BIRTH INJURY TO THE STERNOMASTOID MUSCLE

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The various theories concerning the causation of the "sternomastoid tumour" of infancy and of muscular torticollis have been fully discussed in numerous comprehensive reviews (Middleton 1930; Chandler and Altenberg 1944; Hulbert 1950; Lidge, Bechtol and Lambert 1957; Garceau 1962). Of these the theories of birth injury and of ischaemia have been the two most popular.

The theory of birth injury has been a matter of controversy since it was first postulated by Stromeyer in 1838. Stromeyer believed that the sternomastoid was ruptured during labour, giving rise to the "sternomastoid tumour," which he felt was in reality a haematoma, leading to myositis with replacement by fibrous tissue. Stromeyer's theory has been supported, fully or in part, by many subsequent writers (Spencer 1893, Bevan 1918, Meyerding 1921, Chandler and Altenberg 1944), but others have dismissed trauma and haematoma as possible causes of "sternomastoid tumour" and torticollis (Witzel 1883, Nové-Josserand and Viannay 1906, FitzSimmons 1933, Garceau 1962). Indeed, Garceau (1962) went so far as to declare that the theory of trauma, haemorrhage and haematoma should be removed from textbooks.

The theory of muscle ischaemia was first suggested by Mikulicz (1895), although he also agreed that the muscle was traumatised during birth. Nové-Josserand and Viannay (1906) suggested that the ischaemia was due to arterial occlusion, secondary to tautening of the sternomastoid muscle through extreme rotation of the head. Middleton (1930) believed that venous, rather than arterial, occlusion was responsible for the ischaemia. Although the theory of ischaemia has been disputed by some writers (notably Hulbert 1950), Adams, Denny-Brown and Pearson (1962, p. 309) considered it to be the most satisfactory explanation of "sternomastoid tumour" and wryneck, since the pathological changes were similar to those of Volkmann's contracture. These authors noted that there was no agreement as to whether the ischaemia was due to venous or arterial occlusion.

We consider the following case worthy of publication, firstly because it proves that severe injury may occur to the sternomastoid muscle during birth and clearly demonstrates the striking histological features of such injury, and secondly because it permits a considered opinion as to the manner of development of the "sternomastoid tumour" of infancy.

CASE REPORT

A premature female infant (1,850 grammes) was born after a difficult breech delivery. The mother was a primigravida, aged eighteen, with uncomplicated pregnancy. The baby was in poor general condition at birth, developed respiratory distress and died forty-two hours after birth.

At necropsy there was moderate jaundice of the skin with bruising over the buttocks but no external signs of injury over and around the neck itself. The lungs were firm and poorly aerated, and death was attributed to the respiratory distress syndrome. As part of a current investigation of the cerebral arteries of the newborn, the arteries of the head and neck were routinely injected via the ascending aorta with a suspension of micropaque and gelatin. Upon dissection of the neck, after completion of the injection procedure, the right sternomastoid muscle was found to be swollen and haemorrhagic throughout, firm and of a remarkable

bluish-red colour, contrasting sharply with the normal pale creamy-pink sternomastoid on the left side. No macroscopic abnormalities were noted in the major veins and arteries of the neck.

Histologically, numerous sections from the right sternomastoid showed widespread

FIG. 1

General view of damaged muscle, showing widespread rupture of muscle fibres, many of which are expanded and racquet-shaped, probably due to retraction at time of rupture. (Phosphotungstic acid haematoxylin, $\times 100$.)

FIG. 2

Swollen and fragmented muscle fibres, some with pyknotic nuclei and fine vacuolation. Interspersed are extravasated erythrocytes and occasional polymorphs and histiocytes. Compare size of muscle fibres with that of normal muscle from the left sternomastoid, shown in Figure 8 at the same magnification. (Haemalum and eosin, $\times 500$.)

haemorrhage within and around the muscle bundles, together with striking changes in the muscle fibres themselves. Many of the muscle fibres were ruptured and fragmented (Figs. 1 to 4), the torn fragments often being expanded up to two or three times their normal width,

assuming a clubbed or racquet-like shape. In many instances this expansion strongly suggested that the muscle fibre had undergone sharp retraction at the time of rupture. Some of the torn fragments appeared to be viable, retaining their nuclei, but others had undergone necrosis

FIG. 3

Gross alteration and irregular coarsening of striations in fragmented muscle fibres. Also note great variation in fibre width. (Phosphotungstic acid haematoxylin, $\times 400$.)

FIG. 4

Racquet-like fragments of torn muscle fibre, with alterations in striations. Rupture lines clearly shown; peculiar expansion of fibres probably due to retraction at time of rupture. (Phosphotungstic acid haematoxylin, $\times 400$.)

as shown by pyknosis or disappearance of their nuclei and marked alteration and irregular clumping of the transverse striations of their myofibrils (Figs. 3 and 4). Fine cytoplasmic vacuolation was present in some such fibres. The tearing and fragmentation of muscle fibres

could be seen throughout the muscle, although intact and viable muscle fibres were often present in immediate proximity to fragmented ones. The proportion of fragmented fibres to intact ones varied from area to area, being very high in some areas, much lower in others.

FIG. 5

Reticulin structure of damaged muscle showing extensive irregular tearing and disorganisation of endomysial sheaths. Arrow points to one of the larger tears. (Silver impregnation for reticulin, $\times 250$.)

FIG. 6

Reticulin structure of damaged muscle; another field at higher magnification, showing disorganisation of endomysial sheaths. (Silver impregnation for reticulin, $\times 400$.)

The fragmentation of muscle fibres was obviously accompanied by disruption of the perimysial and endomysial sheaths, best demonstrable in reticulin preparations (Figs. 5 and 6).

In none of the sections examined was there any evidence of venous or arterial thrombosis.

The arteries were all filled with injection medium (Fig. 7). There was a sparse infiltration by neutrophilic polymorphonuclear leucocytes and small groups of young histiocytes were present in some foci (Figs. 2 and 7), but there was no significant fibroblastic proliferation. Sections

FIG. 7

Small arteriole in damaged muscle, containing injection medium. Haemorrhage and occasional polymorphs and histiocytes in adjoining muscle (*right*) and in fibrous septum (*left*). (Haemalum and eosin, $\times 500$.)

FIG. 8

Transverse section from unaffected left sternomastoid muscle. Compare width of normal muscle fibres with abnormal fibres in Figures 2 and 7, which are both at this magnification. (Haemalum and eosin, $\times 500$.)

from the left sternomastoid showed no haemorrhages and the muscle was essentially normal (Fig. 8), although a very occasional clubbed torn muscle fibre was seen on this side also.

DISCUSSION

The lesion herein described is obviously not a simple uncomplicated haemorrhage or haematoma of the muscle. The leakage of blood into the tissues is merely a concomitant of a fundamental damage to the muscle fibres themselves. It is perhaps unfortunate that the haemorrhagic component or "haematoma" has been stressed by some writers in the past, because this has led to assertions that haemorrhages or haematomata in muscles resolve completely and do not lead to contracture. Spencer (1893) in his paper entitled "On haematoma of the sternomastoid muscle in newborn children" indeed described the very same muscular changes as noted in our own case, despite his emphasis on the haemorrhagic element.

In our case there is evidence that the muscle damage was due to a combination of trauma and necrosis. The evidence pointing to trauma is not merely the haemorrhage, but particularly the extensive tearing and disorganisation of the muscle fibres and their endomysial sheaths, and the close juxtaposition of damaged and undamaged muscle fibres in many areas. Morison (1965), who kindly examined sections from our case, noted this interesting feature and remarked that he had seen the same phenomenon in the experimental crush muscles in the mouse and also in the human crush changes in air-raid victims. The necrosis of muscle fibres is evident on histological criteria and is presumably due to ischaemia at the time of trauma. Any primary or sustained arterial or venous occlusion, within or outside the muscle, was confidently excluded as the cause of the ischaemic necrosis in our case. We believe that both the traumatic and ischaemic factors operated on the muscle locally, simultaneously and temporarily, probably through stretching and crushing of the muscle during the birth process. The exact mechanics of the injury is beyond the scope of the present paper, but it is of interest to note that the child in our case was born by a breech delivery, the type of delivery most frequently involved in the "sternomastoid tumour" of infancy and of congenital muscular torticollis.

The nature and extent of the muscle damage in our case is clearly such that complete resolution could not have been expected had the infant lived, and there are sound pathological grounds to support the thesis that, in the ensuing process of repair, a typical "sternomastoid tumour" would have developed. As Adams *et al.* (1962, p. 501) have pointed out in their account of muscle trauma, "If the parallel arrangement of endomysial tubes is destroyed by the injury, fibrous tissue proliferation is extensive and prevents effective regeneration. The muscle is then eventually replaced by a large mass of scar tissue." Extensive disorganisation of the endomysial sheaths has been amply demonstrated in our case, and the gaps in and around the torn sheaths will eventually have been filled by fibroblastic tissue. This is exactly what is seen histologically in the "sternomastoid tumour" of infancy which consists of a mass of fibroblastic tissue engulfing isolated fragments of muscle fibres which usually show considerable variations in size (Garceau 1962), the same variations as were noted in our case.

It is almost certain that the "desmoid tumours" of muscle have a similar pathogenesis. These bear a striking histological similarity to the "sternomastoid tumour" of infancy, showing scattered muscle fibres embedded in fibroblastic tissue. Most abdominal desmoids occur in women after pregnancy with prolonged labour and violent muscular contractions (Adams *et al.* 1962, p. 486), and elsewhere in the body their development is often linked with a history of trauma (Musgrove and McDonald 1948).

SUMMARY

1. A case is described of severe birth injury to the sternomastoid muscle in a breech-delivered two-day-old infant. The affected muscle showed widespread haemorrhage, fragmentation and necrosis of its fibres, and disruption and disorganisation of the endomysial sheaths.
2. Disruptive muscular trauma of this type is known to lead to florid fibroblastic proliferation with formation of a large mass of scar tissue. It is suggested that the "sternomastoid tumour" of infancy develops as a sequel to such trauma occurring during birth.

3. The theories of birth trauma and of ischaemia, in the etiology of the "sternomastoid tumour" of infancy and of congenital muscular torticollis, are not mutually exclusive but may be complementary, the circumstances causing the trauma also leading to ischaemic damage.

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