EXAGGERATED SPREAD OF EPIDURAL ANALGESIA IN ARTERIOSCLEROTIC PATIENTS

DOSAGE IN RELATION TO BIOLOGICAL AND CHRONOLOGICAL AGEING

BY

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From time to time reports are published of massive spinal blockade with coma and cardiovascular collapse complicating epidural analgesia. In some cases there seems to be a fairly obvious reason for the accident in the form of direct or circumstantial evidence that the dura was punctured at some stage of the procedure, either before, or during, or after injection of a considerable volume of local anaesthetic into the extradural space (de Saram, 1956; Sykes, 1958). In these cases it was assumed that a fair proportion of the solution had found its way into the subarachnoid space through the puncture hole in the dura, causing a massive subarachnoid block, involving the cranial nerves and producing complete deafferentation with resulting loss of consciousness (Gordon-Jones, 1953).

However, there have been other cases where no definite reason could be given for the unexpected onset of total spinal blockade (Mostert, 1960). Epidural puncture was technically faultless, and no cerebrospinal fluid or blood could be aspirated from the needle, and yet total spinal blockade and unconsciousness followed the injection of an apparently modest quantity of local anaesthetic solution. Sometimes the accident happened quickly (Stringer, 1954), but often there was a delay of 30 to 40 minutes, or even longer (Morrow, 1959). The published cases seem to have one feature in common. The patients are usually reported as suffering from some degree of arteriosclerotic vascular disease, often associated with diabetes mellitus.

During the course of a previous investigation into the spread of epidural analgesia I noticed that arteriosclerotic patients required smaller doses than normal subjects of the same age in order to block a given number of spinal segments (Bromage, 1962). The present study is an extension of the previous one, in an attempt to arrive at some quantitative conclusions about the effects of occlusive vascular disease on epidural analgesia, for it seemed likely that some of the reported cases of apparently bizarre epidural spread were really conforming to a pattern of relative overdosage, which could have been avoided if the necessary knowledge had been available at the time.

Methods

Controls

A total of 314 normal patients between the ages of 20 and 85 were given epidural analgesia prior to undergoing elective surgical operations. Of these, 255 were studied in the previous investigation already mentioned.

Injectations were given to 175 patients in the horizontal position: 2% solutions of three related local anaesthetic drugs were used in these subjects—lignocaine (60 cases), mepivacaine (60 cases), or L67, a new analgesic, xylydide,

closely related to the preceding two (55 cases). Adrenaline 1/200,000 was added to these solutions.

A further 139 patients were given epidurals in the sitting position, the horizontal posture being assumed five minutes later. Of these, 80 received 2% lignocaine, 23 were given 3%, 21 4%, and 15 5% lignocaine.

Epidural puncture was carried out by the loss-of-resistance method, at the second lumbar interspace, using a No. 16 or 17 Tuohy needle with the orifice directed towards the head (Sicard and Forrestier, 1921). The test syringe was filled with analgesic solution or air, instead of saline, so as to obtain an exact measure of the amount of analgesic solution introduced into the epidural space without dilution by any other solvent. The analgesic solution was injected at a constant rate of about 1 ml. per second. Skin analgesia was determined at minute intervals thereafter, using a No. 22 needle, or by nipping the skin with the finger-nails. The upper and lower margins of analgesia were plotted on graph paper, as spinal segments against time to the nearest whole minute, in order to obtain a visual record of the segmental pattern of onset, as well as the time taken for complete spread of analgesia to its farthest limits. Dosage was then expressed as the ratio of the number of analgesic segments to the volume of solution injected, or, in other words, the number of millilitres of solution required to block one spinal segment.

For example, supposing injection of 20 ml. resulted in analgesia from S5 to T7 inclusive. The segments blockaded would include five sacral (the coccygeal segments were not counted, since they occupy a very small area of the cord), five lumbar, and six thoracic, or 16 segments in all. Thus the dose per spinal segment in this case would be 20/16, or 1.25 ml. per spinal segment.

The dose per spinal segment was then plotted against age for each patient. Regression lines for spread on age were calculated for the 2% solutions, and the 95% fiducial limits for the populations were drawn in. Approximate regression lines were drawn by eye from the individual data of the 3%, 4%, and 5% solutions.

An attempt was made to estimate the amount of motor block in the lower limbs 35 to 40 minutes after injection, in terms of ability to move the toes or bend the knees. This could be carried out in only a proportion of the patients, since many were given light general anaesthesia at about this time for social reasons.

Arteriosclerotic Patients

Fifty-six epidural injections were given to 53 severely arteriosclerotic patients (34 male, 19 female) with vascular insufficiency of the legs or myocardium for surgical or diagnostic reasons. Of these patients, 25 were suffering from diabetes mellitus.

Exactly the same technique was used as for the controls, except that smaller doses of analgesic solution were employed. One patient received 2% L67, six patients were given 2% mepivacaine, and the remainder received 2% lignocaine. Thirty injections were made in the supine and 26 in the sitting position.

Results

Without exception, a given dose of epidural analgesic produced more extensive blockade in the arteriosclerotic patients than in normal subjects of the same age. There was also a tendency for blockade to develop more slowly in the former group.

Spread of Analgesia

Table I shows the spread of analgesia for 3%, 4%, and 5% lignocaine in 59 normal subjects arranged in order of age, the results being expressed in millilitres of solution per spinal segment (as explained under "Methods"). Table II shows the data for spread and latency of analgesia in the 53 arteriosclerotic patients. In some of these cases strikingly widespread blockade was produced by very small volumes of solution. For example, Fig. 1 shows the pattern of spread in one severely arteriosclerotic patient given two epidural blocks five days apart, using 6 ml. of 2% lignocaine on one occasion and 3 ml. on the other.

Fig. 2 shows the data for spread and age in graphic form, combined with control results for 2% lignocaine from a previously published series. In Fig. 2A the regression line for 2% lignocaine in the supine position is surrounded by a shaded area showing the confidence limits within which 95% of all normal patients may be expected to fall (Bromage, 1962). The individual results

from 30 injections in the supine arteriosclerotic patients are shown against this background, and it can be seen that in every case the dose per spinal segment is less than the normal mean. The difference between the two groups is highly significant ($P < 0.001$).

Fig. 2B shows the results obtained in the sitting position. As in Fig. 2A, the regression line for 2% lignocaine from a previous normal series has been shown with its 95% fiducial limits. Approximate regres-

FIG. 1.—Extensive spread of epidural analgesia in 66-year-old diabetic arteriosclerotic man with gangrene of feet (see text). Light and heavy shading represent unilateral and bilateral analgesia respectively.

FIG. 2.—Relationship of age and epidural dose requirements in normal and arteriosclerotic patients (see text). ● = 2% lignocaine. ○ = 2% mepivacaine. × = 2% L67.

TABLE I.—Concentrated Epidural Solutions in 59 Normal Patients. Age Related to Volume of Solution Required to Block One Spinal Segment with 3%, 4%, and 5% Lignocaine

3%			4%			5%		
No.	Age	Dose (ml.)	No.	Age	Dose (ml.)	No.	Age	Dose (ml.)
1	25	1.00	24	20	0.89	45	20	0.75
2	27	1.25	25	21	0.80	46	28	0.60
3	30	0.93	26	27	0.62	47	32	0.62
4	31	0.71	27	30	0.66	48	36	0.67
5	33	0.78	28	33	0.85	49	37	0.64
6	34	1.04	29	35	0.59	50	43	0.43
7	35	0.87	30	38	0.62	51	46	0.56
8	44	0.81	31	40	0.64	52	55	0.42
9	49	0.79	32	40	0.53	53	55	0.38
10	53	0.64	33	41	0.66	54	58	0.44
11	53	0.65	34	42	0.69	55	64	0.44
12	56	0.64	35	43	0.57	56	67	0.34
13	59	0.60	36	43	0.57	57	68	0.39
14	63	0.65	37	47	0.47	58	80	0.35
15	65	0.80	38	50	0.52	59	81	0.25
16	65	0.62	39	56	0.57			
17	66	0.59	40	56	0.53			
18	70	0.70	41	57	0.65			
19	71	0.48	42	67	0.50			
20	71	0.50	43	72	0.45			
21	78	0.49	44	72	0.42			
22	80	0.50						
23	80	0.43						

TABLE II.—Segmental Spread and Latency of Epidural Analgesia in 53 Arteriosclerotic Patients (2% L67, Mepivacaine, and Lignocaine)

Supine								Sitting							
Case No.	Drugs	Age (yrs.)	Dose (ml.)	No. of Analgesic Segments	Dose/Segments	Latency (mins.)		Case No.	Drugs	Age (yrs.)	Dose (ml.)	No. of Analgesic Segments	Dose/Segments	Latency (mins.)	
						Onset	Complete							Onset	Complete
1	L67	66	10.5	15.5	0.68	10	23	28	M	71	8.5	10	0.85	10	25
2	M	70	17	21	0.81	11	23	29	L	72	7	12	0.58	7	15
3	M	55	10	14	0.72	—	12	30	L	54	14	16.5	0.85	5	20
4	M	58	14	19	0.74	10	25	31	L	72	14	18	0.78	—	—
5	M	64	10.5	18	0.58	5	33	32	L	67	10	15	0.75	6	21
6	M	66	12.5	19.5	0.64	8	30	33	L	68	12	13	0.92	5	18
7	L	69	11	19	0.58	7	29	34	L	49	10	13	0.78	8	20
2b	L	70	17	23	0.74	10	24	35	L	72	17	19.5	0.87	6	19
5b	L	64	11	20	0.55	3	19	36	L	58	8	11.5	0.70	7	19
8	L	72	8.5	17	0.50	11	30	37	L	64	8.5	15	0.57	7	22
9	L	72	8	16.5	0.49	9	14	38	L	70	7	16.5	0.42	3	13
10	L	72	8	17	0.47	—	—	39	L	82	7	15.5	0.45	3	12
11	L	49	12.5	17	0.74	7	27	40	L	81	7	17	0.41	4	19
12	L	72	8	19	0.42	4	19	41	L	74	6	18	0.34	8	18
13	L	59	11	19	0.58	10	32	42	L	79	7	16	0.44	6	24
14	L	71	11	18	0.61	6	15	43	L	75	6.5	12	0.54	6	20
15	L	75	6	17	0.37	7	14	44	L	74	9	16.5	0.55	8	18
16	L	76	7	14	0.50	6	14	45	L	58	8	13.5	0.60	3	18
17	L	59	8	14	0.57	—	17	46	L	71	7.5	20.5	0.37	6	20
18	L	66	6	14.5	0.41	8	30	47	L	76	6.25	14.5	0.43	11	23
18b	L	66	3	8	0.38	7	25	48	L	76	8	16.5	0.49	6	18
19	L	64	12	20.5	0.59	8	23	49	L	80	5.1	17	0.30	6	16
20	L	77	5.5	15.5	0.36	6	20	50	L	65	8	15.5	0.52	5	17
21	L	52	10	14.5	0.69	10	22	51	L	66	8	12.5	0.64	16	16
22	L	66	5.5	12.5	0.44	8	17	52	L	64	12	15.5	0.77	7	26
23	L	44	17	21	0.82	6	18	53	L	69	7.5	17	0.44	6	18
24	L	65	10	16	0.63	7	23								
25	L	46	10	13	0.77	6	22								
26	L	58	15	17	0.86	—	20								
27	L	72	5.25	9	0.58	5	21								

M = Mepivacaine. L = Lignocaine.

sion lines for 3%, 4%, and 5% lignocaine have been drawn from the information in Table I. The results for 2% lignocaine in 26 arteriosclerotic patients are superimposed on this control data. It can be seen that the spread with 2% lignocaine in the diseased patients is as great as it would have been with 2½ to 5% lignocaine in normal subjects.

The increased spread of analgesia in arteriosclerotic patients may also be expressed as a reduction of dosage below the normal regression line in any given instance. The percentage reduction in the present series has been presented as a histogram in Fig. 3, with a mean of -42% below the expected normal dosage for 2% lignocaine.

Latency of Analgesia

Latency is plotted against age in Fig. 4. The time intervals from injection to initial onset of analgesia and

FIG. 3.—Distribution of epidural dosage in 53 arteriosclerotic patients, expressed as percentage reduction below normal requirements.

to complete segmental spread are shown for 2%, 3%, 4%, and 5% lignocaine in normal subjects, and for 2% lignocaine in arteriosclerotic patients. The time taken for complete spread is longer than that found by some authors (Scott, 1956), but the criterion used in this series was rigidly restricted to the moment when analgesia reached its farthest segmental limits.

TABLE III.—*Latency of Epidural Analgesia with Lignocaine in Patients Over 40*

Concentration of Lignocaine and Induction Position	Initial Onset			Complete Spread		
	No. of Cases	Mean Time (min.)	S.D.	No. of Cases	Mean Time (min.)	S.D.
Normal patients:						
2% supine ..	28	6.07	1.25	28	16.2	2.66
2% sitting ..	26	5.8	1.78	30	16.5	2.92
3% ..	14	5.7	1.56	15	14.7	2.74
4% ..	13	5.5	1.15	13	17.7	3.32
5% ..	10	6.4	0.82	10	18.3	2.71
Arteriosclerotics:						
2% supine ..	22	7.27	2.05	22	21.9	5.35
2% sitting ..	24	6.0	1.77	24	18.6	3.24

Difference between normal and arteriosclerotic latency with 2% lignocaine:
Supine: $t = 4.55$, $P < 0.001$.
Sitting: $t = 2.44$, $0.25 > P > 0.01$.

There is a tendency for complete spread to be slightly faster in the older than in the younger age-groups; however, the correlation of latency with age is not significant in the normal controls ($r = -0.23$) or in the arteriosclerotic group. Table III shows a comparison of mean onset times for lignocaine in normal controls and arteriosclerotic patients of the same age-group. It can be seen that the time required for complete spread of analgesia is significantly longer and less predictable in the arteriosclerotic patients than in the normal subjects.

FIG. 4.—Latency of analgesia with lignocaine in 126 normal and 48 arteriosclerotic patients. x = Interval between injection and first appearance of analgesia. • = Interval between injection and complete segmental spread of analgesia.

Discussion

This investigation has shown that arteriosclerosis imposes a pattern of spread of epidural analgesia which is very similar to that produced by concentrated solutions of local anaesthetics. A similar pattern is also seen in pregnant women at term (Bromage, 1961, 1962). In all three circumstances sensory blockade extends further than expected for normal subjects and commonly used concentrations of analgesic solutions, while, paradoxically, the intensity of motor block is unchanged. In this series the arteriosclerotic patients reacted to 2% lignocaine as if they had received a solution of 3%, 4%, or 5% concentration, or, to put it another way, as if they were 25 to 50 years older than their chronological age. Clearly, gross overdosage is likely to occur in this type of patient if epidural anaesthesia is administered without due allowance being made for the tendency to exaggerated spread.

Normally, when a standardized technique is used, the area of analgesia following epidural blockade can be predicted within reasonably close limits by reference to the association of diminished dosage with increasing age, as shown in Fig. 2. This inverse relationship has been discussed elsewhere (Bromage, 1962). It is assumed to be mainly caused by changes in the tissues of the spinal canal, particularly around the intervertebral foramina, where increasing condensations of areolar tissue gradually occlude the exits from the epidural space. Injected solutions cannot escape easily, and are forced to travel longitudinally within the spinal canal, thus covering a larger area of the contents. At the same time systemic absorption of analgesic drugs is hindered by sclerosis of the extradural vessels, so that more opportunity is left for absorption by nerves.

Changes within the cord and spinal nerves, as well as their coverings, may also contribute to increased spread. With advancing age the myelinated fibres in the dorsal and ventral roots lose their youthful contours on cross-section, and decline in number, so that by the ninth decade one-third of the myelinated nerve population has disappeared, leaving a smaller number of survivors to compete for any analgesic drugs which may be presented to them (Hodge, 1894; Semenowa-Tjan-Schanskaja, 1941; Rexed, 1944). Indeed, the population curve for myelinated fibres in dorsal roots is very similar in shape to the curve relating epidural dosage and age, rising to a peak at about 20 years and declining steadily thereafter (Corbin and Gardner, 1937). This is accompanied by a steady slowing of conduction in peripheral nerves after the age of 35, amounting to about 0.4 metre per second per year (Downie and Newell, 1961). In addition to these changes in nerve tissue, deterioration in the ground substance of ageing connective tissue weakens the barrier to diffusion of local anaesthetics across nerve integuments into the fibres and interfibre spaces beneath (Ham and Leeson, 1961). Thus the enforced intimacy of injected solutions with the contents of the spinal canal, combined with progressively weakened defences of the spinal nerves and a declining neuronal population, together conspire to bring about increased spread with advancing age.

The exact site of action of epidural blockade is still unproved. In the past, opinions have been divided whether blockade took place in the paravertebral spaces (Foldes *et al.*, 1956), in the subarachnoid space after diffusion across the dura (Rudin *et al.*, 1951; Frumin *et al.*, 1953), or at the dorsal roots (Mocquot, 1924;

Bromage, 1954). Circumstantial evidence is mounting that blockade occurs somewhere within the confines of the spinal canal, possibly by diffusion into the subperi-neural spaces, and then retrogradely along the perivascular spaces of the vasa nervorum into the spinal cord, producing a neuraxial type of spread (Bromage, 1962). The demonstration that small amounts of concentrated analgesic solutions produce widespread blockade without increased intensity or speed of onset is strongly suggestive that diffusion gradients are equally as important as volume, if not more important, in determining transport to distant neuronal tissue. From a small concentrated dose the analgesic is spread thinly over a wide area, and within the concentration range between 2 and 5% the same number of milligrams of lignocaine block approximately the same number of segments at any given age.

In the past there has been some controversy over the effectiveness of connective-tissue sheaths as diffusion barriers to nerves (Lorente de N, 1950), but the evidence now seems conclusive that these structures do play an extremely important part in isolating nervous tissue from the surrounding environment (Feng and Liu, 1949; Crescielli, 1951; Causey and Palmer, 1953; Lehmann, 1953; Krnjevi, 1954a, 1954b, 1955). With advancing age these barriers become weakened qualitatively by the general deterioration in the mucopolysaccharides of ground substance, allowing diffusible substances such as local analgesic solutions to penetrate connective tissue more readily (Meyer and Rapport, 1951; Kirk and Laursen, 1955; Ham and Leeson, 1961). However, at the same time a quantitative hypertrophy of connective tissue around nerve fibres and blood vessels tends to compensate for deteriorating quality.

Degenerative changes in connective tissue are therefore likely to produce two results, both of which tend to increase the spreading effects of epidural solutions: firstly, heightened permeability of nerve integuments (partly offset by hypertrophy); and, secondly, impeded escape from the thickened extradural blood-vessels. Both of these factors will be exaggerated in arteriolar disease, where connective-tissue degeneration is accelerated by diminished blood flow and capillary loss (Jackson, 1958; Dyrbye, 1959; Gough, 1962). Furthermore, sclerosis of the vasa nervorum impairs nutrition of the nerve fibres themselves, and hastens degeneration of their myelin sheaths (Fagerberg, 1957, 1959), so that the overall effect of arteriosclerosis on the nerves and their surroundings is a caricature of the normal ageing processes. Since epidural spread is primarily dependent on the end-result of these processes rather than on the age which normally determines them, it is not surprising that segmental spread should follow the biological rather than the chronological age of the patient.

The slow spread of analgesia observed in some of the arteriosclerotic patients is more difficult to explain, although here again the play of qualitative and quantitative changes in connective-tissue barriers and blood-supply within and about the neuraxis is probably a determining factor. Since spread tends to be slow in these cases, its protracted latency will be most dramatically apparent when the upper limit of analgesia marches over a long distance; in other words, when a gross overdose of local anaesthetic provides a reservoir of solute which is large enough to diffuse through the neuraxis as far as the medulla and brain stem. Then cranial nerve involvement, with unconsciousness and apnoea, may supervene as a seemingly unconnected

event 40 minutes or even longer after epidural injection of what might have seemed a not too excessive dose in a patient of normal biological age.

Summary

Injection of local anaesthetic solutions into the spinal extradural space results in a segmental type of analgesia. In normal subjects the number of analgesic dermatomes can be predicted with an accuracy of 15 to 30% in terms of age and the concentration and volume of local anaesthetic used. The amount of local anaesthetic required to block a given area declines steadily after the second decade, in a manner reminiscent of some other curves relating biological phenomena to age.

Fifty-three patients with occlusive vascular disease reacted abnormally to epidural analgesia. They behaved as if they were 25 to 50 years older than their chronological age, or as if the injected solution had been twice the concentration actually used. In these patients segmental analgesia spread one and a half to three times further than in normal subjects of the same age. This tendency to increased spread can be hazardous if the anaesthetist is not aware of the necessity for reduced dosage in arteriosclerotic patients.

The time taken for epidural analgesia to reach its farthest limits varies quite widely in normal subjects, and does not appear to be dependent on age. This time interval is prolonged in arteriosclerotic patients.

The significance of these findings is discussed in relation to the effects of ageing processes, and to the site of action of epidural analgesia.

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REFERENCES

- Bromage, P. R. (1954). *Spinal Epidural Analgesia*, p. 14. Livingstone, Edinburgh.
- (1961). *Canad. med. Ass. J.*, **85**, 1136.
- (1962). *Brit. J. Anaesth.*, **34**, 161.
- Causey, G., and Palmer, E. (1953). *J. Anat. (Lond.)*, **87**, 30.
- Corbin, K. B., and Gardner, E. D. (1937). *Anat. Rec.*, **68**, 63.
- Crescitelli, F. (1951). *Amer. J. Physiol.*, **166**, 229.
- de Saram, M. (1956). *Anaesthesia*, **11**, 77.
- Downie, A. W., and Newell, D. J. (1961). *Neurology (Minneapolis)*, **11**, 876.
- Dyrbye, M. O. (1959). *J. Geront.*, **14**, 32.
- Fagerberg, S. E. (1957). *Acta med. scand.*, **159**, 59.
- (1959). *Ibid.*, Suppl. 345, p. 1.
- Feng, T. P., and Liu, Y. M. (1949). *J. cell. comp. Physiol.*, **34**, 1.
- Foldes, F. F., Colavincenzo, J. W., and Birch, J. H. (1956). *Anesth. Analg. Curr. Res.*, **35**, 33.
- Frumin, M. J., Schwartz, H., Burns, J. J., Brodie, B. B., and Papper, E. M. (1953). *J. Pharmacol. exp. Ther.*, **100**, 102.
- Gordon-Jones, R. G. (1953). *Anaesthesia*, **8**, 242.
- Gough, K. R. (1962). *Brit. med. J.*, **1**, 21.
- Ham, A. W., and Leeson, T. S. (1961). *Histology*, 4th ed. Lippincott.
- Hodge, C. F. (1894). *J. Physiol. (Lond.)*, **17**, 129.
- Jackson, W. B. (1958). *Amer. Heart J.*, **56**, 222.
- Kirk, J. E., and Laursen, T. J. S. (1955). *J. Geront.*, **10**, 288.
- Krnjević, K. (1954a). *Quart. J. exp. Physiol.*, **39**, 55.
- (1954b). *J. Physiol.*, **123**, 338.
- (1955). *Ibid.*, **128**, 473.
- Lehmann, H. J. (1953). *Nature (Lond.)*, **172**, 1045.
- Lorente de Nó, R. (1950). *J. cell. comp. Physiol.*, **35**, 195.
- Meyer, K., and Rapport, M. M. (1951). *Science*, **113**, 596.
- Mocquot, P. (1924). *Rev. Chir. (Paris)*, **63**, 120.
- Morrow, W. F. K. (1959). *Brit. J. Anaesth.*, **31**, 359.
- Mostert, J. W. (1960). *Ibid.*, **32**, 613.
- Rexed, B. (1944). *Acta psychiat. (Kbh.)*, Suppl. 33, p. 1.
- Rudin, D. O., Fremont-Smith, K., and Beecher, H. K. (1951). *J. appl. Physiol.*, **3**, 388.
- Scott, D. B. (1956). *Brit. J. Anaesth.*, **28**, 187.
- Semenowa-Tjan-Schanskaja, W. (1941). *Z. ges. Neurol. Psychiat.*, **172**, 587.
- Sicard, J. A., and Forrestier, J. (1921). *Rev. neurol.*, **28**, 1264.
- Stringer, R. M. (1954). *Anesth. Analg. Curr. Res.*, **33**, 195.
- Sykes, M. K. (1958). *Anaesthesia*, **13**, 78.

A SYNDROME DUE TO TRANSIENT OR CHANGING HEART-BLOCK

BY

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It is well recognized that angina pectoris can occur with the paroxysmal tachycardias. Pain can also be a direct result of heart-block, the pain and the block starting and ending at the same time.

Four cases of transient or changing heart-block are reported. Two of the patients had normal cardiograms at rest and had severe pain only when exertion was associated with a 2:1 heart-block. The pain was described as bursting, relieved by a varying period of rest but not by trinitroglycerin, and radiating to the neck and down the arms. The pain was associated with the sudden onset of severe dyspnoea, palpitations, a feeling of marked faintness, weakness of the limbs, and alarm. In the other two cases symptoms were associated with a change from 2:1 to a complete heart-block.

Froment *et al.* (1959) report three cases of anginal pain associated with heart-block but without evidence of coronary artery disease. They believe that myocardial anoxia results from the decrease in coronary blood flow with the abrupt slowing of the heart rate. This is discussed later.

Exercise has an important effect on the ventricular rate in both complete and partial heart-block. It was once thought that the bradycardia of complete heart-block was quite unaltered during exercise (Vaquez, 1924), but Gilchrist (1934) showed that the idioventricular centre and the sino-auricular node react qualitatively in the same way to different stimuli. Partial atrioventricular block is also affected by exercise, and in fact two types can be distinguished by their response to exercise. In the benign type 1 partial heart-block of the Wenckebach variety, with progressive lengthening of the P-R interval, exercise eliminates the block; whereas in the more serious type 2 partial atrioventricular block, with the fixed P-R interval, exercise may change a 2:1 to a 3:1 or to a complete heart-block (see Cases 3 and 4). Exercise may also bring to light a type 2 block when under resting conditions a cardiogram is normal (see Cases 1 and 2).

Thomson (1934) found only one case of type 2 partial block among 5,000 patients electrocardiographed. By exercise tests and other methods of auricular acceleration, such as the use of atropine and amyl nitrite, Gilchrist (1958) has shown that type 2 partial block is not rare. He found 24 examples of type 2 atrioventricular block among the 140 patients with high-grade heart-block that he has studied clinically. In that authoritative article, only one passing reference to angina is made: "fatigue, exertional dyspnoea, mild to moderate in degree, and attacks of effort angina may prove more of a handicap than the liability to syncope."

In the cases reported here a characteristic symptom-complex appears to result from the onset of heart-block or change in the degree of block which is usually provoked by exercise. If exercise tests were done more commonly in patients with symptoms described here, and in whom a resting cardiogram showed no heart-block, more cases of type 2 partial block and this clinical syndrome might well be recognized.