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THE GASTRIC FIBRES OF THE VAGUS NERVE.

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Ever since the work of Gaskell and Langley, the vagus nerve has occupied a firmly established position in the parasympathetic nervous system, and amongst its contributions to that system are included the vagal branches to the stomach. In conformity with the general arrangement of neurones in this system, the gastric branches of the vagus are usually considered to be long connector neurones arising in the bulbar nuclei and passing via the vagus trunk to Auerbach's plexus; hence a short effector neurone is relayed to the stomach (Fig. 1, A).

The bulbar roots of the vagus and its trunk above the two vagal ganglia consist almost entirely of medullated fibres, both small and large. Below the ganglia a marked difference is to be found in the composition of the nerve, owing to the appearance of a large number of non-medullated fibres; as the pharyngeal and laryngeal branches leave the parent trunk, they take with them the majority of the medullated fibres so that the proportion of non-medullated increases as the nerve enters the thorax, till finally its abdominal branches consist almost entirely of non-medullated fibres. These changes were recognized by Gaskell, Langley [Schafer, 1900], Ranson [1914] and others, but they made no suggestion that these non-medullated fibres were anything but vagal in their origin, although divergent views were expressed as to how the myelin sheath was lost. Recently Kiss and his colleagues have again drawn attention to this increase in the non-medullated components and have suggested a very different explanation of their presence and origin.

Kiss [1931] has made histological studies of the vagus in man, using an osmic acid technique. He states that non-medullated fibres are not present in the vagus till the lower level of the plexiform ganglion is reached, and he suggests that these fibres are really contributions from the superior cervical ganglion passing to the vagus via the lower anastomotic branches between the sympathetic and vagal ganglia. These anastomotic branches fall into two distinct groups, an upper and a lower; the upper group consists of fine medullated fibres, which Kiss compares

to the white ramus communicans of a spinal nerve; the lower group consists of non-medullated fibres comparable to the grey ramus communicans returning to the spinal nerve. It is, therefore, suggested that the vagus bears the same relationship to the sympathetic as does a typical spinal nerve—fine medullated connector neurones arise in the bulbar nuclei of the vagus, pass via the vagal roots, trunk, plexiform

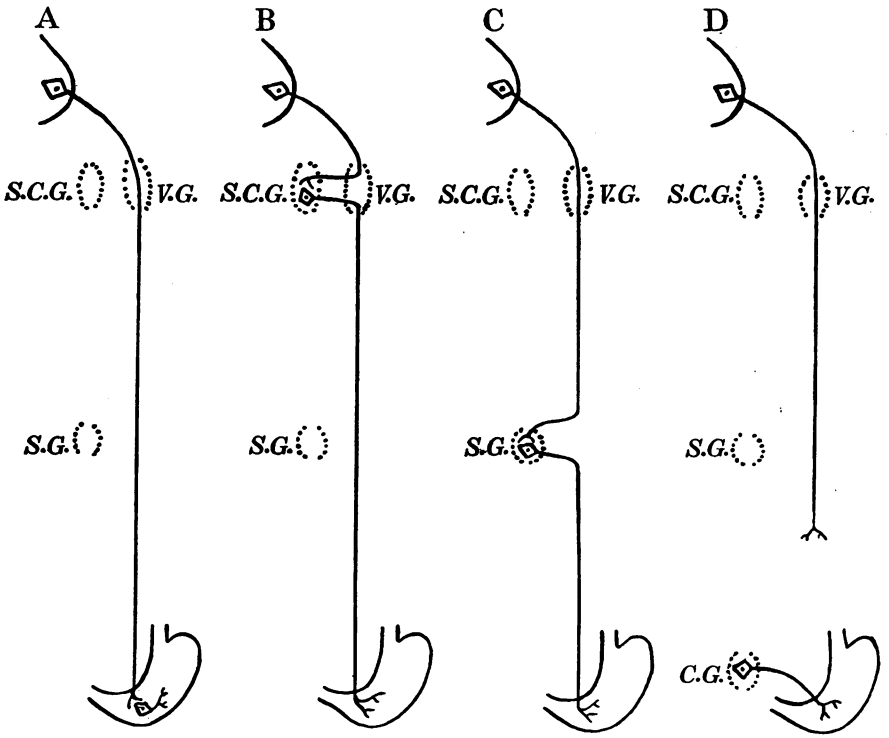

Fig. 1. The possible paths of the gastric fibres of the vagus. A, after Langley; B, C, D, after Kiss. *V.G.* ganglion trunci vagi; *S.C.G.* superior cervical ganglion; *S.G.* stellate ganglion; *C.G.* cœliac ganglion.

ganglion and upper anastomotic branches to a cell station in the superior cervical ganglion; in the superior cervical ganglion a second neurone begins, rejoins the vagus as a non-medullated fibre in the lower anastomotic branches and passes in the main trunk to its peripheral distribution. As the gastric branches of the vagus are almost entirely non-medullated, he therefore suggests that they have this sympathetic origin. Fig. 1 B gives a schematic representation of this suggested arrangement of neurones.

In further support of this idea, Kiss has made a series of comparative anatomical studies in which he claims that the innervation of the abdominal organs is always sympathetic in origin, though the path taken by the fibres varies in different species. Broadly speaking, the alternative paths fall into two groups, which are represented schematically in Fig. 1 (C, D)—in C the sympathetic contribution arises in the stellate or adjacent ganglia and passes to the thoracic vagus, whereas in D the sympathetic contribution passes direct to the stomach and the vagus, as such, does not reach that organ.

This conception of the arrangement of autonomic fibres in the vagus introduces a fundamentally new principle into the arrangement of the parasympathetic: Kiss goes so far as to say that in the abdomen no such separate system exists. Amongst his conclusions are the following statements: "The so-called parasympathetic influence of the vagus has no anatomical basis in the case of the abdomen. The parasympathetic phenomenon can be only a negative phase of the sympathetic."

Langley, Gad and Josephs [Schafer, 1900] considered that these vagal fibres lost their myelin sheath in their course, probably in the vicinity of the vagal ganglia, while Gaskell, from comparative studies, thought that these fibres represented a relay from a cell station on the vagus trunk.

From further histological studies Kiss [1932] states that he is able to identify a nerve cell which is "sympathetic," *i.e.* it gives rise to a non-myelinated fibre of the sympathetic system. These cells are found, not only in the sympathetic ganglia, but also in cranial and spinal ganglia, and they are therefore credited with supplying sympathetic elements within the cranial and spinal nerves. On these grounds he again makes the statement that: "There is no morphological foundation for the supposition of a cranial parasympathetic system."

Apparently Kiss takes the view that all the nervously controlled phenomena termed "parasympathetic" result from impulses passing down a system of non-myelinated fibres which are essentially "sympathetic" and arise from a certain type of cell within a sympathetic ganglion or in one of the cranial ganglia.

In order to test this theory we have performed experiments to determine the functional pathway whereby the vagus influences the stomach, and have tried to establish the source of the non-medullated components of the nerve. The animals employed have been cats. In these animals the histological differences in the vagus trunk are of a similar nature to those described in man—there are a few non-medullated

fibres in the vagal roots, but the number is markedly increased below the ganglion of the trunk. As the vagus does reach the stomach in the cat, condition D (Fig. 1) does not arise, and we have devoted our attention to the three other possibilities, A, B and C.

Effects of stimulation of intracranial vagus.

If the functional path of the gastric fibres is through the superior cervical ganglion, as suggested in B (Fig. 1), then removal of the superior cervical ganglion, and stimulation of the vagus above its ganglia should produce no effect upon the stomach. As the extracranial portion of the vagus above its ganglia is too short and inaccessible for satisfactory stimulation, we therefore proceeded to study the effect of stimulation of the intracranial portion of the vagus and the following technique was developed.

Method.

The animals were anæsthetized with ether, the carotids tied, and decerebration carried out by means of a trephine and scoop. The animals were then given a little more ether and the spinal cord was divided at the level of the axis vertebra; this left the animal with the lower part of the brain stem intact, but isolated from the spinal cord. All the operative procedures involved in the decerebration and spinalization were carried out with the cautery to minimize hæmorrhage. The animal was then allowed to recover for about 30 min.

The abdomen was opened and the pylorus was tied off; the œsophagus was opened in the neck and a stomach tube passed through the cardia and tied in position. The stomach was then washed out with saline and 60 c.c. of warm meat extract were introduced and the animal allowed a further 30 min. rest.

The cerebellum and bulb were now completely exposed by removing the overlying bone. The stump of the cervical cord was lifted up and gently mobilized until the bulbar roots of the vagus were clearly visible—the roots were easily found by following the spinal accessory nerve as it ran up along the lateral border of the bulb to join the lower vagal root. When the vagal roots were identified, two longitudinal cuts were made in the bulb, and its middle third removed—this allowed considerable freeing of the roots and avoided any risk of current spread to the opposite side. At this point, there was frequently troublesome oozing from the divided lateral sinus and bone, but this was controlled by light packing with wool. With this exposure it was possible to identify five bundles of

fibres going to form the vagus trunk—they could either be stimulated together or gently separated to permit of stimulation of individual bundles. There was no difficulty in identifying the spinal portion of the spinal accessory; it was much whiter and firmer than the vagal roots, which were pinkish, and soft in appearance, suggesting the absence of any quantity of supporting fibrous tissue.

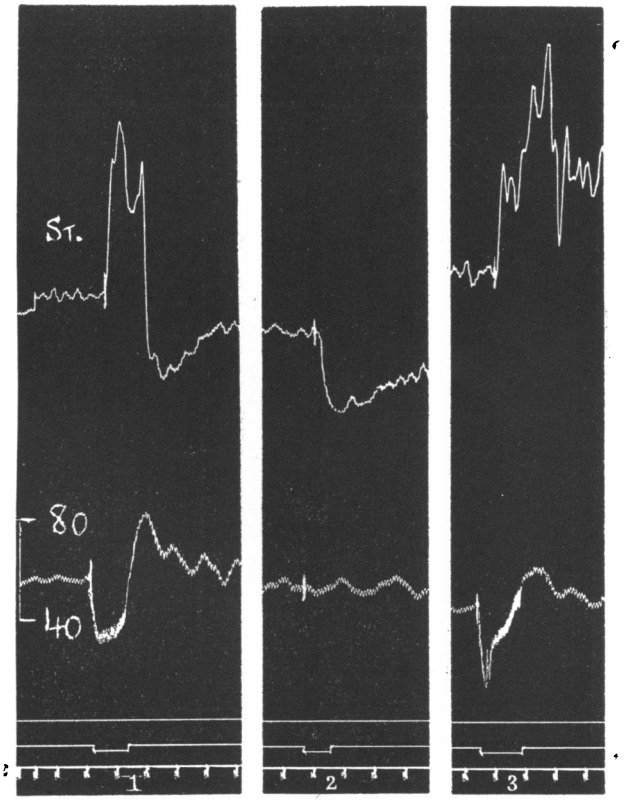

Fig. 2. Gastric response to stimulation of the intracranial roots of the vagus. Cat 12. (1) Motor response—right vagal roots; coil 12, freq. 10: (2) inhibitor response—right vagal roots; coil 10, freq. 60: (3) motor response—cervical trunk of right vagus; coil 8, freq. 20. Time intervals 30 sec.

When the bulbar exposure was completed, the stomach-tube was connected to a recording manometer and the right carotid was cannulated. Stimulation of the intracranial vagus was then carried out either upon the whole nerve or upon individual roots. Towards the end of the

experiment the cervical vagus on the same side was stimulated for comparison of results.

When the results of intracranial stimulation had been established the same procedure was repeated in animals in which the superior cervical ganglion had been removed.

Experimental results.

Stimulation of the intracranial vagus produced exactly similar effects upon the stomach as did stimulation of the cervical trunk—motor or

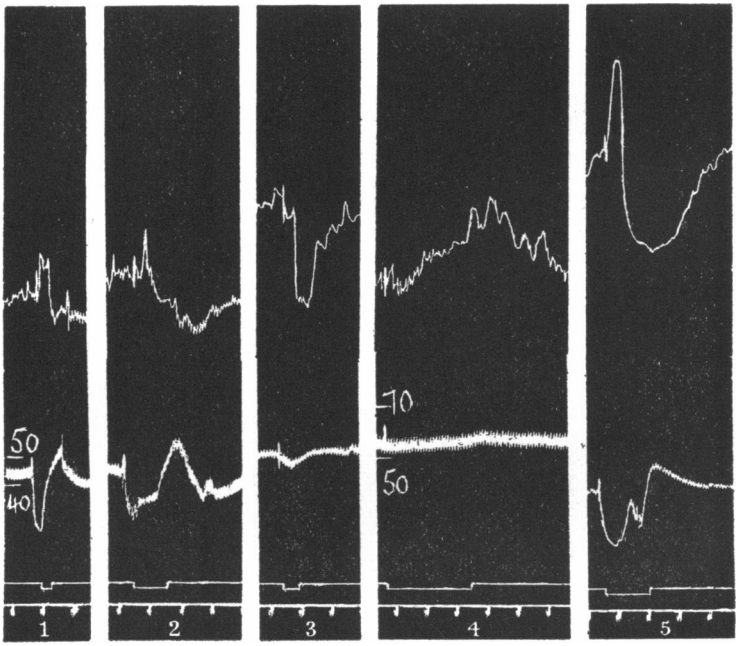

Fig. 3. Gastric response to stimulation of the intracranial roots of the right vagus after removal of the right superior cervical ganglion. (1–4) Stimulation of right vagal roots. Note the progressive diminution in the concomitant cardio-vascular response. (5) Stimulation of cervical trunk of right vagus after (4). Time intervals 30 sec.

inhibitor effects were recorded depending upon variations in the intensity of stimulation, tone of the stomach and the animal (Fig. 2). Selective stimulation of individual roots produced results which only varied quantitatively from those of the whole nerve, *i.e.* it was never possible to identify any particular bundle of fibres as being associated with any particular effect, motor or inhibitor.

The cardiac response to stimulation of the intracranial vagus showed very definite characteristics. At the commencement of the experiment even weak stimulation produced a marked and prolonged fall in blood pressure. As the experiment proceeded, the cardiac fibres became steadily less sensitive, till finally no cardiac effect could be elicited. That this loss of sensitivity of the cardiac fibres was purely a local condition was proved by the ease with which cardiac effects could be elicited via the cervical vagus long after the intracranial portion of the nerve was quite ineffective.

The disappearance of cardiac response did not affect the gastric response, showing that the latter was not dependent upon circulatory changes (Fig. 3).

The gastric response to stimulation of the intracranial roots of the vagus was quite unaffected by removal of the superior cervical ganglion (Fig. 3).

THE STELLATE GANGLION AND THE VAGUS.

Possibility C (Fig. 1) suggests that the gastric branches of the vagus might be derived from the stellate ganglion; if this were so, then removal of the stellate ganglion should render stimulation of the cervical portion of the vagus ineffective. Experiments were performed to test this point.

Method.

Decapitate cats were used. The stellate ganglion was exposed on one side and temporarily covered with a pad moistened with saline. The abdomen was then opened and the pylorus tied—a stomach tube was inserted through the œsophagus and connected to a water manometer. The right carotid was cannulated and the vagus nerve of the side corresponding to the exposed stellate ganglion was exposed and divided high up in the neck. Records of the gastric response to stimulation of the cervical vagus were then made over a wide range of intensities and rates. The stellate ganglion was then excised and a similar range of stimulation was repeated on the cervical trunk of the vagus.

Experimental results.

No difference could be detected between the gastric responses with and without the stellate ganglion.

THE ORIGIN OF THE NON-MYELINATED FIBRES OF THE VAGUS.

Having shown that the motor supply of non-medullated fibres to the stomach was not sympathetic in origin, it was necessary to enquire further into their mode of arrival. Two possibilities exist—they may

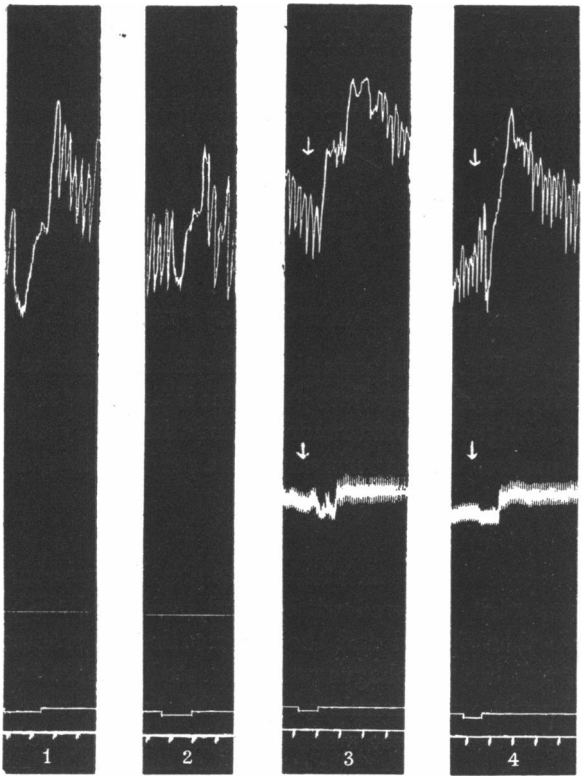

Fig. 4. Gastric response to stimulation of the cervical trunk of the vagus before and after removal of the corresponding stellate ganglion. Cat S 2. (1) Right vagus, ganglion intact; coil 4, freq. 24: (2) right vagus, ganglion removed; coil 4, freq. 24. Cat S 4. (3) Left vagus, ganglion intact; coil 3, freq. 60: (4) left vagus, ganglion removed; coil 3, freq. 60. Time intervals 30 sec.

represent connector neurones arising in the bulb and losing their medullary sheaths en route (Fig. 5, A), or they may represent a fresh neurone arising within one of the ganglia of the vagal trunk (Fig. 5, B). The former view was supported by Langley, Gad and Josephs [Schafer, 1900] but comparative studies suggested the latter explana-

tion to Gaskell. In the cat, longitudinal sections through the ganglion of the trunk show that the change to non-myelinated fibres takes place at that level.

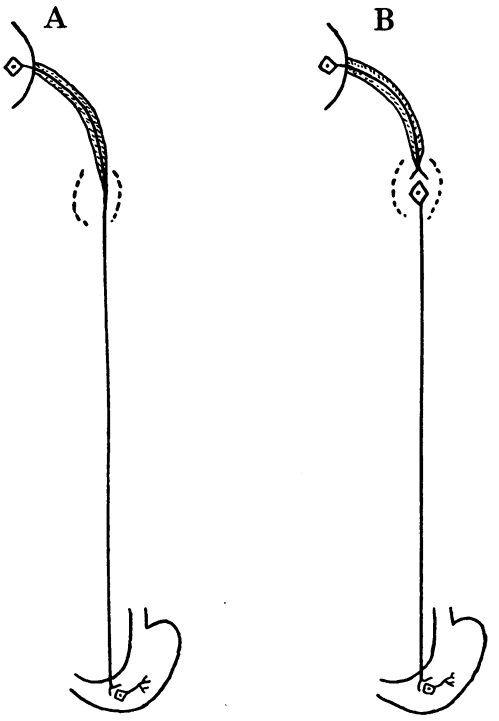

Fig. 5.

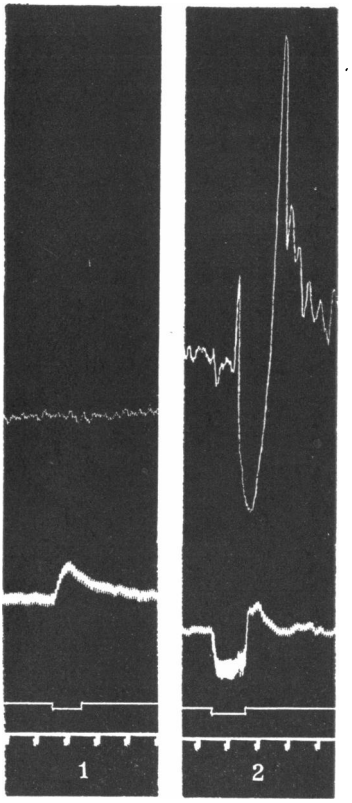

Fig. 6.

Fig. 5. Possible origins of the non-myelinated fibres. A, after Langley, Gad and Josephs; B, after Gaskell and Kiss.

Fig. 6. Effect of section and degeneration of vagus. Cat V 4. Right vagus divided above vagal ganglion 27 days previously. (1) Stimulation of cervical trunk of right vagus; coil 4, freq. 60: (2) stimulation of cervical trunk of left vagus; coil 4, freq. 60. Time intervals 30 sec.

Method.

In a series of cats the right vagus was divided immediately on emergence from the skull; the animals were allowed to recover and 10 to 28 days later the cervical vagus was stimulated and its effect on the stomach recorded.

Experimental results.

The cervical trunk of the vagus on the side of section was decidedly softer and more translucent than on the intact side. Stimulation of the sectioned vagus produced no motor or inhibitor effects upon the stomach in any way comparable to those obtained by stimulation of the intact nerve; the only change recorded was an occasional slight diminution in the amplitude of gastric contraction, most noticeable in those cases where a striking alteration in the circulation was produced: we are inclined to attribute this slight alteration in gastric response to the coincident vascular change.

The vascular response to stimulation of the sectioned vagus was in all cases a definite rise in blood-pressure; there was a latent period of 6 to 8 sec. and little, if any, acceleration of the heart rate. In all animals the right vagus was cut and the results correspond with the preponderance of pressor responses described by Morgan and Goland [1932] in the right vagus of the dog.

Discussion.

The fact that typical gastric responses to vagal stimulation can be obtained in cats deprived of the superior cervical ganglion or stellate ganglion indicates that the nervous pathway involved does not pass through these ganglia. The functional path of the gastric fibres of the vagus is confined to that nerve and derives no essential contribution from the sympathetic.

Section of the vagus trunk above the vagal ganglia results in the degeneration of the fibres which conduct the impulses to the stomach. Therefore no cell station is present in these ganglia, and it would appear that only one neurone is concerned in the conduction between the bulb and the stomach. Histological examination clearly indicates that the bulk of these fibres leave the medulla with medullary sheaths and reach the stomach as non-myelinated fibres; the site of loss of the myelin sheath is in the vicinity of the vagal ganglia.

As far as the efferent gastric fibres of the vagus are concerned, these experiments lend no support to Kiss's theories as to the relationship between the vagus and the sympathetic. Whatever may be the function of his "sympathetic" cells in the vagal ganglia, they are not the cells of origin of the non-myelinated efferent fibres to the stomach.

It has been suggested that the cervical sympathetic may send contributions via the superior cervical ganglion to the vagus, and that these

fibres are responsible for some of the gastric effects elicited by stimulation of the cervical vagus. These experiments show that stimulation of the intracranial roots of the vagus will produce the same range of gastric response as does the cervical vagus, a finding which disposes of any essential contributions from the cervical sympathetic.

Attention is drawn to the apparent difference between the cardiac and gastric fibres in the vagal roots. The cardiac branches are exceedingly sensitive at the commencement of the experiment, but this sensitivity rapidly diminishes till no cardiac effect can be elicited although the gastric response is undiminished (Fig. 3). This high degree of primary sensitivity with rapid subsequent fall suggests that the cardiac fibres have little protective covering in the vagal roots: thus they are readily accessible to stimulation but soon suffer damage thereby.

It has been suggested that the variations in gastric response to vagal stimulation are dependent upon or profoundly influenced by concomitant vascular changes. The fact that the intracranial cardiac roots become insensitive to stimulation, whilst the gastric fibres remain intact, enables one to separate circulatory and gastric effects; the response of the stomach appears to be little affected by the presence or absence of simultaneous circulatory changes (Fig. 3).

Stimulation of individual bundles of the vagal roots produced results which only varied quantitatively from those of the whole nerve. No evidence was obtained for the existence of special groups of motor or inhibitor fibres.

The arrangement of the gastric fibres of the vagus appear to conform to Langley's original conception of the parasympathetic system. A myelinated fibre leaves the bulb and passes via the vagus trunk to the stomach; in its course it loses its myelin sheath but has no cell station in the vagal ganglia.

Summary.

1. Stimulation of the bulbar roots of the vagus produces types of gastric response similar to those elicited from the cervical vagus.

2. Stimulation of individual roots produces no evidence of different types of nerve fibre responsible for motor or inhibitor effects on the stomach.

3. Vascular changes are not responsible for the various types of gastric response.

4. The functional path of the gastric fibres of the vagus is confined to that nerve, and has no essential connection with the sympathetic.

5. There is no cell station for the gastric fibres in the vagal ganglia. Myelinated fibres leave the bulb and pass directly to the stomach, losing their myelin sheaths en route.

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