**PROCEEDINGS OF THE ANNUAL SCIENTIFIC MEETING OF THE
INTERNATIONAL MEDICAL SOCIETY OF PARAPLEGIA
HELD AT STROKE MANDEVILLE IN JULY 1978
(Part III)**

**INTRA-MEDULLARY PRESSURE AND INTRA-OSSEOUS
PHLEBOGRAPHY IN PARAPLEGIA**

A. CHANTRAINE¹, C. VAN OUWENALLER¹, H. J. HACHEN² and P. SCHINAS³

¹ Service de Médecine physique et de Rééducation; ² Centre des paraplégiques and ³ Service de radiologie, Hôpital cantonal, Université de Genève, Switzerland

Key words: Bone circulation; Phlebography; Osteoporosis; Paraplegia.

Abstract. Intra-medullary pressure and intra-osseous phlebography have been studied in the femurs of ten paraplegics. These methods are reliable for studying bone circulation. Results show that there exists an increase of intra-medullary pressure of more than twice the normal value and signs of delayed blood flow as well as a diaphyseal reflux in the bone. The blood gases are also modified (O_2 saturation; pO_2 and pCO_2). These signs indicate that there is a venous stasis. These circulatory disturbances are due to an autonomic nervous system lesion. Furthermore it seems that these vascular modifications below the neurological lesion have an influence on the osteoporosis in paraplegia.

Introduction

IN paraplegia circulatory disorders (GALIBERT, 1959; ROSSIER *et al.*, 1973) and changes in the arterial and venous blood gases (Benassy *et al.*, 1963) have been reported below the neurological lesion, where there also exists an osteoporosis (Abramson, 1948; Lodge, 1956).

These different observations associated with other parameters and mostly chemical bone studies have led us to propose a diagram of our conception of the osteoporosis pathogenesis in paraplegia (Chantraine, 1978). Through this conception, the Autonomic Nervous System (ANS) which is disturbed due to the spinal cord lesion should play an important role in the etio-pathogenesis of this osteoporosis, as it has been already suggested by Maury (1964).

In order to confirm the vasomotor alterations in the bone below the neurological lesion consequent to ANS lesion, we have studied the Intra-Medullary Pressure (IMP) and Intra-Osseous Phlebography (IOP) in these patients.

Measurements of IMP of bone is a useful indirect method of qualitative study of bone circulation (Shim, 1973). The IMP of intact cancellous bone is under normal conditions almost exclusively determined by the pressure in the veins draining the bone (Arnoldi, 1973).

As far as IOP is concerned, this investigation brings important information about the anatomical status of the bone vessels, about the nature of an eventual obstruction, about the dynamic aspect of the venous flow into the bone and the consequences resulting from these problems. For these reasons, it seems that IMP and IOP have a real value in assessing the venous drainage in the bone and to evaluate any vasomotor disorders.

Observation and Method

Ten patients with a complete paraplegia have been studied, aged from 21 to 52, one female and nine males, as shown in Table I, where the duration of the paraplegia is also indicated. Patients were investigated in the supine position and the IMP was measured in both femurs. A cannula, 8 cm long, with an inner diameter of 3 mm was inserted, without anaesthesia, into each great trochanter at the union of the middle and the lower third. In incomplete paraplegia, local anaesthesia may be necessary. The cannula is connected through a catheter filled

TABLE I

Patient	Age	Sex	Level	$\Delta \delta$ in months	IMP		pH		pCO ₂		pO ₂		Saturation O ₂	
					L	R	L	R	L	R	L	R	L	R
					mm Hg									
1	29	M	C5-C6	3	55	51	7.43	—	34	—	82	—	92.5	—
2	27	M	Th12-L1	4	29	41	7.37	7.33	34	30	60	53	91	88
3	35	F	Th10-Th11	72	40	42	7.35	7.34	34	33	42	46	88	85
4	27	M	C5-C6	3	21	25	7.38	7.45	35	36	42	51	85	86
5	36	M	Th5-Th6	4	72	70	7.46	7.43	32.5	35	92.5	78	97	94.5
6	29	M	C6-C7	3	38	50	7.42	7.39	37	39	61	62	92	94
7	57	M	Th10-Th11	12	40	32	7.35	7.46	34	—	—	—	—	—
8	22	M	Th10-Th11	5	30	19	7.35	7.33	34	36	41	38	85	83
9	21	M	C6-C7	6	29	35	7.26	7.34	—	31	—	—	—	—
10	27	M	Th5-Th6	10	60	55	—	—	—	—	—	—	96	95

with sterile heparinised saline to a pressure transducer incorporated in a pre-amplifier and an electronic recorder. The lecture of the IMP is recorded after 5 minutes, time necessary for a stabilised pressure. In addition, a hyperpression test is performed by introducing 5 cc of the solution into the cannula. The IMP is then followed during another 5 minutes. As soon as these measurements are completed, 5 cc of intraosseous blood is taken through the cannula into a syringe rinsed with heparine. Simultaneously, arterial blood is taken from the corresponding femoral artery.

Both blood samples are immediately sent to the laboratory for pH, pCO₂, pO₂ and O₂ saturation study. The analysis is made by the radiometer Copenhagen pH meter 27 apparatus, which allows a direct investigation of the various parameters.

Finally, 10 cc of contrast medium (methylglutamine and sodium ioxitamin acid¹) are injected under manual pressure into the cannula. A series of X-rays are taken, ten within 10 seconds, then one every 5 minutes during the next 30 minutes. In our cases X-rays were also taken an hour later, and on two occasions a control was carried out at the 24th hour.

Furthermore, in six patients, the measure of the deep femoral vein pressure has been performed using selective catheterisation.

Results

Table I indicates that the IMP is increased in all the paraplegics whatever the age of the patients, the sex, the level and the duration of the paraplegia.

Table II gives the mean value of the IMP of our patients compared to the mean values of normal series reported by different authors and one group of hemiplegia measured at the same localisation in the femur. Measurements of the

¹ Telebrix®.

deep femoral vein pressure show an increase of the values in two cases; in the others a thrombosis of that vein was observed.

The results of the IOP show that there is a stasis in the intra-osseous circulation, characterised by some difficulty to inject the contrast medium, the delayed flow and the diaphyseal reflux (Table III). Figure 1 illustrates the diaphyseal

TABLE II
Intra-medullary pressure (in mm of Hg)

Date	Authors	Bone	Series	IMP (mean)
1960	Simon	Up. metaph. femur	Normal	13.5
1968	Arlet <i>et al.</i>	Up. metaph. femur	Normal	17.2
1977	Chantraine <i>et al.</i>	Up. metaph. femur	Normal	17
1977	Chantraine <i>et al.</i>	Up. metaph. femur	Hemiplegia	33
1978	Chantraine <i>et al.</i>	Up. metaph. femur	Paraplegia	42

TABLE III
Intra-osseous Phlebography

Measures	20
Difficulty to inject	16
Delayed drainage	18
Diaphysis reflux	20

FIG. 1

Intra-osseous phlebography in the femur of paraplegic No. 2 (a) & No. 7 (b).
Diaphyseal reflux is well seen.

reflux; the contrast medium penetrates the medullary cavity against the flow creating a reflux. It can stay for a long period in the bone, sometimes for one or two hours and in one instance even after twenty-four hours (Fig. 2).

We have also confirmed in this work the modifications in the blood gases as shown in the Table IV. There is an increase of the oxygen saturation of the blood samples taken from the bone in paraplegia compared to that of the bone of normal subjects and to that of the femoral arterial blood of paraplegics.

FIG. 2

Intra-osseous phlebography, 1 hour later (No. 2); the contrast medium remains in the diaphysis.

TABLE IV
O₂ Saturation in femur

Series	O ₂ Saturation %	pCO ₂ m %	pO ₂ m %	pH m
Normal (6)	81.5	36	43.5	7.32
Paraplegics (10)	90.1	34.3	57.6	7.38
Femoral arteria (7) (Paraplegics)	96.21	33.8	89.6	7.42

Discussion

This functional investigation has allowed us to study the circulatory parameters in the bone below the neurological lesion providing objective data on the hemodynamic and angiographic pattern. The results of this study clearly demonstrate that there exist circulatory disturbances in the investigated bone. The IMP is largely increased and the IOP shows signs of venous stasis. The presence of paraosteoarthritis in some patients did not change the data.

This circulatory perturbation is due to the medullary lesion which undoubtedly provokes alteration of the ANS. Symptoms resulting from the ANS perturbation have been reported (Guttmann & Whitteridge, 1947; Grossiord *et al.*, 1961; Maury, 1966; Chantraine, 1977a, 1977b). It seems that an ANS disturbance is responsible for the vasomotor paralysis which determines the opening of the arteriovenous shunts. Benassy *et al.* (1963) have shown that below the neurological lesion in paraplegics oxygen saturation, pO_2 and pCO_2 are modified as we have confirmed in this study. On the other hand, the results of the IMP and IOP in our series of paraplegics demonstrate the existence of a vasomotor paralysis. This vasomotor paralysis may have direct and indirect consequences on bone metabolism. Such a relationship between osteoporosis and a vascular modification is thought to exist by different authors who have observed such a condition in animals and humans (Geiser & Trueta, 1958; Dhem, 1973; Bergmann *et al.*, 1977). In osteoporosis of a different aetiology this anomaly has also been described. Ficat *et al.* (1973) have shown in post-traumatic reflex sympathetic dystrophy a constant venous stasis in the bone. Fricke & Krokowski (1975) have described the same alterations in diabetes mellitus and rheumatoid arthritis osteoporosis.

This bone circulatory régime may have an influence on cellular differentiation. Trueta (1964, 1968) and Dhem (1973) have insisted on the consequence of the venous hypertension and venous stasis in the bone on osteogenesis. On the other hand, Andrew & Bassett (1971) have shown that applied force on bone may induce transformation of mesenchymal cells. In recent works, we have pointed out a striking observation (Chantraine *et al.*, 1976, Chantraine, 1978): a quantitative radiological study has allowed us to observe a significant increase of the outer and inner diameter of the femur a few months following paraplegia. This observation may also be seen in the Klippel-Trenaunay syndrome characterised by an increase of the length of the long bones due to presence of arterio-venous shunts and deep angioma (Malan & Puglionisi, 1964). In this syndrome there exists a circulatory perturbation with capillary and venous stasis. This passive hyperaemia may increase resorption of endosteal surfaces (Hutchinson & Burdeaux, 1954; Van Der Hoeft *et al.*, 1963).

Brookes (1972) has reproduced the same bone anomaly in experimental studies; Shim *et al.* (1972) have shown that systemic blood pressure, intramedullary pressure and blood flow through bone are remarkably constant through a given period. He demonstrated in the same conditions as Brookes that, if the femoral vein is occluded, IMP is elevated and indicates that there is venous congestion of bone.

The same authors have shown that stimulation of the lumbar sympathetic trunk and the sciatic sympathetic components causes a fall of the IMP and a decrease in bone blood flow. On the contrary, in the dog, the section of the lumbar sympathetic trunk or of the sciatic nerve sympathetic components produces an increase of the IMP (Shim *et al.*, 1972). Furthermore Shim *et al.*, 1972, have also shown that skeletal muscle contraction produces venous congestion of

bone and elevates the IMP, a condition that we see in spastic paraplegia. We thus believe that a vascular perturbation is linked to the neurological lesion and that both factors play a role in the osteoporosis observed in paraplegia.

Conclusions

This functional investigation has studied the circulatory parameters and has brought objective data of hemodynamic and angiographic nature. The circulatory perturbation in the bone is thought to be due to the ANS lesion. These vascular and neurological alterations influence bone formation and probably play an important role in the osteoporosis which appears below the neurological lesion in paraplegic patients.

RÉSUMÉ

La pression intra-médullaire et la phlébographie intra-osseuse ont été étudiées au niveau du fémur chez dix paraplégiques. Ces deux méthodes permettent d'explorer, d'une manière sûre, la circulation osseuse. Les résultats montrent qu'il existe une augmentation de la pression intra-médullaire qui s'élève à plus de deux fois la valeur normale. Cet accroissement est le témoin d'un retard du flux sanguin intra-osseux, comme le précise également la phlébographie intra-osseuse où l'on observe un reflux diaphysaire et une stase médullaire. L'étude des échanges gazeux dans le sang montre également une modification de la saturation en oxygène, de la pO_2 et de la pCO_2 . Tous ces signes indiquent qu'il existe dans l'os du paraplégique une stase veineuse. Ces troubles circulatoires sont dus à une lésion du système nerveux autonome. Il ne fait pas de doute qu'il existe une relation entre ces modifications vasculaires, apparaissant sous la lésion neurologique, et l'ostéoporose présente dans les mêmes segments chez le paraplégique.

ZUSAMMENFASSUNG

Der intramedulläre Druck und die intraossäre Phlebographie sind im Femurbereich bei 10 Paraplegikern studiert worden. Diese beiden Methoden erlauben mit grosser Sicherheit den Kreislauf der Knochen zu erforschen. Die Ergebnisse beweisen, dass eine Erhöhung des intramedullären Drucks besteht, der sich um das doppelte des Normalwertes erhöht. Diese Erhöhung ist der Beweis eines verzögerten Blutflusses innerhalb des intraossären Bereiches, wie es auch die intraossäre Phlebographie zeigt, bei der man einen Rückfluss in der Diaphyse und eine Stauung im Knochenmarkbereich beobachtet. Die Studie über den Gasaustausch im Blut zeigt ebenfalls eine Aenderung der Sauerstoffsättigung, des pO_2 und des pCO_2 . Alle diese Zeichen weisen darauf hin, dass im Knochen des Paraplegikers eine venöse Stauung vorliegt. Diese Kreislaufstörungen beruhen auf einer Läsion im autonomen nervösen System. Es besteht kein Zweifel, dass eine Verbindung zwischen diesen gefässbezüglichen Veränderungen, die während einer neurologischen Läsion entstehen, und der Osteoporose, die in den gleichen Segmenten bei dem Paraplegiker gegenwärtig ist, existiert.

REFERENCES

- ABRAMSON, A. S. (1948). Bone disturbances in injuries to the spinal cord and cauda equina (paraplegia). Their prevention by ambulation. *J. Bone Jt. Surg.*, **30A**, 982-987.
- ANDREW, C. & BASSETT, L. (1971). *Biophysical principles affecting bone structure*. In *Development and growth, Vol. III*, ed. Geoffrey H. Bourne. Academic Press, New York, p. 584.
- ARLET, J., FICAT, P., SEBBAG, D. (1968). Intérêt de la mesure de la P.I.M. dans le massif trochanterien chez l'homme. *Rev. Rhum.*, **35**, 250-256.
- ARNOLDI, C. C. (1973). Intraosseous pressure measurements in degenerative osteoarthritis. In *Circulation osseuse*, ed. J. Arlet et P. Ficat. Inserm, Paris, pp. 119-126.
- BENASSY, J., MAZABRAUD, J. & DIVERRES, J. C. (1963). L'ostéogénèse neurogène. *Rev. Chir. Orthop.*, **49**, 95-116.

- BERGMANN, P., HEILPORN, A., SCHOUTENS, A., PATERNOT, J. & TRICOT, A. (1977). Longitudinal study of calcium and bone metabolism in paraplegic patients. *Paraplegia*, **15**, 147-159.
- BROOKES, M. D. (1972). Blood supply of developing bone and its possible bearing on malformation of the limbs and face in congenital haemangiomas disorders. *Proc. Roy. Med.*, **65**, 13-15.
- CHANTRAINE, A. (1977a). Manifestations neuro-végétatives en dehors de la sphère vésico-sphinctérienne et rectosigmoïdienne dans la paraplégie. *Ann. Méd. Phys.*, **20**, 325-326.
- CHANTRAINE, A. (1977b). Ostéoporose sous lésionnelle et problèmes neuro-végétatifs. *Ann. Méd. Phys.*, **20**, 349-352.
- CHANTRAINE, A. (1978). Actual concept of osteoporosis in paraplegia. *Paraplegia*, **16**, 42-49.
- CHANTRAINE, A., CHAPARD, R. & LEROUX, G. (1976). Etude radiologique de la masse osseuse chez le paraplégique. Comptes rendus du 1er Symposium CEMO. *Méd. et Hyg.*, 44-52.
- DHEM, A. (1973). Les mécanismes de destruction du tissu osseux. *Acta Orthop. Belg.*, **39**, 423-433.
- FICAT, P., ARLET, J., LARTIGUE, G., PUJOL, M. & TRAN, M. A. (1973). Algo-dystrophies réflexes post-traumatiques. *Rev. Chir. Orthop.*, **59**, 401-414.
- FRICKE, M. & KROKOWSKI, E. (1975). Osteoporose-Folge einer verminderten Knochen-durchblutung. *Z. Orthop.*, **113**, 1043-1050.
- GALIBERT, P. (1959). Remarques à propos du syndrome neuro-végétatif des traumatismes de la moelle épinière. *Neurochirurgie*, **5**, 355-359.
- GEISER, M. & TRUETA, J. (1958). Muscle action bone rarefaction and bone formation. *J. Bone Jt. Surg.*, **40B**, 282-311.
- GROSSIORD, A., HELD, J. P., ODIEVRE, M. & MARTIN, M. (1961). Mydriase par excitation nociceptive sous-lésionnelle chez les tétraplégiques. *Rev. Neurol.*, **104**, 310-314.
- GUTTMANN, L. & WHITTERIDGE, D. (1947). Effect of bladder distension on autonomic mechanisms after spinal cord injuries. *Brain*, **70**, 361-404.
- HUTCHISON, W. J. & BURDEAUX, B. D. (1954). The influence of stasis on bone growth. *Surg. Gynecol. Obstet.*, **99**, 413-420.
- LODGE, T. (1956). Bone, joint and soft tissue changes following paraplegia. *Acta Radiol. (Stockh.)*, **46**, 435-445.
- MALAN, E. & PUGLIONISI, A. (1964). Congenital angiodysplasias of the extremities (note I: generalities and classification venous dysplasias). *J. Cardiovasc. Surg. (Torino)*, **5**, 87-130.
- MAURY, M. (1964). Des manifestations neuro-végétatives dans les tétraplégies. In Comptes rendus du IV Congrès International de Médecine Physique, Paris. *Excerpta Medica Foundation*, Amsterdam, pp. 602-607.
- MAURY, M. (1966). Des manifestations neuro-végétatives dans les lésions médullaires. *Presse Méd.*, **74**, 1673-1678.
- ROSSIER, A. B. & coll. (1973). Current facts on para-osteoarthropathy (POA). *Paraplegia*, **11**, 36-78.
- SHIM, S. S. (1973). *Physiology of bone blood flow*. In *Circulation osseuse*, ed. J. Arlet et P. Ficat. Inserm, Paris, pp. 41-56.
- SHIM, S. S., HAWK, H. E. & YU, W. Y. (1972). The relationship between blood flow and marrow cavity pressure of bone. *Surg. Gynecol. Obstet.*, **135**, 353-360.
- SIMON, L. (1960). Nécrose primitive de la tête fémorale chez l'adulte: thèse montpellier.
- TRUETA, J. (1964). *The dynamics of bone circulation*. In *Bone dynamics*, ed. H. M. Frost. Little Brown & Co., Boston, pp. 245-258.
- TRUETA, J. (1968). *Studies of the development and decay of the human frame*. Heinemann, London.