Bulletin of the

University of Minnesota Hospitals
and
Minnesota Medical Foundation

Deterioration of the Bed-Fast Patient

Volume XXII | Friday, April 25, 1952 | Number 24

BULLETIN OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
and
MINNESOTA MEDICAL FOUNDATION

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Published weekly during the school year, October to June, inclusive.

The Bulletin is sent to members of the Minnesota Medical Foundation. Annual membership fee - $10.00.

Address communications to: Staff Bulletin, 3330 Powell Hall, University of Minnesota, Minneapolis 14, Minn.

I. DETERIORATION OF THE BED-FAST PATIENT

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One of the largest items of medical care today is the cost of care of the chronically ill or disabled patient. Of this expense, only a small fraction goes for direct medical care to the patient. The major part of this cost is the cost of hospital or nursing home care made necessary by the patient's disability. In many cases the disabilities which make nursing home or hospital care necessary are not the direct result of disease processes but are secondary changes due to deterioration of the patient while he was confined to bed. These deteriorative changes persist after the patient has recovered from the direct effects of the disease process. If these secondary changes cannot be corrected, the patient remains disabled. Patients are continually being referred to this hospital for treatment of these deteriorative changes which developed as the result of being confined to bed. If deterioration of the bed-fast patient can be prevented, convalescence of these patients would be shortened and the cost of medical care would be reduced.

In recent years there has been an increased emphasis on getting patients out of bed as quickly as possible. This early ambulation has been beneficial in preventing deterioration due to inactivity. This program of early ambulation has been carried on most vigorously on the surgical services. On other services, particularly for diseases which have a protracted course, prevention of deterioration of the patient may be neglected. The military services and the veterans administration have demonstrated that an active program to prevent deconditioning of hospital patients decreases the hospital and convalescence time.

On this background, the program of rehabilitation of the physically disabled in civilian hospitals has developed. Rehabilitation connotes the restoration of the patient to his fullest possible capabilities. This includes consideration of his physical, mental, social, vocational and economic status. To be practical and economical, a rehabilitation program must be completed for each patient as quickly as possible. A planned program decreases not only the time required for hospital care but also the time during which the patient is non-productive. To stop the rehabilitation program before the patient has achieved his reasonable goal of independence, in order to reduce hospital costs, is false economy. Such a patient then remains dependent and a continuing expense to his family or his community.

Not all patients can be restored to full productivity in modern industry. However, a large proportion of physically handicapped patients by proper medical treatment, vocational training and intelligent placement can become economically self sufficient. The Federal Office of Vocational Rehabilitation has demonstrated year after year a wage recovery from rehabilitated workers which far exceeds the cost of rehabilitation. Other patients can become partially self supporting doing piece work production at home or in sheltered workshops. For other patients, cost of care can be reduced appreciably by teaching them to care for their daily needs. This decrease in cost of maintenance of the physically handicapped patients may represent a sizable proportion of any county welfare budget and through it a saving to the community.

Rehabilitation, to be most successful, requires the maximal degree of recovery in minimal time. Such a successful program requires planning in the following categories:

1. Minimization of deterioration of the patient in the early stage of his illness.

2. Efficient use of the patient's time for therapeutic activities.

3. Coordination of all of the necessary therapies to insure continuous progress toward rehabilitation.

This essay concerns itself with the first category, the minimization of deterioration of the patient in the early stage of his illness.

The basis of the development of functional ability by any organ of the body is use. In response to the demands placed upon it, within physiological limits, each part of the body increases in functional ability. Conversely, inactivity or non-use results in regression of the organ with loss of ability to function. Rest constitutes a period of non-function and during prolonged rest of an organ we may observe the progressive loss of ability or deterioration of that organ. During disease or following injury to part of the body, we may observe that its functional ability is greatly decreased. At such times the normal physiological demands on that part may exceed its ability to respond. In such cases in order to protect the part against further damage by exhaustion or to preserve homeostasis, it is necessary to protect the part by rest. In this case rest becomes functional by reducing activity to the level within the capacity of the part. Under these conditions, the metabolism of the injured organ is sufficient to restore the organ and to meet the diminished activity load. As the organ increases in ability as it is restored toward normal, its metabolic reserve increases and if the load upon it is increased at the same time its capacity to do work will increase progressively. Although this principle is demonstrated most easily for voluntary muscle, it appears to apply to other organs equally.

If bed rest is prescribed following injury rather than rest or protection only of the injured parts, there will be a progressive deterioration of the normal parts of the body while the injured part or parts are protected. This loss of functional ability of normal parts of the body by disuse may become a serious factor in producing disability in patients who have a disease or injury requiring prolonged immobilization. During the time of enforced rest to protect the injured part, the uninvolved parts deteriorate to the point that weeks or months of treatment may be necessary before normal function is re-established. Therefore, when rest is prescribed for therapeutic purposes its effect on all parts of the body should be evaluated. Adequate protection must be given to the injured part, but activity should be maintained in normal parts of the body to prevent or minimize their deterioration.

There are many times when normal parts of a patient's body deteriorate faster than the injured parts heal. In some cases permanent disabilities result from these degenerative changes of parts of the body not involved by the original disease or injury. In other cases many months of restorative therapy are necessary to return these deteriorated parts of the body to normal function. In either case rehabilitation to independence is prevented or delayed. If the activities of the uninvolved parts of a patient's body can be maintained during the time he must remain in bed, his convalescence will be shortened and the cost of his care decreased.

The common types of deterioration in patients confined to bed are:

1. Loss of mobility.
2. Loss of muscle power and endurance.
3. Circulatory deterioration.
4. Ischemic ulcers.
5. Metabolic imbalance.

Loss of Mobility

In the normal process of breakdown and repair of body tissues, there is a continual removal and replacement of collagen fibers. In the subcutaneous tissue, around joint capsules, in muscle planes and other moving parts of the body, this collagen is laid down as loose areolar connective tissue. This loose connective tissue allows a considerable range of motion to occur between the moving parts. However, if a part of the body is immobilized there is shortening and tightening of the connective tissue as it is laid. The collagen fibers form a dense, hard scar or contracture. This fibrosis in the joint capsules, fascia, and muscles

causes loss of normal joint mobility.

Certain factors are known to alter the rate of this fibrosis. Impairment of the blood supply hastens the rate of fibrosis. Trauma and edema both limit motion and cause fibrosis to occur more rapidly than normal. The rate of replacement of collagen is not known. However, there is evidence that gross changes in collagen deposition may occur in four to five days[1]. By the end of three weeks the collagen scar between two immobilized tissues is very strong. The implication is obvious that fibrosis develops in the moving parts of the body - the joints, tendons, and muscles - when they are immobilized. To prevent this fibrosis the extremities should be moved through the full range of motion each day.

Clinical experience has shown that intracapsular or intramuscular fibroses or contractures are particularly difficult to combat when they occur in certain joints and their associated muscles. Several factors play a role here:

1. Joints which are not easily moved through the full range of motion in bed develop progressive limitation of motion. This is particularly true of the hip which is kept in partial flexion whether the patient is lying on a soft bed, semi-reclining, or sitting. Pillows under the knees increase the flexion in the hip and maintain flexion in the knee.

2. Any joint that is painful will be protected against full motion unless special exercises are supervised.

3. The weight bearing joints must be extended fully for walking otherwise great muscular strength is required. Contractures of the hips, knees, or ankles prevent many patients who have muscular weakness from standing and walking.

4. Some joints are extremely difficult to stretch. The hip flexors cannot be stretched adequately because it is impossible to immobilize the pelvis. The ankle is very difficult to stretch when there is an equinus deformity because the shortened gastrocnemius is so powerful.

5. Contractures developing in the direction of the pull of gravity are difficult to stretch out because gravity tends to enhance the contracture continually and resist the stretching. On the other hand, gravity helps by its continual action to stretch out anti-gravity contractures.

It is evident that the difficulties are great when one tries to overcome fibrous contractures. It is much easier to prevent them by timely precautions. Preventive treatment may be divided into two types:

1. Proper positioning.
2. Activity.

Both types should be used together for optimal results.

A bed which provides adequate support in the proper position to prevent contractures is referred to usually as an orthopedic bed. The essential features are a firm flat mattress, a bed board and a footboard. A bed with an ordinary mattress on an ordinary spring allows sagging of the hips of two to five inches. The hip flexion which occurs when the patient lies supine in such a bed is usually not apparent. However, if the patient is immobilized in this position or if he is bedfast so that the hips are not extended beyond this position, contractures produce a flexion deformity. The sitting position during the day increases this shortening in the flexor fascia. To provide a firm foundation for the mattress a 3/4" bed board is substituted for the bed spring. On this is placed a firm mattress, either a good felted mattress or a very firm inner-spring mattress. Such a bed prevents the common deformities of flexion of the knees and hips and rounding of the back and shoulders which

may occur in a sagging bed.

An orthopedic bed should be equipped with a solid foot board to prevent foot drop. The sole of the foot against the board should be perpendicular to the long axis of the leg. A good footboard should be adjustable to any position along the bed to accommodate patients of varying height. It should be possible to adjust the footboard beyond the lower end of the mattress so that the patient's heels are off the mattress when the bed is flat. Also when the patient lies supine, the feet should extend beyond the mattress in the anatomical position. A pillow should not be placed between the footboard and the feet or the value of the board to prevent drop foot is lost.

Maintenance of full range of motion of all joints and flexibility of the soft tissues is an important factor in preventing deterioration of the bed-fast patient. Joint mobility is dependent upon full flexibility of the surrounding soft tissues. When joints are immobilized the lack of motion in the surrounding joint capsules, connective tissue, and muscles allows fibrosis to occur tying these structures tightly together. Consequently, joint motion is limited and the patient is handicapped. Only by continued motion between these tissues can this fibrosis be prevented. Active motion, where it is possible, is the simplest way to maintain mobility. Simple calisthenics, to be done several times daily, can be prescribed for the bed-fast patient. Passive motion carried on by a nurse or therapist is also useful when necessary. All joints which need not be immobilized should be moved each day. Joints or extremities which need support should be moved passively by a trained therapist. Painful joints frequently can be moved more freely if the extremity is partially supported. For these cases, a Thomas caliper with a Pearson attachment, counterbalanced or attached by pulleys to a hand grip, can be used for assisted active motion while adequate support is being provided to the extremity. Hip and knee motion may be maintained in this way with minimal stress on a fracture site. Suspension slings, either counterbalanced or connected to a hand grip, also may be used to provide assisted active motion so that the patient may move more easily and frequently. Bed skates gliding on a powder board also make it possible for a patient to carry on motion of the hips and knees with gravity and friction largely eliminated.

When joints may not be moved because of fractures, the motion must be confined to the soft tissues. In these cases maintenance of flexibility of the soft tissues is especially important. Trauma has caused extravasation of blood into tissue planes. Fibrin has precipitated and formed networks on which collagen can be laid down. Edema decreases both the flexibility of the tissues and the nutrition to the cells. All of these factors tend to produce dense scarring. As a result joint motion may be lost although no trauma has occurred to the joint surfaces. Massage is useful in these cases to aid the venous and lymphatic return, reduce edema, and maintain the flexibility in both muscles and periarticular soft tissues. Dense scarring is minimized by daily massage during the period of immobilization. Therefore, the residual disability after bony union has occurred is decreased. Electrical stimulation to cause muscle contraction or voluntary muscle setting also is valuable to maintain the flexibility and strength of the muscles.

If a patient is severely ill, as the patient with acute myelitis, or has a very low cardiac reserve, passive motion is the method of choice to maintain range of joint motion. Gentle passive motion increases the metabolic demand very little, far less than the increase caused by eating, yet if all joints are gently carried through their range of motion twice daily, contractures can be prevented. In patients with acute myelitis or coronary occlusion, it is common to see contractures develop in the shoulders, hips, and knees during the acute stage of the disease when the patients are bed-fast. Later, during convalescence, these contractures are a handicap in recovery.

Other aids may be used on any bed to increase the patient's activity and independence. A trapeze bar suspended from a jury mast at the head of the bed is of great assistance to the patient when he wishes to move. It also provides exercise for his arms. The bar should be hung in such a way that it is level, provides firm support and will not slip. For the more seriously disabled patient, a trapeze bar, 4 feet long, suspended from a Balkan frame is more valuable. If the bar extends over the side of the bed, the patient may use it to swing himself from the bed to a wheelchair and back, and so become more independent.

Loss of Muscle Power and Endurance

The power or strength of a muscle is the maximum force which that muscle can exert. The maintenace of powerful muscles is dependent upon the muscle producing maximal tension at frequent intervals. In response to the production of this maximal tension, the muscle fiber hypertrophies and its power increases. Only a few contractions each day which produce maximal tension are necessary to retain the power of a muscle. However, if these strong contractions are not produced the muscle gradually loses power. Many weak contractions will not substitute for a few strong contractions to maintain muscle power. Patients who are confined to bed for a prolonged period of time frequently do not have the opportunity to contract their muscles strongly against resistance and, consequently, the muscles atrophy and become weak.

A muscle fiber can contract to about 50 per cent of its normal resting length. As the fiber shortens during contraction, the force which it exerts decreases progressively until at a full contraction the force is zero. Experimental studies[2] have shown that maximal force is exerted by a muscle during an isometric contraction at its normal relaxed length. As the muscle fiber is stretched beyond its relaxed length, its power begins to fall off. Therefore, contractions of a muscle in its position of advantage, i.e., at its normal relaxed length, should be most useful for maintaining muscle power.

Certain types of disabilities produce the situations which favor the physiological deterioration of the muscle and cause the rapid development of weakness and atrophy. The inhibition of muscular contraction or joint motion because of pain causes rapid atrophy. Immobilization of a muscle in its shortened position in a cast or splint prevents the development of tension in the muscle and causes atrophy. Immobilized paretic muscles atrophy quickly. A muscle which is short because of section of the tendon is unable to exert maximal tension and undergoes atrophy.

Endurance of a muscle is to a large extent a problem of adequate nutrition and excretion. If the circulation to a muscle is not adequate to supply its needs and if the muscle cannot get rid of its wastes, endurance is poor. Many contractions of a muscle at a relatively low resistance (10-25 per cent of its maximum power) are most useful in building up endurance. Since venous and lymphatic flow are increased by external compression of the vessels, contraction of a muscle through its full length aids the lymphatic and venous circulation and helps to maintain endurance of the exercising muscles.

To maintain power and endurance in his muscles, the patient who must remain in bed for a prolonged period of time should be given bed exercises to do. For this purpose a Balkan frame is useful. Weights suspended from pulleys provide resistance for arm or leg exercises. With a full frame, necessary adjustments can be made to exercise any group of muscles. The trapeze bar can be used as an exercise bar for the arms and shoulders. Sit-up or push-up exercises can be used to strengthen arms and trunk. Nevertheless, the patient should not be kept in bed any longer than necessary but should begin standing and ambulation as quickly as possible.

Circulatory Deterioration

Changes in circulatory control and

performance are the most dramatic of the deteriorative changes due to bed rest. The circulation through the autonomic nervous system adapts to stresses placed upon it. If the normal postural stresses are removed, the ability to adapt decreases. Injury, disease, or fever make the circulation less able to adapt to stress and hasten the deterioration. The adaptation of the circulation to upright posture deteriorates quickly with bed rest. Taylor, Henschell, Brozek and Keys[3] found that when healthy young men were put to bed for 21 days, there was a deterioration of the cardiovascular ability to respond to the upright posture which had not been regained after five weeks of activity. Table I, reproduced from their work, shows a relative tachycardia at rest after 21 days of confinement to bed, and a pronounced tachycardia when tilted to 68° on a tilt table. There was also inability to maintain the blood pressure when erect, although the signs of strong sympathetic nervous activity - palmar sweating, pallor, and restlessness - were present.

The restoration of the cardiovascular system to its normal adaptibility required from five to more than ten weeks. Patients who are older than these subjects or who have been ill may show more rapid deterioration and recover more slowly. Painful congestion of the feet and legs, vertigo and fainting are common when the patient assumes the erect posture after only a few days in bed. Prolonged bed rest always causes these symptoms. The painful tingling and burning of the feet and legs may prevent the patient from resuming full activity for weeks after prolonged bed rest. Pressure bandages reduce the congestion and help to relieve pain. In older patients, the re-establishment of the orthostatic reflexes may occur very slowly and delay the return to normal activity.

As a person remains bed-fast, the cardiac rate at rest increases progressively. Taylor, et al, found that in healthy young men confined to bed the cardiac

Table 1

Average pulse rate and systolic blood pressure responses to tilting to 68° on tilt table (including the Crampton postural adjustment 'score') of six men before and during recovery from bed rest.

Condition	Measurement	Control		Days of Recovery From Bed Rest						Final[1]
				1	2	8	9	15	16	
Supine	Pulse rate, beats/min.	50	51	65	62	58	57	61	60	52
Tilted		63	64	102	89	90	86	84	81	71
Difference		13	13	37	27	32	29	23	21	19
Supine	Systolic B.P., mm. Hg.	113	113	120	113	115	110	114	110	112
Tilted		111	113	106	109	109	109	107	108	109
Difference		2	0	14	4	6	1	7	2	3
Crampton score		56	63	-2	33	21	39	23	44	46

[1]Observations made 36 to 72 days after recovery from bed rest.

From Taylor, H.L., A. Henschel, J. Brozek and A. Keys: Effect of bed rest on cardiovascular function and work performance. Journal of Applied Physiology. 2:223, 1949.

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Table 2

Pulse rate in bed by averages of successive two-day periods for 6 normal young men.

Days	Time	G.W.	L.B.	D.M.	R.M.	E.S.	A.W.	All
1- 2	A.M.			51.0	48.0	44.0	45.0	47.0
	P.M.	62.0	53.5	56.0	53.0	45.0	55.0	54.1
3- 4	A.M.	51.5	47.5	48.5	45.0	43.0	47.5	47.2
	P.M.	66.5	58.0	55.0	55.5	43.0	48.0	54.3
5- 6	A.M.	50.0	44.0	50.0	52.0	42.5	44.5	47.2
	P.M.	67.0	50.5	56.0	58.0	46.0	57.5	55.0
7- 8	A.M.	56.5	47.5	53.5	53.5	46.0	47.5	50.8
	P.M.	74.0	54.0	58.0	63.5	50.0	54.0	58.9
9-10	A.M.	56.5	50.5	53.0	52.0	46.0	49.0	51.2
	P.M.	63.0	57.0	55.5	64.0	50.5	53.5	57.3
11-12	A.M.	60.5	51.5	59.5	54.5	47.0	46.5	53.3
	P.M.	65.0	59.5	55.0	59.0	53.0	51.5	57.2
13-14	A.M.	54.0	51.0	57.5	45.5	47.0	47.0	50.3
	P.M.	69.0	57.5	63.5	65.5	52.5	57.0	60.8
15-16	A.M.	58.0	48.0	58.5	49.0	44.5	50.5	51.4
	P.M.	80.5	61.5	67.0	62.5	50.5	59.0	63.7
17-18	A.M.	57.5	58.0	61.0	57.0	46.0	43.0	53.8
	P.M.	77.5	61.0	70.5	66.0	50.5	57.0	63.8
19-20	A.M.	54.5	53.5	57.0	59.5	43.5	49.0	52.8
	P.M.	77.0	61.0	69.5	75.0	52.0	55.5	65.0
21	A.M.	62.0	54.0	62.0	62.0	50.0	53.0	57.2
	P.M.	81.0	70.0	69.0	70.0			72.5
Mean	A.M.	+0.35	+0.55	+0.65	+0.53	+0.17	+0.23	+0.41
Δ/day	P.M.	+0.88	+0.57	+0.89	+0.88	+0.46	+0.32	+0.67

Pulse rates were counted at 8:30 A.M. under basal conditions and at 3:30 P.M. after 2 hours of supervised bed flat rest. These are the 'A.M.' and 'P.M.' values. The line marked mean Δ/day presents the average pulse rate increase per day of bed rest. The F. value is the result of the F test used to determine the significance of the ratio of the variation attributable to linear regression to the variation attributable to deviations from regression.

From Taylor, H. L., A. Henschel, J. Brozek and A. Keys: Effect of bed rest on cardiovascular function and work performance. Journal of Applied Physiology. 2:223, 1949.

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rate at rest increased approximately 0.5 beats per day.

After three weeks of bed rest, these young men showed an increase of cardiac rate during moderate work of 40 beats per minute greater than before bed rest. It required from five to ten weeks of conditioning before the cardiac rate during work returned to the pre-bed rest level. This progressive increase of the cardiac rate at rest and the greater tachycardia during work indicates progressive decrease of the ability of the myocardium to do work. Increased rate must be substituted for the diminishing power of the cardiac muscle. During tachycardia, the heart has less time to

recover and fatigues more quickly. With this decreased cardiovascular reserve, the ability to do muscular work is greatly reduced. Taylor found that the ability to walk at 3.5 miles per hour on a 10 per cent grade was decreased to 25 per cent of normal after a three week rest in bed.

This study quoted above has demonstrated that there is a rapid deterioration of the cardiovascular ability as a result of bed rest. This deterioration is overcome only very slowly when the subject is ambulating again. To avoid the deteriorating effects of bed rest, patients should not be confined to bed any longer than is necessary to treat the primary disability. The degenerative effects of the enforced prolonged inactivity may be greater than the beneficial effects of the bed rest. More important rest when it is necessary should be confined only to the parts of the body requiring rest. Bed calisthenics, barbell, pulley or trapeze exercises, or occupational therapy may be prescribed to keep the uninvolved parts of the body active enough to prevent deterioration.

Even for the patient with cardiac disease, carefully prescribed activity may help maintain the body without an undue load on the myocardium. Careful passive motion does not appreciably increase blood pressure or cardiac output and yet may be used to keep the body supple. If the patient is assisted to sit and to stand early, the metabolic load is not greatly increased but the orthostatic reflexes are maintained. A trapeze bar over the bed makes it easier for the patient to move and decreases the amount of energy expended. Table 3 indicates the relative increase in the metabolism and energy output with varying degrees of activity[4]. It may be observed that sitting and standing require less energy than does digesting of a meal.

Exercise in order to be effective to increase endurance must cause some fatigue. This is not harmful if the fatigue does not exceed the recuperative power of the body. To re-establish endurance, the patient's activity should be increased as the tolerance increases. One advantage of exercise obtained under the supervision of an occupational or

Table 3

The effect of posture and activity on oxygen consumption and energy output.

Condition	O_2 Consumption cc./min.	Energy Output Cal./min.	Relative Energy
Lying	226-242	1.14	100%
Sitting	234-260	1.19	104%
Standing relaxed	238-239	1.25	109%
Standing at attention	266	1.30	114%
Standing - Arm calisthenics	516	2.53	220%
Walking			
53 meters/min.	667	3.4	298%
112 meters/min.	1296	6.7	590%
146 meters/min.	2240	11.5	1010%
Standing - after light meal	298	1.45	127%
Standing - after heavy meal	320	1.56	137%

Data taken from Benedict, F. G., and H. Murschhauser: Energy transformations during horizontal walking. Carnegie Institution of Washington Pub. No. 231. Washington, D.C., 1915.

physical therapist is that it can be graded to meet the patient's needs and increased as the patient's tolerance increases. Adapting the exercise load to the increasing tolerance of the patient will develop endurance at the maximum rate.

Ischemic Ulcers

A very common type of deterioration of the patient confined to bed is the development of decubital ulcers. These ulcers develop over body prominences or areas where pressure is great enough to prevent the flow of blood through the tissues. Malnutrition of the cells as a result of this ischemia causes death and sloughing of the tissues of the compressed area. Patients who have poor nutrition for any reason are most susceptible to ischemic damage.

Ischemic ulcers occur most frequently in patients who are unable to move or who have areas of analgesia. Normally dangerous ischemia is avoided because pain arising from the ischemic area causes the patient to move and relieves the pain. If a patient has an area of analgesia, pain is not present as a warning signal and ischemia may persist until an ulcer develops. Debilitated patients, paralytic patients and patients in casts or traction may develop ischemic ulcers because they are unable to turn by themselves. These patients are a serious nursing problem. To prevent a dangerous degree of ischemia, these patients should be turned not less frequently than once each hour. To turn a disabled patient this frequently requires a great deal of the time of the nursing staff. The Stryker frame was devised to enable one nurse to turn a patient more easily so that the patient would be turned more frequently. To place a patient on a Stryker frame and then not turn him each hour defeats the prupose of the apparatus and decubiti may still develop.

Recently an air mattress has been devised which automatically shifts the weight bearing areas of the body at regular intervals. The mattress is composed of parallel rows of thin plastic tubes about one inch in diameter. Alternate tubes are connected together. An electric pump automatically inflates and deflates the two systems alternately on a five minute cycle. This change of the distribution of pressure under the body prevents prolonged ischemia. Although this mattress does not cure decubiti which have formed, it is valuable to help prevent decubiti in debilitated or helpless patients.

Since decubiti are the result of ischemia, the most satisfactory method of treating them is stimulation of the circulation. The decubitus should be relieved of all pressure. Wet or greasy dressings should be avoided since they macerate the skin and increase the slough. Dry heat from a light bulb or heat lamp increases the circulation and aids healing. Ultraviolet irradiation has been found useful for two reasons: (1) An erythemal dose of ultraviolet causes prolonged vasodilatation in the area and promotes healing; (2) the short ultraviolet wavelengths (2540 Å) are bactericidal to the organisms growing in the decubitus. Friction massage around the decubitus decreases edema and venous congestion and increases lymphatic drainage. Fibrosis of the subcutaneous tissue around the ulcer is prevented and better capillary circulation is maintained.

Summary

Efficient rehabilitation of the physically disabled patient is possible only if deterioration is prevented and the patient's abilities are preserved. Prolonged inactivity in itself causes deterioration of the skeletal, neuromuscular, and cardiovascular systems. Bed rest for patients with disabilities of long duration may produce deteriorative changes which permanently disable or greatly delay the recovery of these patients. The intelligent use of activity to maintain the abilities of the normal systems of the body, together with adequate protection of the involved parts of the body, will decrease the time required for resoration of the patient to maximal usefulness.

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II. MEDICAL SCHOOL NEWS

Coming Events

May 12-14	Continuation Course in Eye, Ear, Nose, and Throat for General Physicians
May 15-17	Continuation Course in Allergy and Hematology for General Physicians
May 20	Minnesota Pathological Society Meeting; "Crime and the Doctor," Dr. C. Keith Simpson, Reader in Forensic Medicine, Supervisor of Medico-Legal Post-Mortems, and Home Office Pathologist, Guy's Hospital, University of London, London, England; Owre Amphitheater; 8:00 p.m.
June 23-28	Continuation Course in Otolaryngology for Specialists

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Students Visit Lilly Company

From March 26 to 29, 69 University of Minnesota medical students were the guests of the Eli Lilly Company at the company's laboratories in Indianapolis and Greenfield, Indiana. During their visit in Indianapolis, they inspected the company's main research laboratories and various production facilities. They also had the opportunity of seeing a replica of the original Lilly plant. At Greenfield they witnessed demonstrations of biological production. The students who took part report that they had a most pleasant and profitable excursion.

Faculty News

Dr. E. T. Bell, Professor Emeritus of Pathology, spoke last week at the meeting of the Minnesota Pathological Society. His topic was, "The Pathology of Diabetes Mellitus."

Dr. Leo Davidoff, Professor of Clinical Neurosurgery, College of Physicians and Surgeons, Columbia University, New York, took part in a neuroradiology conference with the Departments of Radiology and Neurosurgery on Tuesday, April 22.

Dr. C. J. Watson, Professor and Head, Department of Medicine, and Dr. Wesley W. Spink, Professor, Department of Medicine, attended the recent meeting of the American College of Physicians in Cleveland. Dr. Spink is governor for the College for the state of Minnesota. Dr. Watson took part in a panel discussion on the functional pathology of the liver and also participated in an informal television clinic entitled "The Jaundice Patient".

Dr. Craig Borden, Assistant Professor, Department of Medicine, and Dr. Carleton B. Chapman, Associate Professor, Department of Medicine, represented the University at the recent meeting of the American Heart Association in Cleveland.

New Minesota Medical Foundation Members

Dick Cason, M.D., Hillsboro, Texas
Edward H. Kirschbaum, M. D., Waterbury, Conn.
J. A. Sheinkopf, M.D., Beverly Hills, Calif.
C. Walton Lillehei, M.D., Minneapolis
R. A. Bock, M.D., St. Paul
E. A. Smisek, M.D., St. Paul
W. E. Anderson, M.D., Clearbrook

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

April 28 - May 3, 1952

Monday, April 28

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; W-612, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:30 - Tumor Conference; Doctors Kremen, Moore, and Stenstrom, Todd Amphitheater, U. H.

11:30 - Physical Medicine Seminar; The Minnesota Crippled Children's Service; Geneva Gerischer; Eustis Amphitheater, U. H.

12:15 - Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - Physiology Seminar; Effects of Graded Caloric Restriction Upon Certain Endocrine and Target Organs in Relation to Mouse Mammary Carcinoma; Truman Newberry; 214 Millard Hall.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Pediatric Seminar; Report of Federation Meetings; 6th Floor West, U. H.

4:00 - Seminar on Fluid and Electrolyte Balance; Extracellular Fluid Determination; Todd Amphitheater, U. H.

4:30 - 5:30 Dermatological Seminar; M-346, U. H.

4:30 - Public Health Seminar; 15 Owre Hall.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Minneapolis General Hospital

7:30 - Fracture Grand Rounds; Dr. Zierold; Sta. A.

10:30 - 12:00 Tuberculosis and Contagion Rounds; Thomas Lowry; Station M.

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

12:30 - Surgery Grand Rounds; Dr. Zierold; Sta. A.

1:00 - X-ray Conference; Classroom, 4th Floor.

1:30 - Pediatric Rounds; Robert Ulstrom; 4th Floor.

Ancker Hospital

8:30 - 10:00 Chest Disease Conference.

1:00 - 2:00 Medical Grand Rounds.

Monday, April 28 (Cont.)

Veterans Administration Hospital

8:00 - 9:00 Neuroradiology Conference; B. J. O'Loughlin, R. C. Gray; 2nd Floor. Annex.

9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

11:30 - X-ray Conference; B. J. O'Loughlin; Conference Room, Bldg. I.

2:00 - Psychosomatic Rounds; Bldg. 5.

3:30 - Psychosomatic Rounds; C. K. Aldrich; Bldg. I.

Tuesday, April 29

Medical School and University Hospitals

8:30 - Conference on Diet Endocrines and Cancer; M. B. Visscher; 116 Millard Hall

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:00 - 1:30 Selected Topics, Permeability and Metabolism; Nathan Lifson; 129 Millard Hall.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

4:00 - 5:00 Pediatric Rounds on Wards; I McQuarrie and Staff; U. H.

4:30 - 5:30 Clinical-Medical-Pathological Conference; Todd Amphitheater, U. H.

5:00 - 6:00 X-ray Conference; Presentation of Cases by Veterans Hospital Staff; Drs. Fink, O'Loughlin, et al; Eustis Amphitheater, U. H.

Ancker Hospital

8:00 - 9:00 Fracture Conference; Auditorium.

8:30 - 9:30 Medical-Roentgenology Conference; Auditorium.

1:00 - 2:30 X-ray-Surgery Conference; Auditorium.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Spencer F. Brown; 5th Floor.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station F.

11:00 - Pediatric Rounds; Erling S. Platou; 7th Floor.

12:30 - EKG Conference; Boyd Thomes and Staff; 302 Harrington Hall.

Veterans Administration Hospital

7:30 - Anesthesiology Conference; Conference Room, Bldg. I.

8:30 - Infectious Disease Rounds; Dr. Hall.

8:45 - Surgery Journal Club; Conference Room, Bldg. I.

9:00 - Liver Rounds; Drs. Nesbitt and MacDonald.

Tuesday, April 29 (Cont.)

Veterans Administration Hospital (Cont.)

9:30 - Surgery-Pathology Conference; Conference Room, Bldg. I.

10:30 - Surgery Tumor Conference; L. J. Hay, B. J. O'Loughlin; Conference Room, Bldg. I.

1:00 - Surgery Chest Conference; T. Kinsella and Wm. Tucker; Conference Room, Bldg. I.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

3:30 - 4:20 Autopsy Conference; E. T. Bell and Donald Gleason; Conference Room, Bldg. I.

Wednesday, April 30

Medical School and University Hospitals

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Norman Jacob and L. G. Rigler; Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Pediatrics Case; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

12:30 - 1:30 Permeability and Metabolism Seminar; Nathan Lifson; 129 Millard Hall.

1:30 - Conference on Circulatory and Renal System Problems; M. B. Visscher; 116 Millard Hall.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; Eustis Amphitheater, U. H.

5:00 - 6:00 Vascular Conference; Todd Amphitheater, U. H.

5:00 - 7:00 Dermatology Clinical Seminar; Dining Room, U. H.

7:00 - 8:00 Dermatology Journal Club; Dining Room, U. H.

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; R. Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

2:00 - 4:00 Medical Ward Rounds;

3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

8:00 - Pediatric Allergy Rounds; Lloyd Nelson; 4th Floor.

10:30 - 12:00 Medicine Rounds; Thomas Lowry and Staff; Station D.

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

12:30 - Pediatric Staff Meeting; X-ray Diagnosis of the Chest; Harry Nellins; 4th Floor Annex.

Wednesday, April 30 (Cont.)

Minneapolis General Hospital (Cont.)

1:30 - Pediatric Rounds; E. J. Huenekens and Robert Ulstrom; 4th Floor.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; Conference Room, Bldg. I.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

2:00 - 4:00 Infectious Disease Rounds; Main Conference Room, Bldg. I.

4:00 - 5:00 Infectious Disease Conference; W. Spink; Conference Room, Bldg. I.

7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, May 1

Medical School and University Hospitals

8:00 - 9:00 Vascular Rounds; Davitt Felder and Staff Members from the Departments of Medicine, Surgery, Physical Medicine, and Dermatology; Heart Hospital Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

12:30 - Physiological Chemistry Seminar; Orotic Acid Metabolism; Noel Simmons; 214 Millard Hall.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theatre.

3:30 - Medicine-Pediatric Infectious Disease Conference; Heart Hospital Auditorium.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

5:00 - 6:00 Radiology Seminar; Thoracic Surgery Conference; Drs. Kinsella, Varco, et al; Eustis Amphitheater, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Spencer F. Brown; 5th Floor.

8:30 - Neurology Rounds; William Heilig; 4th Floor.

11:00 - Pediatric Rounds; Erling S. Platou; 7th Floor.

1:00 - Fracture-X-ray Conference; Dr. Zierold; Classroom.

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

8:00 - Surgery Grand Rounds; Conference Room, Bldg. I.

Thursday, May 1 (Cont.)

Veterans Administration Hospital (Cont.)

11:00 - Surgery Roentgen Conference; B. J. O'Loughlin; Conference Room, Bldg.I.

Friday, May 2

Medical School and University Hospitals

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Medicine Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; Sympathetic Ophthalmia; Ernest S. Palmerton; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

4:00 - 5:00 Dermatology Seminar; W-321, U. H.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

Minneapolis General Hospital

11:00 - Pediatric Rounds; Franklin H. Top; 7th Floor.

11:00 - Pediatric-Surgery Conference; Dr. Wyatt, Forrest Adams; Classroom, Sta. I.

12:00 - Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

1:00 - 3:00 Clinical Medical Conference; Thomas Lowry; Classroom, Station M.

1:30 - Pediatric Rounds; Robert Ulstrom; 4th Floor.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

1:00 - Microscopic-Pathology Conference; E. T. Bell; Conference Room, Bldg. I.

1:30 - Chest Conference; Wm. Tucker and J. A. Meyers; Ward 62, Day Room.

3:00 - Renal Pathology; E. T. Bell; Conference Room, Bldg. I.

Saturday, May 3

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; W. H. Cole and Staff; M-109, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater.

9:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; Heart Hospital Amphitheater.

9:15 - 10:00 Surgery-Roentgenology Conference; L. G. Rigler, J. Friedman, Owen H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:30 - Anatomy Seminar; Some Aspects of Protoplasmic Ultrastructure and Their Physiological Implications, J. Francis Hartmann; The Bronchial Arteries in Pulmonary Disease, Martha Pitel; 226 Institute of Anatomy.

Ancker Hospital

8:30 - 9:30 Surgery Conference; Auditorium.

Minneapolis General Hospital

8:00 - Pediatric Rounds; George Lund; 5th Floor.

11:00 - 12:00 Medical-X-ray Conference; O. Lipschultz, Thomas Lowry, and Staff; Main Classroom.

11:00 - Pediatric Clinic; C. D. May and Floyd Denny; Classroom, 4th Floor.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - Hematology Rounds; P. Hagen and E. F. Englund.