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The Value of Freedom

The right to professional freedom is a fragile value.

The independence and liberty offered by the medical profession was probably one of the values which attracted many of us to this way of life in our younger years. Where else could you have a wide choice of different specialties, make a good living, choose where to live, be respected and, in general, maintain options very valuable and hard to find in other occupations. On top of that, you have the rare privilege and opportunity to help other people.

Unfortunately, the climate surrounding the privileges and rights of physicians in the last 5 to 10 years has put these opportunities in jeopardy. The changes possible in the next decade or sooner are even more worrisome. For example, the *Medical Post* of January 14, 1992 states "concern is growing that changed licence portability requirements for a few physicians could restrict mobility for at least 10,000 already licensed doctors".

This infringement on the right to employment mobility is sadly being inflicted by the profession on the profession and perhaps, shows a lack of respect for our own basic rights that others may copy. Our incomes, as an example, are no longer under our control and, for some of the profession, may already (eventually) be completely controlled. The need and desire to place on salary increasing numbers of doctors is recognized but is also disturbing. Certainly ,the respect shown for our incomes did not lead us to feel a protected part of society as we were singled out by the General Services Tax recently in a very unfair manner.

In British Columbia, with the imposition of "billing numbers", we saw the right to work and employment mobility severely limited. The fact that the Supreme Court found the B.C. process unacceptable does not make it impossible, in some other guise. With government ultimately paying the bills and much of medical service being difficult to evaluate for effectiveness, it may seem to funding agencies that there is no limit to the amount physicians might bill the system, if numbers of doctors are not controlled. Without normal market mechanisms, long since gone, this must be considered true. The trick is to limit, with the minimum use of coercion and loss of freedom, the numbers of physicians and amounts of money paid out in a sensible way. Our concerns for our freedom will need to be tempered by reality. (How much freedom do any of the thousands of unemployed physicians in Europe now have?).

Basic freedoms of physicians were ignored in the preliminary report of the Task Force on Sexual Abuse in Ontario. Ordinary protection of reputation and right to be considered innocent until proven guilty were put in question. The final report was improved only after further consideration and great pressure. In this province we have seen physicians put on trial by public opinion, and the media, for suggested offences. In many cases, little consideration was given to the doctors' basic rights.

Often these threats to our freedoms in the medical profession come from well meaning people with a desire to make right an imperfect world.

F.A. Hayek in his book, *The Constitution of Liberty* states in his introduction "Ambition, impatience and hurry are often admirable in individuals, but they are pernicious if they guide the power of coercion and if improvement depends on those who, when authority is conferred on them, assume that in their authority lies superior wisdom and thus the right to impose their beliefs on others". Hayek goes on to discuss many concepts which we might do well to remember as the face of our health care system changes. Most of us realize, with the institution of Medicare and again with the *Canada Health Act,* that some element of coercion had entered medicine. While such state coercion on occasion cannot be avoided, it certainly should be kept to a minimum. Hayek suggests that government must allow the individual to "determine his own sphere by relying on rules which tell him what the government will do in different types of situations".

In these times of change, we have very few rules that allow us to choose a path free of coercion. Those students now in the system see the rules changing before their eyes and must wonder about the control they might have over their lives in the future. Hayek also quotes the four rights, conferred upon slaves once freed: First, "legal status as a protected member of the community":. Second, "immunity from arbitrary arrest":. Third, "right to work at whatever he desires to do", and fourth "the right to movement according to his own choice".

These "essential conditions of freedom: certainly seem fragile when seen from present day physician's perspective. The Barer-Stoddard report, in attempt to solve the funding crisis in education and research, has recommended "block" funding for this segment of our system. With more funding control one must remember that more control is given to government. Do we really wish more government control of teaching research and effectively our tertiary care system? The Barer-Stoddard recommendation was that "the funding of academic medical centers needs to be restructured to link it as explicitly as possible to academic goals ...". With complete funding control, it seems that complete control of "academic" goals may be achieved by government. The recent article in the *Canadian Medical Association Journal* on the Royal Society Symposium on Constraints to Freedom of Scholarship and Science should make academics and researchers pause before giving away any more control to government. In fact, the whole profession should take nothing for granted when considering the short distance between freedom adn its alternatives. □

J.F.O'C.

The Family Doc Who Delivers Babies . . . An Endangered Species?

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A random sample of 126 Nova Scotia primary care physicians responded to a mailed questionnaire examining various factors which may influence their choice of obstetric practice. Fewer physicians are including obstetrics in their practice. Forty-four percent of respondents were currently not delivering babies. The size of their community of practice did not seem to alter this percentage nor the general make-up regarding obstetrics. Of those currently delivering babies, just over one-half have considered stopping. Interference with lifestyle and financial concerns were the deterrents perceived as most important. Concerns regarding inadequate obstetric training and lack of interest were cited as least important. The results are discussed in light of other Canadian and American studies, and strategies suggested to hopefully slow the ever-increasing attrition rate of family doctors from obstetrics.

The practice of obstetrics by primary care physicians is an issue of current concern. Ideally, maternity care is part of the family-centred nature of family practice. However, family physicians in North America appear to be withdrawing from obstetric care at an alarmingly rapid rate.[1-6] This is of particular concern for women in rural and medically under-served areas, where access to obstetric care may be less than optimal. This issue holds particular relevance for Nova Scotia and the other Atlantic provinces of Canada where a relatively large proportion of the population resides outside the urban community.

Various factors have been implicated as contributing to the ever-narrowing spectrum of primary obstetric care. These include rising malpractice insurance premiums, fear of litigation, interference with lifestyle and office practice, and the rapidly changing technological aspects of modern obstetrics.[1-3,5] In the United States, studies have consistently shown malpractice insurance premiums to be the major deterring factor.[2-3]

Fewer studies have been done in Canada; however, Klein and his colleagues related the decreasing participation of family physicians in obstetric care, evident mainly in Ontario and Quebec, to the ratio of obstetricians to the population.[6] They consequently found physicians in smaller communities more likely to practise obstetrics than those in larger urban centres. Other studies have also found this to be true.[1,3] In a survey of

1338 Ontario primary care physicians, interference with personal and family life was found to be the most frequently cited important issue for giving up obstetric practice.[1] Physicians who had never practised obstetrics cited inadequate training and lack of interest as their chief reasons.

In a study comparing family physicians in Ontario and the United States, Kruse and Wesley noted some important differences.[3] The Canadian physicians were significantly more likely to cite interference with lifestyle and, to a lesser degree, office schedules as important deterrents. They also identified the low financial incentive to practise obstetrics more frequently than did their United States counterparts. The American physicians, on the other hand, again placed more emphasis on malpractice issues as a reason for not practising obstetrics.

The purpose of this study is to describe characteristics of primary care physicians in Nova Scotia who practise obstetrics and to determine which factors they perceive as important in influencing patterns of obstetric practice. Perhaps this information may contribute to reducing attrition from obstetric practice by family doctors.

METHODS

A sample of 177 general practitioners/family physicians was randomly selected from The Medical Register of Nova Scotia (both members and non-members of the College of Family Physicians of Canada) and were surveyed with mailed questionnaires. A single mailing was

*This manuscript is the result of a Research Project completed in 1991, during the author's second year as a Family Medicine Resident at Dalhousie Medical School, Halifax, N.S.

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done in early March 1991. No subsequent mailings were required as a high response rate was achieved (72%).

Information obtained by the questionnaire included: age, sex, years in practice, size of community of practice, type of practice, completion of a family medicine residency, distance to nearest obstetrician, and number of babies delivered over the past 12 months. In addition, physicians were asked to rate on a 5-point Likert scale their perceived level of preparation to deliver babies at the end of their training. On this scale, one represented "totally unprepared" and five represented "very well prepared".

Physicians who had never practised obstetrics, those who had discontinued obstetric practice, and those who had ever considered discontinuing obstetric practice were identified. Those who had discontinued obstetric practice indicated if they had done so in the past year. These three groups were then asked to rate the importance of the following possible influencing factors on their choice of obstetric status: 1) lack of interest; 2) inadequate obstetric training; 3) interference with office practice; 4) inadequate obstetric volume; 5) malpractice insurance premiums; 6) difficulty keeping up with advances; 7) interference with lifestyle; 8) low financial incentive; 9) fear of lawsuits; 10) inadequate obstetric backup; and 11) other. The physicians again rated the importance of each factor on a 5-point Likert scale, with one indicating the factor was "not at all important" and five indicating the factor was "very important".

RESULTS

One physician from the original sample could not be reached by the mailing, leaving an effective sample size of 176. A total of 126 physicians responded, for a response rate of 72%. Nine of the returned questionnaires were excluded because they were improperly or incompletely filled out, or the physician was no longer involved in primary care practice. This left 117 responses upon which the results were based.

The demographic and practice characteristics of the respondents are shown in Table I. Forty-four percent of physicians reported delivering no babies in the past 12 month period. Of those delivering babies, 9% delivered fewer than 10 babies, 45% delivered 10 to 24 babies, 32% delivered 25 to 50 babies, and 14% delivered more than 50 babies in the 12 month period prior to the survey.

The current status of obstetric practice by responding physicians is summarized in Table II. A high percentage (88%) of respondents have at some time included obstetrics in their practice. Sixty-five of the total sample of 117 (56%) were currently delivering babies at the time of the survey, with just over one-half of them having considered stopping the obstetric portion of their practice. Of the 44% who were not delivering babies at the time of the survey, 14 (12%) had never delivered babies and 38 (32%) had stopped delivering babies.

In general, the physicians in this sample felt well prepared to practise obstetrics at the end of their training. Forty percent of the physicians indicated they felt well prepared by checking 4, and 20% indicated they felt very well prepared by checking 5 on the 5-point Likert scale described earlier. Only 12% indicated a low level of preparation by checking 1 or 2. The overall mean perceived level of preparation was 3.84. Those who had never practised obstetrics had the lowest perceived level of preparation, with a mean score of 3.20. Those who continue to deliver babies scored higher with a mean score of 3.82. Those who had discontinued obstetric practice had the highest perceived level of preparation with a mean score of 4.00.

TABLE I

COMPARISON OF DEMOGRAPHIC AND PRACTICE CHARACTERISTICS (N=117)

Characteristics	Percent	Characteristics	Percent
Age		**Type of Practice**	
25 - 34	28	Solo	38
35 - 44	39	One partner	16
45 - 54	15	Group of 3-5	23
55 - 64	10	Group of >5	20
≥65	8	Other	3
Sex		**Family Medicine Residency**	
Female	28	No	90
Male	72	Yes	10
Years in Practice		**Distance to Nearest Obstetrician**	
<5	15	In community	58
5-9	22	<50 kms	27
10-19	40	50-99 kms	8
20-29	10	100-150 kms	6
≥30	13	>150 kms	1
Community Size		**Babies Delivered in Past 12 Months**	
<5,000	25	None	44
5,000-14,999	26	<10	5
15,000-24,999	6	10-24	25
25,000-50,000	9	25-50	18
>50,000	35	>50	8

TABLE II

CURRENT STATUS OF OBSTETRIC PRACTICE

Status	Number	(%)
Total sample	117	
Practised obstetrics at some time	103	(88)
Currently delivering babies	65	(56)
Have considered stopping	33	
Have not considered stopping	32	
Not currently delivering babies	52	(44)
Never delivered babies	14	
Stopped delivering babies	38	
Active obstetric practice 12 months prior to receiving questionnaire	71	
Stopped delivering babies in past 12 months	6	

Table III shows the respondents' choices concerning obstetric practice as related to the population of their practice community. When comparing the smaller communities with populations less than 15,000 to the larger communities with populations greater than 50,000, the make up is remarkably similar. The vast majority of respondents from communities ranging in population from 15,000 to 50,000 were currently practising obstetrics. However, these physicians represented only a small proportion (15%) of all respondents.

The relative importance of the factors that may influence primary care physicians to never practise obstetrics, to discontinue, or to consider discontinuation of obstetric practice is shown in Table IV. Four physicians were excluded from the group who had never practised obstetrics for these calculations, since they were military physicians. Six physicians were excluded from the group who had discontinued obstetrics as well, since they indicated only a single reason for discontinuation (ie "too old", "health reasons", "obstetrics unit closed").

TABLE III

CHOICE OF OBSTETRICAL PRACTICE BY POPULATION OF COMMUNITY

	Less than 5,000	5,000 - 14,999	15,000 - 24,999	25,000 - 50,000	More than 50,000
Respondents in community who have never practised OB (n = 14)	21%	7%	0%	0%	15%
Respondents in community who have discontinued OB practice (n = 38)	34%	40%	14%	0%	37%
Respondents in community who practise OB but have considered stopping (n = 33)	17%	30%	29%	80%	22%
Respondents in community who practise OB and have not considered stopping (n = 32)	28%	23%	57%	20%	27%

Sixty-four percent of the youngest physicians (25-34 years) were currently delivering babies. This increases slightly to 67% for physicians aged 35 to 44. One-half (50%) of physicians aged 45 to 54 and one-third (33%) of those aged 55 to 64, maintained an active obstetric practice. One of 9 (11%) physicians aged 65 or older was still delivering babies.

A comparison of the relative importance of the various factors of the physicians who had never delivered babies, those who had stopped delivering babies, and those who were currently delivering babies but had considered stopping is shown in Table V. Interference with lifestyle was cited as being most important by all three groups. Those who had never practised obstetrics placed less emphasis on the other factors felt to be important by the

TABLE IV

FACTORS INFLUENCING THE DECISION TO NEVER PRACTISE OBSTETRICS, TO DISCONTINUE OR TO CONSIDER DISCONTINUATION OF OBSTETRIC PRACTICE (N = 75)

Factor	Mean Score *	Percent Citing Factor Very Important †
Interference with lifestyle	3.99	48
Malpractice insurance premiums	3.24	19
Low financial incentive	3.16	28
Fear of lawsuits	2.99	20
Inadequate obstetric volume	2.69	17
Interference with office practice	2.61	8
Difficulty keeping up with advances	2.44	3
Inadequate obstetric backup	2.19	12
Inadequate obstetric training	1.80	4
Lack of interest	1.68	5

* 1 - Not at all important; 5 - very important
† A factor was considered "very important" if 5 was indicated

other two groups. However, in comparison to the other two groups, they placed more emphasis on the factors generally viewed as less important, such as inadequate obstetric backup, inadequate obstetric training, and lack of interest. Physicians who were currently delivering babies but had considered stopping placed more emphasis, on average, on the top five important factors. Interestingly enough, they cited low financial incentive as the second most important factor, after interference with lifestyle, for considering discontinuation of obstetric practice.

TABLE V

COMPARISON OF FACTORS BY STATUS OF OBSTETRIC PRACTICE (MEAN SCORES, RANGE 1 TO 5)

Factor	A	B	C
Interference with lifestyle	4.20	3.50	4.39
Malpractice insurance premiums	3.00	3.09	3.45
Low financial incentive	2.20	3.09	3.52
Fear of lawsuits	2.60	2.75	3.33
Inadequate obstetric volume	2.50	2.63	2.82
Interference with office practice	2.90	2.44	2.70
Difficultly keeping up with advances	2.20	2.41	2.55
Inadequate obstetric backup	2.60	2.03	2.21
Inadequate obstetric training	2.80	1.69	1.61
Lack of interest	2.10	1.81	1.42

A - Never delivered babies (N = 10)
B - Discontinued obstetric practice (N = 32)
C - Currently delivering babies but have considered stopping (N = 33)

DISCUSSION

This study demonstrates some of the patterns in obstetric care provided by primary care physicians in Nova Scotia, and the perceived factors that influence these choices. As noted in previous studies in both Canada and the United States,the most dramatic change is a decreasing number of physicians who choose to offer obstetric services to their patients.[1-6] Unfortunately, no historical data are available concerning Nova Scotia physicians who practise obstetrics but, in 1982, the Canadian Medical Association determined that 56.5% of Canadian family physicians practised intranatal obstetrics.[7] Bain *et al* found that 40% of primary care physicians in Ontario were practising obstetrics in 1987.[1] This study shows, however, that a much higher proportion of Nova Scotia physicians (56%) were currently practising obstetrics, a percentage very similar to that determined by the CMA in 1982. This relatively high percentage of physicians in Nova Scotia still practising obstetrics is possibly related to the high proportion who have practised obstetrics at some time. The minority (12%) of physicians in this study have never practised obstetrics. This group of physicians is markedly smaller than those of other studies where percentages have ranged from 29 to 37%.[1-3]

However, the news is not all good. Nova Scotia physicians have a higher discontinuation rate of obstetrics (32%) as compared with other populations where reported values range from 20 to 31%.[1-3] Both recent American and Canadian studies have found that roughly one-half of physicians currently practising obstetrics have considered giving it up.[1,3] The results of this study concur with this finding.

As fewer primary care physicians offer obstetric services to their patients, there will be reduced access to adequate obstetric care, particularly for those living in rural areas or who are indigent. It may have been felt that this was not a major concern since previous studies have shown that physicians in smaller communities are more likely to practise obstetrics than those in larger communities where obstetricians are available.[1,3,6] However, the results of this study, as shown in Table III, do not support this finding and are somewhat disturbing. It shows that physicians in the smaller communities of Nova Scotia are just as likely not to practise obstetrics as those in the larger urban communities. Obviously, various factors must be at work to deter small town doctors from delivering babies. One concerned physician from a small community commented, "Very timely study. In our community when I started practice, there were 9 general practitioners offering obstetric care. Soon there will be only 2 for approximately 200 deliveries per year."

An examination of the differences in practice patterns among the age groups shows that, in general with increasing age, fewer physicians maintain an active obstetric practice. This likely reflects the commonly perceived strenuous nature of obstetrics in various aspects of the physician's life and practice. Bain *et al.* also found that physicians who ceased to practise obstetrics were significantly more likely to be the older respondents.[1] One might therefore expect that the highest proportion of physicians practising obstetrics would be the youngest. This, however, does not appear to be the case. In this sample, the age group with the highest percentage (67%) of physicians practising obstetrics was 35 to 44 years. The youngest physicians (25 to 34 years) lagged slightly behind at 64%. It is a disturbing fact that among female family physicians, regardless of age, the proportion of those choosing not to practise obstetrics is 50%, and has remained essentially unchanged. The younger male physicians, in nearly equal proportions, have also chosen not to practise obstetrics.[1] It would certainly appear that there will be further declines in the number of physicians practising obstetrics in the future.

Tables IV and V summarize the reasons indicated by various subgroups of respondents for never practising obstetrics, for discontinuing, or for having considered discontinuing obstetrics. The primary reason given by all subgroups is interference with lifestyle. This factor domi-

nated all others and has been found to be the main influencing factor in other Canadian studies as well.[1,3] Financial losses expressed in terms of malpractice insurance premiums and low financial incentive were the next most important factors in influencing patterns of obstetric practice.

Inadequate obstetric backup and training were factors cited as being relatively unimportant for those who had discontinued or considered discontinuing obstetric practice. These factors were relatively more important for those who had never practised obstetrics. However, interference with lifestyle and financial concerns continued to be viewed as most important. It has been suggested that further exposure to obstetrics during medical training might serve to bolster the number of primary care physicians currently delivering babies. However, this study and others show that concerns regarding adequacy of training and obstetric competence play only a very minor role in the physician's decision regarding obstetric practice.[1,3] This is further supported by the finding that the overall perceived level of preparation at the end of medical training was relatively high (3.84 out of 5). In fact, those who had stopped delivering babies had the highest perceived level of preparation (4.0). One must take into account, however, that the retrospective nature of this survey may have influenced respondents' perceived competence to deliver babies at the end of their training. Those who have never practised obstetrics may recall this factor as an important reason for not doing so, while others who did go on to practise obstetrics may have felt inadequate initially, but gained competence with years of practice.

The interference with lifestyle issue deserves paramount consideration. It is well known that younger physicians are placing more and more emphasis on lifestyle and so this issue is likely to become of even greater concern. One study showed that 81% of Ontario physicians took their own obstetric calls, even though they were otherwise signed out.[1] As noted in their study, this is not surprising given the close relationship that is fostered over a 9-month period. However, if more physicians are to practise obstetrics, better on-call arrangements have to be made to allow for more personal free time and time with family and friends. Other strategies which might be considered to reduce interference with lifestyle include studying the practices and time management of physicians who have never thought of discontinuing obstetrics, and the future possibility of sharing some of the responsibility with a well-trained midwife.[1] This needs to be further explored.

Financial concerns are the other factors leading physicians away from obstetrics. The annual malpractice fee for primary care physicians who practise obstetrics in Canada is $2796, compared with $1284 for those who choose not to deliver babies. These fees are drastically higher in the United States. In addition, obstetric practice in Canada does not appear to be financially attractive. Some relevant comments from respondents follow. "The fee schedule is grossly inadequate for the degree of risk/responsibility involved and the likelihood of litigation. Malpractice premiums are so high that one has to do approximately seven deliveries per year just to cover the extra cost of premiums ... doing deliveries, especially inductions, requires cancelling office appointments — sometimes for the whole day." "Obstetrics is not a well paid part of my practice. Despite this, I enjoy doing obstetrics and feel it is important I continue to offer this service to my patients. Most of them would be very unhappy if obstetric services were centralized to a regional centre." It is obvious that if primary care physicians are to be encouraged to be active in obstetrics, malpractice insurance reform and a more suitable and adequate fee-for-service mechanism must be pursued.

Various studies have suggested that the outcomes for low risk obstetric patients managed by family physicians in their local hospitals are equal to or better than the outcomes of similar patients managed in referral settings.[8-10] A large part of this has to do with a non-interventionalist approach, recognizing birth as a life event rather than a medical procedure. A European author eloquently wrote, "Childbirth in itself is a natural phenomenon. The large majority of women need no interference whatsoever - only close observation, moral support, and protection against meddling. A healthy woman who delivers spontaneously performs a job that cannot be improved upon." (GJ Kloosterman, unpublished report)

SUMMARY

Fewer primary care physicians in Nova Scotia are including obstetrics in their practice and an alarmingly large portion have discontinued such practice. The major factors responsible for the trend away from obstetrics are interference with lifestyle and financial concerns. These factors must be addressed to prevent inadequate access to obstetric care, especially for those in rural and medically under-served areas. Steps will need to be taken to prevent the family doctor who delivers babies from becoming an endangered species. As one respondent states, "I intend continuing obstetric practice as I feel it is an integral part of family practice. There is something very satisfying about monitoring progress and development of children you have actually delivered." The final comment goes to Klein who entitled an editorial looking at the role of the family physician in obstetrics: "Obstetrics is too important to be left to the obstetricians."[11] □

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than to kno' much that ain't so."

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I was reflecting on the physician as a patient just the other day. A physician friend had given birth, and I called with congratulations. As is typical in such conversations, the subject of analgesics commonly given in labour came up. "Just think!" she wailed, "All the women I've given this stuff to, expecting it to relieve their pain, and wondering why they still made all those distressing noises. Nancy, it just does not work, and all they kept telling me to do in there, while I was in agony, was to relax!" "Aha," I thought, "Welcome to life at the pointy end of the needle. You, my dear, will never do a delivery in quite the same way again."

There is a common feeling that pregnant nurses and doctors make the worst patients, and I have certainly done nothing to refute this. I would hate to have me as a patient, and it is a tribute to my long-suffering family doctor that she has cheerfully endured me in this state twice now. The instant the pregnancy test stick turns colour I, who am otherwise pretty good in this regard, turn into a ranting hypochondriac. And there's nothing worse than a hypochondriac who does in fact have some genuine medical problems, armed with knowledge and a lot of long, sonorous Latin phraseology.

One of the problems, you see, is that while everyone knows about deformed babies, stillbirths, etcetera; if you've never actually been involved with one it is easier to keep dark fears nebulous. Every woman wonders "Is my baby normal?", but the pregnant doctor gets to remember the grim reality of the ones who were not.

In my internship I had the sad task of being present at a small regional hospital when a primipara came in at 39 weeks gestation because her baby wasn't moving. Some time in the night before, for no apparent reason, her child had died. Labour was induced, and as the obstetrician was called away, I delivered the babe. The day I first found out I was pregnant, that lady's anguished face came to me unbidden, and she and her baby haunted my dreams for months.

But pregnancy as a doctor has an up side too. Forget all the degrees and the experience, only now did my pregnant patients feel that I was finally gaining credibility. And the ones who had had their babies, depending on their personalities, either tried to assure me that delivery wasn't all that bad, or got to enjoy proclaiming loudly "Just you wait ..." with evident relish. The best part, however, was often the look on their husbands' faces when "the Doctor" waddled in to deliver their wives. I got to reflect during a C-section one night on how much medicine was changing – not only was my patient pregnant, so was her doctor, the obstetrical resident, and the nurse. There were these three bulging females trying to squeeze close enough to the patient to deliver her baby, while the father and the (male) anesthetist complained loudly about being outnumbered in such a "big" way. They were somewhat mollified that the child being delivered turned out to be a boy – but only somewhat.

And then there was the day in the office when my daughter, who in utero seemed normally to be trying out for place kicker on a football team, decided not to move. At all. All day. Between patients I'd frantically drink sweetened juice, lie down, and prod at my swollen belly. Should I call my doctor? Should I go to the Grace and risk being laughed at? Should I call an ambulance? Should I get a scalpel and start sawing a Pfannenstiel's on myself? Finally I could stand it no longer and wandered down the hall to my associate's office. Could he, I asked, with a deceptive attempt at casualness, which I'm sure was completely transparent, just have a quick listen to the baby as she'd been awfully quiet? One look at my doubtless white face and I was being urged into a chair while my blood pressure was checked. And the minute I lay down on the table – boom, biff, bat, the baby started to kick. Ah yes, physician's nerves was the diagnosis. Stubborn baby was my rather embarrassed opinion.

The really interesting part about being a pregnant physician comes, however, at delivery. Two major inhibiting factors are present – the fact that you're a doctor and are assumed to know all about this stuff (which somehow means that it isn't supposed to hurt as much); and the certain knowledge that you'll have to come back and work with all these people at a later date. This means that you are not allowed to call them nasty names when, at the very peak of a contraction, they tell you to relax.

I had the joyous experience in my first delivery of ending up on a magnesium sulphate drip with a large OP baby and a recalcitrant cervix. People with whom I would have felt far happier having a cup of coffee with down in the cafeteria (you know, the Grace cafeteria that always closes three minutes before your patient delivers) were arriving, performing the most intimate of examinations, looking sorrowful, and departing. Muttered consultations between nurses in the corners, which presumably I was unable to comprehend, having made the miraculous transformation from physician to patient, were getting more and more threatening. This did wonderful things for my blood pressure. My husband, who is involved in the teaching of medical students, was getting alarmed as former pupils were seen shaking their heads. I'm sure he

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was reviewing how well they had done in their courses, though he professes a convenient amnesia in regards to all these events.

Paediatrics was called to the delivery, and I vaguely remember seeing a haze of what seemed like three dozen people, all of whom I usually meet on a more vertical basis, and wondering how I would ever attain a professional manner with them again. (Actually this turned out to be easier than you might think). Fortunately our daughter was healthy, a credit to all who participated in her delivery.

Then, post-delivery, comes dealing with "the Floor". "The Floor" had called me concerning a patient at two in the morning, about two weeks before my own due date. "You took your time answering" was the opening sentence; and I had taken great pleasure expostulating in detail and with some acidity, about the difficulties of climbing out of a low bed and navigating a dark hall to answer a ringing phone at two in the morning while 38 weeks pregnant. Then I asked who was calling. Guess which floor I ended up on after my delivery?

Now, just because you are a doctor does not automatically mean that you know where the showers are, how to have a Sitz bath (I never did find out the awful details and was too chicken to ask), or how to breast feed with only two hands and one pillow. Heck, I was absolutely horrified to find out what "pericare" actually was. By the time medical school lets you loose on the world presumably you have figured out how to pick up a baby – but to bathe it? These little facts should be engraved over every patient's door: assume total ignorance until proven otherwise. It took me two days to find a nurse wise enough to heed my pleas to ignore my so-called credentials and actually show me how to keep from drowning my daughter. And after two deliveries I have yet to find the showers.

No knock on the nurses though. Their job is a hard one. And I'll always remember with gratitude their support in the case room one night early in my second pregnancy. I had started spotting myself while in assessing a patient. It all ended well, with a healthy son, but there was no way of knowing it would that night. They couldn't have been kinder.

Pregnancy number two I got to spend lots of in bed. (I really don't do this pregnancy thing well, at all). Now we were told quite firmly in medical school that bed rest is almost a placebo, if a miscarriage is going to happen early it will happen. But what can you do if, in defiance of the books, every time you get up nasty things (cramps, bleeding) start to happen? And every time you go back to bed the bank balance sinks lower? One of the physician-as-a-patient anomalies that hit hard was that my patients who were ill in pregnancy could collect UIC. As a self-employed bedridden physician, Revenue Canada got to collect from me. One really dark day in my pregnancy they showed a news clip of a Senator in Ottawa, who I am sure gets regular pay cheques, decrying "fat-cat" doctors looking for tax breaks on the GST. I cried.

Perhaps there is a hidden benefit to such difficulties. The patients were right in their perception that my credibility would improve. I know now why pregnant women panic over things which previously I would have thought trite. I know why it is impossible not to push over an anterior lip of the cervix. I know that my memory of life will henceforth be divided into that tetanic contraction that lasted three minutes and the rest of my life (and, by the way, it really does hurt). I, too, have lain on the delivery table and wondered how many stitches this was going to require, and I am a better doctor for it. □

ACKNOWLEDGEMENTS

I wish to acknowledge with gratitude the excellent care of Doctors I.A. Perlin, L. Stirk, and M. Van den Hoff; and most of all that of our family physician Dr. Brenda Ashley.

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Intraventricular Hemorrhage in the Preterm Infant

Gerald P. Murray, MD, and William J. Howes, MD, FRCSC

Halifax, N.S.

Preterm infants of low birth weight are very susceptible to brain hemorrhage. Usually, it is in relation to the germinal matrix which is a richly vascular subependymal structure. The ensuing hemorrhage may be limited to the germinal matrix or may rupture into the lateral ventricle or extend into the brain parenchyma. One of the complications of intraventricular blood is interference with CSF circulation, leading to ventricular dilatation, ie. hydrocephalus.

Based on the location of the blood and the presence or absence of hydrocephalus the following classification scheme has been proposed:[1]

Grade 1: Germinal Matrix Hemorrhage Only
Grade 2: Intraventricular Hemorrhage Without Hydrocephalus
Grade 3: Intraventricular Hemorrhage With Hydrocephalus
Grade 4: Intraventricular Hemorrhage With Parenchymal Extension

The incidence of intraventricular hemorrhage is directly related to the degree of prematurity, James[2], in a review of this subject, quoted studies showing an incidence of 40-50% when the birth weight was less than 1500 grams, whereas Bejar *et al.* reported an incidence as high as 90% if gestational age was less than 34 weeks.[3,4]

Of those infants with hemorrhage, 20-50% will go on to require treatment for either transient or progressive hydrocephalus.[5] There is generally a good correlation between the severity of the hemorrhage and the subsequent development of hydrocephalus. The best data supporting this was reported by Volpe *et at.*[6] In 800 cases they found the rates of hydrocephalus to be 5%, 25%, 55% and 80% for grade 1-4 respectively.

Numerous studies have looked at various aspects of outcome. In general, prognosis is related more to the extent of parenchymal injury and other aspects of prematurity rather than the development of hydrocephalus *per se.*

In looking at mortality, the Volpe *et al.* series quote rates of 15% and 20% for grades 1 and 2, whereas grades 3 and 4 were associated with mortality rates of 40% and 60%, respectively.[6]

James cited several studies which examined intellectual function. The majority of children in these series had mild to moderate hemorrhages, ie. grades 1 or 2.[2] In general, approximately 80% of these children were found to be intellectually normal on long term follow-up. Fewer studies are available which examined long term intellectual function in those suffering more severe degrees of hemorrhage. Guzzetta, *et al.* reported on 37 children with severe intraparenchymal injury, which would correspond to grade 4 hemorrhage.[7] Of this group, there were only 7 survivors, all of whom had major motor deficits and only 1 had an I.Q. greater than 80. Boyton, *et al.* reviewed a series of 50 children, which included grade 3, in addition to grade 4, hemorrhages.[8] Of the survivors, only 18% were found to have normal motor and developmental scores. The authors noted that grade 4 patients had lower scores than grade 3.

In summary, the following points can be made:

1. The development of intraventricular hemorrhage is directly related to the degree of prematurity.
2. The development of hydrocephalus is correlated with the quantity of intraventricular blood.
3. Outcome is related to the severity of the brain injury caused by the hemorrhage.
4. Outcome, in terms of motor and intellectual function, is good for grades 1 and 2 with approximately 80% being normal.
5. For grades 3 and 4, prognosis is dismal. Approximately 50% will not survive and among the survivors very few will be free of major motor and/or intellectual impairments. □

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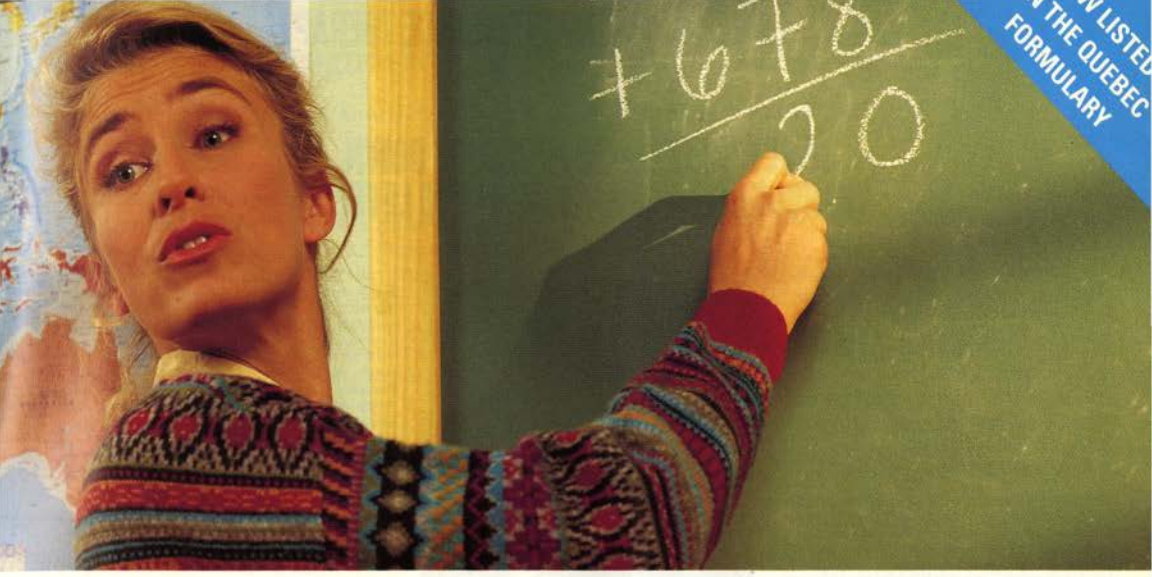

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CONTRAINDICATIONS: In presence of (1) sinus bradycardia; (2) 2nd and 3rd degree A-V block; (3) right ventricular failure due to pulmonary hypertension; (4) congestive heart failure; (5) cardiogenic shock; (6) anesthesia with agents that produce myocardial depression, e.g. ether.

WARNINGS: (a) Increase in **antinuclear antibody** (ANA) titer was observed in approx. 12.5% of patients on chronic SECTRAL therapy. Rare instances (< 1%) of a syndrome resembling lupus erythematosus have been reported with maintenance therapy. Similar symptoms were occasionally observed with some other β-blockers. Other main presenting symptoms included polyarthralgia, myalgia and pleuritic pain. Symptoms and elevated ANA titer appear reversible upon discontinuation of SECTRAL therapy. The drug should be withdrawn if symptoms appear or if the results of ANA testing are significantly positive. Patients should be followed up both clinically and serologically until resolution of symptoms. **(b) Cardiac Failure** – Exercise special caution when administering SECTRAL to patients with history of heart failure. Sympathetic stimulation is a vital component supporting circulatory function in congestive heart failure. Inhibition by β-blockade carries potential hazard of further depressing myocardial contractility, precipitating cardiac failure. SECTRAL acts selectively without abolishing the inotropic action of digitals on heart muscle but negative inotropic effect of SECTRAL may reduce positive inotropic effect of digitals when drugs used concomitantly. Effects of β-blockers and digitalis are additive in depressing A-V conduction. In patients with no history of heart failure, continued depression of myocardium over time can sometimes lead to cardiac failure. At first sign or symptom of impending cardiac failure, digitalize patient fully and/or give a diuretic and observe response carefully. If heart failure continues, SECTRAL should be immediately withdrawn. **(c) Abrupt Cessation of Therapy** – Angina patients should be warned against abrupt discontinuation of SECTRAL therapy because of risk of severe exacerbation of angina, myocardial infarction and ventricular arrhythmias; the latter two may occur with or without the first. When discontinuing therapy, dosage should be reduced gradually over about 2 weeks under careful observation, with frequency maintained. If greater urgency is required, reduce dosage step-wise under closer observation. If angina worsens markedly or acute coronary insufficiency develops, reinstitute SECTRAL promptly (at least temporarily). **(d) Various skin rashes** and conjunctival xerosis have been reported with β-blockers including SECTRAL. **(e) Severe sinus bradycardia** may occur from unopposed vagal activity remaining after β_1-adrenergic receptor blockade; dosage should be reduced. **(f)** Possible deleterious effects of long-term SECTRAL use in **patients with thyrotoxicosis** have not been adequately appraised. By masking clinical signs of continuing hyperthyroidism or its complications, SECTRAL may give a false sense of improvement and abrupt withdrawal may be followed by exacerbation of hyperthyroid symptoms including thyroid-storm. **(g) Usage in Pregnancy** – Since both acebutolol and diacetolol cross the placenta, SECTRAL should not be given to pregnant patients. If used in women with child-bearing potential, anticipated benefits should be cautiously weighed against possible risk. **(h) Nursing Mothers** – Since acebutolol and diacetolol appear in breast milk (respective milk/plasma ratios of 7.1 and 12.2), use in nursing mothers is not recommended.

PRECAUTIONS: (a) In general, patients with **bronchospastic disease** should not receive β-blockers but SECTRAL's β_1-selectivity may be cautiously used in such patients unresponsive to, or intolerant of alternative treatment. Since β_1 selectivity is not absolute and is dose-dependent, the lowest possible

dose should be initially used, preferably divided to avoid higher plasma levels associated with longer dose intervals. A bronchodilator such as a theophylline or a β_2-stimulant, with instructions for use, should be made available in advance. If an **allergic-type reaction** occurs in patients on β-blockers, treatment may be more difficult because reaction may be more severe due to pharmacological effects of the drug, and problems with fluid changes. Administer epinephrine cautiously because it may not have its usual effects in anaphylaxis. Larger doses may be needed to overcome bronchospasm, but may create excessive α adrenergic stimulation with consequent hypertension, reflex bradycardia and heart block, and possible bronchospasm potentiation. Alternatives to large doses of epinephrine include vigorous supportive care, e.g. fluids, and β-agonists including parenteral salbutamol or isoproterenol to control bronchospasm, and norepinephrine to overcome hypotension. **(b)** Administer SECTRAL with caution to patients subject to spontaneous hypoglycemia or to **diabetic patients** (especially labile) receiving insulin or oral hypoglycemic agents. β-blockers may mask premonitory signs and symptoms of acute hypoglycemia. **(c)** Cautious administration is advised in **patients with impaired renal function** because diacetolol is eliminated mainly by the kidney. **(d)** SECTRAL has been used in the **elderly** without specific dosage adjustment but these patients may need lower maintenance doses because bioavailability of both acebutolol and diacetolol are approximately doubled, probably due to decreased 1st pass metabolism and renal function. **(e)** SECTRAL dosage should be individually adjusted when used with other antihypertensive drugs. **(f)** Liver function tests should be done at regular intervals during long-term therapy. **(g)** Management of patients undergoing **elective or emergency surgery** while on β-blocker therapy is controversial. β-adrenergic receptor blockade impairs ability of the heart to respond to β-adrenergic mediated reflex stimuli; abrupt withdrawal can create serious problems (See Warnings). Some patients receiving β-blockers have suffered protracted, severe hypotension during anesthesia. Difficulty in restarting and maintaining heartbeat has also been reported. Thus in angina patients undergoing elective surgery, SECTRAL should be withdrawn gradually as outlined under "Abrupt Cessation of Therapy". Available evidence shows all clinical and physiological effects of β-blockade are no longer present 72 hours after medication withdrawal. SECTRAL is a competitive inhibitor of β-adrenergic receptor agonists and in emergency surgery its effects may be reversed, if necessary, by sufficient doses of such agonists as isoproterenol. **(h)** There is no experience with SECTRAL in **pediatric age groups** and therefore use in children is not recommended.

(i) Drug interactions – Catecholamine-depleting drugs, e.g. reserpine, may have an additive effect when given with β-blockers. Exaggerated hypertensive responses have been reported from combined use of β-blockers and α-adrenergic stimulants, including those in proprietary cold remedies and vasoconstrictive nasal drops. No significant interactions have been noted with digoxin, hydrochlorothiazide, hydralazine, sulfinpyrazone, oral contraceptives, tolbutamide or warfarin. If therapy with β-blockers and clonidine is to be stopped, discontinue the β-blocker several days before gradual withdrawal of clonidine. It has been suggested that withdrawal of clonidine in presence of β-blockade may exaggerate clonidine withdrawal syndrome (See Prescribing Information for clonidine).

ADVERSE REACTIONS: Most serious – congestive heart failure, severe bradycardia and bronchospasm in < 1% of patients. Most common – fatigue (4%), dyspnea (2.5%), nausea (2%), dizziness (2%), hypotension (1%) and rashes (1%). Adverse reactions by systems: **Cardiovascular** – congestive heart failure; secondary effects of decreased cardiac output including syncope, vertigo, light headedness, postural hypotension; severe bradycardia, lengthening of PR interval; 2nd and 3rd degree A-V block; sinus arrest; palpitations; chest pain; cold extremities; Raynaud's phenomenon; hot flushes; leg pain; edema. **CNS** – headache; dizziness; mental depression; tiredness; drowsiness or somnolence; light headedness; anxiety; tinnitus; weakness; confusion; vivid dreams; paresthesia; insomnia. **Gastrointestinal** – nausea and vomiting; heartburn; indigestion; flatulence; abdominal pain; diarrhea; constipation. **Respiratory** – dyspnea; cough; shortness of breath; wheezing; bronchospasm. **Allergic-dermatological** – urticaria; pruritus; sweating; exfoliative dermatitis; psoriasiform rash; lupus like syndrome with arthralgia, myalgia, dyspnea and pleuritic pain, reversible on withdrawal of drug. **EENT** – blurred vision and non-specific visual disturbances; itching eyes; conjunctivitis. **Miscellaneous** – weight gain; appetite loss; decreased libido; shivering; micturition (frequency); nocturia. **Lab Tests** – occasional reports of increased transaminase, alkaline phosphatase and lactic dehydrogenase values; and positive ANA.

SYMPTOMS AND TREATMENT OF OVERDOSE – Most common symptoms – bradycardia, congestive heart failure, hypotension, bronchospasm and hypoglycemia. Treatment – in all cases, SECTRAL should be discontinued and patient closely observed. If required, the following therapeutic measures are suggested: 1. Bradycardia – atropine or other anticholinergic. 2. Heart block (2nd or 3rd degree) – isoproterenol or transvenous cardiac pacemaker. 3. Congestive heart failure – conventional therapy. 4. Hypotension (depending on associated factors) – epinephrine plus atropine and digitalis (See Precautions for epinephrine). 5. Bronchospasm – aminophylline or isoproterenol. 6. Hypoglycemia – intravenous glucose. Isoproterenol is a competitive antagonist of SECTRAL and large doses can be expected to reverse many effects of a SECTRAL overdose. Complications of excess isoproterenol should not be overlooked.

DOSAGE AND ADMINISTRATION – Dose must always be adjusted to individual requirements in accordance with the following guidelines: **Hypertension** – SECTRAL is usually used with other antihypertensive agents (particularly thiazide diuretics) but may be used alone. Initiate treatment with 100 mg b.i.d. If no adequate response in one week, increase to 200 mg b.i.d. In some cases, further increments of 100 mg b.i.d. at 2 week intervals may be needed to a maximum of 400 mg b.i.d. Maintenance – 400 to 800 mg/day. Patients with satisfactory response at 400 mg/day or less may receive total dose once daily in the morning. **Angina Pectoris** – Initial dose – 200 mg b.i.d. If no satisfactory response in 2 weeks, increase dose to a maximum of 300 mg b.i.d. Usual maintenance dose – 200 to 600 mg/day in two divided doses. A maintenance dose of 100 mg b.i.d. may be tried in patients adequately controlled on 400 mg/day. **Use in Elderly** – Older patients may have approx. 2 fold increase in bioavailability and are likely to require lower maintenance dosage. Use in **Patients with Impaired Renal Function** – Reduce dosage by 50% when creatinine clearance < 50mL/min. and by 75% when < 25mL/min. (See Precautions). SECTRAL and its metabolite are dialyzable. **Dosage Forms** – SECTRAL 100 mg, available in bottles of 100 and 500, scored white shield shaped tablets, marked with the logo on one side and SECTRAL 100 on the other. SECTRAL 200 mg, available in bottles of 100 and 500, scored blue shield-shaped tablets, marked with the logo on one side and SECTRAL 200 on the other. SECTRAL 400 mg, available in bottles of 100 and 500, scored white shield shaped tablets, marked with the logo on one side and SECTRAL 400 on the other.

† ONCE MAINTENANCE DOSAGE FOR HYPERTENSION IS ESTABLISHED (RANGE 400-800 mg), DOSES UP TO 400 mg CAN BE GIVEN ONCE DAILY. FOR ANGINA, RECOMMENDED DOSAGE (RANGE 200-600 mg) SHOULD BE GIVEN IN TWO DIVIDED DOSES.

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Prognosis of Hydrocephalus in Infants

David C. Ley,* BSc., BEd and W.J. Howes,** MD, FRCS(C)

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The aim of this paper is to provide a concise source of information on the prognosis of hydrocephalus (HC), for several reasons. The first and most important question arising from parents is the prognosis with and without intervention. The ethical dilemmas arising from prenatal diagnosis by ultrasound or amniocentesis requires this prognostic information. For these reasons, a literature review was carried out by the junior author as an elective project.

Various articles list incidence rates as 0.053%[1], 0.11%[2], 0.1% to 0.4%[3]. Therefore, a rough estimate of 1 per 100 live births appears to be appropriate.

Different types or causes of HC lead to a different prognosis. The following more common causes of HC were reviewed:

1. Spina Bifida
2. Aqueduct Stenosis
3. Dandy-Walker Syndrome

Finally, the outlook for prenatally diagnosed HC, often of undetermined etiology, was examined.

SPINA BIFIDA

Spina bifida is the most common cause of HC. In a study of the incidence rate of spina bifida, the reported rate was 3.69 per 10,000 live births.[4] In one study (n=287), it was shown that 62% of untreated infants with a myelomeningocele survive one month of age and 30% survive six months.[5] The probability of surviving infancy with spina bifida in the period 1952 to 1969 was about one-third, this rate doubled for the period 1970 to 1986.[4]

A rise in survival rates does not necessarily lead to a better situation in all cases. Therefore, it is important to look at the quality of life of an infant or child with spina bifida. In one study, 59% of the survivors of spinal bifida had IQs above 80 which is in the range of educational normality.[6] Other studies which concur show this rate to be 63%[7] and 53%.[5]

It should be noted that only 65% to 85% of children with myelomeningocele develops HC[7,8], and a child with myelomeningocele has a decreased chance for normal intelligence if HC is also present.[7] Also, children with spinal bifida, treated for their HC, have a better chance for normal intelligence than non-treated hydrocephalic children, but still not as good as the chances are for a child without HC[7], 80% of whom will have an IQ greater than 80.[9]

Lorber found that the prognosis of children with myelomeningocele and extreme HC is poor, with only 8% of such affected children surviving and having an IQ within educational normality.[5] Other sources warn against assuming a relationship betwen IQ and degree of HC or cortical mantle thickness.[6,7] In six studies of this possible relationship, five showed that no relationship exists.[10]

Ordinary schools is the schooling for 40% of the children treated with myelomeningocele. From the same study, 51% attend day schools or residential special schools for physically handicapped students, while most of the rest receive no formal education.[5]

Along with the mental capabilities of children with spina bifida, it is necessary to look at motor development in these patients. Although this is more related to the interruption in the spinal cord than to the HC, parents and doctors would still be interested in this area of the prognosis. In one study, 17 out of 134 children (13%) surviving treatment for myelomeningocele had normal legs and could walk without any aid; fifteen children (11%) walked with a limp but did not require aid; twelve (9%) waddled and needed sticks to walk; while 49 (37%) needed callipers and crutches.[5] Forty-one (31%) were completely confined to a wheelchair.

STENOSIS OF THE AQUEDUCT OF SYLVIUS

Aqueduct stenosis in one study had an overall survival rate of 74.6% (106 out of 142 patients).[11] In this study, deaths occurred after the first surgery (4.2%) or related to shunt malformation (13.3%) or after shunt revision (7.7%). Others report a survival rate of 89% for aqueduct stenosis.[12]

Normal intellectual development is found in 58%[12] to 68%[11] of those with aqueduct stenosis. A study of both children and adults reports that out of 90 patients, 61% were capable of normal work or schooling, 23% were able to care for themselves and perform limited work, while the remaining 16% were unable to care for themselves.[13] Epilepsy occurs in as many as 39% of those with aqueduct stenosis.[13]

DANDY-WALKER SYNDROME

Dandy-Walker Syndrome (DWS) is a congenital HC due to obstruction of the foramen of Magendie and Luschka. "DWS is characterized by the association of HC, a cyst in the posterior fossa and a defect in the cerebellar vermis through which the cyst communicates with the fourth ventricle".[14] This syndrome may be found with other brain anomalies such as agenesis of the corpus callosum, aqueduct stenosis, and spina bifida.[15]

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Many babies with DWS found on ultrasound screening may not have HC. Pilu *et al.* report 80% of newborns with DWS do not have HC.[11] Pilu *et al.* reviewed prognosis of infants with DWS and cite one source as reporting a survival rate of 50% with half of the survivors being severely intellectually impaired.[14] Other surveys list survival rates as being 74% and 88% with the survivors having an IQ above 80 in 29% and 60% of the cases.[16,17] Toronto Sick Children experience lists 33% as being the percentage in the range of normal mental development.[18]

PRENATALLY DIAGNOSED HYDROCEPHALUS

By using ultrasound techniques, it is possible to diagnose HC in the unborn fetus, although not always the etiology.

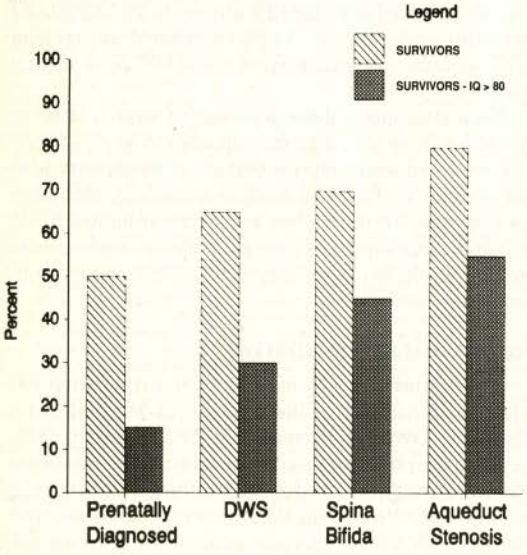
Cochrane *et al.* studied 40 cases of intrauterine HC: 65% were stillborn, and died without any attempts being made at treatment, 27.5% were developmentally delayed and 7.5% were normal.[19] In a literature search of 112 cases, Cochrane *et al.* found similar distributions.[19] Pregnancy ended in abortion or stillborn for 39 (35%) while 37 (33%) died without any attempt at treatment. Nineteen (17%) were delayed developmentally and 11 (10%) were normal.

A study of 75 surviving infants with prenatal HC showed that 28% had an IQ over 80, while 50% had an IQ below 60.[20]

It is obvious from the above figures that the prognosis for a fetus with HC is poor. Because of results like these, intrauterine treatment of et al HC has largely been abandoned.[21]

SUMMARY

A graph outlining prognosis for survival and intelligence follows:



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Bilateral Posterior Humeral Fracture – Dislocation as a Complication of a Generalized Seizure

Deon F. Louw, MB, ChB[1], K. Reddy, MD, FRCSC[2], G.R. Sutherland, MD, FRCSC[2] and L.G. Sank, MB, ChB[3]

Generalized tonic-clonic seizures have been associated with various unusual orthopaedic injuries such as fracture-dislocation of the shoulder. In this report, a case of bilateral fracture-dislocation of the humeri associated with a generalized seizure is presented. Synergistic contraction of the shoulder girdle musculature is causally implicated. A high index of suspicion is required for early diagnosis and management of this injury.

The epileptic population suffers a disproportionately increased incidence of bony injury.[1] This may reflect osteopenia secondary to anti-convulsant therapy and/or trauma, either direct or secondary to seizure-induced muscle contraction. Additionally, they are more likely to sustain atypical injuries, for example posterior dislocation of the shoulder. This report presents an unusual case of bilateral posterior fracture-dislocation of the humerus associated with a generalized convulsion. Five similar cases have been previously documented.[2-6]

CASE REPORT

A 70-year-old female presented in 1987 to the Emergency Room with a complaint of severe, bilateral shoulder pain evidently rousing her from sleep. There was no antecedent history suggestive of trauma or systemic malignancy. The family found her to be "confused" to the extent that she was initially unable to recognize her own son.

Relevant past medical history included a subfrontal craniotomy with subtotal resection of a pituitary adenoma in 1977. This procedure was followed by adjuvant radiotherapy (5,000 rads). She remained asymptomatic until 1985 when she presented with episodes of transient "confusion". The duration of these events varied from 15 to 45 min. Reinvestigation disclosed a large, recurrent pituitary adenoma. In view of her age, she was treated conservatively with a further course of radiotherapy. Electroencephalography (EEG) was not performed at this point, and anti-convulsant therapy not commenced.

The patient was on replacement thyroxine and Ibuprofen (Motrin®, Upjohn Company, Kalamazoo, Michigan) for chronic arthritis. Her endocrinologic follow-up had been unremarkable apart from elevated gonadotropins appropriate for her age.

Clinical examination in the emergency room revealed a mildly obese, hypertensive lady in obvious distress. There was abnormal prominence of both coracoid processes along with marked limitation of adduction and external rotation of both shoulders. Neurologic examination revealed global disorientation, and a residual left optic atrophy. Plain x-rays of both shoulders showed bilateral humeral neck fracture-dislocation (Fig. 1 and 2) with cortical thinning and osteopenia. Attempted closed reduction resulted in suboptimal bony alignment and hence, an elective operative reduction was planned. Following her admission to hospital, the patient had several witnessed generalized seizures, and epileptiform activity was noted on EEG. Anticonvulsant therapy in the form of Diphenylhydantoin (Dilantin® 100 mg, p.o., t.i.d., Parke-Davis Canada Inc.) was instituted.

DISCUSSION

Shoulder dislocations and humeral neck fractures constitute a relatively common presentation to the emergency department. Unilateral posterior dislocation of the shoulder is rare, and accounts for only 1.7% to 4.3% of all shoulder dislocations.[4] Acute fracture of the humeral head has been reported in the setting of chronic posterior subluxation.[7] Bilateral posterior fracture-dislocation is extremely rare with only five other such cases documented in the literature.[2-6] All cases were considered to be secondary to generalized tonic-clonic seizures.

Sequential mechanical events that are peculiar to the phenomenon of a convulsive seizure are likely responsible for the patient's unusual injuries.[4] The position of the shoulder during a convulsion is characteristically one of adduction, internal rotation and flexion. In this position, the humeral head is thought to be vulnerable to posterior dislocation were synergistic contraction of the shoulder girdle muscles to occur. Prolonged muscular contraction could result in shearing off the humeral head from an anatomical neck impinged against the glenoid rim.[4]

The incidence of non-seizure related fractures was reported to be six times greater in epileptics than in the normal population.[1] Osteopenia secondary to anticonvulsant therapy may, in part, account for this increased incidence.[8] Anticonvulsant medications putatively facilitate degradation of vitamin D and its metabolites via induction of hepatic microsomal enzymes.[9] This could result in under-mineralization of bones and a consequent susceptibility to bony injury following trauma. Additional to a generalized increased incidence of all fractures, the epileptic is particularly

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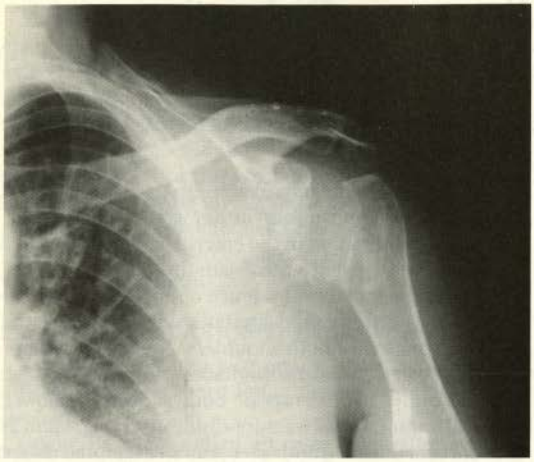

FIG. 1A

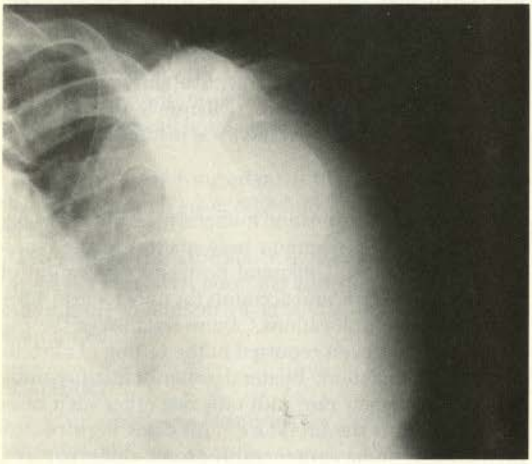

FIG. 1B

Anterior posterior (Fig. 1A) and trans-scapular (Fig. 1B) radiographs showing a fracture of the left anatomical neck with posterior dislocation of the left humeral head. Mild osteoporosis as evidenced by demineralization and thin cortices is also seen.

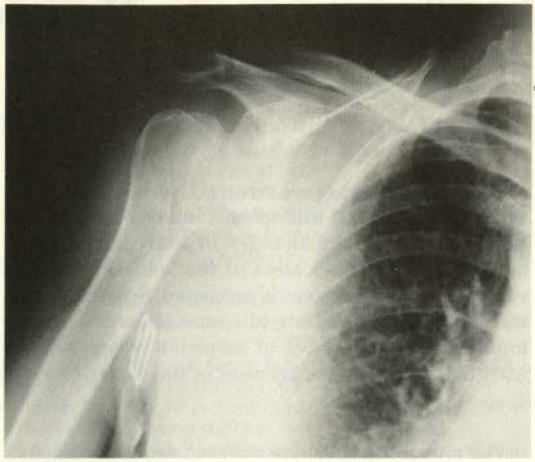

FIG. 2A

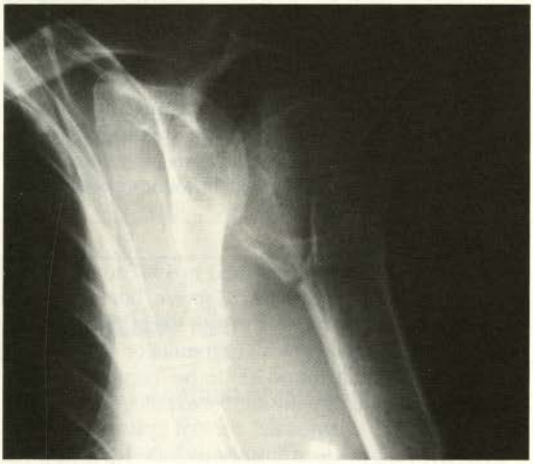

FIG. 2B

Anterior posterior (Fig. 2A) and trans-scapular (Fig. 2B) radiographs showing a fracture of the right anatomical neck with posterior dislocation of the right humeral head. Mild osteoporosis as evidenced by demineralization and thin cortices is also seen.

prone to sustaining vertebral fractures[10], acetabular fracture-dislocations[11] and femoral neck fractures.[12] Rarely, scapular fractures[13], manubriosternal fracture-dislocation[14] and anterior shoulder dislocation may occur.[8]

In conclusion, we emphasize the predisposition of the epileptic to orthopaedic injury. These injuries may be unusual in both nature and location, eg. approximately 50% of posterior shoulder dislocations are overlooked at the initial radiographic examination.[15] Furthermore, typical clinical features of posterior dislocation (eg. prominent coracoid process) may not be present. It is suggested that postictal or "spontaneous" onset of localized pain (particularly if there is impairment of associated joint movement) requires careful clinical and radiographic evaluation. Biplanar radiographs are essential. □

ACKNOWLEDGEMENTS

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Preparation of Medico-Legal Reports

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The medico-legal report is the bane of many physicians' existence, perhaps for several reasons. First, you never seem to have enough time or receive enough money for the trouble of sitting down and drafting the darn thing. As well, you're drifting into somewhat unfamiliar territory where you must attempt to present the imprecise science of medicine to a lawyer who revels in probabilities and likelihoods. You cannot be vague and you cannot equivocate. An "I don't know" from you, the doctor, will almost always prompt the lawyer to respond with, "Yes, but could you hazard an educated guess?"

I'm sure none of you took a course in your medical training on how to prepare a medico-legal report. Well, you can take some comfort in the knowledge that none of us lawyers took a course in what to ask for or how to read the stuff. So, the best that we can do is try to help each other, for the benefit of the patient and client.

One of the most common complaints that I have heard from doctors about lawyers is that lawyers are always trying to put words into doctors' mouths. If the doctor says that there is a chance or a possibility that the traumatized knee may develop arthritis, that never seems to be good enough. The lawyer wants to know how much of a chance – can you put a percentage point on it – can you say that there is a "reasonable likelihood" of arthritis – can you say that it is "more likely than not" that the arthritis will develop? We are not pushing you because we are mean or ungrateful or pushy. We push you because we have to deal with the question of causation and with thresholds of legal proof.

For you doctors, the interest is in treating the injury. The cause of the injury is useful for you to know because it impacts on diagnosis and treatment, but once known, your focus is on treating and assisting recovery. What caused the injury in the first place becomes historical data. In my neck of the woods, causation is a hot ticket. In any personal injury case that I advance or defend, I must deal with a threshold of proof that demands that the case be proved "on a balance of probabilities". Thus, I cannot be content with a vague "maybe" from a doctor when a greater exploration may reveal that the doctor meant to say "likely" or "possible but not very likely", because this change in terminology can make a dramatic difference in the legal result. Recently, this whole conflict in our spheres was discussed by one of the pre-eminent authors in legal circles, John G. Fleming, in a scintillating article entitled, "Probabilistic Causation in Tort Law", which is found in the *Canadian Bar Review* (vols. 68 and 70). He said:

> In a paper recently published in this Journal I addressed the problem increasingly faced by some plaintiffs of meeting the traditional standard of proof of causation against negligent defendants in situations where available scientific or statistical evidence cannot tip the balance of probabilities (more probable than not). Thus where a polluter's responsibility for a particular disease or injury, in competition with other natural sources, can only be expressed in terms of statistical or epidemiological evidence short of 50:50, victims, would most likely fail under the traditional standard. An even more frequent occasion in modern litigation is the inability of medical experts to explain the oetiology of an injury with a degree of exactitude and confidence postulated by traditional formulas like "reasonable certitude" or "reasonable probability". The inherent limitations of medical knowledge, combined with a tendency of physicians to express outcomes in terms of percentage, create problems of compatibility with legal standards, which are both linguistic and substantive.

Therein is the conflict. But it is not fatal to lawyers and doctors communicating effectively. It merely illustrates the challenges inherent in the process.

Now on to the nitty-gritty of report writing. The medico-legal report should enable the lawyer to understand the doctor's methods, treatment and opinions. It should be written in a comprehensive and comprehensible way. And it should *look* credible. A scribbled out note on a prescription pad just doesn't pass muster (for one thing, who can read the writing?). Such a report carries with it an attitude that might trigger the opposing lawyer to think, "Is this a sloppy or uncaring practitioner that I should take to task at discovery or on the stand?" A carefully thought-out and well-presented report will not invite attack or scoffing and will stand up to cross-examination. Take the time to do it right and bill appropriately for your trouble.

The general practitioner who is the patient's doctor assumes a particularly important role in the personal injury lawsuit. He or she should be counted on to be an impartial source of information concerning the patient's post-accident condition as contrasted with the patient's pre-accident condition. The G.P. is the link between past and present. In seeking a medico-legal report from the family doctor, the lawyer looks for three main topics of commentary and delights in receiving a report which is broken down into discussion of the following topics:

1. the doctor's clinical evaluation by
   (a) history (b) physical examination
2. diagnosis
3. prognosis and commentary

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1. a) History

The general practitioner, unlike the specialist who sees the patient for the first time on a referral, is in the unique position of being able to draw on months or years of consultations with the patient in developing a medical history. Summarizing months or years of familiarity with a patient can be difficult, but areas which are of interest to the lawyer are:

- comments as to the patient's pre-injury lifestyle and medical background, such as age, marital status, number of children, particularly the number of children residing at home
- comments as to patient's education and training in specific areas, and future career plans if known
- patient's occupation at the time of the injury and the physical demands of that occupation
- areas of the patient's personal life that make physical demands
- the patient's previous medical history, specifically previous illnesses, accidents or operations with special attention to previous injuries to the areas in issue, and the extent of any functional impairments as a result of the pre-accident conditions
- information gained from the patient about the dynamics of the accident
- post-accident developments

1. b) Physical Examination

The various formal tests and manipulations used should be chronicled and the observations noted. It is also useful to include informal, general observations, ie. whether the patient appeared to be in any physical or emotional distress, or whether the patient appeared stiff or uncomfortable, or had trouble getting dressed or sitting, etc.

2. Diagnosis

The diagnosis should be stated in terms of the particular part of the body involved and the nature of the injury or problem. There are probably two schools of thought on this, but I prefer the medical terminology to the layman's version of the injury, that is, I would rather read the patient suffered a fractured tibia than that she broke her shinbone. The former sounds more authoritative. Of course, using the medical terminology followed by a translation into laymen's talk is extremely helpful.

Sometimes the diagnosis cannot be stated in certain terms. Then, it is very useful to the lawyer if the doctor ascribes his or her confidence level in the diagnosis.

Severity of the injury is always a big issue. The level of compensation for a victim's pain and suffering caused by an injury is not simply a function of the severity of the injury, but severity becomes a large factor by reason of the process we lawyers and the courts use to come up with suitable figures. In assessing compensation for an injury, the court is required to achieve some uniformity or predictability and the court seeks guidance in this endeavour from other cases that were decided before. In comparing the case before it with other previous cases involving the same anatomical part, it is inevitable that the relative severity of the injury is assessed. If the doctor in the medico-legal report has classified the ligamentous neck injury as "mild", "moderate" or "severe" these classifications tend to narrow the focus of the search for comparative cases. The lawyer or judge can search a computer data base for cases where the term moderate is used in conjunction with this kind of injury, and a set of comparative cases is generated. The long and the short of this process is that terminology as to degree of severity is very powerful stuff, and the terminology should be used and explained carefully.

If you are going to venture into the classification of an injury as to degree of severity, then I would encourage you to explain in the report what you mean by "mild" or "moderate" or "severe", so there is no question about this. I find that the patient's family doctor describes the injury as severe much more often than does the specialist to whom the patient is referred. I suspect there may be some notion that a "severe" rating will somehow help the patient resolve his claim, but if the injury is less than severe, this kind of "puffing up" will only serve to discount the value of your report as a whole.

Also be wary that just as we lawyers use legal jargon, you doctors have your own pet phrases and terms that are not readily understandable. For the longest time I equated the term "acute" with "severe" only to find out much later that the doctor using this term meant that the symptoms were produced suddenly rather than gradually.

3. Prognosis

This is the tricky part of the report, because up to this point, the report is largely narrative and factual. Now you must venture an opinion. In a wonderful resource book entitled *Personal Injury: a medico-legal guide to the spine and limbs*, Darrell J. Ogilvie-Harris and Geoffrey J. Lloyd, two orthopedic surgeons, offer the following remarks:

> So far the statements in the report have largely been a matter of fact. The second part of the report is a matter of opinion, and its quality is obviously a function of the examiner's background, training, education and experience. Very useful are comments that deal with the following: Has the treatment conformed to the conventional norm? Has recovery occurred within the expected time frame? Also valuable is comment on factors that add to or take away from the credibility of the patient: for example, is there consistency or discrepancy between the findings on the formal examination and the casual observation of the patient? The final and perhaps the most important aspect of the whole report is an opinion on the effect of any observed anatomical disability on the patient's pre-existing functional lifestyle. In the final analysis it is probably this statement, more than any other, that is going to influence the quantum of damages awarded.

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The only thing to add to this is that the wording of your prognosis may have to be attuned to a specific set of criteria, depending on the use to which your report is to be put. If your report is to be used to send to an insurance company on the question of whether the patient is eligible to receive no-fault benefits under an automobile policy or disability benefits under an accident policy, then there are certain threshold tests to be considered and commented upon in the report.

In an auto policy continued eligibility for no-fault benefits hinges on whether the patient continues to suffer "substantial inability to perform the essential duties of his occupation or employment" within the first two years after the accident, and after two years the question becomes whether the injury "continuously prevents such person from engaging in an occupation or employment for which he is reasonably suited by education, training or experience". These are tests which must be specifically addressed in the prognosis, and a failure to comment about these criteria can mean a significant delay in the patient receiving needed funds.

Example of MedicoLegal Report

Everybody's writing style is different, thank goodness, but a fine example of a specialist's medico-legal report was produced in *Personal Injury: medico-legal guide to the spine and limbs,* mentioned above. □

For copies of this article or the above mentioned *Personal Injury: medico-legal guide to the spine and limbs,* contact the author.

The author of this article has been given the freedom to express his opinions and use his judgement to interpret the law. Nothing in this article is or should be interpreted as legal, medical, or financial advice from a lawyer, doctor or accountant or from this *Journal.*

BILATERAL POSTERIOR HUMERAL FRACTURE

Continued from page 24.

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Current Topics in Community Health

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GUIDELINES FOR THE CONTROL OF MENINGOCOCCAL DISEASE

It is very unlikely there is any physician in Nova Scotia or indeed in Canada that is not aware that meningococcal disease continues to be a significant cause of morbidity and mortality in this country. Generally, invasive disease tends to occur sporadically, with the number of cases rising and falling in a cyclical fashion. Periodically, however, cases can cluster in specific geographic areas producing the need for specific public health measures such as wide spread immunization. As these events have recently occurred in Canada, producing heightened public anxiety about this disease, many physicians and health care institutions in the province have reported a dramatic increase in inquiries from the public with respect to this disease.

Nisseria meningitidis, the organism which causes invasive meningococcal disease, has thirteen serotypes of which groups A, B, C, Y and W135 are most likely to cause disease. The group C strain has been seen more frequently in Canada recently and it has been responsible for current outbreak. In 1989 the average reported incidence rate in this country for all regions reporting cases ranged between 0.6 and 16.9 cases per 100,000 population.

Invasive meningococcal disease is primarily a disease of childhood, occurring most frequently in the first year of life. Infants are at increased risk of acquiring the disease by a factor of ten as compared with the general population. In recent years, cases among adolescents and adults have increased substantially, and the disease causes significant mortality and morbidity. In average years the case fatality rate is in the vacinity of 10%. This has increased substantially in adolescents during the recent outbreak in Ontario, where the mortality rate was approximately 50%. For those who do survive the disease, there is a varying rate of sequelae, particularly with respect to neurological damage.

Public health systems generally pay significant attention to invasive meningococcal disease. As a consequence, it is important that its incidence be reported to the appropriate public health authorities with dispatch. As mentioned, the disease usually occurs with sporadic cases but, from time to time, can occur in geographical clusters which are cause for concern. It is important that primary care physicians be aware of the procedures for management of the sporadic case. This is particularly important in Nova Scotia at this time, as the Province is suffering under a significant shortage of Medical Health Officers.

Sporadic cases of invasive meningococcal disease require aggressive contact tracing for the purposes of identifying persons at increased risk of acquiring the disease. This contact tracing and administration of appropriate antimycrobials to individuals at risk is the basis for the prevention of secondary cases. Chemoprophylaxis will prevent further transmission to suspectible individuals, abort an infection and eradicate colonization in those who may be carrying the organism. As treatment of the disease does not eliminate carriage of the organism in those that are ill, it is important that the index case receive appropriate chemoprophylaxis prior to being discharged from hospital.

Individuals who have had close contact with cases of meningococcal disease are at increased risk of acquiring the disease. *Close contact* is defined as individuals who are likely to have had contact with the oral/nasal secretions of a case of disease. These would include family members living in the residence of the case, individuals in day care or nursery school where a case has occurred, and sexual contacts or others who may have been exposed to the oral secretions of the case. Increased risk of secondary cases has not been demonstrated for those who have had casual contact with sporadic cases. In the health care setting, those health care workers who have had intimate contact with the nasal pharyngeal secretions of a case require prophylaxis. In some instances where travellers have been in close contact with an index case, chemoprophylaxis may be prescribed for some individuals.

Determining the list of potential contacts of an index case and the advisability of prescribing chemoprophylaxis for these individuals, is a critical aspect of the management of sporadic cases. This information should be collected as soon as possible after the diagnosis of the index case. Physicians should be fully knowledgeable of the processes involved. It is recommended that the follow up and decisions regarding prophylaxis treatment should be made in close contact with the medical health officer and/or the provincial epidemiologist.

Either Rifampin orally every 12 hours for a total of four doses, or a single intramuscular dose of ceftriaxone are acceptable drugs for chemoprophylaxis. The dose of Rifampin is 10 milligrams per kilogram per dose for a maximum of 600 milligrams. Some experts recommend the dose be reduced to five milligrams per kilo for infants younger than one month. Although resistance to Rifampin has been reported, it has not yet been shown to be of practical public health importance. Because the safety of Rifampin has not been established in pregnancy, ceftriaxone should be used in pregnant women. The

recommended dose of ceftriaxone is a single intramusular injection 250 milligrams for adults and 125 milligrams for children 15 years of age. The drug should be diluted with 1% lidocane to reduce pain at the injection site.

The choice of drug to be used in any individual depends on the circumstances at the time including the availability of the drug, the number and ages of contacts to be treated, availability of personnel to administer injections and other logistical considerations. Although sulphadiazine has been shown to be effective, it should not be used as a first line prophylaxis unless the strain is known to be sensitive because of the relatively high prevalence of resistant strains.

It should be noted that nasalpharyngeal cultures have no role in the management of cases and contacts.

The management of outbreaks of invasive meningococcal disease is a public health responsibility, although primary care physicians should be knowledgeable of what is involved, particularly at this time in Nova Scotia where Medical Officers of Health are in very short supply.

The proper management of an outbreak requires a process of active surveillance where there is daily reporting of new cases. Complete notification of cases is essential in these situations. Appropriate epidemiological data is required on each case, particularly with respect to age, sex, place of residence, recent travel, attendance or employment at a day care or school, participation in recent athletic or recreational events/recent gatherings. Microbiological data is required for serological grouping of isolates. This information is critical if appropriate decisions regarding control are to be made.

Control measures include expansion of the use of chemoprophylaxis agents in a community where the outbreak is occurring. The use of meningococcal vaccine is appropriate when the outbreak is caused by one of the sero groups contained in the vaccine. The currently available quadrivalent vaccine contains 50 micrograms of each of groups A, C, Y and W135 polysaccharide. The use of a vaccine in a population and delineating high risk groups for which it may be effective, is generally a public health responsibility. If the incidence in a defined group exceeds the acceptable incidence of meningococcal disease and if there is evidence of active spread, the use of the meningococcal vaccine is generally justified. The efficacy of the vaccine differs, depending upon the subgroup and the age of the person being protected. For those under the age of two years, the use of group C meningococcal vaccine is controversial as there is very little published evidence of efficacy in this age group. However, in outbreak situations, the vaccine is usually recommended for children over the age of six months. Physicians may receive inquiries from individuals who are travelling to areas of Canada where meningococcal incidence is increased. Generally, immunization of these individuals is not recommended unless they are planning to remain greater than three weeks in an area where meningococcal vaccination if being used as a control strategy.

In summary, invasive meningococcal disease remains a significant disease from both a clinical and public health perspective. It is important that primary care physicians be knowledgeable of the management sporadic cases as well as understand appropriate procedures to follow when cases occur in clusters in a community. A full range of knowledge by practising physicians of this disease will enable them to provide appropriate and sound advice to the public and hopefully reduce the great anxiety which occurs around this disease. □

Editors Note: The material outlined in this report was obtained from the Canada Diseases Weekly Report, Volume 17-45, published by Health and Welfare Canada, November 9, 1991.

"There is something fascinating about science. One gets such wholesale returns of conjecture out of such a trifling investment of fact."

– Mark Twain (1835-1910)

Appreciations

DR. HAROLD ROSS MACKEAN

Dr. Harold MacKean was born in Tony Mills, Pictou County, in August 1910, the son of a Presbyterian Minister. From the beginning of his boyhood student years in Pictou and Shelburne, his brilliant mind and memory were in evidence. He studied Medicine at Dalhousie University, Nova Scotia, graduating M.D. in 1934, a gold medal winner in his year.

After graduating he took up General Practice in Millertown Newfoundland, where he met and later married Dorothy Phillips, a dedicated senior SRN, and lifelong support to Dr. MacKean in his future career as Eye and Ear Specialist.

Pre-war, he carried out postgraduate study and training in ENT in Edinburgh University, Scotland, returning as FRCS to set up EENT practice in Truro, Nova Scotia. He joined the RCAMC as ENT specialist serving with distinction overseas.

In 1946 he returned to his practise in Truro, working selflessly and single-handed for the counties of Pictou, Cumberland, Colchester and East Hants, helping to establish a 6-bed EENT unit in New Glasgow. During a long professional life of dedicated care of thousands of patients, he earned the well deserved esteem of the citizens of Truro and the Colchester Hospital. He held office as Chief of Staff of the Colchester Hospital, and President of the Colchester East Hants Branch of the Medical Society of Nova Scotia.

A quiet-spoken unassuming man, he enjoyed ornithology and travel after his retirement. He died in September after a long illness, which he bore bravely. He is survived by his four daughters, Libby, Barbara, Kathi and Heather, and three grandchildren.

Dr. Malcolm D. Scott, Brookfield, N.S.

DR. DOUGLAS REID NORMAN

Douglas Reid Norman was a caring and generous person.

He was born in Port Union, Newfoundland and his early years of education were spent in a one-room school house. It was only in High School that he was introduced to a somewhat larger school. From there he went on to Memorial University in St. John's to begin his undergraduate studies. He then came to Halifax and continued his education at Dalhousie's School of Medicine, where he graduated with his Doctor of Medicine degree in 1974.

After graduation, he and his wife Hedy moved back to Newfoundland. There he worked for the Government of Newfoundland as a general practitioner at Markland Cottage Hospital in Whitbourne. Douglas served as the chief medical officer at Markland for ten years, after which time he and his wife and daughter moved to Canning, Nova Scotia.

Douglas practised medicine in the Canning and Kentville area until his sudden death on November 20, 1991. He is survived by his wife Hedy and daughter Andrea Maria, as well as sister Sarah Callahan and four brothers Allan, Albert, James and Barry all of Newfoundland. He was predeceased by his father, Jordan Norman, and his mother Barbara who is still living in Newfoundland.

Throughout his career Douglas worked tirelessly with others for what he believed in. In Whitbourne it was for continuing medical services to outlying districts such as Markland, whose cottage hospitals were being closed one by one. And in Kentville by becoming involved with the Valley Health Services Association and the Department of Emergency Medicine amongst others. He tried to play an active and constructive role whenever he was asked to serve on a committee, and his attitude towards colleagues and patients alike was one of quiet and serious dedication.

Douglas was a devoted father and husband who somehow found time for family and friends, as well as many and varied hobbies such as gardening, wood-working, photography, music and travel. His greatest enjoyment was a deep appreciation for the beauty and wonder of nature.

Douglas died at the early age of forty-two and he will be sadly missed by his loving family and friends for he touched the lives of many in his gentle and caring way.

DR. CHARLES J. DAVID

July 29, 1937 - May 21, 1991

The members of The Medical Society of Nova Scotia and all of his colleagues at the Victoria General Hospital deeply regret the passing of Charles David last summer of cancer.

Dr. David came to Halifax in 1969 after training in the United Kingdom and Chicago. He joined the Department of Psychiatry at the Victoria General Hospital where he was an exemplary clinician and teacher with active research interests for the next 22 years. During his tenure, he was Clinical Director of the Psychiatric Unit at the Victoria General Hospital. He was a past president of the Nova Scotia Psychiatric Association and Provincial Director for the Canadian Psychiatric Association. He was a Fellow of the Royal College of Canada and in 1982 was awarded a "Certificate of Merit" by the Nova Scotia Mental Health Association.

He had a very active career, teaching and practising throughout Nova Scotia, while being very much in demand by his G.P. colleagues for clinical update sessions. He loved his work and had the respect and admiration of his colleagues for his dedication and expertise. In his spare time, he was an active amateur painter and jazz afficionado. He also found the time to help his wife Yasmin raise four wonderful children, Chamin, Farah, Saira and Ariz.

We will all miss him greatly, but his achievements and inspiration will not soon be forgotten.

Dr. M. Ellis, President, Dartmouth Branch Society.

BuSpar*
(buspirone HCl)

A New Class of Anxiolytic for Todays Active Patients.

THERAPEUTIC CLASSIFICATION
Antianxiety Agent

INDICATIONS AND CLINICAL USE
Short term symptomatic relief of excessive anxiety in patients with generalized anxiety disorder (psychoneurotic disorder).

Eight three-way, short term, controlled clinical trials involving buspirone, diazepam and placebo are considered central to the evaluation of buspirone as an anxiolytic agent. In four of the eight clinical trials, buspirone demonstrated a significant difference from placebo. In the other four trials, there was no significant difference between buspirone and placebo, but a significantly greater improvement was observed with diazepam than with placebo. The adverse effect profiles of buspirone and diazepam in these clinical trials were, however, different.

CONTRAINDICATIONS
BuSpar (buspirone hydrochloride) is contraindicated in patients hypersensitive to buspirone hydrochloride.

BuSpar is contraindicated in patients with severe hepatic or severe renal impairment.

WARNINGS
The occurrence of elevated blood pressure in patients receiving both BuSpar (buspirone hydrochloride) and a monoamine oxidase inhibitor (MAOI) has been reported. Therefore, it is recommended that buspirone should not be used concomitantly with a MAOI.

Since buspirone can bind to central dopaminergic receptors, the possibility of acute and chronic changes in dopamine mediated neurological function (e.g. dystonia, pseudo-parkinsonism, akathisia and tardive dyskinesia) should be considered. (SEE PRECAUTIONS)

Since the effects of buspirone have not been evaluated in patients with a history of convulsive disorders and since it lacks anticonvulsant activity in animals, buspirone is not recommended for patients with seizure disorders.

Use of Buspirone in Patients Previously Treated with a Benzodiazepine: Patients who have previously taken benzodiazepines may be less likely to respond to buspirone than those who have not. In two clinical studies to date, substitution of buspirone did not ameliorate or prevent withdrawal symptoms in either abrupt or gradual withdrawal from various benzodiazepines following long-term use. Therefore, if it is considered desirable to switch a patient who has been receiving benzodiazepine therapy to buspirone, the benzodiazepine should first be withdrawn gradually. A drug-free interval is desirable between withdrawal of the benzodiazepine and initiation of buspirone, in order to increase the likelihood of distinguishing between benzodiazepine withdrawal effects and unrelieved anxiety due to possible failure of buspirone in this category of patients.

Benzodiazepine rebound or withdrawal symptoms may occur over varying time periods depending in part on the type of drug and its effective half-life of elimination. These symptoms may appear as any combination of irritability, anxiety, agitation, insomnia, tremor, abdominal cramps, muscle cramps, vomiting, sweating, flu-like symptoms without fever and, occasionally, seizures, and should be treated symptomatically.

Use in Pregnancy and Lactation: The safety of buspirone during pregnancy and lactation has not been established and, therefore, it should not be used in women of childbearing potential or nursing mothers, unless, in the opinion of the physician, the potential benefits to the patient outweigh the possible hazards to the fetus. Buspirone and its metabolites are excreted in milk in rats. The extent of excretion in human milk has not yet been determined.

PRECAUTIONS
Effects on Cognitive and Motor Performance: In controlled studies in healthy volunteers, single doses of buspirone up to 20 mg had little effect on most tests of cognitive and psychomotor function, although performance on a vigilance task was impaired in a dose-related manner. The effect of higher single doses of buspirone on psychomotor performance has not been investigated.

Ten (10) mg of buspirone given three times daily for seven days to healthy volunteers produced considerable subjective sedation but no significant effect on psychomotor performance (no vigilance tasks were used in this study). It also caused transient dizziness, especially on standing and walking.

Until further experience is obtained with buspirone, patients should be warned not to operate an automobile or undertake activities requiring mental alertness, judgement and physical coordination, until they are reasonably certain that buspirone does not affect them adversely.

Significant Interactions: In laboratory studies in healthy volunteers, buspirone in doses up to 20 mg did not potentiate the psychomotor impairment produced by relatively modest doses of alcohol. However, decreased contentedness or dysphoria was observed with a combination of alcohol and a 20 mg single dose of buspirone. Since no data are available on concomitant use of higher doses of buspirone and alcohol, it is prudent to advise patients to avoid alcohol during buspirone therapy.

Food increased the bioavailability of unchanged buspirone in healthy subjects, possibly due to a reduced first-pass effect.

Concomitant use of monoamine oxidase inhibitors and buspirone has been reported to cause an increase in blood pressure. Therefore, concomitant use of these medications is not recommended.

In a study in normal volunteers, no interaction of buspirone with amitriptyline was seen. A similar study with diazepam showed an increase in the levels of nordiazepam.

In another study in normal volunteers, concomitant administration of buspirone and haloperidol resulted in increased serum haloperidol concentrations. The clinical significance of this finding is not clear.

There is one report suggesting that the concomitant use of trazodone and buspirone may have caused 3- to 6-fold elevations in SGPT (ALT) in a few patients. In a similar study, attempting to replicate this finding, no interactive effect on hepatic transaminases was identified.

Because the effects of concomitant administration of buspirone with most other psychotropic drugs have not been studied, the concomitant use of buspirone with other CNS active drugs should be approached with caution.

In vitro, buspirone does not displace tightly bound drugs like phenytoin, propranolol and warfarin from serum proteins. However, there has been one report of prolonged prothrombin time when buspirone was added to the regimen of a patient treated with warfarin. The patient was also chronically receiving phenytoin, phenobarbital, digoxin and Synthroid. In vitro, buspirone may displace less firmly bound drugs like digoxin. The clinical significance of this property is unknown.

There have been no reports to date of interference of buspirone with commonly employed clinical laboratory tests.

Drug Abuse and Dependence: Although preliminary animal and human investigations suggest that buspirone may be significantly devoid of potential for producing physical or psychological dependence, only extensive clinical experience with the drug will provide conclusive evidence. Meanwhile, physicians should carefully evaluate patients for a history of drug abuse and follow such patients closely, observing them for signs of buspirone misuse and abuse.

Use in Patients with Impaired Hepatic or Renal Function: Since it is metabolized by the liver and excreted by the kidneys, buspirone should be used with caution in patients with a history of hepatic or renal impairment. It is contraindicated in patients with severe hepatic or renal impairment.

Use in Children: The safety and effectiveness of buspirone in individuals below the age of 18 years have not been established.

Use in the Elderly: Buspirone has not been systematically evaluated in older patients. Although it would appear from limited pharmacokinetic and clinical studies that buspirone does not behave differently in the elderly, there is little known about the effects of buspirone in this age group at doses above 30 mg/day. Therefore, it is recommended that buspirone should be used in the elderly at doses not exceeding 30 mg/day for a duration not exceeding 4 weeks.

Neuroendocrine Effects: Single doses of 30 mg or higher of buspirone resulted in significantly elevated plasma prolactin and growth hormone concentrations in normal volunteers. No effect was seen at lower doses. In another study, no such increases were observed after buspirone was administered in divided doses (10 mg t.i.d.) for 28 days.

Possible Concerns Related to Buspirone's Binding To Dopamine Receptors: Because buspirone can bind to central dopamine receptors, a question has been raised about its potential to cause acute and chronic changes in dopamine mediated neurological function (e.g., dystonia, pseudo-parkinsonism, akathisia, and tardive dyskinesia). Clinical experience in controlled trials has failed to identify any significant neuroleptic-like activity; however, a syndrome of restlessness, appearing shortly after initiation of treatment, has been reported in some small fraction of buspirone treated patients. The syndrome may be explained in several ways. For example, buspirone may increase central noradrenergic activity; alternatively, the effect may be attributable to dopaminergic effects (i.e., represent akathisia). Obviously, the question cannot be totally resolved at this point in time. Generally, long-term sequelae of any drug's use can be identified only after several years of marketing.

ADVERSE REACTIONS
The most common adverse reactions encountered with BuSpar (buspirone hydrochloride) are dizziness, headache, drowsiness and nausea. During premarketing clinical trials, approximately 10% of the patients discontinued treatment due to an adverse event.

Adverse reactions reported include the following:

CNS: Dizziness, headache, drowsiness, lightheadedness, insomnia, fatigue, nervousness, decreased concentration, excitement, depression, confusion, nightmares/vivid dreams, anger/hostility. Infrequently (<1%) depersonalization, noise intolerance, euphoria/feeling high, dissociative reaction, fear, loss of interest, dysphoria, hallucinations, seizures, suicidal thoughts. Rarely, slurred speech, claustrophobia, cold intolerance, stupor, psychosis.

Neurologic: Paresthesia, weakness, incoordination, tremor, numbness. Infrequently, muscle cramps and spasms, rigid/stiff muscles, involuntary movements, akathisia, slowed reaction time. Rarely, tingling of limbs, stiff neck, rigidity of jaw, ataxia.

Autonomic: Dry mouth, sweating/clamminess, blurred vision, constipation. Infrequently, urinary frequency, retention and burning, flushing.

Cardiovascular: Tachycardia, chest pain, palpitations. Infrequently, syncope, hypotension, hypertension. Rarely, congestive heart failure, cerebrovascular accident, myocardial infarction, cardiomyopathy, bradycardia, EKG change.

Gastrointestinal: Nausea, G.I. distress, diarrhea, vomiting. Infrequently, flatulence, increased appetite, anorexia, hypersalivation, rectal bleeding, irritable colon. Rarely, burning tongue.

Respiratory: Nasal congestion. Infrequently, shortness of breath, chest congestion, difficulty breathing, hyperventilation. Rarely, epistaxis.

Endocrine: Infrequently, decreased and increased libido, weight gain, weight loss, menstrual irregularity/breakthrough bleeding. Rarely, delayed ejaculation, impotence, galactorrhea, amenorrhea, thyroid abnormality.

Allergic or Toxic: Skin rash, sore throat. Infrequently, edema/facial edema, pruritus, chills/fever. Rarely, photophobia, erythema, flu-like symptoms.

Clinical Laboratory: Infrequently, increases in liver enzymes. Rarely, eosinophilia, leukopenia, thrombocytopenia.

Miscellaneous: Tinnitus, muscle aches/pains. Infrequently, redness/itching of eyes, altered taste/smell, roaring sensation in head, malaise, easy bruising, dry skin, arthralgia, blisters, hair loss. Rarely, acne, thinning of nails, sore eyes, inner ear abnormality, pressure on eyes, nocturia, enuresis, hiccups, voice loss, alcohol abuse.

Post Introduction Clinical Experience: Post-marketing experience in the United States has shown an adverse experience profile similar to that given above. Additional reports have included rare occurrences of allergic reaction, cogwheel rigidity, dystonic reaction, ecchymosis, emotional lability and tunnel vision. Because of the uncontrolled nature of these spontaneous reports, a causal relationship to buspirone treatment has not been determined.

SYMPTOMS AND TREATMENT OF OVERDOSAGE
Symptoms: In clinical pharmacology trials, BuSpar (buspirone hydrochloride) up to 400 mg/day was administered to healthy male volunteers. As this dose was approached, the following symptoms were observed in descending order of frequency: drowsiness, ataxia, nausea and vomiting, dizziness, clammy feeling, difficulty thinking, feeling "high", "rushing" sensation, gastric distress, headache, itching, miosis, hypotension, tremor, incoordination, insomnia and hallucinations. In a dose ranging study in acute psychotic patients, up to 2400 mg/day was administered. Dizziness, nausea and vomiting were the most common adverse effects. One patient developed extrapyramidal symptoms at 600 mg/day.

Treatment: There is no specific antidote for buspirone. Management should, therefore, be symptomatic and supportive. Any patient suspected of having taken an overdose should be admitted to a hospital as soon as possible, and the stomach emptied by gastric lavage. Respiration, pulse and blood pressure should be monitored, as in all cases of drug overdosage. As with the management of intentional overdosage with any drug, the ingestion of multiple agents should be suspected. In six anuric patients, hemodialysis either had no effect on the pharmacokinetics of buspirone or decreased its clearance.

DOSAGE AND ADMINISTRATION
BuSpar (buspirone hydrochloride) dosage should be individually adjusted, according to tolerance and response.

The recommended initial dose is 5 mg two to three times daily. This may be titrated according to the needs of the patient and the daily dose increased by 5 mg increments every two or three days up to a maximum of 45 mg daily in divided doses. The usual therapeutic dose is 20 to 30 mg daily in two or three divided doses.

Elderly Patients: Limited pharmacokinetic and clinical data have shown no difference in the effects of buspirone between elderly patients and healthy adult volunteers. However, until more information has accumulated in the elderly, it is recommended that the maximum daily dose should not exceed 30 mg for a duration not exceeding 4 weeks.

Note: If buspirone is administered to patients with compromised hepatic or renal function, careful monitoring will be required together with appropriate dosage adjustment.

AVAILABILITY
BuSpar (buspirone hydrochloride) Tablets, 10 mg, are white tablets. Bottles of 100.

Product Monograph available upon request.

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†Until further experience is obtained with buspirone, patients should be warned not to operate an automobile or undertake activities requiring mental alertness, judgement or physical co-ordination until they are reasonably certain that buspirone does not affect them adversely.

††Since no data is available on concomitant use of higher doses of buspirone and alcohol, it is prudent to advise patients to avoid alcohol during buspirone therapy.

*T.M. Authorized user, Bristol-Myers Squibb Canada Inc.

PAAB
PMAC

BRISTOL
A division of Bristol-Myers Squibb Canada Inc.
2365 Côte-de-Liesse
Montréal, Québec H4N 2M7

P One a day Oruvail®

Sustained-release ketoprofen capsules 150 mg and 200 mg

Anti-inflammatory analgesic agent

ACTION and CLINICAL PHARMACOLOGY: Animal pharmacological studies have shown that ketoprofen possesses anti-inflammatory, analgesic and antipyretic properties. The anti-inflammatory action is not mediated through the pituitary adrenal axis. Its therapeutic effectiveness has been demonstrated by a reduction in joint swelling, pain and duration of morning stiffness, and by increased grip strength and an improvement in functional capacity. Clinical trials in patients with rheumatoid arthritis and osteoarthritis have shown that when given in a dose of 200 mg once daily, the anti-arthritic activity of Oruvail is comparable to that of a twice daily administration of ketoprofen (100 mg ketoprofen b.i.d.). Ketoprofen 200 mg daily induced less gastrointestinal bleeding than acetylsalicylic acid 4 g/day. **Pharmacokinetics properties:** Ketoprofen from Oruvail is slowly but almost completely absorbed from the gastrointestinal tract. Mean peak plasma levels of 2.2 and 4.2 mg/L are achieved about 5 hours following single oral doses of Oruvail 100 and 200 mg, respectively. Pharmacokinetics are linear over a dosage range of 100 to 200 mg. The systemic availability of Oruvail is 95% of that of conventional capsules. In a food-effect study, meal composition did not affect the extent of absorption of ketoprofen from Oruvail, although a heavy meal slightly but significantly delayed the absorption of the drug by about 2 hours by comparison to a light meal; in this study, there was no comparison with the fasted state nor with a conventional ketoprofen formulation. Steady-state plasma ketoprofen concentrations are achieved within 4 days with mean peak and trough levels of 4.3 and 0.91 mg/L respectively, after repeated doses of 200 mg once daily. There is some evidence that C_{max} and bioavailability are increased in the elderly as the result of an age-related reduction in volume of distribution since the apparent elimination half-life of about 8 hours is similar in both young and elderly patients. No or negligible accumulation of ketoprofen was found following repeated once daily dosing of Oruvail 200 mg capsules in either young or aged subjects. In arthritic patients treated with Oruvail 200 mg once daily for up to 3 months, the steady-state disposition of ketoprofen remains unaltered during chronic administration. When comparing to a group of healthy subjects, no differences with respect to AUC, C_{max}, and elimination half-life were found, indicating that inflammatory joint disease has no influence on the kinetics of Oruvail capsules.

INDICATIONS and CLINICAL USES: Oruvail (ketoprofen) is indicated in the treatment of rheumatoid arthritis, ankylosing spondylitis and osteoarthritis.

CONTRAINDICATIONS: Oruvail (ketoprofen) is contraindicated in patients with active peptic ulcers or active inflammatory diseases of the gastrointestinal tract. Oruvail is also contraindicated in patients who have demonstrated hypersensitivity to the drug. Because of cross-sensitivity, ketoprofen should not be given to patients in whom acetylsalicylic acid (ASA) and other nonsteroidal anti-inflammatory drugs induce symptoms of asthma, rhinitis or urticaria. Fatal anaphylactoid reactions have occurred in such individuals.

WARNINGS: Peptic ulceration, perforation and gastrointestinal bleeding, sometimes severe and occasionally fatal have been reported during therapy with nonsteroidal anti-inflammatory drugs (NSAID's), including Oruvail (ketoprofen). Unlike most adverse reactions, which usually manifest themselves in the first month if they are going to occur in an individual, new peptic ulcers keep appearing in patients under treatment with ketoprofen at a rate of greater than 1% per year. Oruvail should be given under close medical supervision to patients prone to gastrointestinal tract irritation particularly those with a history of peptic ulcer, diverticulosis or other inflammatory disease of the gastrointestinal tract. In these cases the physician must weigh the benefits of treatment against the possible hazards. Patients taking any NSAID including this drug should be instructed to contact a physician immediately if they experience symptoms or signs suggestive of peptic ulceration or gastrointestinal bleeding. These reactions can occur without warning symptoms or signs and at any time during the treatment. Elderly, frail and debilitated patients appear to be at higher risk from a variety of adverse reactions from nonsteroidal anti-inflammatory drugs (NSAID's). For such patients, consideration should be given to a starting dose lower than usual, with individual adjustment when necessary and under close supervision. See "Precautions" for further advice. **Use in Pregnancy:** The safety of Oruvail when administered to pregnant or nursing women has not been determined and therefore such use is not recommended. Pregnant rats who received ketoprofen 6 and 9 mg/kg/day p.o. from day 15 of gestation, showed dystocia and increased pup mortality. **Nursing mothers:** In rats, ketoprofen at doses of 9 mg/kg (approximately 1.5 times the maximum human therapeutic dose) did not affect perinatal development. Upon administration to lactating dogs, the milk concentration of ketoprofen was found to be 4 to 5% of the plasma drug level. Data on secretion in human milk after ingestion of ketoprofen do not exist. As with other drugs that are excreted in milk, Oruvail is not recommended for use in nursing mothers. **Use in Children:** The conditions for safe and effective use of Oruvail in children under 12 years of age have not been established and the drug is therefore not recommended in this age group.

PRECAUTIONS: Gastrointestinal system: If peptic ulceration is suspected or confirmed, or if gastrointestinal bleeding or perforation occurs Oruvail (ketoprofen) should be discontinued, an appropriate treatment instituted and patient closely monitored. There is no definitive evidence that the concomitant administration of histamine H_2-receptor antagonists and/or antacids will either prevent the occurrence of gastrointestinal side effects or allow continuation of Oruvail therapy when and if these adverse reactions appear. **Renal function:** As with other nonsteroidal anti-inflammatory drugs, long-term administration of ketoprofen to animals has resulted in renal papillary necrosis and other abnormal renal pathology. In humans, there have been reports of acute interstitial nephritis with hematuria, proteinuria, and occasionally nephrotic syndrome. A second form of renal toxicity has been seen in patients with prerenal conditions leading to the reduction in renal blood flow or blood volume, where the renal prostaglandins have a supportive role in the maintenance of renal perfusion. In these patients, administration of a nonsteroidal anti-inflammatory drug may cause a dose-dependent reduction in prostaglandin formation and may precipitate overt renal decompensation. Patients at greatest risk of this reaction are those with impaired renal function, heart failure, liver dysfunction, those taking diuretics, and the elderly. Discontinuation of nonsteroidal anti-inflammatory therapy is usually followed by recovery to the pre-treatment state. Ketoprofen and its metabolites are eliminated primarily by the kidneys, therefore the drug should be used with great caution in patients with impaired renal function. In these cases lower doses of Oruvail should be anticipated and patients carefully monitored. During long-term therapy kidney function should be monitored periodically. **Hepatic function:** As with other nonsteroidal anti-inflammatory drugs, borderline elevations of one or more liver tests may occur in up to 15% of patients. These abnormalities may progress, may remain essentially unchanged, or may be transient with continued therapy. Meaningful (3 times the upper limit of normal) elevations of ALT or AST occurred in controlled clinical trials in less than 1% of patients. A patient with symptoms and/or signs suggesting liver dysfunction, or in whom an abnormal liver test has occurred, should be evaluated for evidence of the development of more severe hepatic reaction while on therapy with this drug. Severe hepatic reactions including jaundice and cases of fatal hepatitis have been reported with this drug as with other nonsteroidal anti-inflammatory drugs. Although such reactions are rare, if abnormal liver tests persist or worsen, if clinical signs and symptoms consistent with liver disease develop, or if systemic manifestations occur (e.g. eosinophilia, rash, etc.), this drug should be discontinued. During long-term therapy, liver function tests should be monitored periodically. If this drug is to be used in the presence of impaired liver function, it must be done under strict observation. **Fluid and Electrolyte Balance:** Fluid retention and edema have been observed in approximately 2% of patients treated with ketoprofen. Therefore, as with many other nonsteroidal anti-inflammatory drugs, the possibility of precipitating congestive heart failure in elderly patients or those with compromised cardiac function should be born in mind. Oruvail should be used with caution in patients with heart failure, hypertension or other conditions predisposing to fluid retention. Serum electrolytes should be monitored periodically during long-term therapy, especially in those patients at risk. **Hematology:** Drugs inhibiting prostaglandin biosynthesis do interfere with platelet function to some degree; therefore, patients who may be adversely affected by such an action should be carefully observed when Oruvail is administered. Blood dyscrasias associated with the use of nonsteroidal anti-inflammatory drugs are rare, but could be with severe consequences. Anemia is commonly observed in rheumatoid arthritis and is sometimes aggravated by nonsteroidal anti-inflammatory drugs, which may produce fluid retention or minor gastrointestinal blood loss in some patients. Therefore, patients with initial hemoglobin values of 10 g/dL or less who are to receive long-term therapy should have hemoglobin values determined frequently. **Infection:** In common with other anti-inflammatory drugs, Oruvail may mask the usual signs of infection. **Ophthalmology:** Blurred and/or diminished vision has been reported with the use of ketoprofen and other nonsteroidal anti-inflammatory drugs. If such symptoms develop this drug should be discontinued and an ophthalmologic examination performed; ophthalmic examination should be carried out at periodic intervals in any patient receiving this drug for an extended period of time. **Drug Interactions: Methotrexate:** The concomitant administration of ketoprofen and high-dose methotrexate has been associated with prolonged and marked enhancement of serum methotrexate levels resulting in severe methotrexate toxicity. This may also apply to some other nonsteroidal anti-inflammatory drugs. There were no abnormalities in methotrexate kinetics or evidence of toxicity when ketoprofen was given at least 12 hours after completion of high-dose methotrexate infusion. ORUVAIL should not be used in patients receiving high dose methotrexate. The potential for severe toxicity should be kept in mind when prescribing ketoprofen and low-dose methotrexate concurrently. ORUVAIL should not be administered within 12 hours of methotrexate infusion. **Acetylsalicylic acid (ASA):** concurrent administration of ASA decreased ketoprofen protein binding and increased its plasma clearance. The overall result was a 40% reduction in the AUC of ketoprofen. **Oral anticoagulants:** Ketoprofen has been shown to depress platelet aggregation and it can prolong bleeding time by approximately 3 to 4 minutes from baseline values. However, a study conducted in twenty patients undergoing therapy with coumarin and simultaneously receiving ketoprofen, failed to demonstrate potentiation of anticoagulant effect. Nevertheless, close monitoring of patients is recommended when Oruvail is given concomitantly with anticoagulants. **Diuretics:** hydrochlorothiazide, given concomitantly with ketoprofen, produces a reduction in urinary potassium and chloride excretion compared to hydrochlorothiazide alone. Patients taking diuretics are at greater risk of developing renal failure secondary to a decrease in renal blood flow caused by prostaglandin inhibition. **Antacids:** concomitant administration of magnesium hydroxide and aluminum hydroxide does not interfere with the rate or extent of the absorption of ketoprofen. **Lithium:** nonsteroidal anti-inflammatory agents have been reported to increase steady-state plasma lithium levels. It is recommended that plasma lithium levels be monitored when Oruvail is coadministered with lithium. **Probenecid:** concurrent administration of probenecid increases both free and bound ketoprofen through reducing the plasma clearance of ketoprofen to about one-third as well as decreasing its protein binding. ORUVAIL is not recommended in association with probenecid. Ketoprofen is extensively (99%) protein bound to human serum albumin and may compete for binding sites with drugs such as sulfonamides, oral hypoglycemic agents, phenytoin or lithium. Although no significant interaction has been documented, patients with such combination therapy should be monitored. **Clinical Laboratory Test:** The presence of ketoprofen and its metabolites in urine has been shown to interfere with certain tests which are used to detect albumin, bile salts, 17-ketosteroids or 17-hydroxycorticosteroids in urine and which rely upon acid precipitation as an end point or upon colour reactions for carbonyl groups. No interference was seen in the tests for proteinuria using Albustix, Hema-Combistix or Labstix Reagent Strips. Ketoprofen decreases platelet adhesion and aggregation. Therefore, it can prolong bleeding time by approximately 3 to 4 minutes from baseline values. There is no significant change in platelet count, prothrombin time, partial thromboplastin time, or thrombin time.

ADVERSE REACTIONS: Gastrointestinal: Gastrointestinal effects were the most frequently observed adverse reactions and were seen in approximately 13% of patients receiving Oruvail (ketoprofen). Ulceration and gastrointestinal bleeding have been observed in a few patients receiving Oruvail therapy (approximately 0.3%). Other adverse reactions in order of decreasing frequency were: gastrointestinal pain, dyspepsia, constipation, nausea and/or vomiting, diarrhea and flatulence. Such symptoms led to the discontinuation of treatment in 6.8% of patients. **Central Nervous System:** Central nervous system adverse reactions were next in frequency and included headache, fatigue, drowsiness, dizziness, depression, restlessness and nightmares. **Skin:** rash, eczema, flushing, pruritus, sweating and loss of hair were occasionally observed. **Allergic:** These were seen infrequently and included urticaria, angioedema and asthma. **Cardiovascular:** Mild peripheral edema, palpitation, bruising, arrhythmia, chest pain and exacerbation of circulatory disturbances were reported. **Auditory:** Tinnitus and deafness were reported on rare occasions. **Mouth:** The following symptoms were reported: dry mouth, mouth ulcers, sore tongue and inflammation of the mouth and gums. **Laboratory tests:** Abnormal alkaline phosphatase lactic dehydrogenase, glutamic oxaloacetic transaminase and blood urea nitrogen values were found in some patients receiving ketoprofen therapy. The abnormalities did not lead to discontinuation of treatment and, in some cases, returned to normal despite continuation of the drug. There have been sporadic reports of decreased hematocrit and hemoglobin values without progressive deterioration on prolonged administration of the drug.

SYMPTOMS AND TREATMENT OF OVERDOSE: Symptoms: Of 20 cases of overdosage (up to 5,000 mg) reported in Great Britain (5 children, 14 adolescents or young adults, and 1 elderly), only 4 had mild symptoms (vomiting in 3, drowsiness in 1 child). **Treatment:** Administer gastric lavage or an emetic and treat symptomatically: compensate for dehydration, monitor urinary excretion and correct acidosis if present. The drug is dialyzable; therefore, hemodialysis may be useful to remove circulating drug and to assist in case of renal failure.

DOSAGE AND ADMINISTRATION: Adults: The usual dosage is 150 to 200 mg once daily. The capsules should be taken with food and can be administered in the morning or evening. **Elderly and debilitated patients:** The dosage should be reduced in patients with impaired renal function and the elderly. The lower strength should be used in those cases. **Children:** Oruvail is not indicated in children under 12 years of age because clinical experience in this age group is insufficient. **Composition:** Non medicinal ingredients: colloidal silicone dioxide, ethyl cellulose, gelatin, maize starch, shellac, sucrose, talc. Colouring agents: ORUVAIL 150 mg: erythrosine, titanium dioxide. ORUVAIL 200 mg: brilliant blue, erythrosine, titanium dioxide.

AVAILABILITY: Oruvail 150 capsules: each transparent pink capsule with opaque white cap (each half printed "Oruvail 150" in black) contains ketoprofen 150 mg as white pellets. Available in bottles of 100 and 250. **Oruvail 200 capsules:** each transparent pink capsule with opaque blue cap (each half printed "Oruvail 200" in yellow) contains ketoprofen 200 mg as white pellets. Available in bottles of 100 and 250.

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PRESCRIBING INFORMATION

THERAPEUTIC CLASSIFICATION
Bronchodilator

ACTION – Atrovent (ipratropium bromide), a quaternary ammonium derivative of atropine, is an anticholinergic drug which has bronchodilator properties. On inhalation, the onset of action is noted within 5 to 15 minutes, with a peak response between 1 and 2 hours, lasting about 2 additional hours, with subsequent decline from the peak. Bronchodilation is still evident 8 hours after inhalation.
In acute and maintenance therapy of chronic reversible airways obstruction, Atrovent has been shown to provide additive bronchodilating effects to theophylline and beta-adrenoceptor agonists (sympathomimetic amines). Repeated inhalation of Atrovent has not been linked to tolerance towards bronchodilating effects. Significant alterations in mucociliary clearance of sputum were not observed in short term clinical trials. Systemic absorption of Atrovent is poor and the blood levels reached are very low. Metabolic studies with Atrovent in healthy volunteers show an average elimination half-life of 3.5 hours (range 1.5 to 4 hours). The drug is transformed to some 8 metabolites with little or no anticholinergic activity.

INDICATIONS AND CLINICAL USES – Atrovent (ipratropium bromide) solution is indicated for the therapy of acute exacerbations of chronic bronchitis. Atrovent solution, when used in conjunction with a β_2-adrenergic stimulant solution such as fenoterol or salbutamol, is indicated for acute asthmatic attacks. It is to be administered by compressed air or oxygen driven nebulizers.

CONTRAINDICATIONS – Known hypersensitivity to Atrovent (ipratropium bromide), to any of the product ingredients, or to atropinics.

WARNINGS – Atrovent (ipratropium bromide) solution contains preservatives (benzalkonium chloride and disodium ethylene diamine tetraacetic acid – EDTA-disodium). It has been reported that these preservatives may cause bronchoconstriction in some patients with hyperreactive airways.
Atrovent should not be used alone for the abatement of an acute asthmatic attack since the drug has slower onset of effect than that of an adrenergic β_2 agonist.
Care should be taken to ensure that the nebulizer mask fits the patient's face properly and that nebulized solution does not escape into the eyes. There have been isolated reports of ocular complications (i.e., mydriasis, increased intraocular pressure, angle closure glaucoma) when nebulized ipratropium bromide either alone or in combination with an adrenergic β_2 agonist solution has escaped into the eyes. In the event that glaucoma is precipitated or worsened, treatment should include standard measures for this condition.

PRECAUTIONS

- Patients should be instructed in the proper use of the nebulizer.
- Caution is advised against accidental release of the solution into the eyes.
- In patients with glaucoma, prostatic hypertrophy or urinary retention, Atrovent (ipratropium bromide) should be used with caution.
- If a reduced response to Atrovent becomes apparent, the patient should seek medical advice.
- Atrovent solution, when administered to patients with acute severe asthma, should be used with concomitant β_2-adrenergic stimulant therapy.

Use in Pregnancy: The safety of Atrovent in pregnancy has not been established. The benefits of using Atrovent when pregnancy is confirmed or suspected must be weighed against possible hazards to the fetus. Studies in rats, mice and rabbits showed no embryotoxic nor teratogenic effects.

Use During Lactation: No specific studies have been conducted on excretion of this drug in breast milk. Benefits of Atrovent use during lactation should therefore be weighed against the possible effects on the infant.

Use in Children: The efficacy and safety of Atrovent in children younger than 5 years has not been established.

Use with Other Drugs: In patients receiving other anticholinergic drugs, Atrovent should be used with caution because of possible additive effects.
In patients with glaucoma or narrow anterior chambers, the administration by nebulizer of a combined Atrovent-β_2 agonist solution should be avoided unless measures (e.g., use of swimming goggles) are taken to ensure that nebulized solution does not reach the eye. Exposure of the eyes of such patients to a nebulized combination of Atrovent and a β_2 agonist solution has been reported to result in increased intraocular pressure and/or acute angle closure.
Atrovent solution should not be mixed with sodium cromoglycate. If the patient's condition requires the administration of sodium cromoglycate, it should be given separately. Mixing of Atrovent solution and sodium cromoglycate produces a cloudy solution.

ADVERSE REACTIONS – The frequency of adverse reactions recorded in 214 patients receiving Atrovent (ipratropium bromide) solution was as follows:
Dry mouth or throat, 9.3%; Bad taste, 5.1%; Tremor, 4.2%; Exacerbation of symptoms, 4.2%; Burning eyes, 0.9%; Nausea, 0.9%; Sweating, 0.9%; Cough, 0.9%; Headache, 0.5%; Palpitations, 0.5%.
The adverse effect judged to be most severe was exacerbation of symptoms. This occurred in 8 patients treated with Atrovent solution alone, 6 of whom withdrew from the clinical studies.
Bronchospasm occurred in 3 patients with acute severe asthma who received Atrovent solution alone. In two patients, this was reversed after therapy with a β_2 sympathomimetic solution. The third patient received no other therapy.
The following table compares the incidence of adverse effects of the combination of Atrovent and a β_2 agonist (either fenoterol or salbutamol) solution with that of the β_2 agonist alone.

ADVERSE EFFECT	ATROVENT + β_2 AGONIST (% of 94 patients)	β_2 AGONIST (% of 96 patients)
Tremor	31.9	26.0
Dry mouth	16.0	28.1
Bad taste	16.0	13.5
Vomiting	2.1	2.1
Palpitations	2.1	1.0
Headache	1.1	2.1
Cough	1.1	0.0
Flushing	1.1	0.0
Dizziness	0.0	1.0
Numbness in leg	0.0	1.0

There have been isolated reports of ocular effects such as mydriasis, increased intraocular pressure, and acute glaucoma associated with the escape of nebulized ipratropium bromide–alone or in combination with a β_2 agonist solution into the eyes.

SYMPTOMS AND TREATMENT OF OVERDOSAGE – Doses of Atrovent (ipratropium bromide) up to 1.2 mg (approximately 30 times the therapeutic dose) have been administered by Atrovent inhaler without the appearance of serious systemic anticholinergic effects. Should signs of serious anticholinergic toxicity appear, cholinesterase inhibitors may be considered.

DOSAGE AND ADMINISTRATION – In adults, the average single dose is 1-2 mL of Atrovent (ipratropium bromide) solution, containing 250-500 µg of ipratropium. In children, aged 5-12 years, the recommended dose is 0.5-1 mL (125-250 µg of ipratropium). This should be diluted to 3-5 mL with preservative free sterile Normal Saline (Sodium Chloride Inhalation Solution, USP 0.9%) or with a bacteriostatic sodium chloride solution, 0.9% preserved with benzalkonium chloride (see PHARMACEUTICAL INFORMATION).
Nebulization should take place using a gas flow (oxygen or compressed air) of 6-10 L/minutes and the solution nebulized over a 10-15 minute period. The Hudson Updraft™, Bennett Twin Jet® and Inspiron Mini-Neb® nebulizers, with facemask or mouthpiece have been used. The manufacturers' instructions concerning cleaning and maintenance of the nebulizer should be strictly followed.
Treatment with Atrovent solution may be repeated every 4-6 hours as necessary.

PHARMACEUTICAL INFORMATION – Stability and Storage Recommendations:
Unopened bottles of Atrovent (ipratropium bromide) solution should be stored at controlled room temperature (below 31°C). Solutions diluted with preservative free sterile Sodium Chloride Inhalation Solution, USP 0.9% should be used within 24 hours from time of dilution when stored at room temperature and within 48 hours when stored in the refrigerator. Dilutions may also be made with a bacteriostatic sodium chloride solution 0.9% which contains benzalkonium chloride as the bacteriostatic agent (see WARNINGS). This diluted solution may be stored at room temperature and used within 7 days.
Controlled laboratory experiments using mixtures of Atrovent solution with Alupent® (orciprenaline sulfate), Berotec® (fenoterol hydrobromide), or salbutamol sulfate (6 mg/mL preserved with benzalkonium chloride) solutions and diluted with a sterile bacteriostatic sodium chloride solution 0.9% (i.e., normal saline), preserved with benzalkonium chloride, indicated that such mixtures were stable for 7 days at room temperature. For the preparation of such mixtures, it is recommended that only sterile solutions of bacteriostatic sodium chloride 0.9% preserved with 0.01% benzalkonium chloride be used to maintain the level of preservative in the mixture.
The safety of preservatives other than benzalkonium chloride has not been established.

Incompatibilities: Atrovent solution should not be mixed with sodium cromoglycate solution. If the patient's condition requires the administration of sodium cromoglycate, it should be given separately. Mixing of Atrovent solution and sodium cromoglycate solution produces a cloudy solution.

DOSAGE FORMS
Availability: Atrovent solution is provided as 20 mL clear, colourless or almost colourless solution containing 250 µg/mL (0.025 %) Atrovent in isotonic solution preserved with benzalkonium chloride 250 µg/mL and EDTA-disodium 500 µg/mL at pH 3.4 in an amber glass bottle with screwcap.
The complete Product Monograph for Atrovent (ipratropium bromide) Inhalation Solution is available to health professionals on request. Patient Information/Instructions are provided with the inhalation solution.

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OBITUARIES

Dr. Harold R. McKean (81) of Truro, Nova Scotia died on September 1, 1991. Born in Toney Mills, Pictou County, he received his medical degree from Dalhousie Medical School in 1934 with distinction winning the gold medal. He did postgraduate studies in Edinburgh and served in both the Royal and Canadian Army Medical Corps. He practised in Truro until 1984 and he was nominated a senior member of The Medical Society of Nova Scotia in 1984. He is survived by his four daughters to whom the *Journal* extends sympathy.

Dr. George Shimotakahara, (71) of Antigonish, Nova Scotia died on January 25, 1992. He received his medical degree from McGill University in 1944 and taught surgery as an associate professor at McGill's Department of Otolaryngology. After 30 years of specialty practice in Montreal, he moved to Antigonish and founded the Department of Otolaryngology. He is survived by his wife, two daughters and two sons. We offer our sympathy to his wife and family.

Dr. C.R. Benson Auld, (68) of Halifax, Nova Scotia died on February 2, 1992. Born in Prince Edward Island, he received his medical degree from McGill University in 1949. He did postgraduate studies in general surgery in British Columbia and Halifax, and was a member of the faculty of Medicine, Dalhousie University. He was a member of the Medical Society of Nova Scotia and the Canadian Medical Association. He is survived by his wife, a son and three daughters. The *Journal* extends sincere sympathy to his wife and family.

Dr. Rueben S. Shlossberg, (85) of Halifax, Nova Scotia died on February 11, 1992. He received his medical degree from Dalhousie Medical School in 1928. He did post graduate training in ophthalmology and otolaryngology and later established a practice in New Glasgow and Halifax, before locating permanently in Halifax in 1959. He was a member of The Medical Society of Nova Scotia and the Canadian Medical Association. Our sincere sympathy is extended to his daughter and son.

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Internes & Residents	K. Pelekian	C. Zip
Obstetrics and Gynaecology	D.P. Kogen	J.B. Wenning
Ophthalmology	F.J. Carpenter	D.M. OB'rien
Orthopaedic Surgery	E. Howatt	Wm. Stanish
Otolaryngology	C.C. Cron	
Paediatrics	M.B. O'Neill	A.E. Hawkins
Laboratory Medicine	D.M. Haldane	A. Foyle
Psychiatry	A.E. Hipwell	S. Brooks
Radiology	G.J. Butler	C.D. Lo
Surgery	P. Roy	J.P. Curry
Urology	R.W. Norman	B.W. Palmer
Vascular Surgery	D.A. Pyne	R.S. Dunn