Clonus was marked whenever he tried to use his arm. All sensations were intact. A median and ulnar nerve block at the elbow was performed with 3 ml. of 3 per cent phenol in saline injected at each site. After this block the patient was able to grasp and raise to his lips a standard drinking glass filled with water. Clonus in the lower arm no longer presented a problem. His major difficulty now was upper arm spasticity and clonus of the biceps and triceps brachii both on rapid velocity and passive movement. One month later a left brachial plexus block by the suprascapular approach was done using 9 ml. of 3 per cent phenol. Immediately post block, spasticity went to 0 in the biceps. One week after block both biceps and triceps were 0–1 plus. Clonus was gone. Functional activity increased dramatically to the point where the patient was able to do two push ups. In the months which have followed the block there has been some return of spasticity but functionally the arm remains good. He is now on an active regime to improve coordination. Aside from some soft tissue swelling at the sites of injection, which disappeared without treatment in 48 hours, there have been no untoward sequelae. All sensations are the same as before the block.

Discussion

The use of dilute phenol solutions for peripheral nerve injections appears to be a relatively benign procedure. There is occasionally mild but quite tolerable discomfort upon injection. One patient has had a persistent hypesthesia over the ulnar distribution after block. Aside from this there have been no permanent or serious sequelae in over 50 peripheral nerve blocks.

It is our current opinion that when spasticity does not respond to drug and rehabilitation regimes nerve block with dilute phenol should be considered. The advantages of decrease in spasticity and possible unmasking of motor function far outweigh the fact that at present the technique and drugs used are not successful in all cases. Since the nerves to the muscles of the upper arm are technically difficult to isolate for injection further investigation of brachial plexus phenol infiltrations is being pursued.

Further Experience with the Earlobe Algesimeter

Ephraim S. Siker, M.D., Bernard Wolfson, M.D., William D. Stewart, M.D.*

An earlobe algesimeter described by one of us [1] in 1954 has recently been modified and tested. The original apparatus consisted essentially of a standard inductorium connected to a 1½-volt dry cell battery with identically wired primary and secondary coils. Direct interrupted current was produced through the electromagnetic circuit breaker of the inductorium and adjusted to produce a faradic current of 60 pulses per second. The current was directed across the earlobe of the subject by means of an adjustable earpiece, the voltage increasing as the secondary coil was manually moved toward the primary coil. The end point (pain threshold) was distinguishable as a distinct pricking sensation which was preceded by a feeling of vibration in the earlobe.

The current source, type of current, and basic premise in the modified apparatus remain unchanged. The modifications include: (1) an electric motor which drives the secondary coil toward the primary coil at a constant, fixed speed; (2) a variable resistance voltmeter which allows precise reading of pain threshold in volts; (3) a lock switch with release which makes it necessary for the subject to start and stop each pain threshold determination; (4) a test switch which registers the pain threshold voltage on the voltmeter while bypassing the earpiece. (This latter allows leisurely and accurate reading of the threshold after each determination.) The elec-

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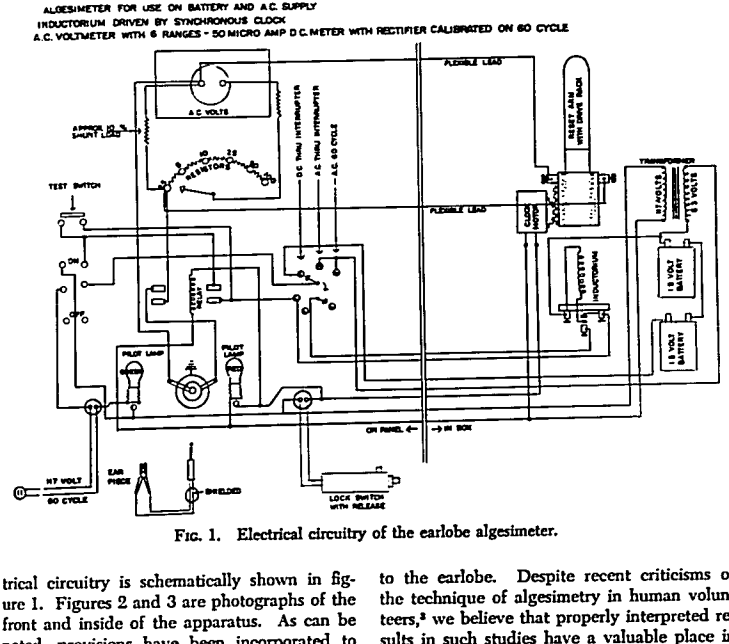

FIG. 1. Electrical circuitry of the earlobe algesimeter.

trical circuitry is schematically shown in figure 1. Figures 2 and 3 are photographs of the front and inside of the apparatus. As can be noted, provisions have been incorporated to allow the future study of alternating 60 cycle line current (via the transformer shown) directly or through the interruptor. The apparatus is capable of delivering from 0.5 to 10.0 volts across the earlobe. Concomitantly there is a linear but disproportionate increase in current from 8 to 30 microamperes. With each subject acting as his own control, unknown levels of inductance, capacitance and resistance in the biological circuit cease to be important determinants.[4, 5]

Use of the earlobe as the site of stimulus application was based primarily upon the work of Sinclair and his associates [2] who found only two types of receptors in the earlobe: a basket-like network surrounding the hair follicles, and undifferentiated bare nerve endings. Their test subjects could nevertheless distinguish equally well between heat, cold, pin prick and touch whether applied to the forearm or to the earlobe. Despite recent criticisms of the technique of algesimetry in human volunteers,[3] we believe that properly interpreted results in such studies have a valuable place in the study of pain relieving drugs.

MATERIAL AND METHOD

Using the above described algesimeter, 8 male and 8 female volunteer subjects were used for pain threshold determinations following the intravenous injection of meperidine. For comparison, determinations were also made after injections of saline. Neither the subjects or the operator of the algesimeter knew the nature of either injection. No attempt was made to define pain and each subject was told only to press the "off" button when he or she felt "pain."

Before the start of the test, each subject reclined in the quiet test room for a period of 20 to 30 minutes following which three control determinations of pain threshold were obtained at 10-minute intervals. The average of these determinations served as the control for

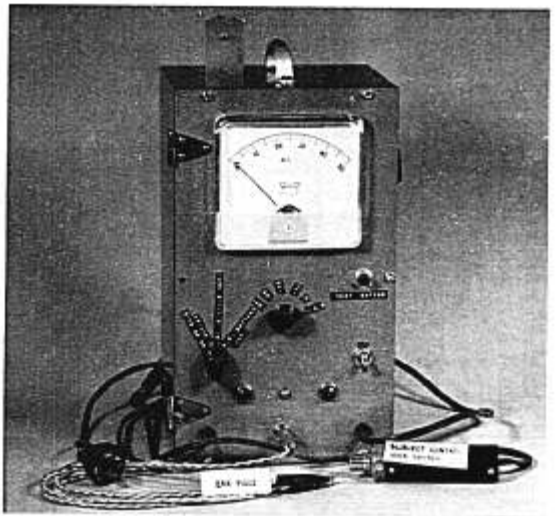

FIG. 2. Earlobe algesimeter.

each subject. Following this, the subject received 5.0 ml. of saline or 1.0 mg. per kg. meperidine in 5.0 ml. of distilled water intravenously over a 2-minute period. The end of the injection was taken as zero time and pain thresholds, recorded in volts, were measured at 10, 20, 30, 40, 50, 60, 80, 100, 120, 140, 160 and 180 minutes.

RESULTS

The average of the control pain threshold in volts for the 16 subjects was 2.12 ± 0.77 volts in the meperidine test and 2.12 ± 0.95 volts in the test with saline. With each subject acting as his own control, all pain thresholds were converted to percentage of control. As can be seen from table 1, the averages of the percentage of control in the meperidine and saline tests vary substantially. The differences in pain threshold are significant from 10 ($t = 2.0$) through 60 ($t = 5.5$) minutes. Although the average pain threshold remained higher in the meperidine than in the control group from the 60- through the 140-minute readings the differences were not significant.

Table 2 lists the average of the pain thresholds in the two tests, measured in volts, which again demonstrates the differences between the two groups. The relation between the pain threshold determinations obtained in the meperidine group and those in the control group are shown in figure 4.

TABLE 1. Average Pain Thresholds, Expressed as Percentage of Control, Following Injection of 0.1 mg./kg. Meperidine or Saline

Time (minutes)	Meperidine	Saline
0	100.0 ± 0	100.0 ± 0
10	118.0 ± 26.2*	101.8 ± 29.2
20	127.1 ± 29.1	97.0 ± 21.7
30	127.0 ± 25.9	97.5 ± 21.1
40	126.6 ± 31.2	99.0 ± 22.3
50	136.9 ± 28.3	97.7 ± 16.0
60	129.0 ± 21.4	98.6 ± 19.5
80	113.0 ± 28.9	90.7 ± 16.5
100	114.1 ± 32.8	100.4 ± 28.6
120	112.8 ± 37.1	96.0 ± 18.3
140	112.4 ± 25.8	100.2 ± 19.0
160	99.1 ± 21.9	99.0 ± 20.0
180	93.3 ± 17.2	98.7 ± 21.4

* Standard deviations.

Discussion

Harris and Blockus [6] have stated that, "to validate an algesimetric procedure it should be demonstrable that a compound generally acceptable as a clinical analgesic can cause the threshold of experimentally induced pain to become higher than it might otherwise be if no treatment or a placebo had been given." The results obtained in the present study satisfy this criterion.

A number of investigators have criticized algesimetry as a technique for the study of pain relieving drugs. The dissimilarity between true pain of organic origin and experimental pain has been stressed by some,[7] while others [8] have questioned the results of experimental pain studies in "drug-wise subjects from whom the use of a narcotic cannot be hidden." In addition, many attempts to reproduce the published results of experimental pain studies have been unsuccessful.[3, 9, 10]

Bishop [11] has separated pain into two components: (1) a sensation with its own sense organs and fibers, and (2) an unpleasant psychological experience. The part played by the second component is obviously different in the subject of an experimental pain study as compared to a "sick and anxious patient whose pain is mysterious, unpredictable and of unknown causation." [7] The authors agree that this limits the ability to transfer the results of algesimetric studies directly to patients with pain but not that these techniques are completely invalidated as investigational aides. Further, limitations must also be placed upon the interpretation of the results in algesimetric studies. It should be stressed that the increase in voltage recorded at pain threshold may not be proportional to the increase in the amount of pain that the subject experiences or can tolerate. The administration of a narcotic may result in a doubling of the voltage which represents the pain threshold, but this does not mean that the subject can now tolerate twice the amount of pain. Thus, algesimetry cannot be said to be a quantitative method of study except in crude terms. In a comparison between different drugs or different doses of the same drug interpretation should be limited to a statement of relative effects: Drug A elevates the pain threshold more or less than drug B; or, 10 mg. of drug A elevates the pain threshold more than 5 mg. of drug A.

Still further limitations on interpretation have been suggested by the recent work of Robson and his associates.[12] These investigators have shown that the same drugs may yield opposite results when different modalities of pain are tested. They found that the administration of thiopental sodium was associated with an increase in the threshold to thermal pain and a decrease to tibial pressure pain. Since electrical pain has been charac-

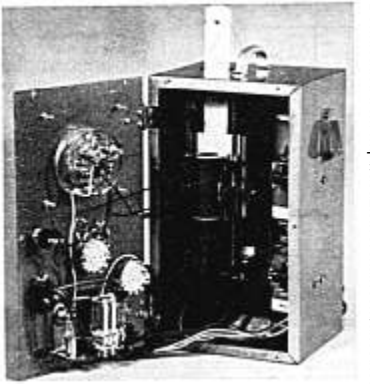

Fig. 3. Earlobe algesimeter with face plate open.

Table 2. Average Pain Thresholds in Volts Following Injection of 0.1 mg./kg. Meperidine or Saline

Time (minutes)	Meperidine	Saline
0	2.12	2.12
10	2.55	2.13
20	2.65	2.33
30	2.48	2.10
40	2.60	2.18
50	2.82	2.14
60	2.67	2.12
80	2.35	1.84
100	2.40	1.99
120	2.33	2.10
140	2.32	2.24
160	2.06	2.06
180	1.78	1.96

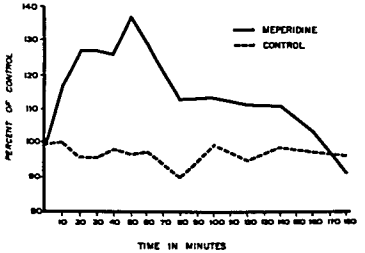

FIG. 4. Average pain threshhold.

terized as the pain of pin prick,[7] the investigator using an electrical source of experimental pain should qualify his results in the light of this possibility. Despite these limitations, the authors believe that algesimetry may yield valuable information on the potential analgesic effectiveness of a new drug. It may well be that this is especially true when assessing the effectiveness of an analgesic to be used in conjunction with general anesthesia. Obviously, there can be no psychological component to pain under these circumstances so that, in this respect, a patient under anesthesia more closely resembles the subject of an experimental pain study than the patient with true pain.

The earlobe algesimeter is no less subject to the above mentioned limitations than are the countless other pain threshold testing devices which have been described. The specific advantageous features of this device include the ease with which the end point is read and the relatively esthetic method of pain application. In addition, the reaction time of the test operator need not be considered since the subject starts and stops each determination.

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