of mild attacks of pain occurred when the patients were startled or when they were markedly anxious. In every patient sustained sensations of burning and aching were experienced over one or more divisions of the fifth nerve on the side of the tic for the first two to six days of nicotinic acid administration. Thereafter, in all but one patient, pain was eliminated during the short period of observation. It would appear that the paresthesias were due to partial ischemia because they were eliminated by inhaling amyl nitrite. In three patients nicotinic acid was effective in reducing the frequency and intensity of attacks for the first two days of administration, but then for two to three days spontaneous attacks occurred more frequently and were more intense, and attacks could be more readily induced experimentally. Thereafter, attacks of pain were eliminated. Cessation of administration of nicotinic acid was followed by a return of the attacks of pain, both the spontaneous and the experimentally induced. In some of these patients the recurrence of tic douloureux was first noted during episodes of anxiety or sudden physical activity.

In one patient an attack of tic douloureux was initiated at the peak of the pressor response when an extremity was immersed in ice water.

None of the vasodilator agents used raised the pain threshold.

CONCEPT OF MECHANISM

It is inferred from these observations that tic douloureux is the result of paroxysmal ischemia of trigeminal structures. The site of the ischemia may be central or peripheral. However, it is difficult to conceive of a central defect so circumscribed as to affect the function of only the trigeminal cells without involving adjacent nuclei and tracts. Also, such a defect must be so discrete as to produce pain limited to one or another division of the fifth cranial nerve. A peripheral ischemia involving Gasserian ganglion sensory root and nerves, however, could result in such a circumscribed disturbance. It is of interest that the vascular bed of the Gasserian ganglion is relatively poor.[15, 16] It is therefore postulated that afferent stimuli (touch, pressure, cold, muscle, etc.) arising from the "trigger area" evoke reflex vasoconstriction either widespread or local, but involving the trigeminal structures. Such reflex vasoconstriction alone, or more commonly when superimposed upon structurally narrowed vessels, results in a sudden and critical increase in ischemia and pain. This postulate is compatible with the observation that blocking such efferents by procaine or alcohol minimizes or eliminates tic douloureux for shorter or longer periods.

The short paroxysm of pain (from one to sixty seconds) can be understood as the effect of periodic vasoconstriction. In patients who are spontaneously having a series of attacks an episode of vasoconstriction may be followed by a short interval of improved blood supply, when the next of a series of vasoconstrictor episodes may induce another attack. Such phases may follow each other for one or more hours.

The time lapse between the administration of a vasodilator agent and reduction of the pain may be explained by assuming a cumulative effect of prolonged meager blood supply to nerve or a refractive state of the local blood vessels. The hyperalgesia of the skin over the painful area that occasionally occurs after a paroxysm of pain is compatible with a state of lowered pain threshold accompanying nerve ischemia.[8, 17]

In those patients who, when startled, apprehensive or subjected to immersion of an extremity in ice water, experience spontaneous attacks of pain, vasoconstriction within the trigeminal structures may be part of a widespread vasoconstrictor reflex or a response to a blood-borne pressor substance. In those patients in whom vasodilator agents modify the syndrome only slightly, structural changes in the blood vessels may be sufficiently advanced to prevent adequate vasodilatation. Because of individual variations in temperament, in the degree and rate of structural vascular changes and in tolerance to vasodilator agents, inferences about long term therapy are not justified. This study is focussed on the mechanism of pain in trigeminal neuralgia.

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SCIENTIFIC APPARATUS AND LABORATORY METHODS

SYNTHETIC LATEX AS INJECTION MASS FOR CLOSED VESSELS

THE use of natural latex as injection mass has been adequately discussed,[1, 2, 3, 4] but to our knowledge nothing has been published concerning synthetic latexes, although at present these are more available.

Two synthetic latex compounds have been used in the present work for vascular injection of cadavers. Present findings are based upon results of injection

[15] H. S. Dunning and H. G. Wolff, *Jour. Comp. Neur.*, 67: 433, 1937.

[16] H. G. Wolff, *Proc. Assn. Res. Nerv. and Ment. Dis.*, 18: 29, 1937 (see p. 52).

[17] N. Bigelow, I. Harrison, H. Goodell and H. G. Wolff, *Jour. Clin. Invest.*, in press, July, 1945.

[1] L. Petrovits and Z. Szabó, *Anat. Anz.*, Bd. 89, pp. 34–45, 1939.

of the entire arterial system in two cadavers, and regional injection, such as arms, legs and abdominal circulation, in an additional eleven cadavers.

The only advantage natural latex is found to possess is that of greater elasticity after it has set so that students may dissect with less danger of breaking or severing smaller twigs. However, synthetic latex mass becomes almost as tenuous, firm and flexible and is entirely satisfactory.

Synthetic latex was obtained from the manufacturer and color added before use. Cost of materials total approximately 75 cents per quart. Chemigum latex Type 100[5] to which has been added a finely divided insoluble water-borne red (A-2989 Toluidine toner)[6] behaves as natural latex and is used and handled with exactly the same technique.

Another latex used was Experimental Latex X-122[7] colored either with the water insoluble red as above or with a soluble dye (Chlorantine fast red 8-BLN).[8] This dye is used in concentrations not to exceed 0.4 per cent. of solids in a given amount of latex, and may be added directly to X-122. X-122 does not behave as natural latex, which coagulates suddenly on coming in contact with weak acid solutions, but may be injected into arteries without previously washing them out with weak ammonia solution.

Injections were made with a glass veterinary syringe, with rubber piston. An air pressure apparatus (5 to 7 pounds per square inch) did not provide sufficient force. Only one specimen had been recently embalmed. The others had been stored in vats for varying periods after embalming and took considerably less injection mass (40–50 per cent.) than can be put into soft pliable material.

Preparations of the cadavers were made through the femoral artery by injections in both directions—other limbs or organs were filled through the main artery normally supplying them. All sized arteries are readily filled. The arteries of the brain were well injected, also vasa vasorum and vasa nervorum as well as arterial anastomoses around joints.

The length of time required for coagulation is variable with the condition of the cadaver. Chemigum type 100 sets at about the same speed as natural latex. Latex X-122 requires a considerable period of time for setting so that preparations should be made several weeks before dissection is to be made. Each has advantages for special situations. X-122 would be preferable to use in a cadaver that had been stored in a vat for a long period. X-100 is more advantageous to use in an area which has been dissected or in mesenteric circulation, for if there is oozing through a rupture or a nick it can be coagulated by sponging with a weak acid solution and the hydraulic continuity of the vascular tubes restored, an impossibility if ligation is done. Also it is easier to sponge the surface of a vessel than to pick it up and tie it. Injection mass in exposed mesenteric circulation can be coagulated almost instantly by flooding the surface with weak acid and thus a preparation may be used immediately after injection.[9]

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[2] O. V. Batson, SCIENCE, 90: 518, 1939.
[3] D. P. Gamble, SCIENCE, 90: 520, 1939.
[4] E. E. Tobin, *Am. Jour. Roentgenology*, 51: 386–388, 1944.
[5] Goodyear, Akron 16, Ohio.
[6] Imperial Paper and Color Corporation, Glens Falls, N. Y.
[7] Dow Chemical Co., Midland, Mich.
[8] Ciba Company, New York, N. Y.

A NEW LABORATORY SHELLAC

THE recent scarcity of satisfactory shellac for the fixation and preservation of kymograph tracings made it imperative to look for satisfactory shellacking materials which can readily be made from ingredients commonly found in pharmacological and other biological laboratories.

At first from 10 to 20 per cent. alcoholic solutions of U.S.P. XII resin (colophony, white lump rosin obtained from the Arthur H. Thomas Company, Philadelphia) were used. These resins in such solutions, however, are quickly oxidized and the shellacked tracings become brittle. The best success was achieved by using the following formula: From 200 to 400 grams of rosin were dissolved in 2,000 cc of absolute or 95 per cent. alcohol; 400 cc of propylene glycol (resins are soluble in glycols) were added to the solution with 10–15 cc of castor oil as a plasticizing agent. The smoked tracing once immersed in this shellacking material must be allowed to dry for 12 hours, and then it may be rolled up and stored. The advantages of this new shellac is that records preserved with it do not become brittle, do not stick, and their surface does not become shiny. The tracings are easily photographed because of the lack of halation.

It may be added that ethylene glycol or other glycols may be used instead of propylene glycol to provide "body" and as antioxidants or any plasticizer may be used instead of castor oil. It is not to be assumed that the use of this shellac is limited to fixation of kymograph tracings.

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