philosophical education during the training of junior psychiatrists. Inevitably, I came across Simpson's comments (*Journal,* December 1988, **153,** 846) on Benjamin's letter, and I would like to express my concern and disagreement with Simpson, on which philosophy to choose to enlighten the dark and sometimes erratic future of the young trainees.

Simpson considers dialectical materialism (DM) as the ultimate pathway that will lead us to clarify the irritating mind–body problem. Moreover, he implies that DM is a philosophy that "in a scientific manner" will help us to explain the problem.

Is there enough room in the decade of the post-anarchist epistemology for DM? Not only did Sir Karl Popper critically attack the scientific basis of DM but Paul K. Feyerabend went even beyond demolishing the sophisticated apparatus built by Popper.

Not long ago, Mario Bunge, Professor of Philosophy of Science at McGill University, published *Materialism and Science.* He concluded that the only philosophical materialism valid was that based on physics, and he, therefore, introduced us to the fascinating territory of quantum mechanics, matter, and reality. Nevertheless, he dismissed DM on the grounds of lack of an adequate scientific basis and an excess of 19th-century rhetoric. Furthermore, he warned us of the danger of omnipotent ideologies, e.g. DM, explaining away every form of human activity.

Having said this, it appears quite clear that any philosophy that wants to address the question of what matter is needs the aid of the new physics. Therefore, to study that complex arrangement of matter called man, we need a non-linear science that should not enter into contradiction with the principles formulated by physics. This science may well be evolutionary biology. Hence, any philosophy must be in accordance with this particular approach to reality.

I therefore think that what should be offered to a psychiatric trainee immersed in a confused materialist paradigm is a sensible blend of history of science and a thorough study of the evolution of the ideas on human nature. It would also be necessary to exploit current contradictions like the paradoxical shift from a materialist paradigm (brain) to a non-materialist one (mind) that is usual in psychiatric clinical practice.

In summary, there are certain properties of matter explained by physics; there are some emergent properties that are not explained by physics but that are not in contradiction with it, hence the need for biology and thus neurosciences. The mind–body duality is a pseudoproblem and what is needed is a paradigm, not to bridge the divide between mind and body, but to explain how matter organises itself to generate that complex set of functions with the impressive name of soul, spirit, and perhaps mind.

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Hyperventilation Causing Asthma

SIR: Hibbert & Pilsbury's paper (*Journal,* November 1988, **153,** 687–689) is interesting, but the important thing to emphasise is that it is possible to make an assessment of the situation by careful attention to the clinical history and physical signs without the use of any special technology. The inter-relationship between hyperventilation and asthma is well known in the chest world. The fact that hyperventilation may proceed to asthma or that asthma may be complicated by hyperventilation or that the two may be closely interwoven has been known to me at least since 1960, when as far as I can remember I was introduced to the idea by Dr J. G. Scadding, who was then Dean of the Institute of Diseases of the Chest at the Brompton Hospital where I was an honorary consultant. It is referred to in a number of reviews, and in particular in my chapter in the Brompton textbook on asthma (Cohen *et al,* 1983). Asthma is a common disorder, and asthmatic patients may be anxious for the same reasons that make other people anxious. If an anxious asthmatic hyperventilates, there is a risk that his disorder will be misdiagnosed as asthma and if that happens and he is treated, say, with steroids, treatment will be ineffective. It is therefore very important to make an assessment whether one or other disorder is present or, much more frequently, since they are both commonly present simultaneously, which is the more important at the particular time and therefore which line of treatment, anti-asthmatic, anti-anxiety, or both, would be the most effective. This assessment can be made clinically as the following two patients illustrate.

Case (1). A married male teacher aged 34 seen in 1982, referred by Dr Duncan Geddes from The London Chest Hospital for an exacerbation of asthma which had prevented him from working for some weeks. He had suffered from asthma since the age of 13, and his presenting complaint was "I think my nerves are precipitating my asthmatic attacks." At the age of 18 he had been treated by a psychiatrist for an anxiety state, and he recalled an attack in which he said he felt "tense, worried, tension in the neck, I went rigid, heart pounding, throat dry, gasping but not

wheezing." Since then he had had many such attacks, some of which continued into typical asthmatic attacks, and he distinguished these quite clearly from his typical attacks of asthma which began with wheezing and in which there were few obvious symptoms of anxiety. The distinction between the two types of attacks had not previously been made by his doctors. Treatment directed towards relieving his anxiety by dealing with certain family problems together with instruction in relaxation led to complete relief quite quickly, and he returned to work.

Case (2). A male plasterer aged 28 was referred by Dr Joy Edelman of Barking Hospital in 1988. She had noticed that his asthma had persisted despite increasing doses of steroids, and that he could change from being completely free of wheezing one minute to having a wheeze quite audible without the stethoscope the next. She noted that the wheeze was always localised in the large airways, with good-quality breath sounds throughout the rest of the lung fields. He also complained of a sensation of a lump in the throat. In my interview with him he described how the lump in the throat appeared in only *some* of his episodes of breathlessness, and in these he experienced tingling in his fingers and around his mouth. These were evidently episodes of panic-over breathing, with incipient tetany. He had himself learnt that in these attacks he could relieve his symptoms by calming himself down. (The production of a wheeze in the large airways by compression of the chest by the voluntary muscles in asthmatics was described by Dekker & Groen (1957)).

In Case (2) Dr Edelman had thus made the distinction between the two types of attacks of breathlessness based on physical signs, and I had made it based on symptoms. Both methods are obviously useful.

Treatment of these patients depends on a careful assessment of the pathogenesis of the symptoms and this requires considerable attention to the detail of the history and physical signs. Where hyperventilation is important simple relaxation, concentrating on the chest, can be done very simply. In most patients a combination of specific anti-asthmatic treatment and treatment directed at the anxiety is probably necessary, and asthmatics are the ideal patients for collaborative management by physician and psychiatrist as first described by Maimonides in the 12th century (see Muntner, 1963).

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Malignant hyperpyrexia syndrome in combined treatment

SIR: We are writing to add another report to that of Brennan *et al* (*Journal,* April 1988, **152,** 578–579) in which a fatal malignant hyperpyrexia was associated with the combined medication of L-tryptophan, lithium, and phenelzine. This appears particularly important in view of the fact that both the aetiological and the pathogenetic factors involved in the neuroleptic malignant syndrome (NMS) are still ill understood (Henderson & Wooten, 1981; Gribb, 1988).

Case report. At the time of her death, this 48-year-old woman had been treated for depression since June 1986. Various treatment regimes, including dothiepin (225 mg *nocte*), mianserin (90 mg/day), flupenthixol (1 mg/day) and ECT (8 sessions), were administered unsuccessfully. In October 1986, she started on phenelzine (up to 60 mg/day) for four weeks, changing to tranylcypramine (10 mg b.d.) for the following six weeks. Both of which made no improvement to her condition, and were abandoned. Her mental state gradually showed evidence of more pronounced agitational symptoms, as well as obsessional and phobic traits. In November 1987 she took an overdose of tricyclic antidepressants, tranquillisers, and paracetamol.

In February 1988 she was started on chlorpromazine (up to 325 mg/day) and trazodone (100 mg t.d.s.), and a month later lithium and L-tryptophan were added (800 mg *nocte* and 3 gm/day respectively). This combination of drugs appeared to have made the first noticeable change in her depressed and agitated state, so that trazodone could be discontinued by April 1988. After four months it was decided to increase the chlorpromazine to 600 mg daily and on 21 July 1988 she was started on phenelzine (45 mg/day); other medication at the time remained unchanged, with lithium (800 mg *nocte*) and L-tryptophan (6 mg/day). Improvement of her symptoms allowed a reduction of the neuroleptic medication to 300 mg daily over four weeks, beginning in mid-July 1988. At that time the patient was well enough to go on a holiday to her sister's. During the journey she began to feel unwell and presented at a casualty department 36 hours later, having been found lying on the floor in an incontinent state. On admission she was incoherent in her speech, with marked reduction of her consciousness. Temperature was 38.8°C initially; muscular rigidity with cog-wheel phenomenon, as well as nystagmus, hyperreflexia and upgoing plantars, were present.

A tentative diagnosis of NMS was made and procyclidine (10 mg) was given. Blood samples for urea and electrolytes, full blood and differential count, blood cultures, MSU and lithium levels as well as random blood sugar proved normal. A CXR was clear and the CSF showed no cells or protein. Creatinine kinase was elevated at 408 IU. Within one hour of admission her temperature reached 42°C, at which point prophylactic antibiotics were started via a CVP line inserted in the meantime. Respiratory and cardiac arrest ensued shortly afterwards, and despite pacing and resuscitatory measures for over one hour, the patient died on 27 August 1988.