ill among the local population"; and later "that the epidemic occurred in the community rather than the building; a class of girls at the public bath was affected and others felt ill when walking home." In fact the girls were taken ill inside and outside the school community and both when in company and when alone. In the same paragraph it is stated "no poison gas could be found despite careful detective work, nor has there been any trouble in the building since." In fact it was found on investigation that an iron foundry adjoining the school had lost a quantity of aromatic hydrocarbon. It was shown that it was possible for this to seep into the school precincts. More convincing was the fact that a second concern, a small wood treatment plant, had premises abutting into the school precincts. It was found that wood shavings impregnated with preservative, improperly burnt in a stove, caused aromatic hydrocarbons to be distilled off into the air. The small chimney was adjacent to the school wall. A small plastic tube passed up to its outlet enabled flue gases to be pumped through a reagent. The test was strongly positive for aromatic hydrocarbons. Charred wood shavings were found in the school playground. The caretaker had not seen them there before. Fresh shavings when burnt in the laboratory produced a strong positive reaction for aromatic hydrocarbons. The architectural layout of the buildings and prevailing climatic conditions at the time made it possible for a known temperature inversion to cause pooling of warmer air on the floor of the playground. Symptoms exhibited by a number of the children were similar to those experienced in aromatic hydrocarbon poisoning. No further cases occurred when these two processes were stopped from operating in the way that they had been. It is noted that in Drs. Moss and McEvedy's article it is stated that when the school finally reassembled on the following Monday nearly 60 girls complained of a recrudescence of symptoms, but none required admission. In actual fact the children returned to school on day 20 and not on day 12, as stated in the article, and, although a retrospective questionary suggested that 60 girls complained of recrudescence of a symptom on the date of their return, there was no evidence of this at the time, although a nurse and doctor were present in the school.

In conclusion we would like to state that:

(*a*) This outbreak should not be confused with outbreaks of winter vomiting syndrome, or gastrointestinal infectious conditions described in the literature.

(*b*) The episode was, in our opinion, due to complex causes of a sophisticated nature, and could not be attributed to any single agency.

(*c*) The suggestion that the affected schoolgirls were of such a constitutional nature as to be particularly psychologically susceptible in contradistinction to their fellows in other schools, and so manifested simple mass hysteria, is not thought to be valid.

(*d*) Of the multifactorial stresses to which these girls were subjected, the one of toxic inhalation carries with it too many positive coincidental features to be ignored completely.

(*e*) Finally, after very careful assessment of all the circumstances and researches it would be most reasonable to keep a completely open mind on the aetiology of the outbreak. We emphasize this latter point because we feel that the danger of accepting Drs. Moss and McEvedy's criteria for diagnosing hysteria in a mass outbreak might result in a previously known or unknown infectious or toxic hazard to be overlooked in some future outbreak. That many girls in the Blackburn outbreak showed signs of hysteria has never been doubted, but to dismiss them all as being hysterical is unwise.

It might be salutary to quote from the *Annual Report of the Chief Medical Officer of the Ministry of Health for the Year 1965* (page 62)[2] regarding, in this case, winter and epidemic vomiting.

"It has been common practice to invoke 'hysteria' as a cause of outbreaks of this type of illness occurring in schools. While psychological factors undoubtedly play a part in some cases—the nature of the illness, its abrupt onset and high attack rate may well give rise to symptoms of a similar nature in certain suggestible children—the pattern and epidemiology of the illness would, in most instances, point to an infective agent as the causative factor.

"Despite careful investigation of a number of outbreaks no causative organism has, so far, been demonstrated. The Public Health Laboratory Service has set up a working party to inquire into non-bacterial gastroenteritis and it is investigating material from a series of outbreaks."

—We are, etc.,

Blackburn. JOHN ARDLEY, Medical Officer of Health.

Preston. PETER GRIME, Deputy Medical Officer of Health.

REFERENCES

1 *An Account of the St. Hilda's School Outbreak, Blackburn, 1965*, 1966. Blackburn County Borough Council.

2 *The Annual Report of the Chief Medical Officer of the Ministry of Health for the Year 1965*] On the State of the Public Health. H.M.S.O.

Bilateral Rupture of Quadriceps Tendons

SIR,—The excellent article by Mr. V. D. Dalal and Dr. D. E. Whittam (3 December 1966, p. 1370) reporting a case of spontaneous simultaneous rupture of bilateral quadriceps tendons performs a service by reviewing previous cases. I would like to add a case reported by Adicoff and myself.[1]

In our case the quadriceps tendons had presumably been weakened by calcific deposits due to hyperparathyroidism. There was also rupture of one triceps tendon in the same injury. After prolonged treatment of a peptic ulcer, and of the hyperparathyroidism, the quadriceps tendons were finally repaired successfully nearly eight months after the original injury.—I am, etc.,

Minneapolis, Minnesota, U.S.A. FRANK S. PRESTON.

REFERENCE

1 Preston, F. S., and Adicoff, A., *New Engl. J. Med.*, 1962, **266**, 968.

King Cholera

SIR,—Dr. A. B. Christie's review of *King Cholera* (28 January, p. 228) reminds me that when that fine upstanding "Adonis of medicine," discoverer of the staphylococcus and former president of the B.M.A., the late Sir Alexander Ogston, was appointed surgeon to the late Queen Victoria he went to Balmoral Castle to write his name in the caller's book and was unexpectedly ushered into the royal presence. He was immediately reduced to a state of knee-knocking stammering impotence by that formidable old lady's first question: "Dr. Ogston, has the cholera come to Aberdeen yet and is Aberdeen fully prepared?"

With cholera but three jet-hours away and with the tropics on our doorstep, the royal question would not be out of place today.—I am, etc.,

Ewell, Surrey. GEORGE R. MCROBERT.

Atrial Function

SIR,—I read the paper by Dr. W. J. Gillespie and his colleagues (14 January, p. 75) with great interest, as much for the discussion as for the original observations. Nevertheless, I feel their general conclusion, that atrial contraction has a greater augmentative effect on the cardiac output in a healthy heart than in a diseased one, may be invalid for the following reasons.

The paper by Kory and Meneely, to which they refer, is misquoted.[1] Kory and Meneely found an average rise in cardiac output of 43% in six patients who had been in heart failure; two patients who were lone fibrillators, with otherwise normal hearts, showed no such rise.

It is true that most workers have found a rise in cardiac output after reversion to sinus rhythm by quinidine, whereas others, using electrical defibrillation, have failed to demonstrate such a marked effect. This may be because those who used quinidine measured the cardiac output some days after reversion to sinus rhythm, while those employing countershock have often measured it immediately after reversion, when mechanical function of the atrium is impaired.

In hypertrophic obstructive cardiomyopathy, when hypertrophy of the ventricle is extreme, atrial contraction may contribute a major fraction of ventricular filling and the same may well apply to other diseases such as pulmonary stenosis.

The quantity of blood transferred forward into the ventricle by atrial contraction will be determined by the relative compliances of the ventricle and the venous system. These depend on structure, transmural pressure, and nervous and hormonal influences. These factors are so complex that it is probably futile to try to predict what the net result will be in any given haemodynamic situation. This complexity may also explain the variable results of changes in rhythm on the circulation.—I am, etc.,

Sheffield. R. E. NAGLE.

REFERENCE

1 Kory, R. C., and Meneely, G. R., *J. Clin. Invest.*, 1951, **30**, 653.

SIR,—Dr. W. J. Gillespie and co-workers (14 January, p. 75) have obviously performed important and interesting work using a method which is itself a technical triumph. I therefore hope I shall not appear a detractor in suggesting a modification of their conclusions with regard to their group B (of patients with symptoms). From the clinical histories of Cases 6 to 11 there was raised left atrial pressure, as indicated by breathlessness. This, as the authors explain, can lead to a reduced right atrial contribution to right ventricular output due to the left atrium acting in an opposing fashion. Why, then, the need to suppose a favourable response is achieved only when the ventricular myocardium is healthy? Surely it is a normal atrial pressure (and larger pulmonary venous compliance) which matters. Examina-