of some local authorities to favour whole-time service, not to mention the proposed extension of panel practice, it is clear that the earnings of the profession will be derived in increasing proportion from salary and in decreasing proportion from fees.

So it should be with the staffing of hospitals. Apart from the professorial units, the larger hospitals will doubtless offer increasing opportunities to men who desire to undertake whole-time hospital service. The London County Council affords a good example of whole-time and part-time men working side by side.

Freedom and Elasticity

Five years will show us the way. We must innovate greatly but quietly. Meanwhile we can pause to consider a whole-time medical civil service on its merits. It would be a more compact and tidy administration, and would probably be run at less cost; but would it have the same value? We have an example of a whole-time service in the R.A.M.C. For war its organization is admirable, but for civil life would not its administration be too rigid, advancement too dependent on seniority, and its day-to-day work without a sharp enough spur of ambition? It is important not to confuse ambition with egoism. Ambition stimulates a man to do his best, and a healthy thought of self consorts well with a desire and endeavour to help others. A whole-time altruism is a futile philosophy.

There are few callings in which there is so big a gap between routine and the best work as in medicine, and no profession needs to be so elastic in its government if it is to be dynamic, not static. Its front line of knowledge is always mobile and often advancing; the men working there need freedom for initiative and should be unfettered by the formulae of administration, and, may be, on the other hand, by the demands of practice. And the same considerations hold in the sphere of clinical practice, for though we need ordered planning we must strive to avoid any cast-iron uniformity. Rather do we seek unity amidst diversity; for man, whatever his political colour, is individual, and in illness even more so than in health, and his doctor must remain an individual and not become an official. It is here that medical planning is up against its crucial difficulty, in that it requires collectivism for its fabric and individualism for human relationships. And individualism will not flourish easily within the rigid boundaries of a State service, but needs the freer atmosphere which belongs to the voluntary hospitals' tradition; for in the difficult conditions of a greatly changing world doctors will need not only knowledge but understanding if they are to guide bodies and minds along the straight road of health and content.

ESTIMATION OF PROTHROMBIN WITH VENOM AND LECITHIN

BY

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AND

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In earlier papers (Witts and Hobson, 1940, 1941; Hobson and Witts, 1941) we described the use of solutions of Russell-viper venom and lecithin in place of the tissue extracts commonly used in the estimation of the prothrombin time. A brief account of our present technique and the normal range of variation may be of interest to workers in this field.

Technical Details

Glassware.—Tubes are checked with a gauge to ensure that they are all of the same internal diameter. Glassware must be scrupulously clean; tubes are left in potassium bichromate and sulphuric acid cleaning solution overnight and then washed in running water for at least 4 hours.

Lecithin.—This is ovolecithin *B.P.C.* as purchased. Specimens are variable and not all give such good effects as we have described. It is therefore wise to try another sample if first results are disappointing. The active principle is not known, but it is probably an impurity or a breakdown product rather than pure lecithin. The strength of the stock solution of lecithin in alcohol should not be less than 10% to avoid an alcohol effect when the lecithin is added to the venom. As lecithin solutions become acid on standing the alcoholic solution of lecithin should be adjusted to a *p*H between 7 and 8 by means of a saturated solution of caustic soda in 95% alcohol; the reaction should be checked at fortnightly intervals. The optimal concentration of lecithin is 0.5%—i.e., 0.05 c.cm. of 10% alcoholic solution of lecithin in 1 c.cm. of the actual dilution of venom which is used.

Calcium.—We use M/40 $CaCl_2$ solution, which is the same as for Quick's test.

Temperature of Water-bath.—The optimal temperature is from 37° to 43° C. As prothrombin is very thermolabile, it is wise to work at the lower end of this range. Our observations have been carried out at 38.5° C.

Plasma.—Nine c.cm. of blood, withdrawn rapidly and with special precautions to avoid trauma, is promptly and thoroughly mixed with 1 c.cm. of M/10 potassium oxalate and centrifuged at 1,500 r.p.m. for 5 minutes. The duration and speed of centrifugalization should be maintained constant. If there is obvious haemolysis the plasma should be discarded, more particularly if the lecithin difference is to be determined. As oxalated plasma is not stable for much longer than an hour at room temperature the oxalated blood or plasma should be put into the refrigerator as soon as possible after venepuncture and removed only immediately before the estimation of the clotting time. It seems immaterial whether the blood is centrifuged soon after venepuncture or just before the estimation. In the refrigerator the plasma will keep its clotting time unchanged for at least 24 hours.

Estimation.—A dry clean test-tube (50 by 8 mm.) is placed in the water-bath and 0.1 c.cm. of plasma and 0.1 c.cm. of the venom–lecithin reagent are added. Mix and leave to attain the temperature of the water-bath; the right time is about 1½ to 2 minutes. Then add 0.1 c.cm. of the calcium solution, which is kept in the incubator alongside the water-bath, and start the stop-watch. The end-point is different when venom alone and venom–lecithin are used. In both cases the tube is held at a slant of 45 to 60 degrees and rotated round its longitudinal axis. The end-point with venom alone is the moment when fibrils of fibrin first appear. If the same criterion is used when the venom–lecithin reagent is employed the end-point is much more difficult to detect, because the opalescence obscures the fibrin fibrils, as Fullerton (1940) has pointed out. As the tube is slowly rotated, however, a fine film of opalescent liquid quite suddenly appears on the inside surface of the tube above the surface of the mixture. This film is actually coagulated plasma, and the end-point is the moment when it is first seen. It is a very sharp end-point, and is best seen by diffuse light. The tube should not be rotated round its horizontal transverse axis or shaken, because these manœuvres accelerate the end-point in a variable manner.

Lecithin Difference.—The lecithin difference is the difference in seconds between the clotting time with venom alone and with the venom–lecithin reagent.

Normal Values and Range of Variations

We have previously shown that whereas different batches of Russell-viper venom may vary in strength, samples from the same batch are constant. In the present investigation we have estimated the accelerated clotting time of the plasma from 56 normal men and women (medical students, laboratory workers, and nurses), using venom from a single batch. The venom was used in two dilutions—1 in 10,000, which is the maximum potency, and 1 in 80,000, which had seemed in our earlier experiments to provide a more convenient speed at which to work. Quick's test was also carried out on these same plasmas. For this we used two different preparations of dried rabbit brain. The first was prepared for us by Messrs. Boots Pure Drug Co., Ltd., who mixed 10 g. of dried rabbit brain so that it was homogeneous and then packed it in evacuated ampoules, 0.1 g. per ampoule. The second preparation was made in our own laboratory as required, and represents several batches. The results are given in Table I.

TABLE I.—*Normal Range of Variation for Quick's Test (Prothrombin Time), using as Thromboplastin Dried Rabbit Brain and Two Different Dilutions of Russell-viper Venom, With and Without Lecithin*

Reagent	No. of Observations	Coagulation Time in Seconds		Coefficient of Variation %
		Mean	Standard Deviation	
Dried rabbit brain (Oxford)	55	20·3	± 1·5	7·4
Dried rabbit brain (Boots)	55	22·0	± 2·2	10·0
Venom 1 in 10,000 ..	56	16·9	± 1·5	8·9
Venom 1 in 10,000 plus lecithin 0·5%	56	5·9	± 1·0	16·9
Lecithin difference ..	56	11·0	± 1·7	15·4
Venom 1 in 80,000 ..	56	22·2	± 2·0	9·0
Venom 1 in 80,000 plus lecithin 0·5%	56	10·0	± 1·1	11·0
Lecithin difference ..	56	12·2	± 2·8	22·9

The results suggest that when the reagent is used at its maximum potency accuracy is lost, presumably because the error in timing is constant and forms a larger proportion of the whole when coagulation takes place more quickly. If coagulation is unduly prolonged the end-point is not sharp, and it is probable that the greatest accuracy is obtained when the coagulation time lies between 10 and 30 seconds. In normal subjects venom alone gives slightly more consistent results than venom plus lecithin, a point made by Fullerton (1941), but the addition of lecithin is a safeguard against errors which may occur if venom alone is used in abnormal subjects or without special care in collecting the plasma. The lecithin difference is higher when the venom solution is somewhat more dilute than the maximum potency, and this, too, has advantages. We suggest that when the venom–lecithin reagent is used for the determination of accelerated clotting times it should be standardized to give a normal value of 10 seconds. This is identical with the clotting time when acetone-extracted rabbit brain is used in Quick's test (Pohle and Stewart, 1939), and the figure has certain advantages when we come to express the results in terms of prothrombin. Any venom which will not reduce the clotting time to 10 seconds when fortified with lecithin should be discarded.

The range of variation of the clotting time of 56 normal human plasmas with a venom–lecithin reagent standardized to give an average value of 10 seconds was 8 to 12 seconds. The coefficient of variation was practically the same when we used dried rabbit brain. Nygaard (1941), using acetone-extracted rabbit brain in 32 normal adults, found an actual range of 11 to 19 seconds and, by statistical computation, an average prothrombin time of 13.3±0.35 seconds. Owen and Toohey (1941), employing fresh emulsions of human brain and rabbit brain in 97 normal controls, found a range of 10 to 16 seconds and an average of 13.97 seconds. Holmboe and Holmboe (1940), who used 0.5% emulsion of an extract of sheep brain, in contrast with the usual 10% emulsions, found a range of 55 to 85 seconds, with an average of 70 seconds. The coefficient of variation with all these techniques is of the same order, and it is probable that this represents a real difference in the coagulability of normal human plasmas. It seems unlikely, however, that it is entirely or, indeed, mainly due to variations in the quantity of prothrombin.

Expression of Results

As normal values for accelerated clotting times vary with the technique, some method of scoring or comparison is desirable. The simplest is an index, in which the reading obtained is compared with the normal value. Illingworth (1939) uses as prothrombin index:

$$\frac{\text{Normal time} \times 100}{\text{Observed time}}$$

and finds indices in health of 100±10. As we have shown, the normal range of variation is a good deal wider than this. This index is nevertheless a rational method of expressing prothrombin values because, as we shall see later, the clotting time is inversely proportional to the concentration of prothrombin.

Others have preferred to use a standard of reference in which the clotting times are related to the amount of prothrombin actually present. A correlation curve of this type may be obtained by making progressive dilutions of oxalated plasma and measuring the accelerated clotting time of each dilution (Quick, 1937). Saline is an unsatisfactory diluent, as it lowers the concentration of protein, and prothrombin-free plasma or a solution of fibrinogen should be used. We use "alumina plasma," which is normal plasma from which the prothrombin has been absorbed by aluminium hydroxide (Quick, 1936). The plasma is shaken with aluminium hydroxide cream (C-gamma reagent) in the proportion of 0.1 c.cm. of cream to 1 c.cm. of plasma, incubated for 15 minutes at 37° C., and centrifuged 15 minutes at 2,500 r.p.m. Prothrombin can also be removed from the plasma by Seitz filtration (Lozner, Kark, and Taylor, 1939). Both methods are simple but unfortunately far from ideal, as R. G. Macfarlane and R. H. Mole (1942), working at this hospital, have shown that the plasma may be considerably altered in other respects when the prothrombin is removed by alumination or Seitz filtration.

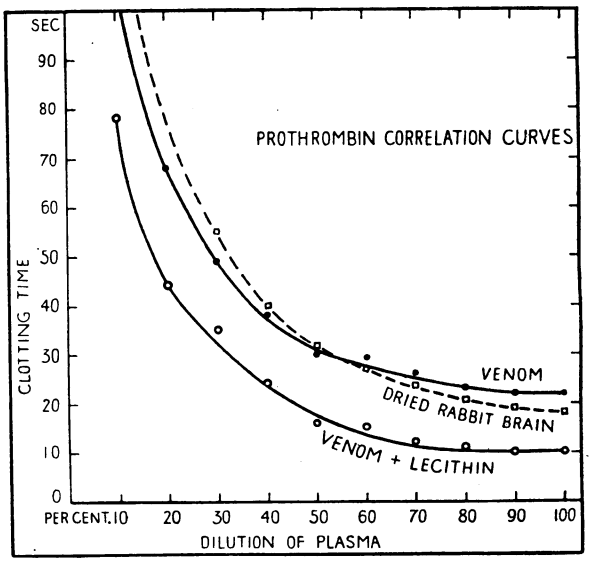

FIG. 1.—Curves correlating the prothrombin time with varying concentrations of prothrombin as obtained by increasing dilution of the plasma. The curves are from different normal subjects, but they show the similarity of the results with different thromboplastins.

Correlation curves for venom, the venom–lecithin reagent, and dried rabbit brain are shown in Fig. 1; they all have the same shape. Coagulation times may be converted into prothrombin percentages by reading the corresponding value from the chart or by using a formula derived from the curve (Quick, 1939), but it is better to make dilutions of the unknown plasma and obtain several readings for comparison with the normal curve. In practice these curves are not very convenient for converting accelerated clotting times into prothrombin percentages because little change takes place in the coagulation time until the prothrombin is reduced to 30% of normal and only the narrow range between 5 and 30% is really suitable for readings. Nygaard (1941) has stated that each concentration of prothrombin of the undiluted plasma has its corresponding dilution curves, or, in other words, that curves from subjects with hypoprothrombinaemia cannot be made to fit the normal curve; but this has not been our experience, and it may well be that Nygaard has been led astray by the use of saline as a diluent.

Jaques (1941) has shown that a straight line is obtained if the logarithms of the clotting time are plotted against the logarithms of the *thrombin* concentration, and we have found the same approximately true of *prothrombin* concentrations in our dilution experiments. When the data from all the available instances were plotted as logarithms of clotting times against the logarithms of plasma concentration, it was clear that each set could be fitted reasonably well to a straight line and that the slopes of the lines did not differ greatly in the different instances. The data from the 13 instances available were therefore pooled and the slope of the line best fitting all the data was calculated. The value obtained was −1.01±0.03. Since

this is not significantly different from unity, it indicates that the clotting time is inversely proportional to the plasma concentration. In human plasma the agreement between the calculated values and the observed values is often close, as shown in Table II and Fig. 2. In plasma from the dog and the rabbit

TABLE II.—*Data of Prothrombin Titration on Human Plasma with Various Thromboplastins. Successive Dilutions of the Plasmas were made with Prothrombin-free Aluminated Plasma*

Reagent	Subject	Plasma Concentration %									
		100	90	80	70	60	50	40	30	20	10
Venom alone : Stypven 213 1/80,000 ..	O'B.	22	22	23	26	29	30	38	49	68	115
Venom + Lecithin : Stypven 213 1/80,000 + lecithin 0·5%	O'B.	10	10	11	12	15	16	24	35	44	78
,, ,,	So.	9	11·5	13	13·5	14	16	18	23	30	175
,, ,,	St.	10	11	12·5	15	17	20	24	29	39	62
,, ,,	Ta.	10	12	13	15	16	19	24	27	35	55
,, ,,	Bl.	11	11	13	16	19	22	26	31	40	70
,, ,,	Al.	11	13	14	16·5	20	24	30	36	52	85
Rusven 1/50,000 + lecithin 0·5%	Mo.	10	11	12	13	14·5	19	23	31·5	55	210
Stypven 226 1/60,000 + lecithin 0.5%	We.*										
20/11/41 ..		7	7	8·5	10	13	15	18	24	32	54
26/11/41 ..		16	17·5	18·5	22	27	35	44	56	80	140
30/11/41 ..		27	29	34	43	48	56	69	100	130	240
Dried rabbit brain	Sm.	23	24·5	25	25	29	30	40	42	62	150
	Mo.	18	19	20·5	23·5	27·5	31·5	40	55	120	290

* Before and after dicoumarin.

the observations often diverge from the straight line a good deal, particularly with the higher concentrations of plasma. The divergence can be expressed by saying that in high concentrations of plasma the clotting time is longer than the ideal, whereas in low concentrations of plasma it is shorter. It is not due to the speed of the accelerated clotting reaction in these animals and consequent inaccuracy in measuring the times, as it occurs with relatively slow reagents such as venom alone.

These methods of expressing the results in man apply whatever type of thromboplastin is used, though it is probable that

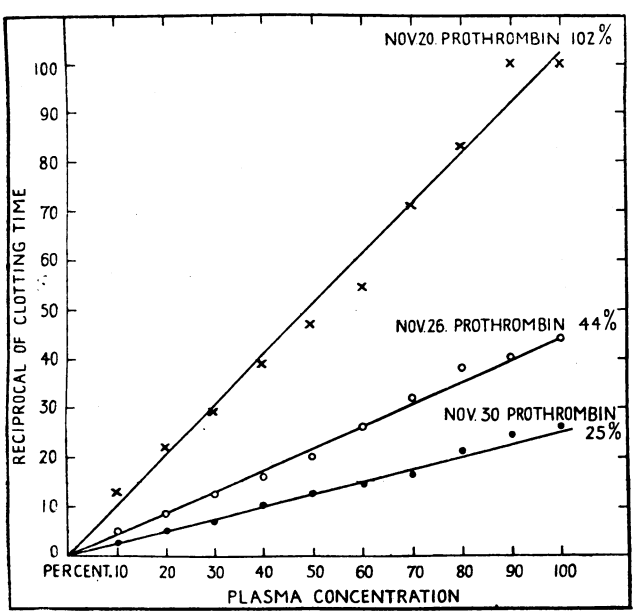

FIG. 2.—Prothrombin dilution curves from patient We. before and after treatment with dicoumarin. The lines are plotted from the values for prothrombin calculated from the data in Table II; they agree fairly closely with the individual observations. As the venom-lecithin reagent used in these experiments had not been standardized and gave a normal clotting time (C) of 7 seconds, reciprocals have been calculated as 700/C instead of the standard 1,000/C.

divergencies from the ideal are higher with venom alone than with the venom-lecithin reagent or tissue extracts. An example of the use in clinical work of complete prothrombin dilution curves, charted in the way we have suggested, is given in Fig. 2, which shows the values obtained from a patient before and during treatment with dicoumarin (Butt *et al.*, 1941). Such a complete range of titrations is unnecessary in routine work, but the graphs show the value of estimating the clotting time of several dilutions of any particular plasma. The results of our observations are expressed somewhat dogmatically in the following conclusions:

1. In estimating prothrombin greater accuracy can be obtained by measuring the accelerated clotting time of several dilutions of the plasma—e.g., 100%, 50%, and 25%. These dilutions are best made with prothrombin-free plasma, but normal saline can be used with little disadvantage.

2. Graphs of prothrombin estimations are best made by plotting the plasma concentration as abscissa against the reciprocal of the clotting time as ordinate. Reciprocals can be obtained from four-figure mathematical tables.

3. If the reagents are standardized to give a clotting time of 10 seconds at 100% plasma concentration, then the amount of prothrombin in a particular instance is calculated from the equation:

$$\%\ \text{normal prothrombin} = 100\left(\frac{\text{mean value of } 10^3/\text{clotting time}}{\text{mean value of plasma concentrations}}\right)$$

Thus an accelerated clotting time of 8 seconds with undiluted plasma would indicate a prothrombin level of 125%, and 12 seconds would indicate 83%. In another case the times actually observed were 10 seconds at 100% plasma concentration, 20 seconds at 50% plasma concentration, and 39 seconds at 20% plasma concentration. Hence the prothrombin level was:

$$100\left(\frac{100+50+26}{100+50+20}\right) = 104\%$$

4. To standardize the venom-lecithin reagent, make up a series of dilutions of venom in saline, adding 0.5% lecithin to each dilution. Now find the concentration of reagent which gives a clotting time of 20 seconds at a 50% concentration of normal plasma, and check it again to see that it clots undiluted plasma in 10 seconds.

Limitations of Test

Although we have shown how accelerated clotting times may be converted into prothrombin percentages, the calculation is based on the assumption that the accelerated clotting time varies only with the amount of prothrombin present. Experience has shown that this is true often enough for the test to be of clinical value, but it is by no means always the case. All that we are measuring is the time in which the plasma clots under certain arbitrary conditions, and when we express this in terms of prothrombin we are making assumptions which may be erroneous. This point is illustrated by the following experiment, in which the patient received an injection of 80 mg. of heparin intravenously (Table III). The clotting power of

TABLE III.—*Effect of Intravenous Heparin on Coagulation and Prothrombin Time*

Time in Minutes after Injection of Heparin (80 mg. I.V.)	Platelets per c.mm.	Bleeding Time in Minutes	Coagulation Time in Minutes			Prothrombin Time in Seconds				Clot Retraction %
			Lee and White	Dale and Laidlaw	Howell	Dried Rabbit Brain	Venom	Venom + Lecithin	Lecithin Difference	
0	414,000	1	9·5	5	2	19·5	24	7	17	Good : 48
5	432,000	1	50	14	15–30	28	120–150	14	120	Nil : 26
30	520,000	1	100	9	6–10	24	90	10	80	Nil : 40
90	523,000	1	37	10	6–7	20	37	7·5	29	Good : 50
240	370,000	1·5	10	4·5	2·2	19·5	24	7·5	16	Good : 42

the blood, as measured by a variety of techniques, was uniformly diminished and therefore the prothrombin time was increased. Heparin acts both as an antiprothrombin and an antithrombin, but there is no evidence that it alters the prothrombin itself either quantitatively or qualitatively (Ferguson, 1939). It would therefore have been inappropriate to express these altered coagulation times in terms of hypoprothrombinaemia, and the only legitimate observation is that the coagulation of the blood was diminished. At 90 minutes the prothrombin times had returned to normal and excluded hypoprothrombinaemia; the unaccelerated clotting times were still high and demonstrated the presence of a coagulation defect; the high lecithin difference was in keeping with the fact that this

was not haemophilia. Conditions which are strictly comparable to this phase of the experiment occur spontaneously in man, and we hope to discuss them on another occasion.

Discussion

These data are presented in the first place to show that a venom–lecithin reagent gives results in the accelerated clotting test which are in all respects comparable to those of thromboplastins such as brain extract; and in the second place to give figures for normal values and range of variation. The most potent accelerator of coagulation which we have used is a mixture of Russell-viper venom with 10% emulsions of brain. When this is added to normal plasma, clotting occurs in 5 seconds after recalcification, which is faster than with any other reagent recorded in the literature. Such a reagent demands for its study instrumental methods such as Nygaard's, which are unfortunately hard to procure at the present time. Without such instrumental aids excessive potency of the clot-accelerating reagent may, as we have shown, be a source of error. We hold no brief for Quick's test in any of its modifications as a means of estimating prothrombin. It is affected not only by the rate of conversion of prothrombin into thrombin but also by the presence of inhibitory or accelerator principles. It is nevertheless a valuable clinical test when due heed is paid to possible fallacies. Russell-viper venom and lecithin are relatively cheap and stable reagents for the test. They allow us to decompose the action of thromboplastin into its water-soluble and lipoid-soluble moieties and thus gain more insight into disturbances of coagulation than is possible with the more complex reagent. They have the additional advantage that they are effective in widely different animal species. Results with dicoumarin suggest that this is a point of great importance, and we shall deal with it in a subsequent paper.

Summary

Quick's prothrombin test is based on the hypothesis that if the coagulation of plasma is accelerated by excess of thromboplastin the speed of coagulation will depend mainly on the amount of prothrombin present. Other things being equal, the speed of coagulation is inversely proportional to the concentration of prothrombin.

When reagents composed of mixtures of Russell-viper venom and crude lecithin are used as thromboplastins the coagulation time of recalcified plasma can be reduced to 5 seconds. Unless instrumental methods of recording are used, such reagents are too swift for accurate work.

The venom–lecithin reagent has been standardized to give an average coagulation time of 10 seconds. The normal range of variation has been determined, and methods of scoring the results in terms of prothrombin are discussed.

We are indebted to Dr. R. H. Mole for much help in standardizing the technique of the test, and to Dr. R. B. Fisher for explaining the mathematical treatment of the data.

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Sixty per cent. of India's medical stores are now made in India as compared with 25% at the beginning of the war. Instrument manufacturers have expanded their works and increased their production, and only such instruments as cystoscopes, microscopes, etc., need be imported. A number of medicinal items have recently been taken off the list of importations, also certain bacteriological stains which are now prepared by the distillation of ordinary commercial and crude dyes. The rubber industry has widened its range of products so that it is now possible to obtain a large percentage of the appliances necessary in hospital.

PERINEURITIS

BY

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In the diagnostic sphere the territories of the neurologist and the physiotherapist overlap at one point—in the elucidation of some of the diffuse pains so often spoken of by the patient as "neuritis." Many, probably most, patients complaining of "neuritis" are suffering from referred pain clearly demonstrable as taking origin from a deeply situated somatic tissue. Other patients with this complaint are found to have some disease of the nervous system. Evidence has, however, been accumulating that cases occur in which, despite the presence of unimpaired conduction along a nerve, the pain originates in a nerve. Such pain is not the lancinating stab of neuralgia but a continuous burning ache closely resembling that caused by severe inflammation of such structures as muscle, tendon, joint capsule, etc. In these cases the following criteria are satisfied: (1) no pain can be elicited from the muscles, joints, etc., comprising the same group of segments as the painful part; (2) conduction along the nervous system is normal; (3) the pain is referred to a region obviously corresponding accurately to the area of cutaneous supply of a peripheral nerve. The conclusion is then inescapable that the pain arises in a nerve but not in its conducting elements. In other words, it is the nerve sheath that is inflamed—perineuritis.

Theoretical Considerations

Since the inflamed structure in perineuritis is a fibrous sheath, the quality of the pain resembles that of fibrositis of muscles and similar tissues. It is in its mode of reference that it differs; for not only does the region in which the pain is felt correspond with a known cutaneous area, but the patient can tell whether the front or the back, say, of the finger is affected. In fibrositis of other structures the pain is usually felt right inside the part. Though at first sight it may appear unlikely, symptoms due to perineuritis are often brought on by using the painful member. This can be demonstrated by a simple experiment. The subject sits on the edge of a chair in the manner known to cause pressure on the sciatic nerve in the upper thigh. When paraesthesiae can be felt in the foot, the subject, keeping otherwise perfectly still, voluntarily flexes his toes. This at once increases the pain, though the movement in no way alters the tension on the affected part of the nerve trunk. Hence, though the cause of this phenomenon is obscure, a complaint of pain brought on or aggravated by exercise does not preclude perineuritis.

Secondary parenchymatous change may occur in perineuritis, presumably because of the pressure exerted by the swollen sheath on the nerve fibres. In sciatic perineuritis this occurred in 10 cases out of 18, and showed itself by wasting of the gluteal muscles and by incomplete ability to contract them voluntarily, wasting of the hamstrings, and loss of the ankle-jerk. When these neurological signs appear the label "sciatic neuritis" would seem warranted, but this name leads to an error in emphasis, focusing attention upon the secondary rather than the primary organic process.

In these figures of proved cases of sciatic perineuritis lies the answer to the school of thought that would confine the use of the term "sciatica" to sciatic neuritis. Since roughly half of all cases of sciatic perineuritis show no evidence of loss of conduction along the nerve, this limitation results in the indefensible position in which some, but not other, cases of sciatic perineuritis are called "sciatica." Moreover, a few patients with perineuritis complain only of backache: this raises the question whether that symptom, if of sciatic periradicular origin, can be called sciatica. The alternative, and to my mind better, course is to retain the word sciatica—well known to the public—as the name of a common symptom, like headache. A medical definition compatible with this use of the word would then be: "A