parts of it; their range goes from "sealed rooms" through the "iron lung" to the chest or abdominal shells and the chest-abdomen cuirass. Body respirators have mainly inspiratory action; in some types inspiration can be supplemented or replaced for a short time by a positive-pressure attachment. (2) This group is represented by insufflation respirators, which may be either positive-pressure apparatus, giving only active inspiration, or combined positive/negative-pressure machines, giving an active expiration as well. As the body respirators have almost unanimously been abandoned for the treatment of acute and severe cases of respiratory paralysis, this discussion is limited to explaining the essential differences between the two types of clinically introduced and commercially available insufflation respirators.

In pressure-cycling apparatus (Mushin and Rendell-Baker, 1954) the ventilatory effect depends not only on pressure variation but also on airway resistance and lung compliance. Increased resistance in the tracheo-bronchial tree means diminished efficiency of ventilation; theoretically ventilation could be made efficient if these factors could be held constant. This is, however, impossible in acute conditions. Leakage in the positive pressure phase prolongs inspiration time; with a big enough leak the valve does not operate, resulting in a constant pressure application (with subcycling values); this might rapidly become very dangerous in the presence of certain complications and circulatory lability. Concerning the circulatory effect of such systems, it has been emphasized (Maloney *et al.*, 1953) that it is important to add a negative phase in order to keep the mean intrathoracic pressure at low levels, which will avoid the harmful effect of positive pressure on the circulation.

The Engström respirator, on the other hand, delivers a predetermined volume at a pre-set frequency. That means that the machine furnishes the pressure needed to overcome the resistance, the latter being recorded on a water manometer so that any developing impediment can be observed. Therefore, the apparatus represents a time and volume cycling respirator, and is the first of this type used clinically and on a large scale. It ensures a quantitatively sufficient ventilation to the patient with a predetermined volume of any desired qualitative composition.

Conclusion

The severe forms of respiratory paralysis in which signs of central lesions are prominent often constitute a rather complicated therapeutic problem for both physician and nurse. The reported method of treatment has been widely adopted in the Scandinavian countries during the past years, and has led to a considerable decrease in mortality. It must be pointed out, however, that artificial respiration is but one side of the intricate therapeutic problem in these cases; the rest of the treatment is at least as important, particularly the control of parenteral nutrition, water and mineral balance, and the maintenance of an adequate air-way.

Summary

The Engström respirator is described, and the results obtained with it at the Stockholm Hospital for Infectious Diseases in a series of 54 consecutive cases (mortality 27%) of acute, severe poliomyelitis with brain-stem involvement are reported. The respirator is suitable for the treatment of all forms of severe respiratory paralysis, such as may occur in polyradiculitis or barbiturate poisoning, for example.

The principles on which satisfactory artificial respiration depend are discussed.

Finally, it is stressed that, in addition to respirator therapy, efficient general medical treatment is essential to success.

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THE PREVENTION OF DEFORMITY IN POLIOMYELITIS*

BY

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It is axiomatic to state that it is better to prevent deformity rather than try to correct it after it has occurred. But the idea is commonly honoured only in the breach. It is too often convenient both for the conservative surgeon and for the timid patient to be lulled into a state of hopeful security by the thought that physiotherapy will not only restore strength but will also prevent deformity. Early surgery is often regarded as interference, and it is usual to recommend that anything more drastic than manipulation ought to be relegated to the stage when muscle recovery is complete. Theoretically this interval may extend to two years, but as a practical policy it is fair to consider all worth-while muscle recovery to be achieved by the end of six months from the onset of the disease. Since the orthopaedic requirements for nursing during the acute phase of the disease have become more generally recognized, surgical adjustment of potential deformity depending upon muscle imbalance can usually be safely left until six months have elapsed, by which time a clear assessment of muscle recovery will have been made. But there are other perhaps less potent, but not negligible, causes of deformity that will require attention before the effects of muscle imbalance are likely to become clearly manifest. In any event it seems only logical to treat and to attempt to counteract any condition or force that will otherwise inevitably produce deformity in the fullness of time. The need for early recognition of real or potential deforming influences is the strongest argument that can be offered for the inclusion of the orthopaedic surgeon in the team of experts that the management of poliomyelitis calls for from the very beginning.

Causes of Deformity in the Acute Phase

Pain.—It is too often forgotten that in a relatively small proportion of patients poliomyelitis is a painful disease, and that in a few of these the pain may persist after the febrile attack has subsided, even up to six or eight weeks after the onset of the disease. Pain means immobility on the part of the patient in whatever position may be found to be comfortable. Pain also means that any attempt to carry out passive movements in order to dispel peri-articular

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oedema and to limit joint stiffness is likely to be resented. With persuasion the important passive movements can and must be carried out and their range gradually increased. Complete immobility caused by pain requires splintage for the joints and limbs in their positions of optimum function until the pain can be relieved enough by local heat and sedatives to enable passive movements to be started.

Muscle Spasm.—This has been a subject for controversy probably because it is so easily confused with tightness. In 1947 Seddon found that muscle shortening occurred in 10 out of 1,500 patients, and in none was spasm unequivocal. In any event muscle spasm subsides very quickly and if protracted it is probably by psychogenic perpetuation. In the unit at Pinderfields I have seen the condition only once in over 900 patients. One leg was affected for almost six months, and in consequence the knee was held in full extension and the ankle in full plantar flexion. The spasm finally resolved after repeated paravertebral blocks, using local analgesic, and eventually there were no residual deformities in the affected limb. Temporary spasm recurred once after accidental sudden stimulation of the leg with a stream of cold water, which suggests that there remained a pre-natural irritability of the muscles.

Fibro-Elastic Tissue Contractures.—The tightness produced by these contractures is so usual that poliomyelitis ought to be regarded as a disease of the collagenous tissues as well as of the motor nervous system (Bennett, 1952). The changes in the fibro-elastic tissues are early and rapid and result in early shortening and loss of normal elasticity and resilience in tendinous, ligamentous, and fascial structures; they occur far too soon to be manifestations of disuse and postural adaptation.

Sites of Potential Deformity from Collagenous Contracture

The sites most affected are the plantar fascia, the tendo Achillis, the ilio-tibial band, the hamstrings, the lumbo-sacral fascia, and the peri-articular ligaments of the spine. The less affected sites are the infrapatellar tendons, the pectoral muscles, teres major, and pronator teres.

If the sites likely to be affected are examined as a routine early contractures will not infrequently be found. It is probable that contracture of the peri-articular structures of the spine is a prime cause of spinal deformity. At any rate when scoliosis does occur and is developing it is the result of a nice mixture of peri-articular contracture and imbalance of the intrinsic muscles of the spine. A similar mixture of contracture and paralysis has to be taken into account with regard to the forces responsible for maintaining a normal relationship of the pelvis to the spine.

The recognition of these early contractures goes far to prevent the development of deformity on their account. Passive, and, later, active stretchings of the tightening structures can be expected to be effective only in the early stages. Established deformity will require tenotomies and fasciotomies, which generally speaking ought to be avoidable. It must be recalled, however, that the modern treatment of respiratory paralysis from poliomyelitis has introduced a new engine for producing deformities in the shape of the cabinet respirator. Consideration of the effect and site of collagenous tissue involvement makes it clear that it will be impossible to avoid the development of certain deformities in patients who need to be isolated in the cabinet respirator for any length of time. Such deformities are avoidable and treatable only by redesigning the respirators with this in view, and the problem is receiving attention.

Passive stretching of early or potential fibro-elastic contractures is the preventive treatment of choice, but it is a two-edged weapon. In the hands of ignorant or irresponsible therapists stretching will result in muscle tears which will not only destroy valuable muscle fibres required for future rehabilitation but will also produce scar tissue and thus eventually exacerbate the condition it was intended to ameliorate. Particular care must be exercised in stretching until the return of power of the controlling muscles is fully known, and only sufficient range of movement ought to be obtained at first to enable muscle action to begin. In the

established contracture passive stretching can only do harm to the muscles and will have no effect at all on ligamentous bands or aponeuroses.

The acute phase of the disease is completed at the end of an arbitrary period of three weeks or a month after the onset of the disease. The next phase of potential recovery is heralded by an estimation of the muscle power, and its distribution and the presence of any deformities are noted. This is the period when, if possible, definitive correction of existing deformative factors should be carried out and the slow formation of new deformities prevented.

The causes of deformity in the phase of potential recovery are: uncorrected fibro-elastic tissue contractures, muscle imbalance, and perverted action of normal muscles and tendons.

Contracted Bands

The chain of deformities that a contracted band can produce must be recognized so that any particular deformity can be related to the single prime cause. Similarly the chain of deformities that can result from overstretching the controlling muscles must be known in order to avoid the evil results of maltreatment. This principle is most convincingly seen with regard to the tendo Achillis and its controlling triceps surae (see Table I), and with the ilio-tibial band and its two motors, tensor fasciae femoris and gluteus maximus.

TABLE I.—*Tendo Achillis*

After detecting apparent contracture of the tendo Achillis it is next essential to obtain a clear idea of the power of the gastrocnemius and soleus, which together constitute the triceps surae and work as a unit in pulling on the calcaneum. The power of the posterior tibial muscles must be equally accurately measured and their power relative to that of the triceps surae assessed. The importance of this lies in a disparity of power in favour of the posterior tibial musculature because that will increase any calcaneus deformity permitted by a weak triceps surae or an overlong tendo Achillis (Fig. 1). The posterior tibial muscles in attempting to supplement the weak triceps surae will actually exacerbate the deformity.

FIG. 1.—Lateral view of ankle in calcaneus to show the action of strong posterior tibials to increase the deformity.

I have on more than one occasion found physiotherapists industriously manipulating feet into dorsiflexion with the object of stretching the plantar fascia. Unless the hindfoot be firmly grasped there will be gradual lengthening of a weak triceps surae and consequently a troublesome calcaneus deformity.

The ilio-tibial band, by reason of its attachment through its motor muscles to the pelvis and by its insertion to the head of the tibia, acts upon both hip and knee. Even when the buttocks and thighs are severely paralysed the tensor fasciae femoris is commonly spared because of its long cell station in the spinal cord. In consequence a combination of

fibroelastic tissue contracture and the preservation of one motor muscle in opposition to much reduced power will produce a chain of deformities (see Table II).

TABLE II.—*Ilio-tibial Band*

Flexion deformity of the hip is more commonly caused by contracture of the ilio-tibial band than by shortening of the hip flexors or periarticular structures. This is demonstrable by the ease with which the flexion deformity can be abolished by widely abducting the hip. It is rare to require section of the ilio-psoas muscle (which is the only real hip flexor) to correct a flexion deformity of the hip, and the sartorius plays no part at all in hip-flexion deformities. Soutter's (1914) operation (or its modifications) is to be condemned for muscle destruction because the contractures lie in the fascia and intermuscular septa and the muscle fibres are normal. Ilio-tibial band contractures cannot be corrected by passive stretchings but only prevented; nor can they be overcome by the application of serial plaster spicas. The established contracture of the ilio-tibial band is best treated by using Yount's (1926) operation. The ilio-tibial band is divided in its lower part, 4 in. (10 cm.) above the knee-joint, the lateral intermuscular septum is excised down to the femur, and the fascia covering the vastus lateralis is sectioned in front and behind.

In total or subtotal paraplegia much thought must be given before breaking down a tight fascial envelope of the thighs and buttocks. In the absence of effective muscle power such an envelope is useful in the prevention of "jack-knifing" when the patient is trying to use callipers; intact contractures may obviate the need for a pelvic band and hip-lock attachment to the callipers. It is often a nice decision whether to have ease in sitting at the expense of an inability to perform four-point, or even three-point, walking.

Muscle Imbalance

When it has become clear from a consideration of the muscle chart that there is obvious muscle imbalance in a part then deformity can and must be anticipated. Muscle rehabilitation in the presence of imbalance can only make matters worse by strengthening the pull of the stronger muscles at the expense of the weaker. Early tendon transpositions are frequently called for, especially in children, in order to preserve satisfactory function, but it is better to avoid operation under the age of 5 years if possible because of the difficulty of muscle re-education in the very young.

Operations to improve the muscle control of the shoulder are not indicated, except transposition of the teres major to the lateral aspect of the humerus to prevent internal rotation deformity of the arm. Early restoration of active flexion in the elbow, preferably by pectoral muscle transposition, is desirable where possible because a working biceps will also give active supination. In default of this, transposition of the flexor carpi ulnaris to the dorsum of the lower end of the radius is effective in correcting a pronation deformity. For the control of the wrist an equitable distribution of available muscles must be arranged to give flexion and extension without deviation. Wrist flexors can be transferred into finger extensors because they normally act synergically; to a lesser degree wrist extensors are suitable for depleted finger flexors; the pronator teres works well as a wrist extensor. Care must be exercised to avoid the subtraction of all wrist flexor power, otherwise a hyperextension deformity of the wrist will ensue associated with complete extension of the fingers at the metacarpophalangeal joints. Loss of opposition is the commonest residual paralysis of the hand in poliomyelitis; it reduces

the power of pinch by 90% and leaves an apposition deformity. Every effort must be made to restore active opposition by transposing a motor tendon to work from the region of the pisiform bone and, by its attachment to the ulnar border of the base of the proximal phalanx, to restore the rotation of the thumb on its own axis.

In the absence of pelvic obliquity caused by contractures of the ilio-tibial band most scoliosis is caused by defects in the spinal structures. When it is known that there is imbalance of the paravertebral muscles or the intrinsic muscles of the spine treatment is not very effective unless the degree of imbalance is small. The most effective measure is to nurse the patient in a hammock in the overcorrected position.

It is also necessary to maintain a balanced active relationship between the pelvis and thorax. Unequal paralysis of the lateral abdominal muscles is best corrected by transposition of the ilio-tibial band (keeping the tensor fasciae femoris and its nerve supply intact) to the ninth rib in the mid-axillary line. Forward tilting of the pelvis in the presence of equal weakness of the lateral abdominals will benefit from bilateral operation, and the improvement in balance and thoraco-pelvic stability will do much to help the gait.

Leg deformities are not usually caused by muscle imbalance, but where there is gross preponderance of the hamstrings the transposition of the biceps and semitendinosus to the patella is a good method of supplementing a weak quadriceps; the semimembranosus must be preserved intact in order to avoid the development of genu recurvatum.

Tendon transposition in the foot has much to offer either as a supplement to the action of synergistic muscles or to an arthrodesis of the foot (Table III). There is no tendency for transposed tendons to lose power when they are acting naturally, but when the new location requires education of

TABLE III.—*Tendon Transpositions in the Foot*

Group	New Insertion	Deformities Controlled
Anterior ..	Dorsum of foot	Equinus, valgus and varus
Posterior ..	Tendo Achillis	Calcaneus
Toe extensor ..	Calcaneum Dorsum of forefoot	Claw toes

FIG. 2.—The cocked hallux.

the muscle to act with an antagonistic group a material loss of power is to be expected. It is not enough to allow a foot deformity to develop with the idea that it can be corrected and fixed by arthrodesis later because muscle imbalance can still act as a deforming agent when the joints are fused. Muscles must be so balanced that the maximum of power of active dorsiflexion and plantarflexion can be achieved and yet the foot be maintained plantigrade.

The results can be improved by making use of synergy of neighbouring tendons. Transposition of the extensor hallucis longus and peroneus longus or transposition of both peroneals medial to the midline of the foot are examples for replacement of weak or paralysed tibialis anterior.

Analysis of the deformations is most important. For instance, the dropping of metatarsal heads may be secondary to cavus induced by an uncorrected calcaneus deformity. Dropping of the first metatarsal head is a very common deformity which will inevitably produce a cocked hallux (Fig. 2) with its painful friction areas, plantar callus, and ruinous wear on the shoe sole.

Perverted Action of Normal Tendon

Two prime examples of this are to be seen in the foot. One has already been noted in the discussion of the calcaneus deformity. The posterior tibial muscles, in the presence of a weak triceps surae, will attempt to take over the function of the weak group. If the calcaneum is already dorsiflexed then the action of the posterior tibial muscles will exacerbate the deformity and the need to transfer the posterior tibial muscles to the tendo Achillis is paramount.

FIG. 3.—Gross valgus foot with perverted action of extensor digitorum longus tendons and peroneus tertius.

The other example in the foot is the action of the extensor digitorum longus tendons and peroneus tertius on a valgus foot resulting from a weak tibialis posterior (Fig. 3). If this action is allowed to persist the foot will assume a bow shape and the long toe extensor tendons will act as the bow string. Mistakenly the peroneal tendons may be used to counteract the deformity, but it will persist if the true deforming agent be not perceived. The foot can only be corrected by shifting the toe extensor tendons to the medial side of the foot to act as a supplement to the weak tibialis posterior.

Summary and Conclusions

The causes of deformity during each phase of poliomyelitis are considered. In the acute stage the effects of pain and muscle spasm are discussed, the contracture of fibro-elastic tissues is detailed, and methods of its control are outlined. In the stage of potential recovery the mechanics of uncorrected fibro-elastic contractures are elaborated and methods of correction are recommended. Muscle imbalance as a cause of deformity is expounded and the surgical correction by tendon transposition is explained as a means of anticipating common deformities. The perverted action of normal tendons caused by paralysis or weakness of other muscles is instanced and its correction is described. The thesis is that surgical anticipation of deformity is more logical and easier to perform than surgical reconstruction, and the results are better.

It is not claimed that all deformities can be controlled by preventive surgery, but it is maintained that patients can be halted in a steady deterioration which would otherwise yield an intractable problem. In poliomyelitis it is the surgeon's duty to anticipate rather than to reconstruct.

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POSITIVE-PRESSURE INFLATION IN RESPIRATORY PARALYSIS OF POLIOMYELITIS

BY

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During the 1952 epidemic of poliomyelitis in Denmark the iron lung type of respirator used in cases of bulbar respiratory paralysis requiring tracheotomy was replaced by a different method of artificial respiration. Instead of the application of negative pressure to the chest wall manual rhythmic positive-pressure inflation of the lungs themselves was performed. It was carried out, by means of an apparatus specially designed by Dr. B. Ibsen (Lassen, 1953), through a cuffed endotracheal tube led into the tracheotomy opening.

Since that time various other methods of artificial ventilation in these cases have been described. Macintosh (1953) redesigned the bellows part of the Oxford vaporizer as a separate unit which he called the Oxford inflating bellows and which are operated by hand. Mushin (1953) added to these the Cardiff inflating valve which automatically allows deflation and prevents re-breathing. Russell and Schuster (1953) constructed a respiration pump which drives the Oxford bellows automatically. Others constructed automatic respirators for inflation where volume, speed, and pressure were either set according to the needs of each case or steered by the intrapulmonary pressure of the patient (Pask, 1953; Beaver, 1953; Bang, 1953; Macrae *et al.*, 1953).

I have utilized the original Oxford vaporizer fitted with a Salt valve (Wislicki, 1953). Its use for two different purposes is described in this paper.

Case 1

A builder aged 29 was admitted to the local infectious diseases hospital on October 29, 1953. For four days he had suffered from poliomyelitis which had resulted in paralysis of both legs and retention of urine. His temperature was 102.6° F. (39.2° C.). During the next two days external eye muscles and pharyngeal muscles became affected, causing diplopia and difficulties in swallowing, respectively. Large amounts of mucus accumulating in his mouth and pharynx necessitated tracheotomy (Dr. A. Fraenkel). On November 1 partial respiratory paralysis of bulbar origin developed. Manual artificial respiration was applied, but his condition deteriorated and little hope was held out for his survival. I was then called in.

On arrival I found a patient of muscular build, in a good state of nutrition, but cyanosed, restless, and with a very anxious expression. His respirations through a tracheotomy opening were laboured, irregular, and shallow. It was decided to try artificial respiration by means of the Oxford vaporizer. As no cuffed endotracheal tube was available the inner tracheotomy cannula was removed while the outer one was left in position. A Magill endotracheal tube No. 5, shortened to 10 cm., was inserted into the cannula until it made a tight fit. The tube was connected by a Rowbotham connexion and a short piece of wider rubber tubing to the Salt valve and from there to the Oxford vaporizer. Rhythmic inflation with air only was started. After a few minutes oxygen at the rate of about 2 litres a minute was led into the vaporizer. Suction was performed by taking the rubber tube out of the tracheotomy cannula and introducing the suction catheter instead. The mouth and pharynx were also cleansed by suction when secretions accumulated.