THE NOVA SCOTIA MEDICAL BULLETIN

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Canadian Armed Forces Issue

Foreword

The articles appearing in this issue of the Nova Scotia Medical Bulletin have been submitted by staff members of Canadian Forces Hospital Halifax through the courtesy of the Surgeon General, Canadian Forces Medical Services.

We wish to acknowledge the honour and privilege extended by the Editorial Staff for their invitation to devote full space in this issue of the Bulletin to articles from our staff members.

R. B. IRWIN SURG/CAPT
Commanding Officer
Canadian Forces Hospital Halifax

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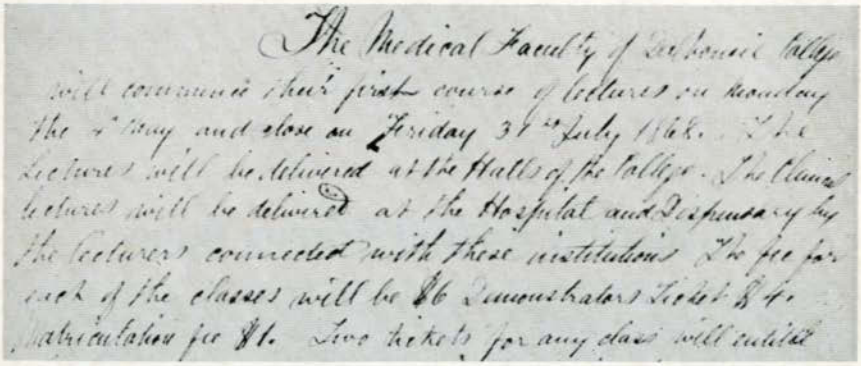
One Hundred Years Ago

January 1868

THE ANNOUNCEMENT OF THE OPENING OF DALHOUSIE MEDICAL SCHOOL



The Medical Faculty of Dalhousie College will commence their first course of lectures on Monday the 4th May and close on Friday 31st July 1868. The Lectures will be delivered at the Halls of the College. The Clinical lectures will be delivered at the Hospital and Dispensary by the lecturers connected with these institutions. The fee for each of the classes will be $6. Demonstrators Ticket $4. Matriculation fee $1. Two tickets for any class will entitle the holder to a perpetual ticket for that class with the exception of the Demonstrators fee. Five lectures shall be given each week in each class one of which shall be an examination on the subject treated of previously and it will be considered a lecture. The course will be illustrated by plates, specimens and apparatus so that the student will have ample facilities for obtaining a knowledge of the subject. The text books are those generally recommended by the schools. The Provincial and City Hospital may be attended daily at 12 by students without paying extra fee and clinical lectures will be delivered twice a week illustrated by cases at the time in the wards in addition to the general instruction at the bedside. The student will here have the best opportunities for becoming acquainted with the treatment of Disease and at the Deadhouse, Post Mortem examinations will make him conversant with Pathological appearances. The city Dispensary is carried on after the Model of De Milt N.Y. being divided into three departments – Surgical including the eye and ear – Medical including the heart and lungs and Diseases of Women and Children: The student can here have the advantage of seeing the treatment of more than 50 cases daily and likewise become practically acquainted with Midwifery under the officers of the institution. Every facility will be furnished to students to become practically acquainted with Anatomy both in the dissecting room and by the lecturers in that department. The Almshouse under the management of the Presdt Dr. Almon will also be at the service of the students and be a valuable means of obtaining information. There is no Medical library or Museum as yet belonging to the college but the Faculty have voluntarily placed their private libraries and collections at the service of the institution so that there will be every facility for students to become acquainted with the literature of Medicine.

Prizes A prize will be given by the Hon. Chief Justice Young to the student that has been most regular in attendance during the session and another by Dr. Almon the Presdt. to the one that has made the most progress as shown by the general answers to questions at the weekly examination due allowance being made for those not having previous opportunities.

The Lecturers in Anatomy and Materia-Medica will give a prize for the best dissection and best botanical collection all competing specimens to be the property of the faculty and go towards forming the nucleus of a Museum. There will be certificates of Honourable Mention in each class for general proficiency. □

The Early Management of the Severely Burned Patient

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Thermal injuries are probably one of the oldest forms of pathology known to mankind. It has been stated that our arboreal ancestors took their first great step towards being man when they came down out of the trees and learned to walk erect. Certainly their second great step forward was when they put fire to their own use. Thus they were able to learn to survive in colder climates, preserve food for short periods by cooking, and ultimately put heat to industrial use, until today, practically everything with which we come in contact is either directly or indirectly related to heat. Unfortunately, when man first learned to put fire to his own use, he also learned to burn himself and has been devising bigger and better ways to burn himself ever since. This culminated in the holocaust of Hiroshima and Nagasaki when tens of thousands of people died from burns. Each year, in Canada, approximately one thousand patients die and another seven thousand patients are admitted to hospital with burns.

Over the centuries, and particularly in the last three or four decades, a great deal of clinical and experimental research has been done on burns. In spite of this, there is still no truly satisfactory way of treating burns. Today, with our better understanding of the patho-physiological changes in burns, of fluid balance, infection, metabolism and nutrition, we have decreased the mortality of the moderately burned patient and increased the survival time of the severely burned patient, only to have the latter succumb to the late effects of burns. There has been no significant change in burn mortality in the last thirty years, and the mortality curve (Figure 1) continues unchanged.[1,2,3,4,5,6] A 50% burn still carries with it approximately a 50% mortality and a 70% burn, for all practical purposes is 100% fatal. In the older age groups, the mortality is vastly increased, and to a lesser extent in the younger age groups. Unfortunately, nearly half of all burns occurring in an average community, occur in children under 12 years of age. Even more discouraging is the fact that the vast majority of all burns are preventable.

In spite of this somewhat discouraging picture, much can still be done for the burned patient and with proper care, particularly in the early phases, the majority of these patients can be saved. The treatment of burns involves the understanding of surgical techniques, respiratory physiology, the physiological changes of fluid and electrolyte balance, nutrition, biochemistry, bacteriology and psychiatry, in other words a working knowledge and understanding of all the disciplines of medicine. Certainly the doctor who undertakes the treatment of burns must be a physician in the broadest sense of the word.

RELATIONSHIP OF EXTENT OF BURN TO MORTALITY

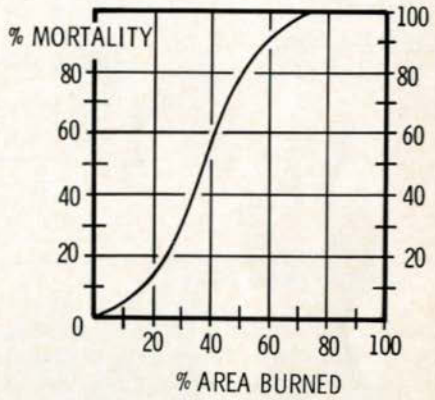

Figure 1. Burn Mortality Curve.

It is the purpose of this short paper to review the management of the severely burned patient during the first 48 hours. It is during this period that the majority of fatal cases will die, the cause of death being burn-shock. The actual cause of death is obscure and the post-mortem changes bizarre. No attempt will be made to discuss the late management of the local burns because this is basically the field of the plastic and the re-constructive surgeon. Similarly, infection, although the great cause of death in the later stages of burns will not be discussed in detail. The responsibility and capability of clinics and hospitals in the various types of burns is outlined in Figure 2.

Classification of Burns

Burns may be classified in many ways depending on whether one is a "lumper" or a "splitter". The oldest, and still the most common method, is to classify burns into first, second and third degree. Others classify them in anywhere up to nine or ten degrees and still others have devised a complicated

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CRITICAL BURNS

2° BURNS OF OVER 30%
3° BURNS OF FACE, HANDS, FEET OR OVER 10%

Burns complicated by:

RESPIRATORY TRACT INJURY
MAJOR SOFT TISSUE INJURY
FRACTURES

ELECTRICAL BURNS

General Hospital

MODERATE BURNS

2° OF 15 - 30%
3° OF LESS THAN 10%,
EXCEPT HANDS, FACE, FEET

Community Hospital

MINOR BURNS

2° OF LESS THAN 15%
3° OF LESS THAN 2%

Figure 2. Level of Care required for various degrees of severity in burned patients.

RULE OF NINES

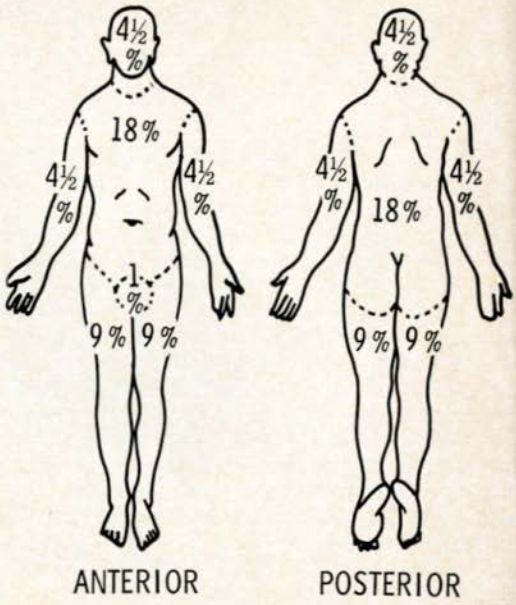

Figure 3. The Rule of Nines. A rapid method of estimating percentage of body surface involved.

burn index based on depth of burn and percentage of body surface involved. Regardless of the type of classification, it boils down to the simple fact that a burn is either superficial or deep. Although this has a great bearing on the later treatment of the burned area, it is often difficult to determine at the time of burning. It is logical that the deeper the burn, the more severe the reaction of the patient as a whole.

Of equal importance in determining treatment, particularly in the early phases, is the percentage of body area burned. Many methods of determining this have been devised but the rule of nine (Figure 3) is still the most common. It makes little difference how one estimates the percentage of area burned as long as the examining doctor makes a thorough, careful and honest estimate.

The management of the burned patient during the first 48 Hours may be divided into seven categories:

1. Sedation
2. Tracheostomy
3. Antibiotic coverage
4. Tetanus prophylaxis
5. Intravenous therapy
6. Diet
7. Initial care of the local burn.

Sedation

Burns, unless they are full thickness, are exquisitely painful and the patient most apprehensive. "Cold water first-aid" is simple, safe, readily available and effectively alleviates pain. However, burn patients require sedation, often in considerable amounts. Whatever sedation is used should always be given intravenously. The painful phase of the burns usually lasts only a day or two and care should be taken not to over-sedate the patient after this period.

Tracheostomy

Many burns produce thermal trauma to the respiratory tract, particularly through inhaled heat or in burns about the head and neck. The tremendous amount of edema which ensues frequently results in laryngeal obstruction. Tracheostomy is not only beneficial but is often mandatory in such patients: one should be alert to the necessity, and always be prepared to do the tracheostomy before the absolute indications arise.

Antibiotic Coverage

Although infection is the great killer of burn patients if they survive the first few days, it is now generally agreed that routine prophylactic antibiotics should not be used because they may well promote the growth of resistant organisms. It is better to obtain a culture and sensitivity of the

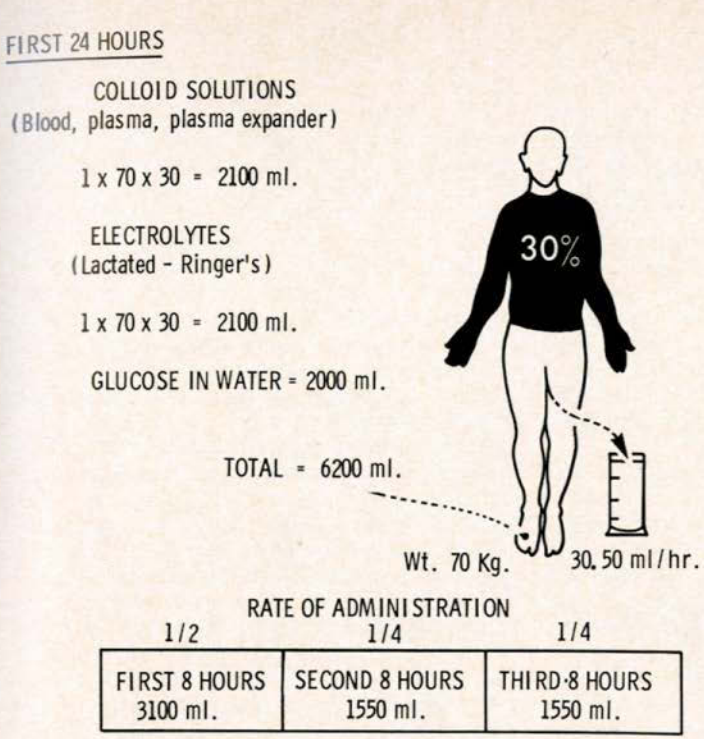

Figure 4.

The Evans Formula in the estimation of fluid requirements in a patient weighing 70 Kg with a 30% burn.

organism or organisms through skin and blood cultures and then to give the appropriate antibiotic in adequate doses for an adequate time. If an antibiotic must be given without waiting for cultures and sensitivities, then Penicillin is still the drug of choice.

Much has been written about burn toxemia and it would appear that this is most likely the toxic end-result of an overwhelming infection. The use of convalescent burn serum as a therapeutic-aid agent is not generally accepted as being beneficial. Recently, interesting work has been done on passive and active immunization through vaccination, particularly with Pseudomonas infections.

Tetanus Prophylaxis

All burn patients who have been previously immunized should receive a booster dose of tetanus toxoid; those who have not been immunized should receive a course of active immunisation.

Intravenous Therapy

Shock is the major cause of death during the first 48 hrs. after the burn, and it is the doctor who initially receives the patient who can do much to determine whether the patient will survive or not. It is important to emphasize that any replacement therapy should be calculated from the time of burning, not from the time of beginning treatment.

Upon arrival at the treatment centre, the patient should be quickly examined and the extent and severity of the burn ascertained. Vital signs should be noted and these should be monitored frequently along with hemoglobin, hematocrit and, if necessary, central venous pressure. An indwelling urethral catheter should be inserted and the hourly urinary output measured.

Much has been written about the "burn formulas" available in the current literature. It must be realized that none of these is a panacea but that all of them are an extremely useful guide to the replacement therapy of the severely burned patient. The first formula was devised by Evans (Figure 4) and his formula is still the most commonly used today. It is simple and straight forward. A clinical example of the use of his formula is shown in Table I.

TABLE I
EVANS FORMULA

1 ml × wt (IN KILO) × % BODY SURFACE BURNED
= COLLOIDS (BLOOD, PLASMA, PLASMA EXPANDERS)
+ EQUAL AMOUNT OF ELECTROLYTES
+ 2000 ml 5% GLUCOSE IN WATER
AUTOMATIC CUTOUT AT 50% BURN

The judicious use of a burn formula, guided by sound clinical assessment of the patient and monitoring of the various clinical parameters available will do much to increase the survival-time during this method. This probably represents the greatest advance we have had in burn therapy in the past few decades.

Colloid may be given in the form of whole blood, plasma, or plasma expanders. Considerable controversy exists as to how much blood should be used and when it should be given. However, most of us feel that probably one-quarter to one-half of the Colloid requirement should be in the form of blood and the remainder in plasma, if available. If plasma is not available, the use of Dextran, or better still, low-molecular-weight Dextran is quite satisfactory.

Many commercial electrolyte solutions are available such as Ringer's solution, Hartmann's solution etc. Theoretically, these solutions represent a more physiological fluid but there is little clinical evidence to substantiate that they have any advantage over the use of normal saline. Normal saline is readily available and is still the electrolyte solution of choice.

Two thousand c.c.'s of 5% dextrose solution are given to compensate for insensible loss and the urinary output of 30 to 50 c.c.'s an hour as measured through the urethral catheter.

Diet

Burn patients very quickly go into a profound catabolic phase. Usually after about 48 hours it is possible for them to take nourishment by mouth. High-protein diets are most unpalatable and there is little point in giving such a diet unless a normal caloric intake can be maintained. Therefore, it is of the utmost importance that the burn patient have a high-caloric intake. To accomplish this, it is much better to give the patient whatever he wishes to eat rather than try and force upon him an unpalatable but high-protein diet. One of the common causes of failure to eat during the early phases of burns therapy is over-sedation, and one must guard against this.

Initial Local Care of the Burn

All burns are sterile at the time of injury and every effort should be made to preserve this sterile state. Consequently strict aseptic techniques should be used in all phases of burn treatment. If the burned area requires cleansing, this should be done in the most gentle way possible, using a bland solution such as water or "Cetavlon". Soft gauze should be used, never brushes. This can usually be done under intravenous sedation and rarely requires a general anaesthetic. The question of whether blisters should be removed or not depends on individual preference. Most surgeons prefer to remove massive blisters or those blisters that have been broken, leaving the smaller blisters intact.

The actual care of the burned area falls into one of two major categories: either a modification of the occlusive-dressing method or some form of open or exposure treatment. The occlusive method of dressing is one of the oldest forms of treatment and is still widely used with excellent results. Unfortunately it requires a large amount of sterile material and an experienced staff to carry it out. These two requirements are usually not readily available, particularly in smaller hospitals. The method is usually limited to the treatment of extremity burns, being impractical in burns of the head and neck or of the trunk. Nevertheless, it is an effective way of treating burns and is the treatment of choice in patients who have to be transported to another medical facility.

The exposure method of treatment was a logical sequence to the closed method. A burn that is left untreated will form a fairly satisfactory eschar within a few days. Over the years, many lotions, potions and poisons have been devised as topical treatment for burns. None of these have stood the test of time. They all have one fault in common in that they do not "get to where they are needed". Tannic acid was one of the first of these described and probably was one of the most effective burn dressings we have ever had. Unfortunately it was extremely toxic and in some cases lethal to the individual. This has been the story of most of the topical applications for burns. More recently, Carl Moyer revived the use of 0.5% Silver Nitrate. Similarly Moncrief has popularized the use of Sulfamylon. Silver Nitrate is now being questioned because of its disturbing effect on electrolytes and other suspected toxic effects. Sulfamylon and other antibiotic dressings have produced excellent results in the hands of their developers, but these results have not been duplicated by others. One cannot help but wonder if the excellent results described are not simply the result of superb surgical technique in the hands of dedicated clinicians.

The open method is the only means we have of treating large numbers of burned patients and it is most efficacious in burns of the head and neck, hands and girdle burns.

Brief mention should be made of the small full thickness burn, i.e. – less than 3% of the body surface, which may be excised and closed primarily. Similarly the trend in the management of burns of the hands is towards earlier and earlier debridement and grafting.

Summary

Although the overall picture of burns still continues to be somewhat discouraging one should not be pessimistic. If the seven points mentioned above are remembered and followed through to their logical conclusion much can be done to save patients and to increase the survival time of the severely burned patient.

The most important aspect of the treatment of any disease entity is its prevention. Approximately one-half of all major burns occur in children and practically all burns are preventable. Therefore we as individuals, and as a profession, have a responsibility to promote fire safety and adult education to eliminate fire hazards particularly in the home. Many minor burns are converted to major burns by highly inflammable synthetic clothing and every effort should be made to educate parents and industry in the use of flame retardant clothing. □

We wish to thank Mr. Gibson and his Staff in the Audio Visual Dept., Dalhousie University for their help.

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The Headache in Your Office

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It is my opinion that the commonest type of headache seen by physicians anywhere is what has been termed in the past "tension headache". It is unfortunate that the term has tended to confuse this headache with other types and recently it has been renamed in most textbooks and by mose people as "muscle contraction headache". This is indeed a more descriptive and better term. Although there may be some argument as to whether or not this is the commonest type of headache, I firmly believe that there can be no argument about the fact that it is the worst handled of all headaches and one of the most misunderstood and badly treated problems in medicine. I firmly believe this, because I see patients not by the dozens but by the hundreds who have had their headaches for 3, 6 or 9 months and who after seeing 3, 6 or 9 doctors receive 3, 6 or 9 different treatments and continue to have their headaches for 3, 6 or 9 more years. The discouraging thing from the patient's standpoint is not only that he still has his headache but as time goes by he receives less and less sympathy from his doctor but still the same treatment that he received last time: treatment that did not relieve his symptoms.

The first and foremost problem to settle is that of diagnosis. What is meant by the term "muscle contraction headache" and how does one recognize it? It is not a throbbing or a pulsating headache and I do not believe it should be referred to as a psychogenic headache or a nervous headache anymore than it should be called by a hundred other names. Although these latter factors may contribute to some headaches they are often of no real significance in many other headaches and the term "muscle contraction headache" relates precisely what the headache is caused by – *namely muscle contraction. It also denotes precisely how to treat the headache or to relieve the symptoms – namely by relieving muscle contraction.* The patients may describe their headaches in a variety of ways: as an ache, as a tightness, as a feeling of constriction or even a feeling of pressure. Depending on the circumstances the headache varies greatly in intensity and in duration. It may be of nuisance value and shortlived or incapacitating and unrelenting. The site of maximum intensity may vary from time to time from frontal to temporal to occipital and be unilateral or bilateral. *The most vital point in the diagnosis is an association of a definite related ache in the neck muscles.* This point is the one frequently missed by most physicians, since it is so rarely stressed by the patient, and the information must often be obtained by direct questioning. It is a very common occurrence in my experience to have patients referred with headaches or to be presented on ward rounds with a very detailed description of the type, the site, the intensity, the duration, the aggravating factors but with no mention of this most vital point. One must remember that to a patient, a headache simply means an ache in the head and he does not mention what he does not consider important. Once he or she is specifically questioned about this fact, however, they will frequently relate in detail the various relationships and the factors affecting and producing both the neckache and the headache. It is on this connection with sustained contraction of neck muscles that the pathophysiology of the headache, the diagnosis and the treatment rests. Complete relief for the headache or the cure of the headache, and I repeat, the cure of the headache, will follow the breaking of this connection. Relief of symptoms will follow in all cases irrespective of whether the headache has been present 9 days or 9 years if this muscle contraction cycle is broken. As sure as fate there will be no relief of headache or rapid recurrence after symptomatic therapy if this spasm is not broken.

As far as the pathophysiology of these headaches is concerned, there is no problem about which comes first as with the chicken or the egg. The sustained chronic neck contraction produces the headache. Most normal people have the ability to read for long periods, to do detailed work over their desks or over any particular job or equipment without producing sustained neck contraction. A great number of people, however, are less fortunate and find it impossible to do almost anything that they have to concentrate on without producing sustained contraction of the neck muscles during this period of concentration. Once this cycle of neck contraction develops with almost any job requiring concentration then the vicious cycle begins and once headache is produced it will recur repeatedly, at first only at intervals, then semi-constantly and finally as a constant headache. These people are seen so often by so many different physicians that eventually they settle into a routine of discouragement, analgesics and so often a bonus of indigestion.

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The condition under which the headache occurs is very important and an aid in the diagnosis because the physician can usually correlate the working conditions that produce the headache as well as occasions when the headache does not occur such as weekends, holidays, periods of relaxation and multiple working conditions which do not produce sustained muscle contraction. Lack of relief from medication again helps to confirm the diagnosis. Without relief of the associated neck contraction there is no headache relief; fortunately the patient is often well aware of this and is more than ready to listen to an explanation and to adopt any method of treatment which does not involve drugs but will involve relief of headache.

I want to dwell now on my rather detailed handling of this problem with a patient sitting with me in the office. On examination the patient often has a headache right at the time and if so, then the marked tension of the neck muscles can be easily felt. This is often confined to one side only or one side may be more in spasm than the other. This can often be relieved simply by extending the neck or getting the patient consciously to relax all his neck muscles. It is important also to ask the patient at this time whether this has any effect on his headache. It is surprising how often 30 seconds of neck relaxation will tend to lessen the headache. The patient will often note that in the past he has often had to relax his neck or have it massaged or rubbed or do various things with his neck in order to relieve the headache. Again this is important from the standpoint of convincing the patient of the cause and in seeking his co-operation for the cure.

The next point that I stress to him very carefully and very slowly so that it sinks in is that there is nothing unusual, strange, uncommon, rare or peculiar about this headache. It is a simple garden-variety type of headache that is seen, treated and cured everyday. This is important because so many people have become convinced that their headache is intractable and that it almost surely represents some underlying disease that has not yet been discovered. I might note that most of them, if they have had their headaches for years have already had their skulls X-rayed, their necks X-rayed and have taken a multitude of muscle relaxants, analgesics and often physiotherapy. It is indeed unfortunate that some of them happen to have unimportant X-ray findings and have been told that this is the cause of their headache. This theory must be forthwith destroyed and laid to rest. It must, however, be done carefully, diplomatically and ethically, but it must be done.

I now tell the patient (and it is usually quite true) that this type of headache usually occurs in hardworking, intelligent, conscientious people – the kind who like to do a job well and to concentrate on what they are doing. I also tell them that they are the kind of people whom I would like to have working in my firm, if I had a firm, or for me personally, and that this type of headache rarely, if ever, occurs in the non-conscientious useless type of person. As of now the patient is on your side and he says to himself: "at last I have found a physician who knows what he is talking about". I would like to note here that a great majority of these people have been told that they are neurotic, or make too much of their headaches or that their headaches are psychological in origin. When they do not respond to analgesics the doctor unfortunately often becomes convinced that there is a big psychological overlay and so often, in spite of his pain and discouragement, so does the patient. The psychological aspect of these headaches is in my opinion overstressed. It is so often not the important problem. It is easy to see, therefore, why this first approach is so important and that so often there just isn't anything you can say or do wrong from here on.

Next comes the pathophysiological explanation to the patient in plain "layman terms". I tell him or her to cock the arm and to contract the muscles very hard and to keep them contracted. I then tend to occupy myself with something at the desk and the moment the patient relaxes the arm I forestall him by saying: "no, keep it contracted". When I finally let him relax it, he is aware not only of a real overall tiredness in his arm but a very definite ache. I then tell him that if he had kept the muscles contracted longer they would have ached more, and after a while would have become not only painful but unbearably painful. I point out to the patient that the moral of this experiment is that muscles are meant to contract and to relax and *not to remain contracted.* He usually nods his head in complete understanding and agreement. I then explain to him that the head is held steady by the muscles of the neck which act like guy ropes and that under ordinary conditions the muscles are continually contracting and relaxing as we read or study at a desk, or paint or use a welder's torch or do any other form of work that requires concentration and a steady hand. I then do a simple experiment with him. I give him something to read or to look at. I then tell him that I want him to read the same thing but to really concentrate on it as if his life depended on it and as if he were really interested in the fine detail. The patient nine times out of ten will be aware of the fact that this added concentration makes his neck muscles literally stand out and forces his head to a steady position. I repeat the experiment while he feels his own neck muscles and I then explain to him carefully that when he works or reads or concentrates that the muscles are able to do this automatically but that the people with tension headaches have entered a vicious circle which must be broken if they are to be relieved.

An important point in the history is that the headache often comes on at the end of the day or even in the evening when the patient is presumably relaxing. It is quite obvious that this is after a whole day of working and contracting neck muscles and that this is why the headache only comes on intermittently to begin with. Eventually however, as the pattern becomes more firmly established the patient may go to bed with the headache, awaken and start the day with the headache. I then ask the patient: "does this make sense"? He usually remarks that it makes an awful lot of sense and you you can see "the wheels going around" as he then tends to fit this explanation into his own pattern of work and to explain to himself as well as to me why it is quite obvious that he has had headaches for so long.

You must now impress upon the patient that now that he understands the exact cause, he must never forget it, and that he must remember that the cure depends on his developing a persistent pattern of working with a relaxed neck just as in the past he has worked with a persistently contracted set of neck muscles. The patient must take time out to relax the neck muscles deliberately and to turn the neck from side to side and then go back to the job with a relaxed neck. *This does indeed result in cure.* I realize that many people who are authorities on this type of headache routinely use many forms of treatment including drugs, analgesics, heat and physiotherapy, but I firmly and absolutely believe that these methods defeat the main purpose, namely, that the patient must be so completely and thoroughly convinced of the cause of the headache that it becomes obvious to him that the cure involves breaking the cycle and nothing else. I have never seen this method of handling really fail to relieve a patient of his symptoms and I have never resorted to the use of drugs or any other form of treatment.

Often in the presence of medical students I have completed this routine and told the patient: "there is no doubt about the fact that your headache will be cured and there is really no reason why you should return, but I wonder if you would be kind enough to do so in a week in order that the students may see how well you are". The students, of course, are almost 100% doubtful that such a serious chronic problem can be "cured" by such simple methods but they are always impressed when a very grateful patient returns not only without a headache but thoroughly convinced that the method works and that such headaches are a thing of the past. □

O Trust in the Lord.

The November 1894 issue of the *Maritime Medical News* has the following which may have some psychological value:—"Medical Journals not Appreciated."—The following reply was received to a circular letter soliciting subscriptions to a certain medical journal:—

> Your copy of the———journal Fairbault, Minn., come, and the letter to,—askin me to sen' fifty cens and get it fur a year. I don't need no journals. When I git a tuff case I go off inter sum secrit plase and tell the Lord all about it and wate for him to put inter my minde what ter do. That's better'n jurnals and cyclopedes and such. If we had more Lord trustin' doctors and less colleges weed fare better. The Lord knows moren all the doctors and if we go to him fur noledge it ill be better'n journal
>
> Fraternally in the Lord,
>
> A CHRISTIAN DOCTOR
>
> P.S. I've practiet medisen mor'n fifty years. Yore can publish this letter if you want ter.— *Northwestern Lancet.*

Getting the Most From X-Ray Examinations

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In this present age of medicine it is the rare patient who escapes some form of radiological investigation. This is especially true of the hospitalized patient. In a great many instances the radiological interpretation will be left to the diagnostic radiologist and such interpretation will depend to some extent on the referring physicians' clinical impression. This information is crucial to the success of the consultation and will ensure that any special views required will be obtained at the time of the patient's first visit, before the examination is considered complete. A woefully incomplete X-ray referral may break the link in the very necessary triangulation so vital in the radiological interpretation.

The referring physician himself should, and may in many instances wish to review the X-rays for himself. In this manner he will build-up his own store of roentgenographic knowledge. Most physicians do this after a fashion; some build at random and without much sense of organization or satisfaction; some do it systematically and deliberately. Those who do usually find that roentgenographic facts which are gathered together in clumps and groups are considerably easier to remember.

Remember, the initial visualization of an X-ray film is a radiological finding and not a diagnosis. In the patient being investigated medically for the first time, the finding of a fluffy soft tissue density in the lung does not make a diagnosis of tuberculosis. If the radiologist phrases his report rather positively in the direction of that disease as a more probable explanation of the shadows he sees, he does so with the certainty that you, the clinician, will not consider the diagnosis established without bacteriological confirmation.

Certain diseases should not be "diagnosed" in the written radiological report. There are other times however when the radiological appearance is pathognomonic, and in that instance time is saved for every one concerned if the radiologist states that the findings are compatible with a specific entity. The more expert and knowledgeable the radiologist becomes, the more confident he will be in undertaking to direct your thinking, and the more reliant you will become on his diagnostic suggestions.

Radiological literature is full of many articles reporting the statistical incidence of this or that radiological finding associated with a certain pathological disease process. Familiarity with such findings helps the radiologist in advising you. He knows for example that some 80 percent of infants who have a congenital absence of the iris will probably develop a malignant mixed embryoma (Wilm's tumor) and some 1 percent of infants with Wilm's tumor may have a congenital absence of iris. In the lung infections, he knows that the patient with bacterial pneumonias of the lobar type, usually begin showing a density in the lung radiologically some 6 to 12 hours after the onset of symptoms; while those with viral or atypical pneumonias frequently show no change for some 24 to 48 hours. Patients with pulmonary tuberculosis may not show any demonstrable radiological lesion for some five months after exposure. Thus interpretation of shadows must be considered in the light of such considerations.

However, your patient is not a statistic and although the radiologist may interpret his findings in the light of such statistical possibilities, you are treating one human being, a statistic of one. Thus the interpretation of the radiological findings must always be fitted into the clinical picture and a triangulation arrived at. The radiologist can also be of service to your patient by ruling out from a radiological stand-point some items included in your differential diagnosis. Often subsequent film, studies and clinical findings will narrow the list of possibilities until a final diagnosis is arrived at.

Some disease conditions, however, produce no radiological change whatsoever, or when such changes are produced they are so late in the clinical pattern of the disease as to be of minimal value in early diagnosis. The bony changes in gout is one case in point. Consultation with the radiologist will soon build up your knowledge of disease conditions in which you can or can not expect radiological assistance and in addition the most suitable type of examination to request.

On the other hand a negative radiological examination in the patient with a compelling story and clinical findings and in whom radiological changes are to be expected, is one of the most constant and disturbing problems the radiologist has to face. He has to be familiar with, and remind you about the weaknesses of a certain radiological method. The patient with a history suggestive of gallbladder

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disease may have a negative X-ray study for years before he finally shows positive evidence of chronic cholecystitis and cholelithiasis. Likewise in patients with scarring from a duodenal ulcer it may be impossible to demonstrate a recurrent crater by barium study. In such instances much more reliance must be placed on the clinical impression which may actually rest on the patient's own assessment of his condition. Thus we have two types of radiological information to accumulate and correlate. One must have a clear visual image of the various sorts of radiological changes a particular disease may produce such as the spectrum of changes in the chest film in a patient with pulmonary sarcoidosis. At the same time you must also be aware of and accumulate information as to the fallibilities of the radiological examination in that same disease. You must be prepared for the negative examination and wise enough to discount it in the patient over whom you are justly worried. In general, positive radiological findings are helpful but a negative study does not provide a clean slate.

A good relationship between yourself and the radiologist is to be not only recommended but encouraged. You must freely be able to say "how sure are you?" and he must be able to reply, "I am not sure at all, but I believe it is the most probable explanation". At other times he must be able to say, "I can not of course guarantee what the micropathology will show, but as far as the gross pathology is concerned I have no doubts whatsoever"; or he may even say, "it is non-specific and has to come out to be spelled out". He must know his limitations and be prepared to say, "I have little experience with this disease entity and I would like to consult with my colleague."

I again caution that, in general, positive radiological findings are helpful but a negative study does not provide a clean slate and you must be prepared to say in the face of a negative report, "that is all very well but I feel strongly that my patient has the disease". You must however also listen to the radiologist when he feels strongly about a diagnosis and insists that no other should be entertained. *Argue with him if you must, but listen.*

In certain diseases however there may be a tendency to over-interpretation. In contrast to the serious disagreement among physicians detecting physical signs, radiologists quite consistently detect those X-ray signs that have been traditionally used to diagnose emphysema. Pulmonary hyperinflation appears as an increased translucency of the lung fields on a film with proper X-ray penetration. Radiologists often use these signs of hyperinflation to diagnose destructive emphysema because hyperinflation and emphysema are characteristically associated. Despite their conformity however, the value of X-ray interpretation is only slightly superior to the physical examination because of the difficulty in differentiating emphysema from pure pulmonary hyperinflation which is commonly present in normal ageing or in any condition that obstructs the airway, such as asthma. The chief value of the chest X-ray is probably to help to exclude cardiac and other causes of dyspnea, rather than to detect emphysema.

Some radiological examinations are almost routine; for example, any patient who had enough low back pain to consult a doctor deserves X-rays of the lumbar spine and lumbosacral area. Although such X-rays are normal in most patients they will reveal or rule out congenital anomalies and diseases of, or injuries to, the bones and joints.

Finally, every radiological department performs many studies of little or no value because the patients were improperly prepared. This probably happens most frequently in colon studies where in spite of the utilization of the most advanced technological equipment and experienced radiographers, the best diagnostic radiological interpretation cannot exclude a carcinoma masquerading as faeces. How much time this takes varies tremendously in different patients and each one requires supervision. Preparation of the older patient with colonic inertia should start much earlier and sometimes include a supplementary saline enema. In examination of the small bowel the success of the examination will quite often depend upon adequate prior cleansing of the colon. This improves the transit time through the small bowel reducing the time spent in the radiological department as well as reducing the radiation to the patient. The alternative to gradual emptying of the colon is a violent purging and severe fasting. These measures may be much more traumatic than the examination itself and they are unnecessary if the referring physician plans ahead.

Planning ahead ensures not only adequate preparation of the patient but also that the necessary equipment will be available for special procedures. Supplemented by an adequate clinical history a more conclusive and satisfactory diagnostic radiological consultation will be obtained. □

Blushing

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The phenomenon of blushing can be either a normal or increased physiological response which varies greatly in individuals depending to a great extent on their inherited, constitutional and congenital attributes. It becomes abnormal when it occurs in almost every situation or when it becomes a hindrance to the individual in his inter-personal relations. As a symptom of expression, it has both a physiological and a psychological basis. Physiologically it is a dilatation of the blood vessels in the cheek, face and neck caused by nerve impulses conducted from higher brain centres via the autonomic nervous system; so that blushing from embarrassment is due to some physiological process in the brain. This same phenomenon can be produced by injection of histamine or nicotinic acid. At the same time however, it is a subjective sensation which can be described in psychological terms and by the person experiencing the embarrassment with blushing. Tickling can cause laughter, a blow can cause weeping or frowning but there is no physical means by which a blush can be produced. The blush itself may be physiological in its manifestation as a reddening of the face but its cause is on a psychological basis. As such, it is the most peculiar and the most human of all expressions.

Charles Darwin in 1872 wrote a book called "Expression of the Emotions in Man and Animals" and little has been added to the facts about blushing as originally enumerated by him. He pointed out that blushing occurs more frequently in women than in men, and much more freely in the young than the old. It is common to all races whether or not there is any change in facial colour. He felt it was primarily a psychological problem, tended to be inherited, was involuntary and the wish to restrain it led to increased self-attention which actually increased the tendency to blush. Certain movements and gestures accompany blushing such as a strong desire for concealment, turning the body away, casting the eyes down and inability to gaze directly at a person. Also, a state of (non-psychiatric) confusion with the physiological reactions of tachycardia and increased breathing accompany blushing.

More recently, Franz Alexander has pointed to blushing as evidence of the connection between psyche and soma in the study of psychosomatic medicine. He points out that "embarrassment . . . is a distinct process in the brain, but at the same time is a subjective sensation which can be described in psychological terms . . . and a psychological phenomenon can be most precisely described by the person who blushes. The appropriate use of psychological and physiological methods of description - each in its proper place - is the essence of Psychosomatic Medicine".

The mental state that produces blushing is embarrassment associated with shame and modesty, along with the general state of shyness, the essential element in all cases being a state of self-attention. Self-attention directed to personal appearance in relation to how others think of us is the exciting cause, and by the force of association, self-attention in relation to moral conduct. The opinions of others either build up or tear down our self-esteem or ego. In either case, attention is drawn to the self and a sense of inferiority occurs with the feeling that the other person is superior. In the one case it is handing out gifts and as such, has something we do not have and in the other is taking away something so that we do not have it.

Fenichel considers the tendency to blush or its more severe component erythrophobia (fear of blushing) to be a neurotic symptomatic picture in which social anxiety is predominant and certain paranoid trends are interwoven. The main difference however is that the person with social anxiety feels that "people might be against me" while the paranoid feels that "people are against me". "Paranoid" as used by Fenichel is a rather extreme term; a better descriptive phrase might be "Ideas of reference" or "referential ideation" (the latter being a term invented by me – I think!)

Feldman, writing in the Psychiatric Quarterly makes a number of observations regarding blushing. He considers that it is a symptom of a neurosis from which men suffer comparatively more frequently than women, that it never occurs until the child reaches the genital phase of its libido development and that it appears only in the presence of others and can be postponed for a time. The face is the only visible organ to show explicit signs of libidinal excitement. Blushing is associated with a feeling of shame which in itself is more often extrasexual than sexual; it is a social phenomenon occurring when the affected person is in contact with his fellows. □

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Physical Examination of the Knee Joint

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The young motorcyclist, the football player, the descending parachutist and the sailor falling from his ladder are all apt to suffer from a knee injury. The young female recruit has a so-called "lateral meniscus tear". The officer after curling develops a warm swollen knee. Many of our hospital beds contain patients with knee problems.

While complete histories and physical examinations are needed, local examination is very important.

No knee examination is complete without a quick verification of normal limb circulation, integrity of the sciatic, medial and lateral popliteal nerves and normal hip joint motion. It follows that "naked to the waist" possibly with jockey shorts is basic to carrying out an adequate examination. The few moments after injury are often a period of clinical opportunity. Many injured people can tolerate gentle manipulation, enough to verify active and passive ability to extend the knee, demonstration of ability to flex to a right angle or beyond, and stability of the knee in the extended position.

Here we should note that instability into varus and valgus of the fully extended adult knee proves not only that a collateral ligament is torn or stretched but also that a similar injury has affected one or both cruciates. It may be possible to flex the knee to a right angle to test the cruciates individually.

Severe knee injuries are often associated with minimal effusion. This is because the synovia and the capsule are close together, and often are both torn by the same injury thus allowing escape of fluids from within the knee-joint cavity.

If lateral instability is extensive, X-rays in displacement should be taken to show the site of displacement and to rule out fractures of the lower femur or displacement at a subluxed femoral epiphyseal plate.

Inspection of the joint will reveal general or local swellings, distortion of alignment, lateral shift of the patella, and, rarely, signs of unusual skin retraction. The latter may mean capsular entrapment.

Palpation will verify normal arterial pulsation, sites of tenderness, the amount of effusion and any abnormal position of the patella. In addition, those rare cases where an intra-articular cartillaginous fragment is broken off can only be detected early by palpation or contrast arthrography.

Aspiration is valuable in non-traumatic knee effusions for diagnosis, but after trauma most of our knees have quite a bloody effusion, diagnostic only of some trauma somewhere, a fact we usually knew before.

X-rays should always be taken - at least three - an anteroposterior, a lateral and an oblique view. The latter will sometimes show tibial plateau fractures not otherwise seen. If the patella is suspected of damage, views of both patellae to help exclude bipartite patellae, as well as profile views may be needed.

In acute injuries certain combinations of findings are ominous. Absence of distal pulses is serious. Instability of the lateral ligament with common peroneal nerve palsy at times has been due to the nerve being caught in the joint space. Inability to extend at all implies an extensor mechanism defect. This defect is often palpable. Instability of the medial collateral ligament and of the anterior cruciate, frequently with minimal effusion, should make one suspect that the medial meniscus may be torn as well

Any knee which after aspiration will not passively extend fully probably should be manipulated under anaesthesia with exploration if the knee will not then extend.

Between acute injuries and chronic injuries lie problems which we wish could be eliminated. The main one is found in the patient with an injured knee which has been allowed to lie flexed 20 degrees on a pillow for 15 to 30 days. Even examination under a general anaesthetic will not make it clear whether he has a torn cartilage preventing full extension or whether this is due to the disuse and adhesions from the flexed position. We mobilize these patients without weight-bearing, but frequently extension is not regained and an arthrotomy has to be performed. These cases are our chief source of "negative" explorations.

In chronic knee complaints, chondromalacia of the patella is the main offender. Patients with this problem have painful grating if the patella is rubbed over the femur. To do this the knee must be fully extended and supported from behind or the tight quadriceps will prevent motion. The margins of the posterior surfaces of the patella can be felt with a finger-tip if the patella is moved to one side or the other. These areas will be more tender than will those of the other knee, if the latter is normal. Effusions may or may not be present.

Cysts of the menisci can readily be seen or felt as firm, hard swellings that often disappear and reappear as the knee is put through a full range of motion in a good light. The important words in the above sentence are the last four, - plus an alert observer. Cysts of the popliteal space are non-pulsatile and can best be felt with the knee flexed and supported by the examiner and with the patient lying on his face.. Loose bodies are often palpable as mobile, firm swellings. Sometimes they are multiple and sometimes not visible on X-rays. If you see a knee which is swollen but you are unable to demonstrate a gross patellar tap, and if tuberculosis and rheumatoid arthritis are excluded, an arthrogram may show villonodular synovitis, synovial chondromatosis or a rare condition in which fat-infiltrated synovial fronds protrude into the joint cavity.

Collateral ligament laxity as an isolated lesion cannot be demonstrated in the fully extended position of the knee, as normal cruciates may prevent lateral instability. If the knee is stable when extended, flex it thirty degrees and then compare the lateral stability with that of the other knee, if normal, or against your knowledge of the normal if both knees are involved.

In older persons over thirty-five years of age and in those with a prolonged history of working or playing in the deep knee-bend position, meniscal tears of the posterior horns can produce pain and episodic instability without effusion, without locking and without a definite history of trauma. In these people, vague palpable crepitus when carrying out McMurray's test, as well as tenderness over the joint line over the involved posterior horn, possibly associated with inability to "duck-walk", may be the only positive findings. Curling is a frequent cause of this condition in the city dweller and the initial episode is usually associated with some effusion.

Conclusion

We have discussed some physical findings which we have found useful. We rarely, if ever, see hemophilia, septic arthritis, acute osteomyelitis or tuberculosis in peace-time military service.

Our overlooked foreign bodies have included a two-inch-square piece of car fender in the knee joint after primary closure elsewhere. There is always a lot to find in any knee, even those regarded as normal if one simply takes the time to first listen to the patient's history and then to do a careful examination with the patient adequately undressed in a well-lit office or emergency room. □

Iatrogenic Eye Disease

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Introduction

Iatrogenic illness is defined as a condition of anxiety produced in a patient by injudicious statements of a physician. Over the years the term has come to be used for disease produced by drugs or other treatments administered by a physician and this article is about drug-induced eye disease. The field of ocular toxicology is large and a recent authoritative text describes some sixteen hundred substances which can produce ocular problems.[1] Many of the drug-induced eye diseases lie strictly within the domain of the ophthalmologist, so that this brief account will cover only those problems produced by commonly used drugs – the type of case which may crop up in general practice or in a general hospital.

Two groups of drug-induced eye diseases will be discussed:

1. Problems with topical eye medications.
2. Problems with systemic medications

Problems with Topical Eye Medications

Three groups of reactions will be outlined

1. Allergic reactions
2. Adverse effects of anti-glaucoma medications
2. Adverse effects of corticosteroids

Allergic Reactions to Topical Eye Medications

Von Pirquet's term "*allergy*" denotes a specifically altered capacity to react as judged from previous experience of the same individual or from experiences of other individuals of the same species. Factors causing allergic reaction are limitless and include numerous chemicals, living and dead microorganisms, heat, cold, light and probably emotional factors as well.[2] Common drugs which may induce allergic reactions include local anesthetics of all types, antibiotics such as penicillin, neomycin, tetracycline and chloramphenicol, sulfonamides and anti-glaucoma drops such as pilocarpine and epinephrine preparations. At times it is the liquid vehicle or the ointment base which is the allergen rather than the active ingredient.

The clinical picture is characteristic with tearing, irritation and itching of the eyes and eyelids. There is redness of the conjunctiva and lids, especially the inner and outer canthal areas and the lower lids, if eyedrops are responsible. Diagnosis is usually made from the history in conjunction with the typical appearance of the eyes. Patch tests on the skin can be used for definitive diagnosis and treatment consists of stopping use of the causative medication. Topical steroids as drops or ointments are often useful but these too may cause an allergy.[3]

Adverse Effects of Anti-Glaucoma Medication

Primary open angle glaucoma is found in about two percent of people over the age of forty years.[4] When a disease is so common there are few practitioners who do not have glaucoma cases amongst their patients. Numerous preparations are available for treating primary open angle glaucoma and the most widely used are pilocarpine, epinephrine and the anticholinesterase drugs such as echothiophate (Phospholine), isoflurophate (D.F.P. or Floropryl) and demecarium (Humorsol or Tosmilen). Pilocarpine is probably the most widely used anti-glaucoma drug. It is very safe and rarely causes trouble. At times the typical allergic blepharoconjunctivitis is produced by pilocarpine, necessitating change to another drug. Epinephrine salts (Epitrate, Epifrin and Eppy) are used alone or in conjunction with pilocarpine and these too can produce a contact dermatitis. After prolonged use, generally over a year, oxidation products of epinephrine resembling melanin are sometimes seen in the conjunctiva. These black dots are actually conjunctival inclusion cysts containing the pigmented material[5] and similar lesions have been produced in rabbits by repeated subconjunctival injections of epinephrine. The black spots are harmless and important only because they may be mistaken for foreign bodies or conjunctival melanomas. Occasionally similar epinephrine deposits form on the cornea and large aggregates may look quite alarming.[6]

The anticholinesterase drugs are strong miotics which depress the cholinesterase levels throughout the body and occasionally produce serious systemic side effects such as nausea, vomiting, diarrhoea, sweating, salivation, bradycardia and many other effects due to accumulation of acetylcholine.[7] These effects are seen in patients using these drugs who have additional exposure to anticholinesterases in the form of insecticides. There is some risk of prolonged apnoea in such patients given succinyl choline during general anesthesia. The most interesting ocular side effect is the increased incidence of cataracts reported recently in patients treated with echothiophate (Phospholine) for six months or longer. First to describe these were Scandinavian workers who reported anterior subcapsular cataracts in 50% of patients treated with echothiophate compared with a 10% incidence in those patients treated with pilocarpine.[8] Similar findings are described by others.[9,10] Usually these lens opacities are very small and cause little or no

loss of vision. It has been pointed out that anterior subcapsular opacities are not uncommon in eyes not treated with miotics and that the studies done so far were retrospective so that the final answer can only be decided when more data is available.[11,12] Nevertheless, it seems important to be on the look-out for this problem, causing failing vision in patients using anticholinesterase drugs. It is all too easy to blame the reduced visual acuity on progression of the underlying glaucoma.

Adverse Effects of Topical Steroids

Topical corticosteroids are widely and indiscriminately used. Some drug advertising gives the impression that nearly all red eyes are helped by topical steroids or, worse still, mixtures of steroids and antibiotics. There is no question that topical steroids are extremely useful but four serious problems can arise from improper topical steroid therapy.[13] These problems are:

1. Aggravation of herpes simplex
2. Development of fungal keratitis
3. Glaucoma
4. Cataracts

It is well known that some virus infections may be disseminated by corticosteroid therapy. Certain virus infections of the cornea fall into this group and the common, serious herpes simplex keratitis is the main problem. Use of topical steroids in such a case will often make the eye look and feel better, while allowing the virus to spread out of the epithelium, where it is fairly easily reached by antiviral drugs, into the corneal stroma where it is extremely difficult to treat. The only safeguard against this is careful examination of the cornea, including staining with fluorescein, to detect either punctuate staining in the earliest stages or the typical dendritic figure later. If these findings are present one should not use topical steroids. The history of recurrent cold sores on the lips associated with redness or irritation of the eye should alert one to the possibility of herpes simplex.

Corneal abrasions from finger nails, tree branches and other objects are common. Usually these lesions heal rapidly without trouble but sometimes fungi are inoculated into the wound at the time of injury and such a case treated vigorously with steroid antibiotic mixture is a good candidate for developing a destructive and intractable fungal ulcer. There is no good reason for using steroids in most of these superficial injuries. Antibiotic drops or ointment are sufficient as a rule.

Glaucoma caused by topical steroids is of great interest and has been recorded by numerous authors since Hans Goldmann described the condition several years ago.[14] This pressure rise, which occurs in eyes which are symptomless and have wide open angles, is usually temporary and subsides when the steroids are discontinued. However, irreversible steroid-induced glaucoma has been reported.[3,15] Typical visual field defects occur just as in primary open angle glaucoma but the condition can only be diagnosed in the early stages by measurement of the intraocular pressure. Many patients can tolerate topical steroids for years without getting into trouble while others develop pressure rise after as little as two weeks therapy. The usual sort of case to develop trouble is the patient with a chronic condition such as vernal conjunctivitis which requires treatment for months.[16] The suggestion was made in 1963 that eyes which responded in this way had latent glaucoma.[17] A great deal of work has been done since then by Becker, Armaly and others to study this interesting effect. The mechanism of the rise in ocular pressure is not known but the generally accepted idea is that the steroids produce a change in hyaluronic acid ground substance in the trabecular meshwork of the anterior chamber angle. Swelling of the meshwork could reduce the pore size in the meshwork and impair aqueous flow out of the eye. This is a reasonable suggestion in view of the known effect of steroids on hyaluronic acid in other parts of the body. At any rate, most reports indicate that the problem is one of reduction in outflow facility[17] although increased aqueous inflow has been suggested.[18] It is worth noting that this steroid-induced rise of pressure has been reported in 56% of patients known to have primary open angle glaucoma and seems particularly likely to occur if glaucoma control has been poor.[19] Clearly, one must be particularly wary of topical steroids in glaucoma patients. Recent work has shown this steroid induced ocular hypertension to be heritable[20] and that the gene responsible seems also to be involved in the genetic control of glucose tolerance and diabetes mellitus.[21] The important point to be made is that no patient should be placed on topical steroids for more than two weeks at a time without periodic measurement of the intraocular pressure.

Finally, the occasional report of cataract occurring after use of topical steroids should be mentioned.[22] These opacities in the lens are posterior and subcapsular in location and seldom cause any reduction in vision, but the fact that they occur at all is significant and its reported occurrence in a five year old child is worrying.[23] It is not yet known if these cataracts are progressive.

Problems with Systemic Medications

Comments will be limited to the more commonly used drugs or to those of particular interest and will be under the following headings:

a. Antibiotics
b. Steroids
c. Phenothiazines
d. Digitalis
e. Antimalarials
f. Oral Contraceptives

Adverse Effects of Antibiotics

Three commonly used antibiotics will be considered briefly.

Streptomycin

Well known for its eighth nerve toxicity, streptomycin can also cause damage to the visual pathways although such effects are rare.[1] Protracted use of streptomycin has caused xanthopsia or yellow vision, central scotomas and optic nerve bundle defects in the visual fields. The presumed mechanism is a toxic optic neuritis and most cases recovered completely when the drug was stopped. Intrathecal streptomycin has caused optic atrophy and blindness.

Penicillin

Apart from allergic reactions, penicillin has a low incidence of adverse side effects, and eye problems arising from its use are rare. Massive intravenous penicillin therapy has produced hallucinations and impaired ocular motility.[24] The reported cases also had muscular hyper-irritability, myoclonic jerks and generalized convulsions. The patients were receiving in the order of 50 million units of penicillin daily and they had impaired renal function. High serum penicillin levels were demonstrated and the patients recovered when the penicillin dose was reduced.

Chloramphenicol

The chief danger of chloramphenicol is, of course, its bone marrow toxicity. Prolonged use of the drug may also cause optic neuritis with severe impairment of vision.[25] Most cases have been in children with cystic fibrosis treated for several months with chloramphenicol. One series reported optic neuritis in 9 out of 33 children so treated. Two of these patients had severe loss of vision which was permanent while others responded to treatment with Vitamin B complex.[26] Similar cases are mentioned by other authors and there has been speculation about the mechanism of the optic neuritis.[27,28] Hypersensitivity to the drug is possible. Direct neurotoxic effect is suggested and a secondary vitamin deficiency is a third possibility. The question is not settled although the response of some patients to Vitamin B complex suggests this as a factor. Most cases improve within one to two months of stopping the chloramphenicol and all patients on long term therapy with the drug should have frequent checks of visual acuity. It is interesting that some patients with cystic fibrosis show retinal vascular changes in the form of venous engorgement, increased tortuosity, hyperemia and blurring of the optic disc suggesting edema. These changes have no effect on vision and seem unrelated to chloramphenicol therapy.[29]

Adverse Effects of Systemic Corticosteroids

This large topic can only be outlined briefly. The chief danger of systemic corticosteroid therapy is the so-called hypercortism which can produce a variety of ocular features including lid edema associated with the moon face, edema of the conjunctiva (Chemosis) and minor refraction changes due to fluid retention. Myopathy is reported, especially after triamcinilone and dexamethasone, and may produce ptosis and impaired ocular motility. Hypertensive retinopathy may occur but is mild and hard to diagnose. Rarely subconjunctival and retinal hemorrhages may occur.[30] The mechanism of these is not known. Some children, when growth has been stunted by steroid therapy, have blue scleras, but the relation of this finding to the steroid therapy is uncertain.

Adrenocortical insufficiency may occur in a patient on steroids when subjected to some stress or when the steroid dosage is reduced too quickly. In this condition there is occasionally an acute rise in intracranial pressure with the usual features of headache, drowsiness, coma and papilledema.[31] A less dramatic rise in intracranial pressure may occur when attempts are made to taper patients off long-term steroids and these cases present the picture of pseudotumor cerebri with mild headache, dizziness and vomiting. There may be papilledema and slight blurring of vision. The signs and symptoms disappear when the steroid dosage is increased.

All the features mentioned so far can be explained by excessive amounts of corticosteroids or by lack of corticosteroids. Three other ocular side effects should be mentioned. Some normal eyes show pupillary dilatation when systemic steroids are given. Some direct action on the dilator muscle of the iris could explain this, or it might be the result of adrenergic innervation potentiation.[30] Glaucoma may occur with systemic steroid therapy. This has been known since about 1950.[32] It is not very common and seems less likely to occur than after topical steroid therapy although the mechanism is probably the same. Most cases are reported in adults.[33,34] but there is an account of glaucoma in a seven month old child treated with ACTH in doses of five units twice daily for six months because of hypoglycemia. The glaucoma disappeared when the ACTH was stopped. The child was thought initially to have congenital glaucoma.[35] Cases like this emphasize the importance of periodic eye examinations of patients of all ages on long-term corticosteroid therapy.

Cataracts Following Systemic Steroid Therapy

This important problem merits a special paragraph. Posterior subcapsular cataracts (PSC) are found in up to four percent of normal persons and in up to eight percent of patients with rheumatoid arthritis who have not been treated with systemic steroids.[13] The report by Black et al[36] in 1960, describing PSC in thirty-six percent of a series of rheumatoid arthritis treated with systemic steroids, created considerable interest and subsequently numerous related articles appeared in the literature.

The incidence of lens opacities appeared to be related to duration of therapy and to daily dosage of steroid. In most cases visual acuity was little impaired. Usually the lens opacities can be seen with an ophthalmoscope but definite diagnosis is made with the slit lamp with which the lens changes can usually be distinguished from those seen in diabetes mellitus, hypocalcemia, myotonia and atopic dermatitis. However, the lens changes induced by steroids are indistinguishable from those produced by radiation and secondary to uveitis.

After Black's article appeared, other workers showed a similar incidence of PSC in steroid treated rheumatoid arthritics[37,38,39,40] but some reports suggested a lower incidence.[41,43] The relationships of lens opacities to dose seems to be definite.

Patients receiving less than 10 mgm of prednisone daily, regardless of duration, and those treated with steroids for less than a year, regardless of dosage, are unlikely to develop PSC although such cases are on record.[39,44] On the other hand, patients receiving 16 mgm per day of prednisone for over a year are extremely likely to develop PSC[45] Patients on long term steroids for asthma,[38] multiple sclerosis[46] and systemic lupus erythematosus[47] may also develop lens opacities but are less likely to do so than are rheumatoid arthritics. Children may have steroid induced PSC which resemble those in adults.[47,49] In most patients the effect on vision is small. The lens opacities may remain static for years[50] or may progress until dense enough to require removal by surgery.[40] The facts that some patients have needed cataract surgery and that lens changes can be induced in children are serious enough to warrant careful follow-up of all patients on long term steroids. Much experimental work is being done on this problem and the mechanism of the lens changes may soon be explained. It is interesting to note that as long ago as 1936, Hans Selye observed that clouding of the lens may be produced in animals subjected to non-specific stress.

Adverse Effects of Phenothiazines

Phenothiazine derivatives are widely used as anti-emetics and tranquillizers. They are particularly useful in the treatment of certain psychoses when large doses are sometimes given. A large number of these drugs is on the market including chlorpromazine (Thorazine), mepazine (Pacatal), perphenazine (Trilafon), prochlorperazine (Compazine), promazine (Sparine), promethazine (Phenergan), thioperazine (Vontil), thioprozate (Dartal), trifluoperazine (Stelazine), thioridazine (Mellaril) and others. Animal experiments have shown that phenothiazine compounds, not phenothiazine itself, are stored in the pigmented uveal tract (iris, ciliary body and choroid).[51] The same is probably true in humans.

Nearly all the phenothiazines can cause the so-called extra-pyramidal effects which are usually seen in children, less often in adults and seldom in the aged.[1] Within a few hours of ingestion of the drug there are convulsions of the head and neck, Parkinson-like staring with inability to close the eyelids and oculogyric crises. These effects disappear spontaneously in a few hours and their resolution can be hastened by use of diphenhydramine or any of the drugs used in Parkinson's disease.[52,53]

More common than these dramatic cases are the oculocutaneous side effects which include bluish discolouration of exposed skin, brown pigmentation of the interpalpebral conjunctiva with reduced tear formation and golden brown, dust-like opacities in the deep layers of the cornea and on the anterior capsule of the lens.[54,55] These changes do not usually interfere with vision and are seen after a total intake of 500 Gm to 1,000 Gm of chlorpromazine. A survey of patients receiving a total of 2,500 Gm of chlorpromazine are more showed the characteristic lens deposits in ninety percent of cases.[56] More recently a corneal epithelial change has been described in patients taking 2,000 mgm chlorpromazine daily for six months or more.[57] The exposed cornea shows white, gray or brown linear or curvilinear opacities and the effect seems related to the high daily intake rather than to total dosage. Although these corneal and lenticular deposits cause significant visual impairment in only a few cases they can lead to significant loss of vision.[58]

Although rare, a chlorpromazine retinopathy is reported causing severe loss of vision.[54] On the other hand, serious retinopathy is well documented after the use of two other phenothiazines, namely NP 207[59] and thioridazine.[60] NP 207 is not used now and the following remarks apply to thioridazine (Mellaril). The clinical picture is that of pigment dispersion in the macular area followed by clumping of the pigment in the form of large plaques and a widespread "pepper and salt" appearance of the fundus. There is usually severe loss of central vision which in seventy-five percent of the cases improved but did not return to normal over several months of follow-up. Patients developing this serious problem had received between 85 Gm and 105 Gm of thioridazine. The electroretinogram remained abnormal for at least a year after cessation of treatment in one report[61] so it is likely that the retinal damage is permanent.

No doubt more phenothiazine derivatives will appear for clinical use in the future so physicians using these drugs in high doses and for long periods will have to be watchful for damage caused by accumulation of these drugs in the eye.

Adverse Effects of Digitalis

It is said that up to seven percent of patients given digitalis develop toxic effects.[62] Of these, as many as one quarter show ocular toxicity. This ocular toxicity has been known for a long time and that great authority on digitalis, William Withering, mentions it in his book published in 1785.[63] Digitalis can produce xanthopsia (yellow vision),

"snowy" vision, coloured halos around objects, coloured spots before the eyes, flashes, sparks and scintillating scotomas, oscillopsia, photophobia, blurring or tilting of objects and failure of central vision. Rarely hallucinations are reported and even total blindness may occur.[64] The clinical signs include conjunctivitis, changes in pupil size and ocular muscle palsies.[65]

Toxic effects may appear as early as one day after starting the digitalis, are commonly seen within two weeks and may rarely appear after several years of treatment. When the drug is stopped the adverse effects usually disappear within a few days or a week or two. Rarely, visual loss may be permanent. Patients developing toxicity on several occasions may have a different set of symptoms each time, even from the same digitalis preparation.

The site of the ocular toxic effects of digitalis is debated. The occurrence of central scotomas suggests a retrobulbar neuritis. Hallucinations imply cerebral cortical stimulation and some investigators have shown elevated dark adaptation thresholds indicating damage to the retinal receptors.[66,67] Biochemists point out that digitalis inhibits the sodium-potassium activated ATP-ase enzyme system concerned with sodium and potassium movement across cell walls and this enzyme is present in high concentraton in the cerebral cortex of cats and in the retinas of humans and many animals.[68] The current view is of multiple sites of action of digitalis to produce all its toxic effects.

As a rule the physician is warned of digitalis toxicity by the characteristic anorexia and nausea but the ocular side effects can occasionally occur before any of the more usual toxic features.[69]

Adverse Effects of Antimalarials

The antimalarials chloroquine and hydroxychloroquine are widely used in the treatment of arthritis, lupus erythematosus and in some cases of asthma. Gastrointestinal upsets, skin rashes, leukopenia and whitening of the hair are known side effects of these drugs. Corneal and lens deposits and pigmentary degeneration of the retina are the ocular toxic effects which concern us here. The corneal changes, which take the form of grayish subepithelial linear deposits are reversible and seldom affect vision although blurring and halos are reported[70] so that glaucoma may be suspected unless a history of drug intake is obtained. Corneal changes are reported in from thirty percent to one hundred percent of cases in different series.[78]

The keratopathy and lens changes seem to bear no relation to the much less common but serious retinal changes which occur in up to fifteen percent of various series.[70] Rarely, other ocular effects are reported due to chloroquine. These include bilateral abducens paralysis,[71] optic atrophy[72] and macular edema.[73]

The typical appearance of the retinopathy is a granular pigmentation of the macula or patchy areas of depigmentation at the macula surrounded by a zone of normal looking retina encircled by another concentric ring of granular pigment. This produces the doughnut or bull's eye appearance which is so characteristic. Unfortunately early cases show only a granularity of the macula which is hard to distinguish from non-specific macular degeneration. Intravenous fluorescein studies may help in these early cases by revealing considerable pigment disturbance otherwise invisible.[74] As the retinal degeneration progresses there is diffuse depigmentation in the peripheral areas and finally narrowing of the retinal vessels. There is no absolute correlation between the appearance of the macula and the patient's vision.

Very early cases of retinopathy may recover if therapy is stopped promptly[75,76] but most cases do not recover and some progress.[70] Most patients who develop retinopathy do so while taking their chloroquine but delayed onset, as long as two to five years after cessation of therapy, is reported.[77] Retinopathy from chloroquine is also recorded in children.[79]

Animal studies have shown that chloroquine is deposited in the choroid and pigmented epithelium of retina, ciliary body and iris in rabbits. Deposition in these structures does not occur in albino rabbits[80] so that pigmentation seems to be necessary for damage to occur. In cats the primary damage from chloroquine is in the retinal pigment epithelium and all other changes are secondary to this.[81] Human cases show segmental disappearance of rods and cones with migration of melanin from retinal pigment epithelium into the inner retinal layers.[82,83]

Clearly it is vital to watch for early chloroquine retinopathy. Unfortunately there is no simple reliable test for early damage. Special tests such as the electro-oculogram have been suggested,[84,85] but the usefulness of this test is denied.[86] The best test is probably the retinal threshold for red light but this requires elaborate equipment which is usually only available in special centres.[87] From a practical aspect, careful visual field studies, colour vision testing, ophthalmoscopy and serial photography of the fundi should probably be done on all patients on antimalarials. The macular dazzle test may be useful but is very variable and its place is not definitely established.

It is reassuring to note that only a few cases of retinopathy are reported in which the chloroquine intake was 250 mgm daily or less or that of hydroxychloroquine 400 mgm daily or less. Cases of retinopathy are also rare in patients who have taken antimalarials for less than one year.

Treatment of an established case is difficult because the damage to the retinal receptors is already done by the time the condition is diagnosed. However, Rubin et al[88] have shown that the urinary excretion of chloroquine in humans who have the retinopathy can be increased fourfold by administration of 8 Gm of ammonium chloride daily and

further increased by the use of dimercaprol (BAL). This is logical treatment and although its efficacy is not absolutely proved I would not hesitate to use it.

Adverse Effects of Oral Contraceptives

The consumption of oral contraceptives is increasing each year. Adverse effects in the form of thrombo-embolism have been reported in numerous cases since 1961 but it was not until 1964 that possible ocular effects were reported by Walsh who had noted papilledema in a patient taking norethylnodrel (Enovid). In 1965 Walsh et al[89] gave details of numerous reported case histories of possible side effects from oral contraceptives which included pseudotumor cerebri, retinal vascular occlusions, optic neuritis, visual field defects, papilledema, exophthalmos, sixth nerve palsy and migraine.

Considering the large number of patients taking "the pill", reports of adverse effects on the eyes are rare and many of the recorded cases could be explained by other diseases and may have had nothing to do with the medication. Isolated reports of ocular effects presumably due to the oral contraceptives have appeared in the literature, for example Flynn's report of a patient developing bilateral retinal edema and periphlebitis after taking norethylnodrel for two months. In this case visual acuity fell to 20/200 and "counting fingers" but returned to 20/20 in both eyes after stopping "the pill" and receiving ACTH.[90] On the other hand there are negative studies such as the series of 212 patients using seven different oral contraceptives reported by Faust et al.[91] In these cases careful study revealed no ocular pathology which would not be expected in a random sample of the population. I have seen one patient with macular edema which developed while she was taking "the pill" but it was impossible to prove the relationship between the two.

The question of ocular toxicity from oral contraceptives is by no means settled but there is sufficient recorded information to suggest some association, so one must be on the look-out for patients with eye symptoms which may be relative to these drugs. Often a patient will not mention the contraceptives when questioned about drugs unless the specific question is asked.

Conclusion

A few of the commonly used medications which may harm the eyes have been discussed briefly. It is hoped that some of the points made will serve as useful reminders to those who use potentially harmful drugs and also that no one will be dissuaded from using any drug when the proper indications exist.

It is difficult to keep pace with the flood of new drugs which become available each year and although manufacturers and food and drug authorities make every attempt to release only safe drugs, it is seldom possible to evaluate a drug fully until it has been in general clinical use for some time. Drug toxicity may appear in only a small proportion of patients treated or only after prolonged use. Some diseases may predispose the patient to certain toxic effects of a drug. This is a possibility with posterior subcapsular cataracts in rheumatoid arthritis patients treated with systemic steroids. The importance of recording and disseminating information about adverse effects of drugs is self-evident. Frequently the toxic effects of drugs are trivial, occasionally the effects are fatal but any effect on vision is always serious. To keep the incidence of iatrogenic eye disease at its absolute minimum, one must abide by the well established rules of drug therapy: - only use drugs when they are really indicated, use them in proper dosage and with constant awareness of their possible harmful effects.

Where there is no vision, the people perish:
Proverbs XXIX. 18.

□

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MEDICAL-LEGAL ENQUIRIES

IAN MAXWELL, M.B., Ch.B.

RESPONSIBILITY FOR THE ACTS OF ANOTHER

PART I

A Physician in Private Practice

This column has received a number of queries concerned with the physician's responsibility for patient care when he has delegated this in part to another person.

As the situation depends somewhat on whether the other person is a physician in private practice, an intern, or a nurse, we shall consider this month the questions concerning the case of the second private physician.

Q: *If the radiological films on a patient are mislabelled and the consultant surgeon, who does not know the patient, removes the wrong organ, who can be sued – the radiologist, the surgeon, or the referring physician?*

A: Fortunately this rarely happens. In June, 1967, Lord Beswick reported to the House of Lords that, out of a total of over 2 million operations performed in Britain during 1965, there were eight claims arising from operations on the wrong digit, the wrong side, or even the wrong patient.[1] The Medical Defence Union and the Royal College of Nursing have printed a joint memorandum on the steps which should be taken to avoid these unfortunate occurrences.[2]

With respect to operations on the wrong side or limb, they find that abbreviation of the words "right" and "left" or illegible writing may be to blame. Sometimes the nursing or orderly staff prepare the wrong region without referring to the notes. The practice of referring to fingers by number, instead of by name, is a very dangerous one.

They recommend that not only should the words "right" and "left" be written in full, both in the patient's notes and on the operating list, but also that the site of the proposed operation should be clearly marked with indelible skin pencil, and that a further mark should be made on the right or left side of the patient's forehead.

The question, however, specifically refers to a mislabelled X-ray film and there is no doubt that radiological markers can be displaced or fallacious. Nevertheless, the surgeon is responsible for all his acts, irrespective of any misinformation he may receive, and he is unquestionably culpable in this case. The crux of the matter would seem to lie in the words "who does not know the patient." It is probable that the courts would have little sympathy for a surgeon who allowed himself to be reduced to the status of a mindless, paid technician.

It would seem to be the responsibility of the radiologist to correct mismarking of the films, and that this responsibility is not discharged by simply giving a correctly oriented report. If, in addition to the mislabelled films, his report was also incorrect, successful suit could probably be brought both against him and against the hospital which provided him with his facilities. The attending physician does not appear to be culpable unless he assisted at the operation.

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Q: *If an anaesthetist uses a second-hand ampoule of a spinal anaesthetic and the patient becomes paralysed, can a suit be brought against the surgeon?*

A: Until a few years ago the principle applied that the surgeon, as "captain of the ship" was in complete executive command of the operating room and of all personnel therein, including the anaesthetist. With the emergence of the specialty of medical anaesthesiology this probably does not hold any longer and it is unlikely that the surgeon will be held responsible for the negligent acts of an anaesthesiologist. There seems little doubt that the anaesthesiologist in the present case would be held culpable, in fact judgment for damages of $100,000 have been granted against an anaesthesiologist in just such circumstances.[3] The question arises, however, could the hospital also be sued for negligence? Although it is axiomatic that hospitals are not engaged in the practice

of medicine, it is considered to be their responsibility to ensure that materials and equipment used in their institutions are not defective, and that if certain supplies are liable to rapid deterioration on opening (anaesthetic ether would be a good example), that regulations concerning their handling will be drawn up and adhered to. Under these circumstances, suit might be brought against the hospital if it can be shown to have neglected these precautions. Not only this, but although a hospital as a general rule cannot be charged with the negligent performance of a medical service by a private physician on its staff, in the case of an anaesthesiologist engaging in private practice within its confines, it may be considered his employer if he uses drugs and equipment supplied by it, or if he takes "on call" duty, even though he draws no formal salary from the hospital.

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Q: *If a surgeon discovers an intra-abdominal sponge left by a previous surgeon, what should he do?*

A: By the terms of his contract a doctor has the duty to tell his patient at once if there has been an accident in therapy, either if the accident has been precipitated by the physician's own misadventure or by another following his instructions. Furthermore, if he fails to do so, an action for damages could be brought against him. Although, in the present case he cannot be considered responsible for the original accident, he clearly has a duty to disclose to his patient the true cause of the symptoms which necessitated the second operation.
He should send a copy of his operative report to the original surgeon if this is possible.

References

1. *Brit. med. J.* **1**: 776, 1967.
2. *ibid.* **2**: 1157, 1961.
3. Cited in Hospital Physician (Sept. 1967): 62.

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You are invited to contribute questions to our **Medical Legal Enquiries.**

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