Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

CORONAVIRUS AND GASTROENTERITIS IN FOALS

SIR,—In an editorial [1] you suggested that coronavirus was involved in human gastroenteritis. We later reported that calf-diarrhœa coronavirus serologically cross-reacted with antibodies in human sera.[2] The existence of an enteric coronavirus in man is supported by electron microscopic observation of such particles in fæcal specimens from an outbreak of human gastroenteritis.[3]

We should like to expand the list of species in which coronavirus may cause gastroenteritis. Fæcal specimens from three foals that died or were killed in the acute stage of disease were examined by electron microscopy. All three foals were found to contain typical coronavirus particles.

The three specimens originated from an endemic area in the U.S. where 40 or more cases of foal diarrhœa have recently been reported. The disease is characterised by profuse watery diarrhœa, fever, extensive lymphatic involvement, and a high rate of mortality despite treatment.

We have also tested 65 equine sera for serum-neutralising antibody against calf-diarrhœa coronavirus. Titres ranged from nil to greater than 181. We believe these serological data support our electron microscopic observation of virus particles from foals, suggesting the existence of an enteric coronavirus for the horse in addition to man, swine, and calves (calf-diarrhœa agent).

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HYPO-OSMOLALITY IN BEER DRINKERS

SIR,—Dr Phillips and Dr Pain (Sept. 6, p. 455) have drawn attention to the fact that in the calculation of urinary osmolality (Aug. 9, p. 245, table II) we have forgotten the anions. This we regret very much. If this correction is made then the supposed urinary osmolality in beer drinkers would be approximately 40 mosmol at diureses of about 5 litres.

Dr Phillips and Dr Pain remark that in 2 patients we found urinary osmolality of 69 and 79 mosmol. However, these urine collections began on the first or second day after admission and thus the patients had already been eating normal food.

They also suggest that our low serum-sodium values could be misleading because of hyperlipidæmia. This was excluded in 2 of our patients. Furthermore, it must be a very heavy hyperlipidæmia to cause "pseudohyponatræmia" to the extent that we found.

Dr Phillips and Dr Pain think that inappropriate secretion of antidiuretic hormone is not excluded. To this, we can say that in 2 of our patients we measured plasma-volume and found it slightly reduced.

We wish to thank Professor Demanet (Sept. 6, p. 455) and Dr Banks and Dr Lecky (Sept. 20, p. 559) for their comments.

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A PAMPHLET TO ANSWER PATIENTS' QUESTIONS

SIR,—We appreciated the interest shown in our paper by Dr Reedy (Sept. 27, p.604), although we believe the problems of communications between doctors or nurses and patients are very different from those between professions or members of the same profession. As we indicated, the final version of the pamphlet was prepared after many interviews and discussions with patients before, and it varying intervals after, the operation. The patients, of different classes and nationalities, were unanimous in welcoming the pamphlet and difficulties or omissions which they raised were taken into account in the next revision. Working-class women certainly understood it and did not appear to find any undue problems.

With regard to the assumptions about the behaviour of friends and relatives, gynaecologists and obstetricians, in the course of clinical work, are repeatedly made aware of the fact that patients do receive inaccurate and misleading information in this way. However, confirmation was provided by patients who, after reading it spontaneously and specifically, welcomed the pamphlet, among other reasons, because it avoided this particular problem. We should add that we found that the pamphlets were borrowed and read by other patients in the ward and many of those admitted for hysterectomy under other firms complained that they had not received copies. We have accumulated considerable information from our interviews which we intend to publish and our study continues.

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ACUTE HÆMORRHAGIC CONJUNCTIVITIS DURING AN EPIDEMIC OUTBREAK OF ADENOVIRUS-TYPE-4 INFECTION

SIR,—A new entity, acute hæmorrhagic conjunctivitis (A.H.C.), has been described in several countries since 1969.[1] The ætiological agent of this condition was first described by Kono et al.[2] and was later classified as enterovirus type 70.[3] Epidemics of A.H.C. have never been reported in Italy; therefore the observation in Rome of several cases of an eye infection characterised by subconjunctival hæmorrhage prompted a virological study to determine whether an enterovirus was the cause. 14 adult patients with A.H.C. and 46 with follicular conjunctivitis (F.C.) sought medical care at the outpatient service of the Ospedale Oftalmico Regionale from the beginning of March up to the end of May, 1974. The number of F.C. cases was at the same level for this period of the year as in the past, while the incidence of A.H.C. appeared to be unusually high. The symptoms of patients with A.H.C. were ocular pain, sudden swelling, congestion, and watering: the subconjunctival hæmorrhage varied from petechiæ to small blotches covering the whole bulbar conjunctiva. The cornea was not involved, hæmorrhagic symptoms generally subsided within a week, and recovery was complete within the follow-

VIROLOGICAL STUDY OF 8 CASES OF ACUTE HÆMORRHAGIC CONJUNCTIVITIS

Patient No.	Age (yr.)	Conjunctival swab (virus isolated)	Serum-neutralising-antibody titres*			
			Adenovirus type 4		Enterovirus type 70	
			Early	Late	Early	Late
1	38	Adenovirus 4	1/8	1/128	< 1/8	< 1/8
2	19	Negative	< 1/8	1/64	< 1/8	< 1/8
3	55	,,	1/32	> 1/128	1/8	1/16
4	59	,,	1/32	> 1/128	< 1/8	< 1/8
5	26	,,	—	—	—	—
6	30	Adenovirus 4	—	—	—	—
7	27	,, 4	1/8	1/32	< 1/8	< 1/8
8	34	,, 4	—	—	—	—

* Titres were determined against 100 T.C.D.$_{50}$ of virus.

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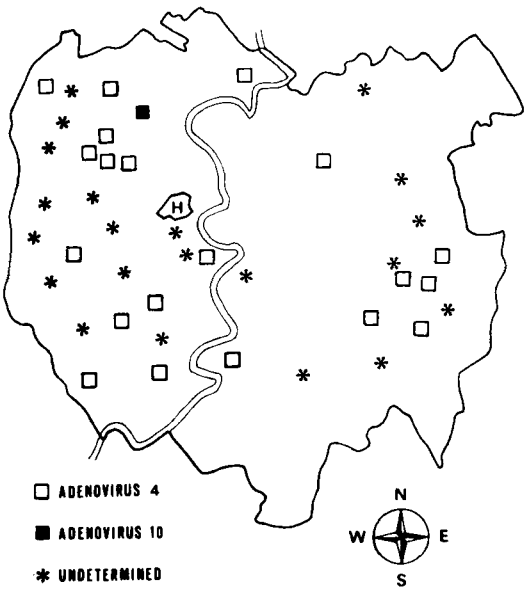

Ætiology and distribution in the metropolitan area of Rome of the 42 cases of conjunctivitis.
H=Ospedale Oftalmico Regionale.

ing two weeks. F.C. cases showed the typical pattern of the disease.

Conjunctival and pharyngeal swabs were obtained from 8 cases of A.H.C. and 34 of F.C. Virus isolation was attempted by inoculation of each sample in hela, primary monkey-kidney (M.K.), and human embryo kidney (H.E.K.) cell-cultures. A blind passage in cell-culture was routinely performed for every sample not showing cytopathic effect (C.P.E.) after the first inoculation. Cell damage indicative of picornavirus C.P.E. was not observed. 14 strains of adenovirus type 4 and 1 strain of adenovirus type 10 were isolated in H.E.K. cell-culture from the conjunctival samples. Reference sera for virus identification were obtained from N.I.H., Bethesda, Maryland, U.S.A. Hela and M.K. cell-cultures were less suitable for the adenovirus isolation. Viruses were not recovered from pharyngeal samples. Acute and convalescent (paired) sera, collected from 16 of the patients, were tested by means of neutralisation test (N.T.) in H.E.K. cell-culture against 100 $T.C.D._{50}$ of Japanese acute hæmorrhagic conjunctivitis (enterovirus type 70, A.H.C. J 670/71 strain) virus, kindly supplied by Dr R. Kono (Central Virus Diagnostic Laboratory, National Institute of Health, Tokyo), and of adenovirus type 4. N.T. results did not demonstrate any ætiological relationship between enterovirus type 70 and the conjunctivitis under study, since the majority of the patients did not have antibody against the virus or showed very low positive titres without significant variations in paired sera. 6 additional cases of adenovirus-type-4 infection were detected by antibody titrations: therefore a total of 20 cases of adenovirus-type-4 and 1 case of adenovirus-type-10 infection were demonstrated by virus isolation and/or serum N.T. amongst the 42 patients under study.

The results obtained in the A.H.C. cases studied are summarised in the accompanying table. These data prove that A.H.C. can be caused not only by an enterovirus [4] but occasionally by an adenovirus.

The relevant incidence of conjunctival hæmorrhagic manifestation as well as the widespread diffusion of the infection in the metropolitan area of Rome (see figure) appear to characterise this adenovirus-type-4 epidemic. The outbreak lasted for three months, only adult cases were found to be affected, and the epidemiological survey failed to demonstrate the usual sources of adenoviral contagion (i.e., iatrogenic transmission, community living and/or work, swimming-pools, &c.) as the origin of the cases studied.

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LATE NEOPLASIA AFTER POLIOMYELITIS

SIR,—We report here two cases of spinal-cord tumours 30 years after childhood poliomyelitis. A 34-year-old woman at the age of 4 had had poliomyelitis affecting the lower limbs. Thirty years later she had a retroperitoneal neurogenic (Schwann cell) sarcoma excised. She died shortly thereafter; necropsy was not performed. In the second patient, a 52-year-old woman, poliomyelitis occurred at age 4 with subsequent partial paralysis of the lower limbs. At the age of 30 she experienced sudden onset of paraplegia, and at operation a meningioma at D10 level was removed. The motor paralysis remained but sensation and sphincter control returned to normal.

No published reports have been found relating poliomyelitis to tumours. In studies showing the late progression of poliomyelitis, Mulder et al.[1] and Andersen et. al.[2] showed an association between poliomyelitis and late onset of neurological and functional changes (amyotrophic-lateral-sclerosis-like picture). Kayser-Gotchalin [3] reported late muscle atrophy many years after poliomyelitis. Many publications refer to the late picture as "A.L.S." rather than poliomyelitis. It is difficult to conceive of tumours causing a poliomyelitis-like picture thirty years before their detection, although spinal-cord tumours have been reported after aseptic meningitis.[4]

Hopkins and Shield [5] described immunological aspects of poliomyelitis. In children with bronchial asthma, a picture of poliomyelitis has appeared after an acute attack of asthma ; the neurological picture here could be due to a hypersensitivity to drugs, for no pathogen was isolated. Pietsch and Morris [6] found an association between HL-A (especially HL-A 3 and 7) and poliomyelitis: "this association might represent an association between HL-A and immune response gene determining an immune response to the virus or a product of viral infection of the central nervous system." Such an association was found in multiple sclerosis, and perhaps the ætiology is similar. The possibility of an immune response to poliovirus, with a neoplastic reaction many years after infection, must be considered.

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