ANGOR ANIMI, OR THE SENSE OF DYING*

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THE study of subjective symptoms is philosophically absorbing but beset with difficulties. It is a type of inquiry which falls particularly within the province of the physician, for if he lacks the accurate methods of the physiologist, the visual advantage of the radiologist, and the special opportunities of the surgeon, at least he has access to symptoms in greater variety and number than any of these, and a fuller insight into their qualities, their circumstances and antecedent causes. But our knowledge of the nature of symptoms remains peculiarly limited. Thus, when we consider the time and attention long given to the subject, it is remarkable that no settled opinion has yet been reached in regard to the seat and essential cause of such a clear-cut and classic phenomenon as anginal pain. It was in the process of pondering the anginal syndrome that it occurred to me to attempt a review of that other awe-inspiring component of some anginal attacks—I mean the *angor animi*, the *angor molestus* of Morgagne, the *meditatio mortis* of Seneca's physician, more familiarly known as "the sense of dying." What a strange force it is that can stir, and with such alarming intensity, a consciousness of the imminence of an event of which there can never have been any actual experience, and yet is so frequently inoperative when death is at hand. These circumstances alone should move our curiosity, but there are other aspects of the phenomenon which call for inquiry.

In considering how to approach the problem it is at once apparent that the symptom is not one which can be assessed by any precise physical method, and that an analysis of it must therefore be based on accumulated clinical observation and the method of induction. For this purpose I would propose to consider—(1) The nature of the symptom itself. (2) Its immediate symptomatic associations. (3) The conditions in which its occurrence has been noted. (4) Some other conditions (particularly those in which there is—or seems to be—a frequent threat of sudden dissolution) in which the symptom might be anticipated but is not recorded.

The symptom is sometimes described as "a fear of impending death". This is incorrect, for it is rather an actual "sense of dying". It is possible that we are confronted with the same

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symptom less frankly expressed when we are told by patients that they have experienced a feeling " as if something dreadful might happen ", or as if they " were fading into space ", but my references are to the more defined angor. Mackenzie (1923) does not appear to me to have drawn sufficient distinction between the associated sensations of depression or oppression and the true *angor animi*. It has generally been agreed by those who have studied it closely that the sensation is a physical one. Clifford Allbutt (1915) particularly refers to it as an " organic sensation ". Although it may engender them, it bears no relation to morbid dread, anxiety or panic. In those nervous diseases in which it occurs independently of any demonstrable organic change (such as the vaso-vagal attacks of Gowers), although the anxiety may to some extent be allayed, the symptom itself cannot be removed by reassurance or any other psycho-therapeutic measure. I cannot ascertain that it occurs in its major form as a symptom of psychological disease. In angina pectoris the feeling is apparently as transient as the pain. In vaso-vagal attacks it may, however, be much more prolonged, is of more frequent occurrence, and is, therefore, more easily observed. In early vaso-vagal attacks the feeling may be so strong that no persuasion will rid the patient's mind of the idea that the end is coming, if not in this in some subsequent attack. After many years he may realize that the threat is never consummated and yet the sensation in the seizures remains as real as ever. A patient of mine in the sixties who has had vaso-vagal attacks for thirty years now knows that she will not die in them, and yet this " sense of dying " is no whit diminished.

Some of the immediate associations of the symptom are common both to angina pectoris and vaso-vagal attacks. These include the fixed immobility, the unwillingness or inability to make even slight movements, and the sense of restricted breathing. In vaso-vagal attacks there is frequently coldness, pallor, palpitation and a quickened or sometimes a very slow pulse. In one of my cases a pulse-rate normally 70 to the minute frequently fell to 40 or lower, and in one attack, in which consciousness was lost, to 19 beats to the minute—presumably a vagal effect, as there was no clinical or electrocardiographic evidence of organic cardiovascular disease. The respirations may be shallow and quick and, perhaps as the result of this tachypnoea and the washing out of carbon dioxide from the lungs, tetany may develop. There is often a strange sense of unreality and consciousness may be lost for short or longer periods. There is frequent mention, but not as the leading symptom, of pain or discomfort between the praecordial area and the left

clavicle, and of a curious tugging pain up the left side of the neck. Collier (1928) mentions a falling blood-pressure, but in the aftermath of the attacks in the case with bradycardia to which I have referred I repeatedly felt a cord-like radial and observed a very considerable rise in pressure. Vaso-vagal attacks are followed by feelings of prostration lasting many hours or days, reminiscent of, but more pronounced than, the prostration after migrainous seizures.

I have encountered *angor animi* not only in angina pectoris, but also in one case each of angina abdominis and angina cruris. It would seem to be less common in angina pectoris than one would infer from the prominence given to it in the literature. Thus I only find it specifically described in 6 cases of 43 cases of which I possess full notes. But it seems possible that the "warning to immobility" may be regarded as a minor representation of the angor. Many patients, without confessing to the angor, feel that it would be impossible to walk another step and to survive. My impression is that *angor animi* is less frequent in coronary thrombosis than the angina of effort, and that it bears (as Clifford Allbutt (1915) observes) no relationship to the severity of the pain. I am doubtful also whether it has any bearing on the gravity of the event. It is much more frequent in vaso-vagal attacks, and was described as the most distressing symptom in 11 out of 13 cases. One of these I saw during her first attack and had under close observation afterwards. One I saw frequently just after attacks, and her husband, a doctor, clearly described the objective phenomena of many of them. One patient, a medical man, started his attacks with a sinking feeling in the epigastrium, such as one has in anticipation of some anxious interview, but this spread and became translated into a most intense sense of dying. He also described general shock-like sensations akin to those which follow a bad blow on the testicle. I have encountered the symptom in one case of spasmodic Raynaud's disease in which it did not appear unless the vascular manifestations were already present, thus suggesting (for those who hold that the cerebral arteries are under vasomotor control) a simultaneous spasm in vessels supplying the medulla oblongata, or alternatively (and as I should prefer to believe) a reflex effect from the contracted peripheral arteries. The symptom or something like it has been mentioned in epilepsy, but I have no note of a case. I have observed its occurrence in association with attacks of labyrinthine vertigo. Clifford Allbutt (1915) refers to a case described in Muller's *Physiology* in which a tumour was found involving the vagus nerve. Mott (1908) described *angor animi* or some closely similar sensory experience

in a case of cerebral tumour. Dr. C. P. Symonds has kindly given me access to his notes of a case of *angor animi* in a child in which there was a tumour involving the medulla oblongata. Thus Clifford Allbutt's statement that the symptom in its extremer degree is almost peculiar to angina pectoris requires modification.

To turn to conditions in which its presence might be presumed to be likely, but in which it does not occur, I need only mention that it is very rarely described as a precursor either of slow or rapid deaths from any cardiovascular cause, unless this be an association of angina pectoris. Mackenzie (1923) refers to the symptom in association with flutter and pulsus alternans, but the cases which he quotes were clearly cases of angina pectoris, although gripping or oppression replaced the more typical pain. In heart block with Stokes-Adams seizures, in which there is so nearly an arrest of the functions of life, Clifford Allbutt (1915) points out, and my experience agrees, that the symptom does not appear. It is not, to my knowledge, encountered in ordinary fainting attacks. I have already stated that it is not a symptom of anxiety-states, although in vaso-vagal cases the storms engender anxiety, and this, perhaps, in turn increases the liability to the attacks, as may also be noted with fainting and vertiginous episodes. In none of the conditions mentioned is the threat of dissolution fulfilled, excepting only angina pectoris and cerebral tumour, and in these there may be many threats before fulfilment.

What is the common factor in the varied conditions in which the symptom is recorded ? It must surely be a powerful medullary stimulus of some kind with physical effects, objective and subjective, so plainly and often simultaneously manifest in disturbances of the cardiac, respiratory and vaso-motor functions. Indeed the sense of dying might be described as *the aura of a nervous storm having its vortex in those medullary centres upon which the act of living depends*. In vaso-vagal attacks, as in epilepsy and migraine, the storm arrives we know not whence, although the liability to it depends in part, no doubt, upon constitutional factors and in part upon other circumstances which make for nervous instability. In angina pectoris, abdominis and cruris and in Raynaud's disease, is it too much to suppose that the symptom is due to an access of impulses arriving from the aorta or the coronary or peripheral arteries in moments of arterial stress or spasm and passing via the medullary nuclei ? Even slight trauma to peripheral arteries can produce very severe constitutional symptoms. Thus Bazett and McGlone (1928) experimenting with arterial puncture, found that a seemingly trivial stimulus which produced no untoward symptom when applied to skin, muscle, subcutaneous fat or veins, could cause coldness, faintness and even loss of consciousness in

the persons of the experimenters when applied to the arteries of their limbs.

There is a motor as well as a sensory part to this strange reflex. It includes the instant immobility which is so striking a feature in angina and which may also be present in vaso-vagal seizures, and the restricted breathing or " constriction " common to both conditions. It is interesting that Mackenzie particularly associated the angor with the sense of constriction. In John Hunter's case, although the sense of dying is not mentioned, it is recorded that he found himself not to be breathing in his initial attack and that he had to re-institute the act by will. In some vaso-vagal cases we obtain the same description, but without pain, of the chest being " drawn in " or " squeezed ", which has so frequently been recorded in angina. Although pallor and vomiting are common enough we do not find either the angor or the immobility of angina pectoris in other crises of severe pain such as biliary and renal colic, conditions in which primary arterial stress plays no part. In three of my 13 vaso-vagal cases (two personally witnessed) the fixed immobility was very striking. In 7 the restricted or difficult breathing was a prominent symptom.

In angina pectoris the biological purpose of this reflex is surely protective. The heart is in such bad case that to give it its best chance of weathering the storm, an arrest or control of all movements, including the respiratory movements, has become a necessity. In vertigo too there is clear need of immobility. I would again, therefore, suggest that the angor may be regarded as an extreme development of the more frequent but less definite " warning ", that is to say, as the sensory part of a strong immobilization reflex. The " freezing " of the terrified rabbit in the presence of a snake (to which my " vaso-vagal " patient with bradycardia once likened her experience) may possibly involve a similar mechanism, but the emotion of fear is here primary.

In describing the sense of dying as a part of a medullary storm and in insisting on its organic nature I am, of course, stating nothing new. I have merely attempted to assemble some evidence, to discuss the character and symptomatic associations of the phenomenon itself, and to indicate that it has certain motor accompaniments which can be observed both in organic and functional disease, and that it occurs in quite a considerable group of conditions other than angina pectoris. In those difficult borderline cases between anginal and vaso-vagal seizure, generally seen in women, the degree and duration of the angor and the character of its nervous sequels may be important differential features, and a fuller understanding of the symptom brings practical advantage.

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