THE SIGNIFICANCE OF HARRISON'S GROOVES

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The origin of the term "Harrison's grooves, or sulcus," has hitherto been obscure. Three standard medical dictionaries—Dorland's, Stedman's, and Gould's—attribute the eponym to *Edward* Harrison (1766–1838); but no account of the deformity can be found in any of his works (Edward Harrison, 1806, 1810, 1820). Dr. Charles Singer told us that because of this uncertainty Sir Frederic Still was unable to include a note on the grooves in his *History of Paediatrics*.

Samuel Gee (1870) and W. Fox (1891) both refer to *Edwin* Harrison's method of determining the height of the diaphragm by means of the lateral thoracic grooves, which were apparently common in the early part of the last century. In following up this clue we have been successful in exhuming the original description of the grooves.

Edwin Harrison (1779–1847), a physician to the St. Marylebone Infirmary, wrote no books and few papers. However, the pages of the *London Medical Gazette* for the years 1835–7 reveal that he was interested in the grooves as a physical sign in examination of the chest. The first inkling comes from an article by a Dr. Williams on Oct. 3, 1835. He refers to the grooves, and states that "the chief object of this communication is to announce them to the profession as original observations of my friend, Dr. Harrison, whose peculiar repugnance to authorship deters him from doing so himself." Then, in July, 1836, Clendinning (in a paper which is printed next to Marsh's original description of his test for small quantities of arsenic) gives more details and an acknowledgment to Harrison. Finally, in 1837, Edwin Harrison managed to overcome his "peculiar repugnance to authorship," and we have a note which included the following observations:

"2. On moving the hand along either side of the chest vertically, on what may be considered as the median line, it will sink into a depression corresponding to the height of the diaphragm on that side.

"3. A depression, or depressions, between the ribs can be felt or seen, or both felt and seen, at each abdominal inspiration, indicating (at least in the physiological state) the presence of the diaphragm in that part of the chest.

". . . If it should turn out that my opinions are not so original as I believe them to be, I trust that I shall always be found ready to make the avowal."

Theories of Causation

The mechanism of the formation of the grooves has also remained obscure, and has been the theme of many fairy-tales told to students in their early clinical days. Rilliet and Barthez (1843) first expressed the view that the grooves correspond to the attachment of the diaphragm to the ribs, and this error has been repeated in textbooks up to the present day (Holt, 1939; Nelson, 1946; Paterson and Moncrieff, 1947). Seventeen years later Jenner (1860) pointed out that the grooves are level with the upper surfaces of the liver, spleen, and stomach, whereas the diaphragm is attached to the margins of the ribs and costal cartilages diagonally along the thoracic outlet. This observation was given publicity by Charles West (1874) in what was for many years a standard textbook on children's diseases.

Rilliet and Barthez (1843) also postulated a gap between the attachments of the major muscles of thoracic respiration and those of the diaphragm: they suggested that the negative intrathoracic tension is in that area able to overcome muscular resistance and so produce indrawing of the ribs. Jenner (1860) was unable to find this gap. Examination of a cadaver confirms his observation. The ribs and cartilages are clothed closely in muscular attachments, and the insertions of serratus magnus interdigitate most intimately with those of the external and internal oblique muscles. Moreover, the line of insertion of serratus magnus runs diagonally across the site of Harrison's grooves.

Jenner (1860) considered that the determining cause of the deformity was atmospheric pressure, aided by the elastic contractility of the lungs. He stated that if the thoracic wall were made of cast-iron the diaphragm would descend only so fast as the air could enter the lungs. If the chest wall were soft, or if there were obstruction to the entry of air into the lungs, recession of the wall would be expected to occur. This recession would occur at the most yielding part of the wall—that is, on either side of the sternum, at the costochondral junctions, and at the anterior ends of the ribs. The abdominal viscera would prevent the collapse of the lower ribs.

Herlitz (1945) states that he has observed hiccuping in infants, and that in them reaction of the chest wall occurs at the same site as Harrison's grooves. Hiccup is due to involuntary contraction of the diaphragm, which sucks in air so suddenly that the vocal cords are drawn together. After this the negative pressure in the thorax must become very great. If further proof of the role of non-expansion of the lung be needed, try the experiment of pinching a newborn baby's nose while its mouth is kept shut. Recession of the ribs at the site of Harrison's grooves then occurs. Dupuytren (1847) noted an association between the grooves and an enlargement of the tonsils in children at the breast, but the children he described seem to have been suffering also from rickets and bronchitis. Fisher (1906) noted the development of a unilateral groove in a child suffering from pulmonary collapse following measles.

Rogers-Harrison (1842) attributed the grooves to the habit of grasping infants tightly round the chest when lifting them. Rees (1850) thought that they were due to the infant's arms being folded across its chest and bound too tightly with swaddling-clothes. McGonigle (1931) stated that they were due to the abdominal enlargement which is found in rickets pressing the lower ribs outwards.

Jenner's reasoning does not adequately explain why Harrison's grooves should develop where they do. Recession of the chest wall, according to him, should occur along the line of the costal cartilages on either side of the sternum, but in fact this type of *vertical* groove is commonly seen only in severe rickets, when the anterior ends of the ribs and costal cartilages are softened by disease. There must be some other explanation to account for the extremely frequent development of the *transverse* Harrison's grooves. Here it is necessary to refer back to the theory of the muscular gap. Although no such gap exists, it is nevertheless true that at the site of Harrison's grooves there is a neutral zone between two parts of the thorax, each moving in a slightly different direction. Thus the upper ribs move upwards and forwards, rotating outwards, while the lower ribs move outwards and backwards, increasing the transverse diameter of the upper abdomen (Johnston, 1932). It would seem likely, therefore, that increased negative intrathoracic tension would tend to cause buckling in of the chest at the hinge between these two movements rather than anywhere else.

Method of Study

The present investigations were undertaken in order to provide some factual basis for discussion, and in the hope of stimulating thought and research in the future. The full results and the analysis are given in a work already published by one of us (J. Naish, 1947).

The following groups were studied : (1) 500 children selected at random from 23 primary schools in the City of Bristol ; (2) 25 children who were diagnosed as suffering from rickets eight years previously ; (3) 100 children attending a clinic for asthmatics ; and (4) 36 children suffering from congenital heart disease.

The depth of the grooves was measured with a home-made instrument, which was calibrated in fractions of an inch. This instrument was applied vertically to the chest at the site of the maximum depth of the groove. A light even pressure was used in order to avoid sinking the flat edges of the instrument into the skin. The width of the measuring blade prevented this from sinking into an intercostal space and so giving a false reading. Girls at about the age of puberty could not be examined because development of the breasts interferes with these measurements.

Control Group

The 500 children, whose ages ranged from 5 to 12, were examined at routine school inspections at 23 primary schools in the City of Bristol. The incidence of the grooves is given in Table I. Analysis of the full figures showed

TABLE I.—*Incidence of Harrison's Grooves in Control Group*

Harrison's Grooves	No. of Children	Percentage of Total
Absent	272	54·4
Unilateral	17*	3·4
Up to 3/16 in. (0.48 cm.) deep ..	177	35·4
3/16 in. or more deep	34	6·8
Total:	500	

* 13 (77%) of these were left-sided. This finding contrasts with the statement that the grooves are commoner on the right side (Holt, 1939).

that there was no significant difference of incidence between males and females or between separate age groups.

A full examination was possible in only 354 of the 500 cases ; in the rest (146), owing to the absence of the parents or some other cause, a satisfactory history could not be obtained. The incidence of grooves in the 354 children was not significantly different from that in the whole group (45.2% as against 45.6%).

Positive Results.—(1) Harrison's grooves occurred more often in those who had suffered from pneumonia in early life. (2) The association of the grooves with bronchitis, measles, and pertussis in early life was probable, but not statistically significant. (3) From the parents' statements it also seemed that grooves occurred more often in children who had suffered from greater degrees of upper respiratory disease (frequent colds or sore throats and ear disease) in infancy and early childhood, but this finding was not significant. (4) Grooves occurred with significant frequency in children with pigeon breasts, and in these children there was often other evidence of past rickets. (5) Grooves were found to be commoner in the children of poorer parents.

Negative Results.—(1) Breast-fed children were no less liable to develop Harrison's grooves than bottle-fed children. (2) Vitamin D fortification of diet in infancy did not reduce the incidence. (3) Neither present objective evidence of past rickets nor smallness of stature was associated with an increased incidence.

In addition the grooves occurred in a number of healthy children who had no history of previous disease.

The association with pneumonia is shown in Table II. The grooves occur more often and are deeper in those who have had pneumonia, especially during the first year of life or on more than one occasion. The results are significant.

TABLE II.—*Association of Harrison's Grooves with a History of Pneumonia*

Harrison's Grooves	No Pneumonia	Pneumonia Occurred			
		At 2 Years or Over	Between 1 and 2 Years	Below 1 Year	Twice or More
Absent ..	179	10	3	1	1
Unilateral ..	9	2	0	1	1
Up to 3/16 in. deep	107	5	2	3	7
3/16 in. or more deep	17	3	0	2	1
Totals: ..	312	20	5	7	10

The social and economic conditions of the 121 children selected at random were investigated by Miss E. C. Moynagh, lady almoner at the Bristol Royal Hospital. On the basis of the average weekly income of the household during the first two years of their lives, the children have been separated into two groups, with a dividing-line at 10s. per head. The results show that grooves are more common in poverty-stricken households (Table III). The difference is significant.

TABLE III.—*Association of Harrison's Grooves with Poverty*

Grooves	No. with Average Income Over 10s. per Week per Person	No. with Average Income Under 10s. per Week per Person
Absent ..	37 (59·6%)	18 (30·5%)
Present ..	25 (40·4%)	41 (69·5%)

Rickets

Dr. Corner (1944) recorded the cases of a large number of children who had been diagnosed as suffering from rickets eight years previously. She kindly allowed us to examine her records, and we chose 60 children in whom the evidence of rickets in infancy had been unequivocal. Only 25 mothers were able to bring their children for re-examination. This poor response was due chiefly to population movements during the war. The results are set out in Table IV. Of the 25 children examined 16 (64%) had grooves. A high proportion of those with deep grooves had shown either unequivocal clinical evidence of rickets in infancy or very high plasma phosphatase values, or else had been diagnosed after the age of 6 months.

TABLE IV.—*Evidence of Past Rickets Correlated with Depth of Harrison's Grooves*

Case No.	Age When Diagnosed (months)	Evidence of Rickets in Infancy in 1938					Depth of Harrison's Grooves in 1946
		Rib Beading		Craniotabes	Plasma Phosphatase (units/100 ml.)	Radiological Rickets	
		Minor	Marked				
1	11	—	—	—	23·5	Yes	Absent
2	2	Yes	—	—	23·5	—	,,
3	15	,,	—	—	13·5	Yes	,,
4	2	—	Yes	—	24·0	—	,,
5	5	—	—	Yes	19·5	—	,,
6	12	—	Yes	—	—	Yes	,,
7	3	Yes	—	—	30·0	—	,,
8	4	,,	—	—	18·0	—	,,
9	N.K.	—	—	—	—	—	,,
10	10	Yes	—	—	17·0	—	Minimal*
11	2	,,	—	—	21·5	Yes	Left unilateral
12	6	,,	—	—	18·0	—	Minimal*
13	1	,,	—	—	30·0	—	,,
14	12	,,	—	—	17·0	—	,,
15	3	—	Yes	—	22·0	—	,,
16	3	Yes	—	Yes	50·0	—	3/16 in. deep
17	2	,,	—	—	20·0	—	,, ,,
18	2	,,	—	Yes	80·0	—	,, ,,
19	19	,,	—	—	12·5	—	,, ,,
20	14	,,	—	—	78·0	Yes	,, ,,
21	2	—	Yes	—	13·0	—	,, ,,
22	4	Yes	—	—	25·0	—	1/8 in. ,,
23	8	,,	—	—	13·0	—	3/16 in. ,,
24	5	—	Yes	—	56·0	—	1/8 in. ,,
25	8	Yes	—	—	—	—	,, ,,

* Minimal = One side less than 1/8 in. (0·32 cm.) deep, but grooves present on both sides.

In drawing conclusions from these findings it should be understood that treatment has probably modified the course of the disease, particularly where the diagnosis was made early. There are two unknown factors in each case: (1) the duration of rickets before diagnosis, and (2) the extent of bone-softening before treatment was instituted. It is tempting to postulate that the children with the highest plasma phosphatase values are those with the greatest degree of bone-softening. This is supported by the results in Table IV, but the number of children examined was too small for significant results to be obtained.

Asthma

We examined 100 children at the Bristol Children's Hospital in 1946. All were suffering from chronic bronchial asthma and were attending a special clinic under the care of Dr. Corner. There were 65 boys and 35 girls, whose ages ranged from 3 to 13. Of these 79% had Harrison's grooves, and 36% had grooves at least 3/16 in. (0.48 cm.) deep. There is a significant difference, therefore, between the incidence in this group and that in the control group. Deep grooves occurred more often in children in whom asthma developed early in life, but the difference in incidence is not significant. There was, however, a significant association between the occurrence and depth of the grooves and the duration of the asthma. This is shown in Table V. A history of pneumonia had no influence on the incidence of the grooves in these children.

TABLE V.—*Harrison's Grooves and Duration of Asthma*

Grooves	Duration of Asthma Less than 4 Years	Duration of Asthma 4 Years and Over
Absent	15	6
Less than 3/16 in. deep ..	18	25
3/16 in. or more deep ..	3	33
Totals: ..	36	64

(*Note*: We have been unable to gain access to the paper written by J. Brock in 1942 entitled "Uber Pseudorachitis Asthmatica.")

Congenital Heart Disease

A. E. Naish (1945) records eight cases of congenital heart disease seen in infancy with marked inspiratory indrawing of the lower intercostal spaces. There was no evidence of rickets or upper respiratory obstruction in these children, and in only one was the heart grossly enlarged. He told us that five of them were observed for three years, and in that time three died and two developed Harrison's grooves.

We examined 36 unselected children with congenital heart disease attending Prof. C. Bruce Perry's cardiac clinic at the Bristol Royal Infirmary. The results are shown in Table VI. The numbers in each group are too small to be analysed separately, but the incidence of grooves in cases other than those of apparently uncomplicated pulmonary stenoses and of patent interventricular septa was significantly greater than in the control group.

TABLE VI.—*Harrison's Grooves in Congenital Heart Disease*

Grooves	Controls	Pulmonary Stenosis or Patent Interventricular Septum	Other Congenital Heart Disease
Absent	272	11	2
Present	228	12*	11*

* Including three cases in infancy with marked indrawing of lower intercostal spaces at the usual site for Harrison's grooves.

Summary and Conclusions

Reference is made to the original description of lateral thoracic grooves by Edwin Harrison.

Theories as to their causation are summarized. The theory that the grooves are caused by the insertions of the diaphragm has been disproved several times and should be abandoned.

A study was made of a sample of 500 Bristol schoolchildren, 25 cases of past rickets, 100 cases of asthma, and 36 cases of congenital heart disease.

Harrison's grooves occurred in 45.6% of normal schoolchildren between the ages of 5 and 12 years. Grooves at least 3/16 in. deep were present in 6.8%. The grooves were often found in children known to have had severe rickets in infancy; on the other hand, in milder cases or cases diagnosed and treated early their incidence was not different from the normal. They occurred very frequently in children with the deformity known as pigeon breast, which is almost certainly due to severe rickets. In these children vertical grooves on either side of the sternum are often also present. Whistler (1645) noted: "At first the sternum is depressed, but as the disease progresses it projects." The conclusions reached by Dalyell and Mackay (1923) that Harrison's sulcus is valueless as an early sign of rickets has been borne out by this study; while the older observations that the grooves develop (frequently in association with a pigeon breast) in severe rickets has been substantiated.

The grooves occurred more often in children who had had pneumonia, particularly if they had suffered from the disease in the first two years of life. They also occurred frequently in children who had had bronchitis, measles, pertussis, or severe upper respiratory infections in early life, but the statistical significance of these findings could not be proved. The heaviest incidence was in children suffering from asthma, and the longer the asthma had persisted the more likely were the grooves to be present.

These results seem to support the suggestion that the most important factor in the production of Harrison's grooves is deficient expansion of the lungs.

Grooves were also frequently found in children with severe grades of congenital heart disease. The explanation of this is not clear. Sheldon's (1938) suggestion that the probable cause of indrawing of the intercostal spaces is pulmonary atelectasis occurring as a complication of the heart disease seems to fit in with the known facts, but this has not been confirmed.

We wish to express our gratitude to Prof. C. Bruce Perry for his help and advice during this investigation; to Dr. Beryl Corner for allowing us access to her cases and records; and to Miss E. C. Moynagh for her work on the social and economic conditions of some of our cases. We would also like to thank Dr. J. Brierley for advice on anatomical considerations, Dr. George Smart for assistance with statistical calculations, and Mr. G. F. Home, librarian of the Royal Society of Medicine, for helping us to find Harrison's original description.

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"MYANESIN" AS A RELAXANT IN CHILDREN

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α : β : dihydroxy-γ-(2-methylphenoxy)-propane ("myanesin," British Drug Houses Limited) is obtainable in 10-ml. ampoules of a 10% solution. Its use as a relaxant in anaesthesia was first described by Mallinson (1947), and its pharmacology has been discussed by Berger and Bradley (1947). Unlike curare, myanesin does not appear to interfere with voluntary movements, but only with spinal reflexes, and therefore, presumably, with muscle tone. It may be suggested that its site of action is the internuncial neurones, which might account for the fact that respiration is not inhibited for long periods. Toxicity is low.

Myanesin has been used by me since February, 1947. In adults its action was not so marked as that of curare, and it was enhanced by ether and by thiopentone, while its effect was minimal when only nitrous oxide or cyclopropane was the anaesthetic used. Further, although abdominal relaxation might occur, laryngeal spasm was not prevented. It therefore seems that its use in adult surgery is limited, although it may be of value in cases in which the respiratory depression caused by curare may be a source of alarm.

Relaxation may be obtained for abdominal surgery in infants by a number of methods. Deep ether anaesthesia is open to obvious dangers; local analgesia is time-consuming, and comparatively large doses of the agent may be needed, and even then relaxation may not be perfect; spinal analgesia, while being unsuitable psychologically for children over 2 years of age, is difficult to give to younger infants, and there are obvious hazards associated with it. Curare, in my hands, has given good relaxation, but has always been associated with profound respiratory depression. This may be serious in the infant, owing to the difficulty of controlling it: closed and semiclosed methods of anaesthesia are contraindicated on account of the "dead-space," while without endotracheal intubation it is almost impossible to inflate the lungs and not distend the stomach. It is questionable whether intubation, with consequent narrowing of the glottis, is advisable in very small infants.

Myanesin was administered to 44 children between the ages of 24 days and 4½ years. The results have been satisfactory. In all cases maintenance of anaesthesia was with open ether, induction being with ether, nitrous oxide, or ethyl chloride. There were 16 cases of intussusception, 16 of pyloric stenosis, and 12 of appendicitis, with or without peritonitis. Anaesthesia was maintained in first plane, third stage; in cases lasting up to 20 minutes it was found that one dose of myanesin sufficed, and that no more ether was required after the peritoneum had been opened. Recovery was extremely rapid, and the post-operative condition was always better than would have been expected had other means been employed. Respiratory depression lasting 15 to 30 seconds occurred in one-third of the cases; in one case respirations were completely inhibited for nearly half a minute. After the initial depression the tidal air assumed normal proportions. There were no obvious changes in pulse rate, although blood pressures were not taken. There were no deaths during operation, but two occurred after operation, in neither case ascribable to myanesin. Relaxation came on rapidly and was good for 10 to 25 minutes after injection, its extent being sometimes obscured by pre-existing distension of the bowel. Clinically, no effect upon bowel movements or tone could be seen.

The injection of myanesin was made into the intravenous drip, if one were set up, or into the longitudinal sinus at the posterior angle of the anterior fontanelle, for at this point the sinus is wider than in front and the approaching edges of bone direct the needle automatically into the vein. Blood is aspirated before injection, but it is unlikely that intrathecal injection would be dangerous. The dose was in the order of 2 ml. per stone (6.35 kg.) of body weight.

The following cases, selected from the series, illustrate the dosage and effect of myanesin in children.

Case Reports

Case 1.—Pyloric stenosis. Aged 24 days. Weight 9 lb. 3 oz. (4.17 kg.). General condition good. Rammstedt's operation. Induction and maintenance with open ether. Myanesin 1.5 ml. into the longitudinal sinus at six minutes. Slight respiratory depression for 30 seconds. Relaxation excellent and still present at the conclusion of operation at 26 minutes. Child began to cry as the last suture was inserted. No ether had been given after the peritoneum was opened. Recovery uneventful.

Case 2.—Pyloric stenosis. Aged 4 weeks. Rammstedt's operation. Induction and maintenance with open ether. Myanesin 1 ml. at eight minutes. Operation concluded at 19 minutes. Relaxation good. No respiratory depression. Recovery uneventful.

Case 3.—Pyloric stenosis. Aged 6 weeks. Weight, 7 lb. 2 oz. (3.23 kg.). Rammstedt's operation. Induction with open ether, maintenance with ether–oxygen endotracheally with Ayre's T-piece. Myanesin 0.75 ml. at five minutes. Operation concluded at 19 minutes. Relaxation poor at first (? under-dosage), although there was slight respiratory depression for 15 seconds. Relaxation good during closure. Recovery uneventful.

Case 4.—Pyloric stenosis. Aged 8 weeks. Rammstedt's operation. Induction and maintenance with open ether. Myanesin 1.5 ml. into the longitudinal sinus at seven minutes. Operation concluded at 20 minutes. Relaxation excellent. Respirations completely inhibited for 20 seconds after myanesin had been given. This inhibition of respiration caused some alarm while it lasted, but there was no change in colour. When respiration was resumed it was at first rather shallow, but it became normal after a further 30 seconds. Recovery uneventful.

Case 5.—Intussusception. Aged 3 months. Weight, 11 lb. 5 oz. (5.13 kg.). Induction and maintenance with open ether. Myanesin 3 ml. at five minutes. Operation concluded at 27 minutes. No respiratory depression. Relaxation good; reduction of the intussusception intraperitoneally. Recovery uneventful.

Case 6.—Intussusception of three days' standing. Aged 4 months. Weight, 10 lb. 10 oz. (4.82 kg.). General condition fair. Intravenous drip (plasma) set up two hours before operation. Induction and maintenance with open ether. Myanesin 1.5 ml. was administered at seven minutes, with slight respiratory