE II	χ^2_{calc}	d.f.	χ^2_{tab}	Status
Schedule for pre-operative preparation	3.51	2	5.99	N/S
Method of pre-operative preparation	2.74	2	5.99	N/S
Treatment of ward shaving razor	6.83	5	11.07	N/S
Location of main wound dressing site	3.07	2	5.99	N/S
Number of staff in dressing team	2.24	2	5.99	N/S
Dress of 'dressing team'	10.69	4	9.488	Sig.
Are gloves used?	0.56	2	5.99	N/S
Method of handwashing - general	3.70	2	5.99	N/S
Method of handwashing - special	2.47	2	5.99	N/S
Method of handwashing - dressings	1.73	2	5.99	N/S
Use of hand cream in wards	7.63	4	9.488	N/S
Occasions when scrubbing is used	0.77	2	5.99	N/S
Treatment of nail brushes	3.43	2	5.99	N/S
Hand cream - container	0.80	1	3.84	N/S
Treatment of Cheatle's forceps	0.26	3	7.815	N/S
Type of dressing - clean undrained wounds	2.26	3	7.815	N/S
Type of dressing - drained wounds	3.76	3	7.815	N/S
Type of dressing - dirty or septic wounds	1.69	3	7.815	N/S
Cleansing lotion used on clean wounds	6.05	4	9.488	N/S
Cleansing lotion used on dirty or septic wounds	0.60	4	9.488	N/S
Appraisal of isolation facilities	0.72	2	5.99	N/S
Isolation of wound infections	4.05	4	9.488	N/S
Isolation of infections due to <u>Staph. aureus</u>	2.55	4	9.488	N/S

[†] modified to account for significant patient parameters

'Method of pre-operative preparation' (when considered independently of patient parameters) appears to have a potentially significant effect on patient wound infection rates, but when the results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting wound infection rates, then any variations in 'method of pre-operative preparation' become non-significant. This result in itself was anticipated, and since the period of the survey, changing practices now mean that hexachlorophane is currently not used because of its reputed toxicity, whilst chlorhexidine is increasingly used. Since the differences between those 'methods of pre-operative preparation' currently in use are non-significant, then this factor can be excluded from further consideration as being a relevant part of that regression model which is specifically concerned with modelling 'patient wound infection rates'.

'Location of main wound dressing site' (when considered independently of patient parameters) appears to have a potentially significant effect on patient wound infection rates, but when the results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting wound infection rates, then 'location of main wound dressing site' proves to be non-significant. This result is accepted, because not only is the dressing of wounds in the ward becoming an increasingly common practice, but also it is illogical for a 'non-ventilated dressing room' where the risk of airborne infection is greater than that for a comparable 'ventilated dressing room', to produce results indicating the opposite to be true. 'Location of main wound dressing room' is, therefore, rejected as an independent factor having any significant effect on 'patient wound infection rates' and is consequently excluded from any further calculations.

'Dress of "dressing team"' appears to have a potentially significant effect on patient wound infection rates. Further analysis of the results reveal that the category of 'no special dress' gives the lowest rate of infection, and so it appears that the use of special dress is a redundant precaution against cross-infection of those patients with wounds. This appears to agree with the results of WILLIAMS and OLIVER (105, 1963), who found no apparent increase in infection rates when nurses gave up wearing masks and gowns. Inconsistencies, however, become apparent when a logical answer is sought for the reason why the wearing of cleaner, more occlusive dress (which is supposedly designed to protect against the risk of infection from special areas of the body), results in infection rates which are higher than those obtained when 'no special dress' is worn. Hence, for these reasons, the variable representing 'dress of "dressing team"' is excluded from any further consideration as being a relevant part of that regression model which is specifically concerned with modelling 'patient wound infection rates'.

'Use of hand cream in wards' and 'treatment of nail brushes' (when considered independently of patient parameters) both appear to have a potentially significant effect on patient wound infection rates, but when these results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting wound infection rates, then both 'use of hand cream in wards', together with 'treatment of nail brushes', prove to be non-significant. Both of these parameters are now considered to be bad practice and so their use has been reduced wherever possible. Although it is not a feasible proposition to ban nurses from bringing (and using) their own into the

hospital, hand cream is now no longer issued because it was felt that communal use caused problems with respect to cross-infection. It is now recommended that nail brushes are not used, but if this is unavoidable, they should be regularly autoclaved in order to thoroughly clean them. Additionally, any individual's use of either hand cream or nail brushes, is somewhat unpredictable, and possibly irregular. It is for a combination of these reasons, that the factors representing 'use of hand cream in wards' and 'treatment of nail brushes', are excluded from any further calculations, because these are not considered to be independent factors likely to have any significant influence on current wound infection rates.

'Cleansing lotion used on clean wounds' (when considered independently of patient parameters) appears to have a potentially significant effect on patient wound infection rates, but when the results are adjusted to take into account those variable patient parameters which are accepted as significantly affecting wound infection rates, then 'cleansing lotion used on clean wounds' proves to be non-significant. This result is accepted because the type and condition of the patient's wound is likely to dictate the choice of lotion, insofar as 'saline' (with no antiseptic properties) is more likely to be used for those wounds which are not infected or considered to be at risk, whilst 'alcohol' is more likely to be used where disinfection is considered to be more important or to dry-up a weeping wound. Therefore, since each of the products is not used under the same conditions, they have not been given equivalent challenges, and so the factor representing 'cleansing lotion used on clean wounds' is excluded from any further calculations.

CHAPTER 8

DISTRIBUTION OF THE PATIENT POPULATION

FREEMAN, ROSNER and McGOWAN (10, 1979) concluded in their paper, that if the hospital was in a mathematically 'steady state', where new patients are admitted at a similar rate to which others are discharged, then the rate at which new infections occur remains relatively constant: whilst MOODY and BURKE (32, 1972) suggest that the results of prevalence surveys give only a single view of a dynamic phenomenon, and consequently, must be considered in the light of previous or subsequent surveys.

8.1 Age distribution of patients

In order to facilitate future comparisons with this study, the distributions for each of the patient populations must first be established. Figure 1 shows the distribution of all patients in the survey, from whom nasal swabs were analysed, in the form of a relative frequency histogram. This histogram is generally skewed to the left, indicating the relative predominance of an ageing patient population, which is supported by the fact that the average age for the overall patient population was found to be 54.6 years. There is an interesting localised peak spanning the ages 15 to 30 years, this increase in patient population, however, can be attributed solely to young women having their babies delivered in maternity units. A further breakdown of the type of patients involved in the survey is given in Appendix D1.

FIGURE 1 - Age of distribution for ALL patients

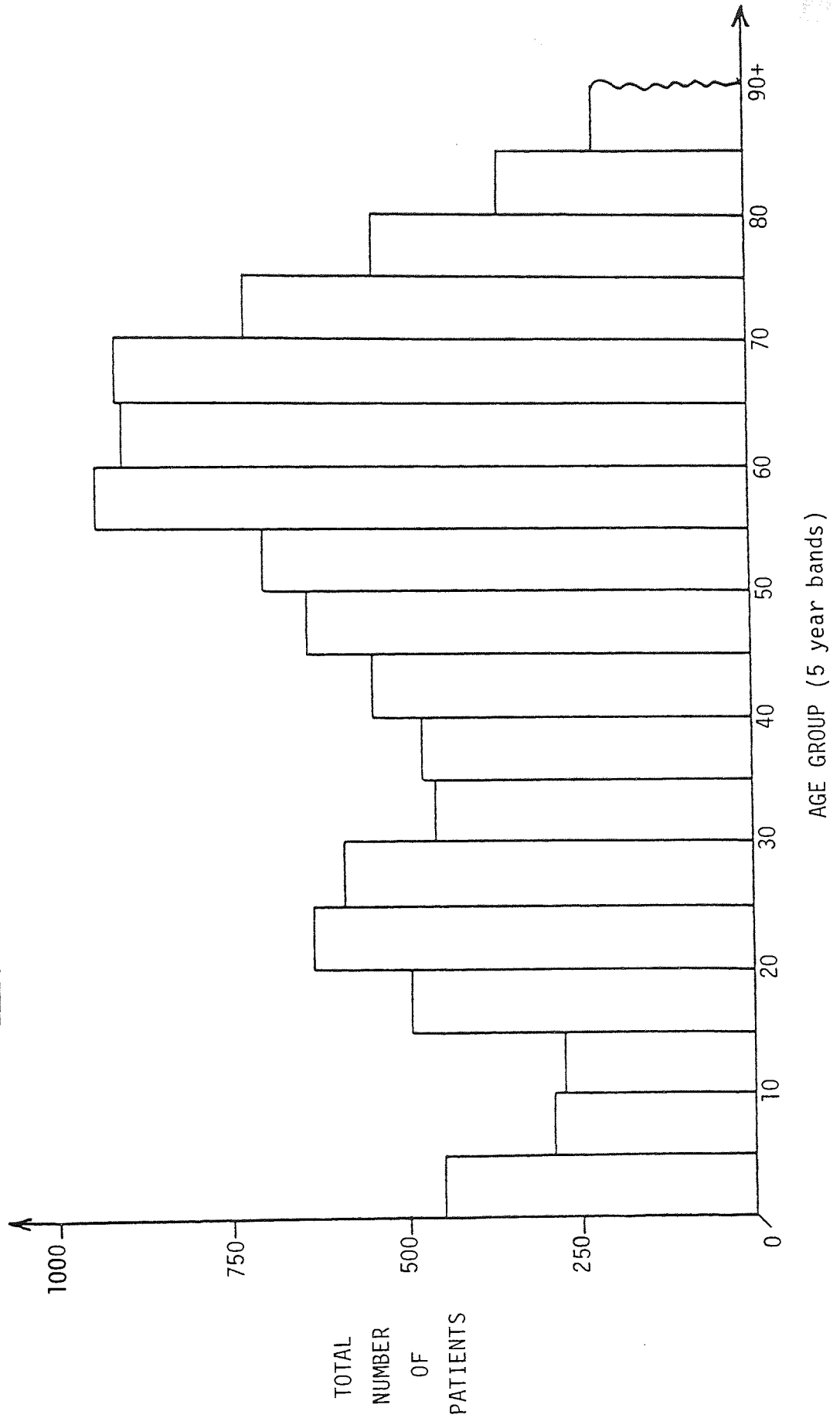


Figure 2 is confined to just those patients who have undergone operative procedures. This histogram is very similar in profile to the previous one, again having a skew to the left with a slightly (but non-significantly) reduced average patient age of 53.2 years. The reason why both overall and operative patient frequencies tail-off rapidly after the age of 70 years, can be accounted for purely because of the decreasing numbers in the population as a whole, who survive beyond this age. More detailed information on the type of operations included in the survey is shown in Appendix D2.

8.2 Relationship between age, drains and wound type

The results shown in Table 7, give the breakdown of wounds into clean, clean-contaminated and contaminated, which are further subdivided into drained and undrained classifications for each of the patient age bands. It is useful to note that 63.2% of operated patients have clean wounds (of which just over one-quarter are drained) whilst clean-contaminated wounds account for another 26.3% (of which over half are drained) and the final 10.5% are contaminated wounds (of which some two-thirds are drained). Clearly, the results of any survey will be dependent on the distribution of wound types which are analysed, and likewise, the proportion of wounds recorded as being drained, will be correspondingly influenced by the types of wound which are predominant in the patient population at the time of any given survey.

The distribution of ages for each of the wound/drain classifications follows a similar format to that shown in Fig. 2, whilst Fig. 3 reveals,

FIGURE 2 - Age distribution for those patients having an operation wound

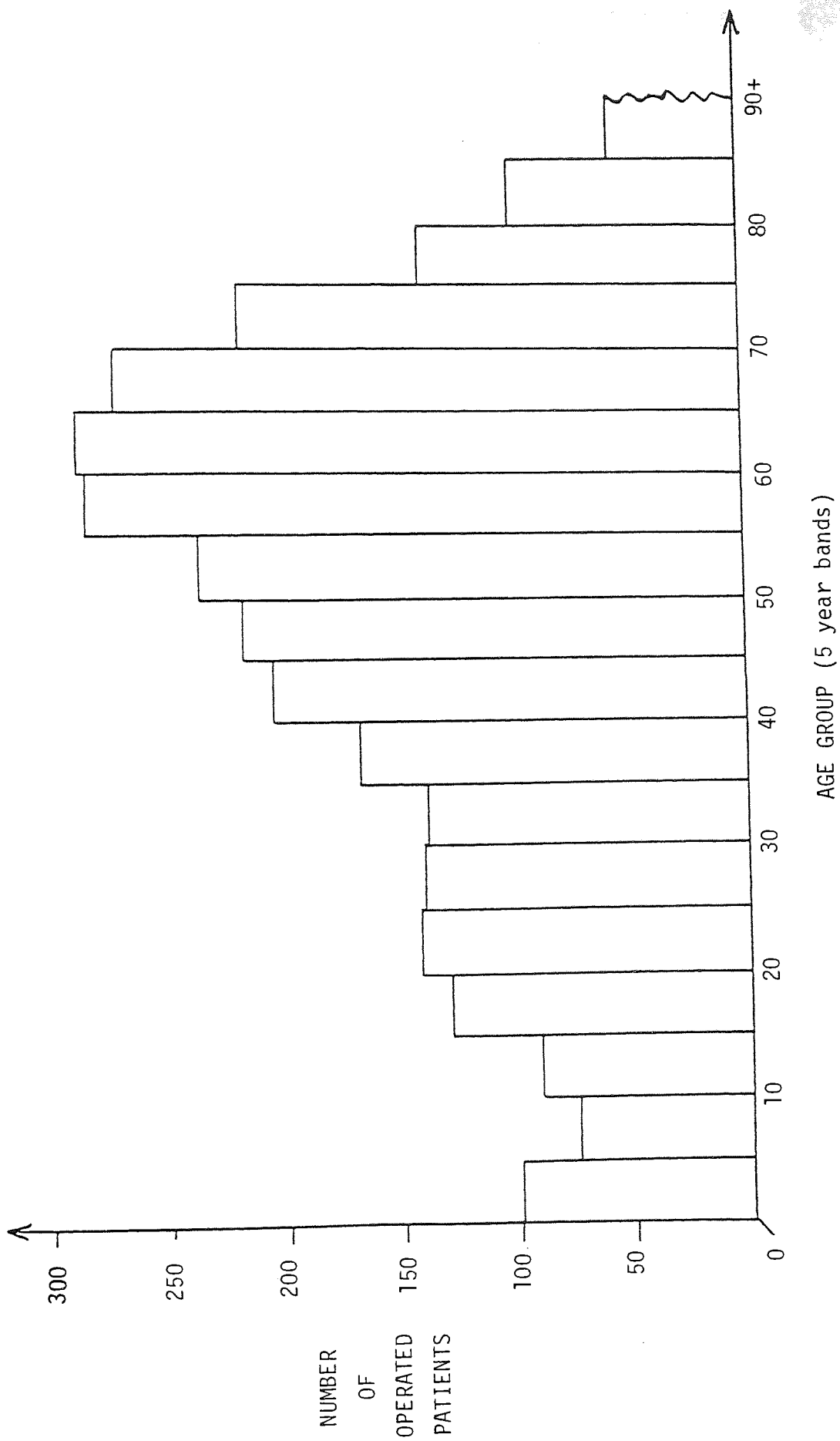
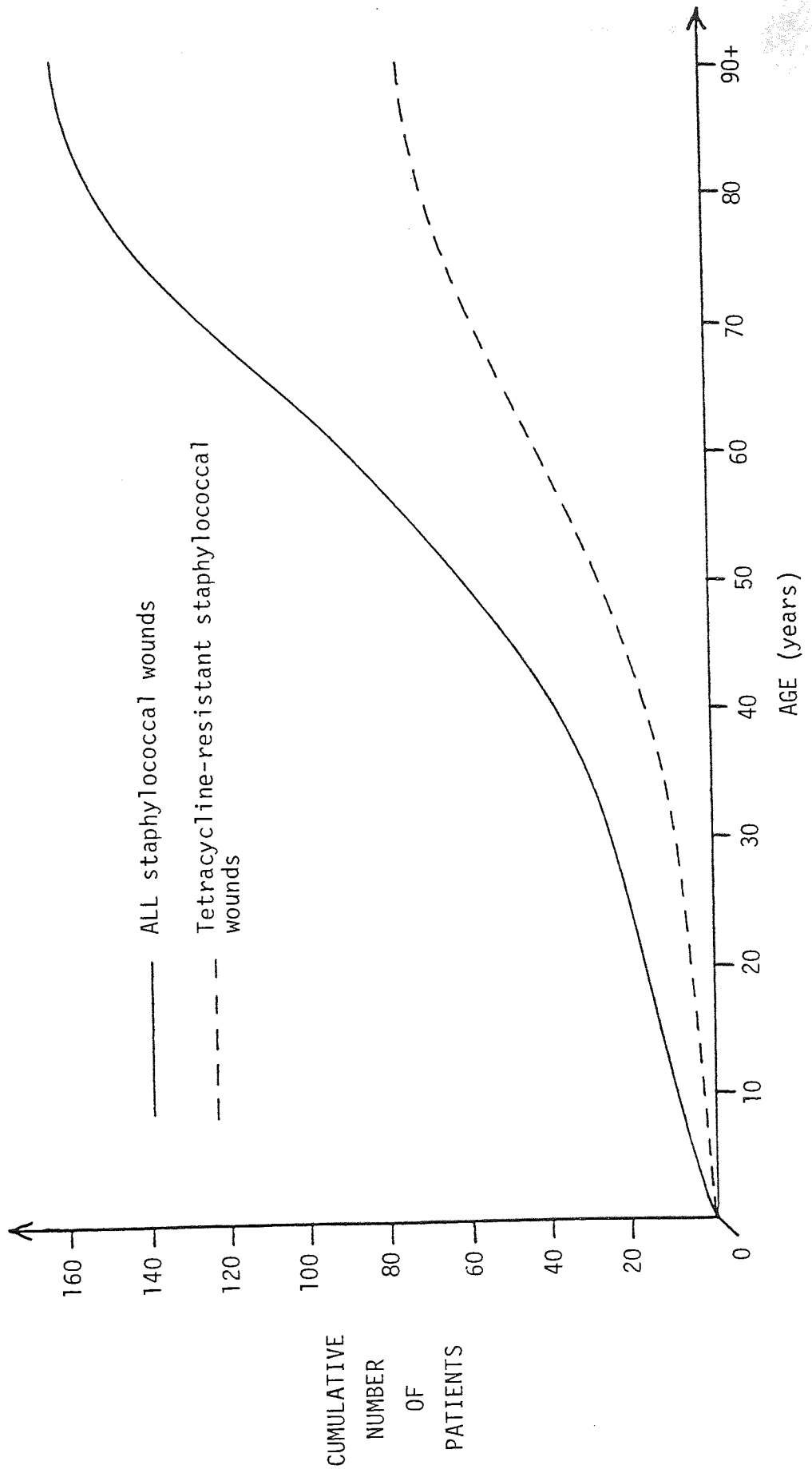


Table 7

Age distribution of patients for different TYPES of WOUND

AGE GROUP (years)	CLEAN WOUNDS		CLEAN-CONTAMINATED WOUNDS		CONTAMINATED WOUNDS	
	Number which are UNDRAINED	Number which are DRAINED	Number which are UNDRAINED	Number which are DRAINED	Number which are UNDRAINED	Number which are DRAINED
1 - 5	69	6	5	4	6	9
6 - 10	39	3	18	7	6	3
11 - 15	38	6	37	1	2	6
16 - 20	72	10	27	13	4	4
21 - 25	77	16	23	15	4	8
26 - 30	70	15	27	16	6	4
31 - 35	76	14	17	15	6	7
36 - 40	89	31	15	27	3	1
41 - 45	106	32	30	24	8	5
46 - 50	111	35	24	31	4	12
51 - 55	109	47	17	41	7	16
56 - 60	131	49	25	47	9	21
61 - 65	105	70	29	43	8	30
66 - 70	93	59	23	51	13	32
71 - 75	67	49	20	52	9	19
Over 75	125	65	26	34	18	22
ALL ages	1,377	507	363	421	113	199

FIGURE 3 - Cumulative distribution of patients having staphylococcal wounds



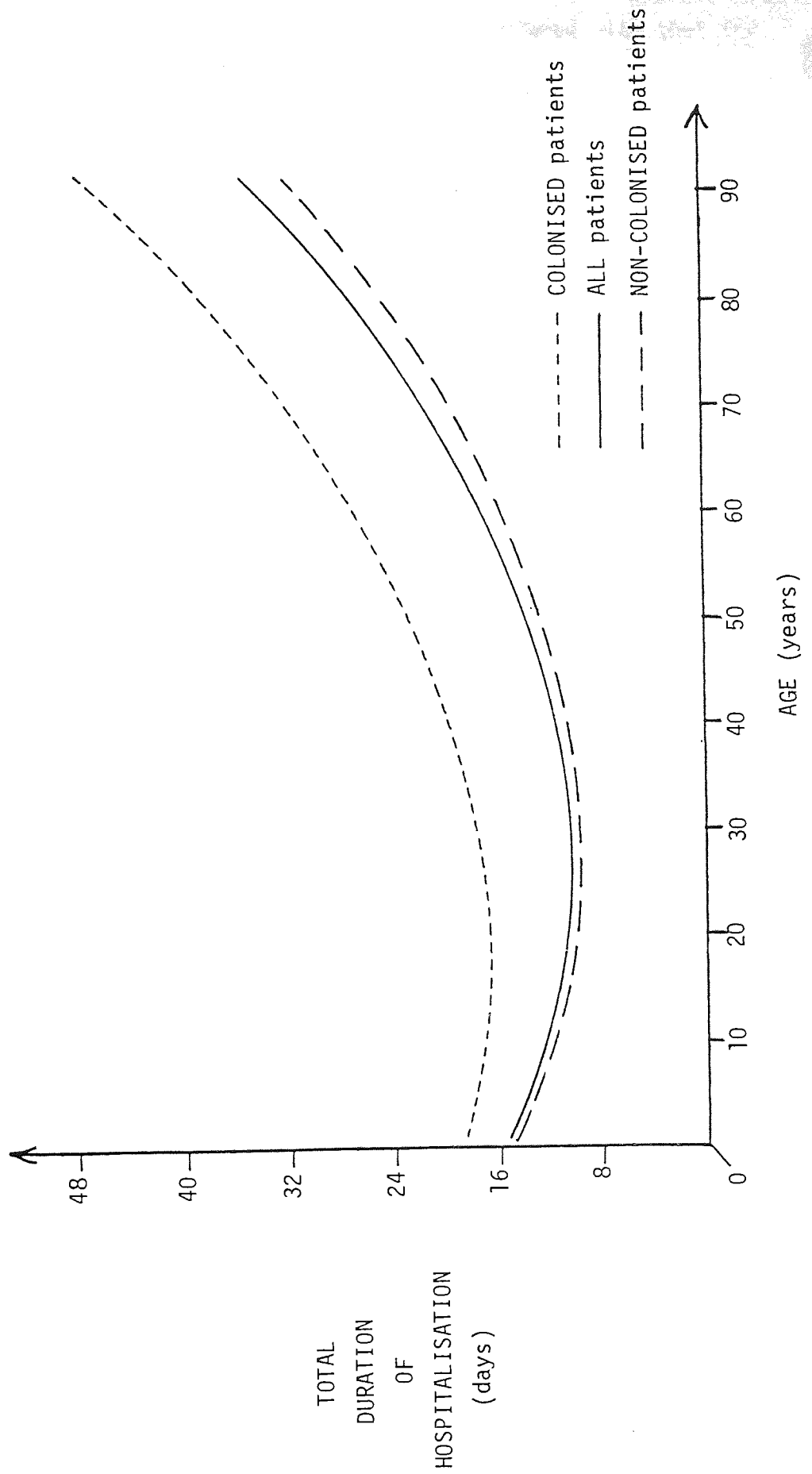
in more detail, the distribution of patients having any form of staphylococcal wound as compared with just those which are tetracycline-resistant. The curves for the cumulative distributions are classical 'S' shapes with wounds which are infected with tetracycline-resistant Staphylococcus aureus accounting for approximately half of all the staphylococcal wounds analysed in the survey.

The slopes of both curves remain reasonably constant up to the age of 35 years, indicating that there are similar numbers of patients in each of these age groups, but from the age of 35 years onwards, the slopes of the curves begin to steepen rapidly up to the age of 70 years, before they again begin to flatten out. This increase in the slopes, reveals that more patients in age groups between 35 and 70 are acquiring staphylococcal wounds with the peak age group corresponding to those parts of the respective graphs with the maximum rate of change of slope, i.e. the steepest part of the curves, which occur between the ages of 60 and 65 years.

8.3 Relationship between age and duration of hospitalisation

Referring to Fig. 4, it becomes clear that, when the patient population is considered as a whole, there is a MINIMUM total duration of hospitalisation which coincides with the 20 to 30 year age group for all patients, except those which are colonised with tetracycline-resistant Staph. aureus. The minimum hospital duration for colonised patients, occurs within the 15 to 25 years age group, and throughout the entire age spectrum, their total duration of hospitalisation is

FIGURE 4 - Age of patient versus total duration of hospitalisation



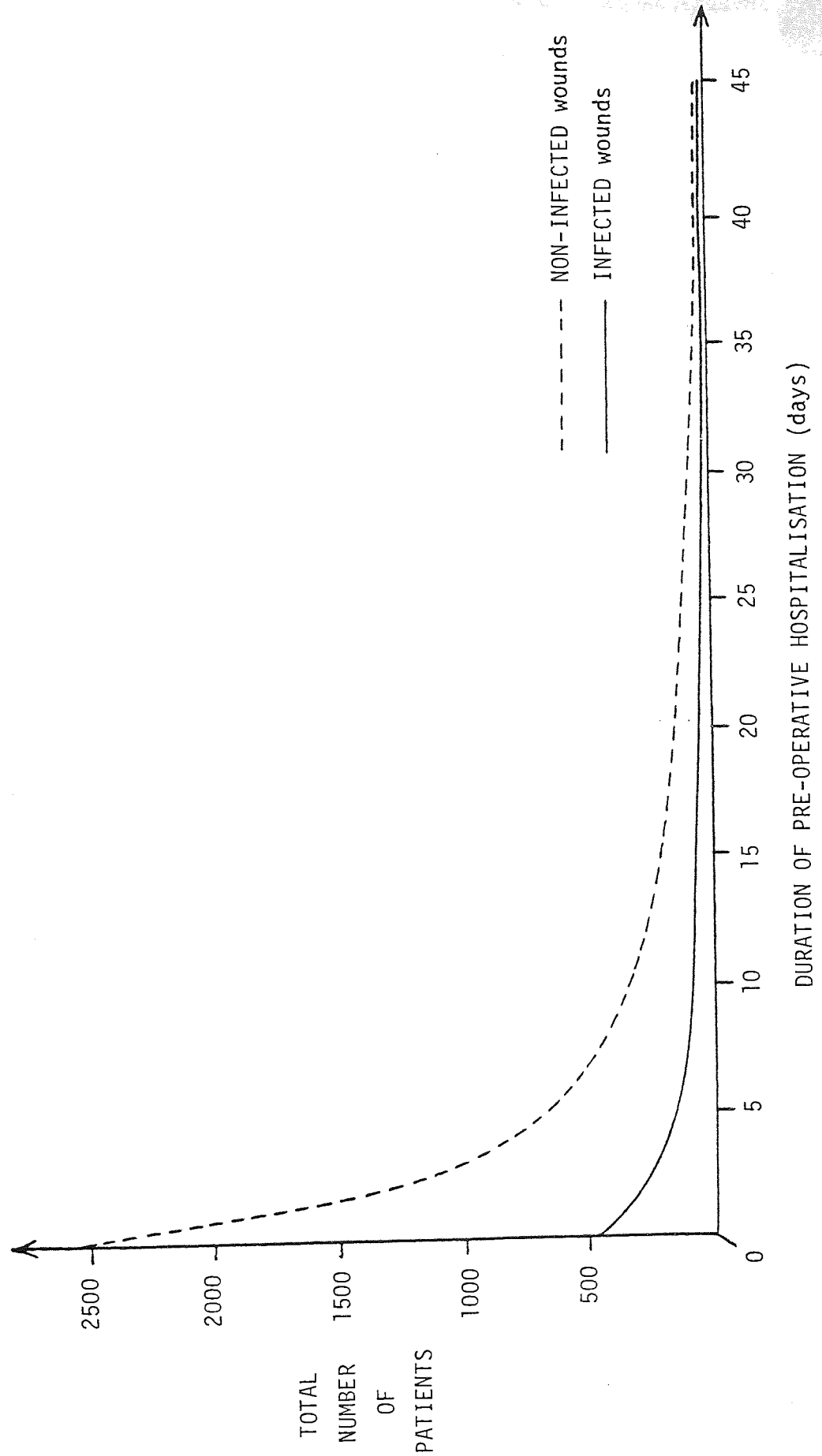
consistently increased by about one-third as compared with that for all patients considered together, whilst for the non-colonised group, the average period of time spent in hospital is reduced by some TEN per-cent.

8.4 Distribution of pre-operative hospitalisation

It is of great value to look retrospectively at the duration of pre-operative stays for hospitalised patients, and to collate this data with additional information indicating which patients subsequently develop a wound infection, in order to determine whether or not there is any link between the two factors. The progress of all patients through the hospital system was monitored, and is graphically illustrated in Fig. 5. Initially, all patients had zero days before their operation, most went on to stay in hospital for 1, 2, or more days pre-operatively, and as the patient underwent his operation he was withdrawn from the ever-decreasing pool of survey patients awaiting operations.

The graph has been divided into two subsections, to show the differences between the progress of non-infected patients and those patients whose wounds became post-operatively infected. Within the first five days, the number of patients with non-infected wounds drops far more rapidly than for those with infected wounds over the corresponding time period. This indicates a larger proportion of the non-infected group have short periods of pre-operative hospitalisation as compared with the infected group, so it is reasonable to postulate that an increased pre-operative stay in hospital, even though it may be essential for diagnostic or other purposes, still has a detrimental

FIGURE 5 - Cumulative distribution of pre-operative hospitalisation



effect on the outcome of whether a patient becomes host to subsequent post-operative wound infection.

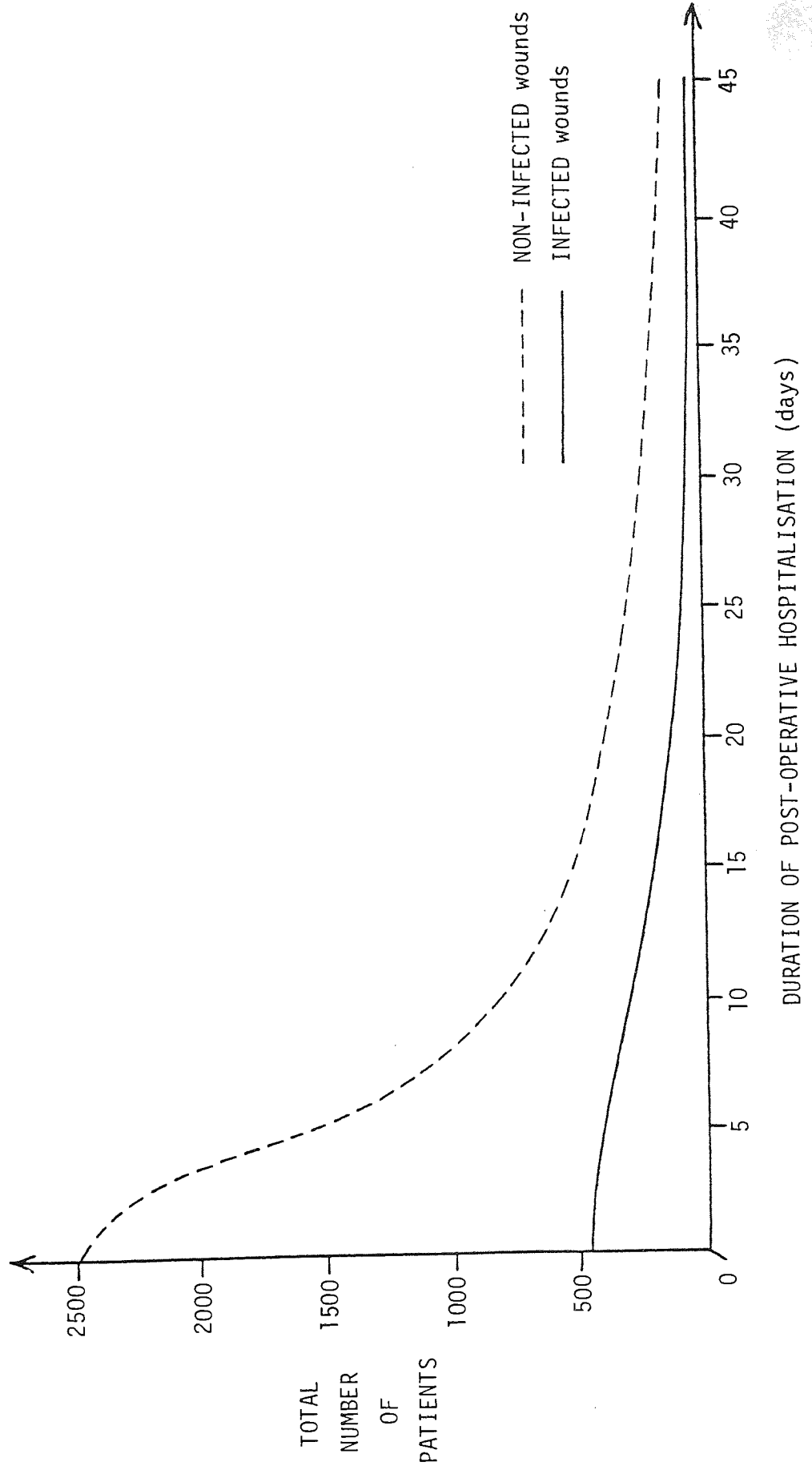
The average duration of pre-operative hospitalisation for non-infected patients is 5.2 days, whilst for the group of patients with infected wounds, this figure rises to 8.1 days, with no detectable difference between male and female patients from either group. It therefore appears, that the patients whose wounds subsequently become infected, were on average, pre-operatively hospitalised for 2.9 days more than equivalent patients whose wounds remained free from infection.

8.5 Distribution of post-operative hospitalisation

Assessment of post-operative stays in hospital is a somewhat controversial aspect of cross-sectional surveys, because conditions can only be observed at one particular instant in time. However, for the purposes of direct comparisons within this closed survey, it is quite justified to perform this particular function. Direct reference to Fig. 6 reveals the distribution of post-operative hospitalisation to be of a similar form to that for patients' pre-operative stays in hospital (shown in Fig. 5). The only difference being a slight 'kink' at the beginning of the post-operative curves, which can easily be accounted for, because very few patients were found to leave hospital immediately after an operation, but normally can expect to spend a period of time in the ward recovering from operative procedures.

The number of hospitalised patients with non-infected wounds drops very rapidly over the first 7 days, after which time the patient

FIGURE 6 - Cumulative distribution of post-operative hospitalisation



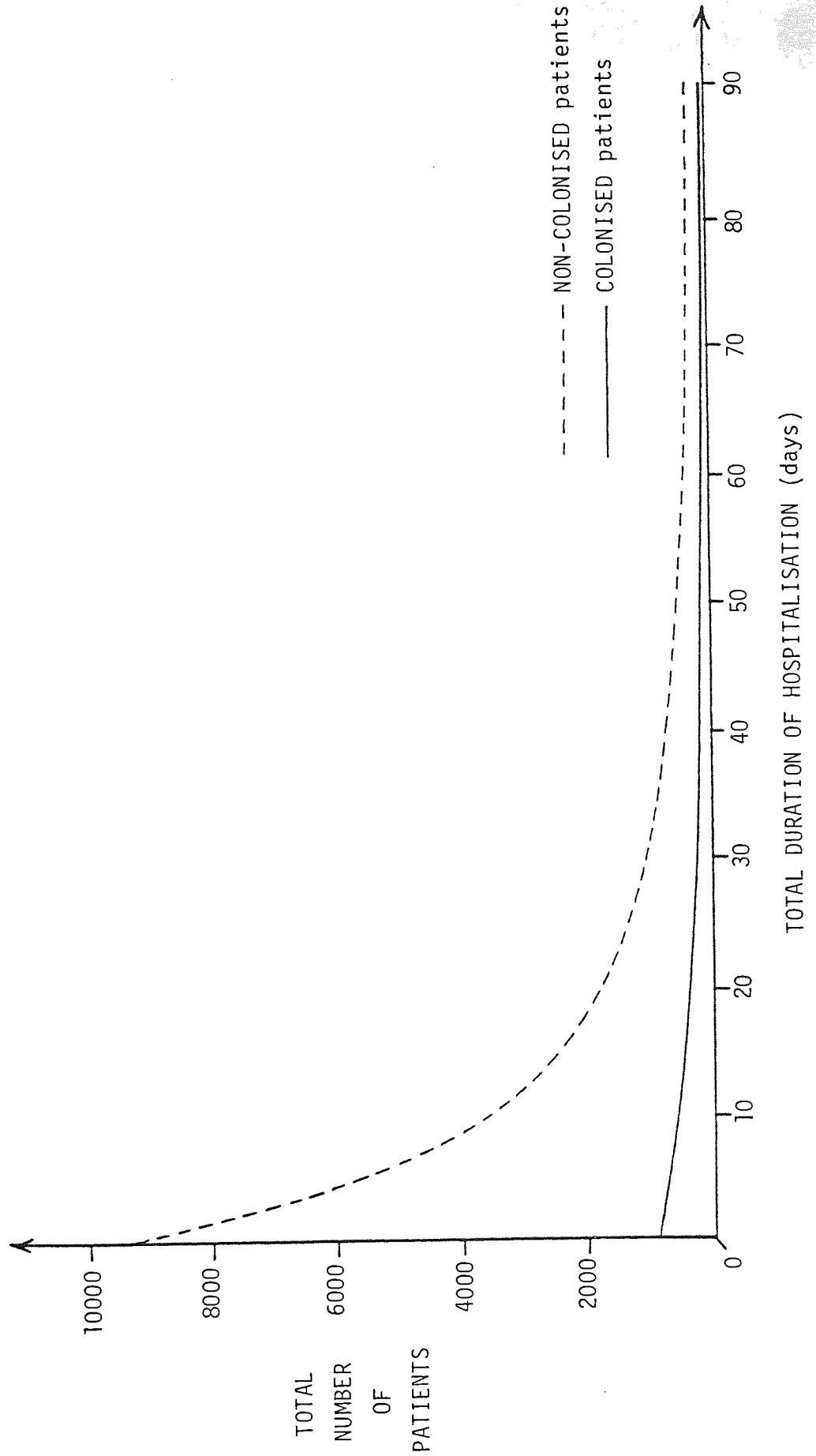
discharge-rate is still quite high, but noticeably reduced. After about 14 days post-operative stay in hospital, the rate of discharge stabilises to be nearly constant - yet very low. Over the same time period, the discharge-rate for patients with an infected wound, remains fairly low and constant over the first 14 days, but after 21 days the discharge rate is so low that it is very close to zero, leaving a small residue of patients hospitalised post-operatively for more than 6 weeks.

The average duration of post-operative hospitalisation for patients with non-infected wounds is 11.9 days, with no detectable difference between the sexes, whilst for the group of patients with an infected wound, the duration of post-operative hospitalisation averages out to 17.3 days for males, and 19.9 days for females (the mean being 18.4 days, after adjusting for differing numbers of male and female patients). Therefore, patients with an infected wound can expect to be hospitalised post-operatively for an additional 6.5 days.

8.6 Distribution of total durations of hospitalisation

Figure 7 illustrates the difference between those patients whose noses are colonised with tetracycline-resistant Staph. aureus and those which are not. It is interesting to note the very steeply descending curve for non-colonised patients over the first 28 days, indicating that the rate of discharges for this group of patients is very much higher than that for the colonised group. After this time period, the rate of discharges for non-colonised patients stabilises to a very low

FIGURE 7 - Cumulative distribution of hospitalisation for ALL patients



but uniform rate, whilst over the entire time-span the rate of discharges for the colonised group follows a similar pattern, but in a more damped and less spectacular fashion.

The average duration of hospitalisation for all non-colonised patients is 14.0 days for males as compared with 14.9 days for females, whilst for the group of patients with colonised noses, this rises substantially to 25.0 days for the males and 31.0 days for the females. This difference between the sexes can be clearly seen in Fig. 8, which shows the cumulative distribution of all patients (independent of whether they are colonised or not), subdivided into male and female classifications.

Throughout the range of total hospital durations, there are always more females present in the wards than males, and after some 70 days in hospital, females outnumber males in the ratio of 2:1.

The total durations of hospitalisation shown in Fig. 9 are for operated patients only, and basically represent an additive combination of pre-operative and post-operative stays. The number in the patient group with non-infected wounds drops more rapidly than that for the equivalent group with infected wounds over the first 21 days, after which time the rate of discharges for all remaining patients slows dramatically leaving a small number in the ward for more than 3 months.

The average duration of hospitalisation for the non-infected patients is 16.7 days, with no detectable difference between the sexes, whilst for the group of patients with an infected wound, the total

FIGURE 8 - Cumulative distribution of hospitalisation for different sexes of patients

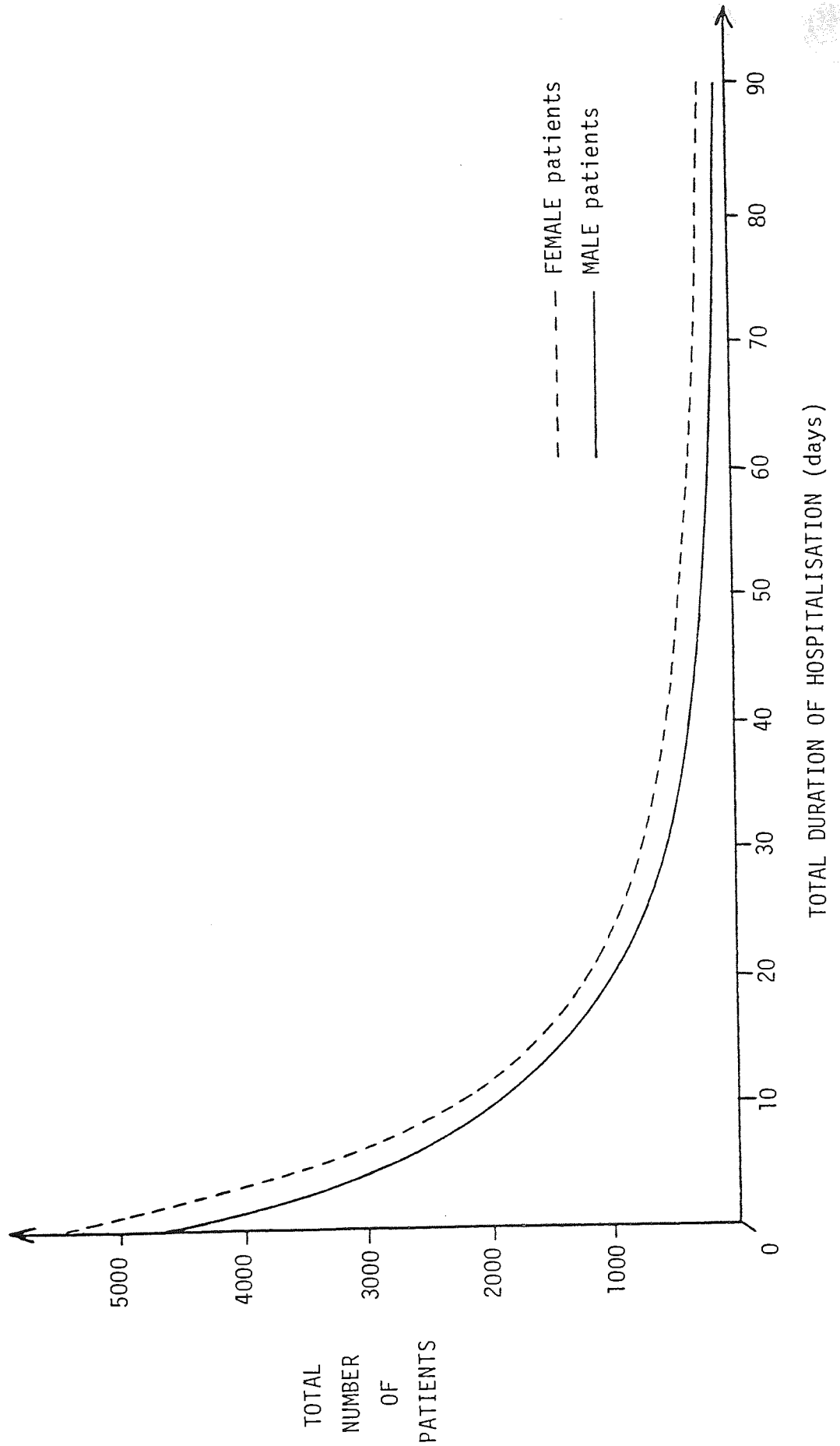
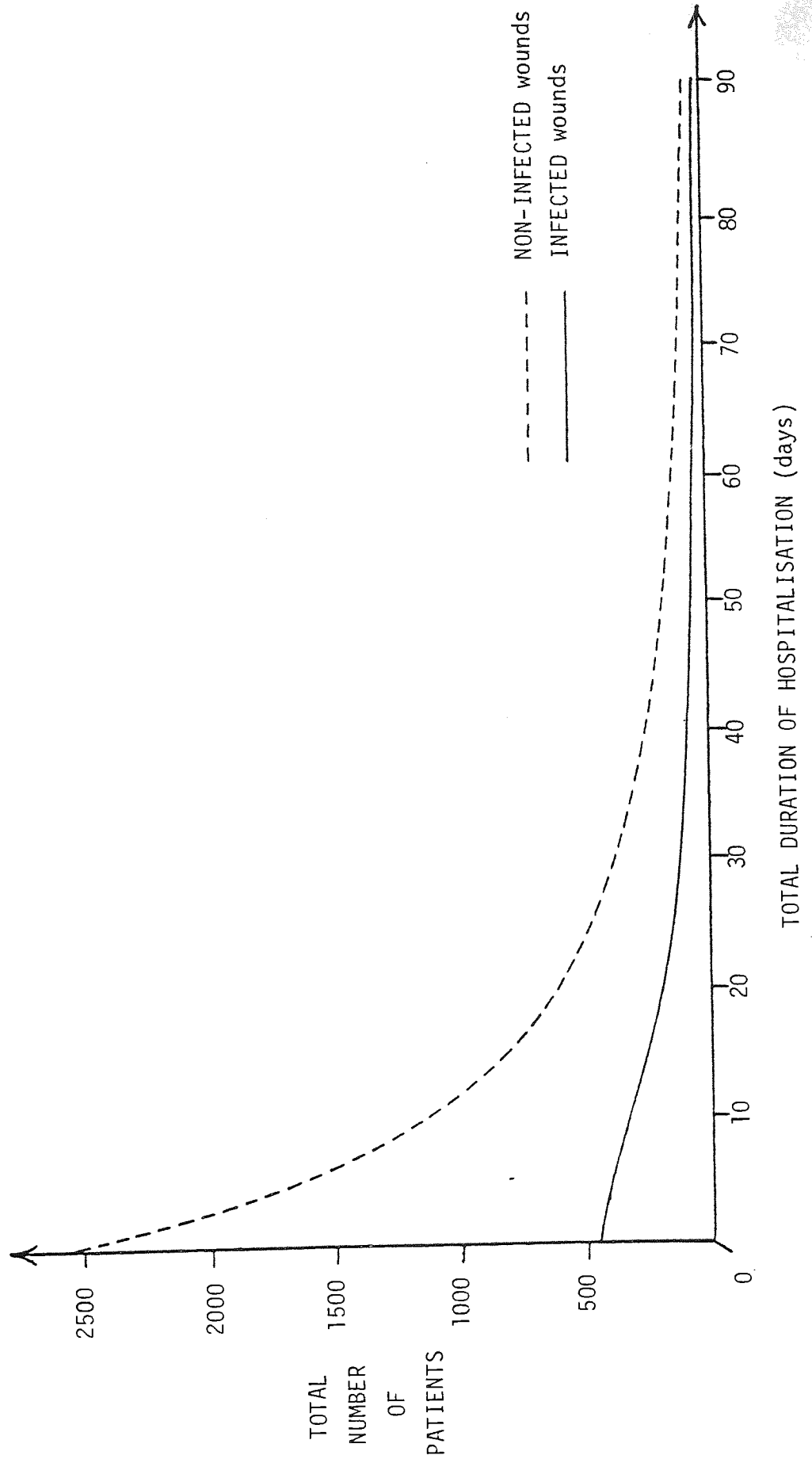


FIGURE 9 - Cumulative distribution of hospitalisation for operated patients



duration of hospitalisation averages out to 24.7 days for males and 27.6 days for females (the mean being 26.0 days, after taking into account the slightly different numbers of male and female patients). Hence, an average of 9.3 additional days were spent in hospital as a result of patients acquiring a post-operative wound infection.

CHAPTER 9

DISTRIBUTION OF COLONISATION AND INFECTION RATES

9.1 Relationship between age and patient risks

A graphical model representing the influence of a patient's age on rates of nasal colonisation is illustrated in Fig. 10 with the minimum risk occurring between the age of 15 and 25 years. The risks of colonisation for male and female patients are very similar in the early years of life, but the difference becomes greater as the age of the patient increases and is nearly 4% at the age of 80 years. A more accurately detailed breakdown of results in Table 8 shows that 1.74% more males than females become host to nasal colonisation with tetracycline-resistant Staphylococcus aureus.

The graphical model appropriate to wound infection rates shows a very smoothed distribution (in Fig. 11) with no localised turning points to indicate any age-range where patient risks are minimised. The risks of wound infection rise very slowly for patients up to the age of 40 years, after which age they begin to increase just a little faster. Specific differences in the wound infection rates are recorded in Table 9, where males are found, on average, to have infection risks which are increased by a factor of nearly one-half over that of equivalent females.

The relative predominance of selected types of post-operative wound infection are shown in Fig. 12, where the rate of change for each of the slopes can be seen to increase more rapidly with advancing

FIGURE 10 - Age of patient versus rate of nasal colonisation

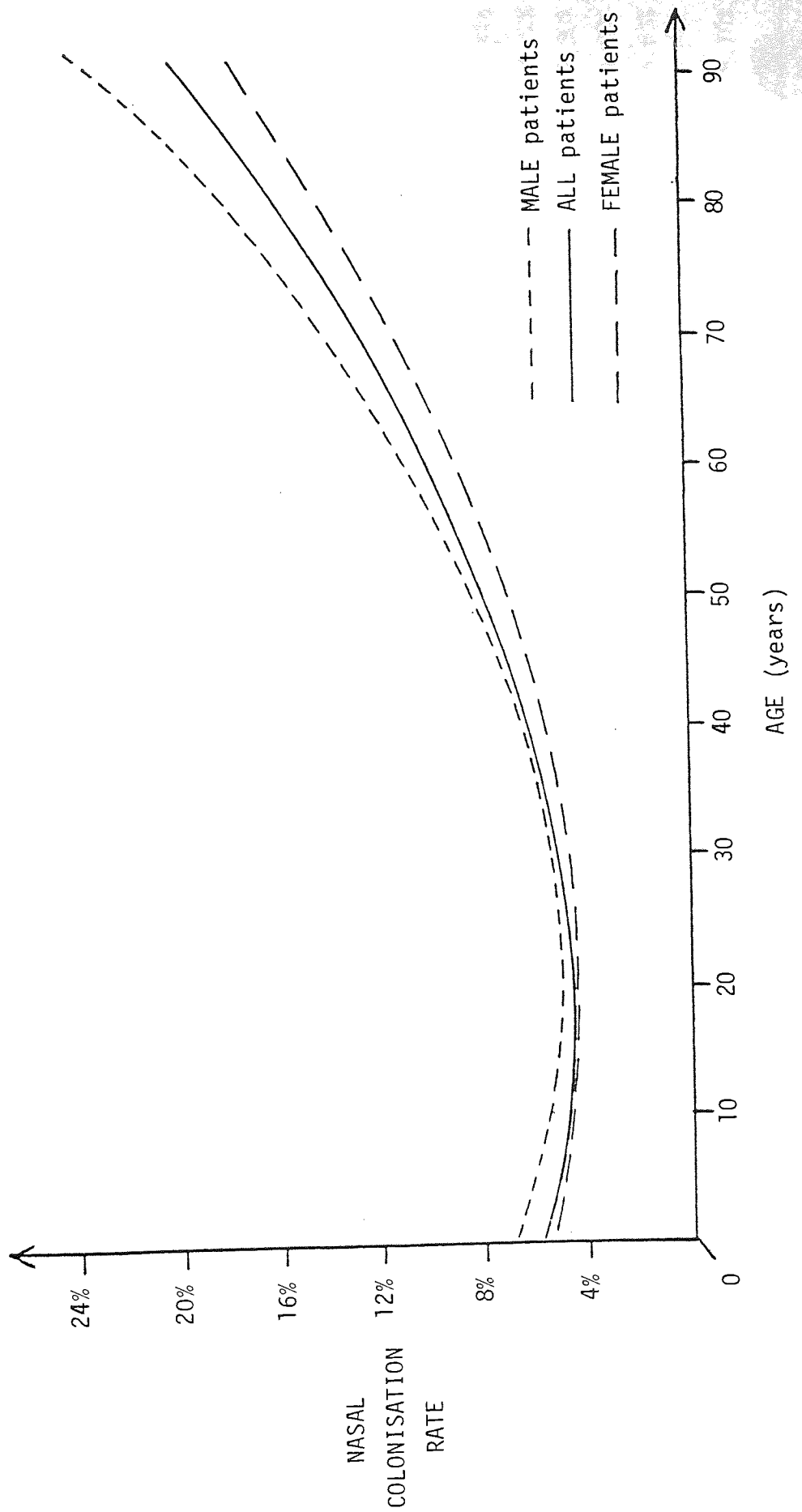


Table 8

Nasal colonisation rates for different AGE groups

AGE GROUP (years)	MALE		FEMALE		ALL patients	
	Number of patients in group	Nasal colonisation rate	Number of patients in group	Nasal colonisation rate	Number of patients in group	Nasal colonisation rate
1 - 5	289	5.54%	196	7.65%	485	6.39%
6 - 10	170	5.88%	118	5.08%	288	5.56%
11 - 15	156	5.13%	124	1.61%	280	3.57%
16 - 20	177	6.78%	320	3.75%	497	4.83%
21 - 25	185	5.41%	448	5.13%	633	5.21%
26 - 30	159	6.29%	435	3.45%	594	4.21%
31 - 35	169	4.73%	290	4.48%	459	4.58%
36 - 40	203	4.93%	276	4.71%	479	4.80%
41 - 45	259	6.56%	288	5.56%	547	6.03%
46 - 50	325	6.15%	313	5.75%	638	5.96%
51 - 55	376	9.84%	326	10.12%	702	9.97%
56 - 60	515	9.51%	430	9.07%	945	9.31%
61 - 65	513	12.09%	387	7.75%	900	10.22%
66 - 70	445	12.58%	459	10.46%	904	11.50%
71 - 75	303	17.16%	420	12.62%	723	14.52%
Over 75	379	18.21%	720	14.31%	1,099	15.65%
ALL ages	4,623	9.65%	5,550	7.91%	10,173	8.70%

FIGURE 11 - Age of patient versus rate of wound infection

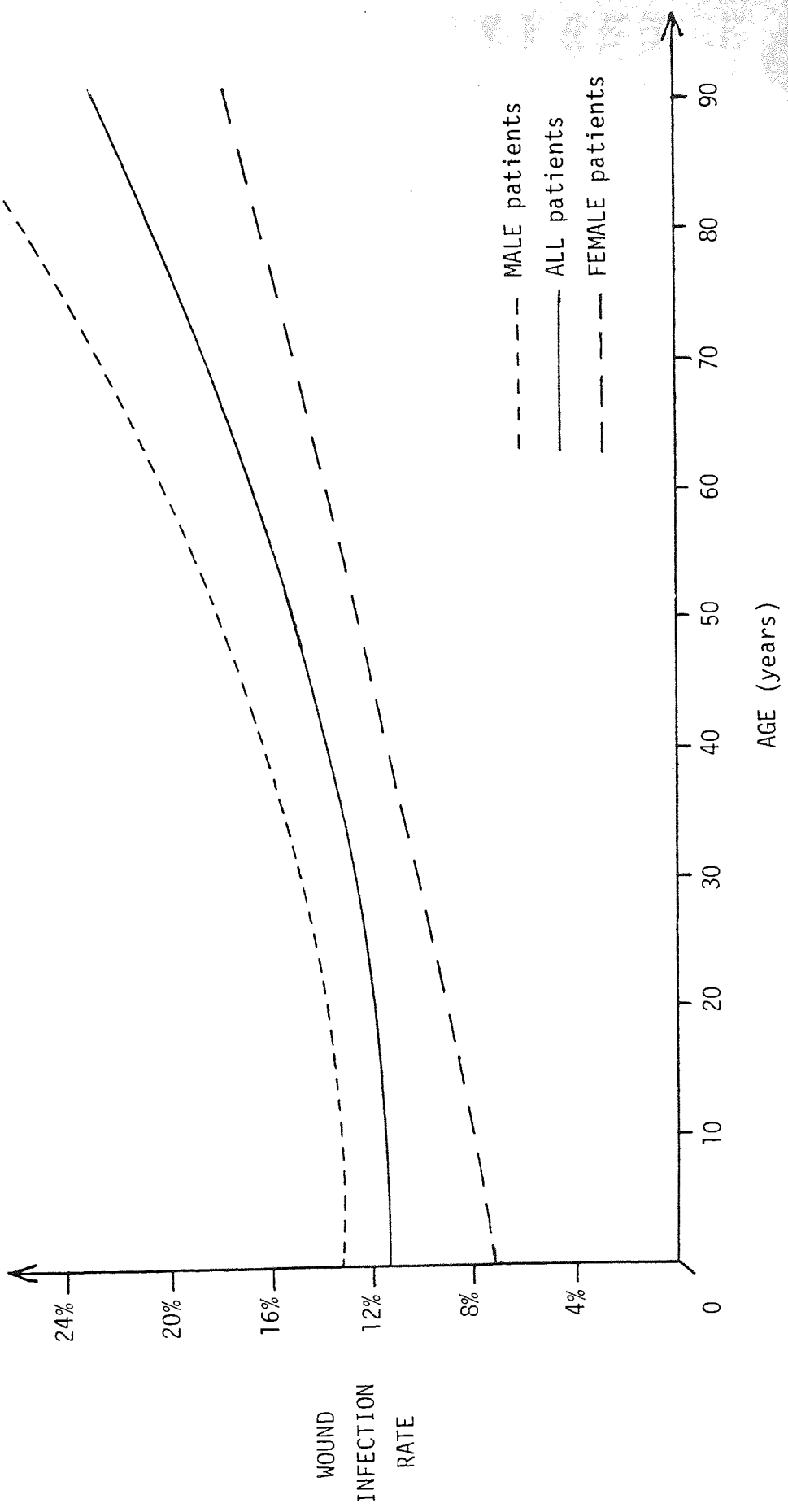
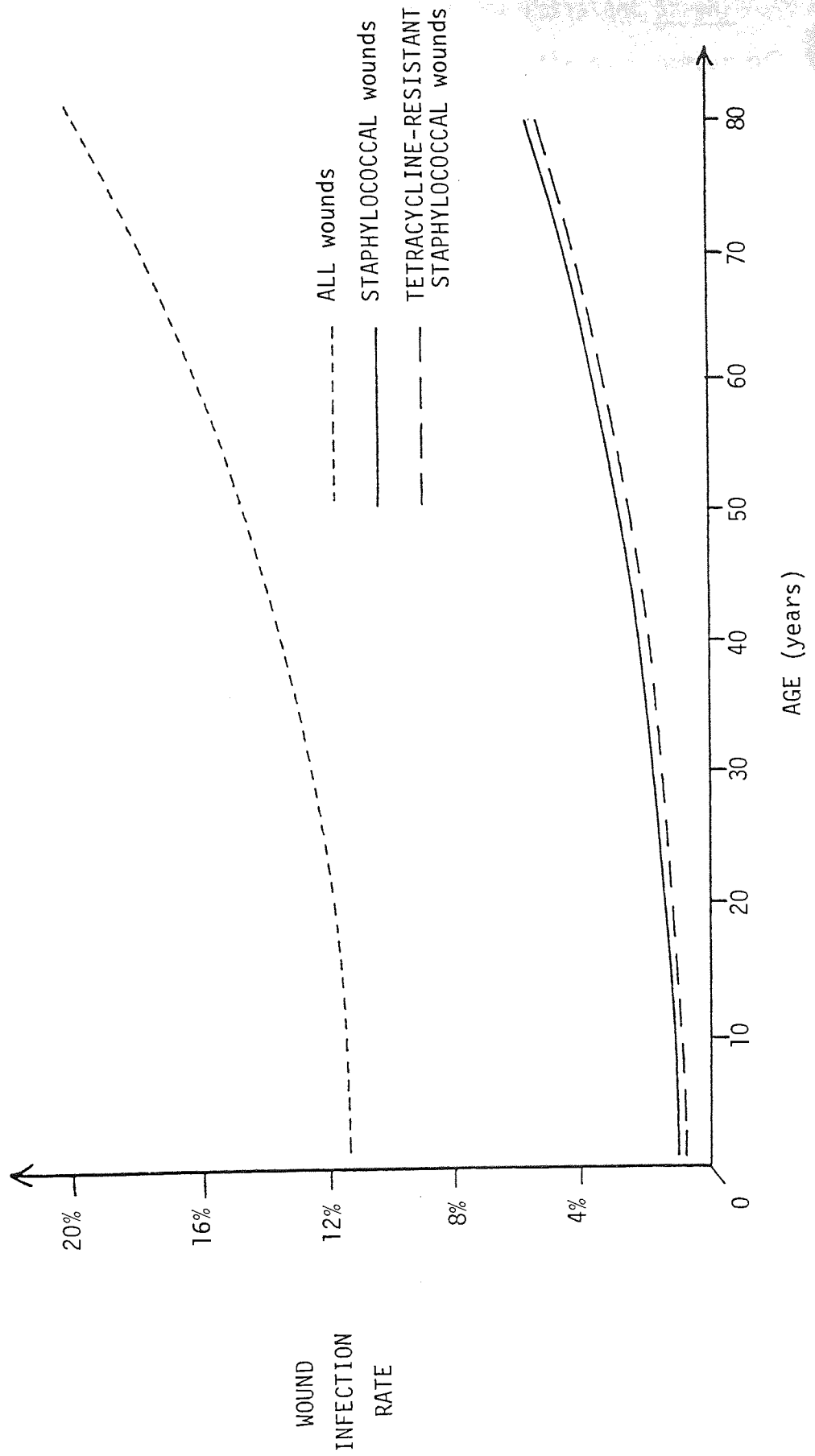


Table 9

Wound infection rates for different AGE groups

AGE GROUP (years)	MALE		FEMALE		ALL patients	
	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate
1 - 5	65	15.38%	34	2.94%	99	11.11%
6 - 10	43	13.95%	31	12.90%	74	13.51%
11 - 15	53	9.43%	37	10.81%	90	10.00%
16 - 20	72	15.28%	58	6.90%	130	11.54%
21 - 25	75	14.67%	68	7.35%	143	11.19%
26 - 30	51	17.65%	87	8.05%	138	11.59%
31 - 35	52	17.31%	85	11.76%	137	13.87%
36 - 40	53	9.43%	113	10.62%	166	10.24%
41 - 45	90	12.22%	115	8.70%	205	10.24%
46 - 50	105	18.10%	112	13.39%	217	15.67%
51 - 55	133	13.53%	104	17.31%	237	15.19%
56 - 60	141	20.57%	141	10.64%	282	15.60%
61 - 65	146	22.60%	139	15.11%	285	18.95%
66 - 70	150	26.00%	121	20.66%	271	23.62%
71 - 75	99	26.26%	117	9.40%	216	17.13%
Over 75	107	20.56%	183	16.39%	290	17.93%
ALL ages	1,435	18.33%	1,545	12.43%	2,980	15.27%

FIGURE 12 - Relationship between different types of wound infection and age



patient age. Wounds infected with tetracycline-resistant Staph. aureus, and other staphylococci, occur in roughly the same number of patients throughout the entire age range. The relative frequency for both types of wound, can be put into a better perspective by comparing the graphical representation of these two forms of staphylococcal wound, with that for all wounds. When added together, the staphylococcal wounds numerically account for about one-third of all post-operative wound infections.

9.2 Relationship between duration of hospitalisation and patient risks

The influence on rates of nasal colonisation which are associated with the total time a patient spends in hospital are graphically modelled in Fig. 13. The overall trends are towards an increasing number of patients becoming colonised with tetracycline-resistant Staph. aureus over the first 7 to 8 weeks of hospitalisation, after which time, there is a marked reduction in the number of new patients becoming colonised. One possible explanation of this phenomenon, is that all patients who are likely to become colonised, are, in fact, colonised within their first 8 weeks in hospital (assuming they are actually hospitalised for such an extensive period) then all other remaining non-colonised patients are thought to have a natural resistance to nasal colonisation with this particular organism.

To give a more complete picture of the overall distribution of nasal colonisation, the results shown in Table 10 illustrate a different facet of the problem, by regrouping the data into slightly modified age-divisions, to counteract the effect of using mathematical approximation

FIGURE 13 - Total duration of hospitalisation versus rate of nasal colonisation

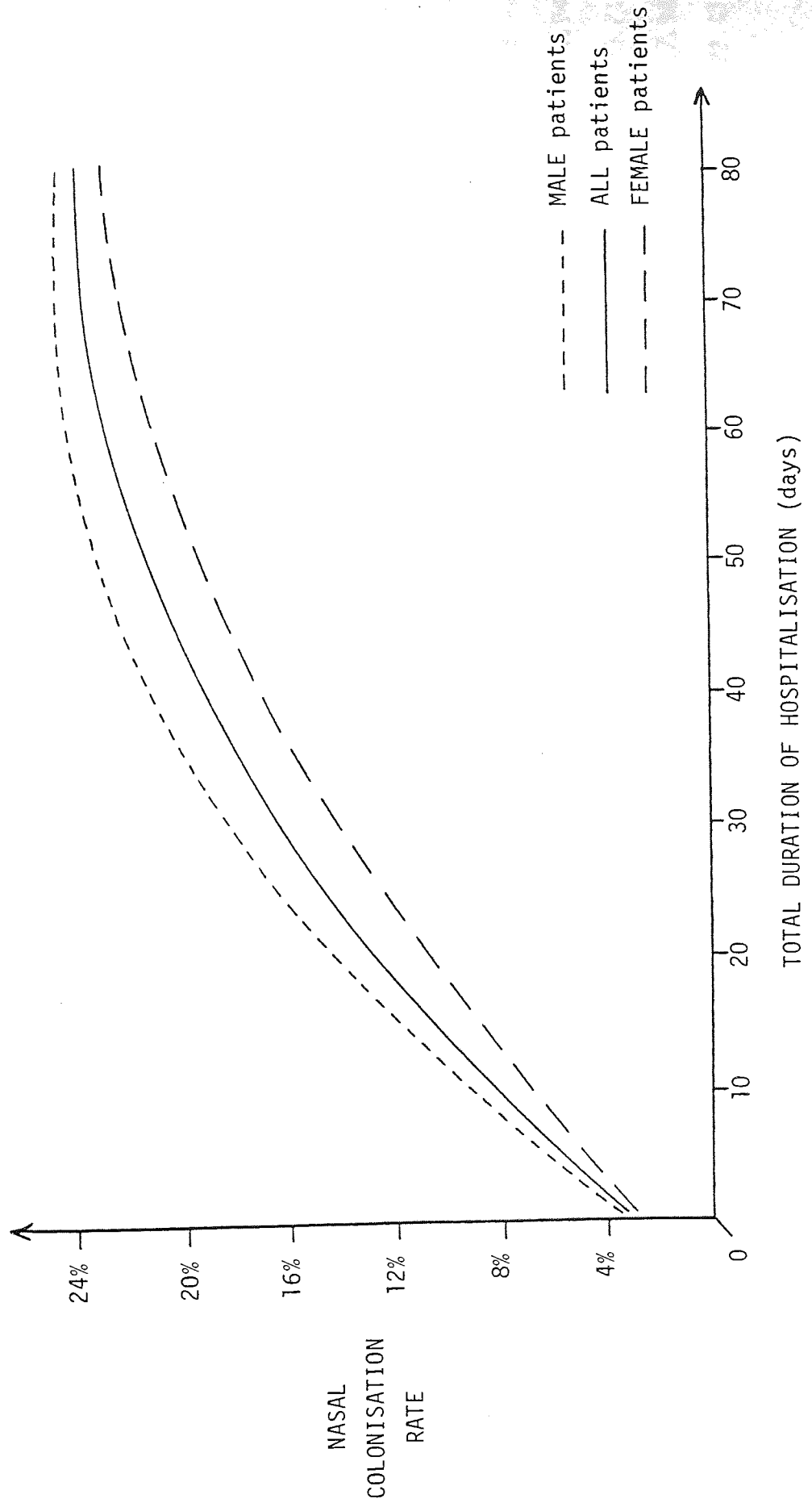


Table 10

Nasal colonisation rates connected with different TOTAL durations of HOSPITALISATION

TOTAL duration of HOSPITALISATION (days)	MALE		FEMALE		ALL patients	
	Number of patients in group	Nasal colonisation rate	Number of patients in group	Nasal colonisation rate	Number of patients in group	Nasal colonisation rate
0 - 5	1,713	3.68%	2,098	3.96%	3,811	3.83%
6 - 10	945	8.25%	1,205	4.65%	2,150	6.23%
11 - 15	593	10.79%	685	7.59%	1,278	9.08%
16 - 20	372	13.44%	365	10.96%	737	12.21%
21 - 25	241	17.01%	266	11.65%	507	14.20%
26 - 30	175	16.57%	153	14.38%	328	15.55%
31 - 35	118	18.64%	119	19.33%	237	18.99%
36 - 45	148	24.32%	163	15.95%	311	19.94%
46 - 55	78	21.79%	110	19.09%	188	20.21%
56 - 75	106	16.04%	122	19.67%	228	17.98%
Over 75	134	21.64%	264	23.11%	398	22.61%
ALL durations	4,623	9.65%	5,550	7.91%	10,173	8.70%

and smoothing procedures (as part of the modelling technique) which tends to mask a certain amount of fluctuation which occurs both between and within certain age groups. The difference in rates of colonisation between those patients receiving antibiotic treatment at the time of the survey and those who are not, is highlighted in Fig. 14. Initially, there is little difference between the two groups of patients with ONE per-cent more of the patients in the antibiotic treatment group being colonised with tetracycline-resistant Staph. aureus. However, as the total duration of hospitalisation increases beyond 10 weeks, the difference in colonisation rates rises to EIGHT per-cent.

The model showing the distribution of the relationship between wound infection and duration of pre-operative hospitalisation is illustrated in Fig. 15. As the period of pre-operative stay in hospital increases, the corresponding risk of wound infection becomes greater in an almost linear fashion, before stabilising to a constant rate, with 6% more male than female patients becoming host to a subsequent post-operative wound infection.

Again, a little of the fluctuation between certain age groups may have been masked by the modelling process, but this is recovered again in Table 11 by redefining the boundaries of the constituent age groups. The breakdown of results show that the rate of wound infection is not ever-increasing, but arrives at a plateau phase (and indeed, decreases a little for the female group, considered in isolation) after patients have spent a period of pre-operative hospitalisation exceeding 40 days.

FIGURE 14 - Influence of antibiotics on rates of nasal colonisation

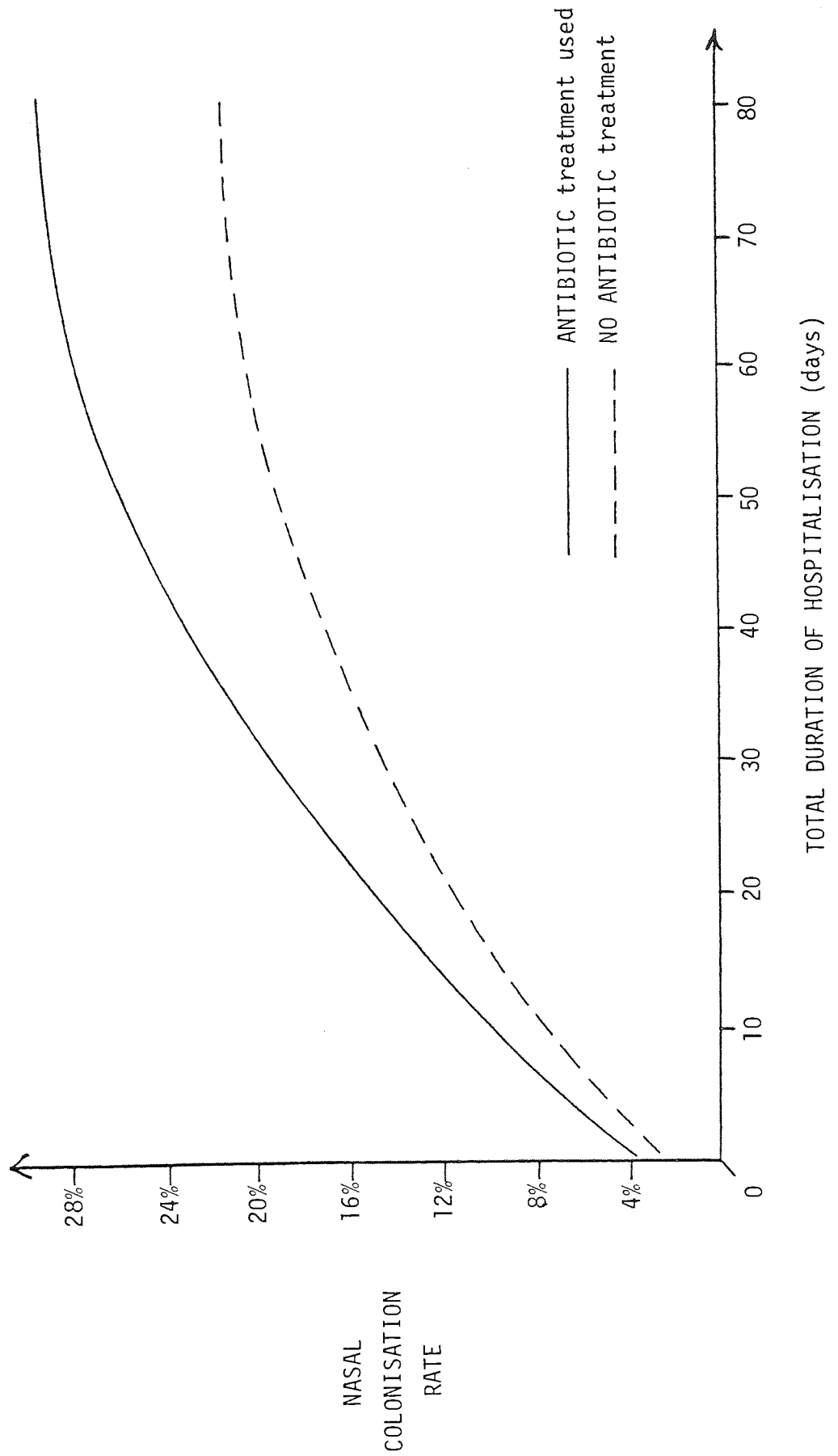


FIGURE 15 - Duration of pre-operative hospitalisation versus rate of wound infection

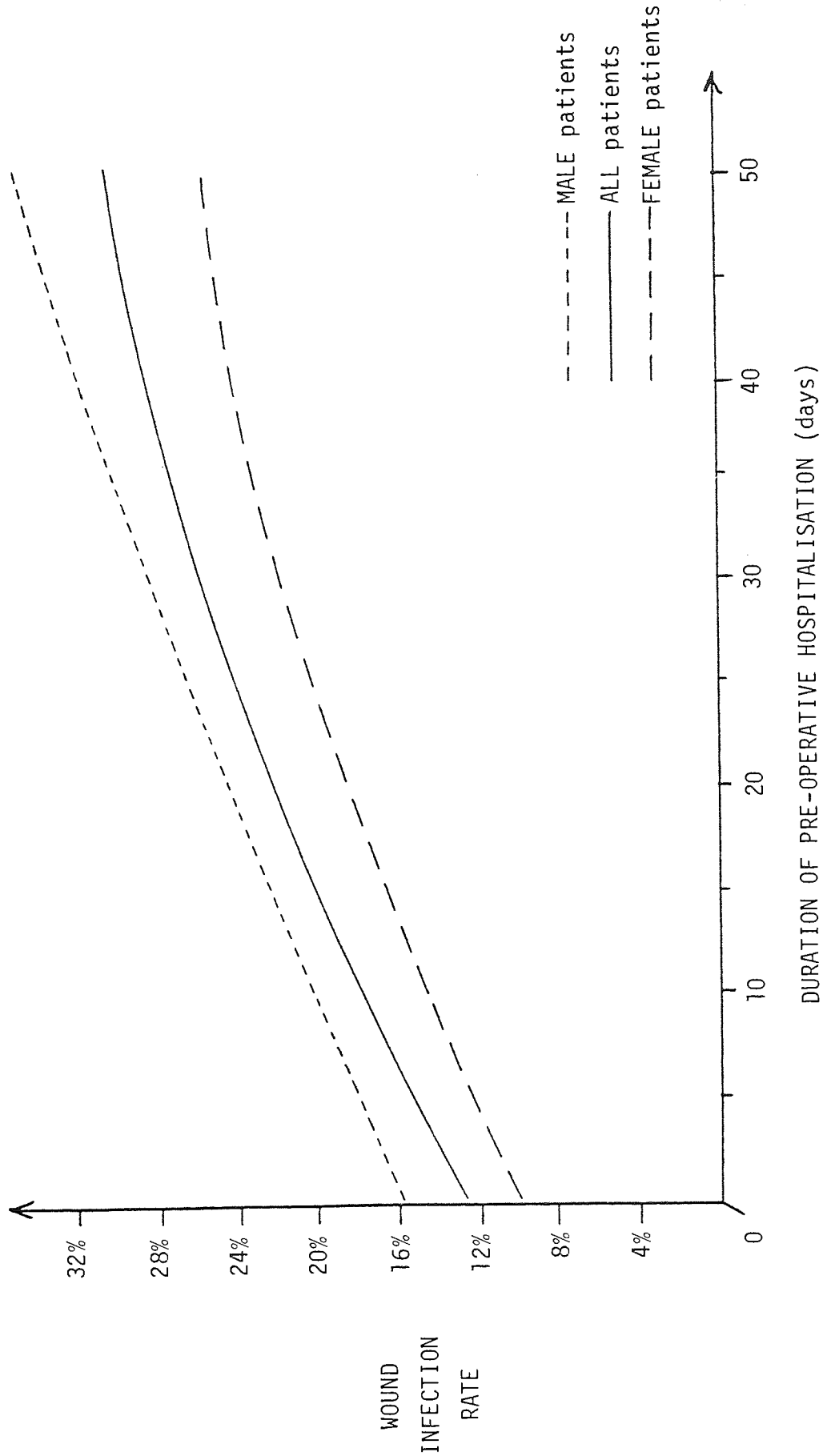


Table 11

Wound infection rates connected with different durations of PRE-OPERATIVE HOSPITALISATION

Duration of PRE-OPERATIVE HOSPITALISATION (days)	MALE		FEMALE		ALL patients	
	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate
0 - 5	1,076	16.64%	1,197	11.61%	2,273	13.99%
6 - 10	151	17.88%	157	8.28%	308	12.99%
11 - 15	78	21.79%	69	17.39%	147	19.73%
16 - 20	33	24.24%	26	15.38%	59	20.34%
21 - 25	26	34.62%	22	18.18%	48	27.08%
26 - 30	21	38.10%	22	22.73%	43	30.23%
31 - 35	17	29.41%	9	22.22%	26	26.92%
36 - 40	8	25.00%	12	33.33%	20	30.00%
Over 40	25	32.00%	31	29.03%	56	30.36%
ALL durations	1,435	18.33%	1,545	12.43%	2,980	15.27%

The effect that increasing durations of pre-operative hospitalisation have on the prevalence rates for different types of wound infection is shown in Fig. 16. Tetracycline-resistant staphylococcal wounds account for approximately the same number of post-operative infections as other forms of staphylococcal wound infections, these two together accounting for some 35.6% of all wound infections.

9.3 Influence of antibiotics

Table 12 shows the effect which various courses of antibiotics have on nasal colonisation rates. Many courses of antibiotic treatment are available, and consequently this results in many groups, with very small patient numbers in many of them. Therefore, it is only valid to compare the difference between those patients receiving no antibiotic therapy, with the group (as a whole) of patients receiving antibiotics. It is of special interest to note that some 25.5% of patients involved in the survey were in receipt of some form of antibiotic, and 4.7% of all patients were receiving more than one.

Analysis of the results derived from the extensive survey, indicate that patients receiving any form of antibiotic treatment at the time of the survey, have a risk of colonisation which is increased by a factor of 0.4, as compared with those patients not receiving any form of antibiotic. Male patients in receipt of antibiotic treatment are subject to increased colonisation risks of about one-third above that for equivalent female patients, whilst for those patients not receiving any form of antibiotic, the difference between males and females is not so great.

FIGURE 16 - Relationship between different types of wound infection

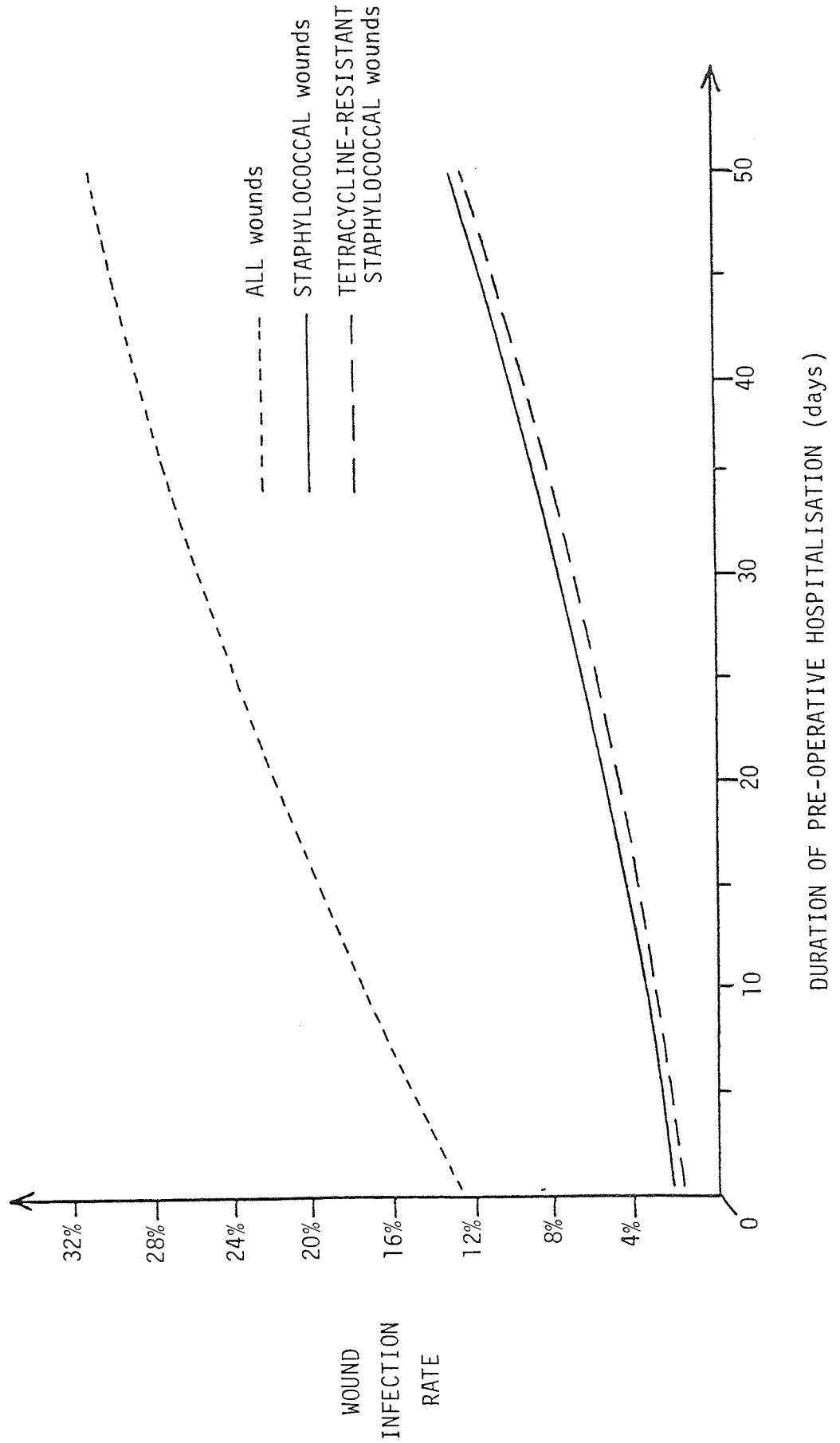


Table 12

Nasal colonisation rates associated with different courses of ANTIBIOTIC TREATMENT

ANTIBIOTIC TREATMENT	MALE		FEMALE		ALL patients		
	Number of patients in group	Nasal colonisation rate	Number of patients in group	Nasal colonisation rate	Number of patients in group	Number with nasal colonisation	Nasal colonisation rate
Penicillin	189	7.94%	186	4.84%	375	24	6.40%
Topical antibiotic	105	4.76%	44	13.64%	149	11	7.38%
Streptomycin	56	12.50%	63	7.94%	119	12	10.08%
Ampicillin	458	12.01%	427	9.13%	885	94	10.62%
Sulphonamide	34	17.65%	49	6.12%	83	9	10.84%
Cloxacillin	85	12.94%	55	9.09%	140	16	11.43%
Tetracycline	316	16.77%	264	11.36%	580	83	14.31%
Nitrofurantoin	46	23.91%	56	19.64%	102	22	21.57%
Neomycin	20	****	20	****	40	4	****
Erythromycin	18	****	12	****	30	2	****
Cephalosporins	13	****	12	****	25	2	****
Chloramphenicol	10	****	14	****	24	4	****
Gentamicin	9	****	5	****	14	4	****
Fucidic acid	4	****	2	****	6	0	****
Other antibiotics	10	****	8	****	18	1	****
ALL antibiotics	1,373	12.53%	1,217	9.53%	2,590	288	11.12%
No antibiotics	3,239	8.37%	4,320	7.43%	7,559	592	7.83%
MORE THAN ONE antibiotic	248	9.27%	229	10.04%	477	46	9.64%

**** not recorded because of the very limited number of patients in these individual groups.

No assessment relating to the effect which antibiotics have on rates of wound infection has been made, because information was not recorded as part of the prevalence survey, on the circumstances in which the antibiotics were used, i.e. prophylactically or to treat specific infections.

9.4 Distribution of different wound types

The results shown in Table 13 indicate that drained wounds are consistently linked with higher rates of infection for every category of wound. Patients with undrained clean-contaminated wounds are two-and-a-half-times more likely to acquire a post-operative wound infection than those with clean wounds, whilst the differential is reduced to two-thirds for clean and clean-contaminated drained wounds. Infection rates for contaminated wounds are double those for both drained and undrained clean-contaminated wounds. The difference between the sexes, reveals that male susceptibility is increased by about one-half over that for the female patient population.

The nasal colonisation rates shown in Table 14 have been subdivided into the same categories as those for wounds, in order to determine whether there is any link between a patient's wound type and his status of nasal colonisation. It is clear that these results follow the same trends (but on a smaller scale) as those for the rates of wound infection, but unfortunately, information is not available in respect of any particular patient's nose becoming colonised before or after operative procedures were performed.

Table 13

Infection rates for different TYPES of WOUND

TYPE of WOUND	MALE		FEMALE		ALL patients	
	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate
CLEAN, undrained	612	4.41%	765	7.06%	1,377	5.88%
CLEAN, drained	207	19.81%	300	11.67%	507	14.99%
CLEAN-CONTAMINATED, undrained	197	18.27%	166	11.45%	363	15.15%
CLEAN-CONTAMINATED, drained	250	28.80%	171	19.88%	421	25.18%
CONTAMINATED, undrained	58	37.93%	55	23.64%	113	30.97%
CONTAMINATED, drained	111	58.56%	88	42.05%	199	51.26%
ALL patients' wounds	1,435	18.33%	1,545	12.43%	2,980	15.27%

Table 14

Nasal colonisation rates associated with different TYPES of WOUND

TYPE of WOUND	MALE		FEMALE		ALL patients	
	Number of patients in group	Nasal colonisation rate	Number of patients in group	Nasal colonisation rate	Number of patients in group	Nasal colonisation rate
CLEAN, undrained	611	6.06%	762	6.04%	1,373	6.05%
CLEAN, drained	207	12.08%	299	9.70%	506	10.67%
CLEAN-CONTAMINATED, undrained	197	10.15%	166	7.23%	363	8.82%
CLEAN-CONTAMINATED, drained	250	15.60%	171	11.11%	421	13.78%
CONTAMINATED, undrained	58	13.79%	55	18.18%	113	15.93%
CONTAMINATED, drained	111	27.03%	88	19.32%	199	23.62%
ALL patients' wounds	1,434	11.09%	1,541	8.63%	2,975	9.82%

9.5 Influence of operation duration and incision length

The results shown in Table 15 reveal that the lowest rates of post-operative wound infection are associated with short operations (taking less than 90 minutes) and small wound incisions (which are less than 2.5 centimeters), whilst higher infection rates occur most often with longer, more complicated operations, during which large incisions are made. These results broadly agree with those produced in an excellent study by LIDWELL (42, 1961), who not only performed a basic analysis on several 'two-level' factors, but also accounted for situations where the predicted risk of wound sepsis could possibly become mathematically negative as a consequence of using certain combinations of factors. He suggested that such groups of patients represent rare, or non-occurring, combinations of factors, and pointed out that this apparent anomaly must be accepted as 'part and parcel' of all approximation procedures.

Within the classification for different operation durations and incision lengths, one particular group of patients (those with longer operations and wound incisions measuring between 2.5 cms and 15.0 cms), appeared not to follow the trend which had been established by the other patient groups, but this is easily accounted for, because the low number of patients in this particular group make its results statistically unreliable.

Analysis of the results for all patient wounds indicate that both duration of operation and length of incision, when considered in isolation, appear to have a significant influence on patient wound infection rates. However, when both of these factors are looked at

Table 15

Wound infection rates associated with different operation duration-incision lengths

OPERATION-INCISION CATEGORY	DRAINED wounds		UNDRAINED wounds		ALL wounds	
	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate
<90 minutes 0-2.5 cms	56	19.64%	250	6.80%	306	9.15%
<90 minutes 2.5-15.0 cms	406	27.83%	1,216	8.47%	1,622	13.32%
<90 minutes over 15.0 cms	294	18.71%	141	12.77%	435	16.78%
≥90 minutes 2.5-15.0 cms	74	14.86%	33	0.00%	107	10.28%
≥90 minutes over 15.0 cms	297	31.65%	213	15.49%	510	24.90%
ALL categories	1,127	25.20%	1,853	9.23%	2,980	15.27%

in perspective, together with other significant patient parameters, then any difference in these two variables become irrelevant with respect to building a model to mathematically represent patient wound infection rates.

9.6 Distribution of different drain types

The summary of information contained in Table 16 shows that patients with a drained wound are subject to a greater risk of wound infection by a factor of 2.7, as compared to those patients with non-drained wounds. The division into male and female patients reveals that males with drained wounds are very much more susceptible to wound infections than equivalent females, by a factor of more than half; male patients with undrained wounds still remaining more susceptible than females though the difference is not really a significant one.

A little caution should, however, be exercised when interpreting the results for individual drain types because of the small patient numbers in each group, but it is of no great surprise to find that 'Redivac' drains (which are currently the ones most commonly used) are associated with the lowest wound infection rates, and corrugated together with more than one drain, the highest.

9.7 Influence of special risk factors

The data contained in both Table 17 (for nasal colonisation) and Table 18 (for wound infection) is somewhat sparse, and consequently only the

Table 16

Wound infection rates associated with different TYPES of DRAIN

TYPE of DRAIN	MALE		FEMALE		ALL patients	
	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate
'Redivac'	208	23.56%	260	10.77%	468	16.45%
Small tube	59	28.81%	72	16.67%	131	22.14%
Wick	8	50.00%	10	10.00%	18	27.78%
Large tube	148	33.11%	96	23.96%	244	29.51%
Corrugated	113	36.28%	89	38.20%	202	37.13%
More than one drain (of different types)	32	56.25%	32	25.00%	64	40.63%
ALL drained wounds	568	31.34%	559	18.96%	1,127	25.20%
ALL undrained wounds	867	9.80%	986	8.72%	1,853	9.23%

Table 17

Nasal colonisation rates linked with different SPECIAL RISK FACTORS

SPECIAL RISK FACTOR	MALE			FEMALE			ALL patients		
	Number of patients in group	Nasal colonisation rate	Number of patients in group	Nasal colonisation rate	Number of patients in group	Nasal colonisation rate	Number with nasal colonisation	Nasal colonisation rate	
Irradiation	41	4.88%	54	1.85%	95	3	3.16%		
Diabetes	134	12.69%	197	12.18%	331	41	12.39%		
Steroids	154	15.58%	189	17.46%	343	57	16.62%		
Immuno-suppressive drugs	1	****	1	****	2	0	****		
Uraemia	1	****	0	****	1	0	****		
Obesity	4	****	17	****	21	2	****		
ALL special risk patients	335	13.13%	458	12.88%	793	103	12.99%		

**** not recorded because of the very limited number of patients in these individual groups.

Table 18

Wound infection rates linked with different SPECIAL RISK FACTORS

SPECIAL RISK FACTOR	MALE		FEMALE		ALL patients		
	Number of patients in group	Wound infection rate	Number of patients in group	Wound infection rate	Number of patients in group	Number with infected wounds	Wound infection rate
Irradiation	2	****	5	****	7	2	****
Diabetes	30	****	52	****	82	22	26.83%
Steroids	15	****	45	****	60	8	13.33%
Immuno-suppressive drugs	0	****	0	****	0	0	****
Uraemia	1	****	0	****	1	1	****
Obesity	2	****	9	****	11	8	****
ALL special risk patients	50	26.00%	111	25.22%	161	41	25.47%

**** not recorded because of the very limited number of patients in these individual groups.

influence caused by the presence or absence of special patient risks (consisting primarily of diabetes and use of steroids) can be detected. Special risks increase a patient's chance of acquiring nasal colonisation with tetracycline-resistant Staph. aureus by a factor of 0.56 as compared with those patients not subject to the detrimental side effects caused by them. Operated patients are also subject to a similar difference in risks, and for the special risk patients, an even larger differential factor of 0.73 applies, over and above that for patients to whom special risk factors are not applicable. Any difference between the sexes is minimal and of no special note, except to point out that males again are marginally more susceptible than their female counterparts.

9.8 Pure risk profiles

Having assessed how nasal colonisation and wound infection rates are affected by age, sex, and so on, the next logical progression is to look simultaneously at the influence which more than one variable parameter may have, with respect to patient risks. In order to check for unusual trends, or indeed apparent discontinuities, a net has been superimposed over each of the upper response surfaces for the various graphical representations of multi-dimensional risk profile models.

The number of patients colonised with tetracycline-resistant Staph. aureus grows at a slow but ever-increasing rate with advancing age. The rate of nasal colonisation also increases as the length of time spent in hospital grows but the increase lessens until the rate

becomes almost constant. The resultant effect of this combination of factors, being the slightly twisted profile for nasal colonisation which is shown in Fig. 17, with 1.8% more male than female patients ultimately becoming colonised with multi-resistant strains of staphylococci.

Figure 18 shows the profile of all post-operative wound infections, and how the rates are affected for patients of different ages, sex, and with differing durations of pre-operative hospitalisation. The response surfaces sweep gently upwards, with the minimum risks being associated with very young patients having short pre-operative stays in hospital, whilst the maximum number of infections occur for the group of very aged patients who have had extensive periods of pre-operative hospitalisation. The difference in infection rates for the male and female groups remains constant at 6.2% throughout the entire age range and pre-operative periods of hospitalisation.

The infection rates for just those patients who have wounds infected with staphylococci are graphically represented in Fig. 19, where the overall difference between the infection rate for the male and female patient groups is reduced to 2.2%. The infection profile is also very much altered, with young female patients who had short periods of pre-operative hospitalisation having an almost zero wound infection rate, rising much more steeply to a maximum of 12.8% for elderly male patients who spent extended pre-operative durations in hospital.

The difference between the infection rate for all wounds, and just those for staphylococcal wounds must be put into perspective, which is done with the aid of Fig. 20. The differences between the two distributions can be accounted for by the fact that only a small

FIGURE 17 - Colonisation profile for all patients' noses

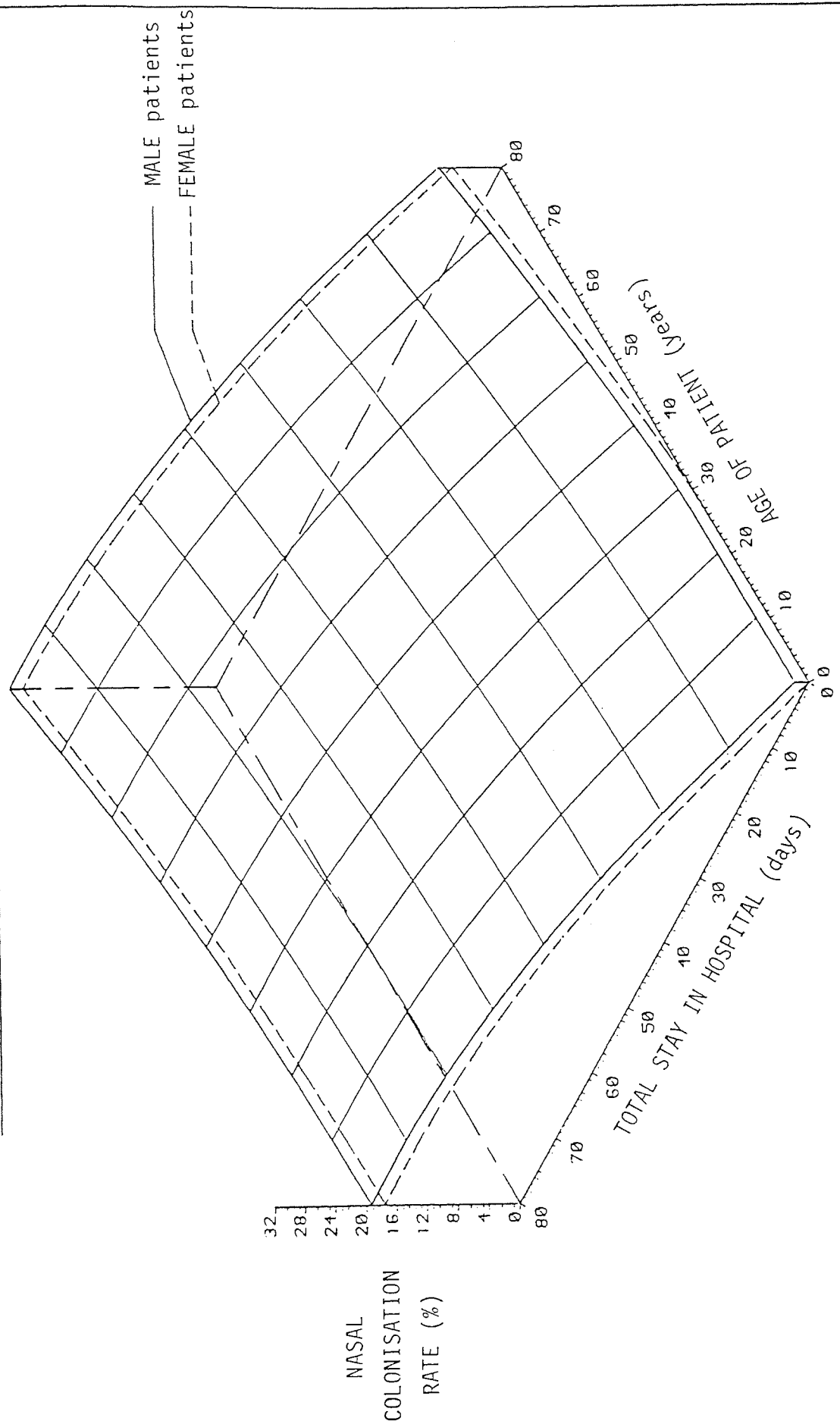


FIGURE 18 - Infection profile for all patients' wounds

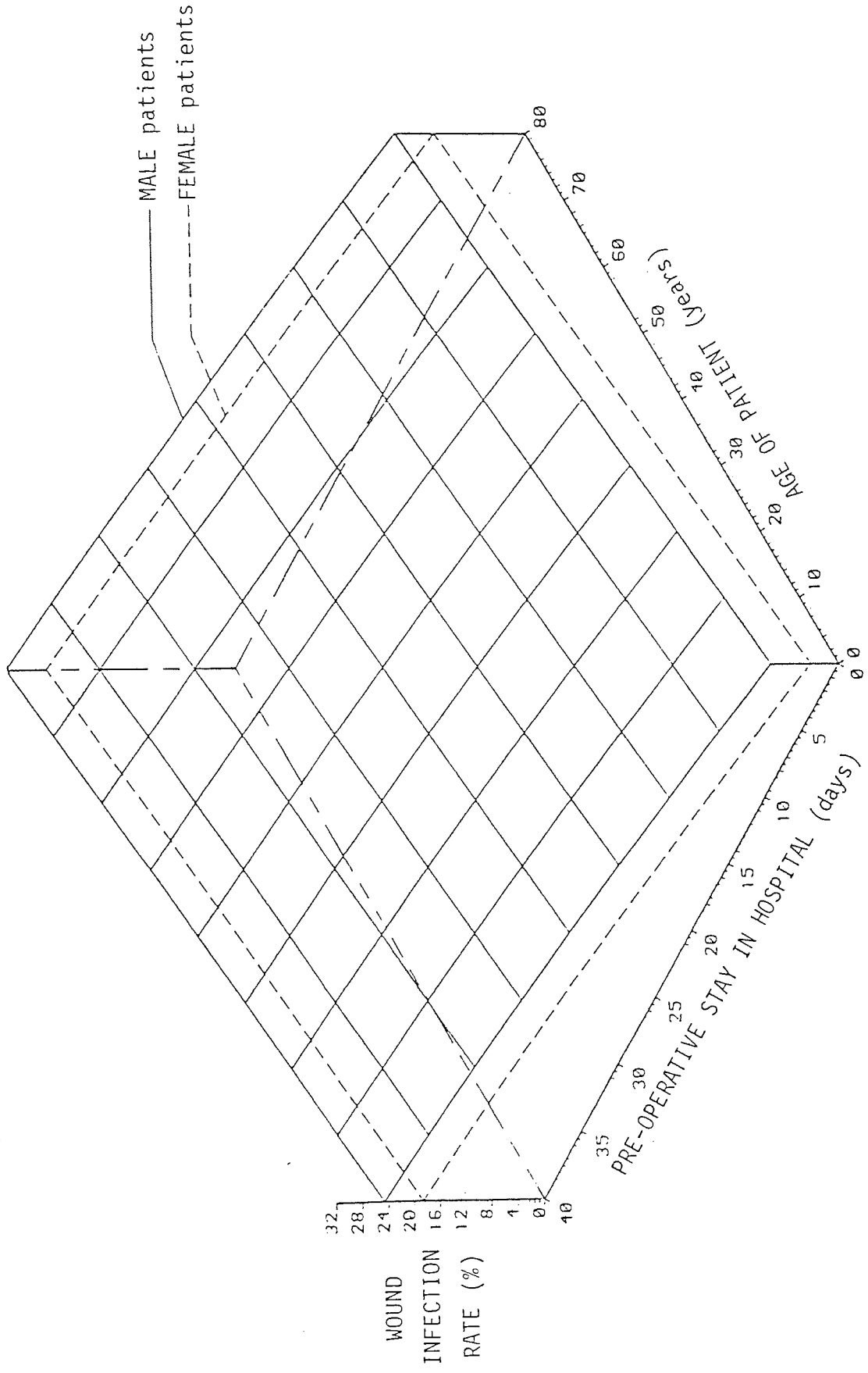


FIGURE 19 - Infection profile for patients' wounds infected with staphylococci

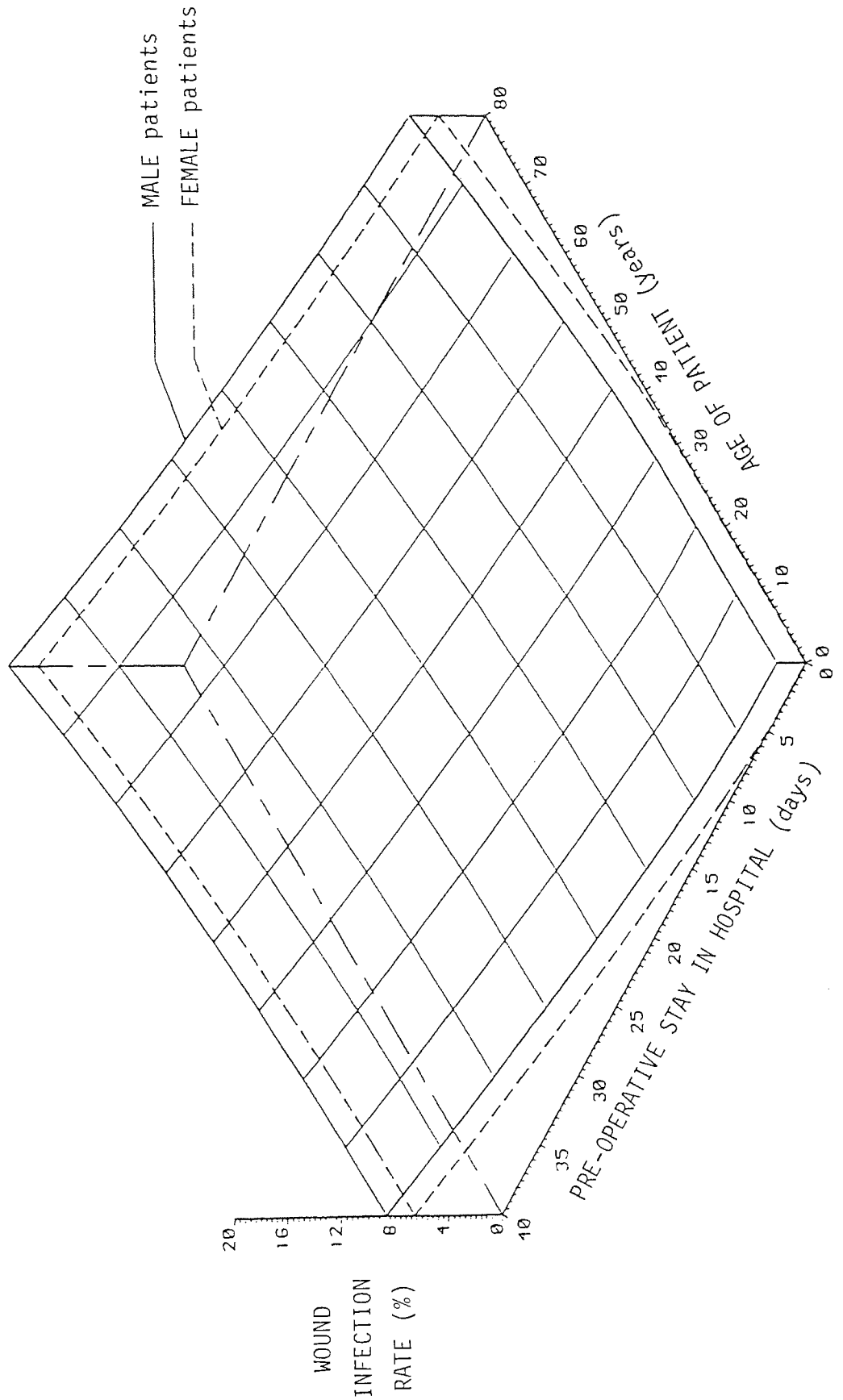
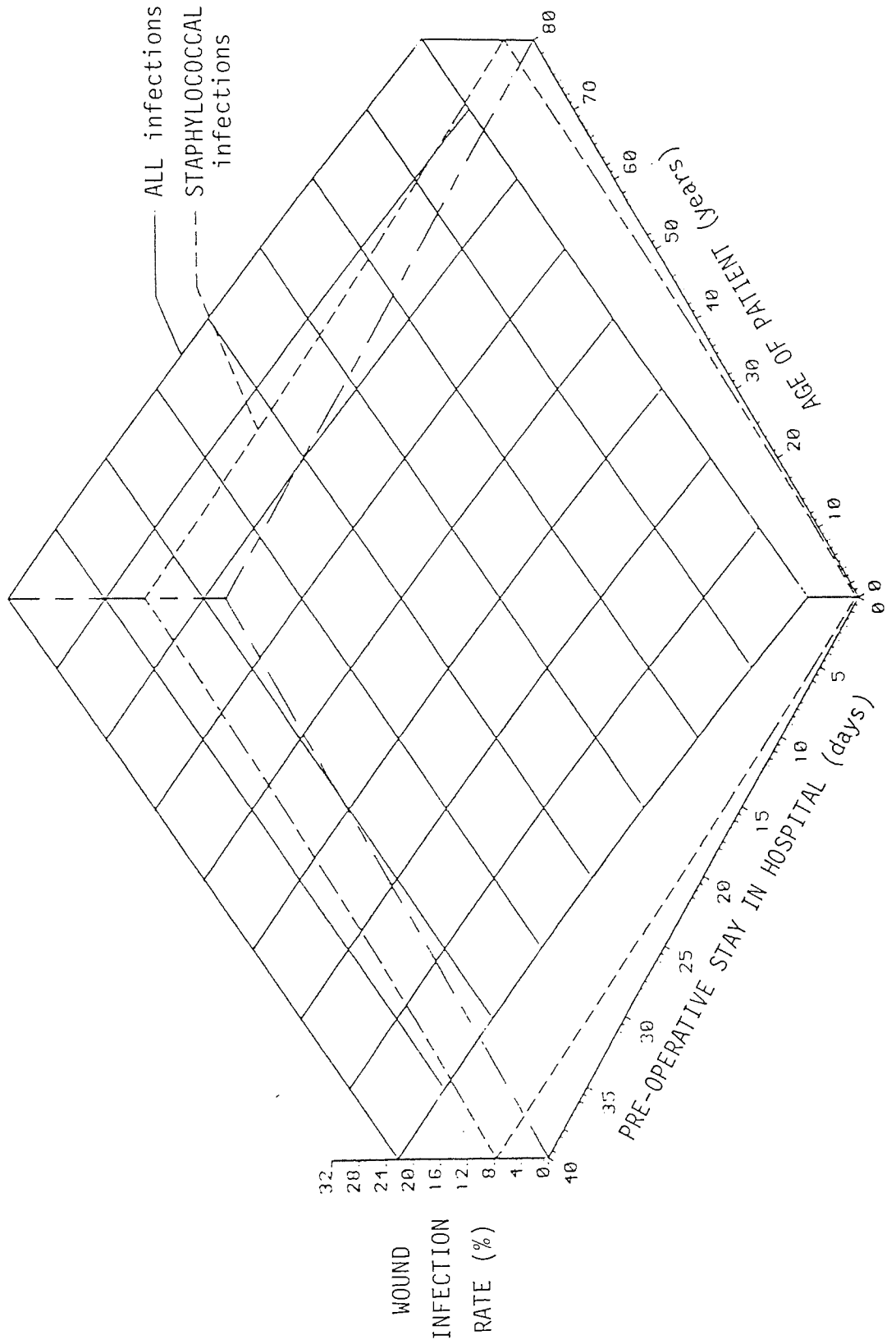


FIGURE 20 - Infection profile for patients' wounds



proportion of the patients acquire a staphylococcal infection in wounds, specifically there are only 161 as compared with the sum total of all wound infections, which number 455. Hence, despite the fact that the infection profile for staphylococcal wounds rises more rapidly than that for all wound infections, it only reaches a peak of 11.6% as compared with 32.0% for all wound infections.

9.9 Partitioned risk profiles

The colonisation profile shown in Fig. 21 has been subdivided into those patients who have undergone operations, and the patient population as a whole. The main outstanding features are that initially, operated patients have lower risks than the patient population as a whole for total durations of hospitalisation which correspond to patient ages which are less than 30 years, after which age the risk of nasal colonisation increases rapidly for operated patients with longer durations of total stay. However, for short durations of hospitalisation, the colonisation rates only begin to overtake that for all patients some 20 years later, at the age of 50 years and beyond.

The colonisation results for operated patients have been split into patients with non-infected wounds, and those with wounds which become post-operatively infected. The difference in the colonisation profiles shown in Fig. 22 is a little unusual insofar as at the age/duration of hospitalisation datum, the probability of colonisation is close to zero for those patients with non-infected wounds, and around 7.2% for the group of patients with an infected wound. In the age/colonisation plane, the difference in risks rises from an initial 7.2% to an upper limit of 12.8% for patients at the age of 80 years. In the total

FIGURE 21 - Colonisation profile for different types of patient

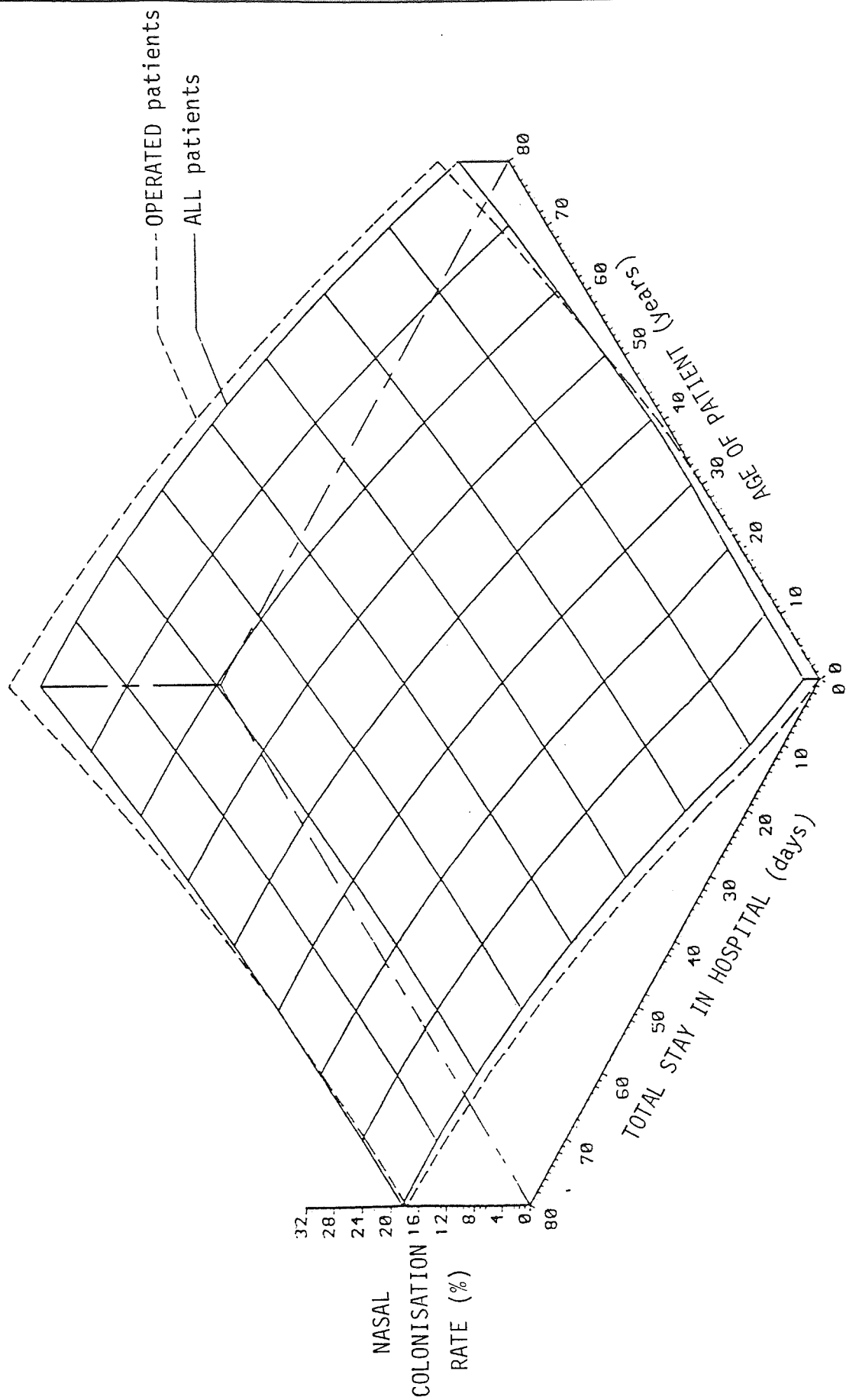


FIGURE 22 - Colonisation profile for different types of patient wounds

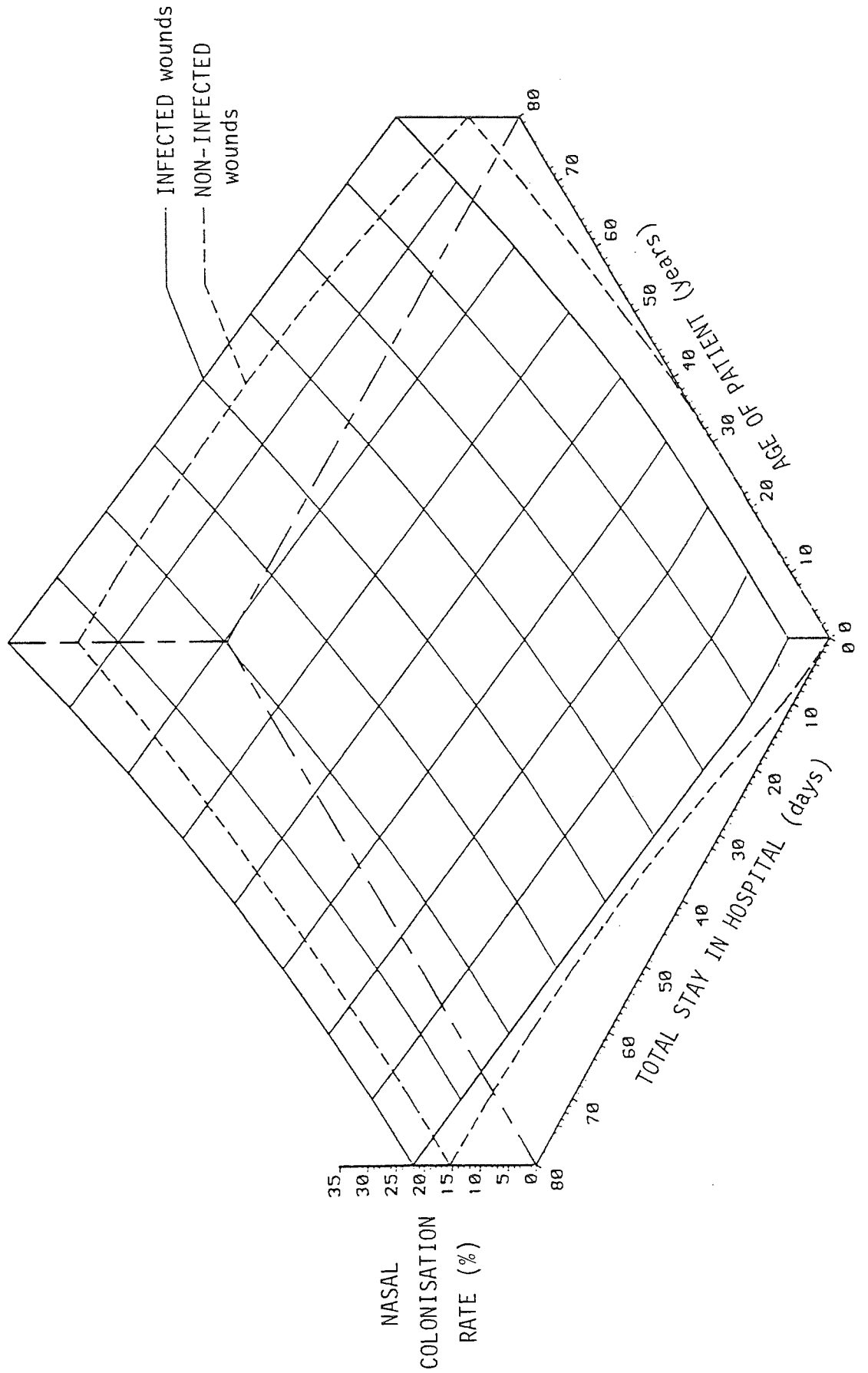
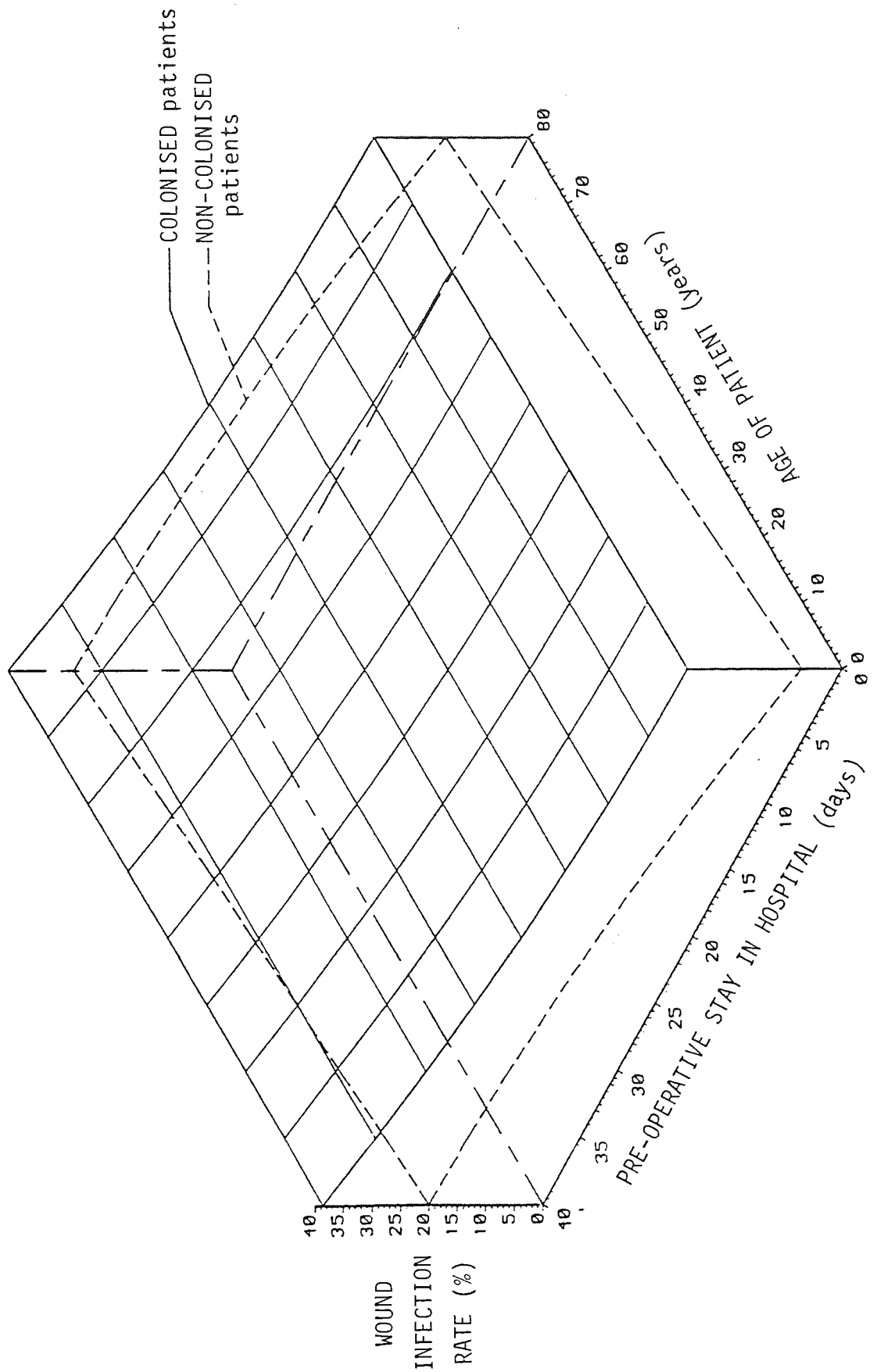


FIGURE 23 - Infection profile for different types of patient



duration of hospitalisation/colonisation plane both the initial and final differences in the colonisation rates are close to 7.2%, whilst in the middle of the duration of hospitalisation range (at around 40 days), the difference drops to only 2.8%.

Figure 23 represents the infection profile for patients who are, and those who are not, subject to nasal colonisation with tetracycline-resistant Staph. aureus. It is interesting to note that for the group of non-colonised patients, the risk of wound infection rises very slowly but uniformly with the advancement of age, whereas, for patients with differing durations of pre-operative hospitalisation, the initial probabilities of wound infection start at 0.07 rising to a peak of 0.20 (after 40 days of pre-operative stay in hospital). When such a long pre-operative stay is combined with an advanced age of 80 years and over, the probability of nasal colonisation rises dramatically to nearly 0.28. For the group of patients with infected wounds, the minimum risk of nasal colonisation is initially a very high 27.0% (at the age/pre-operative hospitalisation datum), rising to 38.6% as the amount of time spent by the patient in hospital prior to operative procedures being performed rises beyond 40 days. Any variations in the age of the patients, does not appear to significantly influence the risk of infection for those patients colonised with tetracycline-resistant Staph. aureus.

CHAPTER 10

STEPWISE REGRESSION PROCEDURE

10.1 Computational method for stepwise regression procedure

Multiple regression analysis is used to obtain the model of best fit, pertaining to a set of observations of independent and dependent variables, which is of the form:-

$$y_i = b_0 + b_1x_{1i} + b_2x_{2i} + \dots + e_i$$

where y_i is the dependent (or response) variable for the i^{th} set of observations.

x_{1i}, x_{2i}, \dots are the independent variables

b_0, b_1, \dots are the regression coefficients to be determined.

e_i is the error term for the i^{th} set of observations.

A multiple regression solution gives the 'least squares best fit' for the particular data sample analysed. The solution also gives a measure of the reliability for each of the coefficients, in order that conclusions may be drawn regarding the population from which the observations were taken.

Multiple regression analysis may also be used to fit more complicated non-linear equations of the form:-

$$y_i = b_0 + b_1x_{1i} + b_2x_{1i}^2 + b_3(x_{1i}x_{2i}) + \dots + e_i$$

where appropriate substitutions will recover the format of the original regression equation.

For problems which involve a large number of variables, any method of regression analysis solution requires a large number of calculations, which makes the problem far too complex for the limited capacity of a desk calculator. It is under these circumstances, that we seek an efficient regression method which is able to cope with a large number of variables, whilst still remaining compatible with the logic available in current high-speed digital computers. It is with these constraints in mind that we turn to STEPWISE REGRESSION as the best means of solution, when efficiently programmed for use on a digital computer.

Use of the stepwise procedure means that intermediate results, which are not usually recorded by the normal methods of calculation, are available at each step in the calculations, to give us very valuable statistical information, on the effect that adding or deleting variables has on the regression model. The intermediate results are also of great benefit in determining the method of calculation for the 'next' or 'following' step in the procedure. Without adding greatly to the number of arithmetic calculations, we have at our disposal, not only the complete multiple regression model, but also all the preceding models which were derived during the intermediate processes. The equations for each of the regression models are obtained by adding (or deleting) one variable at a time. If, for example, variables were only to be added (for the first few steps), the intermediate regression equations may well appear in the form:-

$$y = b_0 + b_1x_1$$

$$y = b'_0 + b'_1x_1 + b'_2x_2$$

$$y = b''_0 + b''_1x_1 + b''_2x_2 + b''_3x_3$$

. . .
. . .
. . .

The variable being added at each stage, is that which produces the greatest improvement in 'goodness of fit'. The coefficients attached to each of the respective x - variables, represent the best value, when the model is fitted by the specific variables currently included in the regression equation.

Two important properties of the stepwise procedure are:-

- a) A variable may be found to be significant at an early stage, and so enter the model at that time.

- b) After several other variables have been added to the regression equation, the initial variable may then be found insignificant. The facility then exists to remove the insignificant variable from the regression equation, before proceeding to add any additional variables.

Hence, only significant variables remain in the final regression model.

10.2 Mathematical discussion for the stepwise regression procedure

A) Mathematical symbols used

- n - number of independent + dependent PATIENT and WARD parameters
- m - number of patients whose records have been analysed in the survey
- x_{it} - t^{th} observation of the i^{th} variable
- x_{nt} - $y_t = t^{\text{th}}$ observation of the dependent (or response) variable
- \bar{x}_i - MEAN of the i^{th} variable
- s_{ij} - RESIDUAL sum of squares and CROSS PRODUCTS for the i^{th} and j^{th} variables
- σ_i - $\sqrt{s_{ii}}$
- r_{ij} - simple correlation coefficient of i^{th} and j^{th} variables
- c_{ij} - element of inverse matrix of r_{ij}
- N_{max} - subscripts of selected independent variables
- V_{max} - reduction in variance caused by adding the independent variable corresponding to N_{max}
- To1 - TOLERANCE LIMIT, below which value, each a_{ij} leading diagonal elements (corresponding to the independent variables) may not fall below, because of possible degeneracy in the calculation of V_i terms
- β_i - TRUE value for the coefficient of the i^{th} variable

- b_i - ESTIMATED coefficient of the i^{th} variable
- S_y - STANDARD ERROR of the DEPENDENT (or RESPONSE) variable
- S_{b_i} - STANDARD ERROR for the coefficient of the i^{th} INDEPENDENT variable
- \hat{y}_t - PREDICTED value of the DEPENDENT (or RESPONSE) variable for the t^{th} observation
- F_1 - critical F- value for a variable ENTERING the regression model
- F_2 - critical F- value for DELETING a variable from the regression model

B) Derivation of the method

Let y be estimated from the equation;

$$y_t = \bar{y} + \sum_{i=1}^{n-1} \beta_i (x_{it} - \bar{x}_i) \quad (t=1,2, \dots, m)$$

The estimate of the t^{th} observed value of y , has error

$$e_t = (y_t - \bar{y}) - \sum_{i=1}^{n-1} \beta_i (x_{it} - \bar{x}_i) \quad (t=1,2, \dots, m)$$

The object of regression analysis is to determine a set of β_i such that the magnitude of the vector $e = [e_t]$ is minimised. Now,

$$\|e\|^2 = [e, e] = \sum_{t=1}^m \left\{ (y_t - \bar{y}) - \sum_{i=1}^{n-1} \beta_i (x_{it} - \bar{x}_i) \right\}^2$$

The error term can be minimised by partially differentiating the above equation with respect to one of the β_i terms and equating the result to ZERO, which gives,

$$\sum_{j=1}^{n-1} \left\{ \sum_{t=1}^m (x_{it} - \bar{x}_i)(x_{jt} - \bar{x}_j) \right\} \beta_j = \sum_{t=1}^m (x_{it} - \bar{x}_i)(y_t - \bar{y})$$

These $(n - 1)$ simultaneous linear algebraic equations in β_j , together form the NORMAL equations, and can be solved by any convenient technique such as the GAUSSIAN ELIMINATION METHOD. Choice of this particular method (when applied to the regression problem), means that we can not only produce the final solution, but also, each stage in the elimination procedure yields a PARTIAL REGRESSION EQUATION. All the variables which have already been eliminated by application of the Gaussian method are in the regression equation, whilst all the other remaining variables are not. We can make use of this valuable piece of information, in deciding which variable will be the next to enter into the regression model.

The technique involved in the Gaussian elimination procedure, is to apply linear transformations to the following PARTITIONED matrix.

$$\begin{bmatrix} S & T' & I \\ T & Z & D \\ -I & B & C \end{bmatrix} \dots \dots \dots (1)$$

where,

S , C and I are $(n - 1) * (n - 1)$ matrices,

T and D are $1 * (n - 1)$ matrices,

B is an $(n - 1) * 1$ matrix, and,

Z is a scalar.

Specifically the contents of the matrices are as follows:-

$$[S]_{ij} = s_{ij} = \sum (x_{it} - \bar{x}_i) (x_{jt} - \bar{x}_j)$$

$$[T]_{1j} = t_{1j} = \sum (x_{jt} - \bar{x}_j) (y_t - \bar{y})$$

$$Z = \sum (y_t - \bar{y}) (y_t - \bar{y})$$

or $Z = \sum (x_{nt} - \bar{x}_n) (x_{nt} - \bar{x}_n)$ (where $x_{nt} = y_t$)

$$[T]_{i1} = [T]_{1i}$$

$$[B] = [C] = [D] = 0 \text{ (initially)}$$

$$[I]_{ij} = \delta_{ij}$$

where δ_{ij} is the Dirac Delta function, taking the values:

$$\delta_{ij} = 0 \quad (i \neq j)$$

$$\delta_{ij} = 1 \quad (i = j)$$

i.e. $I =$ identity matrix

$-I =$ negative identity matrix

Any LINEAR TRANSFORMATIONS will cause some non-zero elements to enter into the sub-matrices B, C and D.

Each successive row elimination applied to the S matrix adds one MORE variable to the regression equation. Application of the same algorithm to eliminate a row in the C matrix results in a regression equation with one LESS variable.

At every stage in the procedure, all the regression coefficients are stored in the B matrix, whilst the C matrix contains the INVERSE of the partitioned part of the S matrix, corresponding to those variables in the regression model at the current stage.

The selection criteria used, when either adding or deleting a variable x_j from the regression equation is as follows:-

- i) Any variable is removed from the regression equation, if the PARTIAL-F value corresponding to that variable, is insignificant at a predetermined critical F - value. If, however, NO variable is to be removed, then we proceed to examine the criteria (shown in ii) for adding a new variable into the regression model.
- ii) Any variable is added to the regression equation, if the VARIANCE REDUCTION achieved by adding that variable, is significant at a predetermined critical F - value.

The form of partitioned matrix given in equation (1) could be used directly, but to assist with efficient digital computing techniques, the S, T, T' and Z matrices (which together form the R matrix) are normalised to obtain UNITY in the diagonal elements. These elements can be transformed to simple correlation coefficients by using the formulae;

$$r_{ij} = \frac{\Sigma(x_{it} - \bar{x}_i)(x_{jt} - \bar{x}_j)}{\sqrt{\Sigma(x_{it} - \bar{x}_i)^2 \Sigma(x_{jt} - \bar{x}_j)^2}}$$

$$\text{or if, } \sigma_i = \sqrt{\Sigma(x_{it} - \bar{x}_i)^2}$$

$$\text{then, } r_{ij} = \frac{\Sigma(x_{it} - \bar{x}_i)(x_{jt} - \bar{x}_j)}{\sigma_i \sigma_j}$$

C H A P T E R 11

SUMMARY OF CALCULATIONS USED IN THE STEPWISE REGRESSION PROCEDURE

11.1 Selection of the key element

All information was initially subjected to a preliminary scan on the computer to check for accuracy and consistency, details of this procedure being shown in Appendix E.

The logic used in selecting a_{kk} , the key element for generating each new matrix is contained in Appendix F, where the modelling technique is shown in detail. For every $a_{ij} > \text{Tol}$, V_i terms are calculated as:-

$$V_i = \frac{a_{in} a_{ni}}{a_{ij}}$$

Control on the size of the diagonal elements a_{ij} , reduces the possibility of degeneracy, which may occur if an independent variable proves to be a linear combination of two or more other independent variables. If the multiple correlation coefficient between a number of (what were thought to be) independent variables, is so large, that most of the variability in one INDEPENDENT variable is related to the other variables, then that variable will not be placed in the regression model.

The criteria used to select the x_j variable which is to enter or leave the regression equation are as follows:-

i) If the partial F-value calculated from,

$$F_{\text{calc}} = \frac{\phi a_{in}^2}{a_{nn} a_{pp}}$$

is LESS than a predetermined critical F-value, then the corresponding x_i variable is removed from the regression model. This rule, it should be noted, takes priority over that for adding a variable. The general algorithm used to generate the succeeding matrix (after a variable has been REMOVED from the regression model) is then as follows:-

$$\text{The new } a_{ij} = \begin{cases} a_{qj} / a_{pp} & \text{if } i = q \\ \frac{a_{ij} a_{pp} - a_{ip} a_{qj}}{a_{pp}} & \text{if } i \neq q \end{cases}$$

(where a_{pp} is the INVERSE diagonal element for the i^{th} variable . . .
i.e. $p = q + n$)

ii) The x_i variable corresponding to the maximum V_i is added into the regression model, providing V_i is positive and the variance reduction caused by adding x_i is significant. The test statistic for the variance reduction is of the form,

$$F_{\text{calc}} = \frac{(\phi - 1)V_{\text{max}}}{(a_{nn} - V_{\text{max}})}$$

and, providing this is GREATER than a predetermined critical F-value, then the variance reduction is deemed to be significant. If these conditions are all fulfilled, then the general algorithm used to

generate the succeeding matrix (after a variable has been ADDED to the regression model) is then as follows:-

$$\text{The new } d_{ij} = \begin{cases} a_{kj} / a_{kk} & \text{if } i = k \\ \frac{a_{ij} a_{kk} - a_{ik} a_{kj}}{a_{kk}} & \text{if } i \neq k \end{cases}$$

(where a_{kk} is a diagonal element, k corresponding to the independent variable being added to the regression equation).

11.2 Calculation of regression coefficients and standard deviations

In addition to the matrix elements a_{ij} (which are recalculated at every stage of the stepwise regression procedure), we also have at our disposal ϕ (the DEGREES OF FREEDOM), the MEAN of each X_i variable, and the standard deviations X_i (which are used to obtain the correlation coefficients). Hence, from this stored information, at the end of every step we can calculate:-

1) Standard error of the dependent variable

The standard error of y at the end of every step is given by,

$$S_y = \sigma_n \sqrt{r_{nn} / \phi}$$

where $x_n = y$, is the dependent variable

2) Calculation of the REGRESSION COEFFICIENTS

At the end of every step, the regression coefficients are calculated as follows:-

$$b_i = b_{in} \frac{\sigma_n}{\sigma_i}$$

where $x_n = y$, is the dependent variable

x_i is a variable in the regression at the current stage.

The CONSTANT in the regression equation is calculated at the end of every step from,

$$b_0 = \bar{y} - \sum b_i \bar{x}_i$$

3) Calculation of STANDARD ERRORS for REGRESSION COEFFICIENTS

The standard errors for each of the regression coefficients (corresponding respectively to those variables in the regression model at the end of the current step) are calculated as follows:-

$$S_{bi} = \frac{S_y}{\sigma_i} \sqrt{c_{ii}}$$

where c_{ii} is a diagonal element from the inverse matrix of r_{ij} .

11.3 Calculation of the square of the multiple correlation coefficient

The square of the multiple correlation coefficient R^2 , is defined in the following manner:-

$$R^2 = \frac{\text{(sum of squares due to REGRESSION } | b_0 \text{)}}{\text{TOTAL (corrected) sum of squares}}$$

It is, however, more usual to see the quantity $100 R^2$ per cent, which gives an indication of how well the regression model fits the particular

set of data under consideration. Larger values of $100 R^2$ indicate better fitting models, since a greater amount of the variation between the data samples can be accounted for.

11.4 Calculation of predicted values for the dependent variable and deviations between 'actual' and 'predicted' values

The final calculation in the stepwise regression procedure, is to predict the value of the dependent (or response) variable for each set of observations, based on the final regression model (equation). The deviation between ACTUAL and PREDICTED values of the dependent variable can be calculated for each set of parameters, but great CAUTION should be taken when applying this comparison to individual patients within a hospital ward, because the dependent variable (in practice) only takes the value 0 or 1 corresponding to a patient being either 'not infected' or 'infected'. Greater flexibility, however, can be exercised with the results obtained by considering the 'sum total' of patients' infection rates from any given ward. For this ward, we would produce PREDICTED ward infection (or colonisation) rates, which could then be compared with the ACTUAL ward infection (or colonisation) rates.

11.5 Results derived from the stepwise regression procedure

Taking into account the results concerning the relevance of many ward structures, practices and procedures (discussed in Chapters 5, 6 and 7), application of the computer-based multiple regression procedure

gives rise to two completely independent mathematical models, one simulating nasal colonisation and the other, wound infection.

Repeated application of the computerised stepwise regression procedure yields an analysis of variance table, which contains all those variable patient and ward parameters which were found to have a significant influence on patient nasal colonisation rates with tetracycline-resistant Staphylococcus aureus. A simplified form of this information is shown in Table 19.

Table 19

Analysis of variance for the nasal colonisation model

Source of variation	Degrees of freedom	Sum of squares	Mean square	Calculated F-value	100R ²
Total	10173	885.000			
Mean	1	76.991			
Total (corrected for mean)	10172	808.009			
Regression	8	40.898	5.112	67.736	5.06%
Residual	10164	767.111	0.075		

For this model, predicting nasal colonisation rates, the critical F-value was arbitrarily set to a value of 1.96 for both entry and deletion of any variable, so that the necessity to consult tables of F-values at each and every stage of the analysis could be dispensed with, since this would have been impracticable to implement, despite the use of a high speed electronic computer. With these criteria in

mind, the following statistical regression model was produced in order to simulate the probability of any patient (or group of patients) becoming COLONISED with tetracycline-resistant Staph. aureus:-

$$P_i = 0.0042 + (0.00001004 \times \text{AGE}^2) + (0.00475 \times \text{TOTAL LENGTH of STAY}) - (0.000031 \times \text{TOTAL LENGTH OF STAY}^2) + (0.05175 \times \text{NUMBER of PATIENTS in WARD} \div \text{NUMBER of BEDS in WARD}) - (0.00812 \times \text{AVERAGE DISTANCE between BED CENTRES}) + \text{SEX}^{\textcircled{a}} + (\text{ANTIBIOTIC TREATMENT})^{\textcircled{aa}} + (\text{SPECIAL RISK FACTORS})^{\textcircled{aaa}}$$

Where

@	{	0.0158, MALE
	}	0.0, FEMALE

@@	{	0.0226, if patient has received any form of ANTIBIOTIC treatment
	}	0.0, OTHERWISE

@@@	{	0.01925, if SPECIAL RISK factors are applicable
	}	0.0, OTHERWISE

Now E_i , the expected frequency of patients colonised with tetracycline-resistant Staph. aureus, in Group 'i' is calculated from:-

$$E_i = \text{SUM of all } P_i \text{ (corresponding to nasal colonisation) in group 'i'}$$

Additional information on those parameters which were found to have a significant influence on patient colonisation rates, can be broken down into the following categories:-

- . Mean for each parameter
- . Standard deviation for each parameter about its mean

Table 20

Summary of statistical information on parameters relevant to nasal colonisation rates

VARIABLE	MEAN	STANDARD DEVIATION	PARTIAL F-VALUE	REGRESSION COEFFICIENT	STANDARD ERROR
DURATION of OPERATION	15.671	21.043	144.983	0.004753	0.0003948
AGE (squared)	2834.860	2124.727	55.781	1.004×10^{-5}	0.134×10^{-5}
LENGTH of STAY (squared)	688.336	1894.128	52.046	-3.131×10^{-5}	0.434×10^{-5}
AVERAGE DISTANCE between BED CENTRES	7.248	1.234	13.411	-0.008119	0.002217
ANTIBIOTIC USE	0.268	0.443	13.181	0.022599	0.006225
SEX of patient	0.454	0.498	8.232	0.015788	0.005503
PROPORTION of BEDS OCCUPIED (NUMBER of PATIENTS ÷ NUMBER of BEDS)	0.879	0.148	7.711	0.051749	0.018636
SPECIAL RISK FACTORS	0.078	0.269	3.536	0.019246	0.010235

- . Partial F-value for each parameter, given that all other relevant parameters are already in the regression model
- . Regression coefficient for each of the parameters
- . Standard error for each of the respective regression coefficients

A summary of these results for the nasal model are shown in Table 20.

The analysis of variance table specifically concerned with wound infection rates for operated patients is shown in Table 21.

Table 21

Analysis of variance for the wound infection model

Source of variation	Degrees of freedom	Sum of squares	Mean square	Calculated F-value	100R ²
Total	2980	455.000			
Mean	1	69.471			
Total (corrected for mean)	2979	385.529			
Regression	13	52.545	4.042	36.003	13.63%
Residual	2966	332.984	0.112		

For this model, predicting wound infection rates, the critical F-value was arbitrarily set to a value of 1.67. With these criteria borne in mind, the following statistical regression model was produced in order to simulate the probability of any patient (or group of patients) becoming host to a post-operative wound infection.

Specifically, the probability of any patient having a WOUND INFECTION is given by:-

$$P_i = 0.0004 + (0.0000063 \times \text{AGE}^2) + \text{SEX}^f + (0.00176 \times \text{DURATION of PRE-OPERATIVE STAY}) + (\text{CATEGORY of WOUND})^{ff} + (\text{TYPE of DRAIN})^{fff} + (\text{SPECIAL RISK FACTORS})^{ffff} + (0.0000278 \times \text{NUMBER OF OCCUPIED BEDS}^2)$$

Where

$$f \begin{cases} 0.0421, & \text{MALE} \\ 0.0, & \text{FEMALE} \end{cases}$$

$$ff \begin{cases} 0.0, & \text{CLEAN undrained} \\ 0.0986, & \text{CLEAN drained} \\ 0.2349, & \text{CONTAMINATED undrained} \\ 0.4109, & \text{CONTAMINATED drained} \\ 0.0896, & \text{CLEAN - CONTAMINATED undrained} \\ 0.1591, & \text{CLEAN - CONTAMINATED drained} \end{cases}$$

For those patients whose wound is DRAINED, append that probability derived from the CATEGORY of WOUND with the respective TYPE of DRAIN as follows:-

$$fff \begin{cases} -0.0520, & \text{'REDIVAC'} \\ 0.0917, & \text{CORRUGATED} \\ 0.0, & \text{LARGE TUBE} \\ 0.0, & \text{WICK} \\ 0.0, & \text{SMALL TUBE} \\ 0.1082, & \text{MORE THAN ONE DRAIN (of different types)} \end{cases}$$

$$ffff \begin{cases} 0.0869, & \text{if SPECIAL RISK factors are applicable} \\ 0.0, & \text{OTHERWISE} \end{cases}$$

Table 22

Summary of statistical information on parameters relevant to wound infection rates

VARIABLE	MEAN	STANDARD DEVIATION	PARTIAL F-VALUE	REGRESSION COEFFICIENT	STANDARD ERROR	
Category of wound	contam. drained	0.07	0.25	203.530	0.41093	0.02880
	clean-contam. drained	0.14	0.35	51.742	0.15914	0.02212
	contam. undrained	0.04	0.19	50.940	0.23492	0.03291
	clean-contam. undrained	0.12	0.33	20.264	0.08958	0.01990
	clean drained	0.17	0.38	15.619	0.09858	0.02494
Sex of patient	0.48	0.50	11.442	0.04209	0.01244	
Special risk factors	0.05	0.23	9.928	0.08692	0.02759	
Pre-operative hospitalisation	5.62	10.74	9.147	0.00176	0.00058	
	0.07	0.25	9.969	0.09175	0.02906	
	0.02	0.14	5.716	0.10824	0.04527	
Drain type	0.16	0.36	4.404	-0.05196	0.02476	
	444.42	456.33	4.208	2.78x10 ⁻⁵	1.35x10 ⁻⁵	
Number of occupied beds (squared)	2888.71	2031.63	4.049	6.31x10 ⁻⁶	3.14x10 ⁻⁶	

Now E_i , the expected frequency of patients having wounds (which are classified as being infected) in group 'i', is calculated from:-

$E_i = \text{SUM of all } P_i \text{ (corresponding to wound infections) in group 'i'}$

Additional information on those parameters which were found to have a significant influence on post-operative wound infection rates, has been broken down into the same categories as those for the nasal colonisation model, and these are shown in Table 22.

11.6 Distribution of predicted patient risks

Using the mathematical models developed, every patient is assigned a probability of nasal colonisation and the operated patients are assigned a second probability, that of becoming host to a post-operative wound infection. Figure 24 shows the distribution of patient numbers that fall into the predicted probability bands which are shown. Roughly, equal numbers of patients fall into the four probability bands spanning the range from 0.0 to 0.8, then the numbers tail-off rapidly over the next ten probability bands covering the range from 0.08 to 0.28 and beyond. This represents some 53.0% of all patients having a probability of nasal colonisation (with tetracycline-resistant Staph. aureus) that is numerically less than 0.08, with an average rate for the whole group being 0.087.

The frequencies representing probabilities of nasal colonisation, for just those patients who have undergone operative procedures, are illustrated in the histogram shown in Fig. 25. The results follow

FIGURE 24 - Distribution of patients associated with nasal colonisation

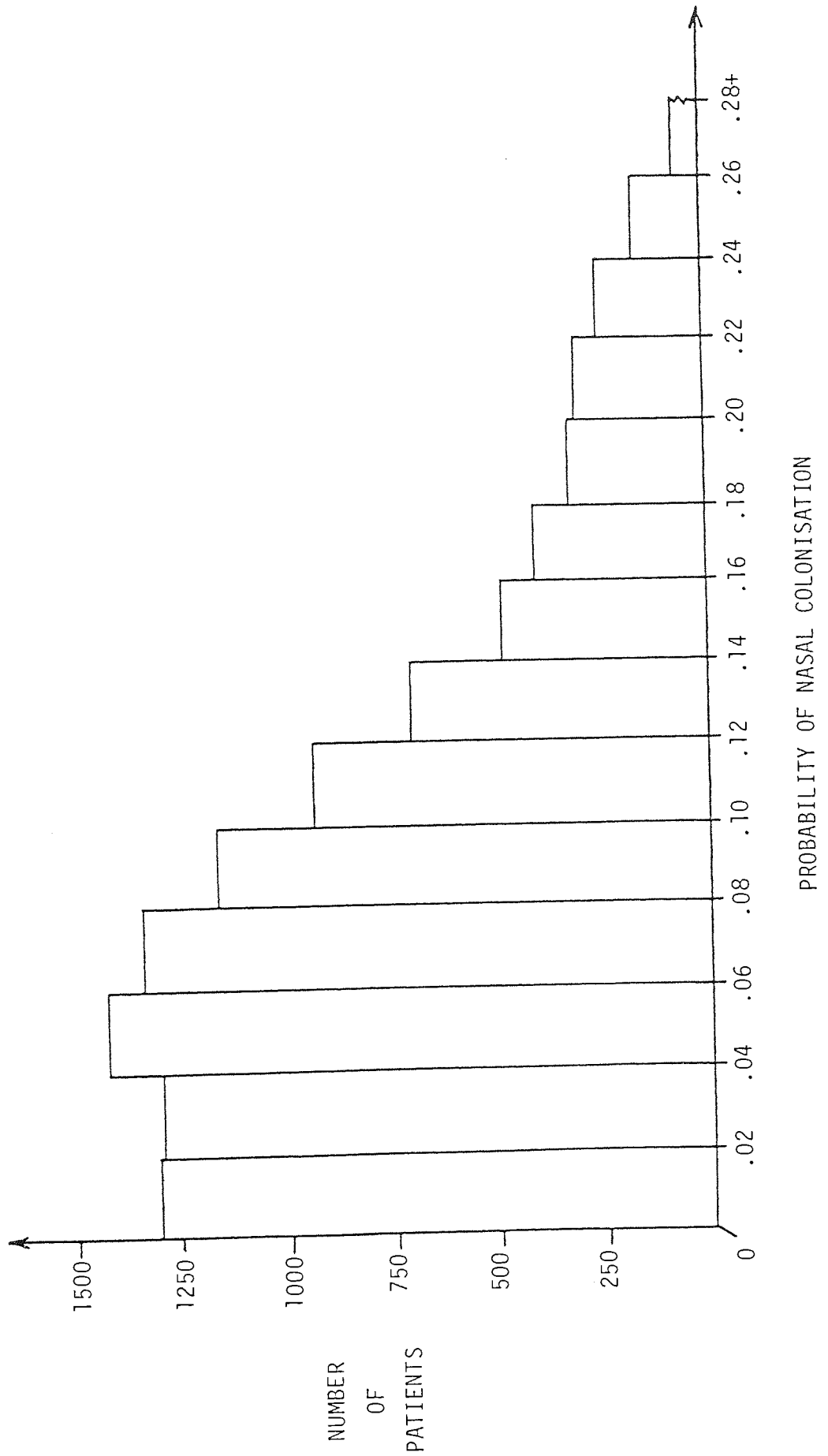


FIGURE 25 - Distribution of operated patients associated with nasal colonisation

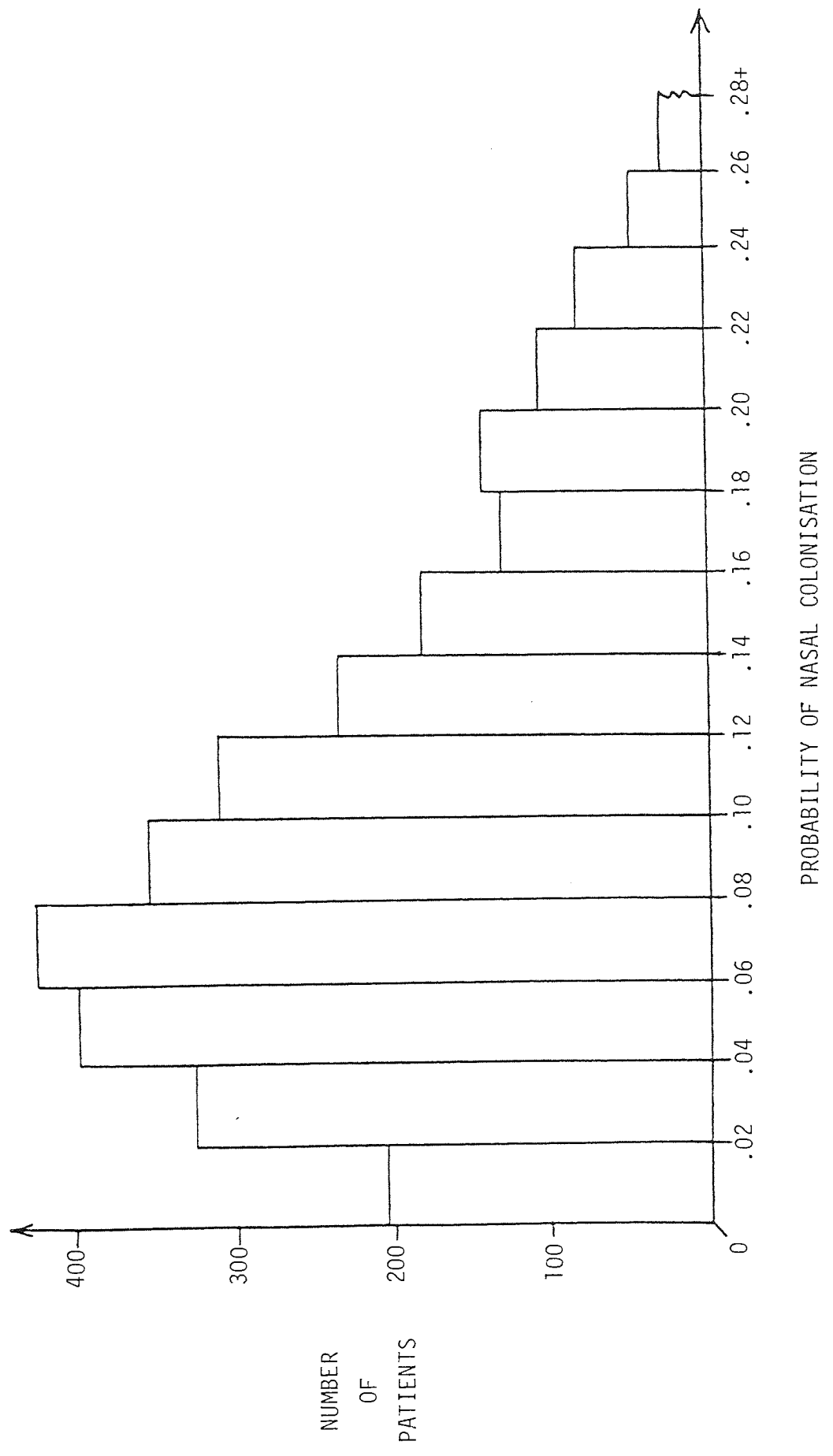
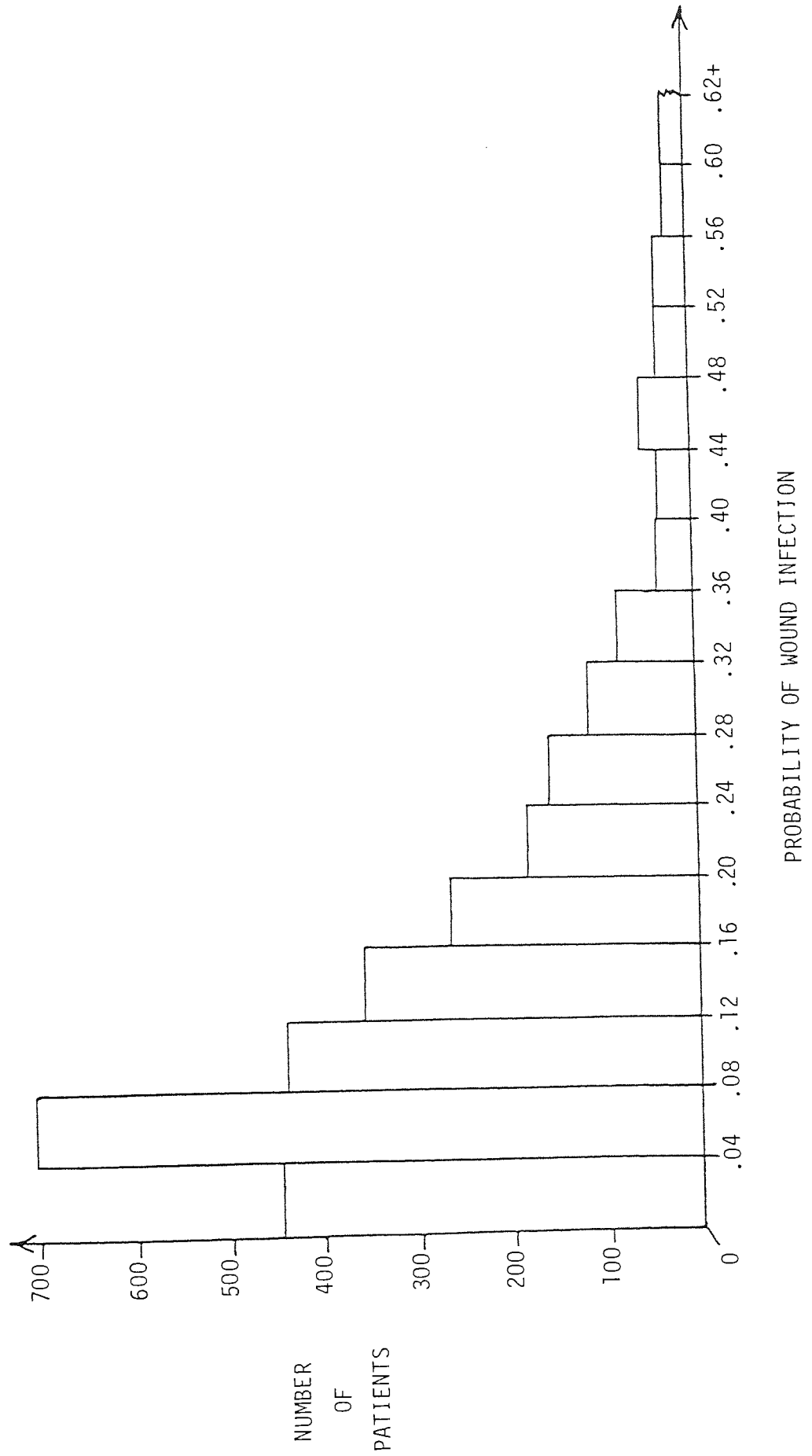


FIGURE 26 - Distribution of patients associated with wound infection



a similar pattern to that for all patients, except that very few patients have probabilities that are less than 0.02. Instead, the majority of patients (61.1%) lie within the five groups spanning the probability range from 0.02 to 0.12, with the peak frequency occurring in the group representing probabilities from 0.06 to 0.08. Hence, the average probability of nasal colonisation for this selected group of patients is increased to 0.098.

Figure 26 represents the frequency distribution for the predicted probabilities of post-operative wound infections, where some 53.2% of all operated patients have probabilities which are less than 0.12, with the majority of these falling into the range 0.04 to 0.08. It is not only interesting, but also very important to note that the distribution of frequencies for all patients whose probability of wound infection exceeds 0.12, are very much strung-out up to the maximum group, which includes probabilities greater than 0.62.

C H A P T E R 12

RELATIONSHIP BETWEEN NASAL COLONISATION AND WOUND INFECTION

12.1 Evaluation of interdependence

For any given patient's risk of becoming colonised with tetracycline-resistant Staphylococcus aureus, there is an associated probability of that patient becoming host to a post-operative wound infection. The graphical representation illustrated in Fig. 27 shows that for any increase in a patient's probability of nasal colonisation, there is also an increase in the risk of developing a subsequent wound infection, but it should be noted that the increases in the colonisation and infection risks are by no means equal. In the lower range of colonisation probabilities there are large increases for the corresponding wound infection risks, but as the probability of colonisation rises beyond 0.20, any increases in wound infection rates prove to be very much smaller.

A further breakdown of wounds into clean, clean-contaminated and contaminated, then in drained and undrained classifications (recorded in Table 23), reveals that in every category, the rate of nasal colonisation for patients with an infected wound was substantially higher than in the corresponding groups for patients with non-infected wounds. Overall, rates of nasal colonisation for patients with an infected wound, were found to be more than double that for the group of patients whose wounds were not considered to be infected.

[WILLIAMS et al (65, 1966) noted that wound infection with Staph. aureus was FIVE times commoner in patients who had staphylococci in the nose.]

FIGURE 27 - Overall relationship between nasal colonisation and wound infection

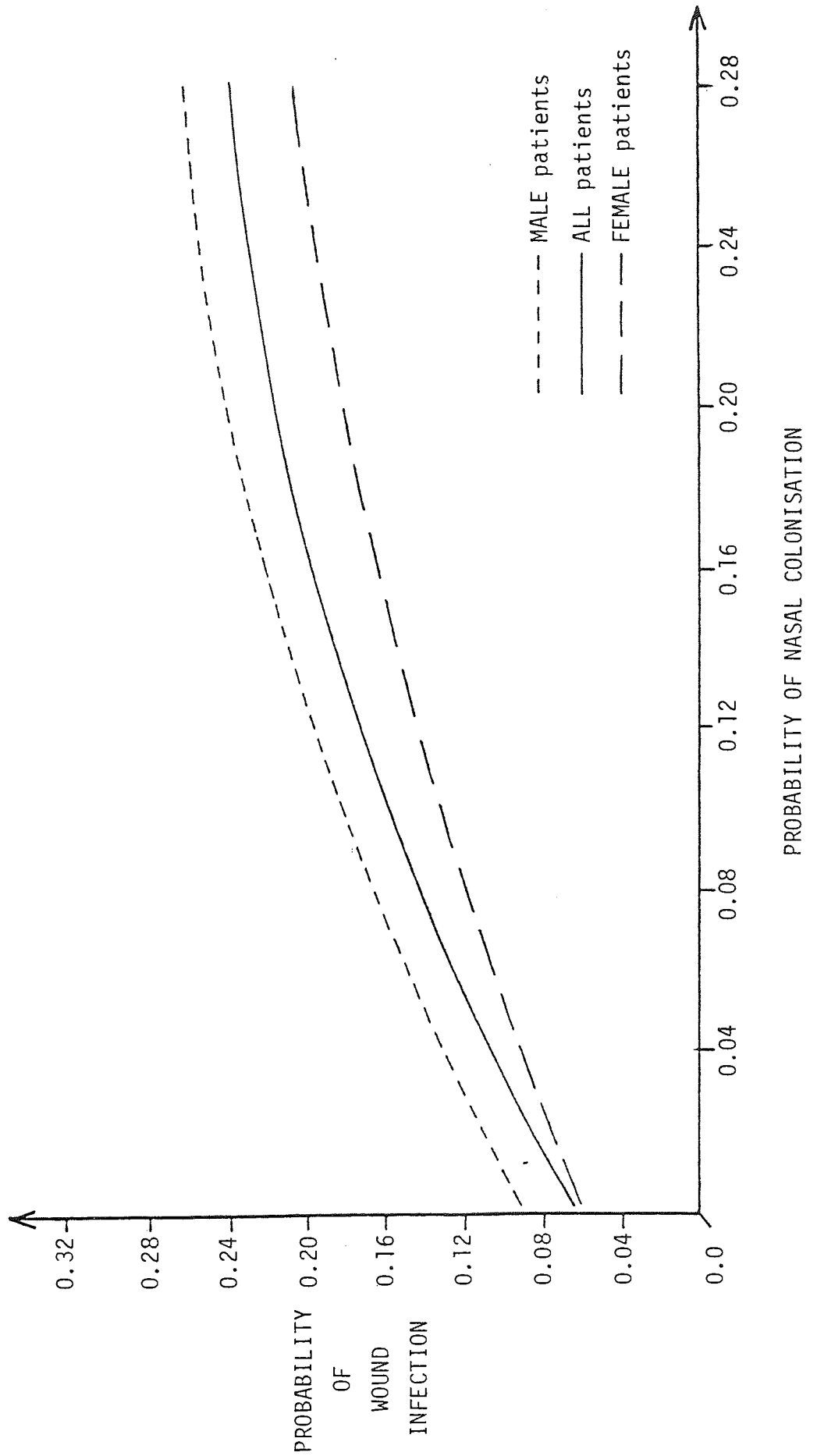


Table 23

Relationship between nasal colonisation and wound infection

TYPE OF WOUND	Patients with an infected wound		Patients with a non-infected wound	
	Number of patients in group	Nasal colonisation rate	Number of patients in group	Nasal colonisation rate
CLEAN	157	14.01%	1,722	6.68%
CLEAN-CONTAMINATED	161	20.50%	623	9.15%
CONTAMINATED	137	23.36%	175	18.88%
ALL UNDRAINED wounds	171	12.28%	1,678	6.67%
ALL DRAINED wounds	284	23.24%	842	11.05%
ALL wounds	455	19.12%	2,520	8.13%

If we postulate a hypothesis whereby one can predict (at the time of admission to hospital) which patients' wounds will ultimately become infected, then the most susceptible patients could well benefit from more effective use of scarce resources, such as isolation facilities, etc. The broad hypothesis being that, if a patient is colonised with tetracycline-resistant Staph. aureus on admission, then we would expect at some time after his operation that the patient's wound would become infected.

To test this broad hypothesis, define TWO classes of SUCCESSFUL predictions:-

Patients with -

- . a colonised nose and an infected wound
- . a non-colonised nose and a non-infected wound

and TWO classes of UNSUCCESSFUL predictions are patients with:-

- . a colonised nose and non-infected wound
- . a non-colonised nose and an infected wound

From Table 23 the following summary of results may be derived.

Table 24a

Influence of colonisation on wound infection

	Number of patients with colonised noses	Number of patients with non-colonised noses
Number of patients with infected wounds	87	368
Number of patients with non-infected wounds	205	2315
All wounds	292	2683

Therefore, under the hypothesis there are -

$$87 + 2315 = 2402 \text{ successful predictions}$$

$$\text{and } 205 + 368 = 573 \text{ unsuccessful predictions}$$

An 80.74% success rate, which is substantially higher than would normally be expected from such a hypothesis where the results can so easily be influenced by the many variable patient and ward parameters.

It is most interesting to note that:- $\frac{87}{292} \times 100 = 29.8\%$ of all patients whose noses are colonised with tetracycline-resistant Staph. aureus, subsequently developed a post-operative wound infection,

whilst ONLY $\frac{368}{2683} \times 100 = 13.7\%$ of those patients whose noses were not colonised, subsequently developed a wound infection.

A quite startling result, indicating that in the presence of nasal colonisation with tetracycline-resistant Staph. aureus, the risk to the

patient in respect of acquiring a wound infection was MORE than DOUBLE that for patients whose noses were not found to be colonised at the time of the survey. Perhaps this result may well indicate that nasal colonisation with resistant strains of Staph. aureus does, in fact, constitute a good and useful index for a patient's general susceptibility to wound infection.

At first glance, these results appear to open up the way for substantially reducing the number of cases where the wounds become infected, if only patients could be protected in respect of their noses becoming colonised with tetracycline-resistant Staph. aureus. However, a little more 'in depth' analysis reveals that unfortunately, at the time of the survey, detailed information was not available on the specific phage typing of organisms, so there is uncertainty as to whether the organisms infecting the patient's wound, and colonising his nose are the same. Additionally, logging the chronological sequence of wound infection and nasal colonisation, may well have proved invaluable in order to determine whether or not a wound infection was generally preceded by colonisation of the patient's nose. Nevertheless, this particular broad spectrum result should justify the inclusion of more detailed microbiological data in future surveys, in order to ascertain whether a more precise link between nasal colonisation and wound infection exists. If one does, then more vigorous measures to prevent nasal colonisation may need to be examined.

Consider now whether there is any mathematical relationship between nasal colonisation and wound infection.

Using the results from Table 24a which is effectively a 2 x 2 contingency table. Test the null hypothesis H_0 : that the current state of colonisation of a patient's nose, has no significant influence on whether that patient's wound ultimately becomes infected, against the alternative hypothesis H_1 : that wound infection is not independent of nasal colonisation but interlinked with it. Under the assumption that the hypothesis H_0 is true, we need to find the expected frequencies for each cell from Table 24a.

Define the following patient conditions:-

WI : a patient with an infected wound

WF : a patient whose wound is free from infection

NC : a patient whose nose is colonised with Staph. aureus

NF : a patient whose nose is free from Staph. aureus

Using the marginal patient frequencies, the probability of each patient condition are summarised as follows:-

$$\text{Prob (WI)} = \frac{455}{2975} \qquad \text{Prob (WF)} = \frac{2520}{2975}$$

$$\text{Prob (NC)} = \frac{292}{2975} \qquad \text{Prob (NF)} = \frac{2683}{2975}$$

Now, if H_0 is true and the two variables are independent, we have

$$\text{Prob (NC} \cap \text{WI)} = \text{Prob (NC)} \text{Prob (WI)} = \frac{292}{2975} \times \frac{455}{2975}$$

$$\text{Prob (NC} \cap \text{WF)} = \text{Prob (NC)} \text{Prob (WF)} = \frac{292}{2975} \times \frac{2520}{2975}$$

$$\text{Prob (NF} \cap \text{WI)} = \text{Prob (NF)} \text{Prob (WI)} = \frac{2683}{2975} \times \frac{455}{2975}$$

$$\text{Prob (NF} \cap \text{WF)} = \text{Prob (NF)} \text{Prob (WF)} = \frac{2683}{2975} \times \frac{2520}{2975}$$

Now, the expected frequencies are obtained by multiplying each cell probability by the total number of observations, i.e. 2975.

For example, the expected number of patients with a colonised nose and a wound infection would be given by,

$$\frac{292}{2975} \times \frac{455}{2975} \times 2975 = 44.66$$

A summary of these expected patient numbers can be appended to Table 24a, as shown in brackets below:-

Table 24b

Contingency table for actual wound infections

	Colonised patients	Non-colonised patients	All patients
Actual number of wound infections	87 (44.66)	368 (410.34)	455 (455)
Actual number of non-infected wounds	205 (247.34)	2315 (2272.66)	2520 (2520)
All wounds	292 (292)	2683 (2683)	2975 (2975)

Now to test the hypothesis of independence, H_0 ,

calculate $\chi^2_{\text{calc}} = \sum_{\text{all FOUR elements}} \frac{(O_i - E_i)^2}{E_i}$

where O_i are the observed patient frequencies
and E_i are the expected patient frequencies

$$\begin{aligned} \chi_{\text{calc}}^2 &= \frac{(87 - 44.66)^2}{44.66} + \frac{(368 - 410.34)^2}{410.34} \\ &+ \frac{(205 - 247.34)^2}{247.34} + \frac{(2315 - 2272.66)^2}{2272.66} \\ \chi_{\text{calc}}^2 &= 52.55 \end{aligned}$$

If $\chi_{\text{calc}}^2 > \chi^2 (v)$, the tabulated chi-square value, then the hypothesis H_0 : of independence between nasal colonisation and wound infection is rejected at the α level of significance, in favour of the alternative hypothesis H_1 . Choose the level of significance, $\alpha = 0.005$

$$\chi_{0.005}^2 (1 \text{ degree of freedom}) = 7.879$$

[where degrees of freedom = (number of rows - 1) x (number of columns - 1)]

Therefore, since 52.55 is GREATER than the tabulated value of 7.879, we must reject the hypothesis H_0 , in favour of the alternative hypothesis H_1 : that wound infection is not independent of nasal colonisation but is very much influenced by its presence.

12.2 Accuracy of the wound infection model

Having determined the results above, the prediction model for wound infections can be used to assess whether or not there is any relationship between ACTUAL nasal colonisation and PREDICTED wound infections.

Consider the results shown in Table 25, with expected patient numbers shown in brackets.

Table 25

Contingency table for predicted wound infections

	Colonised patients	Non-colonised patients	All patients
Predicted number of wound infections	68 (44.66)	387 (410.34)	455
Predicted number of non-infected wounds	224 (247.34)	2296 (2272.66)	2520
All wounds	292	2683	2975

Therefore, under the prediction hypothesis there are,

$$68 + 2296 = 2364 \text{ correct associations}$$

$$\text{and } 224 + 387 = 611 \text{ incorrect associations.}$$

A success rate of 79.46% at correctly predicting whether or not a patient would subsequently become infected, given his current state of nasal colonisation with hospital-acquired strains.

To determine whether or not there is actually any statistical evidence to support this result, test the null hypothesis H_2 : that the predicted wound infection risks are not significantly influenced by the current state of colonisation of a patient's nose, against the alternative hypothesis H_3 : that predicted risks of wound infection are not independent of nasal colonisation but very much interlinked with it.

To test the hypothesis of independence, H_2 , calculate

$$\begin{aligned} \chi_{\text{calc}}^2 &= \frac{(68 - 44.66)^2}{44.66} + \frac{(387 - 410.34)^2}{410.34} \\ &+ \frac{(224 - 247.34)^2}{247.34} + \frac{(2296 - 2272.66)^2}{2272.66} \\ \chi_{\text{calc}}^2 &= 15.97 \end{aligned}$$

Therefore, since 15.97 is GREATER than the appropriate tabulated critical value from the chi-square distribution of 7.879, the hypothesis H_2 is REJECTED in favour of the alternative hypothesis H_3 : that predicted wound infection risk is not independent of nasal colonisation, but very much influenced by its presence.

12.3 Extent of staphylococcal wound infections

If Table 24a is subdivided into staphylococcal wounds and all other infected wounds, then the following table of wound infections results:-

Table 26

Breakdown of infected wounds

	Colonised patients	Non-colonised patients	All patients
Staphylococcal wounds only	44	117	161
Other wound infections	43	251	294
All wound infections	87	368	455

The conclusions that can be drawn from this Table are that:-
 $\frac{44}{87} \times 100 = 50.6\%$ of all patients whose noses are colonised with tetracycline-resistant Staph. aureus, and develop a post-operative wound infection, do in fact, have their wounds infected with staphylococci.

On the other hand, ONLY $\frac{117}{368} \times 100 = 31.8\%$ of those patients whose noses are not colonised, subsequently develop staphylococcal wound infections.

The implications of these results being that in the presence of nasal colonisation with tetracycline-resistant Staph. aureus, if the patient did acquire a wound infection, then the risk of this infection being due to staphylococcal organisms, was increased by MORE than HALF over those patients whose noses were not found to be colonised.

In view of these results, it appears that consideration ought to be given to including nasal colonisation as an additional factor in the patient wound infection model. When nasal colonisation was added to the list of input variables to be evaluated by the computerised stepwise regression programme it did indeed emerge as being an integral part of the wound model, making a significant contribution, in respect of increasing a patient's potential risk of cross-infection.

The analysis of variance table concerned with wound infection rates, when a patient's state of nasal colonisation is taken into account, is shown overleaf in Table 27.

Table 27

Analysis of variance for the wound-nasal model

Source of variation	Degrees of freedom	Sums of squares	Mean square	Calculated F-value	100R ²
Total	2974	455.000			
Mean	1	69.612			
Total (corrected for mean)	2973	385.388			
Regression	14	53.932	3.852	34.390	13.99%
Residual	2959	331.456	0.112		

Specifically, the probability of any patient having a WOUND INFECTION is given by:-

$$P_i = (-0.00481) + (0.0004325 \times \text{AGE}) + \text{SEX}^{\dagger} + (0.001513 \times \text{DURATION of PRE-OPERATIVE HOSPITALISATION}) + (\text{CATEGORY of WOUND})^{\dagger\dagger} + (\text{TYPE of DRAIN})^{\dagger\dagger\dagger} + (\text{SPECIAL RISK FACTORS})^{\dagger\dagger\dagger\dagger} + (0.000026 \times \text{NUMBER of OCCUPIED BEDS}^2) + \text{COLONISATION}^{\dagger\dagger\dagger\dagger\dagger}$$

$$\dagger \left\{ \begin{array}{ll} 0.0403, & \text{MALE} \\ 0.0, & \text{FEMALE} \end{array} \right.$$

$$\dagger\dagger \left\{ \begin{array}{ll} 0.0, & \text{CLEAN undrained} \\ 0.1001, & \text{CLEAN drained} \\ 0.2297, & \text{CONTAMINATED undrained} \\ 0.4025, & \text{CONTAMINATED drained} \\ 0.0871, & \text{CLEAN - CONTAMINATED undrained} \\ 0.1570, & \text{CLEAN - CONTAMINATED drained} \end{array} \right.$$

For those patients whose wound is DRAINED, append that probability derived from the CATEGORY of WOUND (shown on previous page) with the respective TYPE of DRAIN as follows:-

+++	}	-0.0561,	'REDIVAC'
		0.0877,	CORRUGATED
		0.0,	LARGE TUBE
		0.0,	WICK
		0.0,	SMALL TUBE
		0.1022,	MORE THAN ONE DRAIN (of different types)
++++	}	0.0818,	if SPECIAL RISK factors are applicable
		0.0,	OTHERWISE
+++++	}	0.0789,	if patient is subject to NASAL COLONISATION
		0.0,	OTHERWISE

Now E_i , the expected frequency of patients having wounds which are classified as being infected in group 'i' is calculated from:-

$$E_i = \text{SUM of all } P_i \text{ (corresponding to wound infections) in group 'i'.$$

Additional information on those parameters which were found to have a significant influence on post-operative wound infection rates, has been broken down into the same categories as those for the previous wound model (which took no account of nasal colonisation), and these are shown in Table 28.

Table 28

Statistical information on parameters (including colonisation) relevant to wound infection rates

VARIABLE	MEAN	STANDARD DEVIATION	PARTIAL F-VALUE	REGRESSION COEFFICIENT	STANDARD ERROR
Category of wound	0.07	0.25	194.408	0.40246	0.02886
	0.14	0.35	50.296	0.15696	0.02213
	0.04	0.19	48.708	0.22965	0.03291
	0.12	0.33	19.134	0.08708	0.01991
	0.17	0.38	16.115	0.10014	0.02495
Colonisation	0.10	0.30	13.637	0.07889	0.02136
Sex of patient	0.48	0.50	10.479	0.04033	0.01246
Special risk factors	0.05	0.23	8.794	0.08183	0.02759
Pre-operative hospitalisation	5.63	10.75	6.711	0.00151	0.00058
	0.07	0.25	9.108	0.08767	0.02905
	0.02	0.15	5.099	0.10218	0.04525
Drain type	0.16	0.36	5.141	-0.05612	0.02475
	444.60	456.67	3.673	2.60×10^{-5}	1.35×10^{-5}
Number of occupied beds (squared)	49.15	21.84	2.142	4.32×10^{-4}	2.95×10^{-4}
Age					

CHAPTER 13

CONCLUSIONS

The extensive literature survey has confirmed the need for some form of standardisation of both patient and ward parameters, in order that valid comparisons between different surveys may be made. Lack of standardisation accounts for the apparent differences in survey results produced by different researchers, particularly so, when differing types of operative procedures are analysed.

Those results produced in respect of analysing the effects of various ward structures, procedures and practices, should dispense with many of the myths which surround the usefulness, or otherwise, of many factors which were believed, but never conclusively proved, to be useful. On the other hand, if current standards are not maintained, but allowed to deteriorate, then many of the factors found not to have a significant influence on either 'nasal colonisation rates', or 'wound infection rates', may well re-emerge causing additional problems to both patients and staff. This is surely good enough reason for not attempting radical changes without proper evaluation of the possible consequences. Other patient and ward parameters (some of which are under our control and others which are not), have been conclusively proved to affect the risks to which all hospitalised patients are subject.

The nasal model created in the thesis has proved to be of substantial benefit by affording insight into the general nasal susceptibility of each particular section of the patient population. Whilst the wound

models indicate that differing wound types have the greatest significant effect on rates of wound infection, and also highlight the need to avoid excessive and unnecessary use of drains.

The problems caused by the notorious Gram-positive organism, Staphylococcus aureus, are not nearly so great as they were 20 years ago. New problems, however, emerge daily as Gram-negative organisms develop resistance to more and more antibiotics. With the aid of the modelling techniques developed here, it is expected that the effect of any organism can be monitored, in order to prevent its uncontrolled spread, before being identified as a chief source of cross-infection.

Age, sex, type of operation, etc., have all been found to contribute to post-operative infection risks. These cannot under normal circumstances be controlled, however, as the proportion of risk due to these factors can now be quantified, we are much closer to identifying those areas where intervention is likely to prove successful, thus enabling scarce resources to be concentrated more effectively on preventable infection risks.

If it can be shown that a sudden increase in infection rates is due to a temporary change in the susceptibility of a particular group of patients (e.g. an increase in average age, number of diabetics, etc.), then extensive investigations into the cause, together with often costly and probably ineffective measures to combat the transient condition, may well be avoided. If, on the other hand, it could be shown that infection rates had increased without a corresponding

increase in patient susceptibility, then the cost of investigative procedures will prove justifiable, and could probably be targeted more specifically to achieve maximum effectiveness.

Since a correction can be applied to compensate for different age distributions and other differences in patient susceptibility, a potential new use of the mathematical models will be to make better comparisons between different wards, (or the same ward at different points in time) and the wound model in particular could be used to re-adjust infection rates to account for the differences in types of operation performed.

Finally, if the results derived in this thesis were used for any of these purposes, then careful consideration of the following questions must be made:-

- .. Is the difference between ACTUAL and PREDICTED colonisation and infection rates a measure of how well the problems are being managed?
- .. Is any excess of ACTUAL infection rates over and above those which are PREDICTED, due to infections that could have been prevented?
- .. Could the models be used to evaluate the effectiveness of any changes that are actually made?
- .. Would this modelling technique help to identify patients for whom the use of prophylactic antibiotics would be of

most value or those patients who would benefit from intervention prior to surgery by an alteration in their immunological status?

- .. Could the surgeon's knowledge of any particular patient be enhanced if he knew in advance which patients, when subjected to a particular procedure, were at greatest risk from post-operative wound infections, and if so, how would it help?

CHAPTER 14

FUTURE WORK

Although a significant amount of progress has been made in developing and adapting multiple regression analysis techniques to assess the contribution to wound infection or nasal colonisation made by each of the patient and ward parameters collected during the course of the survey, the effect of other factors for which information was not recorded still needs to be appraised. Examples of additional factors which may yield fruitful results, and should consequently be considered for inclusion in the construction of future models, include a subjective assessment of an individual surgeon's skill, more accurate collection of data relating to the length of wound incision and duration of operation, together with information on the use of both prophylactic and therapeutic antibiotics.

The majority of this research work has been targeted on wound infections, but if sufficient information becomes available from future surveys, it is now a relatively simple task to expand the ideas developed beyond wound infections to encompass infections of the urinary tract and respiratory tract, skin and subcutaneous infections, together with those infections caused as a direct result of burns.

Having found a potential relationship between nasal colonisation and wound infection to exist, a more detailed study needs to be carried out to assess under more controlled conditions, the validity of these initial observations. The study ought not to be confined to the carriage of antibiotic-resistant Staphylococcus aureus, since

today, this Gram-positive organism is not nearly such a predominant cause of infection as it was in the 1950's and 1960's. More useful information could be gained from looking at the relationship between nasal colonisation with tetracycline-resistant Staph. aureus and nasal colonisation with other forms of staphylococci, and colonisation or dispersal of Staph. aureus from other skin sites. It would also be of additional value if any associations could be firmly established between nasal colonisation with resistant strains of Staph. aureus and colonisation of wounds or the buccal cavity with Gram-negative bacilli, and the subsequent influence not only on wound infections, but on the broader spectrum of all hospital-acquired infections.

In the past, many conclusions have been proposed in respect of controlling the transmission of hospital-acquired infections, by researchers associated with surveys too numerous to mention. Previously, it has been impossible to carry out direct inter-survey (or even intra-survey) comparisons, because of inherent differences in patient populations, range of operative procedures, ward practices, procedures and environmental structures. With the aid of the mathematical models produced here, it is now possible for all these survey results and conclusions to be re-evaluated in the light of this new generation of modelling information becoming available, so that unavoidable variations contained within the sources of raw data collected, can be eliminated by retrospective standardisation.

Finally, perhaps the most important task to be performed in the future, is to distinguish between preventable and non-preventable

infections, in order that we may strive towards the elusive irreducible minimum infection rate. The technique of mathematical modelling alone will not perform this function, but it will serve as an invaluable tool, going a long way towards managing the global problem of infection control in a more cost effective manner, in order that the scarce resources of professionally qualified staff, specialist equipment and facilities, together with finance, may be channelled into the area of preventable infections, rather than being wasted on those infections which cannot reasonably be prevented.

APPENDIX A

COSTS OF ANTIBIOTIC THERAPY

APPENDIX A1

Relative Cost of FIVE Days ORAL ANTIBIOTIC THERAPY with TABLETS/CAPSULES
at March 1980 Rates

<u>ANTIBIOTIC (dose)</u>	<u>Cost</u>
Nitrofurantoin (100mg, 4 times daily)	8p
Oxytetracycline (250mg, 4 times daily)	14p
Penicillin (250mg, 4 times daily)	20p
Phenethicillin (250mg, 4 times daily)	82p
Ampicillin (500mg, 4 times daily)	89p
Erythromycin (250mg, 4 times daily)	105p
Co-Trimoxazole (2 tablets, twice daily)	108p
Amoxicillin (250mg, 3 times daily)	150p
Flucloxacillin (250mg, 4 times daily)	297p
Cephalexin (500mg, 4 times daily)	307p
Nalidixic Acid (1000mg, 4 times daily)	320p
Cefaclor (250mg, 3 times daily)	349p
Pivmecillinam (400mg, 3 times daily)	360p
Clindamycin (300mg, 4 times daily)	598p

NOTE: No attempt has been made to compare the therapeutic efficiencies for any of the products shown.

APPENDIX B

WARD SURVEY RECORDS

APPENDIX B1

WARD STRUCTURES AND FACILITIES

Hospital

--	--	--

Ward

--	--

Day

--	--

Month

--	--

Year

--	--

AGE OF WARD (years)

1. 2 2. 2-5 3. 5-9 4. 10-19 5. 20-49
6. 50-99 7. 100+ 8. Upgraded

--

TYPE OF PATIENTS

1. General surgical 2. General medical 3. Gynaecological
4. Obstetric 5. Paediatric (medical)
6. Surgical and medical 7. Surgical and gynaecological
8. Geriatric (medical) 9. Orthopaedic 10. Others

--	--

Sex of patient

1. Male 2. Female 3. Male and female in same ward
4. Male and female in different sections

--

STRUCTURAL FEATURES

General layout - grade

--

Position of ward

1. Lower ground floor 2. Ground floor 3. First floor
4. Floors 2-3 5. Floors 4-5 6. Higher floors
7. On more than one floor

--

Ward 1. Number of beds

--	--

Ward 2. Number of beds

--	--

Ward 3. Number of beds

--

Ward 4. Number of beds

--

Ward 5. Number of beds

--

Ward 6. Number of beds

--

Shape of Ward 1

- 1. Rectangular 2. Square 3. Round or oval 4. 'L' shaped
- 5. Polygonal 6. Triangular 7. Other (specify)

Floor area Ward 1 (sq. ft.)

- 1. <125 2. 125-249 3. 250-499 4. 500-999 5. 1000-1499
- 6. 1500-1999 7. 2000-3000 8. 3000-4000 9. >4000

Height of Ward

- 1. 10 ft 2. 10-15 ft 3. 15 ft

Division of Ward 1

- 0. Not divided 1. Cubicles 2. Bays (complete) 3. Bays (low partitions)
- 4. Bays (high partitions) 5. Cubicles - no doors 6. One division only 7. Two separate sections

No. of divisions Ward 1

Single beds

2-4 beds

5-8 beds

Over 8 beds

No. of occupied beds, Ward 1

No. of extra beds, Ward 1

Balcony

- 0. No balcony 1. Balcony no beds 2. 1-2 beds 3. 3-5 beds
- 4. 6-8 beds 5. Over 8 beds 6. Balcony, no beds - day room

Day room - number (in addition or instead of balcony)

Average distance between bed centres - all wards with more 6 beds

- 1. <5 ft 2. 5'-5'11" 3. 6'-6'11" 4. 7'-7'11"
- 5. 8'-8'11" 6. 9'-10'11" 7. >11"

No. of bed spaces less than 6½ ft all wards

No. of bed spaces measured

Windows all wards more than 6 beds

1. Bright daylight 2. Moderate daylight 3. Restricted
4. Variable in different wards

Type of floor, Ward 1

1. Wooden boards 2. Wooden blocks 3. Terrazzo
4. Plastic tiles 5. Plastic sheet 6. Tiles, non-plastic
7. Lino 8. Others - specify

Condition of floor, Ward 1

1. Good 2. Above average 3. Average 4. Below average
5. Poor 6. Average with areas of localised damage

Condition of walls, Ward 1

1. Good 2. Above average 3. Average 4. Below average
5. Poor 6. Average with areas of localised damage

Type of Ventilation

WOUND DRESSING ROOM

0. None 1. Mechanically ventilated with air lock
2. Mechanically ventilated without air lock
3. Not ventilated

SLUICE ROOM

0. None 1. One less than 50 ft 2. One 50-100 ft
3. One over 100 ft 4. Two less than 50 ft 5. Two 50-100 ft
6. Two over 100 ft 7. Two of different sizes
8. More than two

Type of floor of Sluice room

1. Wooden boards 2. Wooden blocks 3. Terrazzo
4. Plastic tiles 5. Plastic sheet 6. Tiles, non-plastic
7. Lino 8. Others - specify

Size and design of Sluice room

1. Good 2. Adequate 3. Inadequate 4. Poor

General condition

- 1. Good 2. Above average 3. Average 4. Below average
- 5. Poor

Position

- 1. Opening on to main ward 2. Annexe adjacent to ward
- 3. Corridor away from ward 4. Other - specify

STERILIZING OR PREPARATION ROOM

- 1. Good 2. Adequate 3. Inadequate 4. Poor 5. None

KITCHEN

- 1. Good 2. Adequate 3. Inadequate 4. Poor 5. None

STORAGE OF LINEN

- 1. In ward area 2. Special room 3. Sister's office
- 4. Other - specify

FACILITIES

Number of baths (excluding side-wards)

Number of showers (excluding side-wards)

Number of wash-basins - patients' (excluding side-wards)

Number of wash-basins - staff private

Number of wash-basins - Ward 1

Taps

1. Elbow operated

Ward staff

2. Hand operated

Private staff

3. Foot operated

Washroom patients'

4. Elbow or hand operated

Side-ward patients'

Sterilizing room

Wash-basins in side wards

1. Yes 2. No 3. Variable

TOILETS

Number of patients (main ward)

Total

- Separate for staff 1. Yes 2. No 3. Yes (2)
4. Yes (shared with one or more other wards)

Type

1. Hand flush high level 2. Hand flush low level
3. Foot flushing 4. Non-flushing, hand
5. Non-flushing, foot 6. Mixed

Toilet wash-basins - site

1. In toilet 2. Adjacent to toilet 3. Not adjacent
to toilet

STERILIZING ON WARD

0. None 1. Boiler 2. Autoclave 3. Autoclave and
boiler 4. Others

Use of sterilizers

0. Not used 1. Used rarely. 2. Used frequently
3. Used frequently (not for sterile equipment)

MULTIPLE USE OF FACILITIES

0. Not used for any other purpose
1. Dressings
2. Examinations and/or admissions
3. Examinations and/or admissions and dressings
4. Disinfection of bed-pans and/or urinals
5. Disinfection of contaminated linen
6. Disinfection of bed-pans and contaminated linen

7. Preparation and storage of flowers

8. Other - specify

1. Bathroom

2. Bath

3. Sterilizing room

Same room for bath and toilet

1. Yes 2. No

Record No.

--	--

APPENDIX B2

WARD PRACTICES I

(Where information not recorded code N)
(Where information not known code K)

Hospital	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Ward	<input type="checkbox"/>	<input type="checkbox"/>	
Day	<input type="checkbox"/>	<input type="checkbox"/>	
Month	<input type="checkbox"/>	<input type="checkbox"/>	
Year	<input type="checkbox"/>	<input type="checkbox"/>	
Total nursing staff	<input type="checkbox"/>	<input type="checkbox"/>	
S.R.N. day staff			<input type="checkbox"/>
S.R.N. night staff			<input type="checkbox"/>
S.E.N. day staff			<input type="checkbox"/>
S.E.N. night staff			<input type="checkbox"/>
Orderlies			<input type="checkbox"/>
Domestics			<input type="checkbox"/>

FLOORS

Frequency of cleaning

1. More than twice daily	2. Twice daily	3. Daily	<input type="checkbox"/>
4. Alternate days	5. Less frequently		

Dry cleaning

1. Nil	2. Vacuum cleaner with filter	3. Vacuum cleaner without filter	4. Broom	5. Polish - hand	6. Polish - machine	7. Kex-type mop	8. Other - specify	<input type="checkbox"/>
								<input type="checkbox"/>

Wet cleaning

1. Nil	2. Mop - string	3. Mop - sponge	4. Mop with two-compartment bucket	5. Hand scrubbing	6. Scrubbing machine	7. Combined scrubbing and vacuum machine	8. Other - specify	<input type="checkbox"/>
								<input type="checkbox"/>
				Routine				<input type="checkbox"/>
				Special				<input type="checkbox"/>

Storage of mops

- 0. No special technique
- 1. Dried
- 2. Disinfectant
- 3. Other

WALLS AND SURFACES

Frequency of cleaning

- 1. Daily
- 2. 2-3 times per week
- 3. Weekly
- 4. Fortnightly
- 5. Monthly
- 6. 2-4 months
- 7. 5-6 months
- 8. Less frequently
- 9. Irregularly

Complete ward

Whole wall routine

Whole wall special

Lower wall

Surfaces

Method

- 1. Dry dusting
- 2. Damp dusting
- 3. Kex-type mop
- 4. Washing
- 5. Spraying
- 6. Others

Walls, routine

Walls, special

Surfaces

SPECIAL RISK SITES

Frequency of cleaning

- 0. Not used
- 1. After use always
- 2. After use sometimes
- 3. After use by infected patients and daily
- 4. After use and daily
- 5. Daily
- 6. Less frequently
- 7. Disposable
- 8. Other

Urinals

Bed-pans

Pots (children)

Toilet

Toilet seat

Bath

Wash-basin

Wash-bowl

Method

0. Not used 1. Washed only, no further treatment
2. Hot water 3. Boiled 4. Steam treatment
5. Disinfectant 6. Disposal unit

Urinals

Bed-pans

Pots (children)

Toilet

Toilet seat

Bath

Wash-basin

Wash-bowl

Covers

Disposable toilet seat covers

1. Yes 2. No 3. Sometimes

Bed-pan covers

0. Not used 1. Linen 2. Disposable 3. Other

BLANKETS - MATERIAL

1. Wool 2. Cotton cellular 3. Synthetic fibre
4. Mixed 5. Other - specify

Frequency of changing

1. Weekly 2. Twice weekly or more frequently 3. Every
2-4 weeks 4. Less frequently 5. After discharge of
patient 6. Irregularly 7. Other

Routine

Infected patients

CURTAINS AND SCREENS

0. No curtains or screens 1. Curtains only
2. Screens only 3. Curtains and screens

Material

1. Cotton 2. Plastic 3. Fibreglass 4. Material
5. Other - specify

Curtains

Screens

Changed or Disinfected

1. Weekly 2. Fortnightly 3. Monthly 4. 2-3 monthly
5. 4-6 monthly 6. Over 6 months 7. Irregularly
8. Other - specify

Curtains

Screens

TOWELS - type for handwashing

1. Linen 2. Roller 3. Roll-o-mat 4. Paper
5. Other - specify

MATTRESSES AND PILLOWS

0. Not used 1. Not covered 2. Linen covers
3. Waterproof covers 4. Waterproof cover, special cases only 5. Variable

Pillows

Mattresses

Treatment of Mattresses and Pillows

0. Not used 1. Not treated 2. Treated after all infectious patients
3. Treated after some infected patients 4. Treated after each patient
5. Irregularly 6. After death only 7. Other - specify

Pillows

Mattresses

Method of Treatment

0. Low temperature wash only 1. Water 80°C-99°C 2. Water 100°C
3. Autoclave 4. Low temperature steam 5. Phenolic - specify
6. Q.A.C. - specify 7. Formaldehyde 8. Fresh air
Other - specify

Blankets

Mattresses (routine)

" (contaminated)

Pillows (routine)

" (contaminated)

Sorting of linen

1. In ward 2. Sometimes in ward 3. Never in ward 4. Other arrangements

Disposal of linen

1. Special sack at bedside 2. Ordinary sack at bedside
3. Special sack in sluice room 4. Ordinary sack in sluice room
5. Other arrangements

Routine

Contaminated

Material of sack

1. Canvas 2. Plastic 3. Synthetic fibre 4. Other cloth
5. Paper 6. Other

Routine

Contaminated

DISINFECTION OF THERMOMETERS

1. Individual thermometers in disinfectant
2. All treated together after temperature round
3. Individually disinfected only on patients' discharge
4. Wiped with disinfectant after each patient
5. Individual thermometers in disinfectant (not labelled)

Record No.

APPENDIX B3

WARD PRACTICES II

Hospital

--	--	--

Ward

--	--

Year

--	--

PRE-OPERATIVE PREPARATION IN WARD (cold surgery only)

1. Day of operation 2. Previous day 3. Two days of treatment
4. Three days of treatment 5. Other

Method

1. Plain soap 2. Hexachlorophane soap or detergent
2. Chlorhexidine 4. Iodine 5. Iodophor 6. 70% alcohol
7. Hexachlorophane soap and chlorhexidine 8. Other - specify

TREATMENT OF SHAVING EQUIPMENT (ward)

0. Not used 1. Boiled or autoclaved after use 2. Boiled or autoclaved daily
3. Disinfected after use 4. Disinfected daily
5. Stored in disinfectant 6. Washed only
7. Disposable 8. Other - specify

Brush

Razor

Electric razor heads

WOUND DRESSING

Wound dressing site

1. Ward 2. Dressing room, non ventilated 3. Dressing room, ventilated
4. Bathroom 5. Sterilizing room 6. Other - specify

Main site

Other site

Use of wound dressing room

0. Not applicable 1. All patients 2. Clean operations only
3. Septic operations only 4. All patients (excluding side-wards)
5. Clean operations (excluding side-wards)
6. Septic operations (excluding side-wards)

Number of staff in dressing team

1. One 2. Two 3. Sometimes two, usually one

Dress

0. No special dress 1. Mask 2. Gown 3. Gown and mask
4. Gown, mask and cap 5. Gown and cap 6. Cap and mask
7. Other - specify

Gloves used

1. Yes 2. No 3. Special wounds only

Handwashing

1. Plain soap 2. Hexachlorophane bar soap 3. Hexachlorophane detergent
4. Hexachlorophane liquid soap 5. Iodophor, e.g. 'Betadine'
6. Other - specify

General

Dressings

Special techniques

Scrubbing

1. Yes 2. No 3. Special cases or techniques only
4. At beginning of dressing round only 5. Beginning of round and special cases

TREATMENT OF NAIL BRUSHES

0. No nail brushes 1. No treatment 2. Autoclaved or boiled daily
3. Autoclaved or boiled daily and stored in disinfectant
4. Stored in disinfectant 5. Autoclaved after each use
6. Periodic treatment only 7. Other - specify -

USE OF HAND CREAM IN WARDS

0. Not used 1. Rarely 2. Sometimes 3. Usually 4. Always

Container

1. Individual tube or jar 2. Communal jar 3. Communal tube

TREATMENT OF CHEATLE'S FORCEPS

- 0. Not used
- 1. Boiled or autoclaved before use
- 2. Boiled or autoclaved daily and stored in disinfectant
- 3. Stored in disinfectant
- 4. C.S.S.D.

DRESSING TECHNIQUE

- 1. Not no touch
- 2. No touch - pre-packed set
- 3. No touch - drum or box
- 4. Other procedure - specify

Number pairs of forceps used per dressing (usual)

CLEANING LOTION FOR WOUNDS

- 1. Saline
- 2. 'Eusol'
- 3. 'Savlon'
- 4. 'Cetrimide'
- 5. 70% alcohol
- 6. Chlorhexidine
- 7. Other - specify

Clean

Dirty or septic

ANTI-BACTERIAL SPRAY

- 0. Never used
- 1. Always used
- 2. Sometimes used

Name of spray

Clean

Dirty or septic

TYPE OF DRESSING

- 0. Nil
- 1. Nil + Norbecutane
- 2. Gauze and cotton wool
- 3. Gauze and elastoplast sealed
- 4. Other - specify

Clean undrained

Drained

Dirty or septic

HANDLING OF CONTAMINATED DRESSINGS

- 1. Ungloved fingers
- 2. Gloved fingers
- 3. Forceps
- 4. Forceps, sometimes fingers

DISPOSAL OF CONTAMINATED DRESSINGS AND INSTRUMENTS

1. Paper or plastic bag 2. Open receiver 3. Closed bucket
4. Open bucket 5. Paper bag and bucket
6. Container with disinfectant 7. Other - specify

Dressings and disposables

Instruments (metal)

USE OF SIDE ROOMS

0. No side room 1. Very ill patients 2. Private patients
3. Infected patients 4. Any patients 5. Any combination

Isolation facilities

1. Inadequate 2. Satisfactory 3. Satisfactory but not used for infections

Isolation of infections

0. Never 1. Rarely 2. Sometimes 3. Usually 4. Always
5. Hospital-acquired only 6. Serious infections only
7. Not applicable

Wound infections

Enteric "

Other notifiable diseases

Infections due to Staph.aureus

" " " Ps.pyocyanea

Other organisms - specify

Other indications for isolation

1. Patients with suspected infection on admission 2. Patients transferred from other hospitals
3. Patients with suspected infection and transferred from other hospitals

Barrier nursing

1. Not done 2. In side ward 3. Side ward if available or main ward
4. Main ward only 5. Not applicable

(continued)

Notifiable diseases

Other infections, non-hospital-
acquired

Other infections, hospital-
acquired

Disposal of infected cases

1. Not transferred 2. Special isolation unit
3. Infectious diseases hospital or ward 4. Not
applicable 5. Other - specify

Notifiable diseases

Other infections, non-hospital-
acquired

Other infections, hospital-
acquired

Terminal disinfection of isolation areas

1. Never 2. Rarely 3. Sometimes 4. Usually
5. Always 6. Not applicable or known

Notifiable diseases

Other infections, non-hospital-
acquired

Other infections, hospital-
acquired

Treatment of isolation areas

1. Routine treatment only 2. Washing with soap or
detergent 3. Washing with disinfectant 4. Formaldehyde
fumigation 5. Sulphur fumigation 6. Fogging or spraying
7. Not applicable or known

Notifiable diseases

Other infections, non-hospital
acquired

Other infections, hospital-
acquired

Time of occupation after treatment

1. Within 6 hours 2. 6-24 hours 3. Over 24 hours
4. Normally 24 hours (less in emergency) 5. Normally
over 24 hours (less in emergency)

TREATMENT OF WARD EQUIPMENT

0. Not used 1. Not treated 2. Boiled 3. Autoclaved
4. Hot air oven 5. Disinfectant 6. Disposable
7. C.S.S.D. 8. Other - specify

Syringes

General instruments

Cutting instruments

Drainage bottles

Suction tubing

Suction bottles

Tracheotomy tubes

L.P. sets

Aspiration sets

Oxygen masks

Crockery and Cutlery

Washing

1. Hand washed 2. Machine washed 3. Machine washed -
heated over 80°C 4. Hand washed and heated over 80°C
5. Disinfectant 6. Other

Drying

1. Heat 2. Drainage only 3. Linen towel 4. Drainage +
towel 5. Paper towel 6. Other - specify

Other information

Record No.

APPENDIX C

PATIENT SURVEY RECORDS

APPENDIX C1

CROSS-INFECTION SURVEY REGIONAL SURVEY

PATIENT'S INFORMATION RECORD

Hospital

--	--	--

Ward

--	--

Day)
Month and Year) of admission

--	--	--	--

--	--

Side ward number

--	--

Type of patient

--	--

Sex 1. Male 2. Female

--

Age

--	--

Hospital unit number

--	--	--	--	--	--

Date of discharge (B4) or date of survey

Days in hospital

--	--

Diagnosis 1.
2.

--	--	--	--

--	--	--	--

Operation

--	--	--

Date..... days after admission

--	--

WOUND

0. No wound 1. Clean, not drained 2. Clean, drained
3. Contaminated, not drained 4. Contaminated, drained
5. Not known 6. Excluded 7. Clean-contaminated, not
drained 8. Clean-contaminated, drained

--

TYPE OF DRAIN

1. 'Redivac' 2. Corrugated 3. Large tube 4. Wick
5. Small tube 6. More than one drain (of different types)

--

DESCRIPTION

0. Not infected 1. Margin red with serous discharge
2. Purulent 3. Sinuses or fistulae 4. Wound break down
5. Deep abscess 6. Cellulitis 7. Previously infected
(now clean or healed) 8. Haematoma 9. Slough

--

--

--

--

SEVERITY OF INFECTION

1. Doubtful 2. Mild 3. Moderate 4. Severe 5. Drain wound only - mild

SOURCE OF INFECTION

1. Ward 2. Theatre 3. Ward or theatre 4. Self 5. Unknown

PYREXIA

0. No 1. Yes 100°F 2. Not known

Onset of infection (days after operation)

OTHER INFECTIONS

A.

B.

C.

ACQUIRED IN HOSPITAL

0. No acquired infection 1. A 2. B 3. C 4. A + B
5. B + C 6. A + C 7. A + B + C

BACTERIOLOGY

1. Site

Organisms

2. Site

Organisms

Antibiotic sensitivity of *Staph. aureus*

0. Not applicable 1. Sensitive 2. Resistant to penicillin only
3. Resistant to one antibiotic other than penicillin
4. Resistant to two or more antibiotics (no *Staph. aureus* - leave blank)

Site 1

Site 2

CHEMOTHERAPY

0. None 1. Penicillin 2. Amp. 3. Clox 3. Sulphon.
5. Strep. 6. Tetra. 7. Neo. 8. Nitrofurantoin
9. Meth.
A. Eryth. B. Novo. C. Poly. D. Kana. E. Ceph.
F. Fuc. G. Linco. H. Topical antibiotic
K. Chloramphenicol L. Gentamicin M. Carbenicillin

<input type="checkbox"/>
<input type="checkbox"/>
<input type="checkbox"/>
<input type="checkbox"/>

ASSOCIATED FACTORS

1. Immuno-suppressive drugs 2. Irradiation 3. Steroids
4. Diabetes 5. Uraemia 6. Obesity 7. Malnutrition
8. Agammaglobulinaemia 9. Other - specify

<input type="checkbox"/>
<input type="checkbox"/>

Haemoglobin

<input type="checkbox"/>

Procedures

<input type="checkbox"/>
<input type="checkbox"/>

Isolation (Regional survey)

0. Nil 1. Side-ward 2. Ventilated side-ward
3. Barrier-nursing in main ward

<input type="checkbox"/>

Other information

<input type="checkbox"/>	<input type="checkbox"/>
--------------------------	--------------------------

Record No.

<input type="checkbox"/>	<input type="checkbox"/>
--------------------------	--------------------------

APPENDIX D

TYPE OF PATIENTS AND OPERATIONS

IN THE SURVEY

APPENDIX D1

TYPE OF PATIENTS IN THE SURVEY

Type of patients	Number of patients	Nasal colonisation rate
Geriatric (surgical)	85	42.35%
Burns	44	22.73%
Geriatric (medical)	264	21.21%
Infectious diseases (children)	70	18.57%
Medical (chests)	121	14.05%
Neurosurgical	60	11.67%
General surgical and orthopaedic	288	11.46%
General medical	2111	10.52%
Dermatology and dental	58	10.34%
Intensive care	40	10.00%
Surgical and medical	217	9.22%
General surgical	2711	9.18%
Tuberculosis	22	9.09%
Orthopaedic	789	8.24%
Surgical (thoracic)	144	7.64%
Trauma	357	7.00%
Radiotherapy	106	6.60%
Paediatric (surgical)	107	5.61%
Metabolic	38	5.26%
Infectious diseases (adults)	107	4.67%
Medical and ophthalmic	23	4.35%
Paediatric (medical)	118	4.24%
Ear, nose and throat (E.N.T.)	289	3.81%
Obstetric	720	3.75%
Paediatric (medical and surgical)	214	2.80%
Gynaecological	545	2.20%

Type of patients	Number of patients	Nasal colonisation rate	
Medical and orthopaedic	55	1.82%	
Urological	112	1.79%	
Ophthalmic	166	1.20%	
Gynaecological and orthopaedic	43	0.00%	
Neurosurgical and ophthalmic	26		
Infectious diseases (babies under one year)	26		
Diabetes	20		
Surgical and E.N.T.	9		
Premature and special baby unit	6		
Surgical and gynaecological	3		
Medical and thoracic surgery	2		
Unknown	57		38.60%
All patients	10,173		8.70%

Summary for selected groups of patients

All geriatrics	349	26.36%
All medical (excluding geriatrics and paediatrics)	2421	10.29%
All surgical (excluding geriatric and paediatric)	3420	9.36%
All infectious diseases and isolation	225	8.89%
All orthopaedic and trauma	1201	7.58%
All paediatrics	439	3.87%
All obstetric and premature or special baby unit	726	3.72%
All ear, nose and throat	298	3.69%
All gynaecological	591	2.03%
All ophthalmic	192	1.04%

APPENDIX D2

TYPE OF OPERATIONS IN THE SURVEY

(Figures in brackets show the wound infection rates for patients having those operations which are contained within each of the different operation headings).

Type of operation	Number of patients	Number with an infected wound
NEUROSURGERY (6.41%)		
Brain and cerebral meninges	26	1
Spinal chord and spinal meninges	38	4
Peripheral nerves and sympathetic system	14	0
OPERATIONS in ENDOCRINE SYSTEM (2.38%)		
Thyroid and parathyroid	29	1
Adrenals	5	0
Pituitary	8	0
OPHTHALMIC OPERATIONS (0.83%)		
Orbit and supporting structures of eyeball	24	1
Conjunctiva	2	0
Cornea	6	0
Iris and cillary body	33	0
Sclera, choroid and retina	20	0
Lens	36	0
OPERATIONS on EAR, NOSE and THROAT (15.94%)		
Otological operations	25	2
Nose and accessory air sinuses	7	0
Larynx and trachea	37	9

Type of operation	Number of patients	Number with an infected wound
OPERATIONS on BUCCAL CAVITY and OESOPHAGUS (11.11%)		
Pharynx, tongue, palate and buccal cavity	5	1
Oesophagus	16	2
Salivary glands	6	0
THORACIC SURGERY (18.95%)		
Heart and pericardium	44	3
Great vessels	5	3
Lung, bronchus and mediastinum	46	12
OPERATIONS on BREAST (12.94%)	85	11
GASTRO-INTESTINAL and ABDOMINAL SURGERY (21.25%)		
Abdominal wall	274	26
Stomach	189	30
Appendix	238	57
Intestines (except appendix and rectum)	169	62
Rectum and anus	56	30
Liver and bile ducts	138	21
Pancreas	1	1
Spleen	3	0
GENITO-URINARY SURGERY (22.47%)		
Kidney	40	9
Ureter	8	4
Urinary bladder	24	3
Urethra	4	1
Prostate and seminal vesicles	91	21
Other male genital organs	60	13

Type of operation	Number of patients	Number with an infected wound
GYNAECOLOGICAL OPERATIONS (12.57%)		
Ovary	62	4
Uterus and supporting structures	120	16
Vagina	2	0
Vulva and perineum	7	4
OBSTETRIC OPERATIONS (6.78%)		
ORTHOPAEDIC SURGERY (10.68%)		
Bone	366	33
Joints, cartilages and bursae	173	8
Muscles, tendons and fascia	45	0
Upper limb amputations and disarticulations	3	0
Lower limb amputations and disarticulations	87	31
OPERATIONS on PERIPHERAL BLOOD VESSELS and LYMPHATIC SYSTEM (11.79%)		
Arteries (except great vessels of thorax)	38	5
Veins (except great vessels of thorax)	63	1
Lymphatic system	19	4
Operations on skin and subcutaneous tissues	109	17
OTHER SURGICAL PROCEDURES		
Localised surgical procedures (with site unspecified)	15	0
<u>ALL OPERATIONS and SURGICAL PROCEDURES (15.27%)</u>		
All operations and surgical procedures	2980	455

APPENDIX E

VALIDATION OF DATA

APPENDIX E1

LISTING OF VARIABLES USED IN THE PROCESSING, SELECTION AND
VALIDATION OF PATIENT DATA FOR BOTH 'NASAL ACQUISITION RATE'
AND 'WOUND INFECTION RATE' MODELS

PX	-	Hospital and ward IDENTIFICATION codes
I3	-	Day of patient survey
I4	-	Month of patient survey
IPYEAR	-	Year of patient survey
I5	-	Side-ward number
I6	-	Type of patient
I7	-	Sex of patient
I8	}	- AGE of patient
I9		
I10	-	Hospital unit number
I11	-	TOTAL number of days spent in hospital
I12	-	Diagnosis (1)
I13	-	Diagnosis (2)
I14	-	Type of OPERATION
I15	-	Number of days spent in hospital (BEFORE operation)
I16	-	Type of WOUND
I17	-	Type of DRAIN
I18	-	Description of wound (A)
I19	-	Description of wound (B)
I20	-	Description of wound (C)
I21	-	Description of wound (D)
I22	-	Severity of infection
I23	-	Source of infection
I24	-	Pyrexia (of unknown origin)
I25	-	Onset of infection (number of days after operation)

- I26 - Other infections (A)
- I27 - Other infections (B)
- I28 - Other infections (C)
- I29 - Is infection acquired in hospital?
- I30 - BACTERIOLOGY - site 1 (location)
- I31 - BACTERIOLOGY - organisms in site 1
- I32 - BACTERIOLOGY - site 2 (location)
- I33 - BACTERIOLOGY - organisms in site 2
- I34 - Antibiotic SENSITIVITY of Staph. aureus - site 1
- I35 - Antibiotic SENSITIVITY of Staph. aureus - site 2
- I36 - Chemotherapy (A)
- I37 - Chemotherapy (B)
- I38 - Chemotherapy (C)
- I39 - Chemotherapy (D)
- I40 - ASSOCIATED FACTORS (1)
- I41 - ASSOCIATED FACTORS (2)
- I42 - Haemoglobin
- I43 - Procedures (1)
- I44 - Procedures (2)
- I45 - Isolation
- AA(530) - Array containing hospital and ward IDENTIFICATION codes
- NAB(530,80) - Array containing information on either:
 - Ward STRUCTURES and FACILITIES, or
 - Ward PRACTICES I, or
 - Ward PRACTICES II

depending on which main programme this selection routine is attached to
- IWYEAR - Year of the WARD survey
- NOWARD - Number of patients whose records CANNOT be 'matched' with a particular ward

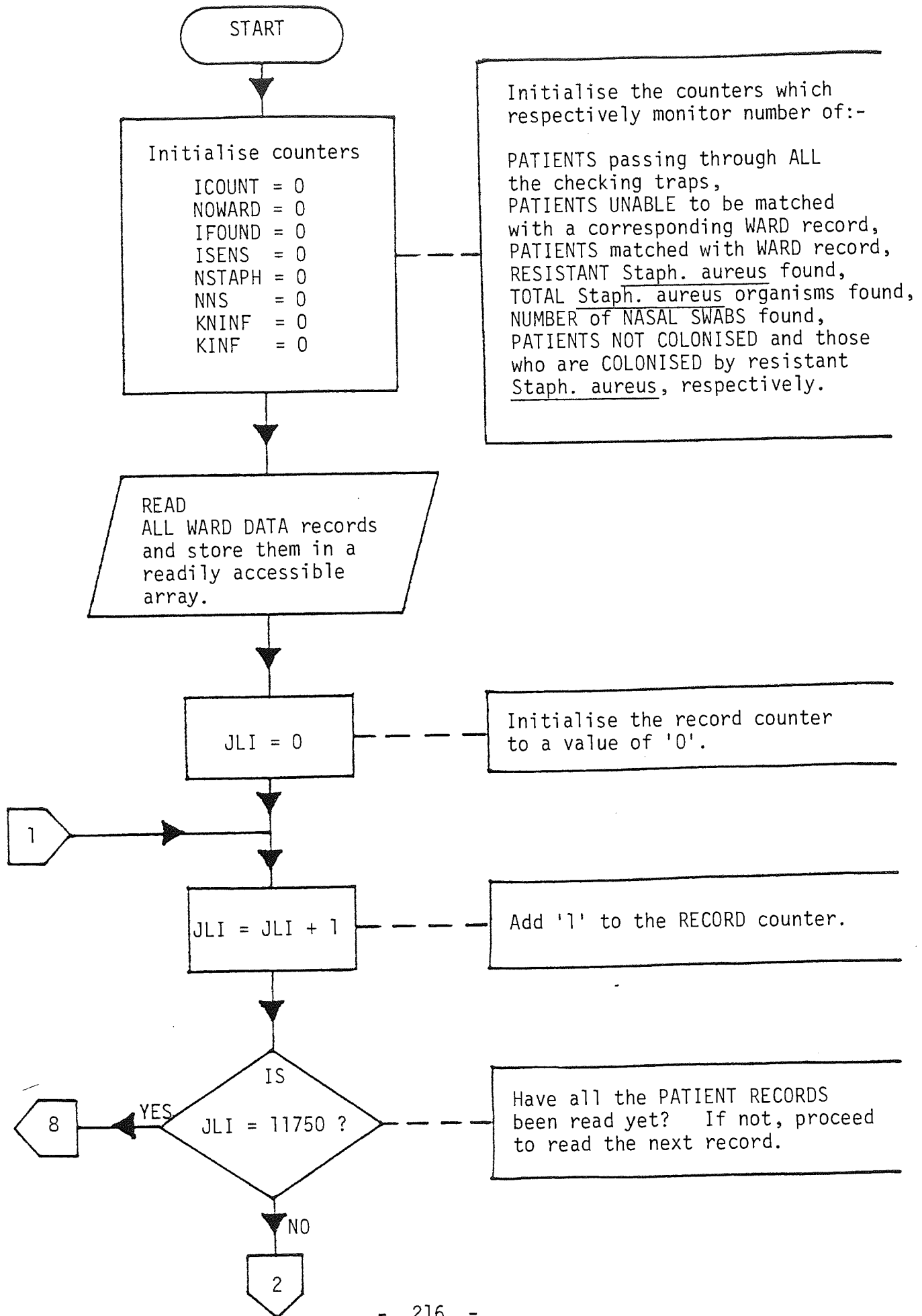
IFOUND	-	Number of patients whose records CAN be 'matched' with a specific ward
ISW1	-	Number of nasal swabs found in SITE 1
ISW2	-	Number of nasal swabs found in SITE 2
NSTAPH	-	Total number of <u>Staph. aureus</u> organisms found
ISENS	-	Number of <u>Staph. aureus</u> resistant to '1' or '2' antibiotics other than penicillin
NWOUND	-	Number of patients found NOT to have WOUNDS
IWOUND	-	Number of patients recorded as having WOUNDS
NNS	-	Number of nasal swabs found
KNINF	-	Number of patients NOT COLONISED by resistant <u>Staph. aureus</u> (or recorded as having a <u>NON-INFECTED</u> wound)
KINF	-	Number of patients COLONISED by resistant <u>Staph. aureus</u> (or recorded as having an <u>INFECTED</u> wound)
IREJ	-	Number of records REJECTED, because NO nose swabs were found for the patient
KY	-	Data variable containing '5' for BACTERIOLOGY SITE
JZQ	-	Data variable containing '0' } Used in connection with testing for patients having antibiotic treatment
JZR	-	Data variable containing 'b' }
AGE	-	Age of patient
INFECT	-	Flag to determine whether patient is colonised by resistant <u>Staph. aureus</u>
NCH	-	Flag to determine whether records conform to varying prescribed conditions within the programme
ANTIBI	-	Flag to indicate whether patient is receiving any kind of ANTIBIOTIC treatment
SPECIAL	-	Flag to indicate whether patient has any SPECIAL RISK factors associated with his case
OCCBED	-	Number of OCCUPIED BEDS in any given ward
AREBED	-	Average FLOOR AREA per bed in any given ward
DIST	-	Average DISTANCE between bed centres in any given ward
OCCPRO	-	PROPORTION of beds in ward which are OCCUPIED

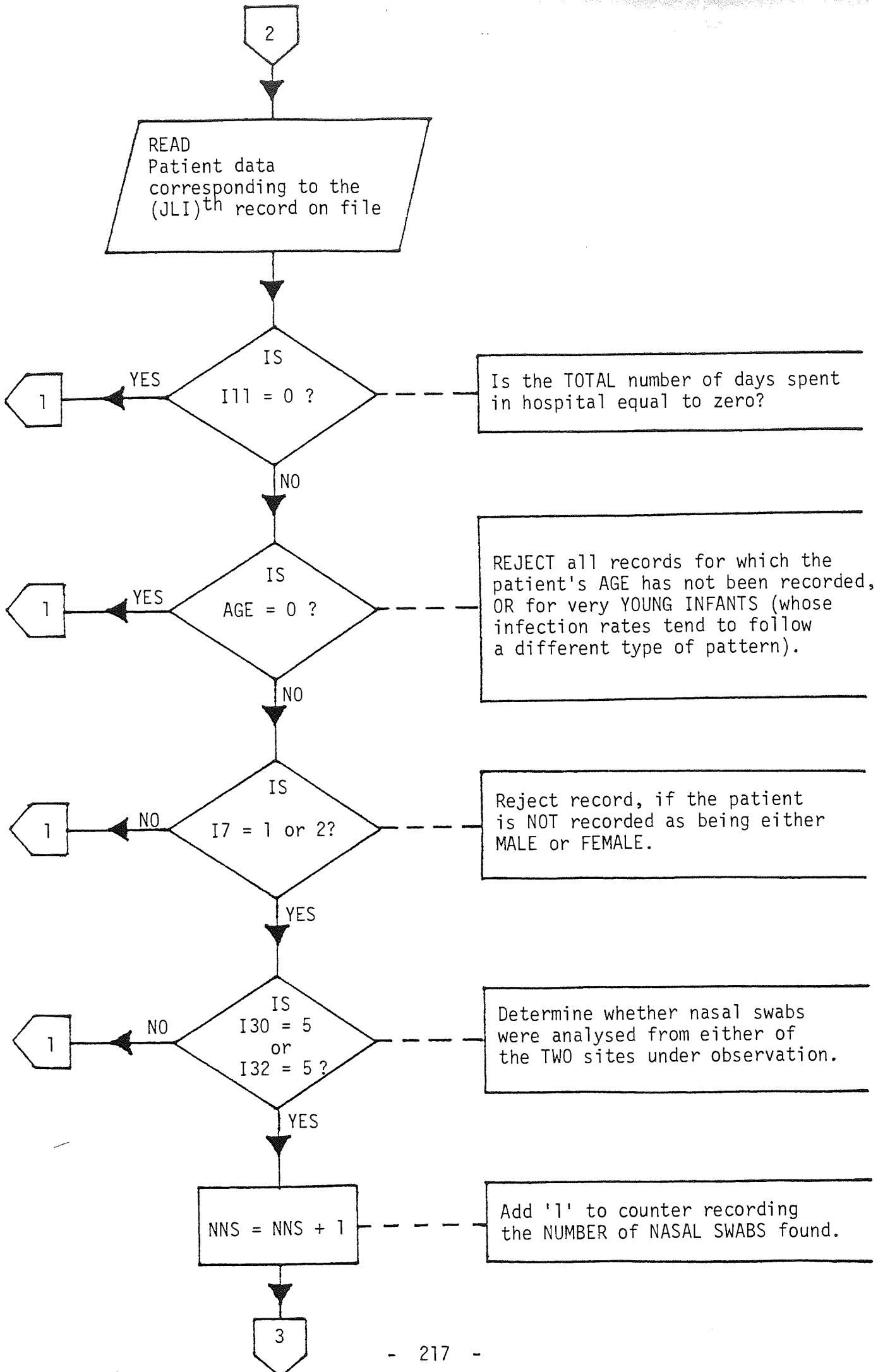
- ICOUNT - Counter to determine the NUMBER of RECORDS passing through a successive series of 'checks'

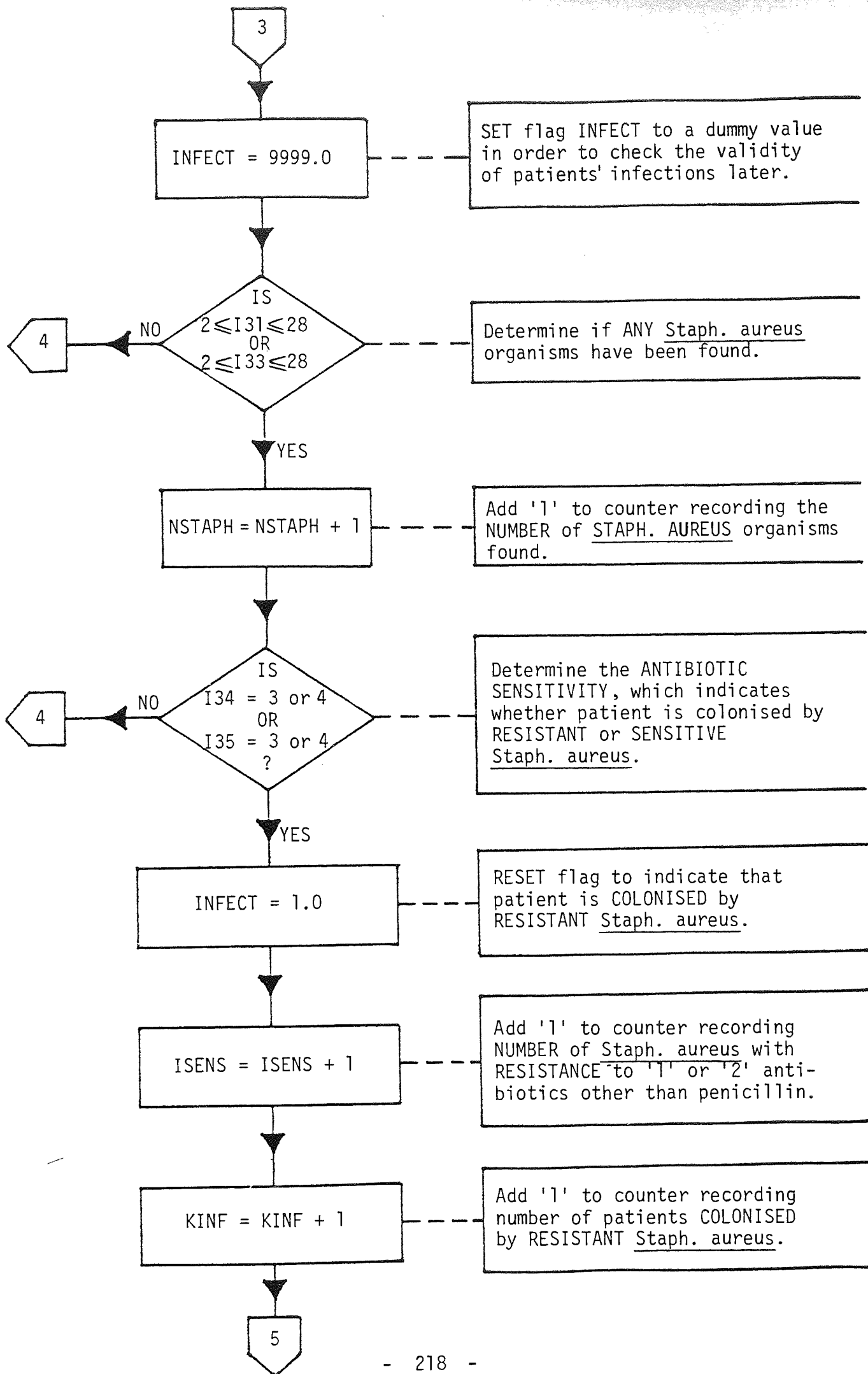
- JLI - Counter to determine the NUMBER of PATIENT RECORDS which are PROCESSED through the computer

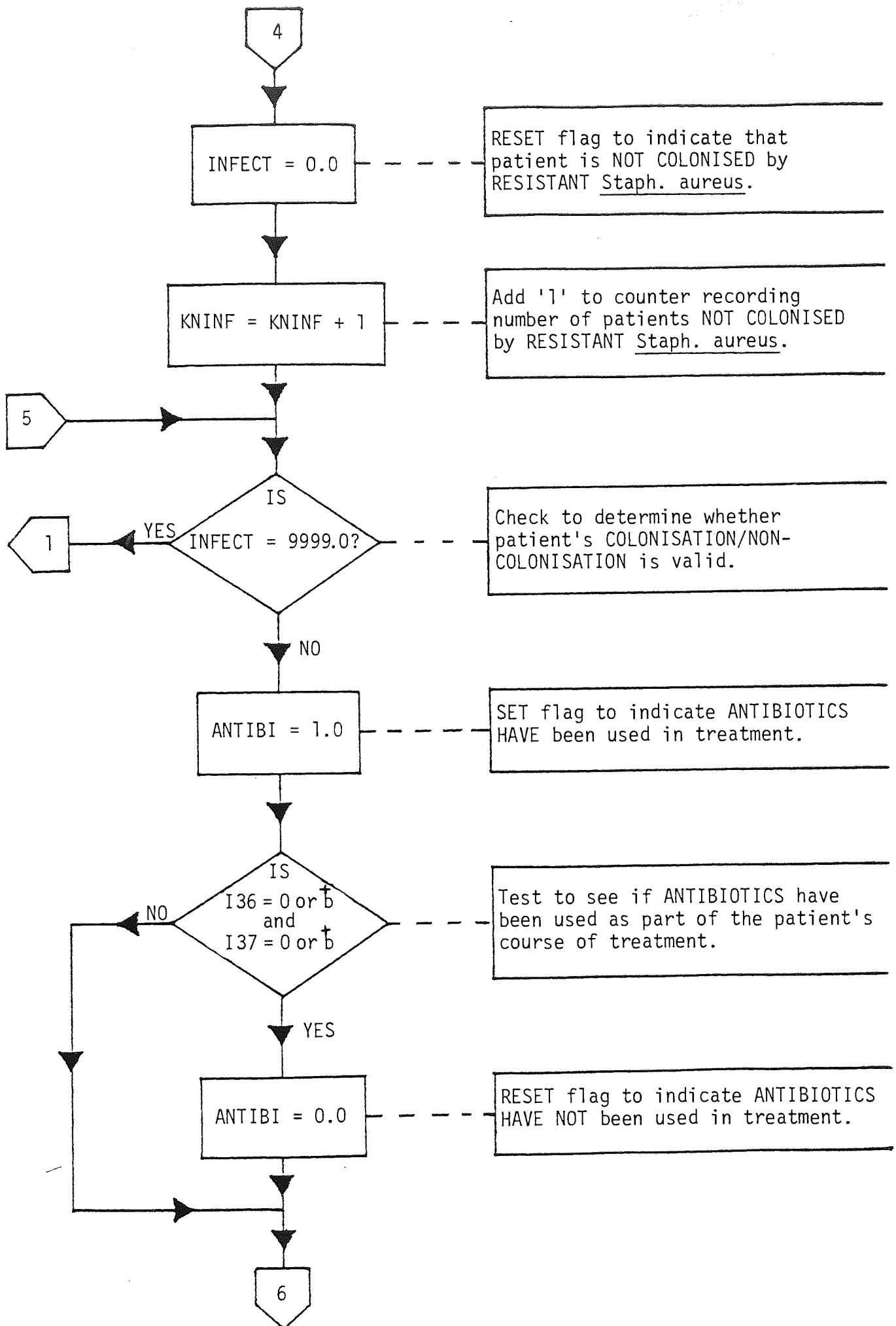
APPENDIX E2

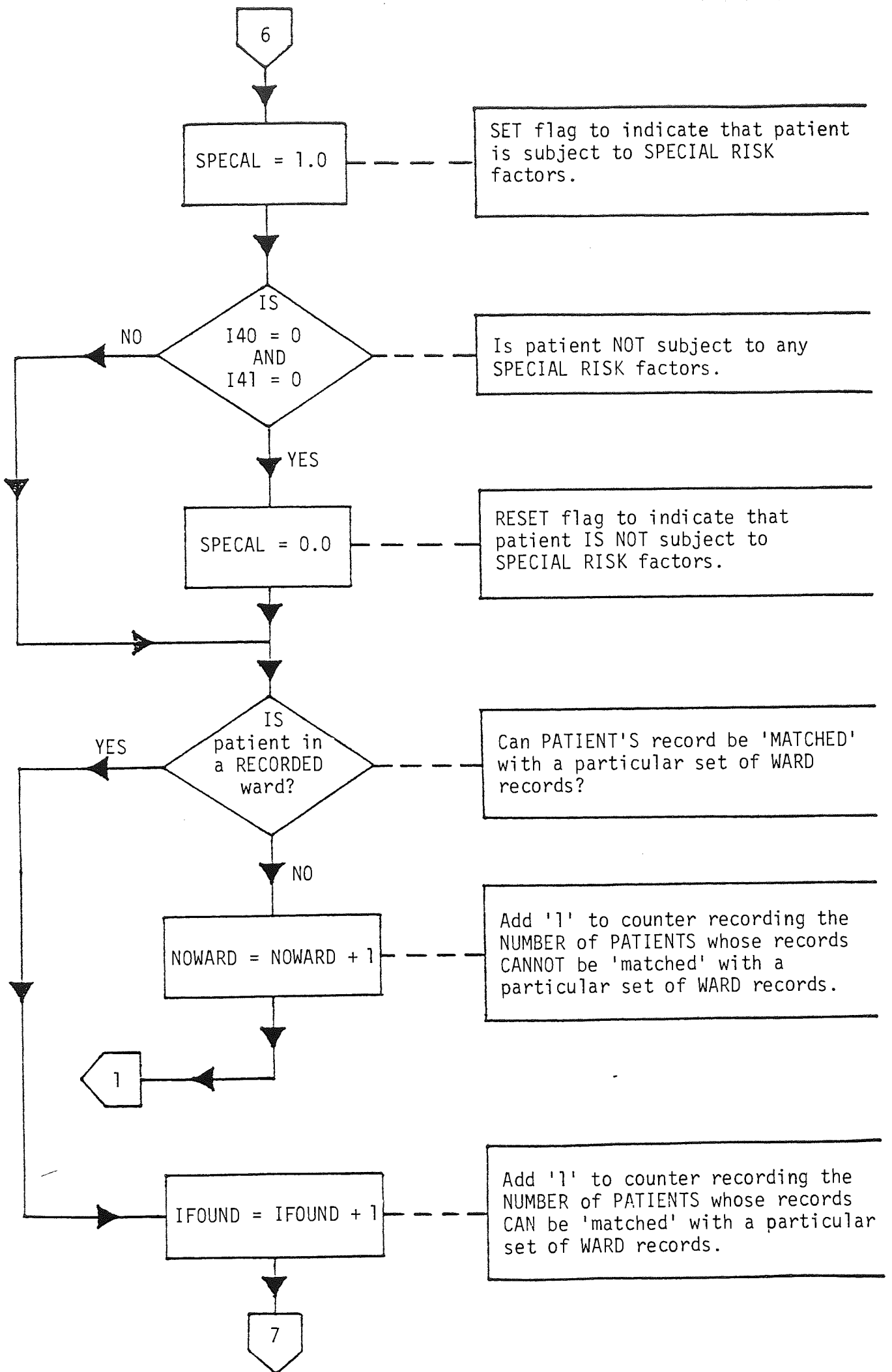
Flowchart for validation of each patient's nasal input data

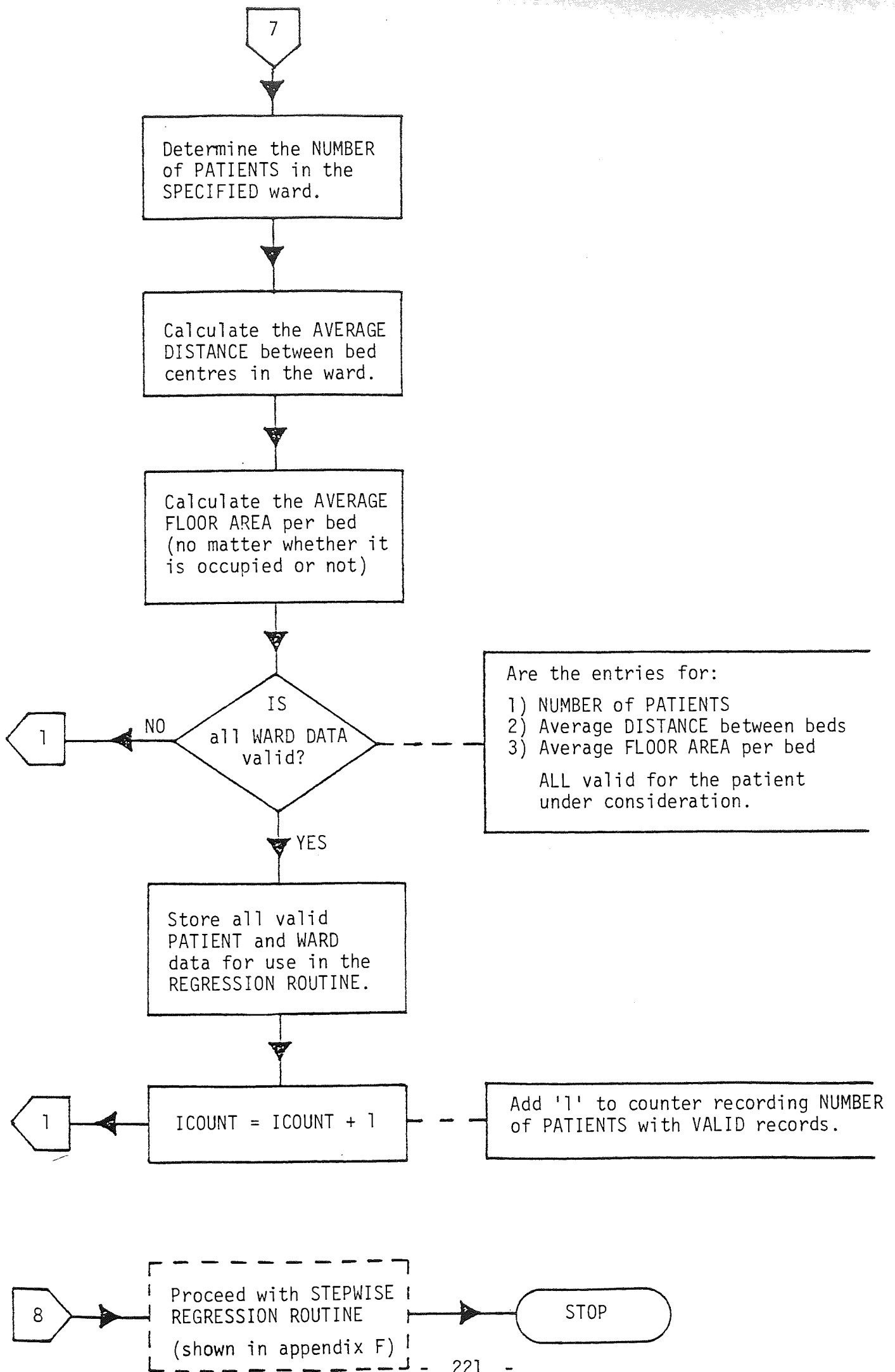






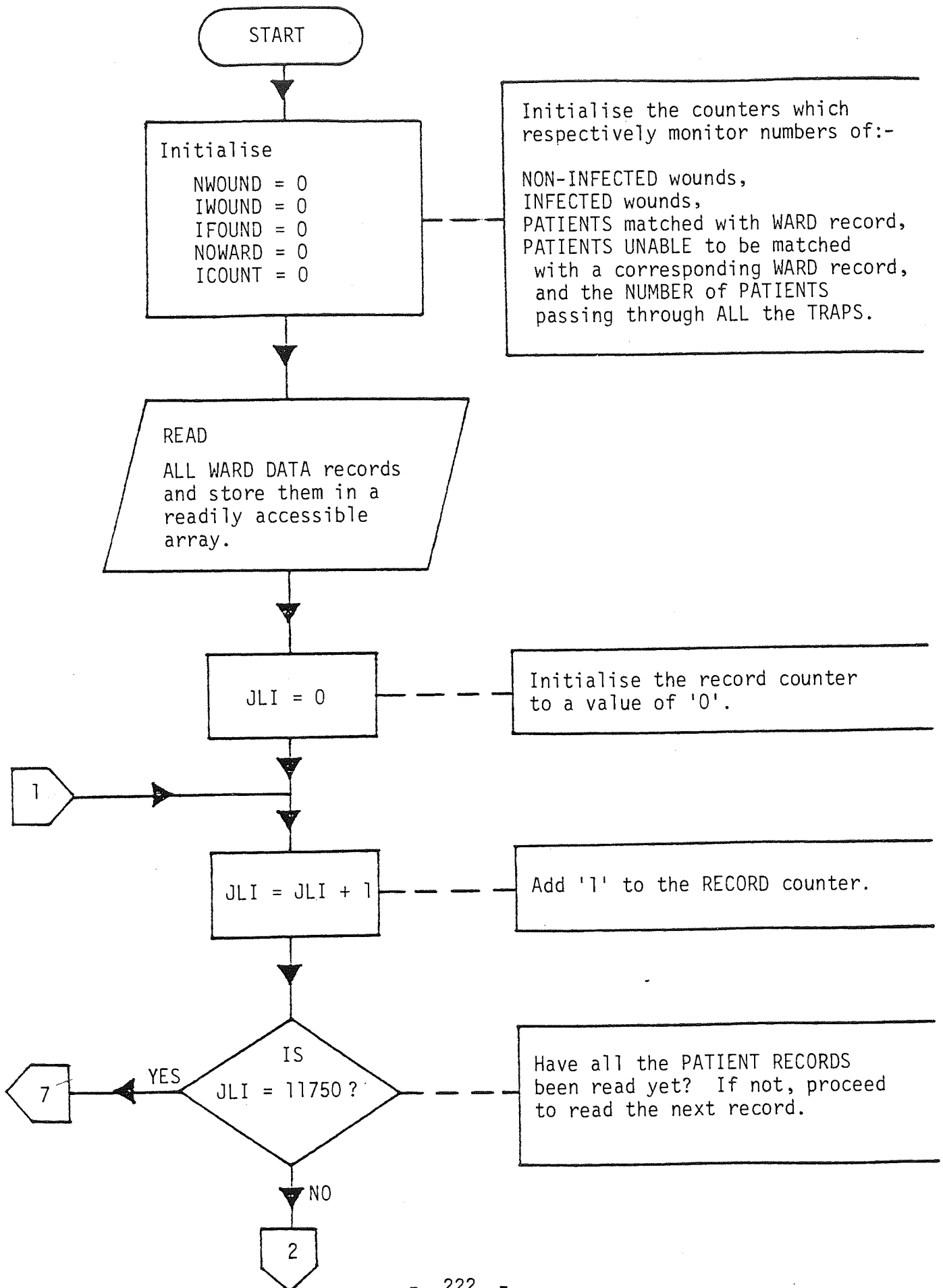


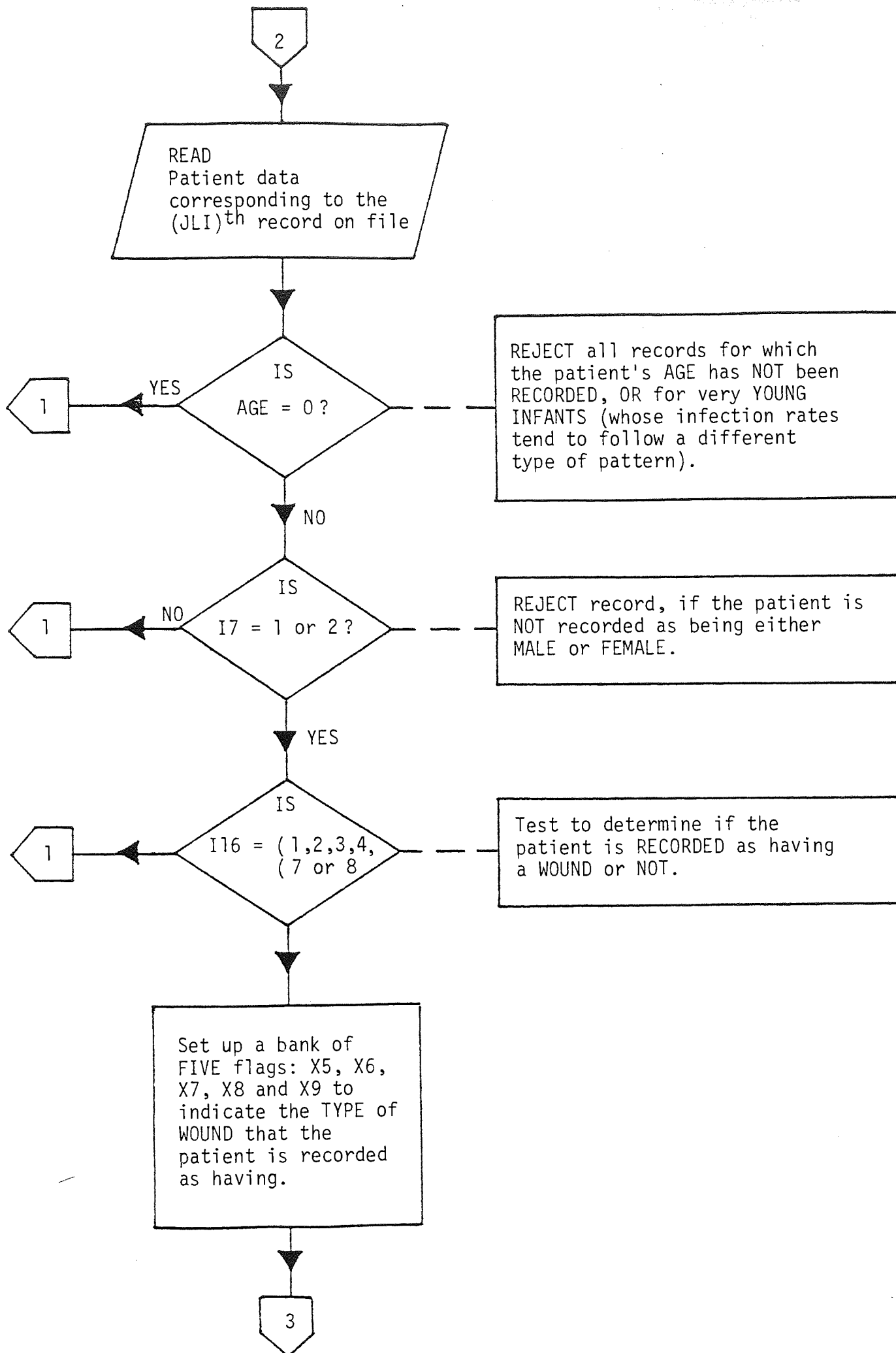


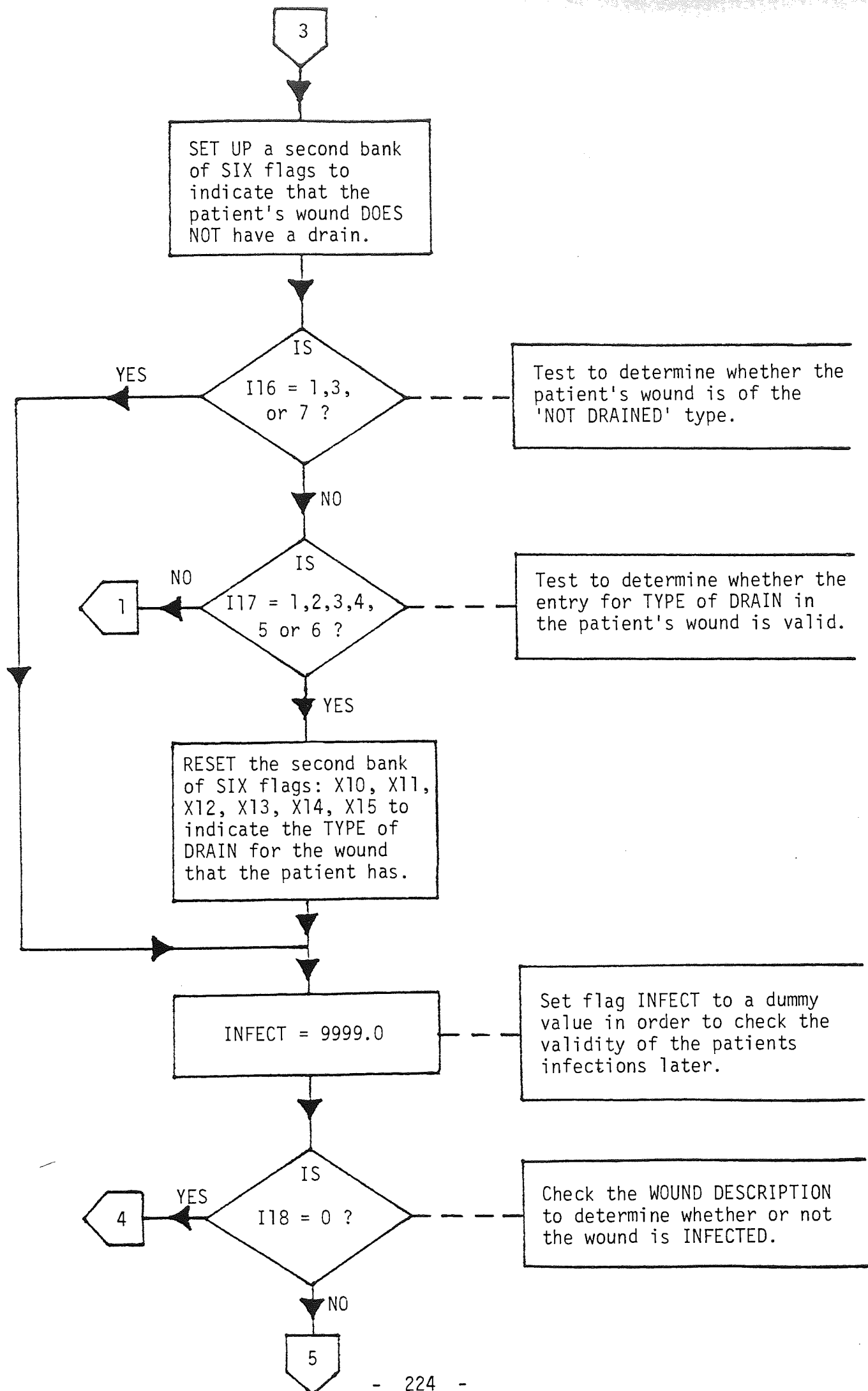


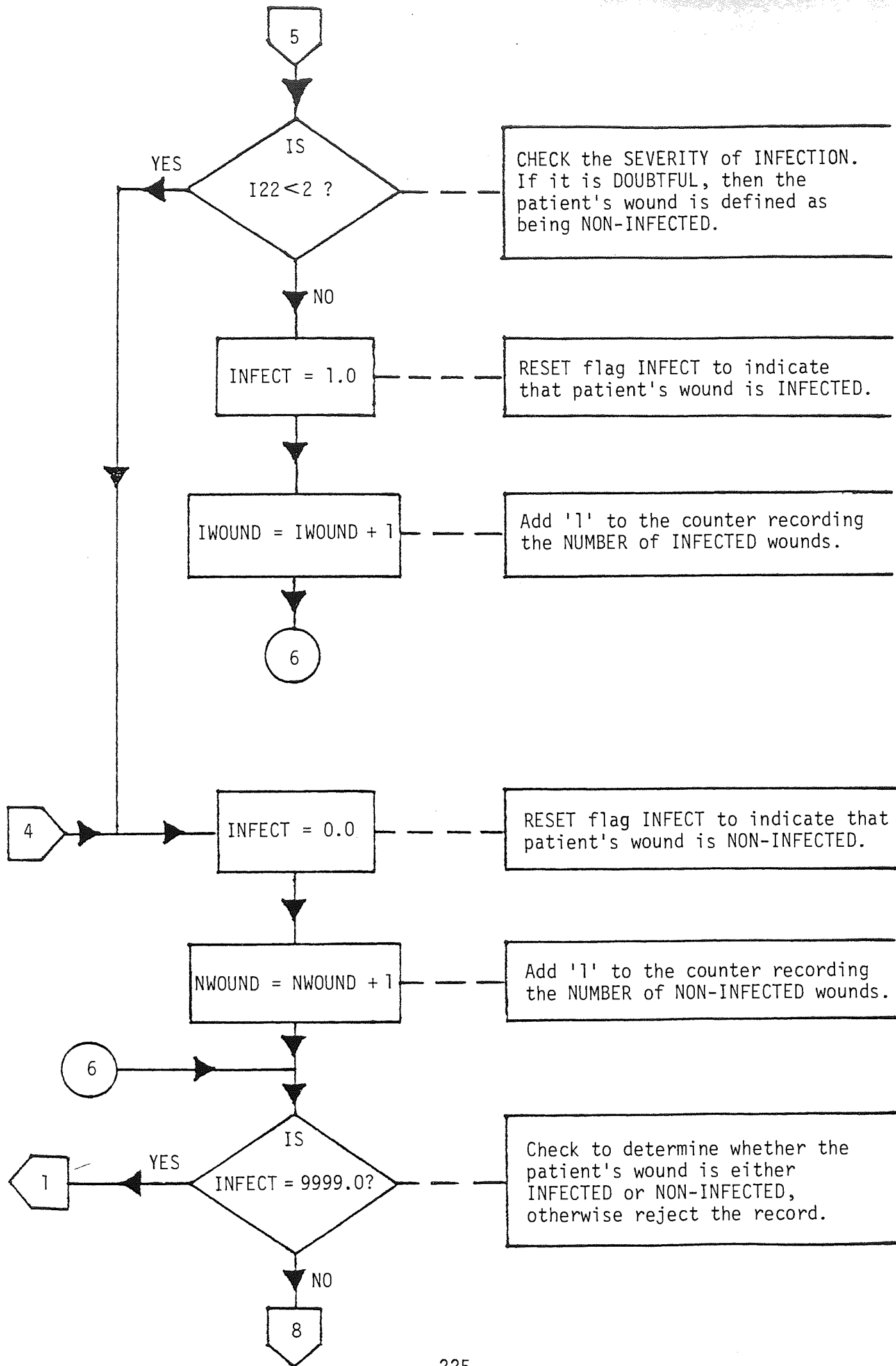
APPENDIX E3

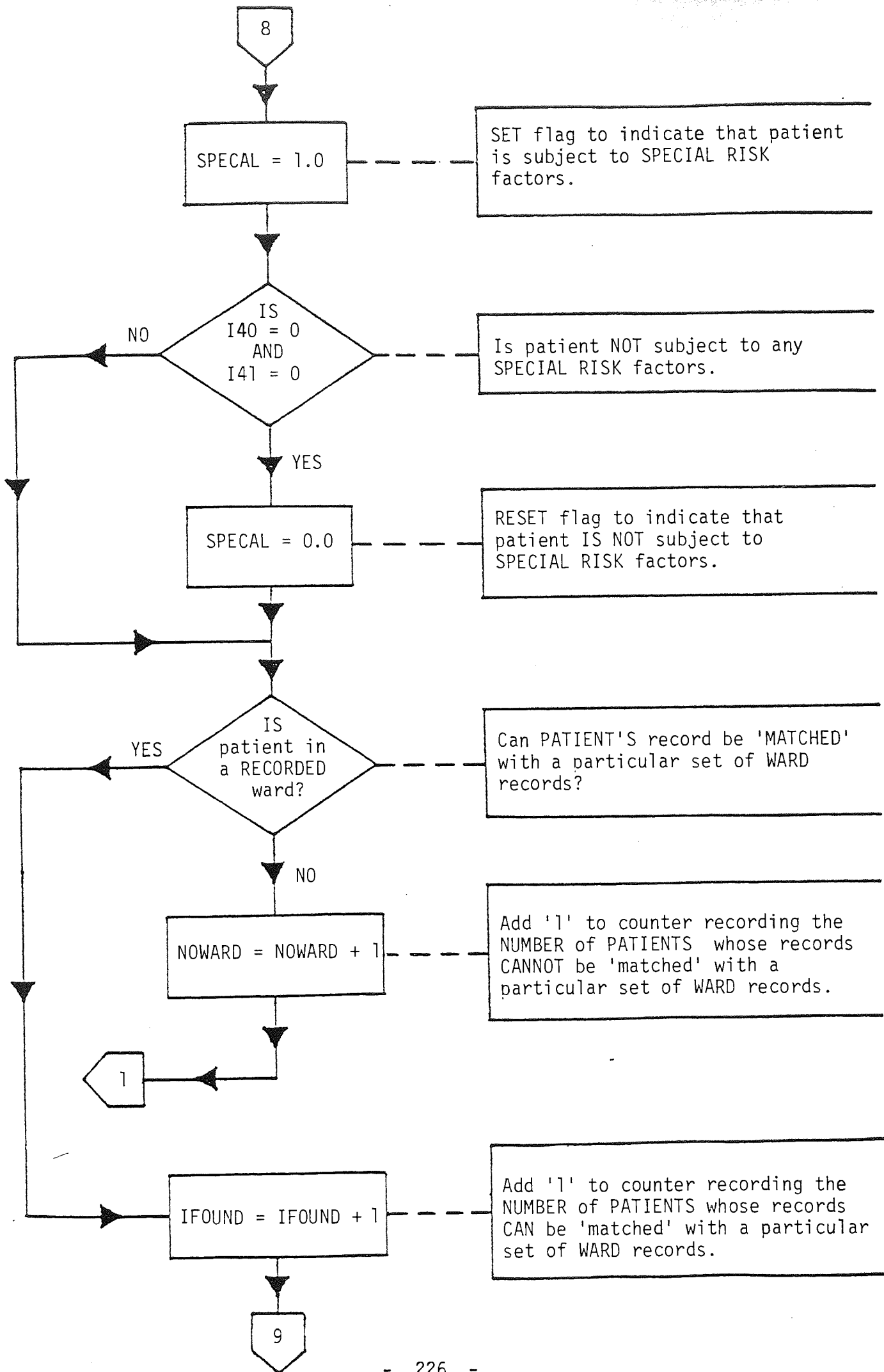
Flowchart for validation of each patient's wound input data

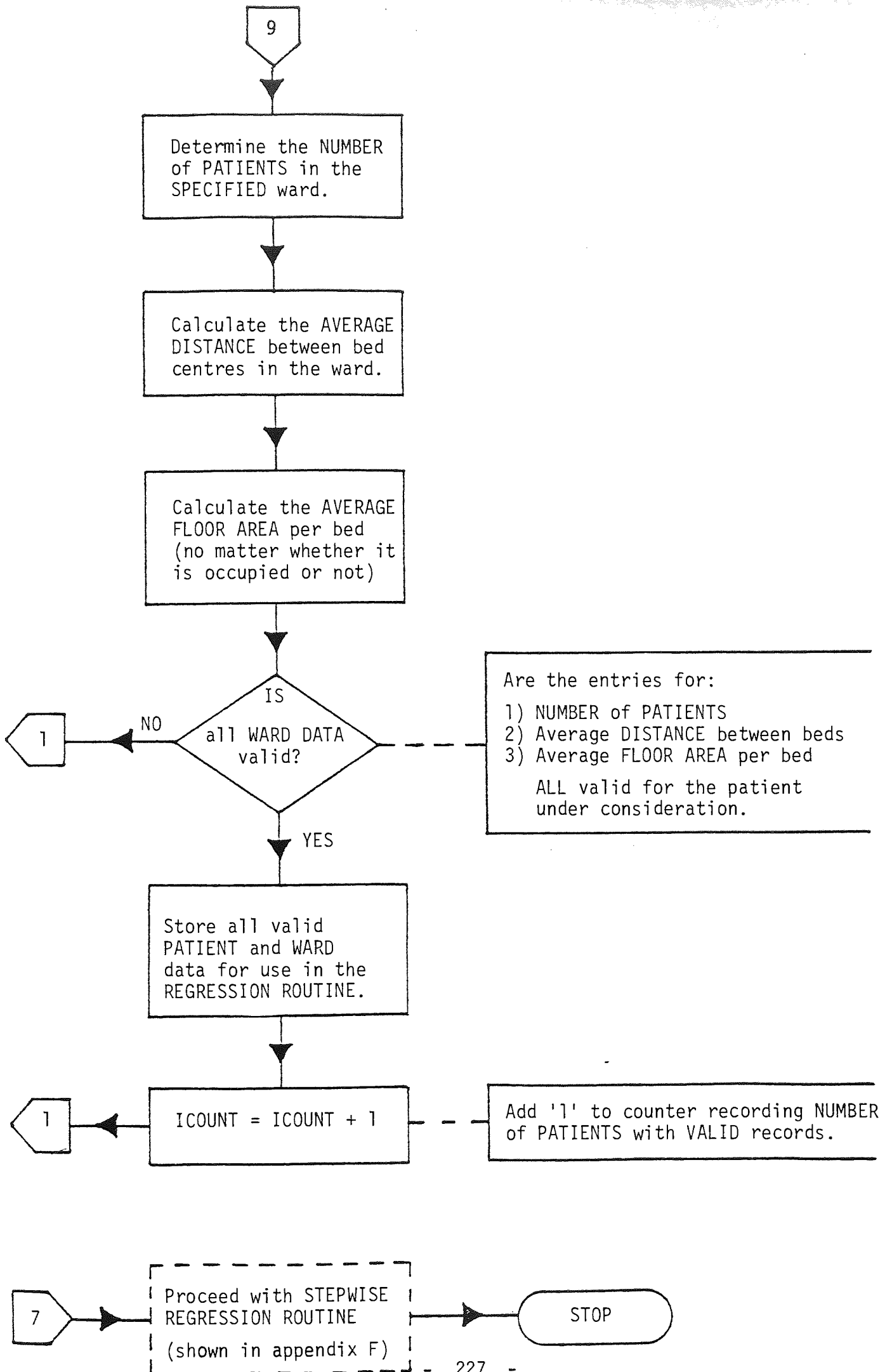












APPENDIX F

MULTIPLE REGRESSION ANALYSIS

$$\begin{bmatrix} R(K*K) & T'(K*1) & I(K*K) \\ T(1*K) & S(1*1) & 0(1*K) \\ -I(K*K) & 0(K*1) & 0(K*K) \end{bmatrix}$$

where $R(K*K)$ is the correlation coefficient for the K independent variables

$T(1*K)$ is the correlation vector for the K independent variables, with the response variable Y

$T'(K*1)$ is the transpose of T

$S(1*1)$ is the correlation of the response with itself (=1)

$I(K*K)$ is the identity matrix

$-I(K*K)$ is the negative identity matrix

- BOR(49,49) - Array containing (NEW or NEXT) CORRELATION COEFFICIENTS which are calculated (as required) from the (CURRENT) matrix of correlation coefficients, COR(49,49). When all elements of BOR(49,49) have been calculated these are channelled back into the array COR(49,49), which is then used as the NEW (CURRENT) matrix of correlation coefficients.
- S(25) - Contains elements corresponding to VAR(I,I)
- JK(24) - Flag to indicate whether I^{th} variable is in the regression model
- MK(24) - Flag to indicate whether the I^{th} variable has 'just left' the regression model
- ITABLE - Indicates which stage of the 'stepwise-regression' procedure we have currently reached by attaching the appropriate ITABLE number to its respective Analysis of the Variance Table

- JIM - Flag to indicate whether the variable that 'just entered the regression equation was the first one to do so

- TOL - TOLERANCE LIMIT, below which value, each leading diagonal element (corresponding to the independent variables) of the correlation matrix COR(49,49) may not fall below, because of possible degeneracy in the calculation of each V(I) element

- V(25) - Array containing V(I) elements, where each
$$V(I) = \frac{COR(I, NPARAM) * COR(NPARAM, I)}{COR(I, I)}$$

- VMAX - MAXIMUM value of V(I) from the array V(25)

- NMAX - Array element (variable) number corresponding to VMAX

- FIXE - Critical F-value for either REJECTION or ACCEPTANCE of any variable respectively leaving or entering the regression model

- PF(24) - Array used for storing Partial-F values corresponding to each of the variables in the current regression model

- CURRFI - Degrees of Freedom used when testing each of the Partial-F values, to determine whether any of the variables are to leave the regression equation

- QI - Degrees of Freedom associated with the TOTAL (Corrected) Sum of Squares

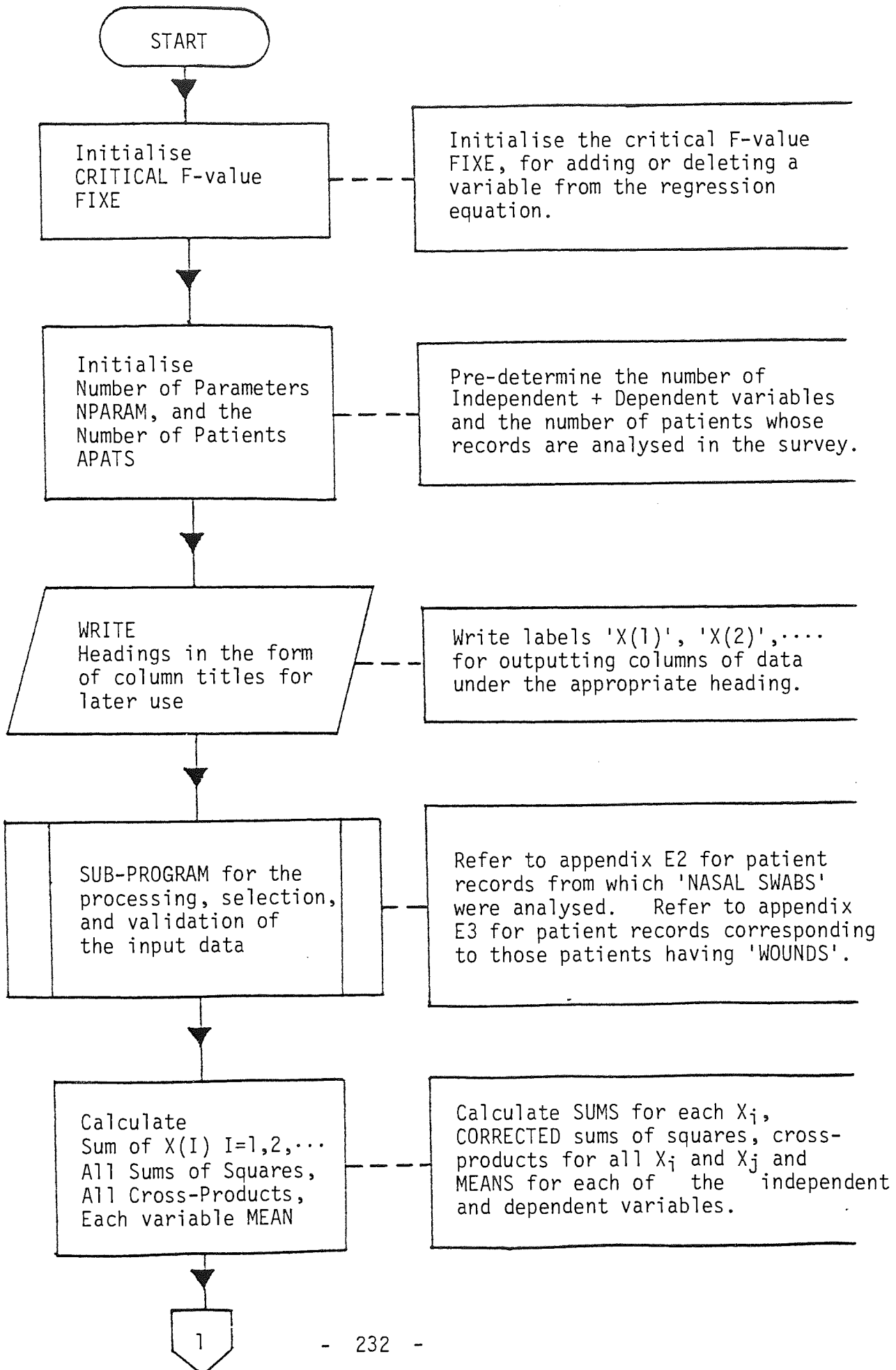
- QJ - Degrees of Freedom associated with the REGRESSION Sum of Squares

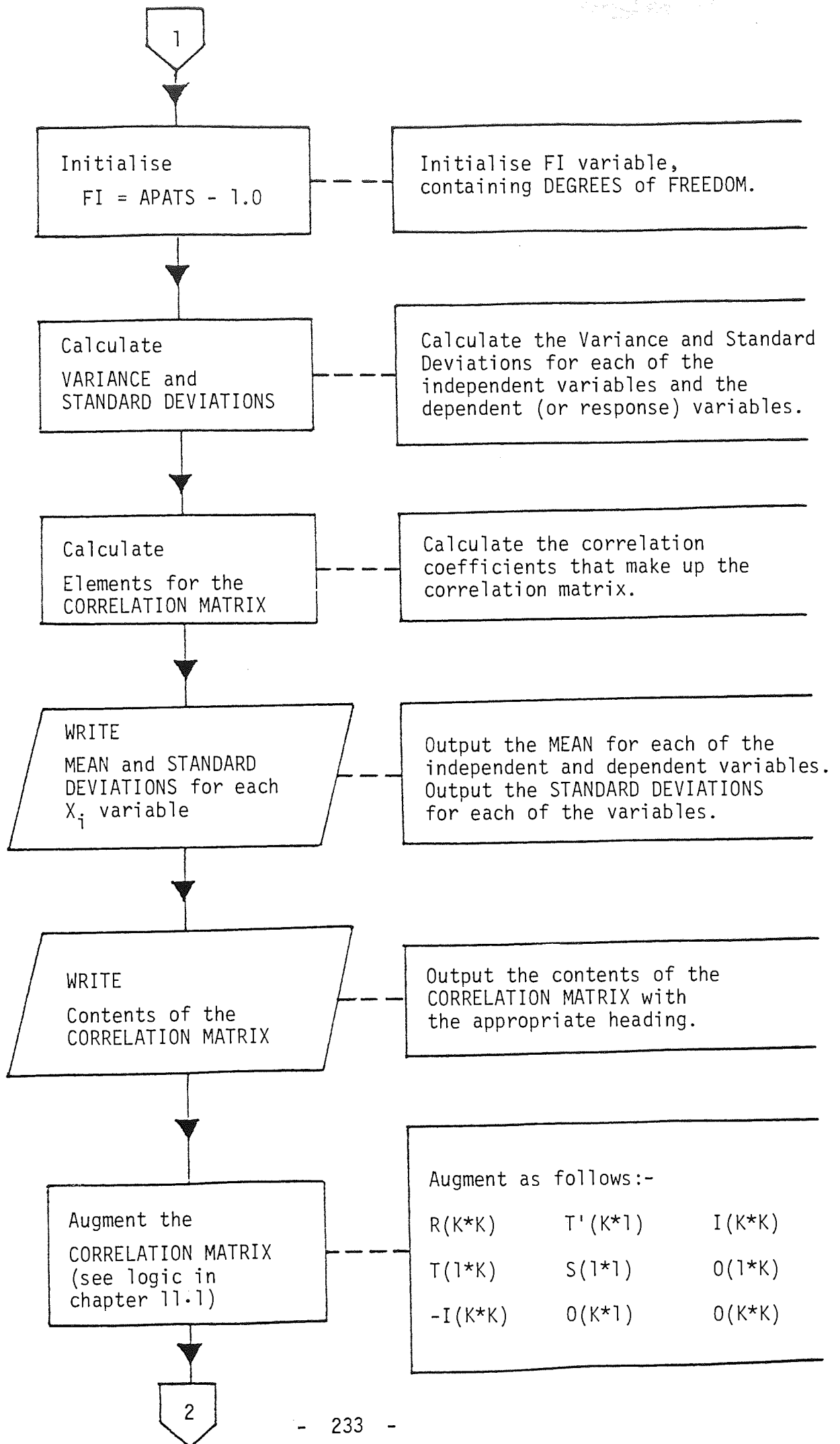
- SSR - Correlation Form for the REGRESSION Sum of Squares

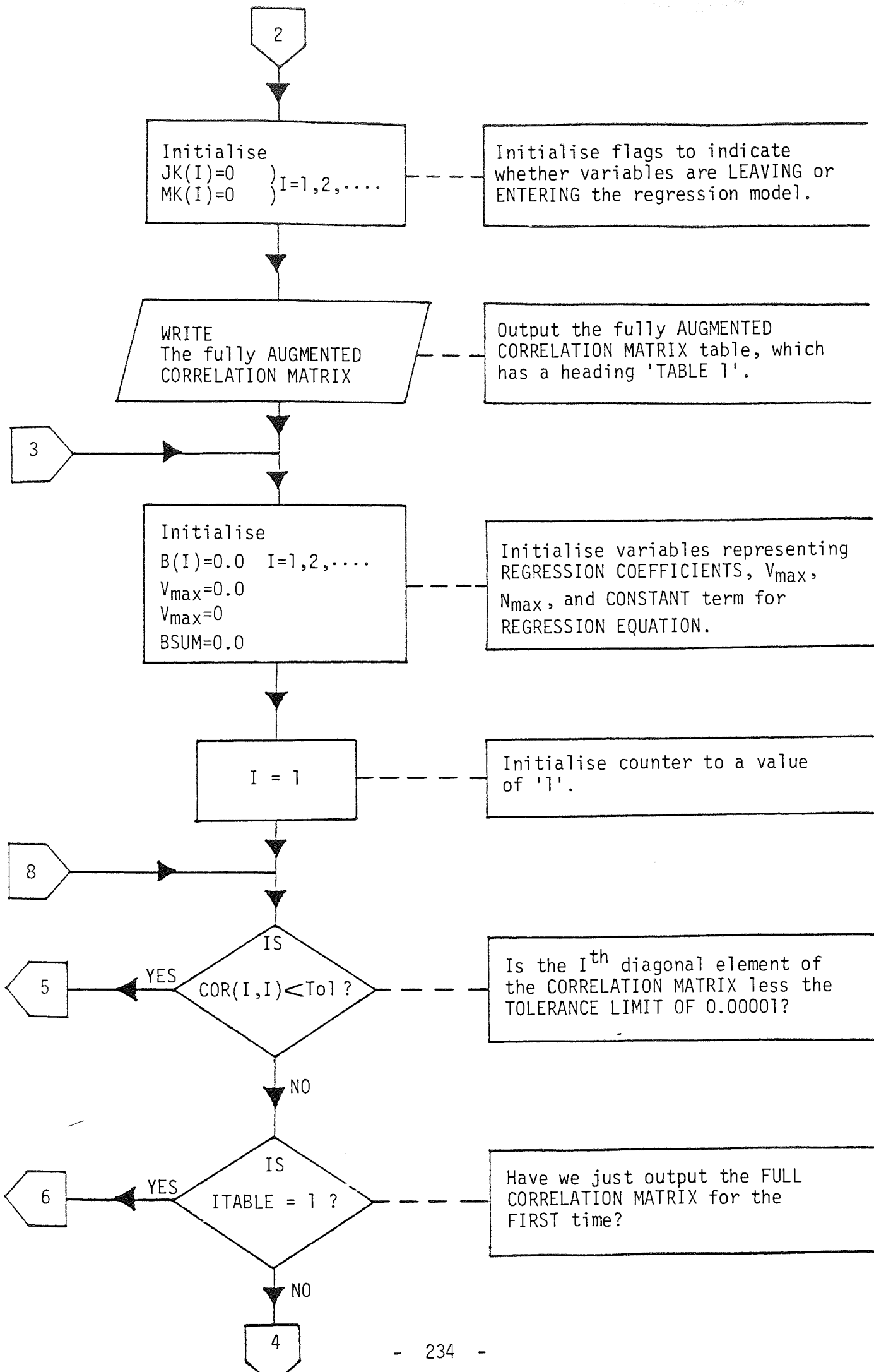
- AMSR - Correlation Form for the REGRESSION Mean Square
- F - Calculated F-value
- RSQUAR - PERCENTAGE of variation in INFECTION RATES which can be accounted for by differences between PATIENT parameters and differences within ward STRUCTURES and FACILITIES
- SSE - Correlation Form for the RESIDUAL Sum of Squares
- AMSE - Correlation Form for the RESIDUAL Mean Square
- B(25) - Array containing each of the COEFFICIENTS associated with the respective variables currently in the regression model
- DSDV(25) - Array containing the STANDARD ERRORS for each of variables currently in the regression model
- BB - CONSTANT of the final PREDICTION EQUATION which has been derived from the 'stepwise-regression' procedure

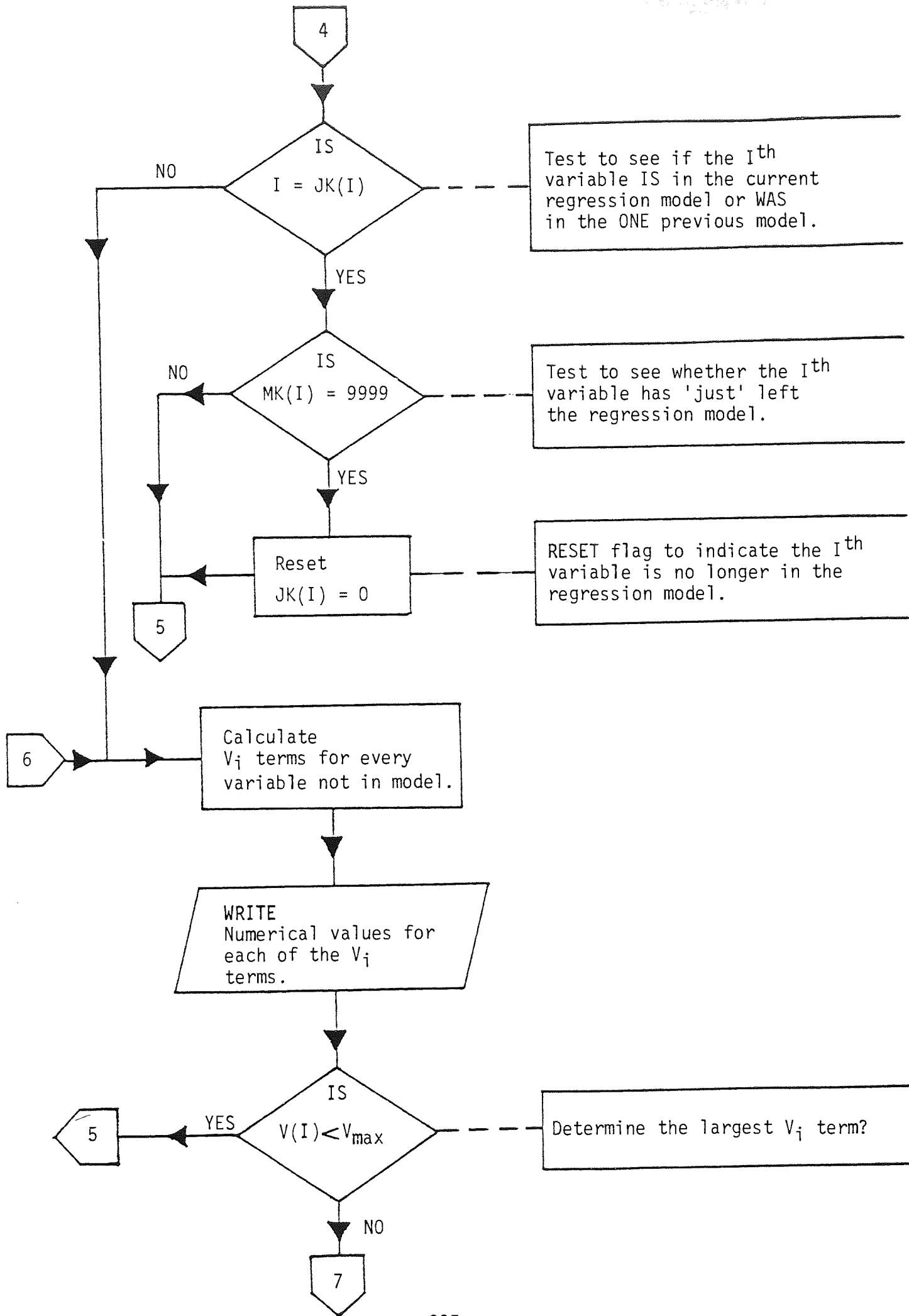
APPENDIX F2

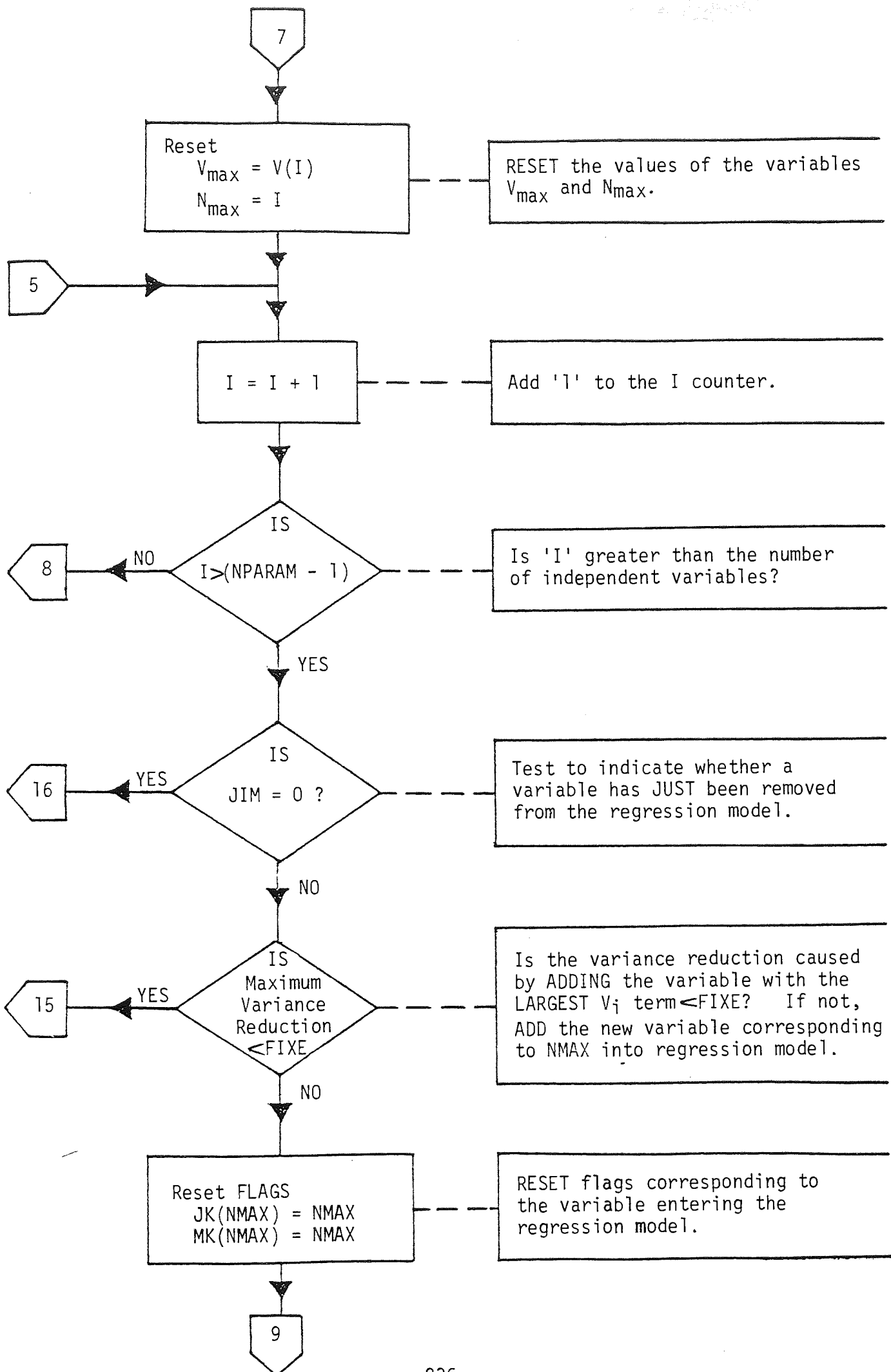
Flowchart for regression analysis program











Reset
 $V_{\max} = V(I)$
 $N_{\max} = I$

RESET the values of the variables V_{\max} and N_{\max} .

5

$I = I + 1$

Add '1' to the I counter.

8

IS
 $I > (NPARAM - 1)$

Is 'I' greater than the number of independent variables?

16

IS
 $JIM = 0 ?$

Test to indicate whether a variable has JUST been removed from the regression model.

15

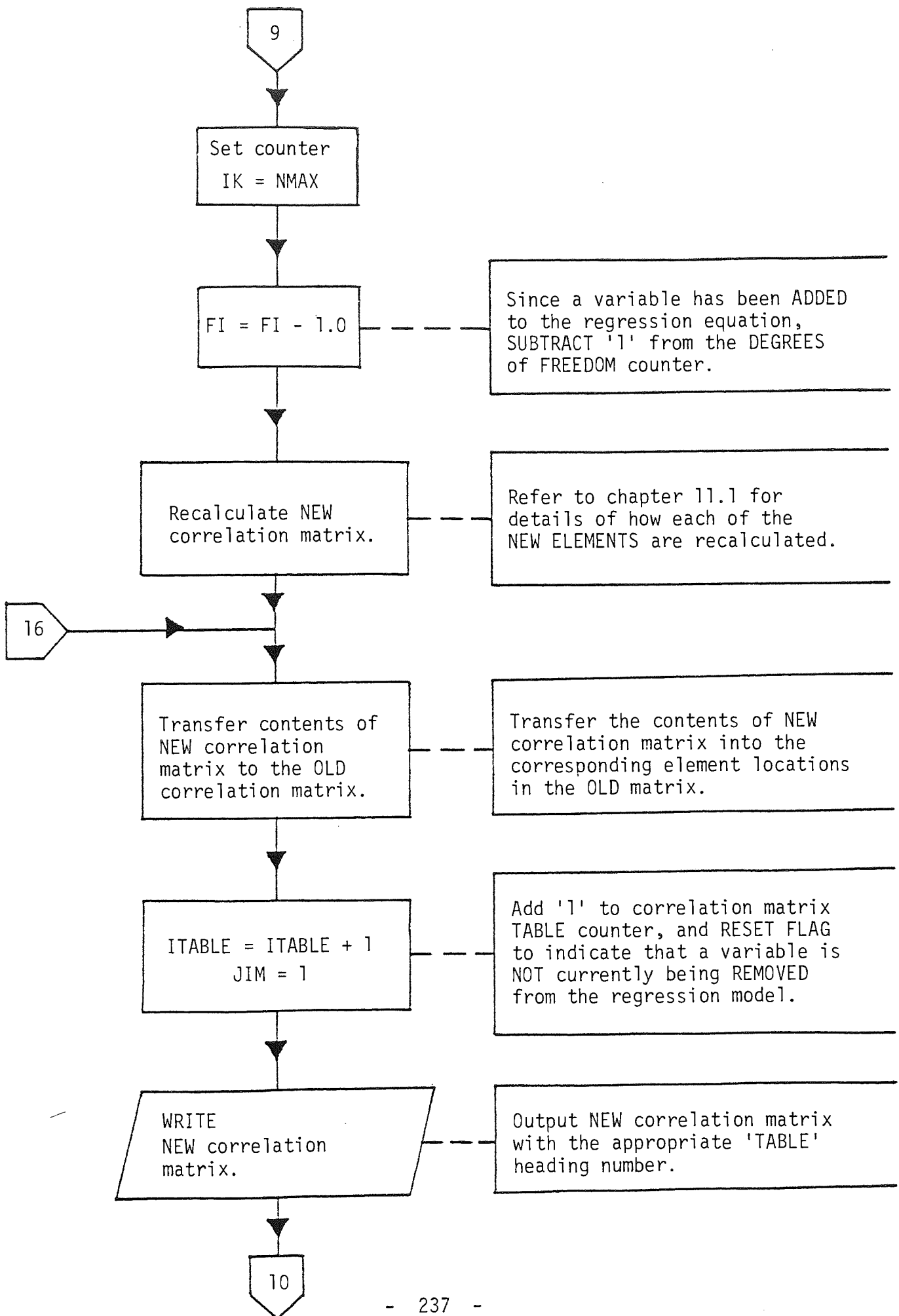
IS
 Maximum
 Variance
 Reduction
 $< FIXE$

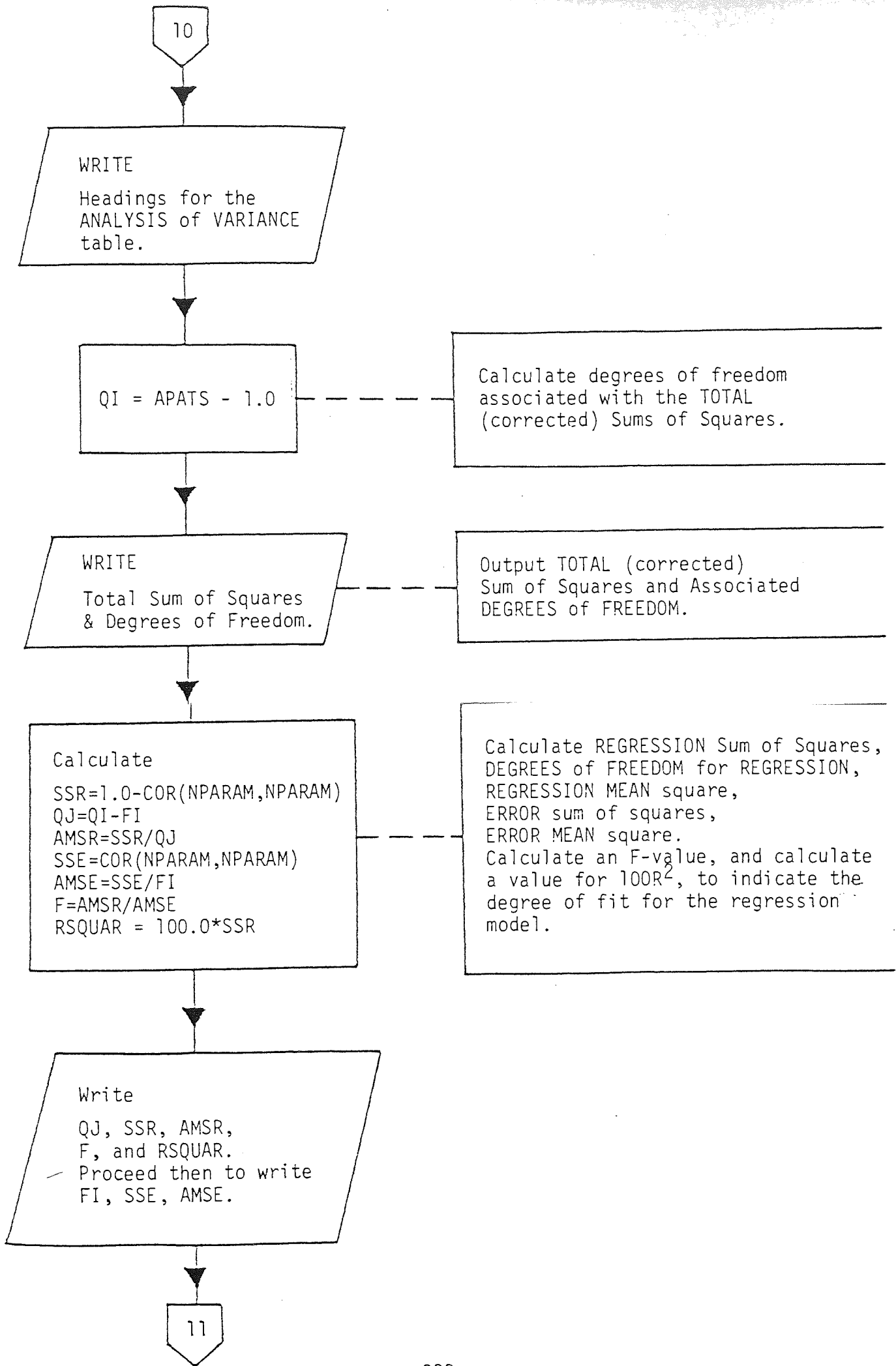
Is the variance reduction caused by ADDING the variable with the LARGEST V_i term $< FIXE$? If not, ADD the new variable corresponding to N_{MAX} into regression model.

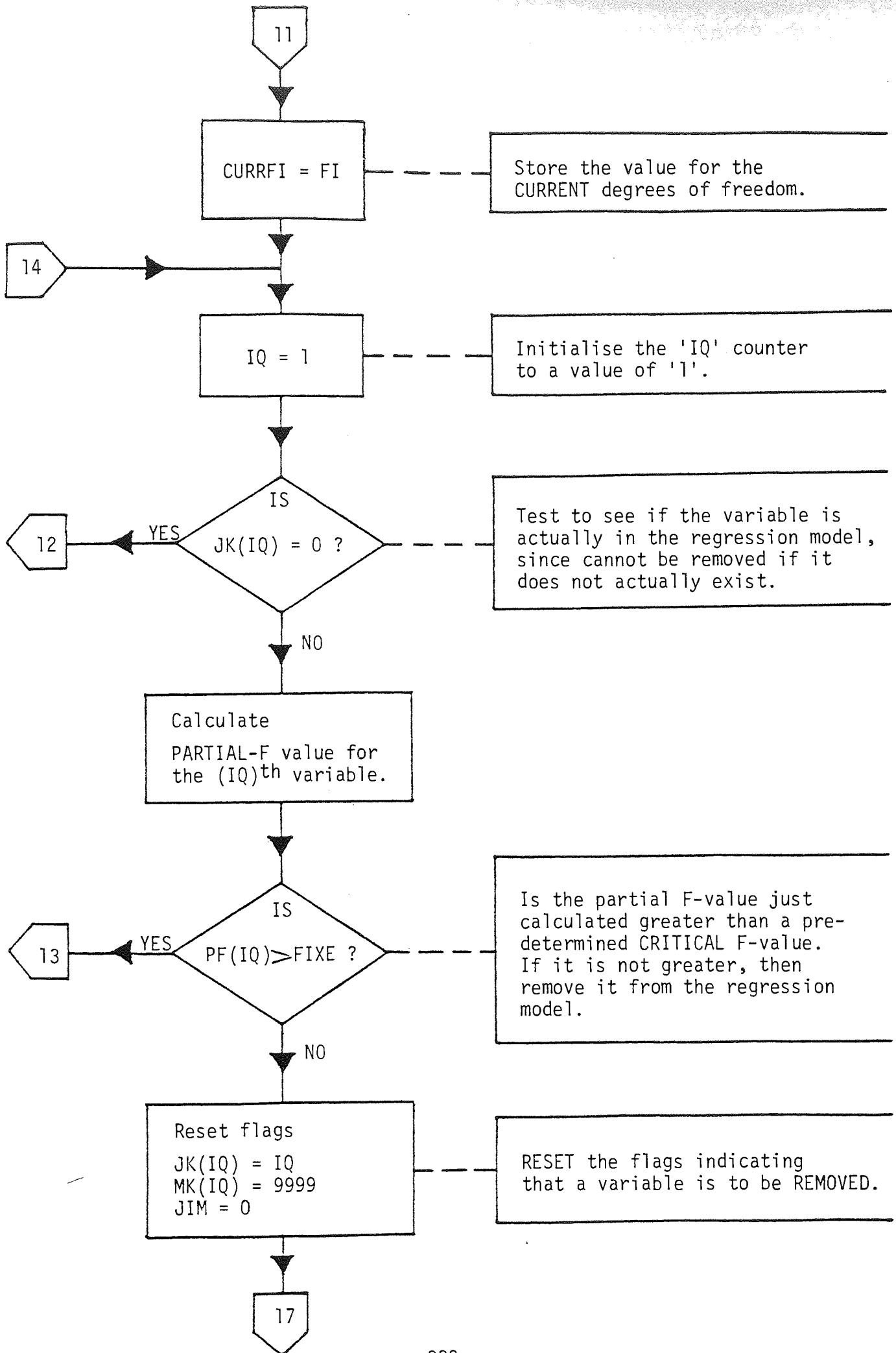
Reset FLAGS
 $JK(N_{MAX}) = N_{MAX}$
 $MK(N_{MAX}) = N_{MAX}$

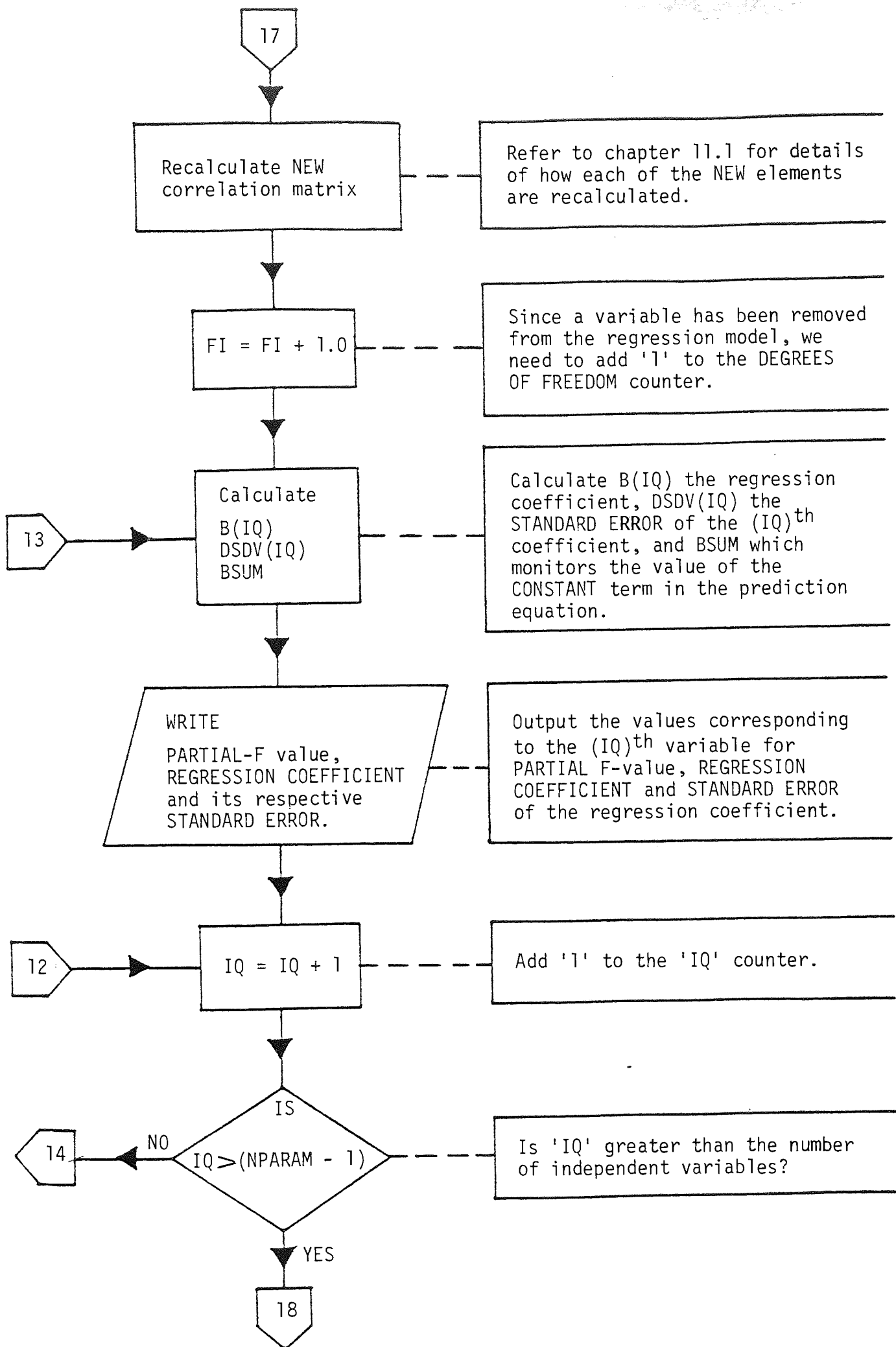
RESET flags corresponding to the variable entering the regression model.

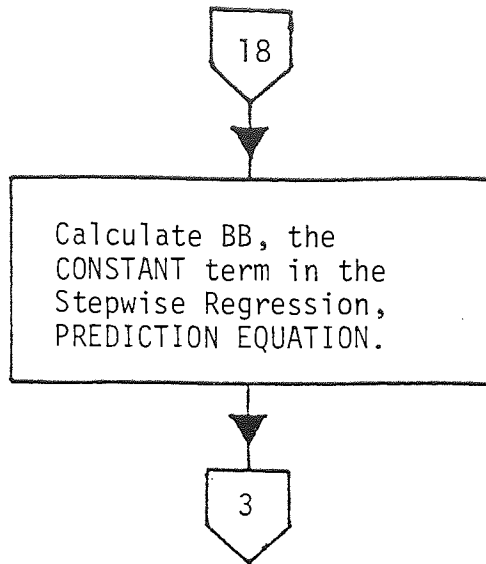
9



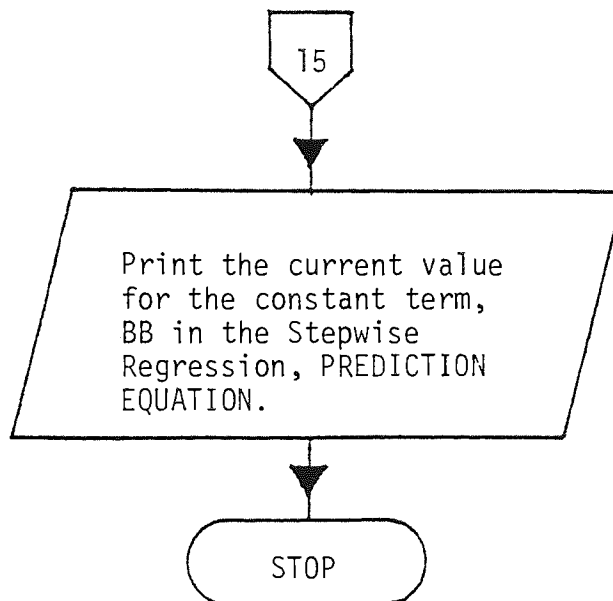








* * * Entry to the terminal section may only be achieved via the off-page connector '15'. * * *



REFERENCES

1. Jeffery, J.S. and Skylaroff, S.A. (1958) Incidence of wound infection. Lancet 1:365-368.
2. Lister, J. (1867) Antiseptic principle of the practice of surgery. Lancet 1:326, 357, 387, 507. 2:95.
3. Guthrie, D. (1949) In: Lord Lister: His life and doctrines E. & S. Livingstone Limited:Edinburgh.
4. Cruse, P.J.E. (1970) Surgical wound sepsis. Canadian Medical Association Journal 102:251-258.
5. Fowler, A.W. (1963) Effect of chlorhexidine on tissues. Lancet 1:387.
6. Altemeier, W.A., Hummell, R.P., Hill, E.O. and Lewis, S. (1973) Changing patterns in surgical infections. Annals of Surgery 178:436-445.
7. Wenzel, R.P., Osterman, C.A. and Hunting, K.J. (1976) Hospital-acquired infections. II. Infection rates by site, service and common procedures in a university hospital. American Journal of Epidemiology 104:645-651.
8. Kislak, J.W., Eickhoff, T.C. and Finland, M. (1964) Hospital-acquired infections and antibiotic usage at the Boston City Hospital - January 1964. New England Journal of Medicine 271:834-835.

9. Adler, J.L. and Shulman, J.A. (1970) Nosocomial infection and antibiotic usage at Grady Memorial Hospital: A prevalence survey. Southern Medical Journal 63:102-105.
10. Freeman, J., Rosner, B.A. and McGowan, J.E. (1979) Adverse effects of nosocomial infection. Journal of Infectious Diseases 140:732-740.
11. Scheckler, W.E. (1980) Hospital costs of nosocomial infections: A prospective three-month study in a community hospital. Infection Control 1:150-152.
12. Ayliffe, G.A.J. and Collins, B.J. (1982) Infection control in the United Kingdom. In: Aspects of Infection Control Series (Imperial Chemical Industries Limited, Pharmaceuticals Division).
13. Beigelman, P.M. and Rantz, L.A. (1950) Clinical importance of coagulase-positive penicillin-resistant staphylococcus. New England Journal of Medicine 242:353-358.
14. North, E.A. and Christie, R. (1945) Observations on sensitivity of staphylococci to penicillin. Medical Journal of Australia 2:44-46.
15. Barber, M. (1947) Staphylococcal infection due to penicillin-resistant strains. British Medical Journal 2:863-865.
16. Harley, H.R.S., Baty, J.A. and Bowie, J.H. (1946) Pathogenicity of penicillin insensitive infection. British Medical Journal 1:639-643.

17. Howe, C.W. (1954) Post-operative wound infection due to Staphylococcus aureus. New England Journal of Medicine 251:411-417.
18. Bennett, I.L. jnr., Minchew, H. and Fekety, F.R. (1959) Some aspects of the epidemiology of staphylococcal disease. Trans. Am. Clin. Climat. Association 71:191-201.
19. Cohen, L.S., Fekety, F.R. and Cluff, L.E. (1964) Studies on the epidemiology of staphylococcal infection. VI. Infections in surgical patients. Annals of Surgery 159:321-334.
20. Ehrenkranz, N.J. (1964) Person-to-person transmission of Staphylococcus aureus. New England Journal of Medicine 271:225-230.
21. Knight, V., White, A.C. and Martin, M.P. (1958) The effect of antimicrobial drugs on the staphylococcal flora of hospital patients. Annals of Internal Medicine 49:536-543.
22. Loewenthal, J. (1962) Sources and sequelae of surgical sepsis. British Medical Journal 1:1437-1440.
23. Williams, R.E.O., Jevons, M.P., Shooter, R.A., Hunter, C.J.W., Girling, J.A., Griffiths, J.D. and Taylor, G.W. (1959) Nasal staphylococci and sepsis in hospital patients. British Medical Journal 2:658-662.
24. Williams, R.E.O., Noble, W.C., Jevons, M.P., Lidwell, O.M., Shooter, R.A., White, R.G., Thom, B.T. and Taylor, G.W. (1962) Isolation for the control of staphylococcal infection in surgical wards. British Medical Journal 2:275-282.

25. Farrer, S.M. and MacLeod, C.M. (1960) Staphylococcal infections in a general hospital. American Journal of Hygiene 72:38-58.
26. Thoburn, R., Fekety, F.R. jnr., Cluff, L.E. and Melvin, V.B. (1968) Infection acquired by hospitalised patients - an analysis of the overall problem. Archives of Internal Medicine 121:1-10.
27. Barrett, F.F., Casey, J.I. and Finland, M. (1968) Infections and antibiotic usage among patients at Boston City Hospital, February 1967. New England Journal of Medicine 278:5-9.
28. Public Health Laboratory Service (1960) Incidence of surgical wound infection in England and Wales. Lancet 2:659-665.
29. Thornton, G.F., Fekety, F.R. and Cluff, L.E. (1964) Studies of the epidemiology of staphylococcal infection. VIII. Seasonal variation. New England Journal of Medicine 271:1333-1337.
30. MacNamara, M.J., Hill, M.D., Balows, A. and Tucker, E.B. (1967) Study of bacteriological patterns of hospital infections. Annals of Internal Medicine 66:480-488.
31. Adler, J.L., Burke, J.P. and Finland, M. (1971) Infection and antibiotic usage at Boston City Hospital, January 1970. Archives of Internal Medicine 127:460-465.
32. Moody, M.L. and Burke, J.P. (1972) Infections and antibiotic use in a large private hospital, January 1971. Archives of Internal Medicine 130:261-266.

33. Ayliffe, G.A.J., Lilly, H.A. and Lowbury, E.J.L. (1979) Decline of the hospital staphylococcus? Incidence of multi-resistant Staph. aureus in three Birmingham hospitals. Lancet 1:538-541.
34. Cruse, P.J.E. and Foord, R. (1973) A five-year prospective study of 23,649 surgical wounds. Archives of Surgery 107: 206-210.
35. Davidson, A.I.G., Clark, C. and Smith, G. (1971) Post-operative wound infection: A computer analysis. British Journal of Surgery 58:333.
36. Minchew, H. and Cluff, L.E. (1961) Studies of the epidemiology of staphylococcal infection. I. Infection in hospitalised patients. Journal of Chronic Diseases 13:354-373.
37. Medical Research Council (1968) Aseptic methods in the operating suite. Lancet 1:705-709, 763-768, 831-839.
38. Streeter, S., Dunn, H. and Lepper, M. (1967) Hospital infection - A necessary risk? American Journal of Nursing 67:526-533.
39. Schreck, K.M. and Hopps, E. (1960) Observations on the epidemiology of staphylococcal infections. American Journal of Medical Science 240:171-185.
40. Dineen, P. (1961) A critical study of 100 consecutive wound infections. Surgery, Gynaecology and Obstetrics 113:91-96.

41. Steinhauer, B.W., Cox, F., Stobie, G.H.C. and Quinn, E.L. (1967) A method of hospital infection surveillance incorporating the use of the computer. Henry Ford Hospital Medical Journal 15:139-147.
42. Lidwell, O.M. (1961) Sepsis in surgical wounds: Multiple regression analysis applied to records of post-operative hospital sepsis. Journal of Hygiene (Camb) 59:259-270.
43. Rountree, P.M., Harrington, M., Loewenthal, J. and Gye, R. (1960) Staphylococcal wound infection in a surgical unit. Lancet 2:1-6.
44. Altemeier, W.A. (1966) Control of wound infections. Journal of the Royal College of Surgeons of Edinburgh 11:271-282.
45. American Hospital Association (1958) Prevention and control of staphylococcal infections in hospitals. Hospitals 32:49, 51, 93.
46. Altemeier, W.A. (1970) Current infection problems in surgery. In: Proceedings of the International Conference on Nosocomial Infections, Atlanta pp.82-87.
47. Knight, V., White, A., Foster, F. and Wenzel, T. (1956) Studies on staphylococci from hospital patients. II. Effect of antimicrobial therapy and hospitalisation on carrier rates. Annals of the New York Academy of Science 65:206-221.

48. Parker, M.T., John, M., Emond, R.T.D. and Machacek, K.A. (1965) Acquisition of Staphylococcus aureus by patients in cubicles. British Medical Journal 1:1101-1105.
49. Scheckler, W.E. and Bennett, J.V. (1970) Antibiotic usage in seven community hospitals. Journal of the American Medical Association 213:264-267.
50. Wenzel, R.P., Osterman, C.A., Hunting, K.J. and Gwaltney, J.M.jnr. (1976) Hospital-acquired infections. I. Surveillance in a university hospital. American Journal of Epidemiology 103:251-260.
51. Edwards, L.D. (1969) Infections and use of antimicrobials in an 800-bed hospital. Public Health Report 84:451-457.
52. Shoji, K.T., Axnick, K. and Rytel, M.W. (1974) Infections and antibiotic use in a large municipal hospital 1970-1972. A prospective analysis of the effectiveness of a continuous surveillance program. Health Laboratory Science 11:283-292.
53. Hinton, N.A. and Orr, J.H. (1957) Studies on incidence and distribution of antibiotic-resistant staphylococci. Journal of Laboratory and Clinical Medicine 49:566-572.
54. Feingold, D.S. (1970) Hospital-acquired infections. New England Journal of Medicine 283:1384-1391.
55. Dixon, R.E. (1975) Techniques for prevention of nosocomial surgical infection. Journal of the American Medical Association 231:1290.

56. Finland, M. and Jones, W.F. jnr. (1956) Staphylococcal infections currently encountered in a large municipal hospital: Some problems in evaluating antimicrobial therapy in such infections. Annals of the New York Academy of Science 65:191-205.
57. Robertson, H.R., Sutherland, W.H. and Colbeck, J.C. (1958) Wound infection. Annals of the Royal College of Surgeons of England 23:141-154.
58. Cruse, P.J.E. (1975) Incidence of wound infection on surgical services. Surgical Clinics of North America 55:1269-1275.
59. Altemeier, W.A. and Culbertson, W.R. (1965) Surgical Infection. In: Surgery: Principles and Practices. 3rd edition. Eds. Moyer et al. Philadelphia. p.51.
60. Shooter, R.A., Taylor, G.W., Ellis, G. and Ross, J.P. (1956) Post-operative wound infection. Surgery, Gynaecology and Obstetrics 103:257-262.
61. Lowbury, E.J.L. (1954) Air-conditioning with filtered air for dressing burns. Lancet 1:292-294.
62. Blowers, R. (1961) Control of infection in hospital wards. Journal of Clinical Pathology 14:18-25.

63. National Research Council (1964) Post-operative wound infections: The influence of ultra-violet irradiation on the operating room and of various other factors. Annals of Surgery 160 (supplement no. 2).
64. Gillespie, W.A., Alder, V.G., Ayliffe, G.A.J., Bradbeer, J.W. and Wypkema, W. (1959) Staphylococcal cross-infection in surgery: Effects of some preventative measures. Lancet 2:781-784.
65. Williams, R.E.O., Blowers, R., Garrod, L.P. and Shooter, R.A. (1966) In: Hospital Infection: causes and prevention. London: Lloyd-Luke (Medical Books) Limited.
66. Eickhoff, T.C. (1975) Nosocomial infections. American Journal of Epidemiology 101:93-97.
67. Matsen, J.M. (1973) The sources of hospital infections. Medicine 52:271-277.
68. Mullholland, S.G., McGarrity, G.J. and Ross, O.A. et al. (1975) Experience with detailed surveillance of nosocomial infections. Surgery, Gynaecology and Obstetrics 140:941-945.
69. Williams, R.E.O. (1970) Changing perspectives in hospital infection. In: Proceedings of the International Conference on Nosocomial Infections, Atlanta pp. 1-10.
70. Meers, P.D., Ayliffe, G.A.J., Emmerson, A.M., Leigh, D.A., Mayon-White, R.T., Mackintosh, C.A. and Stronge, J.L. (1981) Report on the national survey of infection in hospitals, 1980. Journal of Hospital Infection 2:supplement.

71. Schaffner, W. (1976) The ongoing problems of hospital infections. In: Advances in internal medicine. Chicago: Year Book Medical Publishers Inc. 21:175-187.
72. Ayliffe, G.A.J., Lowbury, E.J.L., Hamilton, J.G., Small, J.M., Asheshov, E.A. and Parker, M.T. (1965) Hospital infection with Pseudomonas aeruginosa in neurosurgery. Lancet 2:365-368.
73. Ayliffe, G.A.J., Babb, J.R., Bridges, K., Lilly, H.A., Lowbury, E.J.L., Varney, J. and Wilkins, M.D. (1975) Comparison of two methods for assessing the removal of total organisms and pathogens from the skin. Journal of Hygiene (Camb) 75:259-274.
74. Casewell, M., and Phillips, I. (1977) Hands as route of transmission for Klebsiella species. British Medical Journal 2:1315-1317.
75. Taylor, L.J. (1978) An evaluation of handwashing techniques. 1. Nursing Times January 12th. pp. 54-55.
76. Taylor, L.J. (1978) An evaluation of handwashing techniques. 2. Nursing Times January 19th. pp. 108-110.
77. Bassett, H.F.M., Ferguson, W.G., Hoffman, E., Walton, M., Blowers, R. and Conn. C.A. (1963) Sources of staphylococcal infection in surgical wound sepsis. Journal of Hygiene (Camb) 61:83-94.
78. Dineen, P. and Pearce, C. (1958) A ten-year study on wound infections. Surgery, Gynaecology and Obstetrics 106:453-458.

79. Ayliffe, G.A.J., Collins, B.J., Lowbury, E.J.L. and Wall, M. (1971) Protective isolation in single-bed rooms: studies in a modified hospital ward. Journal of Hygiene (Camb) 69:511-527.
80. Green, J.W. and Wenzel, R.P. (1977) Post-operative wound infection: A controlled study of increased duration of hospital stay and direct cost of hospitalisation. Annals of Surgery 185:264-268.
81. Spengler, R.F. and Greenough, W.B. (1978) Hospital costs and mortality attributed to nosocomial bacteraemias. Journal of the American Medical Association 240:2455-2458.
82. Scheckler, W.E. (1978) Septicaemia and nosocomial infections in a community hospital. Annals of Internal Medicine 89:754-756.
83. Clark, S.K.R. (1957) Sepsis in surgical wounds with particular reference to Staphylococcus aureus. British Journal of Surgery 44:592-596.
84. Cruse, P.J.E. (1970) Surgical wound sepsis. Canadian Medical Association Journal 102:251-258.
85. Gardner, A.M.N., Stamp, M., Bowgen, J.A. and Moore, B. (1962) The infection control sister. A new member of the control of infection team in general hospitals. Lancet 2:710-711.
86. Davis, N.C., Fielding, G., Garlick, F. and Raq, A. (1963) The infection control sister. Her role in a large hospital. Lancet 2:1321-1322.

87. Eickhoff, T.C. (1978) Standards for hospital infection control. Annals of Internal Medicine 89:829-831.
88. Freeman, J. and McGowan, J.E. jnr. (1978) Risk factors for nosocomial infection. Journal of Infectious Diseases 138:811-819.
89. Cohen, L.S., Fekety, R. and Cluff, L.E. (1962) Studies of the epidemiology of staphylococcal infection. V. The reporting of hospital-acquired infection. Journal of the American Medical Association 180:805-808.
90. Mullholland, S.G., Creed, J. and Dierauf, L.A. (1974) Analysis and significance of nosocomial infection rates. Annals of Surgery 180:827-830.
91. Macpherson, C.R. (1968) Practical problems in the detection of hospital-acquired infections. American Journal of Clinical Pathology 50:155-159.
92. Tillet, H.E. and Thomas, M.E.M. (1981) Monitoring infectious diseases using routine microbiology data. I. Study of gastro-enteritis in an urban area. Journal of Hygiene (Camb) 86:49-58.
93. Colbeck, J.C. (1962) Control of infections in hospitals. American Hospital Association Hospital Monograph Series, number 12.
94. Wenzel, K. (1970) The role of the infection control nurse. Nursing Clinics of North America 5:1:89-98.

95. Haley, R.W., Quade, D., Freeman, H.E. and Bennett, J.V. (1980) The SENIC project: Study of the efficacy of nosocomial infection control. American Journal of Epidemiology 111: 472-485.
96. Bradbeer, T.L., Forman, A., Furze, R.M. and Moore, B. (1966) Duties and status of an infection control sister in the Exeter Hospital Group. Monthly Bulletin of the Ministry of Health and Public Health Laboratory Service 25:269-276.
97. Bennett, J.V. (1978) Human infections: Economic implications and prevention. Annals of Internal Medicine 89:761-763.
98. Bartlett, R.C. (1974) Control of hospital-associated infections. A. Infection surveillance and control. American Society of Microbiology. Manual of Clinical Microbiology pp. 841-845.
99. Eickhoff, T.C., Brachman, P.S., Bennett, J.V. and Brown, J.F. (1969) Surveillance of nosocomial infections in community hospitals. I. Surveillance methods, effectiveness and initial results. Journal of Infectious Diseases 120:305-317.
100. Scheckler, W.E. (1978) Nosocomial infections in a community hospital, 1972 through 1976. Archives of Internal Medicine 138:1792-1794.
101. Britt, M.R., Burke, J.P., Nordqvist, A.G., Wilfert, J.N. and Smith, C.B. (1976) Infection control in small hospitals. Prevalence surveys in 18 institutions. Journal of the American Medical Association 236:1700-1703.

102. Dixon, R.E. (1978) Effect of infections on hospital care. Annals of Internal Medicine 89:749-753.
103. Rhame, F.S. and Sudderth, W.D. (1981) Incidence and prevalence as used in the analysis of the occurrence of nosocomial infections. American Journal of Epidemiology 113:1-11.
104. Goonatilake, P.C.L. (1978) Ph.D. Thesis. University of Aston in Birmingham.
105. Williams, C.P.S. and Oliver, T.K. (1963) Nursery routines and staphylococcal colonisation of the newborn. Journal of Hygiene (Camb) 61:83-94.

BIBLIOGRAPHY

Draper, N.R. and Smith, H. (1966) In: Applied regression analysis.
New York: John Wiley and Sons Inc.

Efroymsen, M.A. (see Ralston Anthony and Wilf, Herbert S.).

Graybill, Franklin A. (1961) In: An introduction to linear statistical models. Vol. 1. McGraw-Hill Book Company Inc.

Hald, A. (1967) In: Statistical theory with engineering applications.
New York: John Wiley and Sons Inc.

Lowbury, E.J.L., Ayliffe, G.A.J., Geddes, A.M. and Williams, J.D.
(1981) In: Control of hospital infection: A practical handbook.
2nd edition. London: Chapman and Hall.

Parker, Margaret J. (1978) In: Microbiology for nurses (from
Nurses' Aids Series). London: Baillière Tindall Limited.

Ralston, Anthony and Wilf, Herbert S. (1960) In: Mathematical methods for digital computers. Vol. 1. New York: John Wiley and Sons Inc.

Walpole, Ronald E. and Myers, Raymond H. (1972) In: Probability and statistics for engineers and scientists. London: Collier-Macmillan Limited.

Winner, H.I. (1979) In: Microbiology in patient care. London: Hodder and Stroughton Limited.