SURGICAL CONVALESCENCE*

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Surgical convalescence is of vital importance on a personal, social, and economic level to everyone concerned with an operation, especially the patient, his family, his employers, and the community in which he lives.

It *is*, fortunately a normal occurrence.

A constant review of ideas which seem easily acceptable, of procedures which seem obvious, and of syndromes which seem commonplace is necessary to appreciate the unusual, the abnormal, or the new. A simple example of this is early, controlled, post-operative activity. Where John Hilton said, in his writings on rest and pain, 'under injury, pain suggested the necessity of, and indeed compelled . . . rest', we say today that pain suggests the necessity of the limitation of activity only to the extent that this causes something more than discomfort.

General reading of the works of Hardy,[8,10] from the University of Mississipi, and of Dunphy,[14,15] and above all of Moore[4,11,12] and the Harvard school, suggests that surgical convalescence may be reviewed under the heading of 6 questions: When does it start? Whence come the stimuli for the whole response to injury? Where lie the origins of the whole syndrome? What are the landmarks of its normal progress? How can *we* aid the whole process to follow its normal course? and, When does it end?

In surgery it is always well to consider the end before embarking on the beginning. Let us start, therefore, with the question: When does convalescence end?

Objectively, convalescence ends when the patient has returned to a normal physical and psychological state of well-being, of effort potential, of resistance to fatigue, and to a state of normal social relationship and of economic usefulness.

Subjective convalescence may coincide with objective convalescence, but the patient carries a scar to the end of his days . . . a sensitive-to-the-weather, touchy, or even adherent scar . . . and is never quite the same after the operation. So, although healed to a tensile strength even beyond that of nearby tissue, the wound remains an area of continuing, special metabolism. In experiments on the recovery of the tensile strength of fascia in wounds, Douglas[3] shows that this process continues for at least a year (Fig. 1).

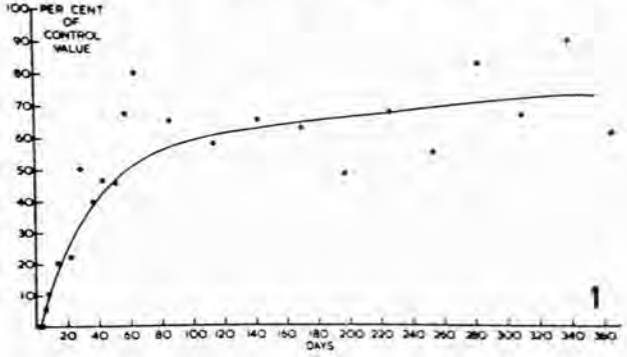

Fig. 1. The continuing recovery of tensile strength in a wound.

In this connection the breaking down of wounds in severe scurvy during the long sea-voyages of exploration comes to mind. In 1772 Lind showed that fresh fruit-juices could prevent such afflictions as beset George Anson in his circumnavigation in 1748, when he recorded that ' . . . old wounds, long healed, broke out afresh', and that ' . . . the callus of a broken bone that had been formed for a long time, was found to be completely dissolved'.

When does convalescence begin? Although it cannot begin before accidental injury, it may, and should, begin before elective surgery. This should be the point of commencement of return to psychological normality.[2] The sufferer's complaints have been patiently and sympathetically explored, his questions answered simply, as far as possible, and his fears have been rationalized. Not every shoal of differential diagnosis, nor treacherous current of prognosis, nor adverse wind of complication has been

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mentioned in discussion, but he is allowed to realize that a course of treatment is laid, a mark is in view, and a pilot has been picked who is competent and who, although aware of his limitations, will never give up the ship.

This pre-operative rapport is most important when it comes to guiding the patient's own evaluation of his post-operative progress, whether in exploring his increasing ability, or in correcting any fearful, needless, or even superstitious self-imposed limitation of activity.

Man's actions may be wise or they may be foolish, futile and self-defeating, but they are not without purpose. For example, a type of person, whom we all know too well, has an attitude of fantasied atonement for fantasied guilt towards pain and disease, and approaches an operation fearful of injury but confidently expecting it. Such a person awakes convinced that the day of reckoning has dawned and, even though he is apparently doing well, feels that he is liable to all sorts of complications and to permanent weakening of his constitution. He seemed stupidly difficult to convince of the obvious need for an operation for a hernia or a gastrectomy in the first place, and now the surgeon is sorry that he ever tried! If he didn't establish firm contact pre-operatively, he has no hope of achieving it now. In case it is felt that this psychological preparation for physical stress is exaggerated, it may be well to recall that Hill and others[3] found that the coxswain and coach of the Harvard boat in 1953 and 1954 gave just as much evidence of adrenal cortical activity as the rowers themselves after a 4-mile race. Adrenal cortical activity, incidentally, interferes with the normal contraction of a wound—which will be mentioned later—as does excess secretion of thyroxin which may of course accompany the excited state.

Physical convalescence commences with the act of wounding. The severity of the wound and the size of the definitive procedure will modify the duration of the whole response, and so will sepsis, haemorrhagic oozing and, in trauma, imperfect wound cleansing. Removal of body fluids by enteral drainage, especially if done to an injudicious extent, and fistulous depletion will also modify the course of convalescence.

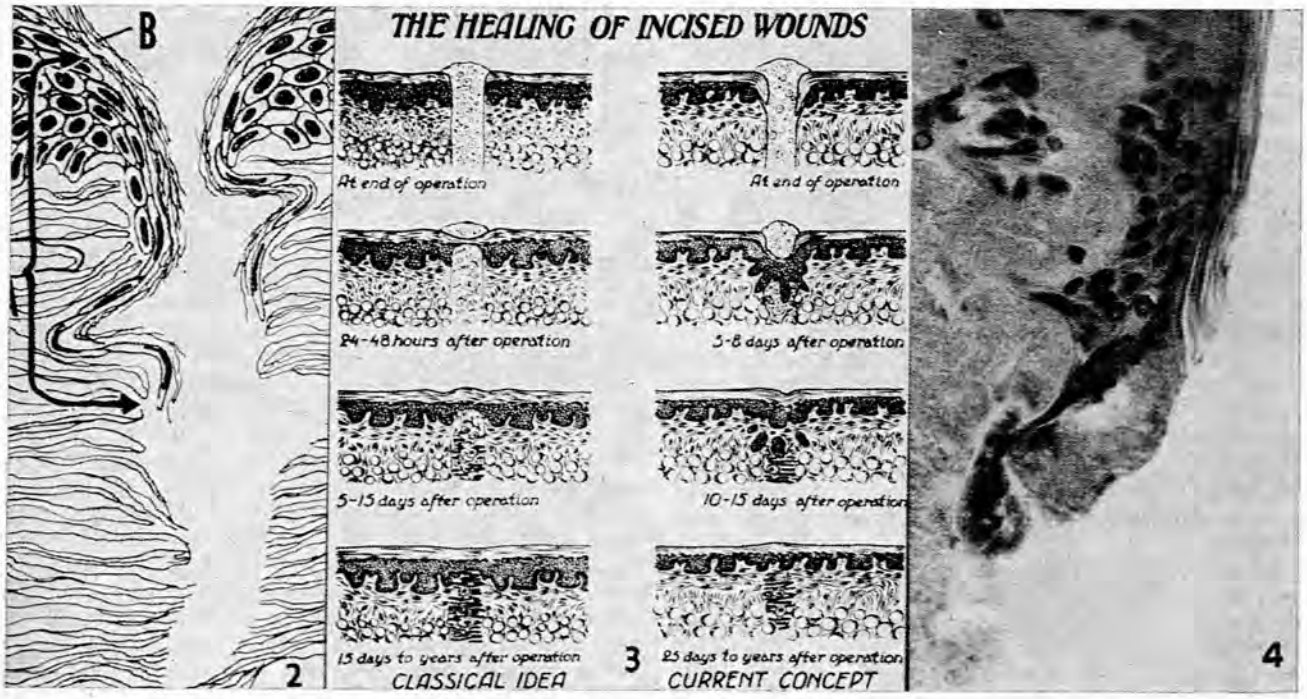
What are the landmarks of the normal progress of convalescence, whence come the stimuli for the response, and where lie the origins of the whole syndrome? According to Moore[4] there are 4 phases, viz. (1) The phase of injury, (2) the phase of returning biochemical balance or homeostasis, (3) the phase of spontaneous nitrogen anabolism and returning muscle strength, and (4) the phase of fat gain and restoration of normal healthy weight.

Some aspects of these phases will now be surveyed in terms of (1) the clinical picture, (2) the picture of the wound itself, (3) the biochemical picture, and (4) the features in the clinical management most likely to affect helpfully these natural, normal processes.

The First Phase

In the first phase the patient is weak, thirsty and listless. He has no appetite, and the limit of his ambition is to avoid pain, to sleep, and to feel a little better than he did some short hour ago.

The wound accumulates an amorphous coagulum of clot, lymph solution, fibrin, leukocytes—especially monocytes—and large immune-protein bodies which are an important part of the matrix of the future collagen and fibrous tissue. There is variable bacterial contamination even under ordinary strict aseptic conditions. The very act of wounding cells has liberated hexosamine, to which reference will be made later. No collagen is detected and no polyblasts, fibroblasts, histiocytes, etc. are found on using the Edwards[5] sponge sampling method.



Figs. 2, 3 and *4*. See text.

Biochemically, however, the healing of the wound has now commenced, although the last-mentioned cells appear in greater numbers from the first day onwards. There is no biological 'lag period'.

Hartwell's 'Law of epithelium'[6,23] commences to operate, and the epithelial cells move down each side of the wound to its depth, covering the raw surfaces. They do this by extending themselves elliptically, by pseudopodic extensions, and by a rolling movement. They do not multiply in number. There is no epimorphosis; there is only morphallaxis (Figs. 2, 3 and 4).

The technique of incision and dissection, and the accuracy of apposition and correct tension of stitching greatly influences this stage.[7] The incision has damaged and dislocated tens of thousands of cells and mingled the intra- and intercellular, and the intravascular contents, and has given rise (amongst other things) to possible auto-immune sensitization effects—an extreme example of which may be found in operations for hydatid disease if anaphylactic shock is precipitated.

Biochemically the patient is retaining water and sodium by withdrawing them from the renal tubules.[8] He is losing potassium, fat, and, fastest of all, his lean muscle mass, which, with his liver, is his greatest protein storehouse.

To explain this correlation of local and systemic response, hormones,[9] 'feed-back mechanisms,'[10] or 'mediators'[4] have been postulated.

What are mediators, what do we know about them, and what are their effects? We don't know what they are; we therefore know very little about them, but Moore[11] discusses their manner of action in the way which seems currently most likely to be clinically helpful. When all this limited knowledge and intelligent guesswork are used as the basis of management of the first phase of convalescence, whether normal or abnormal, we find our results superior to those obtained when we had even less information or more limited ideas to guide us.

'Convalescence is driven by an endocrine engine', said Moore,[12] and in Table I an attempt is made to summarize the concepts which are expressed in different works by Moore, Hardy, and others. It seems that there are endocrine mediators, which, in convalescence, predominate over non-endocrine mediators of the wound itself. The whole process may be likened to a sort of surgical physiological Manicheism—the endocrine mediators are the principle of good and counter homeostatic tensions, while the non-endocrine mediators of the wound are the principle of evil and cause, enhance, and prolong these tensions.

The clinical management of the first phase commences with the proper preparation of the patient for operation, from his psychological outlook to his intracellular electrolytes and his body-fluid contents and their relative proportions, in order to prevent or diminish non-endocrine mediator activity and to give a fair start to what Deaver expressed as ' Cut well, sew well, get well'.

When pre-operative restoration of fluid volume, blood cells, electrolytes, and vitamins has been brought to the best level attainable for the particular patient in the judgment of the surgeon, the sooner the infection, the injury, or the tumour is dealt with, the better. This will avoid an increase of the hazards of toxaemia, tissue anoxia, liver damage, renal damage, and protein, glycogen, and fluid

TABLE I. THE WOUND. FACTORS IN SURGICAL CONVALESCENCE

Infection	*Severity*	*Poor technique*	*Imperfect cleansing*
	↓	↓	
	Wound mediators—non-endocrine		*Neuro-endocrine activation—endocrine mediators*
	1. Loss of normal skin or visceral barrier due to: (*a*) Infection from outside, and (*b*) fluid loss from inside.		1. Medullary epinephrine and synaptic nor-epinephrine control vasoconstrictor response to absorption of the toxaemia (*a*) of infection, and (*b*) 'tissue destruction metabolites', and (?) control of the ACTH response as shown by the eosinopenia for 2 days.
	2. Actual cross-sectional tissue destruction, (*a*) by accident, and (*b*) by operative trauma of all types, especially coarse handling.		2. The antidiuretic hormone partly controls dehydration, especially in poorly-hydrated subjects, and combines with and subsidizes the action of:
	3. Oligaemia due to (*a*) reactionary local oedema at wound, (*b*) reactionary diapedesis of fluid, e.g. from much handled or damaged peritoneum, and (*c*) haemorrhage uncorrected giving inefficient organ function, e.g. kidney, liver, brain; and accumulation of abnormal metabolites, which may contribute to:		3. The 17-hydroxyketosteroids which control nitrogen-metabolism[12] all the better if their function is not diverted to help fluid control, and if there is no cortical focal lipid depletion as in septicaemic infection (Stoner[27]).
	4. Release of intracellular solutes, e.g. enzymes, potassium, etc. into the circulation, and of intravascular immune bodies and other large protein molecules into the tissues, with genesis of auto-immune reactions and anaphylaxis.		4. The aldosterones which are prime movers in fluid retention, and which determine sodium, potassium, and ?other electrolyte values.
	5. Alteration of intestinal motility (Hardy[24]). Alteration of intestinal absorption (Howard[25]). Alteration of gall-bladder function (Howard[26]). Alteration of insulin control with stress glycosuria. All leading, in effect, to starvation.		5. The corticosteroids which may mediate the development of auto-immune bodies, and wound contraction (Grillo[28]).
	6. Hypoxia. ?Impaired vital functions with increase of (2), (3) and (4) above, in gross trauma.		

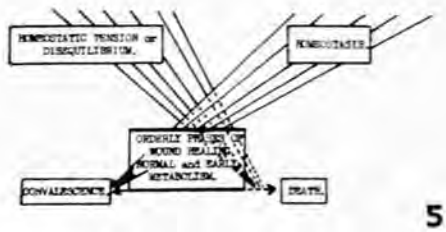


Fig. 5. Graphic representation of factors involved in surgical convalescence.

wastage, which may lead to what Moore calls an 'accretion of biologic components' of such an extent that the very survival mechanisms, e.g. the mobilization of cell-water, will hasten death.

The technique of operation must aim at asepsis, and a constant attempt should be made to damage all tissues as little as possible whether by clamping, crushing, tying, or burning. It should be remembered that an operation is a particular procedure, devised to meet the particular needs of a particular person, even though it follows a general pattern. Sympathy with this approach may bring happier results than adhering to a preconceived idea of a theoretical maximum operative clearance. (As Thomas Fuller might have said, 'the eagle's eye may have to prevent a lion's heart from causing a lady's hand to grab more than it can deal with'.) Soiled wounds and cavities, if unavoidable, must be cleaned and a drain should be used. Stitches and apposition will be mentioned later. To sum up, we may say that the local load of homeostatic response must be lightened to avoid strain upon and depletion of the patient's general endocrine and metabolic resources in favour of the merely local needs.

In the first phase of post-operative care, adequate relief of pain is the most important matter. Sleep, too, is important, but when pain is relieved sleep comes easily. Fluid balance is very simple, provided the temptation to find and then to fill some—usually fancied—endocrinal, electrolytic or protein need is resisted. The routine use of prophylactic antibiotics is wrong. If there are reasonable fears about the asepsis of a wound, then a small corrugated drain is a much better protection. In view of the value of early movement, and the fact that a small ooze of blood into the superficial tissue may result, this drain may be regarded as a double safety valve. Patients should move freely, as soon as possible, and up to the limit of comfort—short of causing actual wound pain.

In many of the very extensive and prolonged modern procedures all these factors demand exacting care for a few days until the end of the first phase. When new theatre accommodation is planned the provision of a post-operative intensive-therapy ward should be considered, where patients who have undergone major surgical operations could spend their first few days. This would lead to great economy in the services of highly trained nurses and to improvement in the use of post-operative physiotherapy.

Post-operative feeding in this phase is unimportant. It is a catabolic phase, and only glucose seems usable as a source of calories at present. Riegel[22] showed that it is important to have a supply of protein and, if possible, calories available when the patient commences to move into the anabolic phase. She is however of opinion that intravenous sources of this supply should be sought, because, up to this point, a scaphoid abdomen and a sense of thirst are preferable to a large quantity of calorie- and protein-sparers being splashed back and forth in a poorly motile bowel.

The actual size of this problem is very difficult to determine. Taylor and Ancel Keys[13] made experiments on healthy young adult males under conditions that simulated injury. They confirm findings by Paquin, cited by Taylor and Keyes,[13] that the rate of nitrogen loss is 2 - 6 (av. 4) g. per kg. body weight per day. Taken over as much as 5 days in a 70-kg. person, this amounts to the nitrogen equivalent of about 5 lb. of the lean-muscle mass. Dehydration can increase this loss up to twice this rate. Inactivity increases the effect of this loss, because not only is the heart muscle losing its substance as one of the larger lean-muscle masses of the body, but it is allowed to get out of condition. The liver loses protein, and so do the large coarse muscles of locomotion, which may account for uncertain balance and weakness if the patient cannot be out of bed by the third day or even earlier, or if he is not taught a proper course of rehabilitative exercises for all his *un*affected muscles.

It is worth noting that in acute peritonitis this lean-muscle mass can lose up to 1 kg. per day!

The Second Phase

This phase of normal surgical convalescence is the phase of returned homeostasis and returning anabolism. It commences from the third day onward depending on the interaction of all the factors mentioned so far. In females it is the time of 'the lipstick sign'.[11]

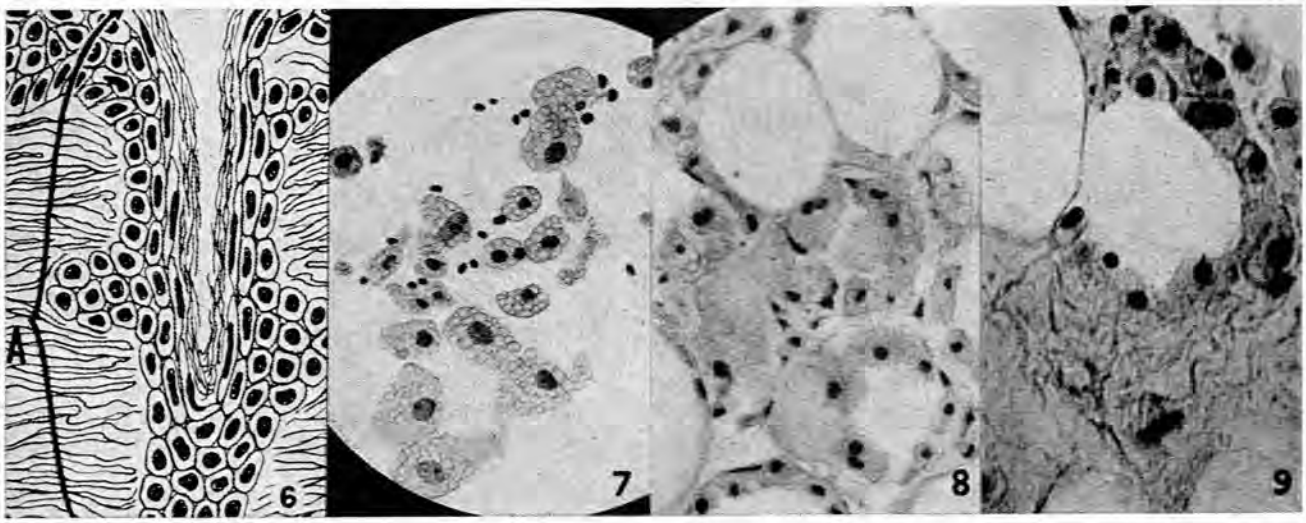


Fig. 6. Epithelialization in the wound (diagrammatic).
Fig. 7. Deposition of small round lymphocytes on fat cells.
Fig. 8. The consumption of fat cells.
Fig. 9. The formation of collagen fibres.

The patient feels better. He suddenly moves much more freely, and the winds of surgical change are to be heard blowing across the ward. His appetite begins to return, his ambitions extend beyond the next hour or two, and his spirit is willing, even if his flesh is still so weak that a 'not-to-be-disturbed' sign turns away late arrivals among his well-wishers and visitors.

In the wound itself epithelialization is almost complete by morphallaxis, as already described, and the subepithelial layers commence mitotic division and raise the epithelium to the surface by epimorphosis[6,14] (Fig. 6).

Tensile strength, which is probably achieved by the co-ordinated laying down of collagen, and the contraction of the wound, is now becoming apparent.

Collagen appears as follows: Of all the cells that stream into the wound margin, the small round lymphocytes are the commonest. They promptly digest the nearest fat cells, or areolar-tissue cells, and swell and become polyblastic cells, or histiocytes, or macrophages, or possibly so-called primary connective-tissue cells[6] (Fig. 7). These cells continue to grow by consuming fat cells and then the amorphous coagulum in the wound line (Fig. 8). They grow so large that they rupture, and collagen is laid down all through the wound line and the damaged layers on each side adjoining it. Obviously, any operative dissection of these areolar and fascial planes further than the minimum necessary for the operative approach is biologically harmful to healing. The collagen fibres are actually preceded by fibres of pre-collagen which are visible through the electron microscope, which arrange themselves in echelon and which coalesce about 5 at a time to form the microscopically visible collagen fibres[15] (Fig. 10). Obviously, the more

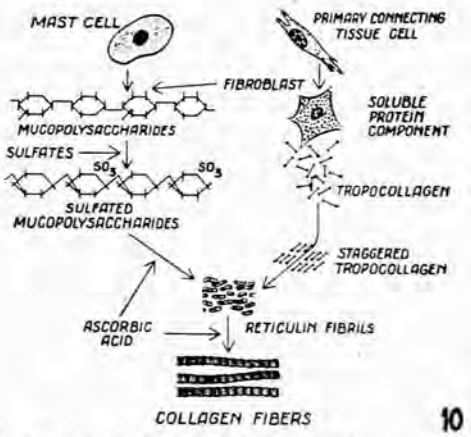


Fig. 10. Hypothetical model of fibrogenesis.

precise the apposition of corresponding layers in the wound and the closer the co-aptation, the less the wound space (in view of what has been described, it can scarcely be called the 'dead space') and the better the chance to establish tensile strength and anatomical integrity as shown by Le Gros Clark in experiments in muscle suture.[7]

Organization of the collagen depends on the ingrowth of a scaffolding of endothelial cells from the underlying capillaries which are sealed with blood clot. These form capillaries themselves. A blood supply to the wound space commences. New fibrin, new protective polymorphs, and new repair cells of all sorts are deposited around the framework. As these cells do their work and become effete they

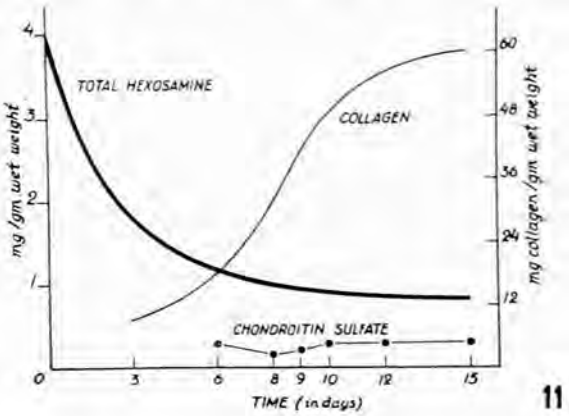


Fig. 11. Hexosamide, chondroitin sulphate and collagen formation in the healing wound.

in turn are consumed by new monocytes, and their polyblastic offshoots, which again swell, burst and form pre-collagen and collagen, but the quality of the pabulum is not as good as it was at first, and the process slows down (Fig. 11).

Negative nitrogen balance does not affect this process.[15] Lack of vitamin C retards it both as to collagen deposition and endothelial ingrowth. Protein starvation, in the clinical sense, affects the processes slightly, if at all, but gross alteration of the serum proteins does interfere markedly by alteration of the osmotic properties of the serum. The reason for this lack of effect may be the fact that sulphur is conserved in protein starvation after wounding. Cortisone in excess does not affect the laying down of collagen, but it does affect contraction of the wound, i.e. the aggregation of this collagen into fibrous bundles which give tensile strength. Thyroxin in excess weakens the tensile strength in the normal closed wound. Irradiation seems not to affect these processes until they are well established and then the effects are those of hypoprothrombinaemia.[15]

A wound in a cachectic, toxic person with absolutely no body fat is seldom seen, but it would not be surprising if the absence of fat interfered with the earliest stage of the processes described. 'Well begun is half done' is truer of wounds than of any other state of affairs, as a brief consideration of wound dehiscence will show.

Wound dehiscence is not part of normal convalescence, but it is always interesting and, of course, is used here to emphasize some of the processes referred to from a clinical standpoint.

Wound dehiscence occurs as frequently in the apparently well-nourished as in the apparently cachectic. The time of its occurrence may be altered by suturing methods and a variety of bursting strains, but there is almost certainly some basic deficiency in the promptness of the earliest healing responses which is not understood, but which is overcome by the time re-suture is needed. Prompt re-suture produces a rate of healing which is little delayed, and a quality of healing which shows a relatively greater tensile strength after one week than would normally be expected, according to experimental investigation.[16] In my opinion this effect

is enhanced if the wound is re-sutured in one full thickness with no re-dissection of layers, and this may be substantiated by what has been said above. It is also my impression that wound breakdown seems more common in persons who for any reason, possibly chiefly emotional confusion and fear, are putting out an undue share of adrenal medullary secretion, or who are, of course, on cortisone therapy.

A useful clinical feature follows from all this. If re-exploration for any reason must follow recent operation, the original wound should be used. It heals faster,[16] it bleeds less, and the patient is less affected than by a new wound.

Biochemically the antidiuretic effect ceases, and a sodium and water diuresis commences. Intravenous nutrition ceases. Potassium loss ceases. Otherwise the biochemical state is based on the decreasing loss of nitrogen and the absence of a positive nitrogen balance in the absence of an adequate exogenous nitrogen intake. Urinary nitrogen falls to normal, after having been raised to 4 times the normal level.

The clinical management must allow for the loss of up to 150 mg. of protein nitrogen, equivalent to about 2 - 3 kg. of the lean-muscle mass. As early as possible the daily intake of food must be about 1,500 calories, including about 1·5 g. of protein per kg. of body weight per day. Most patients can start with 40 - 50 g. of protein, 75 g. of carbohydrate, and 120 g. of fat per day from the third to fifth day. Of course, during the whole convalescence the patient should receive up to 1,000 mg. of ascorbic acid per day, according to Dunphy.[15]

The patient is strongly encouraged to increase his ambition, to think positively about the future, to be active, and to eat well.

Activity does not necessarily mean getting out of bed, which may in any case be impossible, but it does mean active physiotherapy, rehabilitative exercises, and if only it were possible, short-term occupational therapy. So simple a device as a strong bandage tied to the foot-rail of the bed will allow the patient to haul himself upright with no strain on his abdominal muscles, which will both encourage him and give him some exercise. A bed-head 'hockey stick and polly-perch' can provide considerable exercise with no strain on the abdomen.

All this exercise uses calories and this must be allowed for. The patient should be allowed to eat as much as he feels inclined to unless there is some severe contra-indication, and in that case it must be relieved as soon as possible.

The Third Phase

The third phase of spontaneous anabolism and the regaining of muscular strength usually commences at about the eighth day. The patient walks fairly easily. Not only is his spirit willing, but his flesh is now stronger. He is fit to go home if the circumstances of his home are good, and his stitches are removed about this time.

In the wound the epithelium is being thickened by epithelial mitosis, as described, and the scar broadens superficially although contraction is occurring in the deep area. As this progresses the wound scar bulges above the surrounding level of skin.

A fascinating problem arises from this process: What stops it? What interrelation of epithelialization, granulation, and the organization of the stromatous ground substance of the wound comes into play to prevent the process of wound healing continuing and behaving like a sarcoma? Why do wounds not all become keloid?

Dunphy[14] (1958) suggests that in seeking the explanation for the start of the neoplastic process, we should cease to think in terms of the cell only, and that we should pay more attention to the connective stroma around it. He points out[15] that the stoppage of epithelial proliferation and its regression to a normal scar resembles the regression of the epithelium of the breast in carcinoma under the influence of hormone therapy.

This phase of wound healing may in some respects last for a year or longer, as stated earlier, and this far exceeds the period of apparent return to normal general metabolism. In this may lie the explanation of the fact that, although in modern times the patient's initial progress is so much faster, the end point of convalescence is reached after much the same interval as in days gone by.

In fact, human tissue heals by the setting of human glue, and the rate of the reaction has, in normal circumstances, probably remained unchanged since man began. As Iago says:

> 'How poor are they that have not patience,
> What wound didst ever heal but by degree?'

Biochemically the patient is replacing 3 - 10 g. of nitrogen, equivalent to about 100 g. of lean-tissue mass per day. As he has lost about 2,000 g., this should only take about 20 days. Actually this dietary intake probably does not meet his increased caloric need, and nitrogen replacement lags behind this standard and endogenous fat breakdown continues.

The patient, therefore, benefits from muscular activity, but must use for this only a modest portion of his caloric intake. He may and should return to modest physical work, but there is good ground for telling him to 'take it easy'.

The Fourth Phase

The fourth and last phase of convalescence is the return to normal physical habitus and weight. Little is known about the biochemical activity of mediator mechanisms in this and the previous phase, and the opportunity for investigation is relatively limited. Post-operative weight may be quite well below pre-operative level, because before the operation the patient was eating, consciously or unconsciously, beyond his needs, e.g. in tuberculosis or in taking frequent meals and collations so common in the treatment of duodenal ulcer. This lower post-operative weight may be nearer the true desirable weight for the patient.

CONCLUSION

While passing through this jungle of physiological thought and endocrinal speculation, it is easy to forget to treat the patient as a whole man. Karpovitch[17] in an orthopaedic hospital found that young men on an average halved their recovery time when full use was made of all possible ancillary rehabilitation services such as rendered by occupational therapists, social workers, and physiotherapists. The establishment of an experimental post in each ward similar to that of an air hostess should be worth while. In a teaching hospital the constant ebb and flow of the tide of student and staff rounds relieves boredom to a great degree. We have unsuspected entertainment value for our patients.

Nevertheless, a patient in hospital suffers for 10% of his time from pain, and for 90% from boredom.[18,19]

The New York City Hospitals Department is converting areas of certain hospitals no longer required for acute care, e.g. tuberculosis hospitals, into homestead wards, with the emphasis on the recreational, emotional, social, and rehabilitational needs of patients, with a reduction in maintenance costs by over 50%. In 1957, together with Prof. C. E. Lewer Allen, of Cape Town, I made a limited and preliminary study of this sort of approach in Cape Town in the hospitals in the southern suburbs. The average duration of hospital-bed days increased by about 7% compared with 1956. The reason was thought to be the increasing number and increasing severity of traumatic orthopaedic work. Taking this into account and considering also the other types of surgical convalescent patients, it seemed a reasonable conclusion that the establishment of a convalescent or homestead type of hospital consisting of 25 beds for European males, 60 beds for European females and 60 and 30 beds for non-European males and females respectively, i.e. a total of less than 200 beds, would enable the present hospital accommodation for acute cases to cope with the situation and to reduce the large waiting lists to a treatable size. The survey showed that 262 beds were available at that time for the treatment of acute cases.

The daily cost of such beds at that time was 74s. a day for 'acute' beds, and 19s. 8d. for actual existing 'convalescent' beds. The saving effected by creating such an approach would pay a whole body of workers in the ancillary convalescent surgical services.

Howard Rusk[20] says of this part of surgical convalescence; 'If man doesn't use his potential, he vegetates. If a patient has work to do, someone interested in him, congenial company, stimulating and competitive tasks, then he has a desire to live and not to be merely alive, and this is essential to endocrinal, emotional, and physical convalescence'. As soon as the second phase commences we must stimulate the will to live.

The cost of idle convalescence in every aspect, from fees paid to hospitals and doctors to the loss of productive gainful employment, far outweighs its benefits. Bartels and Johnson[21] made a valid comparison of the duration of convalescence in patients of a group of surgeons divided into the following categories: (A) private, chiefly self-employed, patients, (B) military pensioners, and (C) casual seasonal and day labourers. In units of time the groups were convalescent in the ratio of 3 : 4 : 3½, when the date of return to work was set by the doctor. When the patient was allowed to go back to work when he himself felt fit, he did so in abouth 5/8ths of the above times, but the proportions between the types of patient remained about the same, viz., 2 : 3½ : 2, on the same basis of calculation. Those who favour a welfare state will notice that any relative shortening of convalescence within the groups is most noticeable in the private self-employed category.

In conclusion, when we survey the wide field of surgical convalescence and admire the steady progress of investigations into the healing process, by the use of which we may better treat our patients, or better understand some phases of the cancer problem, we can say with Clough, that although we

> 'Seem here no painful inch to gain,
> Far back, through creeks and inlets making,
> Comes silent, flooding in, the main.'

I wish to thank the authors and editors concerned for their kind permission to reprint the following figures:

Fig. 1 from Douglas, D. M. (1952): Brit. J. Surg., **40**, 83.

Figs. 2, 4, 6 - 9 from Hartwell, F. S. (1956): *The Mechanism of Wound Healing*. Springfield, Ill.: Charles C. Thomas.

Figs. 3, 10 and 11 from Dunphy, J. E. (1960): Ann. Roy. Coll. Surg. Engl., **26**, 72, 79, 82.

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