cent. for the more cellular parts (margin and oral lobes). Teissier[8] found 96.5 per cent. water in *Chrysaora hyposcella.*

The foregoing figures apply to medusae taken in water of typical salinity or nearly so. In very brackish water, the water content may increase, at least in *Aurelia.* Thus Möbius[9] reported a water content of 97.9 and 97.94 per cent. for *Aurelia aurita* from the Bay of Kiel with a salinity of 17–18 parts per thousand. This result has recently been verified by Thill,[10] who found 98 per cent. water in *Aurelias* from a port in the Danish Wiek on the Baltic with a salinity of 7.3 per thousand.

During a stay at the Mt. Desert Island Biological Laboratory, Maine, the water content of several large *Aurelias* was determined. The salinity of the water around Mt. Desert Island is given by Bigelow[11] as 31.5–32.6 parts per thousand, a little less than that of the open Atlantic. The animals used were pulsating actively but were not anatomically perfect, all showing some marginal damage. It was not thought advisable to rinse them in fresh or distilled water because of a possible loss of salts; but No. 7 was thoroughly rinsed in fresh water as a check. The others were simply drained for a few minutes. The drained medusae were immediately placed in previously weighed glass or aluminum containers and subjected to dry heat in an electric oven at temperatures varying from 60 to 110° C. The drying was completed in a desiccator over concentrated sulfuric acid.

The data on the nine specimens used are given in Table 1.

TABLE 1

No.	Wet weight grams	Dry weight grams	Per cent. water
1	175.013	7.141	95.9
2	163.892	6.639	96.0
3	82.271	2.831	96.6
4	86.140	3.434	96.0
5	123.745	4.689	96.2
6	129.444	4.915	96.2
7	149.255	5.619	96.3
8	127.802	4.872	96.2
9	264.916	10.382	96.1

From these data and those in the literature it is evident that the water content of medusae in sea-water of typical salinity is 94–96.5 per cent.; in brackish water of less than half this salinity, the water content may rise to 98 per cent.

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[8] *Bull. Soc. Zool. France,* 57: 160.
[9] *Zool. Anz.,* 5: 586.
[10] *Zeit. wiss. Zool.,* 150: 51.
[11] *Bull. U. S. Bur. Fish.,* 40, pt. II: 813.

MEDICAL CLASSICS

PROFESSOR J. M. D. OLMSTED, of the University of California Medical School, contributed to the issue of SCIENCE for December 3 a statement concerning the second number of *Medical Classics,* which was published in October, 1936. This number was devoted to four of the important papers of Sir Charles Bell, those illustrating the original work on Bell's Law, Nerve, Palsy and Phenomenon. Dr. Olmsted overlooked three of the contributions of Bell, especially the "Idea of a New Anatomy of the Brain" which is one of the most important and difficult to obtain of any of Bell's writings. Of the five leading medical libraries in the United States only the Army Medical Library owns a copy.

Dr. Olmsted confines his review to a criticism of the use of Bell's paper, "On the Nerves," and states that the paper as published was not as given before the Royal Society on July 12, 1821. Bell's paper, as we reproduced it, was preceded by a photographic reproduction of the title page of the book from which it was taken, "The Nervous System of the Human Body," published in Washington in 1833. I believe no one would be in doubt as to the actual source of the paper.

When we consider that Magendie himself gives Bell credit for priority, I do not believe it adds to our stature to stress small differences in the texts of these two great men. The battle of Magendie *versus* Bell has raged for a hundred years, and even now there appear many advocates for either side. It is the intention of *Medical Classics* to convey knowledge as we find it in these famous papers and not necessarily to attempt to show old rivalries and differences of opinion as to whom proper credit is due. The modern physician, whom we are trying to interest in the broad aspects of medical history, does not like to be confused and irritated by petty controversies. Both Magendie and Bell were great men, and there is honor enough for both of them.

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POLLEN AND HAY FEVER

THE letter from Dr. Douglas H. Campbell published in SCIENCE for January 7, page 16, is an interesting example of the reappearance of ideas which at one time might have been regarded as plausible. However, a few minutes' inquiry should be sufficient to relegate this one to the shelf where it has lain undisturbed for some sixty years.

If the medical man of whom Dr. Campbell inquired had been an allergist, he would have referred him either to Dr. Charles Harrison Blackley's "Hay Fever," published by Baillière, Tindall and Cox, London, second

edition, 1880, pages 123 to 125, or to Dr. August A. Thommen's part 3 of "Asthma and Hay Fever in Theory and Practice," Charles C. Thomas, Springfield, 1931, pages 503 to 505. Dr. Campbell's theory is thus merely a repetition of Dr. Wilson's theory published with such confidence in 1873 and dealt with so ably by the pioneer in hay fever pollen research, Dr. Blackley.

A casual inquiry into the present-day methods of the preparation of pollen antigen would disclose the fact that the irritating element is obtainable in equal amounts from perfectly fresh pollen and from that which has been denatured and kept in storage for many years. It is not at all necessary for the pollen granule to touch the mucous membrane to cause the characteristic reaction. Instillation of active extracts of pollen will accomplish the same result with the same rapidity. Moreover, skin tests will show equally strong positive reactions, whether they be made with perfectly fresh pollen, with pollen which has been subjected to desiccation and kept for many years, with pollen which has been washed with ether or with extracts of pollen made by any of a score of methods, all of which would prevent any possibility of development of pollen tubes. Further research along this line would not seem to offer any worth-while possibilities.

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SPECIAL ARTICLES

THE INFLUENCE OF IODOACETIC ACID ON THE RESPIRATORY METABOLISM OF MAMMALIAN TISSUES[1]

IN 1929, Lundsgaard[2] observed that iodoacetate (IAA) inhibited the glycolytic mechanism, yet permitted muscular contraction to continue, a fundamental observation which caused a revolution in the concept of the chemistry of the contraction process in muscle. This work led to a further exploration of the nature of the IAA effect, with results that have been of considerable significance for the study of carbohydrate metabolism and the lactic acid cycle in muscle as well as in other tissues.

Although Lundsgaard[3] later pointed out that, with proper concentrations of IAA, the respiration of frog muscle and of yeast cells could be maintained at normal levels for some time, little was known of the nature of the foodstuffs oxidized under these conditions until Meyerhof and Boyland's[4] paper appeared, reporting that the R.Q. of such muscles, with 70 per cent. of the original respiration, was about 0.71, indicating virtual cessation of carbohydrate metabolism. When lactate was added to this preparation, respiration was stimulated, and the R.Q. rose to 0.95, showing oxidation of the added lactate. Krebs's[5] studies on the oxygen uptake of brain, testis and sarcoma under the influence of IAA furnished more support to the view that, in addition to inhibiting lactic acid formation, IAA prevented the oxidation of glucose, although it did not interfere with the oxidation of lactate. Quastel and Wheatley[6] also concluded that exposure of brain tissue to IAA rendered it unable to utilize glucose. These results have had a considerable influence in shaping the theories of the pathway of glucose oxidation in the cell, as well as on the interpretation of the mechanism generally known as the Pasteur-Meyerhof reaction. They would suggest that glucose or preformed carbohydrate may have to go through a lactic acid stage before it can be oxidized. They may also be interpreted as favoring the unitary theory of oxidation and fermentation which postulates a common initial aerobic and anaerobic pathway of carbohydrate degradation. When this pathway is interrupted by IAA, and no endogenous lactate is available for oxidation and resynthesis, oxidation of carbohydrate ceases. Finally, an inhibiting effect of IAA on the phosphorylating mechanism has been demonstrated by Lohmann.[7]

However, Stannard[8] and Saslow[9] have recently reported that non-nutrient R.Q.'s of normal and caffeinized frog muscle lay between 0.90 and 1.00, even though anaerobic glycolysis had been stopped by treatment with IAA and iodoacetamide. We have extended the investigation of the nature of the oxidative processes in IAA poisoning to mammalian tissues, using skeletal, smooth and cardiac muscle, as well as brain, from normal cats and dogs. The brain cortex, freed as much as possible of white matter, was minced. The cardiac muscle was cut into thin slices. Smooth muscle was obtained from the small intestine by a method (unpublished) developed by Eugene Cohen in this laboratory. The skeletal muscle was prepared by careful dissection of bundles of muscle fibers sufficiently thin for microrespiration studies. Prior to study in the Warburg apparatus, the tissues were aerated in Ringer solution, to permit the loss by diffusion of any lactate which

[1] From the New York Hospital and the Departments of Medicine and Physiology of the Cornell University Medical College, New York City. Supported in part by a grant from the Committee on Research in Endocrinology of the National Research Council.

[2] E. Lundsgaard, *Biochem. Zeit.*, 217: 162, 1930.

[3] E. Lundsgaard, *Biochem. Zeit.*, 220: 8, 1930.

[4] O. Meyerhof and E. Boyland, *Biochem. Zeit.*, 237: 406, 1931.

[5] H. A. Krebs, *Biochem. Zeit.*, 234: 278, 1931.

[6] J. H. Quastel and A. H. M. Wheatley, *Biochem. Jour.*, 26: 725, 1932.

[7] K. Lohmann, *Biochem. Zeit.*, 236: 444, 1931.

[8] J. N. Stannard, *Amer. Jour. Physiol.*, 119: 408, 1937.

[9] G. Saslow, *Jour. Cell. Comp. Physiol.*, 10: 385, 1937.