AN ECONOMIC INVESTIGATION OF THE QUALITY OF HOSPITAL CARE IN BRITISH COLUMBIA

by

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Abstract

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The quality of health and hospital care is usually measured by one of three basic approaches. Structure measures assess the inputs used or available for use in the treatment of patients. Assessments of process look at "how" patients were treated. Outcomes measures are concerned with the end-results of care. Outcomes have considerable intuitive appeal to consumers and it is assumed in this thesis that quality is defined in terms of outcomes; providers (e.g. physicians) however may have preferences for hospital structure for its own sake. Structure and process measures are generally regarded as proxies for outcomes measurs. They are used because they are easier to measure than outcomes, but the relations between the proxies and outcomes has not been completely tested.

This thesis is concerned with the empirical verification of the relationship between two types of measures, structure and outcomes of hospital care at the aggregate level, and a possible link between provider preferences for structure and observed "excess" structure. The outcomes measures are based on adjusted hospital death rates. The adjustment factors draw on detailed diagnostic and demographic information available in the British Columbia hospital reporting

system. Several possible adjustment factors (proxies for severity) are considered. The structure measures include measures of inputs per case, and measures of the facilities and services offered by a hospital.

The discussion centres on three hypotheses. The first two concern the empirical relation between structure and outcomes. The first hypothesis that the two types of assessment are equivalent was tested using correlation analysis of alternative outcomes measures and structure measures. The results indicate that structure cannot be substituted for outcomes measures in the evaluation of quality. The second hypothesis is that there is ineffective or "excess" structure. This is demonstrated if the impact of incremental structure on outcomes is not positive. The results generally support the existence of excess structure. Extensive regression analysis and exploration of possible weakness did not result in the modification of the basic conclusion.

The third hypothesis is that such "excess" structure arises and persists because providers value structure for its own sake, and are able to impose their preferences on hospitals. The discussion is essentially theoretical and considerable evidence supporting the hypothesis is provided, although no formal proof is offered. Physicians value structure because it enables them to increase their income and/or leisure, and also to satisfy their professional desires with respect to their working environment. Arguments are presented to support the claim that physicians get some of the structure they want because of imperfections in real-world agency relations and the institutional features of the health care system.

Given the basic premise of the thesis (that consumers would define quality in terms of outcomes), the results of both the empirical and theoretical investigations have implications for policy. Policy changes suggested in the discussion that concludes the thesis are concerned with resource allocation within the hospital system, quality measurement, monitoring of policy changes, and incentives and programs to modify provider preferences.

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Chapter I

INTRODUCTION

I. INTRODUCTION

Economics is concerned with the conversion of resources into outputs that may satisfy alternative desires. The hospital sector is just a set of institutions engaged in economic activity. Inputs are turned into hospital services; indeed there are many studies of the production of hospital services, but these tend to ignore or neglect a most interesting area of analysis - the quality of hospital care. The quality of hospital care is of concern to society, government regulators, insurers, providers and consumers, and it is the topic of this thesis.

Quality is a concept which is handled with reluctance in conventional economic analysis, but it is an important issue in the health care sector because changes in the quantity of resources devoted to hospital care or health care may affect not only the quantity of output, but also the quality of output. This has an impact on both consumers of health care, and on the providers.

Quality has become a central issue in the evaluation of the health care system in Canada. Problems of access and cost have not been exacerbated by the national health insurance system. The system costs no more than its antecedent and costs less than the mixed market American system.[1] The distribution of services has improved somewhat.

So, if there are few problems of efficiency and equity, the last remaining argument may be about quality of care available. Part of the evaluation of the success of the system is predicated on the effect on quality of a cost constraint. Key to this evaluation is knowledge about the relationship of measurable quality to resource inputs.

This relationship has been the subject of much speculation, but little analysis and research. This problem constitutes a doubly interesting topic because it concerns all consumers. Everyone is likely to need health care some time and presumably would prefer "good" care. It is also of relevance to the administrators of the overall health care program since many public funds are allocated to the health care sector.

At a theoretical level, the analysis of "quality" constitutes a difficult area characterized by comparatively little research, the intermingling (and confusion) of quality with quantity, the existence and use of three approaches to quality definition and measurement (structure, process and outcomes), the likelihood that consumers and providers may have diverging views on what "quality" is, and the constraints placed by the social environment on the development of a measure of quality.

This thesis tests several hypotheses about quality measurement, quality production, and choices of quality levels.

II. QUALITY DEFINITION: ECONOMIC THEORY AND APPLICATION TO HEALTH CARE

Economists have exhibited some interest in the conceptual aspects of quality, but analysts of the health care sector have actively pursued the practical implementation of quality assessment.

Traditionally economists have disregarded the existence of similar goods of differing qualities by assuming that a "good" was an iso-quality commodity and that similar products of differing qualities were different goods. These goods entered directly into the consumer's utility function. Standard neoclassical demand theory does not allow for quality variation and changes in "quality" of a good generally require redefinition of the set of goods in the world.

Within standard theory, there has been some limited discussion of quality variation in the context of monopolistic competition where the products were differentiated. In this discussion, goods of differing qualities were "different" goods. Thus goods that were really similar but differentiated in terms of "quality" had high (numerical) cross elasticities and were close substitutes. Quality variation was postulated to act like advertising in shifting the demand curve. Yet Chamberlin (41) and others had problems defining both a good, a group of goods, and quality, without some basic unit of measurement which captured both the possibility of "sameness" and "differentiation".

The use of the characteristics approach (see Theil (296), Houthakker (159), and Lancaster (181)) facilitated the study of heterogeneous goods. This approach requires that a commodity be defined as a set or vector of fundamental elements or attributes called characteristics. These characteristics, not the goods themselves, enter (either directly or via some household production function) the economic agent's utility function. The link to the common concept of a "good" is provided by looking at the functions which various bundles of characteristics serve.

The services provided during the stay of a patient at a hospital are a commodity (good), but each hospital may provide services of differing characteristics, and even different service bundles to each patient. Thus the good is hospital services provided during an episode of care, but the quality of these services may vary, because the characteristics which affect consumer satisfaction are different.

The application of the characteristics approach in health care is not simple. The characteristics of hospital services are not necessarily linearly related to the services themselves. Information about the product and product characteristics is not fully available to all consumers. Other market imperfections such as noncompetitive environments mean that the market is unable to correct for information gaps. Thus conventional economic techniques for determining the shadow prices of characteristics (e.g. hedonic price indices) are inapplicable in a market where there are many imperfections.

Despite some of the insights provided by the characteristics approach, there are still problems in the definition of quality, quantity, and their measurement.[2] Triplett (303) identifies three different, but related, concepts of quality.

The first concept is that quality is a characteristic in itself. "(I)t is an element 'which belongs to something and makes or helps to make it what it is'." (Triplett (303) quoted in Wadman (314) p. 132). The second concept identifies quality as the character, basic nature or essence of a thing - a whole set of characteristics which serve to distinguish one thing from another. In the third concept:

> "Quality" is associated with a ranking of products (or services) according to grade, desirability, usefulness, or degree of excellence. With this concept, quality differences are exclusively differences in the level of quality. Where the second concept (noted above) really referred to a listing of attributes or characteristics possessed by a product, the third is concerned with amounts of quantities of the various characteristics. (Triplett (303) quoted in Wadman (314), p 132).

It is this third concept of quality that is the most appropriate for our purposes.

Thus quality is a relative concept and is defined in terms of relative level of characteristics available (characteristics per quantity unit, if feasible). Assuming that characteristics are arguments in a consumer's preference function, increasing the level of certain characteristics of a commodity will increase the satisfaction of that consumer. Therefore, the consumer's utility and "quality" are directly related. This definition makes it imperative that the set of characteristics used to represent quality be those that enter someone's utility function.

Analysts of the health care sector are less concerned with the elaboration of the "concept" of quality, and have, perhaps for practical reasons, developed and implemented three separate approaches to quality definition and measurement - structure, process, and outcome:

> Structural measurements are concerned with descriptive innate characteristics of facilities or providers (e.g. the soundness of a building, whether a poison chart is posted in an emergency room, or the age and board certification status of the physician). Process measures are those that evaluate what a provider does to and for a patient (e.g. ordering a cardiogram for a patient with chest pain) and how well a person is moved through the medical care system, whether in a "macro" sense (e.g. from first symptom to seeking care to obtaining care) or in a "micro" sense (e.g. from arrival to departure at an emergency department). Outcomes reflect what happened to the patient, in terms of palliation, treatment, cure, or rehabilitation. (Brook (29) p. 1, ftn).

Thus structural measurement is concerned with the quantity and/ or characteristics of resources used (or available for use) in the care of patients. Many standard quality indicators and controls, such as hospital licensure and accreditation are based on compliance with structural standards.

Process assessment is concerned with how inputs are used. Process measurement usually involves the comparison of what was recorded as happening to the patient, to what would have been "optimum", "best", or "average" care.

> ... "process can be applied, by definition, only to examination of services actually rendered, rather than to the experience of a total population, some members of which may receive no services at all. The measurement rests on the assumption that at any time and place there is a scientific consensus among widely acknowledged experts on what constitutes good-or-high quality health services - the consensus typically, though not always, rests on a body of empirical data. (Roemer (250) p. 841.)

Outcomes similarly may require some basis of comparison, i.e.

good outcomes would be characterized by the achievement of an end-result which would be comparable to that expected, given the appropriate application of current technology or the change in health status relative to some expected change in health status. The most basic outcomes measure is either death or recovery.

The choice of quality measurement for an evaluation in the health care sector tends to be the result of practical considerations such as the availability of data. In general, structure is easier to measure (and information is more readily available), than either process or outcomes. Process measurement has some appeal because of the direct relationship between process assessment and what is done to the patient.

III. VALIDITATION OF THREE APPROACHES TO QUALITY ASSESSMENT

(Hypothesis One)

The basic premise of this thesis is that consumers are concerned about their health status. Thus the characteristics of health (hospital) care which would be called "quality" by a concerned consumer would be those related to changes in this health status or outcomes. The better outcomes are, the greater the consumer's satisfaction will be, and the better he will rank the quality of care received. Thus, consumers would measure quality in terms of outcomes.[3] It is assumed that consumers, if fully informed, would not confuse the characteristics of inputs into their care with the effectiveness of these inputs, or the outcome of that episode of care.

Other authorities (such as Williamson (324), Brook (29)) have come to similar conclusions. Tugwell (305) uses the World Health Organization definition of health and its interpretation[4] to note that:

> ... the functional status of the patient (corresponding to outcomes as defined above) should be the definitive measure of the quality of care; this means that measurement of the actions of those providing medical care (clinical process) and of the medical facilities (clinical structure) are only acceptable as indicators of quality where they have been demonstrated to predict the functional status or survival of the patient. (Tugwell (305) p. 113)

Evans and Wolfson (90) present an argument which leads to similar conclusions, but approach the problem as one of establishing a plausible utility function for the consumer with respect to health and health care. The link to quality definition is that their formulation leads to the conclusion that only efficacious health care would be desired both for the consumer himself and for others. Neglecting other elements in the utility function, the utility of a consumer can be described by:

$$U_c = U(HS(H),H)$$

where U_c is the utility of the consumer, HS his health status, and H, health care.

where $\frac{\delta U}{HS} > 0$

but $\frac{dU}{dH} = \frac{\delta U}{\delta HS} \cdot \frac{dHS}{dH} + \frac{\delta U}{\delta H}$

Evans and Wolfson (90) suggest that $\frac{\delta U}{\delta HS} > 0$, but $\frac{\delta U}{\delta H} < 0$.

"Health care <u>per</u> <u>se</u> is in general a dis-good, except in so far as it contributes to health status, i.e. is efficacious in a technical sense. People 'consume' health care for its expected effects on health; absent these, and they would prefer not to use it."[5] (Evans and Wolfson (90) p. 8).

Evans and Wolfson (90) expand the utility function for a representative consumer to include other variables that allow for cross effects, externalities, and the effects of access and technological change. No matter what formulation is used, efficacy of H in producing HS is required. This implies that outcomes assessment is the relevant approach to quality measurement. From the consumer's point of view, the use of structure and/or process as quality measures constitutes the use of proxies.

For practical reasons, structure and process are often substituted for outcomes in quality evaluation. Such substitution implies that all three measures move together. The acceptance of this assumption may subsume the argument that market forces in the health care (or hospital care) sector are sufficiently strong to ensure efficient production of quality. The three types of measures may be related to production of quality as:

$$O = P(S)$$

where outcomes (O) describes the quality that is achieved using inputs (S) combined in a technology described by $P(\cdot)$ (process).

In a perfectly competitive world, where consumers and producers possess full information[6] about all dimensions of output and inputs, the free functioning of the market system would result in an efficient relationship between inputs (S) and output (O) and the most efficient production process (P). Thus any deviation away from optimal S or P would result in reduced O, so the S or P would be equivalent as proxies for O.

For most goods, the market would ensure that technical and economic efficiency is achieved. But this is not an acceptable premise for the market for health care. A number of the conditions required for a free functioning market to achieve optimality do not hold, and the forces which limit the effect of such imperfections are missing in this market. Berki (20) notes that:

> Consumer ignorance, the absence of price competition and entry into the market, atomization of the producing sector without any invisible hand to co-ordinate it, differential accessibility by income, color and location to a set of services proclaimed to be "rights" would suggest that market criteria are deficient. (p. 42)

Thus, if it cannot be assumed that market forces are sufficiently strong to ensure that structure, process and outcomes are equivalent, then substitution of proxies for outcomes for evaluation purposes should be based on empirical evidence of the relationship between the proxy measures (structure and process) and outcomes. The literature reviewed in Chapter Two looks at the measures of structure, process, and outcomes that have been used to evaluate the quality of hospital and health care, and also at some tests which attempt to validate substitution. There are very few recorded attempts to compare outcomes and proxies, but there is some evidence that the result may depend on the method of assessment used. In Chapter Three some aggregate measures of outcomes and structure for British Columbia hospitals are developed. These measures can be tested for equivalence.

The correlation analysis tests the hypothesis that structure and outcomes assessment are equivalent and that structure may be freely substituted for outcomes measures. The results of this analysis (detailed in the first part of Chapter Four) indicate that there is little or no relation between structure and some measures of outcomes based on mortality rates. For some measures the resulting correlation is of the opposite sign to that desired - i.e. structure assessment would yield results contrary to those of outcomes assessment. These results indicate that substitution of structure for outcomes cannot be expected to yield the same result. Similarly policy analysis cannot rely on structure changes as indicative of quality changes.

IV. EXCESS STRUCTURE?

(Hypothesis Two)

The second hypothesis tested in this thesis (second section of Chapter Four), is that the observed low or inappropriately signed correlation is the result of excess structure. This can be tested by looking at the sign, significance, and magnitude of the impact on outcomes of structure variation via regression techniques when a production technology is imposed.

Structure (inputs) is required to produce outcomes. Assuming that the outcomes (quality) dimension of output is separable from the other dimensions (output quantity), a production relationship between structure and outcomes may be posited. Information will be generated not only on the marginal impact of structure on outcomes, but also on the efficiency of hospital production. Evidence of negative or zero marginal products of structure may indicate that excess (or inappropriate) structure exists.[7]

This hypothesis is holds up under rigorous investigation including the use of alternative specifications, different outcomes measures,[8] and many combinations of structure variables (also reported in Chapter Four).

V. EXCESS STRUCTURE: STRUCTURE AND PROVIDERS OF CARE

(Hypothesis Three)

The existence of excess structure is not predicted in a market where consumers are fully sovereign and knowledgeable. Excess structure means that some inputs are ineffective. As noted in an earlier section, the consumer's desires will be satisfied only by health care that has a positive impact on his health status. Markets are not perfect, but unless there are economic agents active in the market, other than consumers, who have preferences for other than outcomes, there would be no reason to expect any other situation to arise.

The third hypothesis of the thesis is that excess structure, as evidenced in the empirical section of the thesis, arises because providers value structure for its own sake, and providers dominate resource allocation in hospitals. There is considerable evidence, assembled in Chapter Five, that providers, particularly physicians, will value structure in hospitals; and that their influence in hospitals (and the health care sector in general) permits them to impose, to a certain extent, their preferences, but the "hypothesis" cannot be formally tested.

The latter argument is supported by consideration of the roles which physicians fulfill as the agents of their patients (and the imperfections in real-world agency relationships), and the institutional importance of physicians in hospitals (hospitals cannot practice medicine).

Producer preferences can be exploited to show why there may be structure in excess of that required to satisfy an effectiveness criterion. Berki (20) notes that:

> Especially where alternative diagnostic and treatment processes are available and considered probabilistically medically appropriate, the chosen course of action is likely to be the one which the physician expects to maximize his preference function ... Service demands may be originated by the physician for purposes of research, for teaching, for demonstrating high esoteric skill achievement, for status, and for increasing his income or decreasing his workload, or both simultaneously. None of these elements need to be detrimental to patient focussed medical outcomes. But they differ from them in that the desired results are expected to benefit primarily the decision maker. (Berki (20) p. 5)

The argument presented in Chapter Five lays most of the blame for over-provision of inputs, excessive duplication of services, and the continued use of inefficacious technology on providers. It may not be legitimate to reject producer preferences, but there may be concern with the extent to which providers dominate consumer choices and hospital decisions. Social institutions determine to a great extent the weighting of consumer and producer preferences. The weighting in observed situations can be questioned, particularly where charges to producers are probably not at socially optimal levels.[9]

The possibility of "excess" structure and the existence of noncongruent preferences for consumers, providers, and payers, gives rise to many policy implications which are detailed in Chapter Six. Programmes to educate providers with respect to the possibility that inefficacious care may be existent, to modify their preferences with respect to where and how medicine is practiced, and incentive schemes figure prominently in this discussion.

FOOTNOTES - CHAPTER I

1. Expenditures on health care in Canada currently are 7.3% of GNP. American expenditures on health care total close to 10% of GNP.

2. Wadman (314) devotes an entire thesis to the nuances of quality and quantity definition.

3. The concern is not with determining the exact characteristics which denote outcomes, and their quantity, but with the justification of concentration on outcomes as the most rational approach to measuring quality.

4. The WHO technical study group interpreted "health" as "a condition or quality of the human organism which expresses adequate functioning under given genetic and environmental conditions." (Quoted in Tugwell (305) p. 113.)

5. Hypochondriacs are an exception, although some consider them unwell.

6. The perfect agency relationship where the agent (the physician (or in some cases, the hospital)) acts on behalf of the patient (as if he were in the patient's place) is subsumed under a perfectly competitive world, since perfectly functioning markets would not permit the agents to deviate far from rational choices. The agency relation is discussed further in Chapter Five.

7. The efficacy condition is assumed to be marginal product = opportunity cost and since resource costs are generally positive, a zero or negative MP is indicative of excess. However it may be argued that MP = 0 is the result of an optimization process that does not recognize the cost of inputs (i.e. resources are to be allocated, in the health care sector, until MP is zero, since human life has no finite value). However, there is still the possibility that a zero MP still represents excess structure because we are past the first point of zero MP, i.e. on the flat portion of the curve.

8. But a limited range of outcomes measures.

9. Most hospital structure is available to physicians at zero monetary cost, which does not reflect the scarcity rent of these resources.

Chapter Two

QUALITY ASSESSMENT: THREE APPROACHES IN PRACTICE

I. INTRODUCTION

This chapter deals with the application of the three approaches to quality measurement used in health care research: structure, process, and outcomes. The choice of approach in any situation is dependent on several factors, both practical and methodological, as well as common practice.[1] The practical considerations influencing the choice of method are linked with the methodological considerations. In some ways the demarkation into two sets is arbitrary.

The major practical consideration is data availability. The feasibility and cost of accessing the appropriate data tends to be an overriding factor.

Almost all types of process evaluation require information on what was done to the individual patient. This information is usually abstracted from the medical record, although a number of other sources are available.[2] Collecting these data for many patients is a resource intensive process, unless standardized computerized records are used.[3]

Structure data are more available. In the USA, much information about the facilities available in hospitals is reported in the August issue of Hospitals each year. In Canada, much structural information is reported to various levels of government.

Outcomes data are of varying availability. In general, "good" data about health status of patients are unavailable on a routine basis. For a study of individual outcomes, it is necessary to establish the end-result for each patient at some point in time, or to have observations on a patient's health status over time. This may involve contacting each patient since the data required are not collected in the normal functioning of the health care system. This is often an expensive activity.

Aggregate outcomes data, such as death rates, are more readily available, but they are, in their crude form, unadjusted for variation in case-mix and other factors, which makes comparison of crude death rates disadvantageous for hospitals that treat the more seriously ill. Problems here stem from difficulties in collecting information on these confounding variables.

Preserving the confidentiality of medical records may also affect the feasibility and cost of any assessment which requires their use.

The cost of an assessment is affected by the choice of who is to do the judging. Hiring "expert" medical judges, and the process of developing measures of norms or consensi, can be expensive. Similarly the requirement that results be quickly available for some purposes may limit choices.

The last practical consideration is particularly important for process assessment. Tugwell (305) notes that a process measure must have "clinical credibility", i.e. the criteria used must be credible to providers or they will not accept the results. This acceptance has extensive consequences for it can limit the range of factors considered, and may also result in enshrinement of "known" factors of "unknown" value.

Other concerns[4] which may be regarded as methodological include what the quality assessment is to be used for (identification of system failures, or regulatory purposes), whether a long-run or short-run viewpoint is desired, how many dimensions or characteristics are to be included, and how optima are to be defined (and who is to define them).

A number of ways of measuring outcomes, structure, and process are described and compared in this Chapter. Practical outcomes measures are based on mortality rates, but there are several ways of adjusting for case severity so that hospitals with different patient mixes can be compared. These are discussed as background (Section II) for the outcomes measures used in the empirical part of the thesis (Chapters Three and Four). Structure measures are basically measures of inputs (Section III), and comparison of structure and outcomes does not always yield the expected results, as reported in Section IV. Similar results are found for process and outcomes as reported in Section V. The literature reviewed in this chapter supports the hypothesis that the relationships between structure and outcomes, and process and outcomes (and even process and structure) are not strong.

II. OUTCOMES MEASURES FOR HOSPITALS

Outcomes are the definitive quality measures in health care, yet practical problems of data availability have limited the development and application of outcomes measures.

> The face validity of patient outcomes measures is more apparent, since measurement of outcome necessitates measurement of "health" itself, or some aspects of it. However, when outcomes measures have been developed and proposed for use or applied to evaluate quality of care, several problems have arisen:
> 1. The outcomes most frequently used, such as death or incidence of major complication, may be so uncommon that detection of significant differences between patient groups requires a sample of patients so large that the feasibility of the study is limited.
> 2. "Ultimate" outcomes or end-results, such as death or restoration of normal function often occur so late in the course of treatment that timely evaluation is impossible.
> 3. Such commonly used measures as mortality or return to function are heavily influenced by intervening factors, such as genetic makeup and the physical and social environment, that are beyond the control of the medical care system.
> 4. Information about many outcomes is not readily available, requiring the use of follow-up patient interviews. These are expensive to conduct and difficult to complete for a high proportion of the relevant patient population.
> 5. Information on the breadth of outcome criteria that should be used in assessing quality of care is absent. Should outcome assessment be limited to physical and physiological measures, or should it include psychological measures, such as sexual function following radical mastectomy for breast cancer.
> (Brook, Avery et al. (31), Vol. 1, p. 3)

As well, some value judgements may complicate assessments, as there is still some variation in opinion about what constitutes a "good" outcome (i.e. is continued "life" after irreversible brain damage a "good" outcome), what is "good" health (i.e. what is normal or abnormal), and who shall decide what the current health status is, the provider, the patient, or some outside authority.

The base of any outcomes measure must be the health status of the individual. Alternative dimensions and depictions of health status range from multi-dimensional health status indices which measure the health status of an individual over time, to death rates.

Health status indices are related to quality assessment by looking at the change in health status resulting from treatment relative to the change expected if "optimal" treatment were given. A simple measure would require that health status be measured before and after treatment. More sophisticated health status indices look at health status as a continuum over time.[5]

The basic problem with the implementation of such health status indices is the amount of information required for each case. It is necessary to know not only the functional level of the patient at each point in time, but also the expected health status with and without treatment in the future.[6] However, some of the scales developed could be put to use in point-of-time evaluation of health status; upon entering the health care system, and at some point when recovery or maximum health status improvement could be expected. This information is not currently collected on a routine basis and can be expensive to collect, even for a small sample of patients.

An alternative to health status indices is to use "intermediate" outcomes as a measure of outcome. These are generally diagnosis specific, and specify some physical state as indicating cure[7] or good health status without waiting for "final" outcomes to occur. These measures can be used as quality measures when compared to expected outcomes. Expected outcomes may be determined implicitly (a rater gives his opinion on whether outcomes could have been better) or explicitly (as a distribution of expected results which have some clinical base (i.e. a specific bacterial count or blood pressure reading)). Generally some sort of follow-up examination or interview is required.

This approach was used by Brook (30) in a study of the care provided in an outpatient department of a hospital, for three conditions; urinary tract infections (UTI), hypertension, and ulcerated lesions of the stomach and duodenum (ulcers), and further developed in Brook et al. (31).

In the implicit rating method, physicians (three per case) were asked if outcomes could have been better, basing their judgement on an abstract which included the results of a follow-up examination and interview five months after discharge from the clinic. Results indicated that two or more judges thought that outcomes could have been improved in thirty-seven percent of all cases.

The explicit method compared the results of the examination and interviews to an expected distribution[8] of health statuses (defined by laboratory results or interview responses re symptoms). Thus for UTI, the results of a urine bacterial count were investigated. Death was a possible health state. The results indicated that oucomes were less than acceptable in many cases.

Only fifty-one percent of those with UTI had acceptable bacterial counts in their urine and only fifty-six percent of the hypertension cases had controlled pressure at the follow-up examination.

There were some problems with this method since some of the categories of outcome such as mortality and decreased activity were expected to occur only in a very small number of cases. In some cases, such as continued symptoms for UTI and ulcers, "the observed outcomes were worse than the physicians estimated they would be if the patients received no therapy" (30, p. 335).

Brook was unable to explain this.[9] The practicality of this approach is limited by the requirement of follow-up interviews, laboratory tests, and the diagnosis-specific nature of the results.

If data at this level of detail cannot be accessed, and information on outcomes for a broader population is desired, more general outcomes measures (such as death rates) which apply to all types of cases are required.

Death aversion is a valid goal of the health care system. A relevant, but not perfect, reflection of the hospital's achievement is its ability to avert death. Thus mortality rates may be used as a base to compare overall hospital outcomes.

An individual will be concerned with the probability of dying if he enters a hospital. This represents one characteristic of the care he would receive. It has the appropriate units for a quality measure (characteristics/unit quantity) since the quantity is a case (episode of care). Aggregation to provide a quality measure for the hospital requires a "representative" consumer, and to establish this "representative" patient some adjustment must be made for factors affecting the death rate which are beyond the hospital's control.

The need for such adjustments has been noted on several occasions. If some hospitals provide services such that the more seriously ill are sent to them for treatment, it would be expected that these hospitals would experience higher crude death rates. There are some types of cases for which the hospital has essentially little control over the outcome. Thus, before crude death rates can be used to compare the care provided by hospitals, some adjustment factor, which captures the variation in case-mix and case severity, should be considered. Two types of adjustment are possible: limit the type of cases considered or adjust for overall variation in case mix.

Unadjusted death rates have been used for comparison purposes, but only in cases where the hospital populations are very similar. This usually involves limiting the type of hospital or type of case considered. This may be described as adjustment for severity variation by disaggregation in an attempt to find common populations.

Some alternatives include:

1) death rate calculated for those who survive at least forty-eight hours in the hospital.[10] This is intended to exclude all those for whom the hospital could offer little hope.

Roemer, Moustafa and Hopkins (254) discuss the use of such a variable but conclude that it offers little improvement over crude death rates as modern hospitals are:

> expected to have efficient emergency services which can save the lives of many such patients. An end-result measure of quality, therefore, should include such deaths - at least until more refined measurements can identify cases that are truly hopeless in spite of any medical or surgical procedures. (254, p. 103)

2) maternal mortality. Deaths in childbirth have declined through advances in technology and asepsis to very low levels. Maternal deaths are so rare, that occurrences usually result in extensive investigation as to cause, process of care, etc. In large populations this measure is occasionally used (e.g. Bubeck et al. (33), Thompson (299)).

3) infant mortality rates. These rates have also been traditional indicators of hospital quality. (See Shapiro et al. (269), Institute of Medicine (162)). However, recent research has indicated that infant death rates are not determined for the most part by hospital care, but, by overall prenatal care, income levels and socio-economic factors which may be beyond the control of the hospital (see Fuchs (111), p. 36). This tends to reduce the usefulness of infant mortality rates for quality evaluation.[11]

4) case-specific fatality rates. Case fatality rates have been used to compare the care given by different hospitals. Lembcke (189), and Lee et al. (184) compared death rates for several diagnoses in teaching and non-teaching hospitals. Roemer (251) looked at size-outcomes relationships. However both Roemer (251) and Lee et al. (184) noted that more information may be required because even within diagnoses there may be differences in patient population. Age and sex variation and severity variation were also considered to have an impact. Thus care is suggested in choosing a case-type for investigation and cross-hospital comparison purposes.

The alternative is to find some general adjustment factor which will capture the variation in severity of illness of those entering hospitals.

If information was available on the health status of each admission, it would be fairly easy to develop an index of case severity;[12] the lower the health status of admissions, the generally more severe the cases. However this information is not usually available so proxies must be used. These proxies try to capture variation in factors which are hypothesized to be correlated with severity.

The demographic mix of the hospital population is postulated to be related to case severity, as the probability that any case is serious (likely to result in death) increases with age. There is some sex-related variation in life expectancies and the severity of cases (i.e. women live longer, and many use the hospital for maternity admission but would not be considered to be severely ill). Thus information on the age-sex mix of admissions to a hospital may be used to adjust mortality rates for variation in factors beyond the hospital's control.

As an alternative or in addition to age-sex mix, case-mix measures could be used to adjust the crude death rate. The mix of diagnoses treated by hospitals varies widely. Either direct information on case-mix (such as proportions of cases of various types) or summary measures (such as factor scores (Evans (85)) or calculated measures like case complexity (Evans and Walker (89))) may be used to capture the variation in case-mix (and case severity) across hospitals.

Other alternatives include the proportion of cases transferred to the hospital from other institutions, the proportion of cases referred by outside physicians, and the average length of stay (ALS).

Roemer, Moustafa and Hopkins (254) claimed that average length of stay was the most widely available variable which was a suitable proxy for case severity. This proposition was based on the hypothesis that more serious cases would be under treatment longer and thus, a longer average length of stay (ALS) would indicate that a hospital had more severely ill patients. However, average length of stay is influenced by many other factors including:

1) common medical practices;
2) availability of beds;
3) availability of alternative treatment modes (particularly for long-stay patients - nursing homes, home care programmes);
4) teaching status and practices;
5) seasonal variation.

Roemer et al. claim that the influence of some of these factors will be measured by the occupancy rate. When occupancy rates are high physicians will be under pressure to move patients through the hospital quickly, to free a bed for another case.[13] They suggest then that the average length of stay variable be linearly corrected by the occupancy rate of the hospital relative to the average occupancy rate in all hospitals. This new variable was called the occupancy corrected length of stay (OCLS).[14] Their outcomes measures were called length-of-stay-adjusted death rates (LSDR - ALS correction) and severity-adjusted death rates (SADR - OCLS correction). The calculation of these variables is discussed further in Chapter Three.

The advantage of LSDR and SADR is that the data to calculate them are readily available. They have been used in quality studies by a number of authors. Roemer et al. (254) applied them to a sample of California hospitals.[15] Roemer and Freeman (252) used SADR as a measure of hospital performance in a study of medical staff organization and its relationship to hospital performance.[16] Neuhauser (218) used[17] SADR as one quality measure in a study of Chicago hospitals and compared it with other measures (see next section).

Thus, outcomes measures of several types have been suggested and used in the analysis of hospital quality levels. Each has varying data requirements. Practical considerations and the desire to develop aggregate measures of outcomes for hospitals limit this thesis to the development and application of outcomes measures based on mortality rates. A number of adjustment factors are available which will permit the development of measures including adjustment for age-sex mix and case-mix specifically, as well as the use of LSDR and SADR (adjustment using average-length-of-stay). The development and calculation of such measures is further discussed in the next chapter.

III. STRUCTURE MEASURES IN HOSPITALS

Structure measures are heavily used in assessing quality in hospitals, particularly for regulatory and certification purposes. In general structure and inputs are directly related. The degree of aggregation over inputs varies. Some studies look at overall resources used in a system of hospitals (Feldstein (97) for example), others use a per institution average (Ruchlin and Levey (258), Holmberg and Anderson (153)). It may also be possible to look at input use on a case-specific basis.[18]

Feldstein (97) used a typical structure approach based on inputs of a highly aggregated nature to look at the quality of American hospital systems. His assumption is that there is a "constant elasticity" relation between the quantity of inputs and the quality of output:

$$Q_{it} = ke^{\gamma t}R_{it}^{\rho}$$

where:

Q_{it} = quality in state i in year t;

R_{it} = measure of resource input per patient day;

k = constant

γ, ρ = unknown parameters.

There is no consideration of the "quality" of the inputs themselves, or the possiblity that there are changes in the capital-labour composition over time. Some shifts in the skill mix are captured in a wage index. Outcomes are not discussed. State aggregates are used.

His choice of units (inputs per patient day) implies that the output quantity is a patient day rather than an episode of care.

Resource input is not ascertained by aggregating inputs, but by the ratio of an index of average cost per patient day ($JACPPD_{it}$) to an index of hospital input prices[19] ($PRIN_{it}$).

Feldstein found considerable variation in the input prices and in the intensity of resource use per hospital day across the states. The rate of quality increase over the study period (1958-69) ranged from sixteen percent in one state to thirty-six percent in another.

Another alternative is to compare staff-hours of various types per bed, per patient-day, or per case, as well as data on physical facilities such as space per bed, bathrooms per room. The use of these invokes the assumption (usually untested) that more is better than less. Thus staffing ratios have been used to give evidence that proprietary hospitals give "different" care from voluntary (non-profit) hospitals (Clarkson (46)), or that proprietary nursing homes give lower quality care since staffing ratios (both total or by specific type (RN hours, aide hours)) are different (Levey et al. (190), Holmberg and Anderson (153), and Solon (278)).[20]

Other studies employ "summary" measures of quality. These can be of two types - those that have been assigned to a hospital by some general rater (such as full accreditation status granted by the Canadian Council on Hospital Accreditation, or certification as a hospital to provide residencies in certain specializations), or those that result from the summarization of several structure measures (e.g. the Technological Adequacy Score (TAS) of Roemer et al. (254) measures the mix of facilities and services available). Some other features of hospitals such as scale, and ownership, might also be regarded as summary indicators of structure because many structural features are subsumed in their use as comparison features in quality analysis.[21]

Some summary measures require the explicit weighting of various dimensions. One heavily used summary measure is cost per case (or in some studies cost per patient day). Wage rates and input prices are used to aggregate inputs. Hospitals that have different input mixes will have different costs. Cost levels have been used by some economists in models of hospital behaviour as indicators of quality levels (see Newhouse (222)). Other weighting schemes are more ad hoc.[22]

Roemer, Moustafa and Hopkins (254) also developed a measure of structure based on facilities and services, but they employed an explicit, if arbitrary, weighting scheme. This index called the Technological Adequacy Score (TAS) was composed of several items as listed in Table IV.[23]

The weights assigned to each component "correspond to the judgement of the authors on the probable relative influence of these inputs on the outcome of patient care" (p. 108). Factors (1), (2), and (3) were given heavy weight because hospitals having these features had been evaluated independently several times. Factors (4) through (9) were indicators of technological sophistication. Factors (10) to (12) reflect the hospital's orientation to the community, and factor (13) gave some indication of whether or not preventive medicine was practiced.

All these structure measures are input based, but in most cases their use is not validated against outcomes. If this is to be done and the "production" of outcomes explored, both structure and outcomes measures are required. The structure measures that are employed are of both types, summary and input, which are further discussed in the next chapter.

IV. STRUCTURE-OUTCOMES COMPARISONS

Most studies that compare structure measures with outcomes measures do not explicitly set out to do so. Such comparisons are hidden in investigations of outcomes for teaching and non-teaching hospitals or large and small hospitals. Goss (128) reports some studies investigating the impact of teaching status, specialization and ownership status on outcomes. Studies such as Kohl (175), Lee et al. (184), Graham and Paluocek (130) and the Commission on Professional and Hospital Activities (49) provide some evidence that case-fatality rates are lower in teaching hospitals.[24,25] The evidence on the impact of specialization and the role of for-profit operation is less clear.

Roemer et al. (254) looked at the relationship between facilities and services in a hospital, scale, and outcomes, as noted in Table I. They did not report any correlations or other tests of the strength of the relationship between the structure and outcomes variables. Goss and Reed (129) in testing Roemer et al.'s methodology on another sample tried to correlate SADR and TAS, but found no strong relationship.

There have been several studies into the organization of hospitals which include investigation of the relationship between quality (defined in various ways) and organizational structure. The approach is generally descriptive, but these studies (Georgeopoulos and Mann

Table I

TAS and other Quality Measures

TAS	Average TAS	Average Number of Beds	Average Autopsy Rate(%)	Average Crude DR	LSDR	SADR
5-20 (8 Hospitals)	15	67	21%	1.88	2.31	3.22
21-35 (8 hospitals)	28	59	23%	2.44	2.90	4.19
36-59 (9 hospitals)	49	111	23%	2.93	2.96	3.77
60-85 (8 hospitals)	81	421	47%	3.64	2.33	2.64

Source: Roemer, Moustafa and Hopkins (254), Tables 4 and 5, p. 109 and 113.

(117), Neuhauser (218), and Roemer and Friedman (252)) provide some evidence on the weak relationship between structure and overall outcomes, even though the data bases are generally derived from case studies and small samples. The extensive use of simple correlations as "tests" of relationships and the inability to control for confounding variables, such as size of hospital, seem to limit the strength of any results.

For example, Neuhauser (218) correlated managerial techniques and organization performance, including quality, for a sample of thirty medium-size Chicago hospitals. His quality measures were: 1) expert evaluation; 2) and 3) measures derived from the Joint Council on the Accreditation of Hospitals (JCAH) evaluation; 4) percentage of physicians who were board-certified specialists; and 5) death rates adjusted for case-severity (SADR). The second, third, and fourth measures are structural in approach. The first measure may be a combination of both structure, process, and outcomes depending upon which characteristics the "experts" are concerned with. The correlations between these measures are detailed in Table II. There is some evidence that outcomes (SADR - see Footnote 17) are correlated with expert evaluation and some accreditation standards and also that expert evaluation is based on structure and outcomes. In another comparison he found that costs and quality were not positively related, but that size was positively related to quality.

The evidence about structure-outcomes relationships is rather weak. Neither summary nor input measures have been shown to be

Table II

Correlations Between the Measures of Quality of Care
(Medical Efficiency)

	% Board-Certified Specialists	JCAH I	JCAH II	Adjusted Death Rates
Mean Expert Evaluation	+.557**	+.089	+.528**	-.456**
% Board-Certified		+.310	+.214	-.270
JCAH I			+.571**	-.103
JCAH II				-.364*

* prob. < .05
** prob. < .005

Source: Neuhauser, (218), Table 7-V, p. 68.

positively related to outcomes (except in the case of teaching status and possibly Neuhauser's JCAH II). Also interesting is the lack of studies in this area. There are very few comparisons of more than the simplest outcomes and structure measures, and in many cases statistical significance is not reported. This thesis does attempt a comprehensive test of the structure and outcomes relation, as reported in Chapter Four.

V. PROCESS MEASURES AND PROCESS-OUTCOMES COMPARISON

Process measures are concerned with what is done to the patient. Most process assessments have focused on medical care practices as related to physician quality. There are two basic approaches. The use of raters or judges (usually peer (i.e. physician)) involves the use of implicit criteria. The care given is compared to what the rater thinks is "good" or "optimal" care. This technique has the disadvantage of variability across judges and the type of case, but the alternative, the use of explicit criteria (usually in the form of check lists), is subject to criticism on the acceptability and applicability of the criteria and their rigidity. The major problem with explicit criteria is that their development by consensus tends to lead to long lists of everything everyone thinks _might_ have an impact on outcomes. In many cases proof as to the most effective course of action is not available, so that "poor" or _status quo_ practices may be enshrined in process protocols (Palmer (133), p. 27-28). Because of the grab-bag nature of criteria it is not surprising that Brook (30) found that very few (less than three percent) of process criteria significantly affected outcomes (p. 334).

Process studies tend to be case-type specific, and there are few general studies.[26] They also tend to be expensive to carry out. There are few process-outcomes studies, and the general conclusions are that there are few strong relationships, although the methodology of such comparisons has been criticized by McAuliffe (197). There are no process-outcomes comparisons performed in this thesis.

The Teamster studies (81, 208) are oft quoted studies of the process of care using implicit rating techniques. The quality and necessity of care were judged by specialists from medical records. There was some general consideration of outcomes (i.e. in some cases, the surveyors were able to say that outcomes could have been improved if the process of care had been better).

The studies indicated that suboptimal care was given in up to forty-three percent of the cases reviewed, and that twelve percent of all hospitalizations were unnecessary. Most information was contained in cross-tabulations of process judgments with physician specialty and qualifications (more formal qualifications were related to good process), hospital sponsorship (better care if hospital was associated with a medical school), and admission type.

Brook (30) used both implicit and explicit approaches in his study of the care of out-patients for three diagnoses (outcomes were discussed in section II). He found that "(i)n effect, the results of the quality assessment are determined by the method used" (30, p. 335), and that the judgments with respect to the quality of care also varied with the condition studied.

The results using the implicit judgments of process were startling. Of all cases, seventy-six percent (227 of 296 cases) were judged by two or three judges to have received inadequate care.[27]

To the abstract used for the process judgement was added a second page providing information on outcomes. Judges were then asked for ratings on outcomes (could outcome have been improved, if process had been better?) and on overall quality of care (was this (based on process and outcome information) acceptable?). Critical examination of improvable outcomes only, improved the assessment of quality in many cases. Now only thirty-seven percent of cases received negative judgments. However, the impact of adding outcomes to process for the overall measure was small. Only twelve cases were judged better under the "overall" judgment than under process criteria. (72.9 percent were still judged negatively by two or more judges.) It seems that in some judges' minds process has more impact than outcome.

When implicit process assessments were compared with explicit outcomes (including laboratory findings) a significant ($p < 0.05$) relationship was found between adequate process and a urine culture for UTI, and process and a follow-up blood pressure reading for the hypertension diagnosis.[28] For other outcomes such as continuing symptoms and activity limitation, a positive insignificant relation with process was observed. The results are mixed, but provide some evidence that bad process may at times lead to poor outcomes.

The explicit process technique produced fewer "good" results. Only four of the 296 cases (1.4%) met all the criteria "agreed on by two-thirds of the Baltimore City hospitals' physicians; six cases (2%) met all the criteria similarly agreed upon by Johns Hopkins Hospital specialty teams" (p. 334). The mean percentage of criteria met was 52 percent (of an average 13.5 criteria per case) for UTI, 58 percent (18.1 criteria) for hypertension, and 35 percent (15.2 criteria) for ulcer cases.[29]

Since the explicit process method produced so few cases judged adequate, Brook noted that "there should be practically no relationship between many of the explicit process criteria and outcome" (p. 334). When the correlations were calculated, 101 of the 204 correlations were positive, but only five of these were significant at the five percent probability level, supporting this hypothesis. This result provokes questions about the validity of current methods of evaluation of care which tend to rely heavily on explicit-process criteria in medical audits. He also found it "ironic that those outcome parameters which are of vital importance to the patient (i.e. his major activity level and symptom level) had nonsignificant correlations with either the implicit or explicit-process judgements" (p. 338).

He recommended the careful and further examination of such methods of quality evaluation and further investigation of the use of outcomes as quality measures.[30]

VI. SUMMARY AND CONCLUSIONS

This chapter has been devoted to approaches to and measures of quality of health care. These approaches basically exist for practical reasons - structure is easily measured, process less easily, but the measurement of outcomes presents some problems. A theoretical justification for the substitution of structure and process for outcomes is possible, but some of the required market conditions such as good information, competitive forces, and disinterested producers, are not thought to hold generally in the real world. Since there is no assurance that the production of health care is efficient (technically or economically), substitution of one measure for another requires testing of the relationship between the various measures.

Evidence on the comparison of various measures showed that inconsistent results could be obtained, even with two measures of process. Certainly more research into the relation between structure and outcomes measures, and process and outcomes, is necessary.

The results using various measures (approaches) have been sufficiently different to prompt Brook, Williams and Avery (78) to note that:

> (i)n assessing quality of care, different data sources and different kinds of data can be used... Each combination will produce different assessments of quality, yet very little work has been done to evaluate individual methods of assessment, let alone to compare the results obtained when the same care is assessed by two or more methods.

The sign of the relation between structure and outcomes, and process and outcomes, has not been proven to be positive in either macro or micro studies. Almost all authors have concluded with a plea for more research. The next two chapters of the thesis discuss the development of consistent measures of structure and outcomes (and several alternatives for each) and the analysis of the relation between the two. It is not surprising, given the evidence summarized above, that the results are not clearly in favour of the equivalence of the two types of measures.

FOOTNOTES FOR CHAPTER TWO

1. There have been historical shifts in the type of approach used. Brook (30) notes that outcomes were first favoured, but shifts to structure and process have occurred in this century. Legislated programs such as the American PSRO (Professional Standards Review Organizations) are generally concerned with what the physician did to the patient (process).

2. See Greene (133), p. 45 or Egdahl and Gertman (78), p. 10 for a tabular presentation of sources of data for all three approaches.

3. This is not common practice.

4. See Palmer (133) for a lengthy discussion of such considerations.

5. See Bush et al. (36) for a complete discussion of how these are measured and implemented.

6 It should be noted that most HSI make some assumptions about these.

7. Some of these constitute the termination of an episode of care and may be regarded as "final". However, the full range of health status of the patient before, during, and after treatment are not observed. The time span of observation is reduced.

8. Variance due to patient compliance and other factors beyond the control of the health care system can be captured by careful definition of the expected outcome distribution.

9. Brook is continuing to work on this approach and is developing outcome protocols for other diseases (see Brook et al. (31). Williamson (324) is pursuing a similar approach.

10. The variable was available for the BC sample for 1966 only. DR48 was used as a dependent variable in regressions and in the correlation analysis for 1966. The results were not different from other variables so the results using this variable are not included.

11. Considerable work was done using infant death rates as an outcomes measure. It appears to assess features very different from outcomes measures based on death rates since the correlation with other measures was very low. However, the results of measuring the relation between infant death rates and structure (either infant care specific or general) were no better than those reported for DIF1, etc. Thus these results are not discussed.

12. If this information were available a health status index approach would probably be used!

13. The effect of the occupancy rate on average length of stay and its relation to the severity of cases may be indeterminate. During periods of high occupancy, one might think that admission would be deferred for the elective, less serious cases, so that on average there would be more severely ill patients, who may or may not have shorter average length of stay.

14. i.e $OCLS_i = ALS_i \times OR_i/\overline{OR}$

15. Adjusted outcomes in large teaching hospitals were shown to be as good as, if not better than, those of small proprietary hospitals. Crude death rates described the opposite relationship.

16. Calculation of SADR supported their hypothesis that hospitals with a looser organizational structure for physicians had poorer outcomes.

17. Neuhauser (218) seems to have applied Roemer, Moustafa and Hopkins' (254) methodology incorrectly. His coefficient for calculating SADR are those calculated for Roemer et al.'s (254) sample, rather than those of his sample. It is possible, but unlikely, that the coefficients were the same.

18. This sort of study tends to slip into the category of process evaluation, since certain resource levels and input patterns may correspond to both structure and process standards.

19. Most of Feldstein's paper is devoted to the calculation of $PRIN_{it}$ across states and over time. Separate Laspeyres indices are developed for labour and non-labour inputs. The labour input price indices take into account some local (state) labour market conditions. The indices for non-labour inputs are laboriously calculated from input-output tables.

20. It is interesting to note that generally studies are unable to find statistically significant differences between the two types of nursing homes.

21. The assumption is often made that bigger must be better, e.g. Roemer et al. (254) test their outcomes measures LSDR and SADR by looking at the relationship between scale and these measures. They expect to have better adjusted outcomes at larger scale hospitals.

22. For example, see Greenfield (184) who simply counts the number of services provided in a hospital and compares this to the maximum found in similar hospitals, or see Levey et al. (190).

23. All the information to calculate the TAS is readily available for American hospitals in the Annual Guide Issue of Hospitals. Unfortunately the same pattern is not used for Canadian hospitals and it is not easy to replicate exactly the TAS for a Canadian sample. See Table IV.

24. Although statistical significance is sometimes not achieved or reported.

25. Goss also noted that only 8% of American hospitals have teaching programmes. Thus the great majority of American (and Canadian) hospitals are compared against a minority, although teaching hospitals are generally large so the proportion of beds found in teaching hospitals is higher. The question remains - how can we distinguish the quality of care for the 92% that are non-teaching. Goss is unable to provide other goals (beyond specialized service and profit) to subdivide the rest for comparison purposes.

26. Anyone interested in pursuing the study of process evaluation is referred to Donabedian (71), Brook (30), Greene (133) or Egdahl and Gertman (78).

27.

By Condition: Process Judgment	Inadequate by two judges		Inadequate by all 3 judges		Total
	No.	%	No.	%	No.
Urinary Tract Infections	17	15.9	77	72.0	107
Hypertension	29	25.4	54	47.4	114
Ulcers	12	16.0	38	50.7	75
Total	58	19.6	169	57.0	296

Source: Brook (30), Table 5, p. 332.

28. It is interesting to note that for UTI, 32.1 percent of cases had high culture counts, and that 44.2 percent of hypertension cases still had uncontroled blood pressure, five months after treatment.

29. This may be the result of the process criteria setting process. Consensus methods may mean that no stone is left unturned (or opinion ignored) so that criteria become too numerous to be fulfilled.

30. To some extent Brook's findings do not support the use of any quality measures! However Brook has not been deterred from further research.

Chapter Three

STRUCTURE AND OUTCOMES MEASURES FOR HOSPITALS IN BRITISH COLUMBIA

I. INTRODUCTION

The purpose of this chapter is to develop and provide details on the calculation of the structure and outcomes measures which are to be used in Chapter Four to test the strength, direction and magnitude of the relationship between structure and outcomes.

In the previous chapter existing measures of structure and outcomes were discussed including evidence on the relation between these two types of measures. Certain structure measures of aggregate hospital quality can easily be adapted and applied to the British Columbia hospitals (see Section II) studied in this thesis. Some of these are direct applications of measures used or developed by other authors, such as the Technological Adequacy Score (TAS) developed by Roemer, Moustafa and Hopkins (254). Other measures, such as the staffing ratios (generally staff hours per case), or skill-mix variables are measures which have not been referenced directly in the quality literature. Those used in this thesis are listed in Table III and discussed in Section III. These measures will be compared in Chapter Four with outcomes measures.

However, before outlining the outcomes measures used in the empirical work, some discussion of the type of measure developed and

Table III

STRUCTURE MEASURES

Variable Type	Name	VN	Calculation	Av. 1966	Av. 1969	Av. 1973	Relation to Good Structure	Expected Impact on Dependent Variable (DR, LSDR/SADR, DRRA1/2)	Comments	References
Summary	Accreditation	ACC	0/1 if accred-ited by CCHA	34.4%	41.8%	57.1%	positive	negative (improved outcomes)	most large hospitals accredited	Goss (128) Roemer et al. (254) Can. Hosp. (327)
Summary	Training Prog. MD's RN's Interns	TPMD TPRN INT	0/1 variable presence of program	4 hosp. 7 6	4 hosp. 6 7	4 hosp. 6 7	positive	negative	very few programmes	Goss (128)
Summary	Technological Adequacy Score	TAS	see Table IV	20.23	29.16 Table V	34.85	positive	negative		Roemer et al. (254)
Summary	Cost per Case	CASEX	total expenditure (net of out-patient educational and depreciation expenses) per case	189.01 (1966)	285.83 Table V (1969)	450.97 (1973)	positive	negative	inputs weighted by prices (3-year variable deflated)	Feldstein (97) Newhouse (222)
Input	Total Staff per Case	TSC	total hours of staff employed in hospital per case	79.23	86.74 Table VI	92.73	positive	negative	measures total labour input	Feldstein (97)
Input	Total Nursing Staff per Case	TNSC	nursing staff hours per case	39.0	43.3 Table VI	44.6	positive	negative	TNSC/TSC declines with scale increases	
Input	Professional Staff per Case	PROSTC	hours of health professionals (except nurses) per case	3.64	7.00	8.46	positive	negative		
Input	Medical Staff per Case	MEDSTC	hospital-employed physicians per case (not hours)	.00053	.00048	.00086	positive	negative	full time + 1/2 part time	Roemer and Friedman (252)
Input	Physician/Popula-tion Ratio for Hospital Districts	DOCS	physicians per 10,000 population in 1975 in hospital districts			14.52	positive	negative	1975 data applied to 1973 only	Roll Call (255)
	Specialist/GP Ratio	SPRIO	specialist/10,000 pop. GP's/10,000 pop.			.311	positive	negative	1975 data applied to 1973	Roll Call (255)
Input (Skill-mix)	Ratio RNA hours to RN hours on nursing services	RNARNT	RNA hours (nursing) RN hours (nursing)	.310	.423	.436	negative	positive		
Input (Skill-mix)	Ratio RN hours to total nursing staff hours	RNTOTT	RN hours (nursing) total nursing staff hours	.6266	.6031 Table VI	.6026	positive	negative		
Input (Skill-mix)	Wage Index	WI.1	wage index for hospital staff	1.0157	.9999	.9968	positive	negative	variables had very little variation	Barer (14)
Other	Size	SIZE	number of beds set up	108.4	117.9	127.6	positive	negative	many small hospitals in B.C.	
Other	Autopsy Rate	AURTH	percent of deaths	.1850	.1925	.1879	positive (?)	negative	difficult concept	Roemer et al. (254)

applied is necessary. An outcomes measure should be an indicator of the aggregate quality of care provided in the hospital, yet be implementable, given data constraints and some theoretical considerations.

As well, if mortality rates are to be used as outcomes measures, some adjustment or correction is necessary for factors which are not the result of quality differences, but influence the overall death rate of the hospital. The adjustment measures used to capture the variation in demographic and case mixes are not original to this thesis, but their application to quality assessment and outcomes measurement is original. The outcomes measure, DIF1 (death rates adjusted with age-sex factor scores and case complexity) is suggested as an improvement over other approaches such as LSDR and SADR. Two other outcomes measures are also developed in this thesis (DRAA1 and DRAA2) based on age-adjustment via weighted aggregation of age-sex specific death rates.

In Section V, each of these outcomes measures is described and discussed. Correlation analysis is used to compare outcomes measures among themselves in Section VI. The results, despite very different adjustment factors and techniques, indicate that all will give similar results for the same hospital.

II. THE SAMPLE AND DATA SOURCES

The sample consists of eighty-three acute-care public hospitals in British Columbia for the years 1966, 1969, and 1973. Specialty hospitals such as maternity and tuberculosis hospitals and the B.C. Cancer Institute are excluded. Data were available for the years 1966-73, but only three years were investigated in depth because of the mass of data and information generated. Each year is treated separately as a cross-sectional analysis. Some analysis was done pooling the three years into a large sample.

Data are abstracted from the HS-1 and HS-2 tapes for each of the three years. These tapes contain information each hospital in Canada was required to report to the Federal Government in the Annual Return of Hospitals (67). The HS-1 report provides information on facilities and services provided, and patient statistics (mortality rates, average length of stay, etc.). The HS-2 provides financial and staffing information.[1]

The second source of data is B.C. Hospital Program discharge tapes which record data abstracted from the individual patient discharge form. This form records patient-specific information such as age, sex, length of stay, diagnosis, and operations undergone. These records are the source of the age-sex composition and diagnostic mix for each hospital. Some of the variables used that pertain to the entire hospital population (COMPL1, AS1, ..., ASn, ASwts, CMPADJ, WI.1 and the CASEX index) were originally calculated by Morris Barer for use in his thesis (14). Variables that pertain to deaths only, such as age-specific death rates, and all input measures, were computed for this thesis.

III. STRUCTURE MEASURES

Measures of structure fall into two major categories. Summary variables are those structure variables which summarize a number of features. Some summary variables such as the Technological Adequacy Score (TAS) are calculated using explicit (but usually ad hoc) weighting schemes. Other summary variables are the result of some evaluation of the hospital, based on structural criteria, by an outside group or investigator.

The summary variables and their calculation methods are listed in Table III. This table summarizes the expected relation of the variable to structure and to outcomes. All these variables tend to reflect the availability of facilities and services in a hospital, so that it would be expected that death rates (after adjustment for case severity) would fall, if the hospital is accredited, trains various groups, and has a high TAS.

Some of the summary measures may be called proxies for capital stock (particularly TAS), but there are no readily accessible measures of capital availability in the hospital. In some ways CASEX might be regarded as an input measure, but CASEX allows for the different mixes of inputs used in different hospitals. It is a summary measure because several types of inputs are added up, the weights being their prices (wage rate for labour inputs). It is the only input measure which captures both labour and some capital inputs.[2]

Several authors (Newhouse (222), Feldstein (96, 97)) have equated resources used or costs with quality. Thus the hospitals with higher costs (CASEX) would be expected to have better outcomes, if this is true.

Since this is a cross-sectional study, deflators do not have to be employed to tie this variable to resource use. But, for the three year combined study (CS-TS), the CASEX variable had to be deflated (inflated). Results are stated in terms of 1970 dollars. The deflators used were 1.465 for 1966, 1.094 for 1969, and .756 for 1973. These deflators were taken from Barer (14) p. 165, Table 5B.4 and were calculated specifically for the British Columbia hospital sample. CASEX values are summarized in Table V.

The TAS measure summary in Table V and the explanatory Table IV indicate a gradual increase in the TAS over time for all scale hospitals. There also appears to be a reporting error, since the maximum reported TAS in 1969 is greater than that of 1973. However, this error was not resolved and has been allowed to stand.[3]

Input measures record resources available per case such as staff hours per case. These are also described in Table III. Included in the input measures category are variables which measure the skill-mix of labour inputs such as the ratio of RNA's (registered nursing assistants (practical nurses)) to RN's (registered nurses) (RNARNT) in the

Table IV

Components of Technological Adequacy Score
(Roemer *et al.* formulation)

	Component	Technological Adequacy Points
1.	Accreditation*	20
2.	Approved residency or internship	10
3.	Approved cancer program	8
4.	Intensive Care Unit	7
5.	Pathology laboratory	5
6.	Blood bank	5
7.	Therapeutic X-ray	5
8.	Post-operative recovery room	5
9.	Rehabilitation service	5
10.	Out-patient department	8
11.	Home care programme (n.a. 1966-68)	8
12.	Social service department	7
13.	Chest X-ray on admission	7
	Maximum possible score	100

Source: Roemer, Moustafa and Hopkins (254), Table 3, p. 109.
* Joint Committee on Accreditation of Hospitals

TAS Calculation Formula
BC Sample

1.	Accreditation**	20 points
2.	Approved residency or internship	10 points
3.	Intensive Care Unit	7 points
4.	Pathology laboratory	5 points
5.	Blood bank	5 points
6.	Therapeutic X-ray	5 points
7.	Post-operative recovery room (this was not available - operating room was substituted)	5 points
8.	Rehabilitation service	5 points
9.	Out-patient department	8 points
10.	Home care programme (n.a. 1966-68)	8 points
11.	Social service department	7 points
	Maximum possible score 1966-68	77 points
	Maximum possible score 1969-73	85 points

** Canadian Council on Hospital Accreditation

Table V

Summary Tables for AUTRH, TAS, CASEX

Autopsy Rate (AUTRH)

Year	1966	1969	1973	3 Year
Average	0.1850	0.1925	0.1879	0.1885
Standard Deviation	0.2002	0.2166	0.1918	0.2023
Lowest	0.00	0.00	0.00	0.00
Highest	0.6296	0.7500	0.6923	0.7500
Scale				
0-50 beds	0.0631	0.0532	0.0899	
51-100 beds	0.1676	0.1856	0.2285	
101-200 beds	0.3389	0.3343	0.2324	
201+ beds	0.4488	0.4392	0.4073	

Technological Adequacy Score (TAS)

Year	1966	1969	1973	3 Year
Average	20.229	29.157	34.855	28.080
Standard Deviation	16.736	19.349	19.220	19.363
Lowest	0.00	0.00	0.00	0.00
Highest	70.00	85.00	77.00	85.00
Scale				
0-50 beds	9.32	15.64	18.97	
51-100 beds	20.74	30.11	39.17	
101-200 beds	31.06	39.00	48.79	
201+ beds	52.80	62.10	60.31	

Cost per Case (CASEX)

Year	1966	1969	1973	3 Year (1970$)
Average	189.01	285.83	450.97	310.18
Standard Deviation	48.727	77.633	148.27	94.37
Lowest	114.82	147.10	239.66	160.93
Highest	382.43	540.50	1243.80	940.32
Scale				
0-50 beds	172.27	261.041	400.786	
51-100 beds	179.73	284.761	439.195	
101-200 beds	191.77	323.461	503.554	
201+ beds	274.36	382.114	570.817	

nursing service of the hospital.[4]

The expected relations here are that greater staff input into a case or more costly and highly qualified staff indicates better structure, and will result in better outcomes. The staffing variables (TSC,TNSC, etc.) are straightforward. The skill-mix variables are simply ratios of hours of one staff type to another, except WI.1. The variable was developed by Barer (14) to capture variations in service mix cost and was:

> an indication of the extent to which a hospital has a relatively costly service or skill-mix. A value greater than 1 would indicate that the hospital in question employs personnel in a more costly combination than the provincial average.
> (Barer (14), p. 148)

WI.1 measures differences in a hospital's personnel mix relative to the provincial average. This variable might be used as a structure variable since a high skill-mix index means a high proportion of more expensive (and thus presumably more highly qualified) categories of staff employed.

Summary tables for some structure measures are provided in Tables V and VI. The number of hospitals in each size class is not constant and over time there was a gradual increase in the scale of hospitals. This may have an impact on average values for larger scale classes for variables such as the staffing ratios. The shift of a hospital from a medium scale (<200 beds) to a larger scale will probably pull down the average staffing ratio (e.g. TSC) in larger hospitals.

It is also interesting to note that there is much greater variability with scale in TSC than in TNSC, and that there is very

Table VI

Summary Tables for TSC, TNSC, RNTOTT

TSC

Year	1966	1969	1973	3 Year
Average	79.231	86.742	92.734	86.235
Standard Deviation	29.189	26.892	32.087	29.864
Lowest	46.391	46.707	50.663	46.391
Highest	187.78	175.43	258.90	258.90
Scale				
0-50 beds	66.312	76.335	79.322	
51-100 beds	74.578	86.885	94.274	
101-200 beds	84.820	91.162	96.379	
201+ beds	139.568	124.997	123.008	

TNSC

Year	1966	1969	1973	3 Year
Average	39.058	43.325	44.639	42.341
Standard Deviation	10.030	11.825	20.159	14.818
Lowest	23.377	22.041	22.974	22.041
Highest	69.360	75.423	197.72	197.72
Scale				
0-50 beds	34.952	40.112	41.762	
51-100 beds	36.995	42.162	44.228	
101-200 beds	44.417	52.921	47.213	
201+ beds	55.562	58.491	54.601	

RNTOTT

Year	1966	1969	1973	3 Year
Average	0.6266	0.6031	0.6026	0.6108
Standard Deviation	0.1013	0.0840	0.0630	0.0852
Lowest	0.4229	0.3969	0.4037	0.3969
Highest	1.0000	0.9911	0.7814	1.0000
Scale				
0-50 beds	0.6207	0.6040	0.5964	
51-100 beds	0.5788	0.5868	0.6295	
101-200 beds	0.6243	0.6144	0.6244	
201+ beds	0.6364	0.5783	0.6022	

little variation in RNTOTT, either across time or with scale.

The variable DOCS (physician population ratios for hospital districts) is an attempt to capture physician input and its quality. No measures per hospital were available so the ratio for hospital districts was applied to all hospitals in the district. Large values of the DOCS variable or the SPRIO variable were expected to reduce crude death rates, cet. par.

The "other" variables are less easily classified. The SIZE variable may be a summary measure.

Scale is a proxy for a number of factors, some of which may be quality related. There are some economies of scale in the provision of facilities and services, so that larger hospitals may attract the patients who require such services. These patients may have more severe prognoses, so that death rates would be expected to rise with scale increases (as they do). Thus size may determine, to some extent, the patient-mix. The availability of such services is also supposed to improve outcomes (quality of care) so that large scale should denote improved ability to care for patients. Thus the overall effect of scale on unadjusted death rates is not clear.

However, the relation of scale to outcomes should be positive (i.e. larger scale implies lower adjusted death rates), as the adjustment factors should remove some of the scale/patient-mix interdependence.

AUTRH is the other variable which was difficult to classify. The use of this variable as a measure of quality was proposed by

Roemer, Moustafa and Hopkins (254). This measure is considered to be a summary or proxy variable for a measure of physician interest in continuing education (also hypothesized to be positively related to quality) or the skill level of the medical staff.

There is little information about this variable. It would be interesting to know what types of deaths are autopsied, and whether this varies across hospitals, as well as to know what motivates physicians to perform autopsies. Although the means by which autopsy rates affect death rates are not known, the general variable is hypothesized to be positively related to outcomes. This is discussed further in Chapter Four.

IV. OUTCOMES MEASURES: ALTERNATIVE ADJUSTMENT FACTORS

As noted in the previous chapters, outcomes measures based on crude death rates should be adjusted to take into account variation in factors beyond the hospitals' control. In particular adjustment for the severity of cases entering a hospital is required, but lack of data on case severity (either micro or macro) forces the use of proxies. Since the basic measure, case severity, is not available for comparison or validation purposes, some exogenous criteria for assessing the "appropriateness" of each adjustment are necessary.

Roemer, Moustafa and Hopkins (254) used the degree of simple correlation with crude death rates as the criterion to determine the "best" adjustment factor. The use of degree of correlation stems from the idea that there is some factor which varies systematically with death rates. Once this commmon trend has been eliminated (captured) then hospitals may be compared. However, it is not clear that the adjustment factor with the highest explanatory power is "the" one that measures the underlying trend.[5]

Roemer et al. (254) have resorted to comparison with other *a priori* hypotheses. They examined the relation of unadjusted and adjusted death rates with scale. They wanted to remove from the series of crude death rates, the positive trend towards higher death rates in larger hospitals, when other studies have shown that diagnosis specific death rates (case-fatality rates) decline as hospital size increases.[6] One should be able to relate the "expected" result with logical reasoning, but care should be taken not to confuse the adjustment factor and its relation to crude death rates with hypotheses relating structure[7] to quality.

However, maintained or *a priori* hypotheses may also be important criteria. A maintained hypothesis in this thesis is that case-mix and demographic mix are strongly related to case severity and thus important determinants of the crude death rates. Once the variation in death rates due to case-mix and demographic mix is removed (such factors do not account for 100 percent of the variation in crude death rates), hospital outcomes may be compared.

Some of the adjustment factors used here are described in this section. All of them adjust the crude death rate and explain variation in this death rate. The overall crude death rate was calculated by dividing total deaths in a hospital during a year by total deaths and discharges (separations).[8]

For 1966, the average crude death rate was 2.02 percent with a standard deviation of 1.14. For 1969, the average was 1.85 percent and for 1973, 1.82 percent with standard deviations around 1.10. The crude death rates vary with hospital size as recorded in Table VII. For all years the larger hospitals had higher average death rates than the smaller hospitals.

There are a number of ways of adjusting for age-sex variation in the hospital population. One alternative is to develop age-sex adjusted death rates (called DRAA1, and DRAA2 in this thesis). The other is to use crude death rates as dependent variables in a regression and include adjustment for age-sex mix in the right hand side variables.

One alternative is to simply include the proportion of separations in each age-sex class. This may present some practical problems such as collinearity when forty (if five year male-female intervals are used) small numbers are used. In studies of hospital costs by Evans (85), Evans and Walker (89), and Barer (14), these problems have been circumvented by the use of factor scores. This procedure reduced the number of independent variables to eight or fewer which explained eighty percent of the variation in the forty proportions. These factors are not easily interpreted and may change from year to year.

These factors will be called AS_j. Residuals of the regression of DR_1 on the AS_j could be used as a direct outcomes measure, but, in

Table VII

Crude Death Rate (percentage)

Year	1966	1969	1973	3 Year
Average	2.0171	1.8530	1.8165	1.8955
Standard Deviation	1.1452	1.0976	1.1372	1.1257
Lowest	0.00	0.00	0.00	0.00
Highest	5.8907	4.2817	4.7597	5.8907
Scale				
0-50 beds	1.7655	1.4289	1.4122	
51-100 beds	1.7980	1.6251	1.6459	
101-200 beds	2.3325	2.4551	2.4507	
201+ beds	3.0790	2.8393	2.5673	

regressions, the age-sex factors can be left on the right hand side of the equation to control for age-sex variation.

As for age-sex variation, the direct use of case-mix proportions presents practical problems. One solution (Evans (85)) is to use factor scores, but a summary measure has been developed and used on Canadian data.

The case complexity (COMPLI) measure provides a single valued variable that describes the case-mix of a hospital. This measure uses techniques of "information theory"[9] to differentiate hospital case-mixes. These measures were originally proposed in Evans and Walker (89), further refined in Walker (315), and used by Barer (14) and others in studies[10] of Canadian hospitals.

The measures are built on the idea that differing distributions of case types across hospitals provide information on the "complexity" of a hospital's case-mix (i.e. if a certain type of case is concentrated in only a few hospitals, rather than spread across all hospitals, then it is likely that type of case is more unusual). To this is added the hypothesis that these are likely to be "more complex" cases.

Case complexities are developed from the hypothesis:

> That complex cases tend to be handled in a few hospitals with more extensive facilities and more specialized staff while relatively straight-forward cases tend to be distributed more evenly over the hospital system. (Evans and Walker (89), p. 399)

These measures depend solely on the distribution of cases, not on facilities, etc.[11] The logic and exact methodology for calculations of the measure is detailed in the above references.

> Basically hospital complexity (CMPXC1) is a weighted sum of the (standardized) complexities of cases treated in the hospital, the weights being the proportion of total case load falling in each case category. (Barer, p. 132)

The hospital complexity[12] measure could be used to adjust crude death rates, if the assumption holds that greater complexity implies that cases admitted are more severely ill. It is expected that death rates would increase as the complexity of cases handled by the hospital increases[13] (i.e. case severity and case-complexity are related).[14]

These variables may be entered alone or used in conjunction with other adjustment variables, particularly those that adjust for demographic factors.

Some adjustment factors are listed in Table VIII giving their corre-lation with crude death rates in 1966, 1969, and 1973. SIZE is included because it is often regarded as a proxy for case severity, and it provides at least one more comparison with the results of other studies using other adjustment factors. The explanatory power of SIZE tends to be lower than other alternatives.

ALS or OCLS (average length of stay and occupancy-corrected length of stay - previously introduced) had the highest correlation with death rates of all the single variables used, except for AGE[15] in 1969. A similar result was found by Roemer et. al. (254) though the degree of correlation is considerably lower than that found in their sample. Similar correlations are found with variables such as COMPL1 and good explanatory power is observed with the age-sex factor scores. Since it is not necessary to assume that these are proxies for case-mix and demographic variation, they seem to be the most appropriate adjustment factors to use.

Table VIII

Adjustment Factors: Correlation with DR

	1966	1969	1973
Variable	Correlation	Correlation	Correlation
ALS	.6345	.4427	.5801
OCLS	.6009	.5984	.7006
SIZE	.3707	.3771	.3128
COMPL1	.5988	.5391	.4940
SPCLC1	.4312		
$AS_1, \ldots, AS_n$*	**	**	**
AGE^{12}	.3392	.5722	.5425

* n = 7 for 1966, 8 for 1969, 6 for 1973.

** Single correlation coefficient not generated, because these variables are always used together. R^2 were .5740 for 1966,.6278 for 1969 and .7979 in 1973 for regression of DR on $AS_1, \ldots, AS_n$ and constant.

V. OUTCOMES MEASURES USED IN THIS THESIS

The adjustments used are ALS/OCLS to calculate LSDR and SADR as developed by Roemer, Moustafa and Hopkins (254), age-sex adjustment using age-sex proportions directly (DRAA1 and DRAA2), and the use of age-sex factor scores and the case complexity measure to calculate DIF1.

A. LSDR/SADR

These two variables were introduced in Chapter Two and are based on average length of stay (ALS) as a severity proxy. ALS has some weaknesses (see Chapter Two for discussion), but is a readily available variable.

The two adjusted death rate - outcomes measures proposed by Roemer et al. (254) were Length of Stay Adjusted Death Rate (LSDR) and Severity Adjusted Death Rate (SADR). Thus:

$$LSDR_i = DR_i - k_1(ALS_i - \overline{ALS})$$

where:

$LSDR_i$ = length of stay adjusted death rate for hospital i;

DR_i = crude death rate for hospital i;

ALS_i = average length of stay for hospital i;

$\overline{ALS}$ = average ALS for the sample of hospitals;

k_1 = coefficient from the regression of DR on ALS (value must be recalculated for each sample).

and $$SADR_i = DR_i - k_2(OCLS_i - \overline{OCLS})$$

where:

$SADR_i$ = severity adjusted death rate for hospital i;

$\overline{OCLS}$ = average OCLS for the sample;

k_2 = coefficient of regression of DR on OCLS.[16]

There is evidence that ALS adjustment has less general applicability than might be desired. LSDR and SADR are most appropriate for crowded short-term urban hospitals that have similar ALS and operate in a common institutional environment. Hospitals which serve the same type of patients should form the comparison group.

Table IX records the average values and standard deviations of LSDR and SADR for the three years under study, and the variation with scale. The negative values for LSDR are for some small hospitals which have low death rates and fairly long lengths of stay. Those small hospitals with relatively long lengths of stays also had relatively low occupancy rates, which reduces the impact of ALS when OCLS is calculated. The negative values for LSDR disappeared when SADR was calculated.

The method for calculating the LSDR and SADR for the three-year sample is not clear. The pooled sample was handled as one big year. There may have been some changes in overall ALS during the time period studied (ALS, in general, fell) so the later year values of LSDR, SADR may be biased upwards. These effects are probably small.

Table IXa

Alternative Outcomes Measures

LSDR

Year	1966	1969	1973	3 Year
Average	2.0383	1.9016	1.8172	1.8956
Standard Deviation	0.8616	0.8759	0.8998	0.8814
Lowest	0.6360	-0.2395	-0.7077	-0.5322
Highest	5.7835	4.2417	3.8303	5.6832
Scale				
0-50 beds	2.0522	1.5849	1.7805	
51-100 beds	1.7716	1.6959	1.6021	
101-200 beds	2.0612	2.2053	2.1460	
201+ beds	2.0597	2.2449	1.8699	

SADR

Year	1966	1969	1973	3 Year
Average	2.0349	1.8653	1.8215	1.8953
Standard Deviation	0.9127	0.8443	0.7667	0.8540
Lowest	0.2441	0.1691	0.4232	0.000256
Highest	5.1430	4.4048	4.2124	5.0128
Scale				
0-50 beds	2.1457	1.9663	1.9702	
51-100 beds	1.6232	1.7502	1.6465	
101-200 beds	1.9414	2.1642	1.9204	
201+ beds	2.1185	2.2192	1.5539	

DRAA1 (percentage)

Year	1966	1969	1973	3 Year
Average	2.0076	1.9328	1.7922	1.9109
Standard Deviation	0.8005	0.8120	0.7129	0.7784
Lowest	0.0994	0.00	0.00	0.00
Highest	4.7725	3.8110	3.2912	4.7725
Scale				
0-50 beds	1.696	1.580	1.444	
51-100 beds	2.061	1.911	1.738	
101-200 beds	2.226	2.430	2.196	
201+ beds	2.774	2.722	2.470	

DRAA2 (index)

Year	1966	1969	1973	3 Year
Average	0.9768	1.0092	1.0170	1.0010
Standard Deviation	0.3851	0.3931	0.3848	0.3865
Lowest	0.1237	0.00	0.00	0.00
Highest	2.3286	1.9872	1.8286	2.3286
Scale				
0-50 beds	0.8145	0.8254	0.8254	
51-100 beds	1.001	0.9767	1.017	
101-200 beds	1.113	1.242	1.231	
201+ beds	1.373	1.379	1.354	

Table IXb

DIF1

Year	1966	1969	1973	3 Year
Average	0.00	0.00	0.00	0.00
Standard Deviation	0.6786	0.5084	0.4878	0.6654
Lowest	-1.6641	-1.2043	-1.3664	-1.4388
Highest	2.5680	1.4166	1.4012	2.8530
Scale				
0-50 beds	-0.0389	-0.0816	-0.0387	
51-100 beds	-0.0756	0.0152	-0.0306	
101-200 beds	0.1314	0.0986	0.0548	
201+ beds	-0.1151	0.1433	0.0989	

B. Age-Adjusted Death Rates (DRAA1 and DRAA2)

Two methods of adjusting death rates for age-sex variation are proposed, DRAA1 and DRAA2.

DRAA1 and DRAA2 are calculated given the average age-sex proportions in the province and age-specific deaths or death rates.

$$DRAA1_i = \frac{\sum_{j=1}^{40} D_{ij}\overline{A_j wts}/A_j wts_i}{\sum_{j=1}^{40} A_{ij}} = \frac{\sum_{j=1}^{40} (DR_{ij} \cdot \sum_{i=1}^{83} A_{ij})}{\sum_{j=1}^{40} \sum_{i=1}^{83} A_{ij}}$$

and

$$DRAA2_i = \frac{\sum_{j=1}^{40} DR_{ij}A_{ij}}{\sum_{j=1}^{40} \overline{DR}_j A_{ij}} = \frac{\sum_{j=1}^{40} D_{ij}}{\sum_{j=1}^{40} \overline{DR}_j A_{ij}}$$

where

i = hospital (1, 2, ..., n);

j = age-sex class (1, 2, ..., 40) by five year intervals;

D_{ij} = number of deaths in class j for hospital i;

A_{ij} = number of patients in class j for hospital i;

$A_j wts$ = average provincial proportion of patients in class j;

$\overline{A_j wts_i}$ = proportion of patients in class j for hospital i;

DR_{ij} = death rate for class j in hospital i;

$\overline{DR}_j$ = provincial average death rate for class j.

DRAA1 provides a death rate adjusted for variation from the provincial average age-sex mix, (i.e. the death rate the hospital would have if its own age-sex specific death rates are applied to the provincial average age-sex distribution) but DRAA2 provides an index of actual to expected death rates given the provincial average death rate broken down by age-sex class (i.e. actual deaths over deaths if provincial average age-specific death rates were applied to the hospital age-sex distribution).

The relative statistics for these two variables are recorded in Table IX. These two alternatives are closely correlated with each other, as expected - .9488 in 1966, .9124 in 1969, and .9450 in 1973.

Upon examination of Table IX, it appears that age-sex adjustment alone leaves some systematic variation with size of hospital, which is more severe than for DIF1. The pattern also differs from that of LSDR/SADR.[17] This may be indicative that further adjustment for case-mix may be advisable. This trend with scale is not the same as that for LSDR or SADR, leading one to hypothesize that they are very different adjustment techniques.

C. Age-Sex Factors and Case Complexity Adjustment (DIF1)

The preferred measure of outcomes was that which incorporated information on both case-mix and age-sex mix. The measure proposed is the residual of actual death rates minus the predicted deaths expected to result from the case-mix and age-sex mix of the hospital. This was DIF1. Thus the regression:

$$DR_i = \alpha + \beta \text{ COMPL1}_i + \sum_{j=i}^{n} \gamma_j AS_{ji} + e_i$$

was run and then DIF1 calculated as

$DIF1_i = DR_i - \hat{DR}_i = \hat{e}_i$

where:

$\hat{DR}_i$ was the predicted value for hospital i for the year in question;

and n = 7 for 1966

8 for 1969

6 for 1973[18]

$COMPL1_i$ = case complexity measure for hospital i;

AS_{ji} = age-sex factor score j for hospital i.

The coefficients for COMPL1 and the age-sex factors are listed in Table X. The coefficients on the age-sex factors do not provide much information, but case complexity has the expected sign. As case complexity increases so do hospital death rates.

Use of age-sex factor scores and case complexity resulted in R^2 of .6485 for 1966, .7854 in 1969, and .8158 in 1973. This implies that in 1966, for example, thirty-five percent of the variation in crude death rates is not explained by age-sex and case complexity variation. This remaining "unexplained" variation is assumed to indicate variation outcomes quality, although it could also include some degree of unaccounted for variation in case severity.[19] Not all of the age-sex factor scores were significant, but the methodology of using factor scores requires that they all be left in. No _a priori_ signs were assigned to the factors because they are difficult to identify.

When DIF1 was to be calculated for the three year sample, some adjustment was needed to the case complexity variable. By

Table X

Coefficients for DIF1

Year	intercept	COMPL1	AS1	AS2	AS3	AS4	AS5	AS6	AS7	AS8	D1	D2	R^2
1966	-2.480	5.392	.5026	-.1524	.1292	-.1184	-.0897	-.2200	.0475				.6485
	(2.06)*	(3.55)	(6.06)	(.96)	(1.69)	(1.33)	(.85)	(1.54)	(.59)				
1969	1.641	3.992	.6821	-.2908	.1432	-.1943	-.0288	.1308	.2308	.0681			.7854
	(1.94)	(4.17)	(10.33)	(2.28)	(2.14)	(2.99)	(.34)	(1.41)	(3.22)	(1.04)			
1973	-.1019	2.265	.8735	-.6417	.4791	.2341	.0659	.1012					.8159
	(.13)	(2.62)	(7.59)	(5.66)	(7.33)	(3.01)	(.712)	(1.11)					
3-year	-1.356	3.831	.6277	-.2975	.2678	-.1841	-.0964	.0179					.6506
	(2.58)	(6.29)	(12.70)	(3.97)	(5.98)	(3.94)	(1.78)	(.30)					
3-year (with	-1.999	4.390	.6095	-.2706	.2718	-.1766	-.0774	.0278			.2500	.1177	.6679
D1,D2)	(3.63)	(7.10)	(12.61)	(3.67)	(6.20)	(3.86)	(1.45)	(.48)			(2.41)	(1.13)	

* t-value in brackets

construction, the average value (province-wide) of the complexity variable is standardized to equal one in each year. However, it was determined that the average value of this variable was rising overtime. To account for this shift an adjusted variable, CMPADJ, which was developed and calculated by Barer (14) was inserted here. The age-sex factor scores were unchanged, but only six could be used, and since the same number were required in each year, the last ones (which contribute the least to explanatory power) were dropped for 1966 and 1969.

Some summary information about DIF1 for the study years are recorded in Table IX. Although the variation (standard deviation) in DIF1 falls over time, the range of values recorded remains quite wide. There is no consistent pattern of variation with scale although there do seem to be generally higher (but not significantly higher) values associated with large scale hospitals.

DIF1 may be used directly as an outcomes measure, or the adjustment factors may be simply added to the right hand side of any regression to explain crude death rates.

VI. OUTCOMES: COMPARISON OF MEASURES

The basic technique for the comparison of alternative outcomes measures is correlation anaylsis. Table XI outlines the correlations between the outcomes variables for the three single years and the pooled series.

All of the relationships are highly significant. The high

Table XI

Correlations for Outcomes Variables 1966, 1969, 1973 and 3-year Sample

1966

Variable 2	DR	LSDR	SADR	DRAA1	DRAA2
			Variable 1		
LSDR	.7762				
SADR	.8149	.8837			
DRAA1	.7551	.4681	.4815		
DRAA2	.7872	.5008	.5109	.9450	
DIF1	.5928	.5505	.6068	.5805	.6791

1969

Variable 2	DR	LSDR	SADR	DRAA1	DRAA2
			Variable 1		
LSDR	.7981				
SADR	.7694	.8539			
DRAA1	.7306	.5733	.4827		
DRAA2	.7874	.6439	.5956	.9541	
DIF1	.4632	.4512	.4425	.4724	.5089

1973

Variable 2	DR	LSDR	SADR	DRAA1	DRAA2
			Variable 1		
LSDR	.7933				
SADR	.6780	.8189			
DRAA1	.7108	.5229	.3034		
DRAA2	.7104	.5387	.2930	.9449	
DIF1	.4229	.4561	.4108	.4725	.4496

3 Year

Variable 2	DR	LSDR	SADR	DRAA1	DRAA2
			Variable 1		
LSDR	.7830				
SADR	.7586	.8578			
DRAA1	.7321	.5087	.4259		
DRAA2	.7548	.5567	.4706	.9433	
DIF1	.5917	.5165	.5525	.5115	.5730

Notes: Significant values are: .2050 for 5%
.2673 for 1%

correlations of the other measures indicate that if low quality care is indicated by one measure, the same result will be found using another measure. The pattern of correlations between measures is very similar for all years. In all cases, DIF1 has the lowest correlation with crude death rates (DR), as would be expected, since its calculation employed the most sophisticated adjustment.[20]

SADR and LSDR are highly correlated (as expected) with each other, as are DRAA1 and DRAA2. They are all correlated with DIF1 to a similar degree which, although significant, is not as large as some other correlations. This lends some support to the hypothesis that DIF1 incorporates some additional adjustment factors.

The high degree of intercorrelation of the alternative outcomes measures, makes it difficult to state that one or the other is really "best". However, given that all information available on case-mix and demographic mix can be used in establishing DIF1, it appears to be the most theoretically appropriate. If the information necessary to calculate DIF1 is not available, it seems that other measures such as LSDR or DRAA1 will give similar results in terms of high and low quality care.

As well, it is interesting to see if the results of assessing the relationship between outcomes, and structure are affected by the outcomes measure used (i.e. does using age-sex adjustment only as in DRAA1/2 result in a different measured relationship with structure than does DIF1, despite the general correlation of the outcomes measures?).

In light of the above results, these measures will also be used when testing the relationship of structure and outcomes quality in the next chapter. However, the concentration is on the use of DIF1 as the outcomes measure with the use of case complexity and age-sex factors as adjustment factors.

FOOTNOTES FOR CHAPTER 3

1. These forms were changed in 1969, but most information was recorded for all years.

2. Some capital measures are excluded because depreciation expense is excluded from the calculation of CASEX.

3. Only one hospital was affected.

4. This variable is the only one which, because of the existence of hospitals with zero RNA hours, had to be calculated in the inverse form. The relation to outcomes is expected to be negative, not positive.

5. One cannot tell if one has "over-adjusted". Some authors (see Goss and Reed (129) seem to indicate that a considerable degree of variation in crude death rates should be "explained" by the adjustment factor, and that the extent of explanatory power should be consistent across samples. They do not say how the critical level of explanatory power should be set.

6. There are probably few disagreements with the argument that case-mix is different (more severe) for larger hospitals. This does not indicate that their quality is better, and does not indicate how much adjustment is necessary (or when there has been sufficient adjustment). Roemer et al. (254) seem to wish to develop a measure which will show better outcomes (lower adjusted death rates) for larger and accredited hospitals. This is, however, the application of even stronger _a priori_ hypotheses.

7. The assumption that any "adjusted death rate" should have lower values for accredited hospitals is really an assumption that accreditation (a structure measure) is positively correlated with outcomes (quality).

8. Maternity separations were _not_ excluded from the patient population before calculating the crude death rate, although hospitals specializing in maternity cases were excluded from the sample. Most hospitals had maternity admissions.

9. The reference for this is Theil (295), but a summary discussion is included in Evans and Walker (89).

10. Previous applications have been for use in cost studies.

11. However, it is true that the distribution of cases will be related to structure, i.e. more severely ill patients will be directed to hospitals with certain facilities. Causality is not clear. Do structures draw patients or has the structure been put in place because patients were coming there?

12. This variable is labelled COMPL1 in this thesis.

13. The *a priori* relationship between death rates and another summary measure of case-mix, also developed by Evans and Walker (89), is not so clear. The measure of "specialization" (SPCLCI) of a hospital is developed from the assumption that "small hospitals are generally prepared to handle a smaller segment of the spectrum of cases than large hospitals" (Barer, p. 141). It is unclear, however, whether increased specialization will result in higher or lower death rates.

14. It is possible that two hospitals could have identical case complexity and demographic mix, yet the severity of cases entering one hospital could be greater. It is thought that the variation across hospitals in case complexity and demographic mix is the most important indicator of variation in case severity, and that other sources of variation will be small. Without a case severity measure itself, it is difficult to test this proposition.

15. AGE is the average age of *deaths* in the hospital. This variable had a strong relationship with crude death rates and indicates that older patients mean more deaths. It backs up the use of the age-sex factors. This variable was used in some regressions without any radical changes in results.

16. In 1966, $LSDR_i$ was calculated as

    $DR_i - .483\ (ALS_i - 8.044)$

    and SADR as

    $DR_i - .234\ (OCLS_i - 8.212)$

    Average ALS was 8.137 in 1969, OCLS 8.244, and the two coefficients .495 and .263 respectively. In 1973, the averages were 7.487, 7.636 and the coeeficients .491 and .314. All coefficients were highly significant.

17. This was consistent with the outcomes measures calculated when only the age-sex factors were used to adjust crude death rates. There was some systematic variation with scale.

18. The number of factors was determined by the number required to explain 80% of the variation in age-sex. See Barer (14) for further discussion.

19. As noted in footnote 13, it is difficult to establish when sufficient adjustment for severity has been made. If one were to assume that <u>all</u> variation death rates unexplained by demographic and case-mix factors represents variation in case severity, that would be equivalent to assuming away all variation outcome quality (at least as measured by death rates). This thesis would then be reduced to relating structure levels and assumed case severity.

    It is interesting to note that adding other possible adjustment factors such as ALS to regressions including COMPL1 and the Age-Sex factors, did not result in any significant shifts in the sign or significance of structure measures.

20. The strength of the relationship between DR and other measures is interesting since a large portion of the variation is supposedly removed when adjustment factors are applied. The correlation results indicate that crude death rates could also be substituted for any other outcomes measure and similar results would be generated. The result that the correlation of DIF1 and DR is lower than the others is still a significant result.

Chapter IV

COMPARING STRUCTURE AND OUTCOMES: CORRELATION AND REGRESSION ANALYSIS

I. INTRODUCTION

This chapter is a report of the empirical investigation of the relationships between structure and outcomes for eighty-three B.C. acute care hospitals.

The first task was to test the hypothesis that structure and outcomes measures are equivalent. This is tested by using correlation analyses of pairs of measures. The results (presented in Section II) depend on the outcomes measures used, but most correlation coefficients are either insignificant or indicate that higher (better) structure implies lower (poorer) outcomes. This is the simplest way of looking at the relationship between outcomes and structure.

This finding has implications for measurement of quality, but it deals only with the appropriateness of substitution of structure for outcomes measures. There may be some interest in whether this state of "no-relation" is the result of too much or too little structure.

The investigation of the second hypothesis, that there is excess structure relative to effective levels, requires some inquiry into the production of outcomes (quality) using regression analysis. Excess structure will be shown if the measured marginal impact on outcomes of additional structure is zero or negative when a functional form is imposed. Essentially, the efficiency of hospital production of one dimension of output, that of quality, is evaluated.

In standard theory, inputs are overallocated to production of some output when the marginal product is less than the opportunity cost of the resources used. If the opportunity cost is greater than zero, a finding of zero or negative marginal product would be indicative of "excess" inputs. It is possible that in health care markets zero marginal product indicates an optimal allocation of resources.

In our society, a decision may have been made that human lives are beyond cost.[1] Hence resources can be allocated to the maintenance of life until their marginal product is zero. Even if this is so, zero marginal product may indicate "excess" if the production function has a flat portion and resources are allocated beyond the start of the flat area.

However, the social opportunity cost of resources is not zero. The finding of zero and sometimes negative impacts of structure on outcomes is indicative of some excess structure.

Multiple regression not only describes the strength of the relationship but also the sign of the impact of additional structure on outcomes. If the coefficient is significant and negative, then the contribution of more structure is negative - i.e. outcomes are poorer.

Underlying the measurement of the impact of structure on outcomes is a simple model. Patients enter a hospital with some characteristics which are described by the "severity" of their illness, and which will have an effect on outcomes. There are two possible outcomes of their hospital stay - death or discharge. It is hypothesized that the split between deaths and discharges is determined, in part, by structure. Additional structure will result in a lower proportion of deaths (death rate), given patient characteristics.

Thus outcome for each case is determined by patient characteristics and inputs (structure). Aggregate quality (outcomes, the probability of death in the hospital (DR_i)) is determined by the characteristics of the hospital population (PC_i) and overall structure (S_i) used or available for use in the hospital.

$$DR_i = f(PC_i, S_i)$$

Various forms may be postulated for this relationship. Patient characteristics are captured by the adjustment factors.

Patient characteristics for a hospital are described by its age-sex mix (AS_i) (assumed exogenous) and/or case complexity (CMP_i). Case complexity may also be partly determined by structure (i.e. certain patients choose or are sent to a specific hospital because of the structure available there).

Thus: $$DR_i = g(AS_i, CMP_i, S_i)$$

or $$DR_i = g(AS_i, h(S_i), S_i)$$

Where $$\frac{\partial DR_i}{\partial AS_i} > 0$$

$$\frac{\partial DR_i}{\partial CMP_i} > 0$$

$$\frac{\partial DR_i}{\partial S_i} < 0$$

$$\frac{dh(S_i)}{dS_i} > 0$$

If only age-sex mix is used to adjust for "severity", the coefficient on S_i would be more positive, than if the CMP variable is included (i.e. the inclusion of CMP_i captures some of the positive relation between structure and DR_i).[2]

This formulation does not completely describe all the activities of a hospital and is thus not a complete specification of the production function. The outcomes dimension is modelled as separable from other dimensions of output which may be jointly produced. These include the education of providers of care, research, amenity levels (which may be proportionate to structure), and even other dimensions of outcomes since death is only one of a large range of outcome states (e.g. reduction of symptoms).

Concentration is on the linear additive model which uses death rates adjusted for case complexity (COMPL1) and the age-sex factors (AS1, ..., ASn) as the outcomes measure. Results are presented in tables for the three years under study, 1966, 1969, 1973 and for the pooled sample.

The results recorded in Tables XVI to XIX are not encouraging to those who would have us believe that structure changes have positive impact on outcomes. Few relationships of appropriate sign (and significance) are noted. However, before accepting a conclusion of no or negative relationship (and hence the existence of excess structure), some investigation of possible deficiencies in and problems with the analysis should be carried out. From Section IV on, such problems are discussed with any empirical evidence that would aid in identification of their severity or their resolution.

One possible problem would be that of errors in the specification of the functional form. The linear additive form is very restrictive, so other forms (inverse, quadratic, interaction, and log-linear forms) were estimated for both the single year and the multiple year samples.

Other problems might be subsumed under the title of errors in variables. Additional empirical evidence on adding variables is presented. Also included is a discussion of the exogenity of certain variables, particularly AUTRH.

Errors in the dependent variable are discussed. The results from using other dependent variables (LSDR, etc) are reported here and compared to those for DIF1. There is also a brief discussion of the limitations of the outcomes measures used.

As the results generally seem to indicate that the measured effect of improved structure on outcomes is at best insignificant, there is a discussion of questions about the nature of the evidence and the degree to which it is evidence of no relation, and implications for policy.

A note on presentation is in order here. Selected regressions are presented in summary tables such as Table XVI. The regressions are numbered for reference. Those pertaining to 1966 are numbered 66XX, those of 1969, 69XX, 1973, 73XX, and for the 3-year sample, 30XX. This numbering scheme is carried throughout the discussion of the results.

In the presentation of results for DR adjusted by case and age-sex mix, the coefficients of the intercept, COMPL1 and the age-sex factors are suppressed - only the coefficients of structure variables and their t-values are reported. For comparison purposes, the R^2 of the base regression (without any structure variables) is given.

When other dependent variables are used, the value of the intercept is suppressed. The base value of R^2 for these regressions is zero.

The most interesting and distinctive results have been abstracted from the volumes of results generated in testing the second hypothesis. For clarity, many variables do not appear in the summary tables. This non-reporting of some variables, for some years, does not mean that they were omitted from the analysis, but that they had insignificant coefficients.

II. COMPARING STRUCTURE AND OUTCOMES MEASURE: CORRELATION ANALYSIS

The calculation of the simple correlation coefficient provides a measure of the strength of the linear relationship between two variables. Any two variables may be compared, but only two at a time. Correlation programmes were run on all the outcomes measures against each structure variable. For a sample of this size, the correlation coefficient for a statistically significant relationship is low. At the five percent level the test value is approximately .205 and at one percent it is only .267.

The computed correlations for each year and the 3-year sample are reported in Tables XII to XV. For all samples a remarkably consistent pattern of results is observable. In no case is DIF1 significantly correlated with a structure variable. There were also few significant correlation coefficients for LSDR. Only PROSTC in 1969 and DOCS in 1973 were significantly related to LSDR, but the sign was positive. SADR had several more significant correlations in each year, but generally these had positive signs (except AUTRH in all samples but 1969).

DRAA1 and DRAA2 exhibited very similar patterns which showed that increased structure was associated with higher age-adjusted death rates, for most variables. The odd significant negative correlation was observed (RNTOTT (1966), WI.1 (1969), and MEDSTC (1973)). Unadjusted death rates (DR) were generally found to be positively related to structure. DR was not significantly correlated with the skill-mix variables (RNTOTT, RNARNT, WI.1) in 1966, 1969, or the 3-year sample .

Looking within years, it can be noted that there is some consistency across outcomes variables. For example in 1966, AUTRH is negative and significant (or close to significant) for DIF1, LSDR and SADR, yet insignificant with DR. In only 1973, MEDSTC is correlated with DRAA1 and DRAA2, SADR, and DR. In the 3-year sample no outcomes measures are significantly correlated with any skill-mix variables.

The difference in the patterns between DRAA1, DRAA2 and DR, and the other outcomes measures seems to indicate that age-sex adjustment

Table XII

Correlations 1966

Summary Variable	DIF1	DRAA1	DRAA2	LSDR	SADR	DR
			Outcomes Variable			
SIZE	.0368	.3583*	.3917*	.0080	.0682	.3839*
ACC	-.0209	.2826*	.3243*	.0049	-.0470	.2902*
TPRN	.0270	.2923*	.3101*	.0039	.0737	.3452*
TPMD	.0730	.3056*	.3184*	.0452	.1192	.3759*
AUTRH	-.1537	.2652*	.2907*	-.2038	-.2476*	.1055
TAS	.0123	.4605*	.4934*	.0581	.0444	.4469*
CASEX	.0845	.3677*	.3895*	.0094	.2376*	.4855*
Input Variable						
TSC	.0827	.4189*	.4341*	-.0100	.1852	.4829*
TNSC	.0799	.3886*	.4054*	.0168	.2283*	.4760*
PROSTC	.0098	.2544*	.2890*	.0164	-.0054	.3099*
TNOPSC	.0728	.4397*	.4605*	.0359	.1909	.5096*
MEDSTC	.0433	.1593	.1385	.1793	.2219	.2925*
RNARNT[1]	.1292	.1163	.1063	-.0316	-.1566	-.0326
RNTOTT	-.1362	-.2285*	-.2317*	-.1161	.1599	-.0879
WI.1	-.0991	-.1409	-.1093	-.1217	.0200	-.1027

[1] Variable expected to have an inverse relation

* Significant $p < .05$

Table XIII

Correlations 1969

Summary Variable	Outcomes Variable					
	DIF1	DRAA1	DRAA2	LSDR	SADR	DR
SIZE	-.0319	.4221*	.4092*	.0863	.0636	.3818*
ACC	-.0177	.1169	.1931	.0941	-.0547	.2271*
TPRN	.0529	.3412*	.3333*	.0796	.0937	.3500*
TPMD	-.0873	.2206*	.2141*	.0573	.0728	.2209*
AUTRH	-.0392	.2820*	.2629*	-.0366	-.1594	.2285*
TAS	.0800	.4780*	.4394*	.1166	-.0066	.4416*
CASEX	.1084	.4657*	.5093*	.1504	.2624*	.5650*
Input Variable						
TSC	.1162	.5233*	.5488*	.1293	.2344*	.5781*
TNSC	.0971	.4185*	.4860*	.1263	.2549*	.5246*
PROSTC	.1408	.5586*	.5144*	.2234*	.0861	.5545*
TNOPSC	.1187	.4975*	.5390*	.1660	.2997*	.5816*
MEDSTC	-.0089	-.0346	-.0528	.1054	.0267	.5816*
RNARNT[1]	.1182	.0613	.0887	.1550	.0847	.0836
RNTOTT	-.1819	.0271	-.0012	.1050	.1213	-.0044
WI.1	-.0462	-.3412*	-.2910*	.0239	.0306	-.1070

1 Inverse relation - positive sign expected

* Significant $p < .05$

Table XIV

Correlations 1973

Summary Variable	DIF1	DRAA1	DRAA2	LSDR	SADR	DR
			Outcomes Variable			
SIZE	.0751	.4362*	.4008*	.0153	-.1282	.3151*
ACC	-.1284	.1989	.2235*	.1244	-.1025	.3663*
TPRN	.1102	.3610*	.3323*	.0495	-.0550	.2717*
TPMD	-.0305	.2108*	.1788	.0331	-.0429	.2081*
AUTRH	-.0505	.2375*	.2110*	-.0966	-.2930*	.1291
TAS	.0725	.5296*	.5176*	.1497	-.1223	.4677*
CASEX	.0519	.1653	.1565	.0408	.1292	.3388*
Input Variable						
TSC	.0781	.1950	.1748	.0301	.0952	.3316*
TNSC	-.0003	-.0392	-.0468	-.0020	.1037	.1150
PROSTC	.1599	.5122*	.4952*	.1238	-.0493	.4872*
TNOPSC	.0303	.0608	.0503	.0218	.0895	.2032
MEDSTC	-.0758	-.3903*	-.3847*	-.0916	.2117*	-.2108*
RNARNT[1]	-.0690	-.1277	-.0978	.0570	.0075	-.0870
RNTOTT	-.0581	.1187	.1507	.1340	.0342	.0545
WI.1	-.1099	-.1789	-.1730	.0876	.2703*	-.0017
DOCS	.1358	.3547*	.3328*	.3320*	.2804*	.5596*

1 Inverse relation - positive sign expected

* Significant at $p < .05$

Table XV

Correlations - 3 Year Sample

Summary Variable	Outcomes Variable					
	DIF1	DRAA1	DRAA2	LSDR	SADR	DR
SIZE	.0494	.4004*	.4009*	.0373	.0107	.3566*
TPRN	.0981	.3287*	.3233*	.0441	.0427	.3231*
TPMD	.1292	.3388*	.3926*	.0772	.0743	.3329*
AUTRH	-.0808	.2612*	.2257*	-.1099	-.2292*	.1540
TAS	.0249	.4251*	.4710*	.1082	-.0257	.4037*
CASEX (deflated)	.0750	.2673*	.3242*	.0684	.1804	.4008*
Input Variables						
TSC	.1059	.3445*	.3758*	.0494	.1529	.4318*
TNSC	.0512	.1719	.2071*	.0347	.1407	.2831*
PROSTC	.0206	.3473*	.4117*	.1139	.0002	.3682*
TNOPSC	.0725	.2682*	.3068*	.0661	.1311	.3667*
MEDSTC	.0421	-.0993	.2806*	.0780	.1418	.0419
RNARNT[1]	.0135	.0009	.0426	.0477	-.0617	-.0336
RNTOTT	.0155	-.0580	.0040	.1271	.1557	.0734
WI.1	.0249	-.1790	-.1790	.0836	.0927	-.0573

1 Inverse relation - positive sign expected

* significant at $p < .05$

alone adjusts death rates in a different manner than using ALS or case complexity and age-sex mix. In particular, the different patterns of DRAA1/2 and DIF1 indicate the importance of including case complexity as an adjustment factor. The signs and significance of the correlation coefficient for several variables are different. These may be "different" outcomes measures. Increasing the degree of adjustment (from DR to DIF1) results in fewer significant correlations.

Several factors could be considered before drawing conclusions. Outcomes measures may not have been sufficiently adjusted for case severity. Insufficient adjustment would tend to mask the true relation between structure and outcomes, and would most likely bias the correlation coefficient upwards. The proposition that there is insufficient adjustment cannot be tested without other outcomes measures. However, given that all the outcomes measures are significantly correlated, the biases should be consistent across all measures. But they exhibit different patterns of correlations with the structure variables even though there are many insignificant correlation coefficients.

Another possibility is that some intermediate factor (such as process or physician quality) has been neglected in the direct comparison of structure and outcomes.

The third possibility is that the relation between structure and outcomes is not linear. Correlation analysis investigates the strength of the linear relationship between two variables. The use of regression techniques allows the investigation of non-linear relationships. Similarly, interactive relations between structure variables can be investigated using regression.

The evidence presented here does not support the wholesale substitution of structure measures for outcomes measures. In particular the substitution of cost measures for outcomes measures in quality studies is not justified. Even the assumption of improved quality with scale increases, or increased service availability (greater TAS), are shown to be inappropriate for this sample. The sample is not thought to be uncharacteristic. The hypothesis that structure and outcomes measures are equivalent is not supported.

The general results of the correlation analysis are further tested in the regression work which follows. It should be noted that the lack of significant and appropriately signed correlation coefficients implies that, unless interactive effects are very powerful, the regression coefficients on structure variables are unlikely to be significant and of the expected sign.

III. COMPARING STRUCTURE AND OUTCOMES MEASURES: REGRESSION ANALYSIS WITH AGE-SEX AND CASE COMPLEXITY ADJUSTMENT

The first investigation uses death rates as the dependent variable with case complexity and age-sex factors as the adjustment variables. Structure variables are added in a linear additive form.

The econometric methodology is ordinary least squares, supported at the University of British Columbia under *TSP (141) (some of the work for 1966) and *SHAZAM (White (320)) (all other work).

The base for comparison for this discussion is the regression of

$$DR_i = \alpha + \beta \; COMPL1_i + \sum_{j=i}^{n} \gamma_j AS_{ji} + e_i$$

The structure variables are added to this base. The coefficient β is always positive (as expected) and significant (death rates rise as case complexity increases) (See Table X for sample coefficients). In each year the coefficients on both case complexity and AS factors were relatively invariant to the combination of structure variables used.

<u>1966</u>: The summary of regressions for 1966 is Table XVI. The base for this year has an R^2 of .6485.

All the recorded regressions include AUTRH because it was a variable which had an appropriate relationship with DR, and was generally significant ($t \geq 1.96$). The coefficients were generally insensitive to the combination of other variables used, except that the use of staffing variables inputs (TSC, TNSC) changed the value of the coefficient on AUTRH from around .97 to around 1.40 (the significance also increased, t=1.8 to t=2.5). Regression 6601 indicates that the R^2 for using AUTRH alone was .6642. This could be increased slightly by adding RNTOTT (6602) or RNARNT (6603). These variables had coefficients of the appropriate sign and t-statistics of over one.

Adding in staff variables increases the R^2 somewhat, but the coefficients have a positive sign indicating that more total staff or more nursing staff will increase the adjusted death rate. Also, if CASEX is used in a regression with TSC or TNSC, the significance (or near-significance) of TSC and TNSC disappears. These variables may be

Table XVI

Regression Summary

Dependent Variable: DR *100
Year: 1966
Specification: Linear

Base Regression: R^2 = .6485

Reg No.	R^2	AUTRH	TNSC	TSC	RNTOTT	RNARNT	WI.1	TAS	CASEX
6601	.6642	-.984							
		(1.84)							
6602	.6692	-.955			-.913				
		(1.78)			(1.04)				
6603	.6709	-.979				.389			
		(1.84)				(1.20)			
6604	.6830	-1.433	-.002						.006
		(2.51)	(.11)						(1.29)
6605	.6819	-1.347		.008					
		(2.56)		(2.01)					
6606	.6818	-1.424	.018					.066	
		(2.54)	(1.62)					(.70)	
6607	.6840	-1.288		.007	-.939				
		(2.31)		(1.82)	(1.09)				
6608	.6868	-1.455							.006
		(2.74)							(2.31)
6609	.6875	-1.380		.009		.353			
		(2.63)		(2.15)		(1.19)			
6610	.6896	-1.426	-.006		-1.061				.007
		(2.50)	(.30)		(1.21)				(1.48)
6611	.6850	-1.266	.019				-.384		
		(2.38)	(1.73)				(.19)		

collinear since much of CASEX is staff costs. When CASEX and TNSC are used together (6609) the sign of TNSC switches to negative, but is insignificant.

Summary variables such as TAS, SIZE, ACC, and training programmes, were generally insignificant and had unexpected signs. CASEX had the wrong sign. The "best" variables for 1966 were AUTRH and the two skill-mix variables, RNTOTT and RNARNT. The value of the coefficient on RNTOTT was such that if all nursing staff was changed to RN's (an approximately 65% increase on average RNTOTT of .6) then crude death rates would fall by about .4 (or 20%). Similarly the impact of increasing the autopsy rate is small (doubling the autopsy rate would decrease DR by approximately .24 or 12%).

1969: Case complexity and the eight age-sex factors explained a higher percentage of the variation in crude death rates (.7854) than they did in 1966. The results of adding structure variables, as recorded in Table XVII, in 1969 were not the same as for 1966. AUTRH, although it carried the appropriate negative sign, had much lower t-values. RNTOTT and RNARNT had the expected sign, but significant t-values were not achieved.

In general, there were no strongly significant determinants of crude death rates among the structure variables. The highest R^2 (.8048) was achieved when AUTRH and PROSTC (6907) were used together. PROSTC was the only variable to have a t-statistic calculated over 1.96, but its effect was positive (increasing PROSTC raised the death

Table XVII

Regression Summary

Dependent Variable: DR *100
Year: 1969
Specification: Linear

Base Regression: R^2 = .7854

Reg No.	R^2	AUTRH	TNSC	RNTOTT	RNARNT	PROSTC	TAS	CASEX
6901	.7943			-1.390				
				(1.77)				
6902	.7916	-.312	.011					
		(.83)	(1.39)					
6903	.7984	-.341	.008	-1.257				
		(.91)	(.97)	(1.53)				
6904	.7960		.006	-1.219				
			(.75)	(1.48)				
6905	.7887				.299			
					(1.05)			
6906	.8048	-.612	.005	-1.428			.009	
		(1.49)	(.62)	(1.73)			(1.51)	
6907	.8048	-.489				.070		
		(1.32)				(2.61)		
6908	.8010					.050		.0008
						(1.77)		(.58)
6909	.8009		-.012	-1.458				.003
			(.77)	(1.74)				(1.32)

rate).[3] Similar levels of explanatory power were achieved using (6906) AUTRH (negative), TNSC (positive but insignificant), RNTOTT (negative), and TAS (positive). The variables such as CASEX, SIZE, TAS all had the unexpected signs or were insignificant.

The results for 1969 are, in some ways, even less encouraging than those of 1966. Once again AUTRH and the skill-mix variables, RNTOTT and RNARNT, are the only variables with the expected signs, but t-statistics are generally less than 1.96. However, they are, at least, greater than 1.

1973: The results for 1973, as reported in Table XVIII are again different. The base regression for 1973 has an R^2 of .8159. Variables that had some explanatory power for 1966 and 1969, such as RNTOTT and AUTRH, are insignificant determinants of DR in 1973. They do, however, have coefficients of the expected sign. There are no other variables of the appropriate sign and significance. PROSTC has relatively large t-values (≈ 1.65), but enters positively. Again the use of TNSC and CASEX together has some impact - the coefficient on TNSC is negative (but still insignificant) and the coefficient of CASEX remains positive.

There are no strong positive results emerging from the analysis for 1973.

Generally the results for the three years do not encourage the formation of strong positive conclusions about the relation between structure and outcomes. It appears that there is no strong and

Table XVIII

Regression Summary

Dependent Variable: DR *100
Year: 1973
Specification: Linear

Base Regression: R^2 = .8159

Reg No.	R^2	AUTRH	TSC	TNSC	RNTOTT	PROSTC	TAS	CASEX	WI.1
7301	.8168			-.009 (.26)	-.606 (.59)				
7302	.8170				-.333 (.33)			.002 (.37)	
7303	.8177	-.256 (.69)	.002 (.68)		-.154 (1.55)				
7304	.8212	-.322 (.86)	.002 (.72)						-2.670 (1.20)
7305	.8221			-.012 (1.43)	-.606 (.59)			.002 (1.46)	
7306	.8235		.0007 (.31)			.033 (1.71)			
7307	.8237			-.004 (.42)		.030 (1.36)		.0006 (.45)	
7308	.8266					.034 (1.65)	-.004 (.86)		-1.518 (.66)
7309	.8290	-.491 (1.22)				.036 (1.70)	.004 (.72)	.00005 (.09)	

consistent (across years) relationship between structure variables, (summary or input varieties) and outcomes. Indeed, it seems that, in some cases, increasing staff and technological adequacy results in higher, not lower, adjusted death rates. The estimated impact of structure on outcomes is zero or negative.

3-year sample: The three years were pooled together to make a large run. Adjusted case complexity (CMPADJ) and six age-sex factor scores were used.

The entire sample may be run with a single constant term and no consideration of the effect of time. Since the average of many structure variables increased in the period under study, and outcomes generally improved (average death rates fell), this technique allows for the most impact of structure on outcomes. To allow explicitly for the effect of time, dummy variables were added to some three-year runs (D1 and D2 for years 1966 and 1969).

The coefficient for D1 was constantly significant and had values around .32 to .35. This indicates that death rates in 1966 were generally higher than death rates in both 1969 and 1973 by about .35 percent. D2 was also positive and had a value about 50 percent of D1, but t-values were below 1.96 (generally around 1.60). This implies that 1969 death rates were not significantly different from those of 1973, if strict 5 percent probability levels are applied. Loosening standards a little, 1969 death rates are slightly higher than those of 1973, but below those of 1966. Results of regressions using D1, D2 as recorded in Table XIX were not very different from those without D1, D2.

AUTRH performs well in the three-year sample. It enters as consistently negative and significant. RNTOTT and RNARNT have the appropriate signs but the t-values are less than significant, although above one, when D1, D2 are included. Other variables, SIZE, ACC, TSC, CASEX, TAS and PROSTC either have very low t-values or are significantly positively related to death rates. This indicates that increasing structure, for the most part, does not improve outcomes, even when trends over time are considered.

When CASEX and TNSC are included in an equation, an appropriately negative coefficient on TNSC and a positive coefficient on CASEX results. The effect is more pronounced when D1, D2 are in the regression. This effect was also observed for each of the yearly samples but was somewhat weaker (t-values were lower). Calculations[4] indicate that at average wages for the years studied the net effect of an additional nursing staff hour per case is very nearly zero, all other variables except CASEX held constant. It is important to note that it is nursing staff increases, not total staff hours, that have this effect.[5]

In conclusion, the investigation of the linear additive relationship between outcomes and structure supports the hypothesis that there is excess structure in the hospital system. The impact of additional structure on outcomes is insignificant or negative. Some variables have strong (and consistent across samples) negative relationships with outcomes, including TAS, TSC, and CASEX. All of

Table XIX

Regression Summary

Dependent Variable: DR *100
Year: 3 year
Specification: Linear

Without D1, D2
(Base Regression R^2 = .6506 (without year dummies, D1, D2))

Reg No.	R^2	AUTRH	TSC	TNSC	RNTOTT	SIZE	TAS	CASEX	RNARNT	PROSTC
3001	.6600	-.407		-.007	-.497			.002		
		(1.61)		(.98)	(.91)			(1.60)		
3002	.6588	-.432		.003	-.355	.0005				
		(3.02)		(.76)	(.65)	(1.32)				
3003	.6613	-.532		-.005	-.516		.004	.002		
		(1.86)		(.78)	(.94)		(.96)	(1.25)		
3004	.6643	-.608	.004		-.343		.004			
		(2.16)	(2.04)		(.64)		(.93)			
3005	.6674	-.697	.010		-.349		.004	.002		
		(2.43)	(2.22)		(.65)		(1.01)	(1.47)		

With D1, D2
(Base Regression R^2 = .6679 (with year dummies, D1, D2))

Reg No.	R^2	AUTRH	TSC	TNSC	RNTOTT	SIZE	TAS	CASEX	RNARNT	PROSTC
3006	.6767	-.526			-.778					
		(2.11)			(1.49)					
3007	.6752	-.507							.189	
		(2.03)							(1.05)	
3008	.6873	-.585		-.012				.004		
		(2.36)		(1.79)				(2.83)		
3009	.6954	-.999	.004		-.686		.010	-.0001		
		(3.52)	(.96)		(1.32)		(2.51)	(.07)		
3010	.6873	-.944		.004			.011			
		(3.34)		(1.23)			(2.84)			
3011	.6914	-.739	.004							.032
		(2.94)	(2.37)							(2.08)
3012	.6905	-.592		-.014	-.828			.004		
		(2.40)		(2.08)	(1.56)			(3.01)		

these have been used as quality indicators under the assumption (not validated) that they improve outcomes.

However, several variables showed some appropriate relationships with outcomes. AUTRH showed appropriately signed coefficients (significant in some samples), as did the skill-mix variables which measured the proportion of registered nurses in various functions (RNTOTT and RNARNT). This seems to indicate that the type of staff employed or added to a staff complement may have a positive impact on outcomes; specifically more highly qualified nursing staff has an effect different from general staff additions or other professional staff.

Before such conclusions are accepted it is necessary to consider other reasons why such results may have occurred. The following discussion considers possible problems. In most cases, further study only reinforces the above conclusions.

IV. **PROBLEMS AND THEIR EMPIRICAL RESOLUTION**

The possible problems fall into several broad categories. These are specification errors, problems with independent (right hand side) variables, and problems with the dependent variable. Each of these will be discussed in turn.

A. Specification Errors

The linear additive form was used in the original investigation of the relationship between structure and outcome, but this is a rather restrictive form of the possible relationship. Alternatives include inverse, interaction, quadratic and log linear forms. The information required to estimate more complex alternatives (such as the CES) was not available, and the required assumptions were judged heroic. The impact of structure on outcomes is not the same for all hospitals when such forms are used, but the calculation of the impact for each hospital would be time consuming, so average values are used in examples.

1) Quadratic: The form specified here is of the type:

$$DR_i = \alpha + \beta\ COMPL1_i + \sum_{j=1}^{n} \gamma_j AS_{ji} + \delta_1 TNSC_i + \delta_2 TNSC_j^2 + \ldots$$

In this case the marginal impact of structure (such as TNSC) on DR is $\delta_1 + 2\delta_2 TNSC_i$. If structure is related to outcomes in an appropriate manner, $\delta_1 + 2\delta_2\ TNSC_i$ should be negative. If δ_1, is positive, then δ_2 should be negative.

The results using this specification are recorded in Table XX. The use of higher order terms in the quadratic specificiation does not affect the magnitude or sign of the coefficients of COMPL1 and the age-sex factors.

The results are generally not encouraging.[6] There are very few significant coefficients (on either first or second order terms) and few second order terms with negative signs. Those that did have a negative sign were generally very small.

Table XX

Regression Summary

Dependent Variable: DR *100
Years: 1966, 1969, 1973
Specification: Quadratic

1966 Base Regression: R^2 = .6485

Reg No.	R^2	AUTRH	TNSC	TNSC2	TSC	TSC2	CASEX	CASEX2
6612	.6756	-1.211	.029	-.00007				
		(2.20)	(.46)	(.11)				
6613	.6812	-1.422			.019	-.00005		
		(2.46)			(1.15)	(.73)		
6614	.6849	-1.885						
		(2.59)						

1969: Base Regression: R^2 = .7854

Reg No.	R^2	AUTRH	AUTRH2	RNTOTT	TNSC	TNSC2	PROSTC	PROSTC2	SIZE	SIZE2	CASEX	CASEX2
6910	.8016	.569	-1.643	-1.253	.021	-.0001						
		(.56)	(.99)	(1.46)	(.58)	(.35)						
6911	.8071	-.507					.117	-.003				
		(1.36)					(2.01)	(.91)				
6912	.8133				.001				.002	-.0000002		
					(.18)				(1.47)	(2.84)		
6913	.7956	-.352									-.0005	.000005
		(.92)									(.08)	(.50)

1973 Base Regression: R^2 = .8159

Reg NO.	R^2	AUTRH	TNSC	TNSC2	SIZE	SIZE2	PROSTC	CASEX	CASEX2	TAS	TAS2
7310	.8233		-.0008		-.002	-.0000004					
			(.25)		(1.62)	(.83)					
7311	.8188	-.290	.011	-.00005							
		(.75)	(.87)	(.90)							
7312	.8253						.038	.0003	-.0000003		
							(1.85)	(.16)	(.22)		
7313	.8239		.0007							.007	-.00006
			(.23)							(1.31)	(1.43)

Thus in regression 6912, the net impact of SIZE on DR is .002 + 2 x (-.0000002) x $SIZE_i$. Only if the hospital has a bed complement of more than 5000 beds will the net impact be zero or less. For the size of hospitals in the sample, the net impact is still positive (i.e. as the size of the hospital increases, so does the adjusted death rate).

For most other variables with negative coefficients on higher order terms, the calculations of impact had the same result - a positive impact on death rates. The exception was TAS in 1973. The TAS net impact is appropriately signed if the TAS of a hospital is greater than 50. A TAS of 50 is well above the sample average TAS. This effect was not observed in other samples.

The quadratic specification performs no better than the linear additive form. Any further investigation of this form is not expected to yield better results.

2) Inverse: The functional form specified here is:

$$DR_i = \alpha + \beta\ COMPL1_i + \sum_{j=i}^{n} \gamma_j AS_{ji} + \delta_1 \frac{1}{TNSC_i} + \delta_2 \frac{1}{TAS_i} + \ldots$$

If structure is related to improved outcomes $\frac{-\delta_1}{TNSC^2}$ should be less than zero, so the presence of a negative δ_1 indicates an inappropriate relationship. Only variables that are always non-zero can be used in the inverse form, so that the number of variables that can be used in the inverse form is limited. Regressions using some inverse forms are found in Table XXI.

There are many insignificantly and negatively signed coeffi-

Table XXI

Regression Summary

Dependent Variable: DR*100

Years: 1966, 1969, 1973

Specification: Inverse

1966: Base Regression: R^2 = .6485

Reg No.	R^2	AUTRH	TNSC	1/TNSC	1/RNTOTT	1/CASEX	1/SIZE
6615	.6611			1.874	.426	-122.21	
				(.06)	(1.18)	(.79)	
6616	.6819	-1.405				-194.76	
		(2.49				(2.00)	
6617	.6739	-1.179		-24.388			
		(2.16)		(1.46)			
6618	.6551		.011				-4.741
			(.88)				(.57)

1969: Base Regression: R^2 = .7854

Reg No.	R^2	TNSC	RNTOTT	1/TNSC	1/SIZE	1/CASEX
6914	.8052	.008			-12.266	
		(1.08)			(2.38)	
6915	.7964		-1.374			-70.204
			(1.74)			(.83)
6916	.7951		-1.329	-5.929		
			(1.66)	(.51)		

1973: Base Regression: R^2 = .8159

Reg No.	R^2	AUTRH	TNSC	TAS	PROSTC	1/TNSC	1/TSC	1/SIZE	1/CASEX
7314	.8333		.004					-12.719	
			(1.09)					(2.75)	
7315	.8169	-.217							-30.315
		(.57)							(.27)
7316	.8189			.006		1.209			
				(1.07)		(.11)			
7317	.8252				.036		-4.182		
					(1.82)		(.20)		

cients in all years. In the 1966 regressions, 1/CASEX is significant (6615, 6616) but has a negative sign. 1/TNSC and 1/RNTOTT have positive coefficients (6615) but 1/TNSC has a t-value of .06 while 1/RNTOTT has one of 1.18. Adding other variables in the inverse form did not affect the sign or significance of variables such as AUTRH or TNSC.

In 1969 the inverse forms had negative signs and the same was true in 1973 (except 1/TNSC but this coefficient was not significant). Overall explanatory power was not increased when the inverse forms were used. Runs with variables in the inverse forms confirm the observation of no strong or appropriately signed relationships between structure and outcomes.

3) Interaction: In this functional form, explicit recognition of possible interaction between pairs of variables involved including the cross products as variables in the regression. The form specified is:

$$DR_i = \alpha + \beta\ COMPL1_i + \sum_{j=1}^{n} \gamma_j AS_{ji} + \delta_1 TNSC_i + \delta_2 TNSC_i \cdot SIZE_i + \delta_3 SIZE_i + \ldots$$

or a number of other interactions such as

$$\delta_2 TNSC_i \cdot COMPL1_i$$

$$\text{or } \delta_2 TNSC_i / COMPLI_i$$

The marginal contribution of structure ($TNSC_i$) to death rate reduction would be:

$$\delta_1 + \delta_2 SIZE_i$$

$$\text{or } \delta_1 + \delta_2 COMPL1_i$$

or $\delta_1 + \delta_2/COMPL1_i$

This is less than zero if δ_1 is positive when δ_2 is negative and greater than $\delta_1/SIZE_i$ or $\delta_1/COMPL1_i$.

Only the results using crossterms are reported in Table XXII. None of the interactive variables for any of the three years was significant, nor had an impact on the coefficients of the variables without interaction (i.e. the sign and significance of TNSC was not affected by the addition of say, TNSC·SIZE). Some of the variables had t-values above one or 1.5. The combination of RNTOTT and RNTOTT·COMPL1 had, in 1966 (6624) a net impact near zero as the negative value of δ_1 (on RNTOTT) would be offset by the positive value of δ_2 when multiplied by COMPL1 (which averaged close to one).

In 1969, the combinations of TNSC and SIZE (6918) and CASEX and COMPL1 (6920) have relatively high t-values. In the former case, only if the hospital has more than 150 beds, is the net impact of TNSC on death rate reduction positive. Thus the net impact of SIZE is positive for about half the hospitals in the sample. In the latter case, the sum of $\delta_1 + \delta_2 \cdot COMPL1_i$ is close to zero for CASEX·COMPL1. Now the negative sign on CASEX is cancelled by the positive sign on CASEX·COMPL1. The same is true for (7321) TNSC and TNSC·COMPL1.

Except for the one result in 1969 for TNSC and SIZE, the addition of interactive variables did not provide results which would seriously bring into question the conclusion of no relationship between structure and outcomes.

Table XXII

Regression Summary

Dependent Variable: DR* 100
Years: 1966, 1969, 1973
Specification: Interaction

1966: Base Regression: R^2 = .6485

Reg No.	R^2	AUTRH	RNTOTT	RNTCMP*	TSC	CASEX	CASCMP*	TNSC	TNCMP*	TNSIZE*	SIZE	TAS	TNTAS*
6619	.6840	-1.285	-.666	-.199	.008								
		(2.27)	(.11)	(.04)	(1.81)								
6620	.6550					-.0001	.003						
						(.01)	(.22)						
6621	.6548							-.028	.044				
								(.36)	(.52)				
6622	.6540							.012		.000007	-.0004		
								(.92)		(.10)	(.07)		
6623	.6566							.018				.001	-.0001
								(1.25)				(.14)	(.80)
6624	.6677		-17.090	18.578	.005						.00002		
			(1.33)	(1.25)	(.99)						(.03)		

1969: Base Regression: R^2 = .7854

Reg No.	R^2	RNTOTT	RNTCMP*	TNSC	TNSIZE*	TNCMP*	SIZE	CASEX	CASCMP*
6917	.8050	-25.502	28.212	.007					
		(1.89)	(1.80)	(.97)					
6918	.8009			.011	-.00008		.005		
				(1.57)	(1.79)		(1.55)		
6919	.7920			.062		-.062			
				(1.04)		(.90)			
6920	.7979							.011	-.011
								(1.68	(1.43)

* Variable Defintion
RNTCMP = RNTOTT*COMPL1
CASCMP = CASEX*COMPL1
TNCMP = TNSC*COMPL1
TNSIZE = TNSC*SIZE
TNTAS = TNSC*TAS

1973: Base Regression: R^2 = .8159

Reg No.	R^2	TNSC	TNCMP*	TNSIZE*	TNTAS*	SIZE	TAS	CASEX	CASCMP*
7318	.8231	-.00007			-.00004		.003		
		(.02)			(.79)		(1.04)		
7319	.8225	.005				-.00008		.007	
		(.92)				(1.21)		(1.26)	
7320	.8235							-.006	.007
								(1.58)	(1.67)
7321	.8213	-.068	.082						
		(1.47)	(1.41)						

4) Log Linear: This functional form can be used if adjusted death rates (DIF1) are used as the outcomes variable. However, since DIF1 may include zero and negative values it had to be scaled upwards before logs were taken. A constant value of five was added to DIF1 to eliminate all negative values but retain the result that higher values of DIF1 implied poorer outcomes. The use of DIF1 means that the R^2 observed are improvements on zero.

The hypothesized relationship is:

$$DIF1_i = A\, S_{1i}^{\alpha}\, S_{2i}^{\beta}$$

which is estimated as

$$\ln DIF1_i = \ln A + \alpha \ln S_{1i} + \beta \ln S_{2i}$$

where non negative structure variables may be substituted for S_{1i} and S_{2i} (structure variables 1 and 2). Negative α and β are desired. No efforts were made to constrain the sum of α and β (as in the Cobb-Douglas production function) because a unitary elasticity of substitution was not thought to be applicable.[7]

Several other log-linear forms such as

$$\ln DIF1_i = \alpha + \beta S_{1i} + \gamma S_{2i}$$

or $$DIF1_i = \alpha + \beta \ln S_{1i} + \gamma \ln S_{2i}$$

were tested, but with no better results.

The regression summary for the log-linear form is Table XXIII. The R^2 are very low for all years.[8] Only the constant term had t-values greater than 1.96. The log linear form yielded results no different from other specifications.

It seems that the investigation of alternative functional forms

Table XXIII

Regression Summary

Dependent Variable: ln(DIF1+5)
Years: 1966, 1969, 1973
Specification: Log Linear

All variables (except constant) are ln X_i

1966: Base Regression: R^2 = 0

Reg No.	R^2	CONST	CASEX	SIZE	RNTOTT	TNSC	TSC
6625	.0341	1.756	-.164	-.011	-.101	.191	
		(3.48)	(.96)	(.47)	(.87)	(1.19)	
6626	.0313	1.846	-.177		-.092	.174	
		(3.96)	(1.06)		(.81)	(1.12)	
6627	.0171	1.485		-.009	-.128		.021
		(4.85)		(.36)	(1.14)		(.25)

1969: Base Regression: R^2 = 0

Reg No.	R^2	CONST	CASEX	SIZE	RNTOTT	TNSC	TSC
6921	.0061	1.498		-.012			.035
		(6.97)		(.67)			(.59)
6922	.0056	1.709			-.010	-.029	
		(10.10)			(.11)	(.66)	
6923	.0169	1.439	.109	-.005		-.115	
		(4.30)	(.95)	(.30)		(1.10)	

1973: Base Regression: R^2 = 0

Reg No.	R^2	CONST	CASEX	SIZE	RNTOTT	TNSC	TSC
7322	.0285	1.594		-.017		.023	
		(12.13)		(1.53)		(.60)	
7323	.0096	1.347	.089		-.005	-.076	
		(4.19	(.86)		(.04)	(.76)	

or specifications has strengthened the conclusions from the linear-additive model. Using the alternative forms gave rise to similar results - structure variables are not related to outcomes either strongly or in the expected (hypothesized) manner.

B. Problems with Independent (Right Hand Side) Variables

There are a number of questions that can be asked about the exogenous variables used in this analysis. They fall into two general categories: 1) questions about missing variables or adjustments to variables used; and 2) questions about the independence of some variables. Problems of the first type may be partly resolved by adding such variables (if they exist). Problems of the second type are less easily investigated.

1) Missing Variables:

Given the generally weak performance of all the variables used, it is not thought that the addition of other structure variables would give rise to radically different results.

Under the topic of missing variables falls a major shortcoming of the analysis - the inability to integrate physician quality into the analysis. The implicit assumption has been that physician ability is either evenly or randomly distributed among hospitals.[9] Data limitations severely restrict the possibilities of including variables that are physician related.[10]

It is plausible that hospitals that offer better structure (best facilities, etc.) would attract the better quality physicians.[11] Structure would then be correlated with physician quality and correlated with outcomes. Any structure-outcomes effects would be reinforced by physician quality considerations. Thus omission of physician quality, in a sense, biases the analysis in favour of strong structure-outcomes relationships.

The opinion that the omission of physician quality has a minor impact is buttressed by consideration of the conclusions of some physician quality-hospital quality studies. Palmer et al. (231) suggest that some selection mechanism such as that mentioned above may be in effect (i.e. best physicians to best hospitals) but that the organizational structure may determine overall quality levels. For example, teaching hospitals or highly structured Kaiser-plan hospitals, exert more control over the medical staff and encourage certain forms of staff activity which in turn may affect quality. The Teamster studies (81, 208) concluded that "the site of practice was more associated with the quality of care provided than were the qualifications of the physician" (Palmer et al. (231, p. 698). Another study quoted in Palmer et al. (231, p. 698-9) stated that "the variance in outcomes attributed to the effect of individual surgeons was 0.2 percent, while that attributed to the 'hospital effect' was 1.11 percent" (231, p. 698). The impact of physicians was relatively small, so that the omission of such variables would not radically affect results.

However, an attempt was made to include some variables which were physician-related.

Some variables describing physician availability (definition outlined in Chapter III under DOCS) were added to the linear additive models, for 1973. None of the physician-population variables were significant at the five percent level. The only variable that consistently had t-values greater than 1 (1.5+) was DOCS (total physicians per 100,000 population), but the sign of the coefficient on this variable was positive indicating that more patients died when there were more physicians available. The only variable with a consistent negative sign was SPRIO (the proportion of physicians that were specialists), but all t-values were well below 1.[12] The addition of physician variables did not significantly increase explanatory power or modify the results.

A second concern may be that the data used do not capture quality variation in the inputs (structure) other than physicians. This is particularly important for staffing variables, as the quality of the workers themselves may vary from hospital to hospital, and it would be expected that hospitals with higher quality staff would have lower death rates at similar comparative staffing ratios. Differences in experience and qualification within groups, are not easily captured.

High quality staff has been hypothesized to be related to a number of factors including experience, qualifications, interest (aptitude), and goals of the institution. Some of these, such as qualifications, have already been included with the use of skill-mix variables such as RNTOTT and RNARNT. In some samples these skill-mix variables were significant, and it seems that increasing the proportion of RN's on staff in nursing roles has a small positive effect on outcomes.

The only variable that seemed to be of use was the skill-mix variable, WI.1. A hospital with a higher proportion of staff in more highly paid employment categories would have a larger WI.1. However, inclusion of this variable had no strong effect on results.

Similarly, some staff quality variation may be captured in variables reflecting the teaching status of the hospital. If teaching hospitals are expected to have a different mix of staff (more educationally motivated) then training programme variables should be significant. These variables did not perform well.

It appears that the staffing variables, although crude, cannot be easily disaggregated or adjusted to capture any variations within staff groups across hospitals.

The omission of either physician quality, physician inputs, or quality variation in other inputs (particularly staff inputs) cannot be shown to seriously bias the results. The addition of variables which might represent such factors had no impact on either explanatory power, or the sign and significance of other structure variables.

2) Problems of the exogeneity of "independent" variables:

The variables used on the right hand side in our equations, the adjustment variables, and the structure variables, are assumed to be independent of each other and exogenously determined. In some cases the exogeneity of certain variables and the independence of death rates and these variables may be questioned.

Two major questions emerge here. One concerns the independence of AUTRH (the proportion of deaths autopsied) which is strongly tied to the existence of those deaths. The other concerns staffing rates and their dependence on other variables also used in the equation (COMPL1 or Age-sex factors).

AUTRH was a variable that performed well in that it was significant in several samples, and had a negative relationship with death rates. One explanation for this result involves consideration of AUTRH as a proxy variable for physician skill-levels and interest in learning from deaths. Alternatively it may signify the presence of other factors such as hospital style and attitude which imply that quality is better. But even when AUTRH is used as a proxy it is necessary to consider the relationship between AUTRH, quality, and death rates.

It is evident that hospitals with zero death rates will have zero autopsy rates. However, there are very few hospitals with zero death rates. It is likely that hospitals with very few deaths will perform few autopsies because they may not have the facilities. This biases the expected sign of the relationship in the opposite direction to that observed (i.e. hospitals with high autopsy rate would also have high adjusted death rates).

Essentially what is modelled is a production process that has two major inputs, hospital provided inputs, and patient characteristics. These are combined and result in two types of outputs; deaths and discharges. Deaths may, however, fall into two categories, those autopsied and those that are not autopsied.[13] Clarification is required to link these autopsies to quality improvement and lowered death rates. This link is found in the quality input - which has a positive relationship with autopsies, and a positive relationship through the production process with outcomes. The regressions do not include any variable directly representing the quality input, but AUTRH, since it may be related, replaces it.

Criticism may also be made of the way the relationship between AUTRH and DR is specified. In particular, one would like to consider the existence of a lag between changes in the proportion of deaths autopsied and in the death rate. Autopsy rates were rather constant across hospitals over time and it was not practical to check out such effects.

No strong conclusions can be made about the relationship between outcomes and AUTRH. It is most likely a proxy for certain factors. The measured relationship between AUTRH and adjusted death rates is not sufficiently strong to encourage extensive analysis of the relation. Some caution is then suggested when noting the encouraging results for AUTRH.[14]

The independence of other explanatory variables, particularly staffing variables may be of concern. To a great extent, the level of staffing in a hospital will depend on the case mix in the hospital, but it is also true that the case mix admitted may depend on the staff available to care for them.

Generally, multiple regression analysis methods allow for such interactions when both variables are present in the equation. Thus, when looking at the coefficient on TNSC when COMPL1 is in the regression, the net effect of changes in TNSC on DR when COMPL1 (and other factors) are held constant is estimated. This allows for some interaction between TNSC and COMPL1. Further adjustment was possible, if interactive terms were used, but the evidence presented in the section on specification errors indicated that results (in terms of the relationship between TNSC and outcomes) were not improved.

To sum up, consideration of possible problems with right hand variables has not substantially modified conclusions. Although the possibility of such problems confounding the analysis cannot be dismissed, there is little evidence that such problems can be easily solved. Even consideration of physician quality is unlikely to change results.

C. Problems with Dependent (Left Hand Side) Variables

Problems with dependent variables are of three general types. The first two are concerned with the possibility that the age-sex factors and case complexity are not appropriate or sufficient adjustment factors. The third set of concerns are with the outcomes variables themselves and their limitations. It is less easy to come to strong conclusions about the impact of such shortcomings on the results of the empirical analysis.

1) Hospital-Specific Effects Not Captured in DIF1:

Age-sex factors and case complexity have considerable intuitive appeal as adjustment factors (proxies for case severity), but it is possible that they are insufficient to such an extent that the results of the comparison of structure and outcomes are drastically affected.

The key question is whether or not complexity is a suitable proxy for case severity. No other way of incorporating diagnositic information other than case complexity, was tested. This is an area of possible future research. The complexity variable cannot pick up cross-hospital variation in severity within diagnostic categories that is also unrelated to age-sex mix. If these variations across hospitals are constant over time, then a pooled time series cross-section analysis employing hospital specific dummies would capture this effect.[15]

Hospital specific variations in outcome would be observed, if hospital dummies[16] were significant. Inadequate or inappropriate adjustment (when COMPL1 was used) would be shown, if staffing variables (structure) had negative and significant coefficients when the eighty-two hospital specific dummies were used in the regression. The large number of variables and observations pushed the computer programmes to the limit, and the results were confusing.

When the dummies were used with the age-sex factors and case complexity variables, these variables explained .8754 (R^2) of the variation in crude death rates. Some of the coefficients[17] on individual dummies had low t-values, but a test of the significance of the dummies altogether indicated that they significantly improved explanatory power. This implies that there are some hospital specific effects, but no explanation of what causes these effects.

However, when hospital specific dummies were included, the complexity variable coefficient became negative, indicating that increasing the case complexity significantly reduced the death rate. This effect persisted even when year-specific dummies were added to capture any time series effects. The effect on the case complexity variable is difficult to explain.

But, more importantly, the signs or significance of the coefficients on the structure variables were unchanged. Thus even when allowance was made for some hospital specific factors affecting outcomes, the tested relationship between outcomes and structure was in the unexpected direction. Increasing structure does not result in lower death rates. The exception was AUTRH; it carried a negative sign. Conclusions were not changed.

2) Use of Other Outcomes Measures:

All of the empirical results for regression analysis reported so far have been generated when age-sex and case-complexity adjusted death rates have been used as the dependent variable. It is possible that the adverse results are a consequence of the use of this variable, so runs were made with the alternative outcomes variables (LSDR, SADR, DRAA1, DRAA2) used as dependent variables.

Once again the amount of information generated was formidable, so much abstraction was necessary. Some investigation of the problems in section (1) and (2) were also carried out when the alternative outcomes measures were used, but no results were generated that would modify the general conclusions reported here.

The consistency of results for the alternative measures with the previously reported measure is striking. No matter what variables and combinations of variables are used, there are few significant and appropriately signed relationships between structure variables and outcomes measures. If anything, more relationships are strongly in the inappropriate direction.

a) Length of Stay Adjusted and Severity Adjusted Death Rates (LSDR and SADR):

As noted in Chapter Three, adjustment for case severity using average length of stay or occupancy-corrected length of stay resulted in two outcomes measures which were significantly correlated with DIF1. LSDR, SADR and DIF1 also exhibited similar patterns of correlation with structure measures. Thus, it would not be expected that the regression analysis would yield results that were at a great variance with those that resulted when death rates were adjusted using case complexity and the age-sex factors.

LSDR and SADR perform in a similar but not exactly the same manner. If anything, SADR shows a stronger relation (but in the wrong direction) with structure than LSDR.

The regression results for LSDR in Table XXIV for 1966 indicate that structure variables do not explain much of the variation in LSDR

Table XXIV
Regression Summary
Dependent Variable: LSDR
Specification: Linear

Year: 1966

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	CASEX	AGE
6628	.1401						.035
							(3.63)
6629	.1596			-.609			.033
				(1.36)			(3.35)
6630	.0143	.0009			1.014		
		(.26)			(1.09)		
6631	.0321		.008	-.895			
			(.81)	(1.67)			
6632	.0410	.003		-.826	.928		
		(.88)		(1.52)	(1.01)		
6633	.0306	.005		-.881		-.002	
		(.52)		(1.60)		(.31)	

Year: 1969

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	RNARNT	TAS	CASEX	PROSTC	AGE
6924	.1678									.027
										(4.04)
6925	.1908		.008		1.170					.027
			(1.05)		(1.10)					(3.98)
6926	.0451			-.510	.930			.002		
				(1.01)	(.80)			(1.65)		
6927	.0747			-1.058		.736	.014			
				(1.69)		(1.66)	(2.05)			
6928	.0803				2.278		.010			
					(1.87)		(1.86)			
6929	.0848			-.429	2.193		.013			
				(.63)	(1.78)		(1.78)			
6930	.1038	-.003		-1.143					.097	
		(.53)		(2.07)					(2.52)	

Year: 1973

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	TAS	CASEX	AGE
7326	.1092		.007	-.239				.023
			(1.28)	(.48)				(2.98)
7327	.1282		.009	-.335	2.041			.023
			(1.63)	(.67)	(1.31)			(2.90)
7328	.0417	.003		-.706	2.42			
		(.92)		(1.32)	(1.53)			
7329	.0835			-1.516		.017		
				(2.38)		(2.54)		
7330	.0849			-1.531		.017	-.0002	
				(2.32)		(2.49)	(.30)	

Years: 3 Year

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	TAS	CASEX	PROSTC	WI.1
3013	.0229		-.004		1.286		.001		
			(.52)		(1.92)		(1.00)		
3014	.0338	.005		-.662					2.488
		(2.12)		(2.20)					(1.65)
3015	.0438		-.005	-.668	1.290		.001		
			(.66)	(2.30)	(1.95)		(1.46)		
3016	.0597		.002	-.824				.047	2.993
			(.40)	(2.73)				(3.13)	(2.02)
3017	.0804		.003	-1.301	1.238	.013	-.0004		
			(.36)	(3.73)	(1.93)	(3.10)	(.26)		

NOTE: Value of constant term not reported in all tables for LSDR.

since R^2 are low. The variable AGE (average age of deaths in the hospital) explains more than any structure variable. Its significant positive relation with LSDR indicates that despite adjustment using average length of stay there is still some systematic variation with the age of deaths. However, the inclusion of AGE does not significantly affect the observed relation with structure variables. The significance of AGE is noted in all other years and for SADR.

Generally, the results for LSDR in 1966 do not contradict the results generated when death rates adjusted for case complexity and age-sex mix were used. In some cases, such as the RNTOTT variable, the sign has changed to the inappropriate sign. AUTRH seems to be the strongest variable.

In 1969, similar results were shown. Variables with larger t-values such as CASEX, PROSTC, TAS, and RNTOTT (in certain combinations), all have positive coefficients, indicating that higher structure levels are associated with higher LSDR. AUTRH is the strongest negatively signed variable.

AUTRH is also the strongest variable in 1973, although including the AGE variable wipes out the high t-values for AUTRH. Other variables with high t-values, TAS and TNSC have inappropriate signs.

The results were similar for the 3-year sample. AUTRH was negative and significant, and other variables were of the inappropriate sign, but several had relatively high t-values (TSC, TAS, RNTOTT, PROSTC, WI.1). R^2 as high as .08 were reached with some combinations of variables (3017).

The results using SADR as the dependent variable were very similar since the two outcomes variables are derived from the same adjustment factor. Comparison of LSDR and SADR summary tables (Tables XXIV and XXV respectively) bears this out. In 1966, for example, AUTRH is the strongest determinant of the outcomes variable with the appropriate sign. If anything, the relationship is stronger (t-values are higher) for SADR. Overall, R^2 are higher for SADR than for LSDR. In 1966, R^2 as high as .1600 were obtained (6640) using TSC (positive), AUTRH (negative) and RNTOTT (positive).

Similar results are found for 1969 and 1973. AUTRH performs well, but other structure variables do not. In the 3-year sample, the TNSC and CASEX (3018), combination showed some promise, but changes in the combination of variables used (3021) result in the disappearance of this effect. PROSTC, in this sample only, carries a significant negative coefficient, for some reason.

The results using LSDR and SADR are similar to those obtained when DIF1 was used, which reinforces the idea that DIF1 and LSDR (SADR) may be close substitutes, and that outcomes and structures are not strongly related in a positive manner.

b) Age-adjusted death rates (DRAA1 and DRAA2):

These outcomes measures are age-specific death rates which adjust only for variation in the demographic mix of the hospital. In the correlation analysis it was noted that DRAA1 and DRAA2 were more strongly correlated with structure variables than DIF1 or LSDR/SADR

Table XXV
Regression Summary

Dependent Variable: SADR
Specification: Linear

Year: 1966

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	RNARNT	CASEX	AGE
6634	.1525							.039 (3.81)
6635	.1852			-.838 (1.79)				.036 (3.48)
6636	.0685	.007 (1.96)			1.014 (1.09)			
6637	.1273		.027 (2.76)	-1.653 (3.14)				
6638	.1321	.006 (.62)		-1.659 (3.03)			.003 (.56)	
6639	.1499			-1.477 (2.83)		-.539 (1.46)	.005 (2.45)	
6640	.1626	.012 (3.16)		-1.604 (3.05)	1.777 (2.00)			

Year: 1969

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	TAS	CASEX	PROSTC	AGE
6931	.1191								.022 (3.31)
6932	.1922		.017 (2.38)		1.356 (1.33)				.021 (3.26)
6933	.0385	.003 (.82)			2.03 (1.79)				
6934	.0498			-.738 (1.22)	1.631 (1.48)	.007 (1.06)			
6935	.0885						.004 (2.66)	-.039 (1.31)	
6936	.1771			-1.367 (3.02)	.823 (.79)		.005 (3.66)		

Year: 1973

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	SIZE	AGE
7331	.1159						.019 (3.27)
7332	.2604		.013 (3.03)	-1.064 (2.70)	1.246 (1.01)		.023 (3.85)
7333	.0963			-1.235 (2.65)	.793 (.63)	.00003 (.07)	
7334	.1381	.005 (1.98)		-1.499 (3.46)	1.536 (1.20)		

Years: 3-year

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	PROSTC	CASEX	SIZE
3018	.0579		-.014 (1.65)			-.039 (2.46)	.005 (3.03)	
3019	.1291	.005 (.90)		-1.450 (5.15)			.001 (.79)	
3020	.1560		-.004 (.51)	-1.387 (5.37)	1.641 (2.72)		.003 (2.66)	
3021	.1606	.010 (4.41)		-1.479 (5.25)	1.818 (3.08)			-.0002 (.59)

NOTE: Value of constant term not reported in all tables for SADR were, but these strong correlations were in the wrong direction. If such effects carry over to the regressions, it would be expected that there will be no outstanding positive results when DRAA1 or DRAA2 are used as outcomes variables.

The regression summaries for DRAA1 are Table XXVI and for DRAA2, Table XXVII. Several regressions were run with case complexity (COMPL1) included as a right hand side variable. The significance and positive sign of the coefficient on this variable indicates that age-adjusted death rates still have a significant relationship with case complexity.[18] However, results with and without COMPL1 are presented. In some years the addition of case complexity seems to affect the significance of other variables (6644 vs 6645). The R^2 are generally quite high for all years.

When DRAA1 was the dependent variable for 1966, AUTRH was not a significant determinant. TSC is positively related to DRAA1, but the significance level is dependent on the presence or absence of COMPL1. RNTOTT, RNARNT and CASEX have the expected signs in some situations, although the t-values for the skill-mix variables are only around one. The sign of the coefficient depends on the presence of TSC in the regression. If TSC (positive and significant) is included, CASEX has a negative sign (6648), but if TSC is not included, the coefficient on CASEX is significant and positive.

In 1969 the results are also not encouraging. AUTRH, RNTOTT are not significant. TSC, TNSC, PROSTC and CASEX, have significant coefficients, but they are positive. In this year WI.1 is negative and

Table XXVI
Regression Summary
Dependent Variable: DRAA1*100
Specification: Linear

Year: 1966

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	RNARNT	TAS	CASEX	COMPL1
6641	.2148								4.205 (4.82)
6642	.2245		.009 (.89)	.152 (.32)					3.358 (2.76)
6643	.2325		.013 (1.22)			.282 (.90)	-.004 (.44)		3.838 (2.45)
6644	.2439	.003 (.76)			-.976 (1.30)				3.424 (3.03)
6645	.1602	.010 (3.28)			-.815 (1.04)				
6646	.1295			.698 (1.51)		.302 (.92)		.004 (2.25)	
6647	.1788	.008 (1.85)				.287 (.90)	.009 (1.34)		
6648	.1794	.020 (2.44)			.423 (.91)			-.006 (1.37)	

Year: 1969

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	WI.1	PROSTC	TAS	CASEX	COMPL1
6937	.3528		.029 (3.41)				.054 (1.68)			1.05 (.70)
6938	.3670	.138 (4.45)								2.27 (2.25)
6939	.2852			-.504 (.97)	.033 (.03)			.017 (2.16)	.003 (2.33)	
6940	.3110		.024 (4.24)	.225 (.55)		-.594 (2.22)		-.0003 (.07)		
6941	.3374	.017 (5.84)		.247 (.64)	.790 (.83)					
6942	.3689	.013 (3.95)					.052 (2.31)			

Year: 1973

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	RNARNT	CASEX	PROSTC	MEDSTC	TAS	COMPL1
7335	.3714				-.0839 (.30)			.039 (1.80)	-2.489 (-2.45)		2.450 (2.65)
7336	.0953	.004 (1.60)		.634 (1.53)	1.601 (1.31)						
7337	.2302		-.034 (-3.88)	.512 (1.35)		.155 (.47)	.005 (4.08)				
7338	.3152					-.204 (.73)		.074 (4.27)	-2.414 (-2.29)		
7339	.3119		-.004 (-1.31)	-.611 (1.36)						.024 (5.37)	
7340	.3425			-.684 (-1.54)			-.0005 (-.98)	.054 (2.27)		.017 (3.03)	

Year: 3-year

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	SIZE	TAS	CASEX
3022	.0932		-.017 (2.37)		.005 (.00)			.005 (4.09)
3023	.1807	.004 (1.77)		.315 (1.24)	.343 (.64)	.001 (3.58)		
3024	.1843		-.006 (.82)	-.027 (.09)	.036 (.06)		.016 (4.24)	.001 (.98)
3025	.2210	.016 (3.48)		-.272 (.93)	.266 (.51)		.015 (4.43)	-.004 (2.94)

NOTE: Values of constant term is not reported in Tables for DRAA1.

Table XXVII
Regression Summary

Dependent Variable: DRAA2
Specification: Linear

Year: 1966

Reg No.	R^2	TSC	AUTRH	RNTOTT	RNARNT	TAS	CASEX	COMPL1
6649	.2393							2.111
								(5.17)
6650	.2689	.001		-.454				1.722
		(.83)		(1.29)				(3.25)
6651	.1753	.005		-.374				
		(3.51)		(1.01)				
6652	.1885	.004	.253	-.347				
		(2.69)	(1.15)	(.94)				
6653	.1915	.008	.240				-.002	
		(2.24)	(1.09)				(1.09)	
6654	.2062	.003			.137	.006		
		(1.76)			(.92)	(1.74)		

Year: 1969

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	PROSTC	SIZE	CASEX	COMPL1
6943	.2712	.004		.0003	.146				1.555
		(2.16)		(.0014)	(.30)				(2.66)
6944	.2785	.0001				.016			1.084
		(3.95)				(.97)			(1.50)
6945	.1711		.009	.325	.312				
			(3.17)	(1.67)	(.64)				
6946	.2190	.005		.208	.433				
		(3.96)		(1.06)	(.92)				
6947	.2589	.003				.034			
		(1.87)				(2.97)			
6948	.2484					.032	.0002	.0007	
						(2.19)	(.58)	(1.32)	
6949	.2187		.007	.133	.273		.0005		
			(2.45)	(.63)	(.59)		(2.22)		

Year: 1973

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	RNARNT	MEDSTC	PROSTC	CASEX	COMPL1
7341	.3371					-.0106	-135.97			1.068
						(.07)	(-2.40)			(2.07)
7342	.0899	.00218		.285	1.062					
		(1.57)		(1.27)	(1.61)					
7343	.1992		.00533	-.0283		-.158	-303.71			
			(2.04)	(.12)		(.92)	(-3.80)			
7344	.2454					-.0630	-132.74	.0383		
						(-.41)	(-2.30)	(4.05)		
7345	.3047						-184.87	.0297	.000378	
							(-2.57)	(2.45)	(1.12)	

Year: 3-year

Reg No.	R^2	TSC	TNSC	AUTRH	RNTOTT	TAS	CASEX	SIZE
3026	.1386		-.011		-.071		.003	
			(3.06)		(.25)		(5.18)	
3027	.1639		.002		.117			.001
			(.89)		(.43)			(5.92)
3028	.1896	.002		.125	.134			.005
		(2.49)		(1.00)	(.51)			(3.25)
3029	.2357		-.004	-.113	-.093	.009	.001	
			(1.33)	(.79)	(.35)	(4.89)	(1.73)	
3030	.2475	.005		-.210	.044	.009	-.001	
		(2.37)		(1.48)	(.17)	(5.35)	(1.67)	

NOTE: The value of the constant term for all regressions using DRAA2 is not reported.

significant, but the impact of having a wage index twice the provincial average is small (less than a 10% decrease in deaths).

In 1973, MEDSTC, the measure of full time physicians employed by the hospital per case, shows up as a strong determinant of DRAA1 with a negative effect. However, the impact of increasing this variable (which has a mean of .00087 per case) is small, (i.e. doubling the MEDSTC results in a net change in DRAA1 of .02 or about 2% reduction). TNSC has the appropriate sign and some significance (depends on other variables used). The sign of AUTRH depends on the other variables used in the equation. If TAS was included the coefficient on AUTRH was appropriately negative.

The three single year samples all seem to indicate that there are some definite non-positive relations between structure and outcomes (i.e structure improves but outcomes do not),[19] but this result is not consistent across samples. The 3-year sample did not yield any significant results. The sign of TNSC depends on the combination of variables used, and most strong relations (CASEX, TSC, SIZE, TAS) are inappropriately signed.

The results for DRAA2 (detailed in Table XXVII) are very similar to those for DRAA1. In 1966 there are few significant relationships. Only TSC is consistently significant, but the sign is positive. In 1969 negative signs are conspicuous by their absence in the summary table. In 1973 MEDSTC is, as for DRAA1, the outstanding variable.

The 3-year sample has few negative signs. The presence of a negative sign on TNSC depends on the presence of CASEX (positive) in the regression. TAS and SIZE are strongly related to DRAA2, but increasing the size and facilties in the hospital increases DRAA2.

The use of DRAA1 or DRAA2 instead of LSDR, SADR, or DIF1 as the outcomes variable does not change the results. There are some appropriate signs, but they are not consistent over time. Changing the combination of variables tends to affect drastically the sign and significance of the coefficient on such variables. This may be the result of multi-collinearity. In general, the evidence supports the results when DR was used as the dependent variable.

The key conclusions that result from the comparison of the relations between structure and various outcomes measures is that, while there is some variability, there is consistency across outcomes measures. No structure variables are shown to have any significant positive relationship to quality as measured by any of the outcomes measures in all samples. This is not surprising when the high correlation of all the outcomes measures is recalled.

The consistency of the results indicates that substitution among outcomes measures will not drastically affect results. Thus, the choice of death rates adjusted with case complexity and age-sex factors as outcomes measures did not determine the result of the analysis.

3) Shortcomings of the outcomes variables used:

All of the outcomes measures used in this thesis are based on death aversion as the goal of the hospital and as the quality measure. This is a crude measure of outcomes. Other alternatives such as health status improvement in the short run, health status improvement over time, and reduction in discomfort during the hospital stay all require information which is not currently available.[20] There should be further consideration of the shortcomings of the outcomes variables used and their possible impact on the results of the empirical analysis.

The first possible problem is, in a sense, an econometric one. There is very little variation in the dependent variable across hospitals after adjustment. For some years, despite the large cross-section sample, the adjustment factors accounted for more than eighty percent of the total variation in death rates. Although this underscored the importance of adjustment, very little is left for the struture variables to explain.

If insufficient variation in the dependent variable is a problem, random coefficients may result. The usual solution to such a problem is to increase the sample size, but it is already relatively large, and tripling its size (when the three years were pooled) had little effect on the results. There is no obvious answer to this problem or a test for its severity.

However, the small variation in death rates after adjustment is an important result in itself. The lack of variation may indicate that, for the hospital system as a whole, some sort of plateau (as far as death rate reduction is concerned) has been reached with the general level of resources allocated to hospitals. If this is the case, then differential changes in staffing levels will be expected to cause little or no change in adjusted death rates. This implication has policy relevance, when combined with the existence of negative impacts of structure increases on outcomes. There may be more effective ways of reducing death rates than pouring more resources into the hospital sector.

A second confounding factor may be that patient movement among hospitals[21] may occur. This is important only if the movement is systematic (say all patients about to die in hospital X are transferred to hospital Y) and not picked up by the case complexity variable. Analysis of the extent of this movement is feasible only if a case-by-case analysis is possible; for this data is not generally available.

There is also the possibility that multicollinearity is so severe that the coefficients are very different from the actual. However, in this case, unless multicollinearity is pervasive, changing the combination of variables should result in changing values of coefficients on some variables. In some cases the magnitude of coefficients was affected by the variables used in combination, but this was not a consistent observation. Collinearity of the adjustment factors and structure is possible, but results when the adjustment factors were dropped from the equation were even worse. The possibility of spurious results should be considered in future research.

The fourth concern is with the definition of output and quality used. It is possible that hospitals may have commitments to goals other than death aversion.

The focus has been on the production of discharged cases (hopefully in better health than when they arrived) as the output of the hospital. The education of providers is an important part of the activity of some hospitals, and much facilities, staff, and effort may be devoted to this goal. The concentration on outcomes as output, means that staff committed to education may be included with staff who are assigned to patient-care only.[22]

Two approaches to resolving this limitation were used. One involves removing the education components from some staffing variables and the expenditure variables, so that only staff directly attributed to in-patient care is included. The second approach controls for the existence of educational commitment by the inclusion of variables representing teaching status, or by removing from the sample such hospitals.

There was no difference in the performance of variables which included staff whose main duties were education related (such as TSC) and those which were strictly made up of patient-care staff hours (such as TNSC).

The variables which denoted teaching hospitals, TPMD or TPRN, were insignificant determinants of death rate variation, and their inclusion in a regression had no effect on the magnitude or significance of other variables. Dropping the teaching hospitals from the sample also did not improve results. It is thought that the educational component of hospital output was not a factor which seriously distorted results.

However, separability of the quality dimension from other output dimensions has been imposed on the analysis. The assessment of the impact of such an assumption can only be carried out through further research into the overall production function for hospital output.

Consideration of the narrowness of the definition of quality may give rise to some unease. Consumers may be concerned with more than just outcomes, (e.g. have secondary concerns about structure) and with more than outcomes defined simply in terms of mortality aversion.

Consideration of consumer concerns about the level of amenities provided in the hospitals may also be included in the analysis. These amenities may take many forms, but most of them provide a link between structure and a definition of quality. Thus patients may think that the amenity level is improved because there are more nurses, greater space for each bed, attractive decor, shiny equipment surrounding their beds, and TV in every room. All are elements of structure. While one cannot dispute that such considerations may be important to the patient, they are of secondary importance after outcomes. There has been little research on patient perceptions of amenity levels and their association with quality, and patient "willingness to pay", but the Iowa Nursing Utilization Study (2), indicated that increasing the availability of nursing staff (increasing structure) did not affect patient satisfaction levels. The staff were the chief beneficiaries.

If amenities are valued in a positive sense, it is necessary to define amenities, and establish the extent of their impact on the structure measurements. It is not clear that variations in the amenity level will show up in many of the structure measures used, since much of the "amenities" may take the form of non-staff inputs. The relation between "amenities" and "excess" structure has not been established.

Some of the amenities may be subject to charges. If, as postulated, the fully informed consumer regards these as "extras", then charging for them would permit patients to choose amenity levels. To a certain extent this is observed in the Canadian health insurance system. Hospital care is provided up to a certain amenity level (standard ward care), but consumers must pay (either directly or through supplementary insurance) for the greater privacy (and amenity) of a private or semi-private room. TV's are rented.

The implications for quality assessment are that before testing the hypothesis regarding excess structure, consumer desired amenity levels should be cleaned out of the structure measures. Systems to control hospital costs (including amenity levels) could use charges to patients, or regulation of amenity levels. However, if informed consumers do value amenities and amenity levels do affect the structure measures used, some of the conclusions about "excess" structure are weakened.

Consideration of broader outcomes measures has been discussed in previous chapters. Although consumers may look at outcomes in terms of their overall health status before, during, and after their hospital stay, data limitations do not enable any further investigation of such ideas. However, death as an outcome would still form a subset of any broader outcomes measure. The narrowness of the outcomes definition may be the cause of the small variation in quality noted across hospitals. Broadening the definition of outcomes may result in more variance in the sample.

In summary, the investigation of several problems with the dependent variables has not established the existence of any conclusion at strong variance with that drawn in the first part of this chapter. The use of alternative outcomes measures, dropping teaching hospitals and adding hospital specific dummies did not change results.

If anything, the consideration of problems and queries about the analysis has strengthened our conclusions. Considerable investigation and experimentation has not disclosed any reason why the results should not be regarded realistic and reliable.

V. **CONCLUSION**

This chapter has summarized the empirical research into the relationship between aggregate measures of structure and outcomes for eighty-three hospitals in British Columbia for the years 1966, 1969, and 1973. The empirical work provided the evidence for the testing of two hypotheses. The first was that structures and outcomes measures were equivalent.

The correlation analysis led to the conclusion that the one-to-one relationship between structure and outcomes was, at best, weak, and the hypothesis was rejected. Wholesale substitution of structure for outcomes measures was not supported. As well, assumptions that costs, facilities, and staffing are positively correlated with outcomes were not supported.

The second hypothesis was that there was excess structure in hospitals. This would be established if the marginal impact of structure on outcomes was zero or negative (i.e. inputs (structure) were supplied to the point of zero or negative returns in terms of effectiveness in reducing overall adjusted hospital mortality rates).[23]

Regression analysis of the relationship between structure and outcomes was used to estimate this marginal impact. Again the conclusion is overwhelmingly negative. Very few structure variables are significant determinants of outcomes, and most of these have an estimated coefficient which indicates that increasing inputs or improving structure will result in poorer outcomes. This conclusion held under rigorous cross-examination when alternative specifications, errors in variables, alternative outcomes measures, and several other factors were considered and investigated. The consistency of all results was striking.

Several popular proxies for outcomes or indicators of quality were either insignificant or of the wrong sign. These included scale, accreditation, teaching status, overall facilities as measured by the technological adequacy score (TAS), and costs per case. There was no evidence that costs may be substituted for outcomes (quality) or that larger scale hospitals provide better care.

However, some limitations could not be fully investigated. For example, only the quality dimension of output (outcomes) has been investigated. Separability of outcomes from other dimensions of output has been assumed. The testing of a full system would require much more rigorous and complex specification, data which are not readily available, and/or the use of assumptions (such as cost minimization) which may not be justifiable. In addition, the extent to which case-mix and demographic variables adequately adjust for case severity across hospitals, may be questioned.

As well, it is possible that the observed lack of relation is because both outcomes and structure measures are too aggregate and gross to be sufficiently sensitive to measure the impact of structure on outcomes. The testing of this possibility requires new and improved outcomes data, severity measures, and more information on structure.

Another possibility is that some key determining factor has been omitted. This factor could be process or physician quality. The discussion of the role of physician quality in determining mortality rates led to the conclusion that including physician quality was unlikely to affect results. Future analyses should include process, if possible.

These results have implications about the technical efficiency of production of quality in the hospital sector. The observed insignificance of the measured relationship could be the result of either of two situations:

1) that production is taking place at a point(s) completely off the production frontier (i.e. observed points bear no relationship to the technically efficient production set). This may be because the production frontier is unknown[24] or because there are no incentives in the market that force producers to use cost-minimizing production

techniques. Given the lack of profit as a motivating factor, non-existent competitive forces, and the possibility that outcomes are not the only concern of providers, this is a realistic possibility.

2) that hospitals have reached the flat portion of the production function, so that adding resources to production of hospital care does not affect death rates (outcomes). For this case two possible situations exist - one is that production is taking place on a local plateau and that beyond some point, additional structural improvements will result in a further improvement in outcomes. The other is that production is at a global maximum or the final plateau. Either situation cannot be ruled out econometrically, but the existence of such flat spots is important for policy. The onus is on those advocating increased structure or the use of structure measures as quality proxies to show that it is simply a local rather than a global situation.

Policy implications of such results are important. If hospitals are operating off the production function, policies of two types can be implemented. One policy would attempt to provide incentives for producers to move towards cost-minimizing techniques, while another policy would reduce resources available to hospitals in an attempt to force awareness of resource constraints. Resources would be cut back until further cuts were reducing outcomes quality. Unfortunately there is no assurance that such a program will result in the elimination of "excess" structure only, because an inefficient producer (the hospital) may not know where the inefficiencies lie. Tighter resource constraints may make hospitals more aware that resources are not free, but cuts may be made in "essential" structure as well as "excess" structure.

A monitoring system, based on outcomes, must then accompany such cost-cutting programs, to ascertain whether the program is having a negative effect on outcomes (either system-wide or for any one hospital). Combined with information on outcomes and patient mix (which may change in response to such programs), should be information on what cuts or changes are made by each hospital. This may increase knowledge of what constitutes "essential" structure. Such structure or process may then be excluded from the cost-reduction scheme by regulation on quantities. This might be called the "trial and error" method.

It may not be possible to link changes in structure to changes in outcomes. Then a number of other factors would have to be considered. These include that the program has resulted in a change of attitudes (patient or provider) which has been detrimental to outcomes, or even that there has been systematic sabotage of the program. These are unlikely. More possible is that the rigidity of current resource allocation patterns does not permit innovation or cuts of the type that would reduce excess structure. Equipment and programs in place will continue to exist, even though continued use contributes little to outcomes. Cuts may be feasible, in the short run, only in variable inputs, and these may have some positive impact on outcomes. Changes to a more specific resource constraint program may be required, in this case.

Emphasis on effectiveness of resource employment is required.

The existence of an insignificant relationship between structure and outcomes indicates that some resources or programmes have not been effective in reducing mortality, although they may have been effective in other dimensions.[25]

If the flat portion of the production frontier has been reached, and the marginal productivity of additional resources in reducing death rates is zero or lower,[26] then it may be possible to reduce the resources applied to hospital care without affecting outcomes.[27] This implies that policy measures such as reduction in bed availability and the application of budgetary restraint on hospitals may be effective in reducing costs without endangering quality of care. There will be limits to the extent that cuts can be made, as it is not known how far the plateau extends. But it should be easy to detect when the limit is reached. Assessment of the impact of such policies requires monitoring of outcomes.

Additional concentration of resources in acute care is not indicated. Concentration on other forms of care, particularly extended care, palliative care and home care (given an aging population) may be more efficient and effective. Policies aimed at changing life styles may be more effective in improving the quality of life and reducing mortality rates than applying more resources to the acute care hospital system.

There is some evidence that redistribution of resources currently devoted to hospital care may also affect death rates. However, the weak positive results of RNTOTT or RNARNT are not sufficient to make strong statements. More research into the effectiveness of various types of staff is suggested.

The evidence presented in this chapter is certainly suggestive of excess structure. Criticisms that would focus on the possibility that the adjustment factors are not valid proxies for case severity, or that the quality measures are too narrowly defined (i.e. other dimensions of health status may be affected by improved structure) cannot be ruled out altogether. Thus before pursuing any one of the policies suggested above (such as massive cutbacks of resources), more supporting evidence should be provided.

However, the evidence contained in this chapter provides a rejoinder to those who say that the relationships are obvious, or that there is no reason to do more work on outcomes. The burden of proof has been shifted to those who must now find and use other information to establish the weaknesses in this analysis.

The subsequent discussion will assume that excess structure exists. Assuming consumer preferences for efficacious care, the existence of "excess" structure can be explained only by the demands or preferences of other economic agents in the market who must to some degree be able to impose their preferences on hospitals. The next chapter explores these ideas in more detail.

FOOTNOTES FOR CHAPTER FOUR

1. The acceptance of this premise would invalidate cost-benefit and cost-effectiveness analysis.

2. This is observed to some extent. The runs for age-sex adjusted death rates have more significant positive coefficients on structure than do runs using age-sex and case complexity.

3. It is possible that PROSTC is positively related to case severity which has not been completely captured by the adjustment factors used, so that the sign result is not entirely unexpected.

4. The net effect on death rates of an increase in nursing staff hours per case is $\delta_{TNSC} + \delta_{CASEX}$ x change in CASEX as the result of an increase in nursing staff. An additional nursing staff hour resulted in an increase in CASEX of approximately $3.00.
   Average CASEX (3 year) = $310.78 (1970$)
   Total staff costs ≈ 80% CASEX
   TNSC ≈ 50% TSC
   Total nursing staff costs were:
   40% x 310 ≈ $124/case
   TNSC ≈ 42 hours/case
   1 hour nursing staff/case costs approximately $3.00.

   Net impact on DR:
   a) 3 year without D1, D2
   (3001) -.007 + 3x.002 = -.001
   (3003) -.005 + 3x.002 = +.001

   b) 3 year with D1, D2
   (3008) -.012 + 3x.004 = 0.000
   (3012) -.014 + 3x.004 = -.002
   is very close to zero.

5. Questions may be raised about the possibility that adjustment for severity is not perfect, so that some of the staffing measures, particularly PROSTC, may be picking up a further severity effect. At this point it is not possible to disprove that this may affect the sign of the coefficients or such variables.

6. The results were so poor that no runs were made on the pooled sample.

7. Several runs were made constraining α and β to sum to one. The results were nonsensical since high t-values were attained on the coefficient, but the R^2 were negative (even as negative as -10.000).

8. But these low R^2 would be in the range of those found when DIF1 was used as the dependent variables in linear additive models instead of using DR and including COMPL1 and the Age-Sex factors on the other side.

9. Other alternatives include assuming that physician ability is correlated with case-mix, hospital size, and even structure availability. Some of these variables would then be proxies for physician ability.

10. It is difficult to tie physicians to specific cases or hospitals because medicare and hospital insurance data are collected separately, and to combine them is a project of some magnitude.

11. The reasons for this, and the form of physician preferences, is discussed at some length in Chapter Five.

12. An example: 1973, linear additive:
Base Regression: R^2 = .8159

Reg. No.	R^2	AUTRH	TNSC	TAS	DOCS	SPRIO
7324	.8298	-.432	-.001	.009	.028	
		(1.08)	(.33)	(1.62)	(1.84)	
7325	.8315	-.440	-.0005	.009	.030	-.284
		(1.10)	(.15)	(1.65)	(1.95)	(.82)

13. Not only the death rate but also the likelihood of autopsy, is determined by patient characteristics. Patients dying of "old age" are less likely to be autopsied. If a hospital has many long-term patients in short-terms beds it will have a higher death rate, but most likely, a lower autopsy rate too. This implies that the autopsy rate is picking up incomplete age-sex adjustment. However, such incomplete adjustment would not be a problem when ALS is used as the adjustment factor since ALS is correlated with age. Yet, AUTRH has a negative sign as a determinant of LSDR and SADR, too.

14. The results are not clear enough to suggest that policies that would require an increase in the AUTRH would mean that outcomes would improve. (i.e. a quality improvement programme would not start by saying perform more autopsies!)

15. An alternative is to add other adjustment factors such as ALS to COMPL1 and the Age-Sex factors. The results with respect to the impact of structure on outcomes were not affected by the addition of ALS to the regression.

16. A large file was set up with a dummy variable for each hospital (82 dummy variables, since the constant was included).

17. The coefficients on each of eighty-two dummy variables are not reported.

18. Using age-adjusted death rates and COMPL1 is an alternative way of adjusting for age-sex mix and case complexity without having to run factor analysis, etc.

19. There is a possibility that this is due to insufficient correction for severity in these outcome variables.

20. In some ways, the poor results of structure-outcomes relation tests should spur efforts to develop and implement more sophisticated outcomes measures. The lack of relation is still not proven until it has been shown for quality measures based on more sophisticated (and different) outcomes measures (i.e. use of a measure that is not totally determined by death rates).

21. It is also possible that patients are sent home to die by certain hospitals, but investigation of the proportion of deaths in the province that occurred in hospitals, indicates that the proportion was steady for the period under study.

22. There may be some effect on outcomes of the commitment to education as there may be improved care, more supervision, more highly developed in-service education programmes in teaching hospitals. But some staff allocation may only have impact on educational activities.

23. The assumption that the optimal point of supply is where marginal impact is still positive (not zero), is invoked here.

24. This is possible given the rather uncertain state of medical knowledge about appropriate techniques of treatment.

25. Some new technologies (such as diagnostic imaging) fall into this category. Studies indicate that although diagnosis is facilitated, the impact on mortality is difficult to assess. See Russell (259), Bunker, Barnes and Mosteller (35) for other examples.

26. See Mushkin and Dunlop (215), p. 3.

27. In a sense, it is assumed that hospitals are efficient (i.e. all on the production possibility frontier). Thus cuts would not have a differential impact on outcomes, until the constraint actually has an effect.

Chapter 5

PHYSICIANS, HOSPITALS, AND STRUCTURE

I. INTRODUCTION

In the previous chapter the empirical relationship between structure and outcomes was investigated at some length. The conclusion was that there may be more structure available in hospitals than necessary for effective care. Structure has been supplied to the point where the differential impact on outcomes is zero or negative.

In a world of fully informed consumers and agents of the consumer (physicians) who possess full information, only efficacious care would be demanded by rational consumers, and only effective structure would be employed at the aggregate level. In a world which is less than perfect, if all economic agents had preferences similar to consumers, again, only effective care would be observed. The existence and persistence of excess structure cannot be easily explained unless there is another source or type of demand for structure, such that structure is valued for other than its impact on health status.

This chapter investigates several sources of this demand. Some authors have suggested that such demands can be attributed to consumers. Feldstein's (96, 97) and Newhouse's (222) models of hospital behaviour are of this type. The hypothesis that excess structure is the result of physician dominance of hospital decisions is also examined.[1] Physician preference for structure is further investigated.

Physician dominance over patients, and over hospital resource allocation decisions, is discussed in Section III.

Section IV discusses two ways in which providers' preferences and structure can be linked. Structure (capital and labour in hospitals) can affect the constraints on a physician's general ability to maximize his utility. Secondly, structure can be of direct concern to the physician in that structure (or structure features) enter the utility function directly.

If prices of inputs to the provider are not set at appropriate levels (social user cost), there can be demands for excess structure in hospitals. This has policy implications which are discussed in Section V.

II. **SOURCES: CONSUMER DEMAND**

In this thesis, the maintained hypothesis has been that consumers would want only effective care, hence increments in structure would be demanded only if they had a positive impact on outcomes. However, some authors have hypothesized (either directly or indirectly) that consumers are also concerned about structure levels in hospitals for their own sake. The Feldstein (96, 97) models of hospital price inflation and hospital quality are of this type.

These models employ assumptions about consumer preferences which make it impossible to observe "excess structure". Since consumers are hypothesized to be willing to pay for structure for its own sake, then consumer sovereignty dictates that all structure is "not excess".[2] "Excesses" will occur only if the prices facing consumers are in some way "inappropriate". It is impossible to refute Feldstein-type models, using the same assumptions. The evidence and theoretical discussion presented in this thesis is based on a different assumption about consumer preferences.

Feldstein's definition of quality simply equates resources used (structure) and quality via a constant elasticity relation discussed in Chapter Two. He then postulates a model which has quality in both the objective function of the hospital and the demand constraint.

According to Felstein (96, 97), hospitals with the number of beds fixed in the short run are constrained in their ability to maximize quality, by the production function for quality, and by the zero profit (or maximum loss) condition. Revenue is determined by demand which is a function of both quality and price. In a situation of excess demand (at current quality (resource-use) levels), hospitals can only eliminate the excess demand by increasing costs so that prices rise. These increased costs are now "quality" increases for which consumers are willing to pay. Hospital administrators are willing to increase resource use because they are short-run "quality maximizers."

Only in the long-run model does Feldstein permit quantity to vary. Increasing the number of beds decreases the equilibrium price and the corresponding quality level. A negatively sloped quality-quantity trade-off is developed. The long-run preferences of the hospital decision-maker are a function of quantity and quality and he chooses this preferred point of operation. This leads to the conclusion that "anything that limits the expansion of the stock of beds will increase the average cost per patient" (96, p. 1690). He also noted that under certain conditions, increasing the number of beds will decrease total expenditures on care, a result which contradicts most observations about expenditure and the number of beds.[3]

Feldstein comes to no conclusion about the relative quality levels in the hospital market. He is only concerned with determining some of the factors underlying hospital cost inflation. "Excess" structure cannot exist in this model, because consumers get what they are willing to pay for. His assumption that both consumer and producer perceive quality in the same way, i.e. resource use is of direct concern to patients and recognized as "quality", is a rather subtle shift that puts the responsibility for cost inflation onto the consumer.

Any problems in the hospital care market (i.e. "excess" resource use), by assumption, are the result of inappropriate prices facing consumers. The chief culprit (generating inappropriate prices) is increased insurance coverage. Feldstein noted that to control hospital cost inflation, "the government must either control hospital care or stop increasing hospital insurance" (96, p.1691) in the U.S.

Newhouse (222) uses a similar approach in his model of hospital behaviour. In his model, both costs and demand curves shift with quality changes. Because hospitals must satisfy a non-profit constraint, output quantity levels are set where the demand and cost curves for a given level of quality intersect. Changing the quality level and observing equilibrium quantity defines a quality-quantity trade-off. Hospital decision makers choose the point on the quantity-quality trade-off which maximizes their utility.

Newhouse claims that overall quality levels will be too high. He states "(i)n normative terms, the problem is that the decision-maker has picked a point on the quantity-quality trade-off curve which is optimal for him but not necessarily socially optimal" where social optimality is defined in a footnote as "the outcome observed in a market dominated by knowledgeable consumers" (p. 70). But, relative to what standard this quality level is too high, remains to be determined,[4] for in Newhouse's model, as stated, cost-minimization at each level of quality holds and consumers are sovereign (they are willing to pay for what they get).

Both these models and their predictions about the source of "excess" structure or quality demands are based on consumer sovereignty and the assumption that consumer and producer perceptions of quality are the same. Any structure provided beyond the point of effectiveness must be knowingly paid for by consumers. Although difficult to define since consumers are part of society, any deviations from a social optimum can be corrected by changing relative prices.

The alternative to models which allow the consumer to confuse inputs into their care with the outcome of that care (hospital care becomes a direct source of utility), is to consider the possibility that the providers of care are concerned with structure and may be able to impose their preferences and perceptions of quality on the market.

III. SOURCES: PROVIDERS

Before considering what form provider preferences may take, some investigation of the means by which they are able to influence market outcomes is necessary.

Physicians may be able to dominate consumers' decisions in their roles as agents. There are information differentials in the health care market. Consumers do not generally feel they possess sufficient information about health care and its impact on health status to make informed decisions. The response has been to delegate responsibility for making choices to the physician who possesses more information and is expected to act in the patient's best interest. This is described as the agency relationship. If it functioned perfectly[5] there would be no problems at all about whether efficacious care would be provided, because consumer tastes would be perfectly represented. But agents (physicians) are human too, and some of their preferences may interfere in the transmission of their clients' ideas, if some changes are relatively advantageous to the agent, or information is costly to procure.

In particular, if the agent will also supply the service required, there may be some conflict of interest. This means that consumer preferences may not be adequately reflected in the professional's actions and decisions made on the consumer's behalf (i.e. demand).

However, such effects are compounded by considerations of the important role that physicians play in the functioning of hospitals.

The major reason for the institutional significance of physicians is that physicians are vested with the legal authority to provide medical care. "The hospital itself cannot legally practice medicine, nor can it lawfully control the professional practices of its physicians. It is these circumstances which result in the relatively independent status of physicians." (Stevens (285), p. 232).

At one time paying patients of physicians were necessary to ensure the fiscal solvency of all hospitals in both Canada and the USA.

> To balance the cost of the care of such "charity" cases, well-paying patients were obviously desirable to the hospital. The physicians who could bring such well-paying patients into the hospital were obviously of great importance to its survival and, as might be expected, were dominant in setting hospital policy in spite of the fact that they were neither owners of the hospital nor committed to it by employment or capital investment. (Freidson (106), p. 112).

Although the economic dominance of physicians has been reduced by increased insurance coverage (particularly in Canada), the pattern was established.[6] The threat of withdrawal of services (or admissions) by large users is a threat that no hospital administrator can afford to ignore. This gives physicians, particularly those that have major affiliation to one hospital, a strong voice in determining policy and resource allocation. Physician influence manifests itself in the organizational structure of hospitals.

Physicians have self-governance. They are organized within the hospital, but not under the direct control of the administration. They are part of the hospital, but not of the operating hierarchy or management.

The organization of the medical staff, establishment of by-laws, control over quality (via tissue committees, credential committees) are all the responsibility of the medical staff.[7] Physicians usually are members of hospital management committees. The chief of staff[8] may be an unofficial hospital spokesperson. Some hospitals have administrators who are (or have been) physicians. Physicians may be on boards of trustees, as elected or appointed members.[9]

Physician advice will be sought after and given in matters which will affect the medical care given by a hospital. Thus, as Granfield (131) notes, physicians' lobbying can affect the capital stock of hospitals in the U.S. Harris (142) also discusses how and why physicians will exert pressure on boards and government for more beds and services.

Short run allocations of resources are also controlled by physicians to a great extent. Only physicians can order certain types of care. This care is part of the total package of treatment that a patient gets from the physician and hospital, but even though the physician is able to order the use of some inputs which affect his ability to earn income, he does not have to pay for these inputs.[10]

Pauly and Redisch (237) (or also in Pauly (235)) take the idea of hospitals serving physicians one step further. They hypothesize that physicians totally control hospitals, but they cannot say what will attract physicians to a certain hospital. The hospital becomes part of a vertically integrated firm.

In such models the hospital now functions as the producer of an intermediate good (hospital care) into the overall health care package provided to a patient by the physician. Normally a market exists in which hospitals would sell such intermediate goods and services to the physicians, but the institutions have not developed this way.[11] Instead physicians have a certain degree of control over the amount and type of inputs from the hospitals used in the care of their patients, but do not have to be in a market situation to do so. So, in a sense, the hospital exists to serve physicians.[12]

Lee (185) also comes to this conclusion. His model shows that hospitals will compete for physicians. Administrators in hospitals are hypothesized to maximize their utility which "is a function of the status of the hospitals" (185, p. 50) where "status" is a function of the "variety, quantity, and complexity of inputs available to the hospital" (p. 50). The status of the hospital also reflects the status of its physicians, leading to competition for physicians. Lee implies that such competition may lead to provision of excess inputs.[13]

One cannot ignore the fact that physicians gain by having access to the hospital, and as agency relationships are less than perfect, may not act in a manner which corresponds to that which informed consumers would prefer. Incomplete information and market imperfections do not guarantee correction of deviations from the consumer desired optimum. However, this does not preclude the possibility that physician determined solutions are socially optimal.

IV. PHYSICIAN PREFERENCES FOR STRUCTURE: DOCTORS AND THEIR WORKSHOPS

Understanding physician preferences may be the key to explaining the existence and persistence of excess structure.

The availability of hospital facilities (hospital-based capital and labour) may affect physician utility[14] indirectly by affecting their ability to maximize their utility. For example, more hospital-based capital and labour (including many new technologies), while perhaps[15] permitting the patient to recover more quickly and comfortably, may also substitute for physician labour input (time).[16] Substitutes act mainly upon the time constraint. Physician incomes, however, may not decline because physicians must supervise the use of hospital-based non-physician inputs.[17] In some ways structure complements physician inputs. In some systems, such as the American one, the availability of (and access to) such inputs may be used as rationale for increasing physician prices in the name of improved quality.[18]

Pauly and Redisch (237) use such complementarity in their model of the hospital as a physicians' cooperative but only in an extension of the model to imperfect cooperation.[19] They assume that hospitals practice average cost pricing so that a physician would order more inputs for "his" patient, and, in their model, be able to charge the patient more for the total package of care, but have his costs spread over all patients. The increase in the price of the package of care would exceed the increase in the cost of the hospital portion, so the physician would gain, as long as he is the only one doing this.[20]

The model with imperfect cooperation is set up so that structural measures must reflect quality to physicians and patients. But, quality changes are modelled to affect physician behaviour only through their effect on income (net revenues to physicians) or leisure. If physicians are also hypothesized to be concerned with the style of hospital care provided, then there is another reason for concern about the amount and type of inputs used.

It is possible to add hospital structure and quality considerations explicitly by expanding the list of maximands in the utility function. There are several ways of doing this.

Murphy and Satterthwaite (214) in their model of physician behaviour under different incentive systems, moved hospital structure from affecting the constraints on physician utility maximization to having a direct effect on utility, by adding a "practice style" variable to the utility function. This variable included hospital inputs (structure) as one of its elements. This argument may be broadened by consideration of the style of work, or the working environment which is hypothesized to be very important to the physician and any professional.

There is some discussion about the importance of "maintaining common medical practices", the "technological imperative in medicine" (Fuchs (111)), the "prestige" of a hospital affecting the utility of those that use it (Lee (185)), and the importance of "professional freedom and independence" (Tuohy and Wolfson (308)) to practice medicine in a manner which the physician feels is "best" for each of his patients, but such ideas are generally not easily quantified. It is necessary to relate these ideas to variables that increase the physician's utility. The hypothesis of this thesis is that these are structure type variables - i.e. inputs are what physicians look for.[21] If the "right" set of structure is there, the physician satisfies his desire to practice in his more favoured style. The effect of such concerns on medical practices, both inside and outside the hospital, should not be underestimated.

Since the physician's standards are imprinted during his training, it is likely that the style of care provided in the teaching hospital (high technology and investigative) will determine his goals.

> During their medical school and residency training, physicians are "imprinted" with what they understand to be the "best medical practice to which they try to conform throughout their careers. This can be a mixed blessing because it is closely related to what I have called the "technological imperative" - namely the desire of the physician to do everything he has been trained to do, regardless of the benefit-cost ratio. (Fuchs (111), p. 60).

He adds that "the young physician develops the view that the hospital is the place to practice medicine, a "repairshop" view of medical care that tends to crowd out the ideals of preserving and enhancing health in the community." (Fuchs (111), p. 90). This will affect the demands the physician places on the hospital system. The physician will pressure for the structure he feels is necessary.

Another possible reason for structure preference by physicians is rooted in the lack of information about the effectiveness of structure. Physicians are concerned about outcomes (i.e. the patient's health status is important to the physician). However, there may be some uncertainty about the impact of structure on outcomes, and the loss function is asymmetric. Not supplying or using the appropriate inputs may have serious consequences for the patient, while use of ineffective inputs has fewer or no observable consequences.[22] Thus to be on the safe side, excess structure may be demanded and used.

This approach is consistent with Freidson's (106) description of physician attitudes. "There is a tendency for the practitioner to take action for its own sake on the spurious assumption that doing something is better than doing nothing" (Freidson, p. 168). Freidson describes the physician as a fairly crude pragmatist, prone to rely on apparent results rather than theory, and "prone to tinker if he does not seem to be getting 'results' by conventional means" (p. 169). This tinkering may include the use of technology or structure without regard for efficacy.

These tendencies may result in physicians overlooking outcomes when suffering from the desire to "do" something. If that "something" involves the use of esoteric equipment which provides some satisfaction to the physician, it will be used. The technological imperative drives one not only to do something, but also to use the most recent technology to do it.

This approach is formalized in Harris's (142) definition of quality which is defined in terms of reduction of risk for both the patient and his physician.

> There is a putative set of scientific standards which serve to define the minimum acceptable level of each medical input. But exceeding the minimum is not the same as failing to satisfy it. How far it is exceeded has something to do with the "quality" of medical care the patient receives... The real problem is that in a decentralized regime of special cases, it may be operationally impossible for anyone but the patient's doctor to determine where these minimum cutoff points are. As a result, production must be organized <u>as if</u> every input received by the patient is potentially an <u>absolute</u> necessity. (p. 470)

It suffices to note that this definition of quality allows all structure to be accepted as "necessary" or "required" and as such, to enter directly the utility function of any physician concerned about his patient's welfare.[23] Harris (142) claims that patients also share this view, noting that "no patient... would want his doctor to be compelled to make repeated marginal decisions about the costs and benefits of an angiogram or unit of blood" (142, p. 473).[24]

"Excess" structure is somewhat difficult to define in this approach, but if information on lack of efficacy or effectiveness is available and is ignored, then we may define that structure as "excess". Systematic biases in the provision or acceptance of information or the choice of treatment (structure use) can then lead to situations of general "excess" structure.

As noted, structure may affect physician choices in several ways. Hospital-based capital and labour may modify the constraints on a physician's ability to maximize his utility. Or structure may enter the utility function directly as reflecting concern about the working environment, or because of basic uncertainty about the effectiveness of structure. Thus structure, which is an intermediate good for consumers, becomes a final product for providers.

The pricing system, if it were operating in the hospital system, might be able to take care of such discrepancies in consumer and producer perceptions of this value of structure. Hospitals provide capital and labour for physicians to use in the care of their patients at zero private monetary costs,[25] but the social cost of these resources is not zero.[26] Unless strong central control is exerted excess structure will be demanded, and may be supplied by administrators, themselves interested in structure (see (Lee (185), Newhouse (222), and Harris (142)).

V. IMPLICATIONS

Thus, there are many reasons why providers of hospital care, particularly physicians, should have preferences for structure apart from its effect on their patients' health status. For the provider, structure may be a final good, rather than an intermediate good. The provider may also, because of his unique position as the "gate-keeper" to hospital care and agent of the patient, be able to impose some of his preferences on the observed resource allocation. This may lead to the situation which was discussed in Chapter Four, of excess structure in hospitals. "Excess" is defined as that which does not have a positive impact on outcomes.

Whether this constitutes a non-optimal situation in the sense of resources being overallocated to hospitals depends on one's view of the social welfare function. In this thesis health status, and hence outcomes, has been deemed of concern to all individuals as consumers in society. In the most basic form, physicians and providers also share this view. Outcomes would enter directly into the social welfare function and the observed ineffective structure is "excess". The existence of this "excess" structure cannot be easily explained in such a world, unless there are informational gaps. If there are information gaps, increased investigation of the effectiveness of structure should result in a gradual reduction in the amount of "excess" structure observed.[27]

However, if one employs the more common formulation of social welfare in which welfare is some function of the utilities of all members of society, this conclusion may be modified. If the utility of an individual is simply a function of commodities consumed and resources supplied by the individual, any ineffective inputs will not be "excess" if the social opportunity cost of the "ineffective" resources was paid by whoever got the utility out of using them. In such a formulation consumers and providers may share concern over both outcomes and structure (inputs) but as long as inputs are paid for at their opportunity cost, there are no true excesses. Even if only providers are affected by the presence of some levels of structure, there will then be no misallocation or redistributive effects, as long as their incomes are adjusted by the value of these inputs. Under the present institutional structure of hospitals, no charge is made for inputs used by physicians. In a full insurance system, such as Canada's, patients do not directly pay for the inputs used in their care. Thus further misallocation, because of faulty price signals, is very possible if there is no strong central control.

However, the problem becomes more complex if the situation is such as that described in this chapter. For some members of society, structure is a final consumption good, not just an input or a constraint. Misallocations can occur if pricing systems are faulty, and will be compounded by imperfect agency relations. Thus, if providers value structure because they are concerned about inputs available and about the style of practice and they do not pay for these, serious misallocations may exist and persist unless:

1) provider preferences can be modified;
2) providers are forced to pay for such inputs;
3) consumer information and voice are increased so that provider dominance of decisions is reduced.

It is not the provider preferences *per se* with which one may be in disagreement, but the extent to which provider preferences dominate resource allocation decisions, combined with the possibility that providers do not have to pay for the structure that satisfies their preferences.

Thus physicians are likely to get what they want - more structure. Since the emphasis is on structure by itself, then it is possible that some of this structure is ineffective, particularly since there are few automatic review mechanisms for new technologies, current techniques or patterns of resource use. The empirical findings of Chapter Four are plausible in this context.

Implications arising from this situation are wide ranging. Models of hospital behaviour should be broadened to include the role and importance of physician preferences. Any policies to change "hospital" behaviour or that attempt to modify resource utilization patterns indirectly will have to be targetted at all groups in the hospitals - administrators, trustees, and doctors. Omission of consideration of physician roles in policy actions, will blunt the impact of policy changes.

If changes in the pattern of and trends in structure use are to be affected, attempts can be made to modify physician preferences so that costs, efficiacy, and delivery modes other than teaching hospital methods, will be of greater concern and acceptance. Since physician preferences are moulded in medical school, it is important that changes be made here in the emphasis and process of education.

Alternative policies would modify relative prices to make hospital use less remunerative. The fee[28] for a hospital visit could be reduced relative to preferred (more efficient) methods of treatment. Systems of charging physicians for the inputs they use in the hospital would require major changes in the institutional structure of the health care system, but could be effective.[29] This is a form of central control, which is also a possible solution.

FOOTNOTES FOR CHAPTER FIVE

1. Other groups in the hospital may not oppose physician choices or decisions because structure may also satisfy their desires. Administrators, nurses, other professional staff may also have preferences for structure.

2. In a Feldstein world outcomes measures and the concepts of efficacy and effectiveness are not important. Structure measures would automatically capture all that is important to consumers (and providers). "Excesses" only arise, if price signals are faulty.

3. However this result is possible. If beds are a constraint, only the more severely ill will be admitted which will lead to more resources for the average patient. Similarly, when bed space is at a premium average length of stay will fall, patient turnover will increase, but since there are more "first-day" (most expensive days of care) average cost per patient admitted may rise.

4. It seems that some sort of ethical observer (see Culliis and West (59)) has been imposed on a market which was assumed to be functioning successfully or that Newhouse feels that consumer preferenes are very different from those of hospital decision-makers.

5. Tuohy and Wolfson (308) discuss real world problems with agency relationships at some length.

6. In the US, physicians who can still bring in insured patients may be favoured. Non-insured patients often cannot pay!

7. Certain forms are required for accreditation and honoured by most hospitals. Licensing and accreditation requirements may also affect the existence of some committees. Both the American Joint Commission on Accreditation on Hospitals (see Manual (167)) and the Canadian Council on Hospital Accreditation (Guide (38)) provide detailed "advice" on how the medical staff shall be organized, its rights and responsibility, categories of staff, and some standardization of formal interaction between the medical staff, trustees and administrators. They include such standards as:
   Standard VIII - Medical Staff Appointment and Clinical Privileges:
   The governing body shall delegate to the medical staff the authority to evaluate the professional competence of its members and applicants for staff privileges; it shall hold the medical staff responsible for making recommendations to the governing body concerning initial staff appointments, re-appointments and

the assignment or curtailment of privileges ... and
Standard IX - Communication between Governing Body and Medical Staff:
The governing body must ensure that by-laws, rules and regulations pertaining to the medical staff are developed and adopted by the medical staff. They shall be subject to governing body approval, which shall not be reasonably withheld, and shall include an effective formal means for the medical staff to participate in the development of hospital policy, in so far as it affects patient care. (CCHA (38), p. 7-9).

8. The chief of staff may not be the same as the medical director who is a contractual physician in charge of medical care administration.

9. Both Canadian and American accreditation guides make explicit recommendations about the relationship of medical staff and governing boards.
The governing body should include a broad representation of the community served by the hospital, and its members should be selected for their ability to participate effectively in fulfilling the governing body's responsibilities. Physicians, including those who are members of the medical staff, where it is legally permissible, shall be eligible for membership on hospital governing bodies in the same manner as are other knowledgeable and effective individuals. (CCHA (38), p. 1)

10. Pauly (235) and Pauly and Redisch (237) would dispute this - they claim that demand is a function of the total price of care (hospital and medical) and that any inputs ordered (which increase the cost of hospital care) decreases the price that the physician is able to charge, since the total price is constant. The Pauly and Redisch (237) physician always orders the cost minimizing combination of hospital inputs except under situations of imperfect cooperation among physicians.

11. See Penchansky and Rosenthal (239) for some discussion of this.

12. An alternative would be for hospitals to hire physicians and sell the total care package themselves, but once again institutional constraints against the corporate practice of medicine do not (or have not) encouraged this.

13. Denton et al. (65) provide some evidence that supports Lee's (185) hypothesis that prestige maximization boils down to providing structure that will attract physicians. In 1967, Denton et al. (65) asked physicians to rate Cleveland hospitals for quality. The raters showed remarkable agreement. Their rankings were shown to be correlated with structure measures such as the number of residency programmes offered, number of

beds, and average length of stay. These three variables predicted well for other communities and raters. The results of this study indicate that physicians are impressed by certain factors such as bigness, number of facilities, and presence of training programmes (which in turn depend on the presence of some types of structure).

14. The maximand used in this discussion is utility which is hypothesized to be a function of income and leisure. Other hypotheses such as target incomes could be substituted without substantially altering the analysis.

15. Considering the reported inefficacious nature of some forms of treatment for some patients, the "perhaps" is very relevant, but there are also the concerns such as lowered anxiety levels and better diagnosis. See Russell (259).

16. Discussions of physician productivity in hospitals, such as those of Daniels (63), and Johnson (166), suggest that hospital services could be further reorganized to increase physician productivity by substituting hospital-based capital and labour for physician own time.

17. Computerized axial tomography (CAT-scanners) is commonly quoted as an example of a technology which is quite expensive to buy and operate, and perhaps over-utilized, but requires physicians to order, and interpret the results of studies. In the U.S. these scanners are so popular that some are being placed in physician offices, despite current prices of $700,000 per unit.

18. Prices may be increased for all services, not just those that use the technology, on the basis of improved overall levels of quality.

19. In the basic model, physicians who own the hospital, will practice cost-minimization in the hospital and act in aperfectly cooperative manner.

20. The imperfect cooperation described here only exists under the assumption of average cost pricing which enables individual physicians to spread their patients' costs to all patients. A question then may arise as to why such a pricing scheme would be used when itemized bills would eliminate this incentive. Recent experience in the US indicates that itemized bills are the norm rather than the exception. However, to support the Pauly-Redisch model, such changes should also lead to reduction in unnecessary hospital services, etc. This does not seem to have been observed. There may be some other explanation for this. Pauly (235) does not modify this assumption in later writings.

21. Harris (142) notes that increasing structure may also serve to resolve conflict within the hospital over resource allocations.

22. Iatrogenic illnesses and risks of some diagnostic techniques and treatments cannot be ignored, but may be considered to be minor relative to risks of no treatment.

23. Harris (142) notes that reduction of defensive margins may be used as arguments to increase structure in hospitals.

24. However, Harris does not attempt to relate this to "willingness to pay" for all these inputs (with or without insurance). He also seems to neglect the risks to the patient involved in some treatments such as angiograms!

25. It is interesting to note that in recent years physician incomes have not risen as quickly as those of other groups in the hospital system (non-professional staff, nurses). Is this a "social" payment for the structure available to them? Possibly, but this is most likely the result of increases in the number of physicians which results in pressure on income.

26. The problem in hospitals of zero-priced inputs may be likened to arguments about labour-managed firms in socialist economies (see Vanek (312)) where the ownership of capital is not vested in the workers who control its use. In the hospital system, physicians would be called usufruct owners (those who use the asset in production and control its use once it is in place). The province, or the board of trustees would be the basic owner who extracts the scarcity rent from the usufruct owner. Such a system will work only if the basic owner charges a rent to the usufruct owner which equals the opportunity cost of the assets in question. Zero prices are unlikely solutions. Since the captital used by physicians is not being appropriately priced, then distortions in its use are likely. (Some authors (e.g. Granfield (131)) claim that time costs are not zero.) To this should be added the common property problem of many physicians dipping into a common pool of hospital provided inputs, with subsequent problems of coordination and cooperation.

27. Or in an income transfer.

28. In Canada, in general, a follow-up hospital visit pays the physician less than a follow-up office visit where the physician must pay the expenses. It is interesting to note that the reverse is true in the United States. The average fee for a follow-up hospital visit is greater than an office visit (see <u>Profile of Medical Practice, 1979</u>, (114) tables 46 and 51).

29. Such changes would be expected to have an impact on physician behaviour, but the extent and direction is unknown. Choices other than the use of "excess" structure may be affected. There is some further discussion of this in Chapter Six.

Chapter Six

CONCLUSIONS AND POLICY IMPLICATIONS

I. SUMMARY

The evaluation of the quality of care provided by a health care system (or any subset of it) is a requisite for the assessment of the overall performance of the system. Policies which affect the health care system, either directly or indirectly, may also affect the quality of care provided. This impact must be critically examined in a consistent and appropriate manner.

This thesis is concerned with the way in which such assessments are made.

The basic assumption of this thesis is that consumers are concerned about health care as it affects their health status. Economic theory emphasizes the link between quality and that which affects the well-being of consumers. Consumers would desire, both for themselves and others, only care that is efficacious; and would assess quality in terms of outcomes. Structure and process measures are also used to evaluate quality, but are proxies for outcomes measures.

In many cases, the three approaches to quality evaluation have been assumed equivalent, and substitution has been freely undertaken. Consideration of the conditions in the health care market means that equivalence cannot be assumed on *a priori* grounds, but must be based on empirical evidence.

The first hypothesis tested is whether aggregate structure and outcomes measures are equivalent. This is tested using correlation analysis to assess the strength of the relation between the structure and outcomes measures developed in this thesis.

The outcomes measures used are based on death rates in the hospital. Any variation across outcomes measures is the result of using different adjustment factors to correct for factors beyond the hospital's control. These factors are proxies for case severity and vary in sophistication from average-length-of-stay to the use of measures of case complexity and age-sex factor scores. All the alternative outcomes measures are significantly correlated.

Outcomes measures are then compared to structure measures which are based on average inputs per case, or indices of or judgements about facilities and services. The results suggest that substitution of structure for outcomes measures will not yield the same results. The structure and outcomes measures (in all their alternative forms) are either uncorrelated, or have correlations of inappropriate sign. The first hypothesis is not supported.

The test of the second hypothesis further investigates these negative results. The second hypothesis is that these negative results are generated by the presence of excess structure in hospitals. Excess is defined as ineffective. This hypothesis was supported by the regression results which indicated that no matter what outcomes measures, what combination of structure variables, or what functional form was used, there was zero or negative marginal impact of structure on outcomes. Some ineffective structure is evident.

This result implies that there may be inefficiency in the production of outcomes (i.e. production is not on the production frontier) or that production of quality in hospitals is taking place on a flat portion of the production frontier.

The last hypothesis of the thesis states that these possible inefficiencies may be the result of the preferences of physicians. The arguments in Chapter Five lend support to the statement that providers' preferences may not be the same as those of consumers, and that providers may be able to impose their preferences on the hospital system.

II. POLICY IMPLICATIONS

Many policy implications arise from this thesis. They are divided into two types - those resulting from the empirical analysis, and those resulting from the theoretical discussion.

The policies proposed in this chapter and in Chapter Four are based on the assumption that excess structure does exist. However, immediate implementation is not suggested because of possible limitations in the analysis. Thus the policy recommendations are really two fold:

1) cautious consideration of implementation of some of the proposals, particularly those calling for increased monitoring of effectiveness; and

2) consideration of a moratorium on the provision of more

structure combined with increased research into the relationships between structure and outcomes. This research could be pursued at both the aggregate level (as in this thesis) and the disaggregate level (both clinical efficacy and economic efficiency studies on treatment modes).

Thus, the results in this thesis should provide a spur to increased research in the area of quality of care.

A. Implications of Empirical Results

The implications for policy which result from the empirical section of this thesis are generally based on the conclusion that measuring structure is not the same as measuring outcomes, and that the contribution of improved structure to outcomes is not positive.

The result that structure and outcomes assessments are not equivalent indicates that there may be shortcomings in current assessment techniques. A change in emphasis towards outcomes is indicated, particularly for procedures such as accreditation.[1]

Future development and testing of outcomes measures are indicated. Because of the relative weakness of current data sets re outcomes, more information is necessary. Policies to encourage the use of outcomes measures would have to include improvement in data.

Outcomes measures should be used to identify system weaknesses and failures, and as indicators of overall system quality. Improvement can result only if failures and weaknesses are investigated as to the structure and process (including physician quality) of the care that resulted in the failure. It is corrections to structure and process which will result in improved outcomes.[2]

Some of the empirical results should be considered on a more specific basis. In particular, the results with respect to structure variable SIZE (the size of the hospital), CASEX (costs per case), and teaching status have strong implications for both the theoretical and practical analysis of hospital performance. In all cases, assumptions about the positive relation between size, accreditation status, teaching status, and quality of care, seem to require more support than is available here. The results indicate that these variables are not strongly related to outcomes in the appropriate direction.

Policies of overall cost-containment which have incentives to encourage efficiency may not seriously compromise quality since there is some room to reduce structure. Showing, for example, that budgetary restraint reduces input levels only leads to the conclusion that quality has been compromised, if outcomes[3] have also been adversely affected. Similarly outcomes should be assessed when analyzing delivery modes for health care services.

Increasing overall resources allocated to the hospital sector would not be expected to improve overall outcomes, although reallocation of resources may have some effect. More research is suggested on this point, to determine the extent and type of oversupply of structure in the acute care sector.

The result that additions to structure have no or little effect on aggregate outcomes may be tied to technological change in hospitals and the form that it has taken. There is no doubt (see Thomas (297)) that the rate of improvement in life spans (reductions in mortality) has slowed in the last few decades. Current medical advances have not had the broad general impact of some advances in the 20's and 30's.

Today much more complex and expensive medical technology is used. Not all are ineffective, but the point is that the measured effect of new technology has been to increase structure greatly, with little effect on outcomes of the general hospital population. In the pursuit of "prestige" and the "technological imperative", technology may be desired by physicians without sufficient consideration of its usefulness and efficacy.

> If these applications of technology to the critically ill were the only aspects of hospital technology that aggrandized the cost of care, I suspect that we would not be dealing with the subject today ... the problem rather is the <u>institutionalization</u> of technology; that is, the development of conventions that call for broad applications of technology to an intensity and extent that the capacity to sustain the cost reaches the limits of toleration... Furthermore, to the extent that any new technology is not fully proven, it tends to be additive rather than substitutive, and pushes overall costs higher. (Rabkin and Melin (244), p. 240-1, emphasis in original).

As well, indications are that there should be more efficacy and cost-effectiveness analysis of current treatment modalities, future "technological advances", and overall levels of resource provision in hospitals.[4] This would improve the level of knowledge for providers, consumers, and government agencies. Increasing provider and consumer awareness of the concept of efficacy should have impact on long-run resource allocation decisions in the health care sector.

Efficiency in the hospital sector has not been proven. The forces that would normally ensure efficiency in production in the market are absent. Altruistic service motives are generally not thought to be sufficient to ensure adequate quality at lowest cost, so other constraints (regulatory) have been added. The Canadian system has been to impose general budgetary restraint rather than specific regulatory controls. Relative to the experience of American and some European hospital systems, this system has been successful. As well, Canadians have been more successful in controlling the spread of add-on technologies.

> Remarkably little attention has been given at the governmental level to the evaluation of particular forms of care. The general philosophy seems to have been to rely on individual physicians and hospitals to carry out the process of "technology assessment", as they respond to the overall constraints placed on their available funds, time and energy. (Evans (88), p. 2)

However, the neglect of the importance of efficacy is not justified. Monitoring of the effectiveness of the health care system should be continued.

To summarize, the empirical results give rise to general policy implications which call for increased investigation of the efficacy and efficiency of current delivery systems and the level of resource use in hospitals. Current efforts at cost containment in several Canadian provinces should provide some new observations, as for the first time, data will be available on the hospital system for a time period when there has been little or no growth in the amount of resources devoted to hospital care.

B. Implications of Theoretical Discussions

The theoretical discussions of Chapter Five serve to reinforce the conclusions and implications of the empirical work. The implications of the theoretical work tend to revolve around the role of physicians in determining resource allocation in hospitals.[5]

An underlying theme of this thesis is that consumers, if fully informed, would assess quality by measuring outcomes, and only efficacious care would be demanded. Given the assumption that consumers are concerned with outcomes, then the empirical observations are inconsistent with consumers having full information. Producer sovereignty seems to dominate health care markets. No proof of this proposition is established, but consideration of the institutional features of the health care market leads to the conclusion that the role of providers (especially physicians) is significant. Provider preferences may not coincide with those of consumers. Under some circumstances, the pricing system would be able to internalize these variations in perceptions of quality. Consumers would only pay for efficacious care and providers might be able to get a little more structure by giving up income. But, consumers do not, in general, possess sufficient information to make "fully informed" decisions about resource utilization and have delegated much of the decision-making to the providers. Because of imperfections in real-world agency relations, and failures in the pricing system, physicians may be able to impose their preferences on the hospital system. In hospitals, this has led to the over-provision of structure in order to satisfy the desires of providers, and reduced consideration of the efficacy and effectiveness of treatment modes.

Correction, if correction is deemed necessary, would be aided by the increased provision of information to the lay-population on the efficacy of new technology, the efficacy (and cost) of alternative treatment modes, and on outcomes (quality) of the available units of care. There is no theoretical reason why increased information about health and health care should not be made available to consumers.

However, some practical problems may be associated with the provision to consumers of increased information about possible weaknesses in the health care system. Psycho-social attitudes may play an important role in the decision to seek care and in the healing process itself. Increased information may erode some positive factors by increasing uncertainty about the care received or to be received.

As well, it is possible that there is a random and uncontrollable element in outcomes. Information about outcomes may unfairly penalize an institution suffering from the vagaries of fate.[6] As well, it is not clear that consumers wish such information or will respond to it.[7]

Increased competition has been proposed by some authors (Enthoven (84), Havighurst (144)) as another remedy. Strengthening market forces should bring consumer and producer preferences (and the observed results) into closer congruence. In the Canadian institutional framework the use of increased competitive forces which involve patient charges is unlikely to be introduced.

In the U.S., however, there is some scope for the use of competition among different modes of practice, and delivery of complete packages of care. There are already some alternatives in operation but there has been little analysis of quality differences, if any.

The results of the theoretical investigation lead to the conclusion that physicians like structure for a number of reasons. Structure in hospitals may enable them to increase their income (or leisure time) by substituting for physican supplied inputs (including office-based inputs) or by complementing physician inputs.

Structure enables physicians to satisfy their desires with respect to their working environment. It is hypothesized that the standards most physicians strive for in both their own performance and in the hospitals they work in, will be those of the teaching hospitals in which they trained.

> I think it's critical that we reorient medical education away from what appears to be a substantial attraction for the benefits of technology, and perhaps replace that with some healthy skepticism about technology. The problem we see from the regulation point of view is an attraction to technology that is highly uncritical. (Steven Weiner quoted in Egdahl and Gertman (79) p. 271).

One solution to such a "problem" is to reorient medical training so that physicians are made more aware of the economic impact of their decisions, of the existence of alternatives, and of the efficacy and effectiveness of treatment modes.

Policies could also be implemented which increase physician awareness of costs of various techniques. In Canada, there are no itemized bills as in the U.S.[8] to which the physician may refer in order to estimate the costs of what has been ordered. It is not certain that physicians are influenced by such information.

Another alternative is to charge physicians for some of the inputs (structure) they use. One of the institutional features which allows physicians to pursue "structure" is the fact that they do not pay directly for the resources to which they have access in hospitals. This gives rise to common property problems with resulting depletion consequences. Incentive problems also arise when the prices faced by providers do not equal the costs.

New pricing schemes include: 1) changing the relative prices of care provided in different settings to make hospital work, in general, less remunerative; 2) promotion of the use of salaried, hospital-based specialists;[9] 3) schemes which do not fully reimburse unproven technology and its application; 4) charging physicians for the hospital-based capital and labour they use.[10]

Charging physicians for the use of structure has considerable intuitive appeal, but such schemes are not simple to design within the context of full insurance, and imperfect information about the effectiveness of structure (there is considerable uncertainty about which elements of structure are ineffective). Some care must be taken in imposing charges on physicians so that patients are not penalized for the use of hospital-based inputs. Programs may apply to all physicians collectively (in which case the common-property problem (Pauly-Redisch non-cooperative behaviour) may arise) or may be designed to capture physician-generated "excess" structure.

Individual physicians could be charged (in part) for all hospital services used by their patients or just specified types of services (e.g. ancillary),[11] and restricted from passing on these charges to the patient directly.[12] Monitoring of outcomes would be required to ensure that incentives to reduce services used do not have a derogatory impact on outcomes. This may be combined with a regulatory scheme (also not easy to design) to ensure that patients are hospitalized as necessary.

An alternative is to charge physicians collectively for the structure they use in hospitals and allow physicians to allocate the charges among themselves. Physicians could be assessed all costs above a budget for specified services, or responsible for cost increases beyond some percentage. The physicians (or regulators) will have to devise some system for reducing common property incentives. A monitoring scheme based on outcomes would also be required here.

The major alternative to physician charges is stronger central budget control which rations the availability of all structure or just certain types, but this scheme also may be flawed because of lack of effectiveness information.

In summary, the theoretical discussion leads us to realize the need for further consideration of the role of physicians in the hospital, in both theories of hospital behavior and in policies which seek to modify hospital resource allocation decisions. These policies may take many forms, but are basically aimed at increasing a physician's awareness and/or responsibility for his economic impact. The source of demand for excess structure in hospitals is more likely to be the physician than the consumer.

Overall resource allocation levels may be too high, but efforts to reduce this or effect internal change are likely to be met with resistance. As noted in this thesis, physicians, administrators, and trustees all have interests in maintaining and even increasing the structure levels in hospitals. Any changes must target all the groups in the hospital to be effective.

The most effective changes which will impact long-run resource use in hospitals and the health care system will be those that attempt to change and redirect the preferences of physicians towards outcomes-based ideals. Only if physicians and others come to recognize and acknowledge the economic impact of their decisions will consumer oriented efficacious care be produced and utilized.

Outcomes and structure measures such as those proposed in this thesis will assist in monitoring the progress of such changes.

The relationship between quality (outcomes) and the structure in hospitals has been investigated at some length. There is evidence that the measured relationship is not positive. The conclusion that there is "excess" structure is dependent on the assertion that outcomes based on mortality rates are the relevant measures of quality. If the argument that structure is quality determining (separate from its impact on outcomes) is acceptable, the "excess" structure conclusion may be weakened on the basis that the defined measure of quality was too narrow. The admissibility of this argument depends on what is postulated as a social objective for the health care system. There are two general approaches: that of health promotion and that of consumer sovereignty.

Use of the health promotion approach implies that mortality reduction is the social objective of the health care system. Effectiveness becomes the operational criterion of system functioning. Measures such as those proposed in this thesis are required to assess the achievements of the system. The implications of this model include that, in the absence of self-correcting market forces and perfect information, stronger central regulatory control will be required (and accepted) to eliminate "excess" structure. "No-frills" hospitals would be the objective.[13]

Use of the consumer sovereignty (or consumer-producer sovereignty) model may imply that a broader definition of quality is required. One alternative is to broaden the range of outcomes to include health status other than death. The other is to admit that quality is not only outcomes, but also structure (amenities only, or structure in general). Since structure may be valued for its own sake, the measured "excess" structure is possibly socially acceptable. However, the current allocation is only a possible optimal solution; the limits to acceptability have yet to be defined.

Two arguments may be considered. The first assumes that consumers (and producers) are fully aware of the cost of structure provided beyond the effective level and have accepted those costs. Consumers agree with physician choices, even though the motives for demanding additional structure may be different. The acceptance of this argument leads to the general conclusion that no corrections are necessary.

However, consideration of the imperfections in the health care market, and insecurity about the degree of consumer knowledge of costs and effectiveness, makes this argument suspect. Consumers may have been dragged along by physicians and others. The best one can say is "we don't know." Further research into consumer tastes, willingness to pay (directly or indirectly), and the social cost of such structure and the form it takes is needed to define the limits of acceptabilty. Outcomes measures are still required to identify system failures and to assess the size of the gap between "effective" and "total" structure.

In theory, health promotion as a goal is acceptable, but as our society becomes more affluent, other features may be of concern. However the form that "quality of life" improvements take may not be the result of conscious decisions by consumers. In many cases the costs and benefits of such improvements are not generally known.

The only way to finalize the conclusion with respect to "excess" structure is to do more research into consumer tastes, the impact of market imperfections on the market, and on the definition of social objectives.

FOOTNOTES CHAPTER SIX

1. It should be noted that some steps in this direction are being pursued by CCHA. More emphasis is being placed on outcomes. However, there is still heavy reliance on structural features. See Woods (327).

2. This system underlies Brook et al.'s (31) approach and that of Williamson (324). These have been implemented in some American PSRO's (Professional Standards Review Organization). Unexpected or poor outcomes are identified and then these cases are further examined.

3. Evidence of a negative effect of minor cuts on quality should lead to a search for the one subset of "costs" that resulted in the decline of quality, and may result in a change in policy.

4. The control of the diffusion of technology is a key topic for both national and international discussions (see Egdahl and Gertman (79)). These discussions tend to centre on the lack of efficacy and effectiveness evaluation before new technologies are adopted.

5. A recent (Feb. 1981) (124) strike of physicians in Chicoutami was the result of a neurosurgeon's decision to leave the town because some equipment (notably a CAT scanner) was not available in town. Eventually, in the face of a general strike by 160 physicians, the government agreed to install one.

6. This point is worthy of more research. Do some hospitals consistently come out poorly?

7. Why do opted-out physicians still have patients (in areas where there are non-opted-out physicians)?

8. Actually the figures noted on the bill are charges not costs. There is considerable cross subsidization of services in American hospitals so that the price (charge) may not equal the cost.

9. Hospital-based physicans may also like structure and demand it. It may also affect their leisure time. However some of the incentives inherent in the fee-for-service system of payment are removed. This scheme only works to reduce demand for more structure if future incomes are reduced by structure purchases.

10. The use of free hospital capital may increase the productivity of the physician, and in free market situations to capitalize on this free capital by charging higher prices for his services. (This, in turn, leads to pressures for more structure.) In Canada, the hospital capital is provided by the community (society). In some situations (particularly Ontario) it has been noted that opting out is concentrated in the surgical specialties (including anaethesiology) which require hospitals in which to do their work, and among physicians with teaching appointments at medical schools. This implies that those physicians who are most dependent on hospital structure, are also those who are able to extra-bill because of their access to it! Pressures to increase structure in hospitals might be reduced if those physicians (particularly medical educators who are often models for their students) were prohibited from extra-billing. Such situations are the logical place to start policies to reduce extra-billing over-all, even though other factors may influence the decision and ability to opt-out.

11. Another alternative would be to charge for structure use over and above the average "profile" of structure use on a diagnosis-specific basis. This scheme is very information-intensive.

12. It would be expected that pressures would be placed on insurers to increase fees so that physician incomes would not fall.

13. There is no reason why some consumers would not be allowed to purchase a few "frills" at cost in this system.

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