

EFFECT OF HIP DISLOCATION ON THE BLOOD SUPPLY TO THE FEMORAL HEAD

An Experimental Study in Rabbits

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This work consists of a series of experiments which were made in order: a) to examine the possible alterations of blood flow to the reduced femoral heads of rabbits, after inducing a closed dislocation of their hips and b) to determine the influence of the early or late reduction of the dislocated femoral heads on their blood flow. The estimations of blood flow to the normal and reduced femoral heads were made using radioactive microspheres.

The findings are as follows: a) The blood flow of the femoral heads of the reduced hips was never interrupted completely. b) The initially decreased femoral head blood flow progressively increased, in association to the time elapsed from the reduction. c) No statistically significant difference was found in the femoral head blood flow between hips reduced in early and those reduced late. d) Pure traumatic dislocation of the hip can only rarely be the cause of aseptic necrosis of the femoral head.

Key words: aseptic necrosis; blood flow; radioactive microspheres; traumatic dislocation of the hip joint

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Traumatic dislocation of the hip joint occurs most commonly after road accidents. Early reduction of the dislocation is thought to be essential for the survival of the femoral head because this ensures early restoration of its blood supply. Even early reduction, however, does not eliminate the danger of avascular necrosis of the femoral head, the consequences of which are serious and long lasting. General opinion is in favor of early reduction in order to avoid avascular necrosis. After many years of work at a hospital dealing mainly with accidents, however, our observation is that despite many traumatic dislocations of the hip being reduced several hours after the accident, the incidence of avascular necrosis in the long term follow-up is not statistically significant.

The purpose of this study was to determine the significance of the time of reduction.

MATERIAL AND METHODS

Seventy-eight adult male New Zealand white rabbits weighing between 3.3 and 5.3 kg were used. The animals were anaesthetised with intravenous veterinary Nembutal Sodium® (60 mg/ml) using a dose of approximately 20–35 mg/kg and were allowed to breathe spontaneously until they were killed shortly after the introduction of the radioactive microspheres.

Closed posterior dislocation of one of the hip joints of each animal was carried out by force and the reduction was performed under general anaesthesia 15 min or 48 h later. The position of the dislocated and reduced hips was checked by X-ray examination.

The rabbits were killed at defined times after the reduction of the hip joint. The rabbits were divided into two groups, group 1 and group 2, according to the time of the hip reduction and into subgroups according to when they were sacrificed. There were seven subgroups in each group and each subgroup included five animals. Another group of eight animals with intact hips was used as control.

At the appropriate time for each animal a veno-catheter was passed to the heart through the right common carotid artery for injection of radioactive microspheres. The microspheres, which were labelled with Sr85, had a diameter $25 \pm 5 \mu\text{m}$ and were suspended in 10 per cent Dextran®. Because of their size the microspheres cannot pass through the capillaries and remain within the vascular bed of the organ, thus allowing an estimation of blood flow. It has been estimated that only 5 per cent of microspheres of this size recirculate (Ya Po M et al. 1960, Neutze et al. 1968). The animals were killed with a large dose of anaesthetic (5 ml of Nembutal Sodium) 2 min after injecting the microspheres. The entire femoral head of the reduced and the normal hips and the hips of the control animals were removed and cleaned manually of all soft tissues. The external surface was thoroughly washed with tap water to minimize contamination with extraosseous microspheres during removal. The femoral head was then weighed on an electronic scale and placed in a plastic tube. The radioactivity was measured by placing the tube in the centre of a gamma scintillation counter with a NaI (TL) detector, flat type 3" $\times$ 3", whose efficiency was approximately 10 per cent.

A background count was performed before and after each counting session. The radioactivity was expressed in counts per minute per gram of weight of the femoral head. In each subgroup the average value of radioactivity of the dislocated heads was compared with that of the contralateral normal femoral head.

RESULTS

The results of this experimental study are as follows:

A. The level of radioactivity in the subgroups of animals killed 30 min after the early and late reduction of the dislocated hip was lower than that in the normal contralateral femoral head but was never zero (Tables 1 and 2), which indicates that the blood supply of the femoral head was never completely interrupted during the traumatic dislocation of the hip.

B. The initially low levels of radioactivity of the reduced femoral heads, increased progressively after the reduction (Tables 1 and 2). The

Table 1. Radioactivity values in femoral heads reduced after 15 min, expressed in counts/min/g

Group 1

Subgroup	Interval between hip reduction & sacrifice	Normal femoral heads	Reduced femoral heads
1	30 minutes	2500	1500
2	24 hours	2850	2350
3	3 days	2200	2450
4	7 days	2200	2550
5	15 days	2600	3000
6	21 days	2200	4400
7	28 days	3200	5600

Table 2. Radioactivity values in femoral heads reduced after 48 h, expressed in counts/min/g

Group 2

Subgroup	Interval between hip reduction & sacrifice	Normal femoral heads	Reduced femoral heads
1	30 minutes	2900	2500
2	24 hours	3200	2700
3	3 days	3450	3300
4	7 days	3100	3400
5	15 days	2000	3800
6	21 days	2700	3950
7	28 days	2800	5500

radioactivity levels of the reduced heads even 24 h after the reduction were higher than those measured after the first few hours. The most likely explanation for this increase in the values after the initial fall is the reversal of the vasoconstriction which occurs at the beginning in response to stress. The increased blood supply to the femoral head 72 h after reduction produced values of radioactivity in the reduced head that approximated those in the normal head.

C. The differences in the radioactivity levels between the reduced and the normal femoral heads of the two groups were not very marked at the beginning, but became significant ($P > 2$) after the third post-reduction week (Tables 1, 2 and Figure 1).

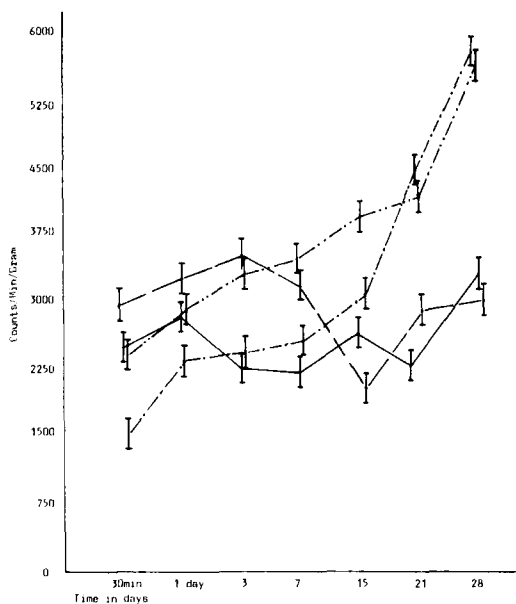


Figure 1. The graph shows the radioactivity values of the femoral head of the animals of both groups, expressed in scintillation counts/min/g. The standard deviation P is 500 counts/min/g.

— Normal femoral heads 1st group
 — Normal femoral heads 2nd group
 - - - Reduced femoral heads 1st group
 - - - Reduced femoral heads 2nd group

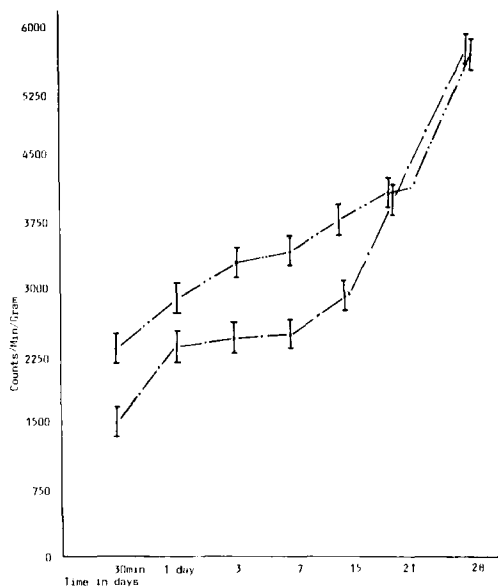


Figure 2. The graph shows the radioactivity values of the reduced femoral head of the animals of both groups, expressed in scintillation counts/min/g. The standard deviation P is 500 counts/min/g.

— reduced femoral heads 1st group
 — reduced femoral heads 2nd group

D. The levels of radioactivity of the reduced femoral heads in subgroups 6 and 7 of both groups, which include animals sacrificed 21 and 28 days after the reduction, showed that there were no significant differences between them ($P < 5$), although there were significant differences between them in the first five subgroups ($P > 1$, $P > 2$) (Figure 2).

E. In the control group of the eight animals sacrificed 2 min after the supply of radioactive microspheres, the average radioactivity value of the femoral heads was 2700 scintillation counts/min/g, which is close to the radioactivity value of the normal heads of the two groups (Tables 1 and 2).

DISCUSSION

Traumatic dislocation of the hip is the result of a high energy injury which is transmitted to the hip

joint through the femur. The resulting interruption of the continuity of the blood vessels varies according to the position of the dislocated head and the degree of violence which produced the injury.

Rabbits were used for this experiment because the blood supply of the femoral head of the rabbit is very similar to that of the human (Brookes 1971, Brookes & Harrison 1957, Trueta & Harrison 1953). The injection of microspheres was made into the heart to allow adequate mixing with circulating blood before the vessels of the hindlimbs were reached. The amount of radioactivity present in each of the femoral heads was expressed as counts per minute per gram, so that the amount of radioactivity present could be adjusted for femoral head size. Assuming that the 25- μ m-sized microspheres are trapped in the circulation just proximal to the capillary bed, the amount of radioactivity present in each head is proportional to the number of microspheres

present (Ya Po M et al. 1960, Kaihara et al. 1968).

Changes in the vascularisation of the femoral head of rabbits which may occur in experimentally produced states of aseptic necrosis have been described extensively (e.g. Rosingh & James 1969, and Rosingh et al. 1969). The observations showed that the restoration of the blood supply takes place through the development of new vessels arising from the intact areas and progressing towards the areas of the head. There is increased vascularity in the area of the affected head even in the first few days after the dislocation. The new vessels multiply continually, form anastomoses and spread towards the injured area of the femoral head. The speed at which new bone is deposited in the necrotic area of the head is very impressive and, as Bobechko & Harris (1960) showed in their experimental studies in rabbits, within 6 weeks after the injury a reasonable quantity of new bone is already present in the subchondral trabeculae. The degree of absorption of the dead bone varies and can take place only at the revascularized interfaces, which is probably the reason why some of the dead bone surrounded by new bone is absorbed so slowly. The osteoclasts come into action simultaneously with the osteoblasts and new bone is laid down 10–20 days after the injury (creeping substitution phenomenon). The re-ossification is usually completed in 6 weeks, but of course this depends on the extent of the necrotic area of bone. It is believed that the bone is alive until the time of the disappearance of the osteocytes and Patrick (1960) stated that if the blood supply is restored within 15 days, necrosis of the bone may possibly be avoided. Woodhouse (1969), however, produced bone necrosis in the femoral heads of dogs when he cut off the blood supply to the head for only 12 hours. Bonfiglio (1954) and Catto (1965a, 1965b) noticed that the disappearance of the osteocytes is not complete even 3 weeks after the interruption of the blood supply in the femoral heads of dogs. The conclusion is that some part of the bone of the femoral head dies some time after the damage to the blood supply and that in cases where re-vascularization of the bone occurs, it is difficult to estimate the speed at which this will take place.

Septic necrosis of the femoral head appears as a complication of traumatic dislocation of the hip. The incidence varies but usually is about 10–15 per cent. It is more common in posterior than in anterior dislocation of the head and this is due to damage to the most important blood vessels to the head (Thomson & Epstein 1951). Another factor which seems to be important is the time of reduction of the hip. Brav (1962) and Stewart & Milford (1954), supported the theory that early reduction of the femoral head weakens the influence of some of the factors involved in aseptic necrosis. In 262 dislocations or fracture dislocations (excluding central fracture-dislocations) Brav studied the relationship between the time of reduction and the incidence of avascular necrosis; avascular necrosis developed in 17.6 per cent of 204 dislocations reduced within 12 h after injury and in 56.9 per cent of 58 reduced 12 hours after injury. After a study of 128 dislocations of fracture dislocations of the hip Stewart & Milford found that the result was invariably poor when the reduction was not done within the first 24 hours after injury. They found that the incidence of avascular necrosis of the femoral head was 21.2 per cent for the whole group and 15.5 per cent for those treated by manipulation. Also they noticed that regardless of the type of treatment, whether closed or open reduction, the average time required for any avascular necrosis to become clinically apparent is 7 months after injury.

Stewart & Milford expressed the opinion that it is possible that avascular necrosis after dislocation of the hip is not always caused by destruction of the circulation through the ligamentum teres and the posterior part of the capsule; it may sometimes be caused by some disturbance whose nature is yet unknown, which affects not only the vessels but also other sources of nourishment of the femoral head. They suggested that the force of the injury may be important in determining whether avascular necrosis develops. That a force may cause molecular changes resulting in necrosis of bone is strongly suggested by the development of idiopathic avascular necrosis of the femoral head after a severe automobile accident in which the knee has struck the dashboard and in which there is no clinical or roentgenographic evidence of injury of the hip.

Brookes (1971) stated that the stress caused by the injury to the soft tissues and vessels in the area of the hip produces contraction of the vessels of the dislocated as well as of the contralateral normal hip, via the sympathetic system and he believes that early reduction counteracts this stress effect. In conclusion, it seems from all the above studies that early reduction of the femoral head significantly diminishes the factors leading to avascular necrosis of the femoral head, though it cannot entirely eliminate this complication.

In the rabbit the blood supply of the femoral head is quite similar to the human with the exception of the medial circumflex femoral artery, which arises from the medial part of the femoral artery and never from the deep femoral artery, and does not exist in rabbits (Brookes 1971, Brookes & Harrison 1957).

The posterior dislocation of the hip affects the medial circumflex femoral artery and the artery of the round ligament, so the blood supply to the femoral head from the remaining arteries remains basically unaffected, although even these arteries can reduce their supply, due to the reactive sympathetic stress (Brookes 1971). This reactive sympathetic stress, which is due to the damage of the soft tissues and vessels of the affected hip, is responsible for the angiospasm of the arteries of the contralateral normal hip, thus explaining the initial post-trauma decrease of the blood flow to the femoral heads, which is later restored to normal levels as the sympathetic stress subsides. The above mechanisms influencing the arterial supply to the femoral heads explain: a) the preservation of the femoral head blood supply in pure dislocations of the hip, b) the initial low levels of the radioactivity values of the normal and reduced hips of both groups, c) the progressive increase of the radioactivity values in the femoral heads corresponding to the time elapsed from the reduction of the hip.

The progressively increasing higher radioactivity values in the reduced femoral heads of both groups comparing to those of the corresponding normal femoral heads, are due to the biologic phenomenon of creeping substitution at the necrotic area of the affected femoral head, which indicates an intensive effort of the organism, supported by the increasing vascularity from the un-

affected areas of the femoral head, to replace dead bone with new.

The higher radioactivity values of the reduced and normal femoral heads in the late reduction group than in the early reduction group during the first 15 days following the reduction of the hips, it is probably due to a) the ongoing biologic phenomenon of creeping substitution in the affected area of the femoral head, which starts soon after the induced dislocation and before reduction is achieved and b) the decrease of the intensity of the sympathetic stress.

The results of this study stress that in the long term follow-up, after reduction of the hip, the radioactivity values in the reduced and normal femoral heads of both groups are quite equal. It is not easy to predict the equalisation time for the radioactivity values of the reduced and normal femoral heads. This may occur after a long period of time, as can be predicted from the still increasing radioactivity values of the reduced femoral heads, 28 days after the reduction of the hips. In conclusion, it seems that in the case of pure dislocation of the hip, the probability of complete interruption of the arterial blood supply to the femoral head, which can cause avascular necrosis, is low. In our experiments the reduction time (early vs. late) did not affect the restoration of blood supply and is therefore not considered a critical factor in the appearance of avascular necrosis.

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