PAINFUL DISABILITY OF THE SHOULDER AND CARDIOVASCULAR DISEASE

(From the Rheumatism Unit at St. Stephen's Hospital, London)

By PHILIP ELLMAN, M.D., F.R.C.P.

Physician to the Rheumatism Unit at St. Stephen's Hospital, London
and

A. SCHOTT, M.D., M.R.C.S.

Medical Officer in Charge of the Cardiographic Department, Queen Mary's Hospital for the East End, London

Attention has already been drawn by one of us to the not infrequent occurrence in patients with coronary disease of a painful disability of the shoulder resembling peri-arthritis (Schott, 1947). It was pointed out at the time that this association seemed to have been noticed chiefly by cardiologists and that, since both coronary disease and peri-arthritis of the shoulder are common in patients over 40, investigations of the cardiac condition of a larger number of patients with this kind of shoulder lesion are required to throw light on the frequency and nature of this association. Such investigations have recently been carried out at the Rheumatism Unit at St. Stephen's Hospital in the Department of one of us (P.E.) and their results form the subject of this communication.

Method

Thirty-one consecutive patients over the age of 40 presenting themselves with a painful condition of the shoulder, the nature of which justified a provisional diagnosis of peri-arthritis on routine examination in the out-patient department were referred for a more detailed examination especially of the shoulder and cardiovascular system. This included, in addition to full clinical examination, X-ray films of chest and shoulders and electrocardiograms with at least the two chest leads IVR and CR₃, and additional chest leads where required. This selection was confined to patients in whom there was no evidence of a traumatic origin of the shoulder condition.

The diagnosis of peri-arthritis, the unsatisfactory nature of which is admitted, was based on a history of pain in the shoulder radiating down the arm and often worse at night, tenderness over the deltoid, the greater tuberosity of the humerus, and often the anterior aspect of the shoulder joint, limitation of movement in the affected joint, chiefly of abduction and external and internal rotation, and pronounced alteration of scapulo-humeral

rhythm (Codman), the scapula moving continuously on abduction of the arm at a much earlier stage than normal. The presence and degree of rest pain and tenderness varied considerably in different cases according to the stage at which the patients were seen. In the subacute and chronic cases, at which rest pain was mild or absent, limitation of movement was the outstanding sign. The movements most readily affected were those involved in brushing the hair or putting on a coat. In the more chronic cases wasting of the deltoid and spinati was noted. The symptoms and signs were sufficiently pronounced for the diagnosis to be made with a fair degree of certainty in all cases except one (No. 17), and are not mentioned in the table; reasons for the inclusion of this particular case are given below.

No conclusions can be drawn from our series as to the incidence of calcified deposits, since special radiological examinations for this purpose (Bosworth, 1941, Baird, 1944) were not undertaken. For the purpose of this investigation this aspect is irrelevant.

Cases of arthritis of the gleno-humeral joint were rejected. Of the 31 patients 11 were excluded on re-examination, since the provisional diagnosis of peri-arthritis could not be upheld, so that this study is based on observations on 20 cases.

Results

The main results are summarized in table 1. There were 9 males and 11 females, the ages varying between 40 and 68. The left shoulder was affected in 11 and the right in 7 cases; in the remaining 2 the condition was bilateral. The onset of the painful shoulder disability was described by the patients as gradual, except for 2 cases (Nos. 16 and 19). In the first of these, shoulder pain started suddenly 16 days after the patient had carried a heavy suitcase; during that interval she had had no discomfort. While the

possibility of a traumatic origin can not be excluded with certainty in this case, the interval is too long for attributing the shoulder lesion to trauma only. In the second case there was a history of sudden onset of a painful disability of the right shoulder following severe exposure to weather; this was followed five years later by a similar affection of the left shoulder with gradual onset.

Three points stand out as being of special importance. The first is that no fewer than 17 out of the 20 cases were found to be suffering from some systemic disorder. Of these 9 had established cardiovascular disease, viz. hyperpiesia 4, arteriosclerosis with intermittent claudication 1, myocardial infarction 1, congestive heart failure with auricular fibrillation 1, and electrocardiographic signs of myocardial damage 2. In one of these cases (fig. 1) there were signs of pronounced disturbances of intraventricular conduction, the QRS complexes being grossly notched or slurred and widened to 0.16 sec., i.e. double the average normal. There was also slight enlargement of the shadow of the heart and the abnormal cardiac condition was presumably a remnant of an inflammatory or toxic condition of the myocardium which had been associated with one or several of the infections the patient had had in 1945. In the other case (fig. 2) abnormal electrocardiographic signs were mainly confined to the final deflections in the two chest leads, CR₃ and CR₁; there was also depression of the S-T junction 1 mm. below the iso-electric line, and slight distortion of the S-T intervals in lead 3. The presence in this case of some myocardial lesion due to coronary sclerosis was made more probable by the patient's age and by a history of dyspnoea on exertion of six months' duration, which could not be adequately explained by the pulmonary or any other findings. Two other patients were suffering from asthma and chronic bronchitis, and in yet another the painful disability of the shoulder had closely followed an attack of herpes zoster on the same side in the distribution of D.6. Of those classified as doubtful three had mild hyperpiesia and one showed slight abnormalities in the final deflections of leads 2 and 3, possibly due to vitamin B₁ deficiency resulting from alcoholism (Schott, 1944). The one remaining case of this group is the only one which was included although movements in the affected shoulder were neither painful nor restricted. Inclusion of this case, in which pain in the left chest, shoulder and arm had followed an attack of atypical pneumonia, seemed justified in view of the tenderness in the left bicipital groove and over the coracoid process, and of pain in the bicipital groove on maximum flexion of the elbow, since nontraumatic tenosynovitis of the long head of the biceps is most

frequently due to a lesion of the supraspinatus tendon, itself the commonest cause of, and intimately related to, periarthritis. The patient was also suffering from angina of effort and hyperpiesia; the association of continuous dull pain in the left arm with both coronary disease and periarthritis has been reported (Spillane and White, 1940) and may be present in the same patient (Schott, 1947).

Another important finding is the high incidence of vascular disease. No fewer than seven showed evidence of advanced vascular changes, viz. pronounced hyperpiesia 5, intermittent claudication 1, myocardial infarction 1. To these should be added three cases of mild hyperpiesia, making 10 out of 20.

Lastly, in only two out of these ten cases did anginal pain occur. This low incidence stands in marked contrast to the findings reported in the papers which deal with this problem from the cardiological angle.

Discussion

It is now accepted that in periarthritis the primary site of the lesion is the conjoined tendon of the short rotators of the shoulder joint (supra- and infraspinatus, teres minor and subscapularis) termed musculotendinous cuff by Codman. Tarsy subdivided such cases into primary periarthritis, i.e. of unknown origin, and secondary, i.e. of known, usually traumatic, origin. If this classification is adopted our series will consist of cases of primary periarthritis.

Perusal of the papers dealing with this subject from the cardiological angle (Howard, 1930, Edeiken and Wolferth 1936; Boas and Levy, 1937; Leech, 1938; Ernstone and Kinell, 1940; Spillane and White, 1940; Askey, 1941) shows that the connecting link between coronary disease and a painful disability of the shoulder, periarthritic in type, was sought in the presence of pain. While valid reasons exist for assuming such a possible connection, such as a tendency of the ipsilateral shoulder to be affected on the side of radiation of anginal pain, or radiation of anginal pain into areas which are the site of another pathological process (Boas and Levy, 1937; Cohen, 1944), in the present series, in which it was the shoulder lesion and not cardiac pain or any other cardiac symptom which made the patient seek medical advice, the incidence of anginal pain was very low while that of vascular disease was comparatively high. For an understanding of a possible relationship between painless vascular disease and periarthritis of the shoulder it will be helpful to consider some recent work on the pathology of periarthritis and allied conditions of the shoulder.

Of the various tendons which jointly form the

FIG. 1.—(Case No. 7). Intraventricular disturbances of conduction. Note also the variation in shape of the Q.R.S. complexes in lead 3, which is due to alterations of intraventricular conduction. The isolated upstrokes (one each in leads 1, 2 and 3, and two each in leads CR2 and CR1 respectively) are calibration marks. Time : 0.04 and 0.2 sec.

FIG. 2.—(Case No. 8). Depression of 1mm. below the isoelectric line of the S-T junction and slight distortion of the S-T intervals in lead 3; abnormal final deflections in leads CR₃ and CR₁. Time: 0.04 and 0.2 sec.

musculotendinous cuff, that of the supraspinatus is generally considered to be most frequently the site of pathological changes. Codman and Akerson (1931) found evidence of rupture of the supraspinatus tendon in 39 out of 100 shoulders examined post-mortem in 52 subjects aged from 46 to over 80. Wilson and Duff (1943) encountered complete rupture of this tendon in 22.2 per cent. of 108 unselected necropsies of persons over 30 (average age: 65) and partial rupture in 20 per cent. of 74 necropsies (average age: 62). These findings combined show that in 225 unselected necropsies 30 to 40 per cent. had demonstrable

lesions after the age of 30 and that in one half of these the lesions were large (Jones, 1944). Wilson and Duff also found degenerative lesions in the supraspinatus tendon, as evidenced by changes in the staining qualities of the connective tissue, particularly with Mallory's stain. The vascularity of the supraspinatus tendon seemed normally to decrease with age to such an extent that 'after the age of 40 arterioles are seldom seen in normal tendons.' Such changes were considered to be the weakening factor which predisposes to rupture; thus the repeated small daily traumata, mainly caused by impingement and friction on the acromion process, which are easily withstood by the normal tendon of the younger individual, could result in a rupture of the tendon without the patient's being aware of a trauma in the sense accepted by the layman.

The reason why the tendon of the supraspinatus is especially prone to rupture has been made clearer by Jones (1944). He pointed out that, while degenerative changes occur in connective tissue generally, 'they are present in aggravated form at the central portion of the supraspinatus tendon just above its point of final attachment to the horizontal line of the greater tuberosity of the humerus.' At this point a pull in four different directions is exerted, which not only tends to weaken this particular area, but also, once a partial rupture has occurred, tends to keep the edges of the rent apart: an upward pull of the supraspinatus, a downward pull of the weight of the arm, a pull directed medially by the subscapularis, and one laterally by the infraspinatus portions of the musculotendinous cuff. Moreover, phylogenetically the function of the supraspinatus has undergone considerable changes with the assumption of the erect posture and its mass, relative to that of the total scapulo-humeral muscles, has progressively decreased from 26 per cent. in the opossum to 13 per cent. in the macacus and 7 to 8 per cent. in man, whereas the corresponding changes of the relative mass of the other short rotator muscles were, by comparison, small (Inman, Saunders and Abbott, 1944). Thus, the focal point of the divergent pull of the various forces, mentioned above, is situated in the tendon of a muscle which has undergone the greatest degree of phylogenetic retrogression.

The relationship between vascular disease, painless or not, and lesions in the supraspinatus tendon can thus be understood. It is obvious that vascular disease will be apt to produce changes, resulting in clinical disease, especially at places which are normally subjected to special mechanical stress and in which, in addition to and perhaps because of this reason, normally degenerative changes are particularly pronounced.

TABLE I
CASES WITH ESTABLISHED SYSTEMIC DISEASE

No.	Name, Sex, Age	Duration of Shoulder Disability	Side	Clinical Features	Ang. Pain	Remarks
1.	M.C. M. 63	1 year	both	Hyperpiesia : 215/135, intermittent claudication. E.C.G. marked left ventricular strain. Remnants of cortical thrombosis sensory area.	No	Dupuytren's contracture right hand.
2.	D.C.J. F. 50	3 weeks	left	Hyperpiesia : 170-160/100-95. Systolic murmur with maximum in left second intercostal space. Dyspnoea on exertion. Pain in knees last three years. Osteoarthritis right thumb, 1946. X-ray : Moderate enlargement of heart. X-ray of shoulder : Normal.	No	
3.	S.O'S. F. 68	2 months	left	Obesity, congestive heart failure, auricular fibrillation. X-ray of chest : Considerable enlargement of heart to left, aortic configuration, calcification of aortic knuckle. X-ray of shoulder : Osteoarthritis acromio-clavicular joint. Osteoarthritis of both knees due to marked valgus position of feet. Sudden onset of shoulder condition.	No	
4.	L.A. F. 58	6 months	left	Obesity (16 st. 6 lb.). Hyperpiesia : 240/120. X-ray of chest and shoulder : Normal. E.C.G. moderate left ventricular strain.	No	
5.	R.B. M. 60	3 years	right	Cough since gassed in 1917. Coal miner for ten years, subsequently plasterer (cement and sand). Intermittent claudication due to obstructive vascular disease, oscillometric index almost nil both calves. X-ray of chest : Marked increase of normal linear striation of both lungs, enlarged hilar shadows, heart normal size, rounding of the contour of the left ventricle. X-ray of shoulder : Irregular outline of greater tuberosity.	No	
6.	R.W.M. M. 57	2½ months	left	Trauma to right shoulder, 1931. Myocardial infarction, February, 1946. X-ray of chest : heart enlarged, mainly left ventricle, aorta dilated. X-ray of shoulder : N.A.D. E.C.G. : myocardial damage subsequent to myocardial infarction.	Both r>1	Slight swelling of fingers, left hand (see Askey, 1941).
7.	G.H. F. 56	2 years	right	Dysentery, malaria and hepatitis in the Far East, 1945. Marked psychological element. X-ray of chest : Slight enlargement of transverse diameter of heart. Shoulder : Slight decalcification in head of humerus. E.C.G. : Disturbances of intraventricular conduction.	No	See Fig. 1.
8.	H.C.C. M. 63	2 months	right	Tonsillitis and quinsy 2-3 times a year up to age of 15. Pleurisy, 1935, hematuria, 1944. Dyspnoea on exertion last six months. Left hand amputated, 1902 (accident). X-ray of chest : moderate emphysema. B.P. : 170/80. Bicipital groove tender. E.C.G. : Abnormal final deflections in CR ₃ and CR ₁ . X-ray of shoulder : Irregularity of the upper end of the greater tuberosity. Osteoarthritis of acromio-clavicular joint. Subdeltoid bursitis.	No	See Fig. 2.

No.	Name, Sex, Age	Duration of Shoulder Disability	Side	Clinical Features	Ang. Pain	Remarks
9.	McD. F. 64	4 months	right	Family history of vascular disease. Hypertension: 190/105. X-ray of chest: Moderate enlargement of heart to left, with rounding of contour of left ventricle. Shoulder: Slight irregularity of the right greater tuberosity. E.C.G.: Slight distortion of S-T intervals in lead 2.	No	Similar attack, more severe in left shoulder, 1944. Dupuytren's contracture both hands for about 20 years.
10.	H.C.H. M. 50	2 months	left	Subject to 'influenza' with bronchitis about once every two years since 1931. Short of breath and wheezing since 1931. Underweight. Poor chest expansion. Emphysema with chronic bronchitis. B.P.: 120/70. X-ray of chest: Old infiltration and fibrosis right apex, pleural involvement of right lower zone, heart displaced to right. Shoulders: N.A.D.	No	
11.	E.S. F. 57	1 month	right	Sudden onset of shoulder pain. Had been carrying a heavy suitcase 16 days prior to onset of shoulder pain. Asthma since menarche, aged 14, family history of asthma. Dyspnoea on exertion for ten years. X-ray of chest: Emphysema. Heart: N.A.D.	No	
12.	W.P. M. 66	2½ months	left	Herpes zoster C.6 left preceded onset of pain in left shoulder by about three weeks. Radial arteries greatly thickened, second aortic sound +. B.P.: 160/80. X-ray of chest: Long asthenic chest with uncoiled sclerotic aorta. E.C.G.: Unusually high T waves in chest leads. X-ray of shoulder: 'Degenerative joint disease.'	No	

DOUBTFUL CASES

13.	L.W. F. 58	10 months	both	Numerous sore throats up to age of 17 when tonsils were removed. Slight accentuation of second sounds over base. B.P.: First reading at hospital. O.P.: 190/110, subsequently 165/85-160/80. X-ray of chest: Old fibrotic changes right apex, numerous large calcified shadows both upper zones. Shoulders: Osteoporosis of bones about both shoulder joints, l>r. Heart shadow normal.	No	
14.	T.H. F. 57	3 months	left	Frequent tonsillitis up to age of 40. ? rheumatic fever. Menopause, 1944, with flushes ++. Anxiety state. Moderate degree of obesity. B.P.: 150/100-105. X-ray of chest: Heart transversely placed within limits of normal. X-ray of shoulder: N.A.D.	No	
15.	R.A. F. 48	9 weeks	left	Family history of vascular disease, both parents died of cerebral haemorrhage. B.P.: 170/90-95. X-ray of chest and shoulder: N.A.D. E.C.G.: Left axis deviation.	No	
16.	J.W. M. 66	2 months	left	Retired brewer. History of trauma to left shoulder over two years ago, which cleared up in a few weeks. Symptom-free until two months ago. At least over three pints of beer for the last 50 years. E.C.G.: Slight abnormalities in leads 2 and 3 (? due to Vitamin B ₁ deficiency). X-ray of shoulder: N.A.D.	No	

DOUBTFUL CASES—(continued).

No.	Name Sex, Age	Dura- tion of Shoulder Disability	Side	Clinical Features	Ang. Pain	Remarks
17.	J.C. M. 54	4 months	left	Atypical pneumonia July, 1947. Pain in left chest radiating to left arm and shoulder started during convalescence in August, 1947. Continuous pain in left arm, on and off, aggravated in the evenings, above and medially to elbow and on volar aspect of wrists. Tenderness in left bicipital groove, coracoid, spinous process of D.5 and pain in bicipital groove on maximum flexion of elbow. No limitation of movement of shoulder. X-ray of chest and left shoulder: N.A.D. B.P. 225/115.	left chest	

CASES IN WHICH NO SYSTEMIC DISEASE WAS FOUND

	F.T. F. 48	6 months	right	Short of breath on walking upstairs. Apical systolic murmur (functional). X-ray of chest: N.A.D. Shoulder: Slight irregularity of greater tuberosity both sides.	No	
19.	F.P. M. 55	3 years	left	Pain and limitation of movement in left shoulder, started 1944, manipulated under anaesthetic at St. Stephen's Hospital, April, 1947, movements improved. Residual limitation of movement and soreness of left shoulder. On flexing left elbow pain down left arm to elbow. Some residual limitation of movement in right shoulder where he had pain of sudden onset in 1942 following severe exposure to weather in Navy. Manipulated under anaesthetic in 1942. X-ray of chest: N.A.D. Shoulder: Small calcification in subscapularis tendon. Heart: N.A.D.	No	Similar attack right shoulder 1942.
20.	J.S. F. 40	6 months	right	Tonsils very scarred with crypts, small submandibular glands palpable. X-ray of chest and shoulders: N.A.D.	No	

This view accords well with McLaughlin's investigations (1944, 1946) on the pathology of subdeltoid bursitis with calcific deposits in the shoulder. As the earliest change he found hyaline degeneration in the collagen of the tendon fibres of the short rotators, which are later detached as foreign bodies from their attachment to the tendon and form rice-like fragments which may calcify. Such foreign bodies set up a peritendinous inflammation and, since in the short rotators the tendon sheath is represented by the floor of the subdeltoid bursa, the result is subdeltoid bursitis. As long as the calcified deposits are situated in the tendons the condition remains asymptomatic; this explains the observation that calcified deposits may be found in one or both shoulders with little or no symptoms for months or years prior to the attack of pain (Bosworth, 1941). It is the reactive inflammation of the bursa to the degenerative changes in

the tendons of the short rotators which causes the symptoms. In this way it can also be understood that there is no correlation between the presence or extent of calcific deposits and the presence or severity of periarthritis (see also Howorth, 1945; Wilson, 1943).

The only paper in which, as far as we are aware, mention was made of a high incidence of hypertension and arteriosclerotic disease in patients with pain and disability of the shoulder and arm is that of Travell, Rinzler and Herman (1942). In their series of 58 cases the incidence was about 40 per cent.; about 20 per cent. had pulmonary tuberculosis and 40 per cent. other general medical conditions. Although the authors consider the painful disability of the shoulder in their cases to be distinct from periarthritis and attributable to painful foci in certain muscles, ('idiopathic myalgia'), the clinical features resemble in so

many respects those of the condition commonly called peri-arthritis that a sharp distinction between these two conditions does not seem convincing.

Two of our patients suffering from hyperpiesia had Dupuytren's contracture. In one, in whom the shoulder lesion was bilateral, the right hand was affected, whereas in the other the shoulder lesion was on the right and both hands showed the contracture. While the connection between Dupuytren's contracture and both peri-arthritis and vascular disease is obscure, it may be mentioned that Dupuytren's contracture has been described as a sequel to myocardial infarction in six cases by Kehl (1943). The occurrence of a painful disability of the shoulder following upon an attack of herpes zoster in the distribution of D.6 of the same side is interesting, since a relationship has been described between anginal pain and both herpes zoster in the distribution of the same or neighbouring segments (Spillane and White, 1939) and peri-arthritis of the shoulder.

Summary

In view of reports in the literature and personal observations of the occurrence in patients over 40 with coronary disease, of a painful disability of the shoulder resembling peri-arthritis, 20 consecutive patients over 40 with peri-arthritis of one or both shoulders were examined for general systemic and particularly for cardiovascular disease. Of these, 17 were found to be suffering from some systemic disorder, viz. established cardiovascular disease 9, asthma and chronic bronchitis 2, recent attack of herpes zoster 1, mild hyperpiesia 3, electrocardiographic abnormalities possibly due to vitamin B₁ deficiency 1 and marked hyperpiesia with angina of effort in 1 patient with bicipital tenosynovitis, but without typical peri-arthritis. While the incidence of anginal pain was low (two cases), that of vascular disease was high (ten cases). It is pointed out that, while the presence of pain has so far been considered to be the sole connecting

factor between coronary disease and peri-arthritis of the shoulder, this cannot hold good for the present observations. Recent work on the pathology of peri-arthritis is reviewed which shows that the primary lesion giving rise to peri-arthritis is degenerative in nature, and reasons are given for the assumption that, irrespective of pain, the vascular lesions are the essential factor and connecting link, since they are bound to precipitate and accelerate in the musculo-tendinous cuff of the shoulder the degenerative lesions which are a common normal feature in subjects over 40.

ACKNOWLEDGMENT

We are indebted to the Medical Registrar of the Rheumatism Unit, Dr. R. E. Ball, for his valuable help and co-operation in this work.

BIBLIOGRAPHY

- ASKEY, J. M. (1941), *Amer. Heart J.*, **22**, 1.
 BAIRD, L. W. (1944), *Radiology*, **37**, 316.
 BOAS, E. P., and LEVY, H. (1937), *Amer. Heart J.*, **14**, 540.
 BOSWORTH, B. M. B. (1941), *J.A.M.A.*, **116**, 2477.
 CODMAN, E. A. (1934), 'The Shoulder,' Boston.
 CODMAN, E. A., and AKERSON, J. B. (1931), *Ann. Surg.*, **93**, 348.
 COHEN (1944-6), *Trans. Med. Soc., Lond.*, **64**, 65.
 EDEIKEN, J., and WOLFERTH, C. C. (1936), *Amer. J. Med. Sci.*, **191**, 201.
 ERNSTENE, A. C., and KINELL, J. (1940), *Arch. int. Med.*, **66**, 800.
 HOWARD, T. (1930), *Med. J. Rec.*, **131**, 364.
 HOWORTH, M. B. (1945), *Surg. Gynaec. Obst.*, **80**, 337.
 INMAN, V. T., SAUNDERS, C. M. and ABBOTT, L. C. (1944), *J. Bone and Joint Surg.*, **26**, 1.
 JONES, L. (1944), *Arch. Surg.*, **49**, 390.
 KEHL, K. C. (1943), *Ann. Int. Med.*, **19**, 213.
 LEECH, C. B. (1938), *R. I. med. J.*, **21**, 104.
 McLAUGHLIN, H. L. (1944), *New York State J. Med.*, **44**, ii, 2231.
 McLAUGHLIN, H. L. (1946), *Ann. Surg.*, **124**, 354.
 SCHOTT, A. (1944), *Brit. Heart J.*, **6**, 27.
 SCHOTT, A. (1947), *Proc. Royal Soc. Med.*, **40**, 733.
 SPILLANE, J. D., and WHITE, P. D. (1939), *Brit. Heart J.*, **1**, 291.
 SPILLANE, J. D., and WHITE, P. D. (1940), *Brit. Heart J.*, **2**, 123.
 TARSY, J. M. (1944), *New York State J. Med.*, **44**, ii, 2109.
 TRAVELL, J., RINZLER, S. and HERMAN, M. (1942), *J.A.M.A.*, **120**, 417.
 WILSON, L. C. (1943), *Arch. Surg.*, **46**, 397.
 WILSON, L. C. and DUFF, G. L. (1943), *Arch. Surg.*, **47**, 121.

H. K. LEWIS & Co. Ltd.

Medical Publishers
and Booksellers

136 GOWER STREET
LONDON, W.C.1

(Adjoining University College and Hospital)

Telephone: EUSton 4282 (5 lines)

Telegrams: Publicavit, Westcent, London

Established 1844

Medical Lending Library

ANNUAL SUBSCRIPTION from ONE GUINEA

For the CONVENIENCE of POST-GRADUATE STUDENTS SHORT
PERIOD SUBSCRIPTIONS ARE ARRANGED — for 3 or 6 months

Detailed Prospectus on application

The Library Catalogue revised to December, 1943, containing
classified index of authors and subjects. To subscribers,
12s. 6d. net; to non-subscribers, 25s. net, postage 9d.

SUPPLEMENT, 1944, TO DECEMBER, 1946

To Subscribers, 2s. 6d. net; To Non-Subscribers, 5s. net, postage 4d.

NEW BOOKS ADDED IMMEDIATELY UPON PUBLICATION